The Formation and Malleability of Dietary Habits: A Field Experiment with Low Income Families^{*}

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Abstract

We conduct a field experiment to evaluate the short and long term effects of two interventions targeting the dietary habits of low income families with young children. In one treatment, families received food groceries at home for free for twelve weeks and were asked to prepare five specific healthy meals per week. In the other treatment, families were simply asked to reduce snacking and eat at regular times, also for twelve weeks. We evaluate the impact of the interventions on diet and BMI over the course of three years. We find evidence that children's BMI distribution shifted significantly relative to the control group, i.e. they became relatively "thinner". This effect persists three years after the intervention for the first intervention, but fades away for the second. We find evidence that children reduced their sugar intake following both treatments. However, we find little evidence that their preferences changed in favor of healthier foods. A possible explanation is that children were restricted access to foods high in sugar in the treated groups. Parents, on the other hand, do not appear to have changed their diet as a result of the interventions, neither in the short run nor in the longer run.

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I Introduction

Poor diet is a major issue in most developed and developing countries. It is estimated that 11.3 million deaths per year globally can be attributed to a poor diet (Global Disease Risk 2013 Collaborators). While there are many policies targeting diet such as information campaigns and, more recently, a series of interventions based on insights from behavioural economics¹, most studies show that long term changes are difficult to achieve. This is one reason why many interventions target children, presumably at a stage where dietary habits are still forming.²

This paper evaluates two interventions targeted at young children and their families. The goal is to gauge the extent to which there is scope to intervene in the formation of dietary habits, and once these habits are formed to examine how malleable they are. We focus on low income families (with children aged between 2 and 6) because there is well documented evidence of a strong socio-economic gradient in chronic diseases and in obesity. Low socio-economic status (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). Income has also been shown to be correlated with nutritional deficiencies³.

The first intervention (*Meal treatment*) is strong and invasive. Families receive food and recipes at home to cook 5 meals a week over a period of twelve weeks. The recipes have been chosen for their combination of healthiness and simplicity of execution. The food is provided free of charge and the costs of the meals have been calibrated to the average weekly budget of low socioeconomic status (SES) families in the UK. This intervention draws on the theory of habit formation (Becker and Murphy (1988)), and asks whether exposure to a healthier diet for a sustained period of time has persistent effects on diet. The protocol has been designed to maximise the chances of exposure to a healthy diet over a period of three months.

The second intervention (*Snack treatment*) is much cheaper and simpler to implement. Families are simply instructed to eat three meals a day, at regular times, and to avoid snacking between meals. Children are allowed two additional healthy snacks during the day, at regular times. This treatment draws on evidence of how snack foods are often high in

¹See Lang et al. (2009), Capacci et al. (2012) for reviews, French et al. (2003) for a discussion of 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).

 $^{^{2}}$ There is a related literature that examines the long run benefits of early childhood investment see for example: Campbell et al. (2014), Cunha and Heckman (2010), Heckman and Masterov (2007).

³For 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.

calories and offer lower nutritional value, and irregular and unstructured eating patterns are associated with poorer diets overall. Snacking is often referred to as a possible culprit for rising obesity rates (Cutler et al., 2003; St-Onge et al., 2003) and the most recent public health advice in England recommends parents to target children's snacking as a means to reduce their excessive sugar intake.⁴ Of course in both protocols compliance cannot be taken for granted, and we will devote great attention to this issue in the analysis.

These treatments should not be seen as policy proposals, but rather as a way of gauging the extent to which dietary habits are malleable - particularly early on in life - in the longerrun after the intervention is stopped. The "treatments" are of greater intensity than most large scale policy interventions (Ludwig, Kling and Mullainathan, 2011), and therefore can illustrate whether invasive interventions are able to improve notoriously unmalleable dietary habits.

We evaluate these interventions with a randomized controlled field experiment with 285 low income families, conducted in two different locations in the UK – Colchester (England) and Edinburgh (Scotland). Our main outcome of interest is diet. We are interested in actual dietary intake, as well as preferences. However, measuring dietary intake and preferences is notably difficult. To obtain the most accurate and complete picture possible of those, we collected a rich set of measures, including objective measures (Body Mass Index and blood biomarkers – the latter only for adults), measures based on self-reports (of preferences and dietary intake), and measures based on incentivized choices. We collected these measures over a period of three consecutive years, and are therefore able to evaluate long term effects.

Our resuts show evidence of significant changes in the children's body mass index (measured as the percentile in the distribution for 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 effects are large (between 5 and 6 percentage points) and are sustained in the longer run for the Meal treatment, while they attenuate over time for the Snack treatment and are no longer significant in the third year. The changes in calorie consumption, while not precisely estimated, are in line with the changes that we find in BMI. We also find significant differences in the intake of sugar of these children, again more pronounced in the Meal treatment than in the Snack treatment. One year after the intervention, the reduction in "added sugar" in the Meal and Snack treatments accounts for 66% and 50%, respectively, of the drop in calories. In addition, children's reported preferences for processed food fall in the Meal treatment after the 12-week intervention, but the effect does not persist over time.

⁴See Public Health England's new Change4Life campaign https://www.gov.uk/government/news/phe-launches-change4Life-campaign-around-childrens-snacking.

They do however seem to value sweets more, perhaps because they have more limited access to them. Food preferences are less affected in the Snack treatment. The most plausible story here for the patterns we observe is that the interventions had an impact on what the parents fed their children, rather than on the children's dietary preferences.

The results for adults show little evidence for malleability of dietary habits. Neither subjective nor objective measures show any evidence of improvement in the healthiness of their diet or their preferences for healthier foods, in the short or longer run. Overall, our results confirm that interventions later on in life are much more challenging. We have considered two very different types of intervention, neither of which seems to have any effect. The Meal treatment is a very invasive intervention - which is certainly far above the upper bound of policy instruments that could be considered. Still, we find little evidence of any change. The Snack treatment is very cheap, but appears harder to follow, and again, does not lead to changes in diet choices or preferences of adults.

The paper relates to the recent body of experimental work on habit formation of healthrelated behaviours. A few recent experimental studies targeting children in schools (Belot et al. (2016), Loewenstein et al. (2016)) provide promising evidence that children's dietary habits are malleable, at least for fruit and vegetable consumption. But the evidence on long term effects of interventions is limited. Also related, 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 and less nutritious foods (subsidy, taxes, information) in order to increase the consumption of the former. These experiments show that framing matters (low income families purchased more of both nutritious and lessnutritious food under the subsidy framing (Cawley et al., 2016)) and that incentives can lead to sustained changes in the purchase of fruit and vegetables even when the incentives are removed, suggesting habit formation had taken place (List et al., 2015). However, overall spending in the store involved in the experiment was low, suggesting that most other food purchases were taking place elsewhere.

The paper also relates to a medical literature that evaluates, under experimental conditions, the impact of specific diets on health outcomes (see for example Serra-Majem et al., 2006 and Esposito et al., 2011) for reviews of RCTs of the health impacts of the Mediterranean diet. These studies are typically interested in specific health outcomes of a specific diet. They are not interested in assessing whether dietary habits change once the intervention has ended.

Aside from experimental studies, this paper is also connected to the literature on food preferences and in particular on how stable they are. Bronnenberg et al. (2012) documents how migrants take their food preferences with them and that this leads to implications for the market share of brands. Atkin (2016) not only shows how preferences are carried with migrants but also that food cultures can constrain caloric intake. Preferences are also shown to be an important factor in understanding cross-country differences in food purchases, Dubois et al. (2014). In particular, they find that interaction of the environment (i.e. the prices and attributes of foods) and preferences explain the cross-country differences between the US, UK and France. Allcott et al. (2018) use a structural model that extends Dubois et al. (2014) and show how preferences are important in explaining the differences in food consumption between high and low-income households. They find a large proportion (over 90%) of the nutrition-income relationship is because of demand-side considerations related to preferences with supply side issues explaining less than 10 percent. The aforementioned studies show the apparent persistence of preferences but none use experimental variation to show the extent to which food preferences and habits are potentially malleable. Also, all of these papers focus on adult consumers. We focus on children whose preferences are potentially less fully formed.

This study makes several contributions to the literature. First, the scope of the interventions tested is unprecedented. Particularly for the meal treatment, we would argue that it is the strongest intervention one could probably think of implementing with families over a period of time. While this setup makes it harder to identify the specific mechanisms that may lead to change, it is an excellent test of whether dietary habits are at all malleable or not.

Second, the study involves adults but also very young children (aged between 2 and 6) some of whom are still pre-school age. This is unusual and enables us to examine the malleability of preferences and habits while they are still forming. Most of the habit formation literature has conducted experiments in schools with older children (Just and Price, 2013; Loewenstein et al., 2016; Belot et al., 2016).

Third, most studies rely on partial measures of dietary choices (such as isolated one-shot choices or consumption of specific items). It is very difficult to obtain a complete picture of one's diet, 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." Our measures collectively provide us with a more reliable picture of diet than each of them would individually.

Fourth, we are able to evaluate effects on a much longer horizon than other studies. We follow the families for three consecutive years. Most studies are typically short run or, if they include a follow-up at a later point in time, the horizon considered is usually limited to a year.

Finally, while most of the literature in economics has focused on how incentives can induce habits, we depart from this. Our aim is to use a relatively invasive intervention to ensure that participants change their diet during the treatment phase, and then to evaluate whether such changes lead to lasting changes in dietary habits once the intervention is removed.

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

II Experimental Design

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

Participants received only general information about the study - such as the study being related to health and lifestyle choices, and the study duration of 3 years. Families were not yet informed about the details of the two treatments. We excluded families for whom we considered the study to not be suitable, for example, families with individuals with pre-existing medical conditions, such as Diabetes Type I and II, or those with severe food allergies (see Appendix A Table A.1 for the full list of exclusion criteria).

We collected data on at least two people per household: The youngest child in the family aged between 2 and 6 and his/her main carer (most often female). Whenever possible, we collected data on both parents. Regardless of the number of children in the household, the "study child", for which we collected measures, was the youngest child of the household aged between 2 and 6. Consent forms were obtained for each participant and from the main carer for the child.

Randomisation — When registering to take part, families were asked to indicate several dates where they would be available to come to our facilities for the first session of measurements.

⁵The experiment was conducted with ethical approval from the University of Edinburgh.

⁶We used a range of different recruitment strategies which consisted of adverts, posters and stalls in community centers, nurseries; and shopping malls; letters sent to school principals; advertisements in buses and on radio. Samples of our recruitment materials (leaflet and poster) can be found in Appendix A, Figure A.1a and A.1b. Recruitment took place in January and February 2015 for Edinburgh, and in July and August 2015 for Colchester, the interventions were conducted in March-June 2015 in Edinburgh and September-December 2015 in Colchester.

All initial sessions' dates had been randomly pre-assigned to a specific treatment (control or one of the two treatments), and families were randomly assigned to one of their selected dates, without knowing these corresponded to the two different treatments and/or the control group.

Timing — Overall, 91 families in Edinburgh and 194 in Colchester took part in the study. Each treatment lasted for twelve weeks, and the baseline and post experiment measurements were collected during 2-week time windows before and after this twelve week treatment period, for each of the three groups (treatments and control). The 12-week treatment started in March 2015 (Edinburgh), and September 2015 (Colchester), and follow-up sessions took place two weeks after the end of the intervention, as well as in February (Edinburgh) and August (Colchester) 2016, 2017 and 2018. The families were not asked to follow specific guidelines beyond the twelve weeks of treatment for the two treatment groups. Table 1 provides further details on attendance and attrition. The attrition rate after the twelveweek intervention was very low (3.85%). The attrition rate was only 13% in 2016, 16% in 2017 and 18% in 2018 relative to the first "baseline" session.

Treatments

Treatment 1 - Provision of recipes and ingredients

The first treatment, referred to as "Meal treatment", consists of providing ingredients and recipe booklets every week, for twelve weeks, directly at participants' homes for five main meals for the *whole* family. Families could select between a regular or vegetarian food basket. The main objective was to maximize the chances that families, and more importantly children, are exposed to a range of healthy meals for twelve weeks. We are then interested in evaluating whether these changes are sustained in the longer run. The protocol was 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. An isocaloric comparison (fixed at 365 calories, the average calories of the meals) between the recommended nutritional guidelines and our recipes shows that our recipes are overall consistent with the recommendations, and are lower than the maximum thresholds on sugar and fat (and saturated fat), compensating for these calories via higher carbohydrate and protein contents. This can be seen in Table B.1, in Appendix B, by comparing the second and the third columns. A similar analysis on the participants' diet will be discussed in section III.F.2.

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 may prioritize problems that require immediate attention over issues that have consequences in the more distant future (such as health or saving). The protocol was deliberately chosen over stricter protocols that would impose constraints on families on all meals and food consumed, to ensure its feasibility. These design choices aimed at maximizing the chances of implementation in the short run and of sustainability in the longer run.

Third, the food is provided free of charge, which addresses the potential obstacle of perceived unaffordability of "healthy foods" (e.g. Dibsdall et al., 2003). Recent survey data from the UK suggest that 36% of low income households indicated they could not afford balanced meals. In addition, low income parents may be somewhat risk averse and less willing to try to cook new meals for their children for fear of the children not liking the food (Dowler et al., 2001). By providing the food for free, we alleviate the potential costs of wasting food that may discourage parents from buying and trying new foods. Furthermore, the costs of the meals have been calibrated to the average weekly budget of low SES families in the UK so it should in principle be possible for families to continue buying the ingredients and recipes once the treatment is over. Based on ONS household expenditure data for 2015^7 , average food expenditure for the 5 first income deciles was £28.81 per person per week (average taking into account both expenditure for adults and children). Our sample has an average household composition of 1.65 adults and 1.9 children per household. Based on this we calculate that our families would spend on average £102.28 per week on all inhouse food and non-alcoholic drink expenditure. No data exists to capture the amount of this spent on the evening meal, though we hypothesise that this will be circa one third of this budget, since the evening meal is typically also the largest, while breakfast and lunch tend to be an informal affair. Based on this assumption we calculate that $\pounds 34.09$ per week is

⁷see https://www.gov.uk/government/statistical-data-sets/family-food-datasets and https://www.ons.gov.uk/peoplepopulationandcommunity/personalandhouseholdfinances/ expenditure/datasets/householdexpenditurebygrossincomedecilegroupuktablea4

spent on 7 evening meals, and thus $\pounds 24.35$ is spent on 5 evening meals per week on average in our sample. This final figure was our benchmark for calibrating the cost of the meals we delivered to reflect affordable levels for this population group.

Note that the last two dimensions (convenience and free of charge) 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 are exposed to the healthy meals.

To maximize compliance families were asked to take photos of their meals – we provided cameras and SD cards. We asked families to fill in a feedback sheet reporting on how easy it was to cook the meals (on a 4-point Likert scale) and to what extent families liked them (again on a 4-point Likert scale). It is worth noting that such simple compliance tasks are inevitably part of the treatment. An example of the first page of the feedback sheet can be found in Appendix A, Table A.4. Food baskets rotated on a four-weekly basis, so families received the same food baskets and recipes three times in the twelve week treatment in order to allow for possible habituation. With this group, we also talked through, and provided a handout with general advice on healthy eating, which also included advice about alcohol consumption (Appendix A, document A.2.).

Treatment 2 - Regulating food intake

The second treatment, referred to as "Snack treatment", consists of regulating the timing of food intake, again for twelve consecutive weeks. Adults in the family were asked to eat three meals per day at regular times (selected by participants) and to avoid consuming any 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 snacks can be found in Appendix A, Table A.3. Snacks are arguably less likely to be results of conscious decisions (Wansink, 2006; Wansink et al., 2009). Piemas and Popkin (2010) find that children in a US sample get 27% of their daily calorie intake through snacks, which are often nutrient poor, and high in sugar and saturated fats. A review paper by Bellisle (2014) suggests that snacking often seems to contribute calories but little nutrition, especially among obese children and adults. Factors which determine nutritionally poor snacking include choosing energy-dense foods, eating when not hungry or in an irregular fashion, and eating in contexts which promote 'mindless eating', such as watching TV (Bellisle, 2014). A review on changes in childhood food consumption patterns by St-Onge et al. (2003) suggests that the rising proportion of calories coming from snack foods, which are in turn associated with 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).

This protocol aims to address the potential 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 with diet found that only skipping breakfast was consistently associated with poorer diets across studies (Leech et al., 2015). A randomised controlled trial on healthy participants found that compared to an irregular meal treatment, those on a regular meal protocol experienced metabolic responses which may favour weight management and metabolic health (Alhussain et al. 2016). With respect to children, a recent study on UK survey data focusing on metabolic markers rather than food consumption found that larger variability in eating frequency was associated with higher total and LDL cholesterol concentrations in children aged 4–10 years, but there was no association with BMI, waist-to-height ratio, and commonly tested biomarkers (Murakami and Livingstone, 2015). A related body of literature in biology hypothesizes that irregularity of food intake could have a significant impact on diet and total calories, although this hypothesis is not supported in non-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 dietary planning and self-regulation are argued to be good strategies to deal with habit-driven impulsive consumption of unhealthy food (Naughton et al., 2015). In light of the evidence, a protocol encouraging reduction of snacking and more regular food consumption was expected to lead to positive dietary outcomes.

Other than being given recommendations about timing of food consumption, families were not given any additional instructions or recommendations as to what they should eat. This protocol is of course difficult to enforce, and so the main goal of this intervention is to create a source of exogenous variation in frequency and regularity of food intake across groups, and study how that has an impact on diet and total calorie intake in particular. To increase compliance, families were to follow this protocol for six 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 twelve 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 (this is discussed in Section III.B).

Finally, our control group consists of participants recruited in the same way as those for the treatments, but instructed to just carry on as usual with their daily routines.

Monetary Compensation— Families received £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 were given an incentive for every measurement session they attended.⁸

III Empirical Analysis

III.A Descriptive Statistics

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

III.B Compliance

The experiment is an intention-to-treat. For the Meal treatment, families had to prepare five meals per week; while for the Snack treatment, families were requested to adhere to regular eating times. Compliance was not directly incentivised for either protocol 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 with a camera we provided, and we asked them to 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

⁸Families 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.

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

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

Table 3 shows differences in self-reported measures regarding the ease of implementation of the protocols. We find interesting differences between the two treatment groups. For instance, 42.5% of the Snack sample found it difficult or very difficult not to snack between the meals. In contrast, 83.7% of those in the Meal treatment say they found it easy or very easy to cook the recipes. Complying with the Snack protocol has not been straightforward and probably meant a substantial change in routine for most participants.

Table 4 presents several variables capturing how participants felt they were affected by the protocols. We find that 48.7% of the people assigned to the Snack treatment felt they were eating less food during the day. In the Meal treatment, 64.8% of the adults self-report and 79.6% 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 10.14 out of 19 recipes⁹. This number fell one year later 6.05 recipes, but then remained stable at 5.7 two years later, and finally 6.2 recipes three years later.

The second measure of compliance we propose is based on the number of photographs provided by participants in the Meal treatment. Since they were supposed to cook five meals per week during twelve weeks and to photograph each of them, a complete set of pictures would include 60 photographs. On average, we received 38 unique pictures back (hence 63%). This could of course be an underestimate of the meals that were cooked and eaten, it could well be the case that a meal was cooked but participants forgot to take the picture. Conversely, it could be the case that a family cooked the meal, took the picture, but did not eat the meal, implying that compliance is lower than the rate of picture returns suggest. While it is a possibility, it seems more likely that the family forgets to take the picture than they would cook the meal and not eat it. Hence, the compliance measure from the return of the unique photographs is likely to be an underestimate. Furthermore, only 11% of the treated families returned no pictures back at all. These figures suggest that compliance was relatively high.

Finally, the last measure of compliance we have is based on the information provided in the leaflets. For the Meal treatment, 80.6% of the households who came back after the intervention brought their completed leaflet back to us. They report a mean liking of the meals of 2.9 (0.38 s.d.) (on a 4 point Likert scale). When children specifically had a different taste than the parents, this was also reported, yielding an average liking by the children of 2.7 (0.67 s.d.). Children's overall liking of the recipes is significantly lower than that of the adults (a Wilcoxon signed-rank test yields a p-value=0.00). Turning to the difficulty of cooking the meals, on a 5-point Likert scale (from 1 being very easy to 5 being very difficult) adults report an average of 1.7 (s.d=0.5). Those results corroborate the self-reports displayed in Table 3, showing that this treatment has been perceived as relatively easy to follow.

In the Snack treatment, among the families that came back after the intervention, 69.0% brought the leaflet back, which is a lower rate than in the Meal treatment. It was also possible to evaluate the extent to which the forms were filled in a "robotic" fashion, which we interpret as a possible signal of misreporting. We use two main criteria to characterise the households as filling the leaflet out in an automatic manner or not: first if they were writing the same times of the meals over the twelve weeks, with the same pen and without

 $^{^{9}15}$ recipes in total for vegetarian families who represent 8% of the sample.

any noticeable differences on each of the pages. We find that 37.5% of the leaflets fit this profile. Second, because the families could deviate from the regular food intake one day of their choice every week, another "robotic" attitude would be to tick the same day every week, with the same pen, and without ever deviating to choose another day off from the protocol. We find that 20.8% fit this profile.

Returning to the "day-off" allowed within the Snack treatment, if every family was taking this option, this would mean that out of the 84 days of the treatment duration, 12 (14.3%) should be marked as a day-off. We find that 14.5% of the days in the leaflets have been reported as the day off. Families were explicitly asked to indicate additional deviations from the protocol. 19.9% of all days were reported as additional days where families did not follow the exact protocol. 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 twelve weeks. For each week, we set the mode time as the regular time and we look at the frequency of a deviation of at least 30 minutes 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 degree of irregularities of the Snack treatment, which corroborate results from Table 3 and Table 4.

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

III.C Self-reported changes in habits

During each session over the three years, adult participants were asked to answer a survey about their health in general, changes in diet and food consumption, snacking habits, smoking and drinking habits. We now briefly comment on the results of these self-reported changes.

First, we see no significant differences in reported health, smoking or drinking habits across the three groups (not reported here). However, answers to questions related to diet quality and food item consumption show significant differences between the treatment groups and the control group. Table B.2 from Appendix B shows self-reported answers about how the quality of the diet and snacking behaviours changed over time. Specifically, participants report whether they have changed diet and snacking habits one, two and three years after the treatment relative to the baseline period. This is reported for both adults (Panel A) and children (Panel B). Significant differences are observed. Meal participants reported that the diet of their children improved compared to the control group and that both adults and children are eating food they were not eating before. This effect is evident up to three years after the start of the experiment. Snack participants also report that their children eat some foods they were not eating before, but there are no significant differences for the adults. This is probably because children ate new snacks along with the Snack protocol, which was not the case for the adults. Adults significantly report snacking less between meals, compared to the control group, one year after the start of the study, but these differences do not persist two or three years later.

We also asked participants to report any changes the consumption of specific food items. Those results are reported in Tables B.3a (children) and B.3b (adults) of Appendix B. We show the proportion of participants reporting increases in consumption of fresh and low-calorie items (for example fresh fruits, fresh vegetables, and fresh meat and fish) and decreases in consumption of high-calorie items (for example processed foods, cakes, salted nuts, crisps). Adults were asked to report any changes for themselves and for their children, 3 months, 1 year, 2 years and 3 years after the study had started. After the twelve-week intervention, Meal participants are significantly more likely to report an increase in the consumption of fresh food (fruits, vegetables, meat, pulses) and more likely to report a decrease in the consumption of processed meat, deep fried food, pies and pastries, both for themselves and for their children. The Snack participants are also more likely to report an increase in consumption of fresh fruit and vegetables for their children (compared to the control group). However, no changes are reported in the consumption of fresh meat for instance, which is in line with the fact that they were not asked to change the meal content of their children. The children in the Snack group significantly decreased their consumption of high-calorie snacks such as pretzels, cakes and pastries, energy and breakfast bars. This is also observed for adults, who were asked not to snack between meals. Over the subsequent years, these results are less robust. Meal participants continue reporting having increased their consumption of some fresh foods (fruits, vegetables and poultry) and decreased consumption of high calorie items such as ice cream and energy bars (relative to the pre-intervention period). The changes appear less persistent for the snack group.

III.D Measures

We collected a rich set of measures to provide a more complete picture of the diet and health of participants. Measures were collected at baseline, after completion of the twelve week treatments, one year, two and three years 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 should collectively provide a picture of participants' diet. The goal of the empirical analysis will be to identify a consistent and robust pattern across these different measures.

Anthropometric measures The first measures are anthropometric measures. Adults and children were weighed and measured by a trained member of the 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.

Dietary intake The second measure is of dietary intake. It is based on a well-known method in the nutrition literature – "24-hour diet recall". Participants are asked to recall in detail what they have eaten in the last 24 hours. They are helped and guided by a professional nutritionist, trained to collect data using this method. For children, we collect information from the child's parent, primarily the mother. The data was first recorded face-to-face with nutritionists, and then entered into a nutritional analysis software¹⁰, which computes measures of dietary intake based on a large database of food items available in the UK. This provided us with caloric intakes, as well as macro-nutrient composition.

Studies validating the 24-hour diet recall as a method for measuring dietary intake compare it to energy expenditure measured by doubly labelled water (DLW).¹¹ These studies show that the 24-hour recall under-reports from 1% to 17% depending on a number of factors including the number of consecutive recalls obtained (each additional consecutive recall gives more accuracy), and whether these have been done in person or over the phone (Hill and Davies, 2001; Livingstone et al., 2003; Ma et al., 2009). While three consecutive recalls are recommended to assess individual intake, one recall does capture the average intake of a group fairly well (Biro et al., 2002).

For the one, two, and three-year follow-ups we used Intake24¹² - a computer-based recall method designed for the British population. Unlike the nutritionist led face-to-face 24-hour dietary recall described above, with Intake24, the participants recall their own intake using the software. Measured outcomes are similar to the face-to-face 24h dietary recall and can thus be compared to the face-to-face recall.

¹⁰Nutritional analysis was carried out using NetWISP 4.

¹¹The doubly labelled water (DLW) method is typically seen as the gold standard way of capturing energy expenditure. Participants are required to drink the 'doubly labelled water' which is enriched with two naturally occurring isotopes which allow, from the analysis of urine samples, the measurement of the expenditure of carbon dioxide and in turn energy. This can be therefore used to measure overall energy expenditure.

¹²See intake24.co.uk more details and a demonstration.

Food preferences Questionnaire The third measure focuses on dietary preferences. Due to the young age of the 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 (see Appendix B, Table B.4 for the full list of items). For each item, participants had to answer on a 4-point scale how much they liked the item (really dislike to really like), with the additional possibility of an 'allergic' or 'never tried' option. Items were then grouped into food categories following the 'eatwell plate'¹³ food categories (fruit, vegetables, meat/fish/eggs, cheese, bread, unhealthy processed food and sweets).

Incentivized measures for food preferences We collected additional incentivized measures of food preferences in years 2 and 3 for adults, and in year 3 for children. The measure for adults was based on the choice of a supermarket basket worth GBP 30, using the tool developed by Spiteri et al. (forthcoming). Participants are asked to select food items among a choice set of 120 popular items from a supermarket, organized in categories that resemble typical aisles in a supermarket (fruit and vegetables, meat and fish, dairy, etc.). Screenshots of the tasks can be found in Figure 1. The basket was delivered to 1 in every 10 families randomly selected, two weeks after the intervention (to avoid that choices would depend on current food stocks). The tool allows us to extract information on calories and nutrients such as sugar and saturated fat. We also construct a measure of percentage spent on "unhealthy" items. Unhealthy items are identified through the nutrient profiling technique developed by the UK's Food Standards Agency (FSA).¹⁴ We chose to collect this measure only ex post to avoid contamination with the interventions conducted in year 1 in the treatment groups. Participants are instructed to shop as they normally would. This means that the shopping basket could be intended for the whole family rather than for themselves individually.

The measure for children was collected in year 3, when children were older and presumably better able to understand an experimental protocol. They were offered a choice between a low calorie food item and a high calorie item. Children were presented with four different pairs of items involving either sweet or savoury food, fresh or storable (See Figure 2). To ensure that the low calorie item was attractive, we chose to price each item by allocating a

¹³The eatwell plate is a policy tool used to define the British government recommendations on eating healthily and achieving a balanced diet.

¹⁴Points are allocated on the basis of the nutrient content of 100g of a food or drink. Points are awarded for energy, saturated fat, total sugar and sodium (A-nutrients), and for fruit, vegetables and nut content, fibre and protein (C-nutrients). The final score is then given as the score for C-nutrients subtracted from the score for A-nutrients. The unhealthy items are then classified as foods with 4 or more points and drinks with 1 or more points. For full details of how the points are calculated please see https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_ data/file/216094/dh_123492.pdf

risk of not obtaining it. Each item was presented next to three closed capsules, and the child was shown the content of each capsule before she was asked to pick. The low calorie item was associated with two capsules with a picture of a smiley face and one with a picture of a bomb, the high calorie item was associated with one capsule with a smiley and two with a bomb. The child was told that she could actually get the item if she would pick the capsule with a smiley face. She first indicated her choice for each of the four pairs and then was told which choice would be implemented for real. She would then be asked to pick one of the capsules next to the item she selected. The outcome measure of interest is a simple count of the number of times the child picks the low calorie (healthy) item. Since the price involves risk, we also collected a measure of risk preference, inspired by the bomb task (Crosetto and Filippin, 2013). Children were presented with a bag containing 10 capsules. In 9 of them, there was a picture of a smiley face and in one of them the picture of a bomb. They could earn a sticker for each smiley face they would pick but if they picked the bomb, they would lose everything. They were then asked to indicate how many capsules they would like to pick. We use the number of capsules picked as a measure of risk tolerance, and control for this measure in the analysis of the incentivized food choice.

Additional measures in year 1

In Edinburgh only, study participants (excluding children) provided fasted blood samples prior to and after the twelve week treatment. The full list of biomarkers screened and their short description is presented in the Appendix B in Table B.5. We tested for biomarkers typically measured in dietary interventions in the existing literature, and which could be expected to change significantly within the 12-week study period, though some markers, like triglycerides, can change after 1 day's exposure to significant dietary change, and the implications of this are discussed in section III.F.5 (Purkins et al., 2004).

In Colchester, we implemented an incentivized measure of snack/drink choice in year 1, before and after the experiment.¹⁵ Every adult participant was asked to pick two combinations of a snack and a drink, one of low calorie (less than 100 Kcal) and one of high calorie (more than 200 Kcal). They were endowed with £4 and were asked to spend part of this money in buying the pair of snacks. They had 7 choices to make in which they had to decide whether they wanted to buy the low-calorie pair or the high-calorie pair of snacks. The price of the low calorie pair of snacks was set to £2 for all 7 choices. The high calorie pair of snacks' price ranged between £1.40 and £2.60, with an increment of 20 pence for each choice. The task is shown in Figure 3. Choices in this task tell us how much the participant is willing to pay to receive the high-calorie option compared to the low-calorie option, choice 4

¹⁵We chose to collect this additional measure as it was logistically not possible to collect blood samples.

displays the same price for both, and choices 5 to 7 display a lower price for the high calorie option.

Note that all 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, diets were assessed with the 24-hour dietary recall method performed face-to-face with a nutritionist the first year (pre- and post-treatment sessions) to limit under-reporting. Surveys were conducted in a computer lab so participants could ask questions if something was misunderstood.

III.E Empirical Strategy

Our econometric specification aims at estimating an intention to treat (ITT) effect:

$$\begin{aligned} Outcome_{it} &= \alpha_i &+ \beta_1 After_{it} + \beta_2 \text{ 1-year}_{it} + \beta_3 M_i \times After_{it} + \beta_4 S_i \times After_{it} \\ &+ \beta_5 M_i \times 1\text{-year}_{it} + \beta_6 S_i \times 1\text{-year}_{it} \\ &+ \beta_7 M_i \times 2\text{-year}_{it} + \beta_8 S_i \times 2\text{-year}_{it} \\ &+ \beta_9 M_i \times 3\text{-year}_{it} + \beta_{10} S_i \times 3\text{-year}_{it} + \epsilon_{it} \end{aligned}$$

Where α_i is an individual fixed effect, $After_{it}$ indicates the period is immediately after the 12-week treatment, with $1 - year_{it}$ indicating being 1 year after the treatment, $2 - year_{it}$ indicating being 2 years after the treatment, $3 - year_{it}$ indicating being 3 year after the treatment. M_i and S_i are the two treatment assignments, Meal and Snack respectively. The estimation of the ITT effects are β_3 through to β_{10} .

III.F Results

III.F.1 Anthropometric measurements

We first present the analysis of anthropometric measures. Table 5 shows the mean BMI and proportion of each weight category of our sample at baseline. Around 64% of our adult sample is overweight or obese (32%). These figures are in line with the national rates reported in the National Diet and Nutrition Survey.¹⁶ For 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

¹⁶https://www.gov.uk/government/uploads/system/uploads/attachment_data/file/310995/ NDNS_Y1_to_4_UK_report.pdf

groups at baseline. However, women in the control group have significantly higher BMI than women in the Snack treatment (a Wilcoxon signed rank test yields a p-value of 0.04).

Table 6 presents the results of the impact of the experiment on BMI. Note that both treatments were not designed as weight-loss programmes and did not impose any calorie restriction. Nevertheless, these protocols were expected to have longer term effects on BMI through a change in dietary habits.

For children, we find a precisely estimated and negative treatment effect on the BMI percentile, seen in the first column, in the short run but also in the years after. There appears to, therefore, have been a sustained impact. The size of the effects (between 5 and 6 percentage points) is initially very similar across both treatments. Children in both treatment groups appear to have moved down in the distribution, that is, they are relatively thinner than the children in the control group. The effect is remarkably robust and persistent for the Meal treatment, while it fades out for the Snack treatment.

We do not find that children are more or less likely to be overweight or obese (Column 2), however there were few children in this category at baseline. 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). BMI results are further complicated by two peculiarities in our data, which show that at baseline, control group mothers had a higher starting BMI than treatment group mothers. Nevertheless, we find no evidence that the treated mothers experienced a lower weight gain that those in the control group.

III.F.2 Dietary intake

We now turn to the analysis of dietary intakes. Table 7 presents the baseline statistics (before the treatment) for different categories of food intakes and average quantities: total calorie intake, number of fruits and vegetables, quantities (in grams) of fruits and vegetables, total fat, carbohydrate, protein, saturated fat, sugar, Non-Milk Extrinsic Sugar (NMES, also called free sugars, which are generally considered to be added sugar), fibre, sodium and alcohol. The first column of Table 7 shows the daily recommendation given by the National Obesity Observatory Document Standard evaluation framework for dietary treatments¹⁷ and the Manual of Dietetic Practice (Thomas et al., 2007). We distinguish between total fat and saturated fat as well as total sugar and NMES. On average, the self-reported intakes imply that a male adult participant consumes 2216 calories over 24 hours, whereas a female adult consumes 1907 calories. The average calorie intake in children is 1434 calories. These

¹⁷British Nutrition Foundation (BNF), 2015. Nutrition Requirements. Available at: https://tinyurl.com/nutrition-requirements

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.¹⁸ By comparing the different intakes with the daily recommendations, we see that a relatively large proportion of food intake for our sample comes from carbohydrates, although those amounts still fit with the recommendations. The intake of protein is above the minimum requirement, and the intake of saturated fats and sugars also exceeds the recommended amounts. The participants also fail to meet the recommended intake of fruit, vegetables and fibre.¹⁹ Nevertheless, at baseline, we find no significant differences in calorie intakes or other macro-nutrient intakes between the groups.

We also compare the baseline diet of the participants compared with the nutritional content of the recipes in the Meal treatment. This allow us to check for a possibility of improvement in the diet of the Meal participants. In an isocaloric comparison, held at 365 calories, of our participants' consumption and our recipe profiles, we note that our participants ate twice the amount of our recipes' fat (15g versus 8g) and twice the amount of recipes' sugars (20g versus 10g) (see Appendix B, Table B.1). Participants' diets at baseline were lower in carbohydrates and protein than our recipes.

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

First, we should point out that the coefficients of the time dummy variables are almost all statistically significant for children and many of them are for adults as well, that is, significant dietary changes are measured for everyone in the sample. For children, it is clear that one would expect them to eat more as they grow, and the effects we find point in

¹⁸The 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

¹⁹Another 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, Tables B.6 and B.7. The outcomes are obviously similar.

that direction. For adults, the time dummy effects suggest that they all have a healthier diet, eating less calories overall, more fruit and vegetables, less fat and sugar (although the effect on added sugar goes in the opposite direction in the longer run). So these results are suggestive of an overall time trend, which could be due to participating in the study or to a general trend in the underlying population.

Second, looking at the treatment effects, the coefficients are not precisely estimated, but we do find interesting significant effects in the longer run. We find that children (panel A) consume less sugar and fat, specifically in the Meal treatment. In the Snack treatment, the only significant effect is on sugar. Even if the effects are not statistically significant, it is worth pointing out that the amount of calories is systematically negative in all post experimental periods in both treatments. The signs of the coefficients are mostly going in the expected direction except for the fruit and vegetable intakes, i.e. the coefficients are negative for calorie intakes, fat, sugars, proteins and sodium, but also negative for fruit and vegetable intake.

As for the adults, no consistent or significant patterns are found in contrast to the children. These results suggest that dietary intake may have changed for children, but we do not 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. If anything, we find positive significant coefficients on fat and protein one year after the intervention. This effect does not persist in the subsequent year.

As an alternative analysis (not reported here), we have also computed two healthy eating indices. In a first index, we use the recommendation threshold for each macronutrient and create a dummy variable equal to 1 if the participant belongs to the recommendations' ranges, 0 otherwise. We then sum those dummy to create an index. A second index is inspired by Handbury *et al.* (2016). We use the share in the recommendation quantities and percentage from calories allowing us to have an index informing on the diet quality. We find no significant treatment effects on any of these two indices, for children or adults (not reported here).

III.F.3 Food preferences

Table 9 presents the self-reported food preferences of both the children and the adults at baseline (before the twelve week treatment). We report the mean of each category of item. These items are ranked based on the response of the control group. There are overall no significant differences in liking at baseline between the treatment groups. It is worth pointing out that the ranking follows an expected pattern for children with sweets, bread and processed food at the top, while the meals are situated at the bottom of the ranking.

For parents, in contrast, the ranking is perhaps more surprising, with processed foods and sweets appearing relatively low in the ranking. One might worry that adults are less likely to report their true preferences with such questionnaire, and are perhaps tempted to report desired preferences instead. This is speculative, of course, but worth keeping in mind when we turn to the results.

We present the ITT estimates of food preferences in Table 10 for three points in time: in the short run, right after the treatment ("After") and in the long run, one, two and three years after the treatment ("1-year", "2-year" and "3-year"). For space reasons, we only report the results for the different food groups. The results for individual food items and meals are reported in the Appendix B, Tables B.8a, B.8b, B.9a, B.9b and B.10.

For the Meal treatment, we find that children in the treated group report a lower level of liking of processed food, as well as for cheese, which are two categories for which consumption is advised to be reduced because of their salty and fatty composition. However they also report liking sweets more. The effects on sweets appear to remain for two years after the intervention, while the effects on processed foods and cheese are no longer statistically significant. We do not find any differences three years later.

Adults in this group show very little changes in reported food preferences. The estimates are precise and close to zero. The only significant effect we find is related to processed food, which they report liking more on average than the control group right after they have been treated. This effect is only statistically significant at the 10% level. Finally, we found no significant changes in meal preferences for children and adults who were exposed to those meals, compared to the control group (see Table B.10. in Appendix B).

For the Snack treatment, we find fewer significant effects. For children, we find again a significant positive effect on the preference for sweets. We also find a positive effect on the preference for bread. None of these effects subsist in the longer run. We find no effects on reported preferences of adults.

III.F.4 Incentivized measures

We now turn to the incentivized measures of dietary preferences, which were collected in years 2 and 3 for adults and in year 3 for children.

For adults, Table 11 shows few significant differences in purchasing behaviour across groups. We do, however, find that the total amount of sugar in the foods chosen is significantly lower for participants in the Meal treatment in Year 2. This could be a statistical artefact, but it does match well with the finding of reduced sugar intake among children observed in the Meal Treatment in Year 2. The effect is not sustained in Year 3 (and it is also not sustained in the dietary intake data). On top of that, we find that participants in the Meal treatment spend significantly less (between 4 and 5 percent) on unhealthy items than the control group. These effects are observed both in Years 2 and 3. We find no statistically significant differences between the Snack group and the Control group.

For children we look at the number of high calorie items chosen out of the 4 pairs presented. We regress this number on the treatment, the age and gender of the child, as well as the measure of risk preference (see Table 12). We find no statistically significant difference across treatments. That is, we have no indication that children in either treatment group developed a preference for "healthier" foods.

III.F.5 Additional measures in Year 1

Incentivised measure of snack preference - In Colchester (England) we included an incentivised measure of snack preference for the parents, before and after the treatment in Year 1. The measure is described in section III.D. Table 13 indicates the changes in the number of times participants chose the low-calorie option over the high-calorie option. We find that participants are significantly less likely to pick the low-calorie option after the treatment, compared to the control group. This means that compared to before the treatment, they are willing to pay a higher price for the high-calorie snack and drink after the treatment. A possible interpretation of this could be that participants experience a rebound effect after having implemented a healthy meal or snack plan for twelve weeks and allow themselves to buy an unhealthy snack at a more expensive price to reward themselves, or it could be a manifestation of cravings after the twelve-week programme for this high-calorie snack (Fishbach and Dhar, 2005).

Blood biomarkers In Edinburgh, adults were asked to provide a fasted blood sample before and after the treatment (although not at the 1 or 2 year follow-up). Table 14 reports the levels of the different blood biomarkers levels at baseline, compared to the normal ranges as advised in the UK. Overall, our participants have normal levels for all biomarkers. This is not surprising as they are non-elderly adults (aged 35 on average) with no serious health conditions (one of the recruitment inclusion criteria). However, the mean low-density lipoprotein (LDL) reaches the upper limit of the normal range in the control group and is significantly higher than in the Meal and Snack treatments. 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). In our sample, Snack and Control participants have a CRP level slightly above normal ranges but those levels are not significantly different from the Meal participants' CRP level, that reaches the normal range limit.

ITT estimates are reported in Table 15 showing two main treatment effects. First, the estimated effect of the level of LDL (colloquially called the "bad cholesterol") is positive and statistically significant for the Meal participants compared to the Control group. Second, the estimated effect on the glucose level is positive, but not particularly precisely estimated (being only significant at the 10% level) for the Snack participants, compared to the control group.

In Table 8 we noted that no significant differences emerged in adults for the Meal treatment post treatment in terms of calorie and macronutrient intake. The estimated coefficients point to a slight increase in calories (88.4 calories), and a small increase in carbohydrates (11.6 grams) though other macronutrient changes remain in the single figures. Based on the above changes in diet, it is unclear why the Meal group experienced a small statistically significant rise in LDL values after the study. LDL has been shown to be elevated in diets higher in saturated fats (Mensink et al., 2003), yet post treatment there was no significant change in the amount of saturated fats those in the Meal treatment were eating. Table 8 showed no significant differences in calorie and macronutrient intakes for the Snack group post treatment. The signs of the coefficients point to a slight drop in calories (178 calories), a drop in total carbohydrates (24.5 grams) mainly caused by a drop in sugars (23.2 grams), and a fall in sodium (370 mg, approximately equivalent to 0.9 grams of salt). The above dietary changes appear to be somewhat consistent with changes in fasting blood glucose, which for the Snack group increased slightly but significantly post treatment. Fasting glucose levels tend to be higher on low-glycemic index diets than on high-glycemic index diets (Sacks et al., 2014), so a rise in blood glucose would be consistent with a post-treatment diet lower in sugars, which we indeed observe for this group albeit these are not precisely estimated.

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

III.G Correlation between parents and children

As the experiment is focused on the family, we are also interested in behaviour within the family unit, and 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 the latter two become closer or further apart as a result of the experiment, hence if the within-family correlation of preferences or intakes changes while being treated. Specifically, we may expect to see a convergence in preferences and food intake of the parent and child in the Meal treatment.

Body measurements — Panel A of Figure B.1 in Appendix B shows the scatter plot of the child's and main adult's 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.11. We find a positive correlation in preferences with one exception, that of carrots which is negatively correlated but this is statistically insignificant. The correlations range from -0.043 (carrots) to 0.244 (melon), these estimates appear to be in line with earlier evidence on the resemblance of food preferences between parents and children (Pliner, 1983). Preferences for 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 re-estimate equation 1 where the dependent variable takes a 1 if the preferences of the parents and children are the same and 0 otherwise. We present the results of this exercise in Figure B.2, Appendix B. In summary, these figures show that the preferences for most foods have not become more alike because of the experiment, neither immediately after the intervention nor one year later. *Dietary intake* — Looking at baseline only, we see a positive correlation between the food intake of the main parent and the child. Figure B.3 in Appendix B shows scatter plots of food intake with the child's intake on the y-axis and the main adult's on x-axis, with a linear fit through those points. We find that this correlation is statistically significant for energy intake, for fruit and vegetables and almost all the macronutrients. The only exception is for protein intake, which could be due to young children not eating as much meat as their parents. However, most correlations are weak with most estimates being around 0.2, the exception being vegetables with a higher correlation 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 these correlations change as a result of the treatments, we first calculate the absolute difference in intake, be that overall energy or a specific macronutrient, between the adult and child. In particular, we again estimate equation 1 with the absolute difference as the dependent variable and evaluate the treatment effects on this absolute difference. Table B.12a, panel A, of Appendix B, presents the results of this analysis where we find that there is a statistically significant increase in absolute difference in overall energy consumption for the Meal intervention, but not for the Snack intervention. Panel B (Table B.12b) shows the actual difference (adult's intake - child's intake). We find that the absolute gap is driven by an increase in the main parent's calories, although this effect is not statistically significant. Panel A also shows there was a significant increase in the gap between adults and children with respect to carbohydrates. Overall, there is a positive correlation between parents and children in the intake prior to the experiment and we find that the gap in overall energy intake between the parent and child increases although there is not a statistically significant treatment effect for any of the food types or macronutrients.

III.H Overall picture from multiple measures

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, the different measures aim at providing a richer and more complete picture of diet. 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 (-33 calories immediately after and -101 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 and 2-year follow-ups. To put this decrease into context, a 5-year old girl on the 50th centile for height and weight would be 108cm tall and weigh 18.10kg. To be on the 45th centile, this same girl would need to weigh 17.88kg, i.e. 220grams less, keeping height constant (NHS Healthy Weight Calculator). Based on calculations developed for adults (Hall et al., 2011), a weight loss of approximately 220 grams, would require a 770 calorie deficit over twelve 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 33 and 40 for the Meal and Snack groups respectively.

We find significant decreases in the intake of "added sugar" (NMES) which could be a key reason behind calorie reduction. At the one-year follow-up, the reduction in NMES for the Meal and Snack groups at -22g and -20g respectively accounts for 66% and 50% of the reported drop in calories (at -101 and -120 respectively). We find no effect on fats and no increased intake of fruit or vegetables. We also find a decrease in the percentage of unhealthy items and amount of sugar in the foods chosen by the Meal participants in the incentivized purchasing task.

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. We have little indication that this change occurred through a change in *preferences* for healthier foods. If anything, these children show a stronger preference for sweets. A more plausible explanation is that children got restricted access to foods high in sugar.

The story is somewhat similar for children assigned to the Snack treatment, although the effects are less consistent with each other. We also see a significant decrease in the number of fruits consumed (one year after treatment). Thus, there is less of a consistent story for the Snack treatment than for the Meal treatment.

Turning to adults, it is much harder to find a consistent picture here. We find no change in self-reported preferences (almost 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, 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 ways to assess compliance of families: self-reported 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 it easier to follow the protocol than participants in the Snack treatment and were more likely to return the feedback leaflets.

IV Conclusion

In this paper, we evaluate two treatments in a randomized controlled trial to evaluate the extent to which dietary habits are malleable early on in childhood and later on in life. We tested two interventions. The first targets what people eat (Meal treatment), while the second targets the regularity of eating patterns (Snack treatment). 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, as well as one, two and three years after the treatments had taken place, which enables us to estimate average treatment effect in both the short and long run.

We consider our treatments to be quite invasive. 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, diets were assessed with the 24-hour dietary recall method performed face-to-face with a nutritionist the first year (pre- and post-treatment sessions) to limit under-reporting. Surveys were conducted in a computer lab so participants could ask questions if something was misunderstood.

We show that, prior to the treatments, both adults and children had diets that would be considered out of the national recommendations with too much saturated fat and sugar, and not enough fruits and vegetables. This unbalanced diet is corroborated by a high proportion of overweight and obese individuals in our sample.

The treatments appeared to have affected children's dietary habits, but not those of their parents. In the short run, children's food preferences decrease for high-calorie food items (processed foods, bread and cheese). NMES intake decreases significantly in the longer run in both treatment groups, compared to the control group. Children in both treatments are moving down the distribution in terms of weight and BMI meaning that overall they become relatively thinner than the children in the control group. A result that is found for both the short and long run. However, our treatment did not alter consumption of, or preferences for, recommended low-calorie foods such as fruits and vegetables. We find no significant effects on parents.

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, so it is not entirely clear that their behaviour has been completely unaffected.

Our results suggest that dietary habits are more malleable early on in life than later and, perhaps interestingly, it appears possible to affect children's habits even if those of their parents are unchanged. The changes do not seem to operate through changes in preferences for healthier foods though, thus we have little evidence that food preferences are malleable, even early on in childhood.

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Tables and Figures



Figure 1: Screenshots from the incentivised supermarket basket choice
Figure 2: Screenshots from the incentivised food choice task for children



Figure 3: Incentivized measures of food choices. In the lab (Colchester sample)

If your choice is randomly drawn, the price will be deducted from your £4 and you will keep the rest of the money and get the snack and paired drink you chose. Please choose one option between the two proposed for each of the 7 rows.		n each row, you will see the referred option in each ro	he prices corresponding to eac w.	h of the
Option 1: Option 2: Image: Constrained on the state of				nd you will keep
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£1.80 Choice 6:		0	O	£2.00
		O	O	£2.00
		0	O	£2.00
Choice 7: £1.40 © £2.00		0	O	£2.00

You are asked to take 7 decisions between the two options, with different prices for the ¢

You are asked to take 7 decisions between the two options, with different prices

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).

	Control	Meal	Snack	Total
Essex baseline	76	66	52	194
Essex after	74	64	47	185
Essex 1 year follow-up	67	55	39	161
Essex 2 year follow-up	68	54	38	160
Essex 3 year follow-up	61	56	36	153
Edinburgh baseline	35	37	19	91
Edinburgh after	35	37	17	89
Edinburgh 1 year follow-up	33	37	17	87
Edinburgh 2 year follow-up	31	33	15	79
Edinburgh 3 year follow-up	31	34	15	81
Total baseline	111	103	71	285
Total after	109	101	64	274
Total 1 year follow-up	100	92	56	248
Total 2 year follow-up	99	87	53	239
Total 3 year follow-up	92	90	51	233

Table 1: Sample size. Number of participating households.

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

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

Table 2: Demographic characteristics at baseline and across groups

Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a t-test of equality of estimated parameters in Col. (1) and (2) and in Col. (1) and (3) respectively. "Higher Degree" includes higher grade, andvanced higher, CSYS, A level, GNVQ/GSVQ advanced, SVQ level 3First Degree, Higher degree, SVQ Level. "No Qualifications corresponds to respondents who ticked the "No Qualifications" option. A descriptive statistics table for panel A Edinburgh and panel B Colchester can be found in Table B.13 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/ easy	Neutral	Difficult/ Very difficult
A. Meal (N=123)			
To cook meals	83.7	13	3.3
To stick to the recipe	61	25.2	13.8
B. Snack (N=80)			
To stick to meal times	41.2	30	28.8
To stick to meal and snack times (child)	57.5	25	17.5
Not to snack	33.7	23.8	42.5
Not to snack (child)	27.4	41.3	31.3

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

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

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

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

	Control	Meal	Snack	$\begin{array}{c} \text{P-value} \\ (1)=(2) \end{array}$	$\begin{array}{c} \text{P-value} \\ (1) = (3) \end{array}$
	00110101	mean	Dilacit	(1)-(2)	(1)-(0)
A. Children					
BMI percentile	64.9	63.2	60.0	0.21	0.57
% Underweight	3.9	3.1	4.6	0.99	0.98
% Normal weight	71.8	78.6	78.5		
% Overweight	18.5	12.2	13.8		
% Obese	5.8	6.1	3.1		
# Obs	103	98	65		
B. Adults (main & second)					
Mean BMI Men	27.9	27.6	28	0.71	0.76
Mean Dim Men	(4.8)	(5.2)	-	0.71	0.70
Mean BMI Women	(4.8) 29.5	(3.2) 27.8	(4.3) 27	0.14	0.04
Mean DMI Women				0.14	0.04
	(7.5)	()	· · ·	0.05	0.65
% Underweight	0.7	1.5	2.2	0.65	0.65
(BMI < 18)					
% Normal weight	29.3	38.6	38		
(BMI 18-25)					
% Overweight	32.7	28.1	32.6		
(BMI > 25)					
% Obese	37.3	31.8	27.2		
(BMI > 30)					
# Obs	150	132	92		

Table 5: Descriptive statistics of body measurements

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

		Children			Adults	
	Perc. BMI	Overweight or Obese	Weight	BMI	Overweight or Obese	Obese
After	0.023	0.022	0.338	0.090	-0.004	-0.006
	(0.015)	(0.037)	(0.543)	(0.200)	(0.026)	(0.023)
1 year follow-up	0.004	-0.045	0.698	0.250	-0.004	0.020
	(0.016)	(0.038)	(0.570)	(0.210)	(0.027)	(0.024)
2 year follow-up	0.011	0.053	0.744	0.276	0.015	0.023
· -	(0.016)	(0.038)	(0.570)	(0.211)	(0.027)	(0.024)
3 year follow-up	0.040**	0.084**	2.632***	1.087***	0.032	0.035
· -	(0.017)	(0.039)	(0.592)	(0.219)	(0.028)	(0.025)
Meal*After	-0.054**	-0.056	0.069	0.020	-0.012	0.006
	(0.023)	(0.054)	(0.795)	(0.294)	(0.038)	(0.034)
Meal*1 year	-0.064***	0.014	0.046	-0.057	-0.018	-0.024
	(0.023)	(0.055)	(0.829)	(0.306)	(0.040)	(0.035)
Meal*2 year	-0.055**	-0.016	1.553^{*}	0.602^{*}	-0.009	0.002
v	(0.023)	(0.055)	(0.831)	(0.307)	(0.040)	(0.035)
Meal*3 year	-0.069***	-0.048	-0.502	-0.137	-0.004	-0.045
·	(0.024)	(0.056)	(0.851)	(0.314)	(0.041)	(0.036)
Snack*After	-0.063**	-0.074	-0.271	-0.166	0.009	0.026
	(0.026)	(0.062)	(0.900)	(0.333)	(0.043)	(0.038)
Snack*1 year	-0.051^{*}	0.071	0.266	-0.070	0.028	0.025
	(0.027)	(0.064)	(0.968)	(0.358)	(0.046)	(0.041)
Snack*2 year	-0.019	0.001	1.228	0.383	0.037	0.006
	(0.027)	(0.064)	(0.966)	(0.357)	(0.046)	(0.041)
Snack*3 year	-0.026	0.026	0.280	0.080	0.033	0.018
•	(0.028)	(0.066)	(1.006)	(0.372)	(0.048)	(0.042)
Constant	0.630***	0.200***	78.274***	28.248***	0.626^{***}	0.336***
	(0.007)	(0.017)	(0.243)	(0.090)	(0.012)	(0.010)
Observations	1,244	1,223	1,592	1,592	1,599	1,599
R-squared	0.035	0.030	0.050	0.065	0.008	0.007
Number of ind.	290	287	381	381	381	381

Table 6: The impact of the Meal and Snack treatment on BMI, overweight and obesity

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

	UK daily Recommendation	Control	Meal	Snack	$\begin{array}{l} \text{P-value} \\ (1) = (2) \end{array}$	P-value (1)=(3)
A: Children					. , . , ,	. ,
A: Children Total calorie intake (Kcal)	1800	1438.9	1463.8	1383.2	034	0.93
Iotal calorie liitake (Real)	1800	(538.6)	(475.4)	(378.0)	054	0.95
# fruit	5 portions fruits and	0.9	1.1	1.1	0.42	0.45
π if all	o portions in this and	(1.26)	-1.4	-1.42	0.42	0.40
# vegetables	Veg.	0.3	0.4	0.4	0.26	0.23
//	8.	(0.58)	(0.85)	(0.78)	0.20	0.20
Fruit and veg (g)	Min 400	101.5	122.4	123.5	0.23	0.27
0 (0)		(124.7)	(126.8)	(141.1)		
Total Fat (g)	Max 70	56.5	` 59.5´	55.1	0.4	0.9
		(24.0)	(25.8)	(20.7)		
Carbohydrate (g)	Max 220	194.7	190.2	182.2	0.77	0.91
· (0)		(86.7)	(65.3)	(50.5)		
Protein (g)	Min 24	47.8	52.5	48.9	0.08	0.5
(0)		(18.8)	(20.2)	(16.7)		
Saturates (g)	Max 20	23.9	25.9	23.8	0.23	0.94
		(11.9)	(12.9)	(11.6)		
Sugar (g)	Max 85	94.2	97.0	87.3	0.2	0.83
		(58.6)	(47.1)	-34.3		
NMES (g)	Max 23	18	25.9	18.1	0.21	0.41
		(22.6)	(34.8)	(20.1)		
Fibre AOAC (g)	Min 15	11.0	10.5	12.0	0.77	0.24
		(5.1)	(5.2)	(5.7)		
Sodium (mg)	2000-3000	1575.9	1621.7	1625	0.93	0.71
(0)		(699.9)	(899.8)	(692.8)		
# Obs		112	104	73		
B: Adults (main & second)						
Total calories intake (Kcal)	2000-2500	2036.1	1843.9	2036.5	0.07	0.91
Total calories meane (Iteal)	2000-2000	(798.1)	(685.2)	(809.2)	0.01	0.91
Portions of fruit	5 portions fruits and	0.94	0.81	1.03	0.53	0.78
	Veg.	(1.86)	(1.48)	(2.67)	0.00	0.10
Portions of vegetables	v تح.	(1.30) 0.77	0.88	(2.07) 0.87	0.48	0.62
i ormono or vegetables		(1.15)	(1.35)	(1.03)	0.40	0.02
Fruit and veg (g)	Min 400	(1.15) 137.5	(1.55) 135.6	(1.03) 151.9	0.93	0.66
r and veg (g)	WIIII 400	(184.8)	(160.9)	(295.6)	0.30	0.00
Total Fat (g)	Max 70	(184.8) 84	(100.9) 74.7	(295.0) 83.4	0.13	0.9
IOUAL L'AU (g)	IVIAX (U	(42.7)	(35.0)	(42.3)	0.10	0.9
Carbohydrate (g)	Max 260	(42.7) 241	(33.0) 223.9	(42.3) 248.9	0.45	0.68
Caroonyurate (g)	WIGA 200	(118.6)	(90.0)	(122.1)	0.40	0.08
Protein (g)	Min 45	(118.0) 79.2	(90.0) 70.1	(122.1) 69.1	0.05	0.21
r roteni (g)	WIIII 40	(55.1)	(32.6)	(27.2)	0.00	0.21
Saturates (g)	Max 20	(35.1) 30.2	(32.0) 28.8	(27.2) 30.2	0.63	0.69
Daturates (g)	1VIAX 20				0.05	0.09
Sugars (g)	Max 90	(17.5) 107.1	$(16.1) \\ 97.9$	(16.0)	0.76	0.65
ougars (g)	wiax 90	107.1 (88.2)		116.1	0.70	0.05
NMES (g)	Max 30	(88.2) 33.2	(56.7) 31.5	$(99.1) \\ 41.3$	0.08	0.06
TATATO (B)	IVIAX OU			(63.0)	0.08	0.00
Fibro $AOAC(a)$	24	(61.7)	(41.0)	· · ·	0.05	0.79
Fibre AOAC (g)	$\angle 4$	13.8	13.5	14.6	0.95	0.72
Sodium (mg)	2400	(6.9)	(6.2)	(8.2)	0.17	0.89
sourum (mg)	2400	2329.4	2139.1	2440.4	0.17	0.88
Alcohol (g)	0	(1246.3)	(1244.6)	-1817	0.71	0.00
Alconol (g)	0	7.1	6.0	9.4	0.71	0.09
(8)		(22.5)	(16.6)	(21.1)		

	D 1'	measures	c	1. 1	· / 1
	Racolino	moggiirog	OT.	diofary	intako
Table 1.	Dascinic	measures	OI.	uluary	mane

Note: Means with standard deviations in parentheses. Col. (4) and (5) report the P-value of a Wilcoxon test of equality of means. 1 portion of fruit or veg approx 80g. NMES: Non-Milk Extrinsic Sugar (NMES, also called free sugars, which are generally considered to be added sugar).

	$_{\rm (cal)}^{\rm Energy}$	Veg (g)	Fruit (g)	Total fat (g)	Carbs (g)	Protein (g)	Sat. fat (g)	Total Sugar (g)	NMES (g)	Fibre (g)	Sodium (mg)	Alcohol (g)
A: Children Sign recommendation - baseline	+	+	+	+	+	-	-	-	+	+	+	
After	-73.1	3.2	9.8	-2.9	-14.2*	1.4	-1.7	-8.4	-3.4	0.1	-89.7	
1 year follow-up	(51.8) 109.3	(5.6) 50.4^{***}	(12.6) 102.8^{***}	(2.6) -0.7	(8.4) 30.6^{**}	(2.2) 2.0	(1.2) -1.9	(5.5) 23.2***	(3.0) 58.8***	(0.7)	(84.7) 121.0	
2-year follow-up	(74.4) 216.5***	(13.8) 50.7***	(20.7) 99.6***	(3.2) 3.8	(12.0) 45.4^{***}	(3.1) 4.7^*	(1.4) -0.5	(7.4) 29.9***	(6.3) 66.3^{***}		(112.1) 304.5^{***}	
3-year follow-up	(81.8) 115.2 (72.5)	(15.0) 61.7^{***}	(18.4) 76.0*** (17.0)	(3.6) 0.8 (2.7)	(13.5) 29.6*** (10.6)	(2.7) 0.7 (2.6)	(1.6) -2.1 (1.7)	(9.6) 11.4 (7.1)	(8.0) 51.9***		(112.6) 274.0** (116.2)	
Meal*After	(72.5) -32.8	(11.5) -9.9	(17.9) -4.8	(3.7) -4.1	(10.6) 4.8	(2.6) -3.0	-2.4	(7.1) -0.4	(6.1) -4.0	0.7	(116.2) -69.0	
Meal*1 year	(71.6) -101.4	$(9.6) \\ 14.6$	$(19.0) \\ 3.0$	(3.8) -6.0	(11.0) -8.9	(3.4) -3.7	(1.8) -3.1	(7.9) -15.9	(5.4) -21.9***	(0.9)	$(115.2) \\ -80.5$	
Meal*2-year	(106.2) -162.0	(24.8) -9.1	(41.9) -33.4	(5.3) -8.6	(15.7) -18.7	(4.4) -3.9	(2.5) -3.1	(10.2) -24.6**	(8.0) -26.3**		(159.8) -154.0	
Meal*3-year	(115.3) 87.3 (116.2)	(22.0) -22.6 (15.2)	(28.2) 9.5 (28.6)	(5.4) 0.3	(17.6) 17.9 (16.5)	(4.4) 4.1 (5.0)	(2.5) -0.1 (2.6)	(12.0) 8.5 (10.2)	(10.2) 2.4 (8.0)		(166.8) -18.6 (170.5)	
Snack*After	(116.3) -40.8 (76.0)	(15.2) -5.5 (11.3)	(28.6) -10.6 (20.2)	(5.5) -2.8 (4.1)	(16.5) 1.8 (11.7)	(5.0) -4.5 (3.4)	(2.6) -1.1 (2.1)	(10.3) -0.7 (7.4)	(8.9) -0.0 (4.8)	-0.7 (1.1)	(179.5) -109.9 (138.4)	
Snack*1 year	-120.5 (99.8)	-39.6^{**} (17.4)	(20.2) -17.9 (34.6)	-1.8 (4.8)	(11.1) -21.2 (15.1)	-5.9 (4.2)	-1.0 (2.4)	-13.6 (10.0)	-20.4** (8.7)	(1.1)	(100.4) -115.4 (174.3)	
Snack*2-year	(108.5) (108.5)	-9.2 (22.2)	(3.10) -43.5 (28.5)	(4.9)	(22.9) (17.1)	-6.3 (4.0)	-2.7 (2.4)	(10.0) -14.7 (12.3)	-20.5^{*} (10.7)		-327.2^{**} (162.7)	
Snack*3-year	-57.6 (103.8)	-37.5^{*} (19.1)	11.8 (30.3)	-2.9 (5.4)	-7.9 (14.6)	-0.2 (4.2)	-0.6 (2.5)	-0.4 (10.1)	-7.6 (9.0)		-130.1 (188.4)	
Constant	$^{1,416.0***}_{(22.6)}$	32.5^{***} (2.9)	87.7^{***} (5.9)	56.7^{***} (1.2)	$ \begin{array}{c} 187.4^{***} \\ (3.3) \end{array} $	49.1^{***} (1.0)	24.5^{***} (0.6)	91.8^{***} (2.3)	20.4^{***} (1.8)	(0.2)	$1,570.5^{***}$ (37.8)	
# Obs	1,261	859	1,030	1,261	1,261	1,261	1,261	1,261	1,257	554	1,261	
R-squared # of ind.	$0.04 \\ 292$	$0.15 \\ 292$	0.12 290	$0.02 \\ 292$	$0.07 \\ 292$	0.01 292	$0.02 \\ 292$	0.06 292	0.33 291	$0.01 \\ 287$	0.04 292	
B: Adults Sign recommendation - baseline	+	+	+	-	+	-	-	_	-	+	+	-
After	-268.4^{***} (85.9)	-14.8 (9.4)	-25.9 (15.7)	-9.8** (5.0)	-34.4^{***} (10.2)	-7.4 (6.1)	-2.7 (2.1)	-22.6^{***} (7.3)	-12.2** (5.4)	-2.0^{**} (0.8)	-280.4^{*} (152.0)	$^{-2.9}_{(2.0)}$
1 year follow-up	(33.9) -422.3^{***} (107.9)	(9.4) 59.0*** (21.1)	(13.7) 132.7^{***} (24.3)	(3.0) -26.8^{***} (5.3)	(10.2) -19.5 (14.2)	(0.1) -22.1^{***} (6.3)	(2.1) -8.2^{***} (2.6)	(7.3) 1.2 (10.1)	(3.4) 41.7^{***} (8.3)	(0.8)	(132.0) -461.7*** (177.8)	(2.0) 28.6^{**} (12.1)
2-year follow-up	(107.3) -131.9 (151.3)	63.6^{***} (18.7)	(24.3) 98.9*** (30.9)	(3.3) -10.2 (7.1)	6.7 (19.3)	(0.3) -10.5 (7.9)	-3.4 (2.9)	-3.8 (8.9)	32.7^{***} (7.1)		(177.8) -4.3 (271.9)	(12.1) 34.0*** (8.9)
3-year follow-up	-368.2^{***} (125.4)	(10.1) 118.2^{***} (27.1)	98.7^{***} (30.2)	(7.1) -17.7^{**} (7.2)	(15.0) -24.4 (16.1)	-23.5*** (6.2)	-6.2** (2.6)	-18.5** (8.2)	23.3^{***} (7.1)		-388.1 (240.2)	9.9 (13.1)
Meal*After	(123.4) 89.1 (112.9)	-2.9 (15.6)	-5.0 (20.0)	2.6 (6.2)	(10.1) 11.1 (14.3)	2.0 (7.3)	-1.3 (2.8)	5.2 (9.4)	(7.1) 1.1 (7.0)	1.3 (1.2)	(240.2) -29.5 (208.8)	(13.1) 1.7 (2.6)
Meal*1 year	337.3^{*} (182.8)	(20.0) -23.4 (29.3)	(20.0) 10.5 (35.5)	(3.2) 14.3* (8.3)	(23.5)	16.7^{*} (8.9)	2.0 (3.7)	8.0 (12.7)	-3.8 (9.9)	(112)	(200.0) 274.7 (297.5)	-7.6 (14.3)
Meal*2 year	52.7 (192.5)	9.2 (29.2)	9.6 (43.7)	2.7 (9.1)	(25.0) -1.3 (25.2)	3.4 (9.5)	0.2 (3.7)	(12.1) 1.4 (12.3)	-2.4 (9.9)		-57.8 (353.6)	-0.5 (12.8)
Meal*3 year	(152.0) 110.6 (151.9)	(23.2) -57.9^{*} (33.4)	(40.1) 35.3 (47.9)	(3.1) 4.8 (8.6)	6.3 (19.9)	(3.6) 13.9^{*} (7.2)	1.0 (3.3)	(12.0) 15.8 (11.4)	9.7 (9.8)		-31.7 (282.3)	-2.9 (16.2)
Snack*After	(131.9) -176.0 (132.5)	(33.4) -21.4 (18.6)	(47.9) -14.9 (42.8)	-8.4 (7.3)	(19.9) -23.2 (18.8)	-3.5 (7.3)	-4.0 (3.1)	(11.4) -21.3 (13.8)	-8.9 (9.3)	-0.8 (1.3)	(282.3) -381.3 (272.5)	(10.2) 0.2 (3.9)
Snack*1 year	(132.5) 23.7 (167.8)	-9.2 (36.4)	(42.8) 47.6 (67.5)	2.3 (8.1)	(13.8) -13.6 (24.5)	(7.3) 18.6** (8.4)	(3.1) 1.3 (3.6)	-26.0 (17.3)	(3.3) -25.0^{*} (13.3)	(1.0)	-254.3 (320.3)	(3.3) -15.3 (15.5)
Snack*2 year	(107.8) -67.2 (236.4)	(30.4) 4.5 (34.9)	(67.3) 28.4 (63.0)	(3.1) -5.1 (10.5)	(24.3) -10.7 (33.1)	6.9 (10.0)	(3.0) 0.9 (4.2)	-2.3 (18.9)	(13.3) 7.6 (16.9)		(320.3) -150.6 (466.8)	(13.3) -20.3^{*} (11.9)
Snack*3 year	(230.4) -351.4^{**} (176.4)	(34.9) -61.7 (38.6)	9.3 (50.9)	(10.3) -16.6* (9.3)	(33.1) -58.4^{**} (25.2)	(10.0) 7.2 (7.9)	(4.2) -4.2 (3.6)	-22.1 (15.9)	(10.9) -14.3 (12.5)		(400.8) -771.7** (364.3)	9.2 (19.3)
Constant	(170.4) $1,970.9^{***}$ (40.0)	(38.6) 73.6^{***} (4.9)	(50.9) 84.6^{***} (8.1)	(9.3) 80.3^{***} (2.0)	(25.2) 238.0*** (5.3)	(7.9) 74.1^{***} (2.1)	(3.6) 29.7*** (0.9)	(15.9) 106.4^{***} (3.4)	(12.5) 35.2^{***} (2.5)	14.2^{***} (0.2)	(304.3) 2,330.7*** (76.8)	(19.3) 8.6^{***} (0.9)
# Obs.	1,473	1,037	1,002	1,472	1,473	1,473	1,472	1,473	1,458	628	1,473	736
R-squared # of ind.	0.04 366	0.14 353	0.21 354	0.05 366	0.04 366	0.03	0.03 366	0.04 366	0.15 366	0.05	0.03 366	0.19 342

Table 8: The impact of Meal and Snack treatments on total calorie intake and intake of macronutrients

 $\frac{1}{\# \text{ of ind.}} = \frac{1}{366} + \frac{1}{353} + \frac{1}{354} + \frac{1}{366} + \frac{1}{3$

	Control	Meal	Snack	P-value	P-value
				(1)=(2)	(1)=(3)
A. Children					
I. Item categories					
Sweets	3.6(0.7)	3.6(0.8)	3.6(0.6)	0.25	0.99
Bread	3.4(0.9)	3.6(0.6)	3.3(0.9)	0.03	0.65
Processed food	3.3(0.6)	3.5(0.5)	3.3(0.7)	0.03	0.67
Fruit	3.2(0.8)	3.3~(0.7)	3.1(0.8)	0.28	0.47
Cheese	3.1(1.1)	3.4(1.0)	3.4(0.9)	0.02	0.04
Meat/Fish/Eggs	2.6(0.9)	2.7(0.9)	2.8(0.9)	0.77	0.32
Vegetables	2.6(0.8)	2.6(0.8)	2.5(0.8)	0.88	0.35
II. Meals					
Tuna pasta	2.5(1.3)	2.7(1.3)	2.7(1.2)	0.33	0.36
Omelette	2.4(1.3)	2.3(1.3)	2.2(1.2)	0.8	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
B. Adults					
I. Item categories	24(07)	$\mathbf{D} \mathbf{F} (\mathbf{O} \mathbf{F})$	$\mathbf{a} \mathbf{a} (\mathbf{a} \mathbf{r})$	0.11	0.01
Fruit	3.4(0.5)	3.5(0.5)	3.3(0.5)	0.11	0.21
Meat/Fish/Eggs	3.3(0.6)	3.3(0.6)	3.3(0.6)	0.62	0.87
Cheese	3.3(0.7)	3.5(0.7)	3.5(0.7)	0.11	0.06
Vegetables	3.2(0.6)	3.2(0.6)	3.2(0.5)	0.96	0.81
Bread	3.2(0.7)	3.2(0.7)	3.3(0.7)	0.93	0.29
Processed food	3.1 (0.5)	3.0(0.5)	3.1(0.5)	0.71	0.35
Sweets	2.8(0.7)	2.8(0.6)	2.9(0.6)	0.68	0.59
II. Meals					
Turkey stir fried	3.2(0.9)	3.2(1.0)	3.3(0.9)	0.62	0.38
Salmon with onions	3.1(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

Table 9: Baseline food preferences

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

			Meat				
	Fruits	Vegetables	Fish Eggs	Processed Food	Sweets	Bread	Cheese
Panel A: Children	114105	regetableb		1000		Dicad	Cheebe
After	0.13**	-0.06	0.00	0.07	-0.14**	0.05	0.20**
	(0.06)	(0.07)	(0.10)	(0.05)	(0.07)	(0.09)	(0.10)
1-year	0.05	-0.01	0.16	0.06	-0.09	0.01	0.02
	(0.07)	(0.08)	(0.10)	(0.07)	(0.08)	(0.10)	(0.11)
2-year	0.09	-0.09	0.02	0.08	-0.16**	0.03	0.03
	(0.09)	(0.09)	(0.12)	(0.07)	(0.08)	(0.11)	(0.13)
3-year	0.03	-0.11	0.11	-0.07	-0.29***	-0.14	-0.24*
	(0.09)	(0.09)	(0.11)	(0.07)	(0.09)	(0.10)	(0.14)
Meal x After	-0.10	0.01	-0.07	-0.21***	0.17^{*}	-0.17	-0.33**
	(0.09)	(0.10)	(0.14)	(0.08)	(0.09)	(0.13)	(0.13)
Meal x 1-year	-0.01	0.03	-0.08	-0.09	0.10	-0.11	-0.08
	(0.10)	(0.11)	(0.15)	(0.09)	(0.13)	(0.13)	(0.16)
Meal x 2-year	-0.06	0.04	-0.08	-0.13	0.23*	-0.21	-0.19
	(0.12)	(0.12)	(0.16)	(0.09)	(0.13)	(0.14)	(0.18)
Meal x 3-year	-0.04	0.08	-0.20	-0.03	0.20	-0.13	0.03
	(0.12)	(0.12)	(0.16)	(0.09)	(0.13)	(0.14)	(0.18)
Snack x After	0.03	0.13	-0.14	0.02	0.21**	0.24^{*}	-0.18
G 1 1	(0.11)	(0.11)	(0.14)	(0.09)	(0.10)	(0.15)	(0.16)
Snack x 1-year	0.15	0.13	-0.26	0.04	-0.04	0.13	-0.12
Smoole as 0 areas	(0.12)	(0.12)	(0.19)	(0.10)	(0.15)	(0.14)	(0.17)
Snack x 2-year	0.16	0.26^{**}	-0.19	0.15	0.18	0.12	-0.21
Smoole er 2 eroom	(0.13)	(0.12)	(0.18)	(0.12)	(0.11)	(0.17)	(0.20)
Snack x 3-year	0.09	0.06	-0.17	0.07	-0.10	-0.04	-0.23
Constant	(0.15) 3.19^{***}	(0.11) 2.58^{***}	(0.18) 2.66^{***}	(0.12) 3.35^{***}	(0.14) 3.52^{***}	(0.17) 3.47^{***}	(0.21) 3.29^{***}
Constant	(0.03)	(0.03)	(0.04)	(0.02)	(0.03)	(0.04)	(0.04)
	(0.03)	(0.03)	(0.04)	(0.02)	(0.03)	(0.04)	(0.04)
# Obs	1,243	1,254	1,254	1,257	1,251	1,248	1,244
R-squared	0.01	0.01	0.01	0.02	0.04	0.03	0.03
# individuals	288	289	289	289	289	289	288
Panel B: Adults							
After	-0.01	0.00	0.00	-0.04	-0.02	-0.01	0.02
	(0.03)	(0.03)	(0.04)	(0.03)	(0.05)	(0.06)	(0.05)
1-year follow up	0.01	-0.01	0.02	-0.05	-0.04	-0.16**	-0.10*
•	(0.03)	(0.03)	(0.04)	(0.04)	(0.05)	(0.06)	(0.05)
2-year follow up	0.01	-0.03	0.01	-0.01	-0.12**	-0.09	-0.10*
	(0.03)	(0.03)	(0.05)	(0.04)	(0.05)	(0.07)	(0.06)
3-year follow up	0.01	0.01	-0.00	-0.03	-0.09	-0.03	0.00
	(0.03)	(0.04)	(0.05)	(0.04)	(0.06)	(0.07)	(0.06)
Meal*After	0.06	0.00	-0.03	0.10**	0.04	-0.11	0.02
	(0.04)	(0.04)	(0.06)	(0.05)	(0.07)	(0.08)	(0.08)
Meal x 1-year	-0.01	0.04	-0.08	0.03	-0.02	0.11	0.08
	(0.05)	(0.04)	(0.06)	(0.05)	(0.07)	(0.09)	(0.08)
Meal x 2-year	-0.08*	0.06	-0.07	-0.04	0.07	0.07	0.06
	(0.04)	(0.04)	(0.06)	(0.06)	(0.07)	(0.09)	(0.09)
Meal x 3-year	-0.03	-0.01	-0.03	0.04	0.08	-0.06	0.01
	(0.05)	(0.05)	(0.07)	(0.06)	(0.08)	(0.10)	(0.09)
Snack x After	0.09	0.06	-0.09	-0.01	-0.00	-0.11	-0.10
a 1 -	(0.06)	(0.04)	(0.06)	(0.06)	(0.08)	(0.10)	(0.08)
Snack x 1-year	-0.04	0.00	-0.06	-0.00	0.06	0.04	0.03
	(0.06)	(0.05)	(0.06)	(0.07)	(0.08)	(0.09)	(0.10)
Snack x 2-year	-0.05	0.01	0.00	0.06	0.13	0.05	0.00
C	(0.06)	(0.05)	(0.07)	(0.07)	(0.09)	(0.10)	(0.10)
Snack x 3-year	-0.01	0.03	-0.09	-0.06	0.04	-0.19	-0.09
Constant	(0.06)	(0.06)	(0.07)	(0.08)	(0.10)	(0.12)	(0.10)
Constant	3.43^{***}	3.20^{***}	3.30^{***}	3.07^{***}	2.84^{***}	3.23^{***}	3.40^{***}
	(0.01)	(0.01)	(0.02)	(0.01)	(0.02)	(0.02)	(0.02)
# Obs	1,588	1,589	1,576	1,589	1,580	$1,\!550$	$1,\!551$
R-squared	0.01	0.01	0.01	0.01	0.01	0.02	0.01
# individuals	380	380	380	380	379	378	377

Table 10: The impact of Meal and Snack treatments on food preferences

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

	% spent		D. A	Saturated	% spent on	
	Calories	on fruit	Fat	Fat	Sugar	unhealthy
	(kcal)	& veg	(g)	(g)	(g)	items
Panel A: Year 2						
Meal	-634	0.01	-19.7	-8.4	-99.2***	-0.041**
	(429)	(.019)	(14.3)	(6.1)	(37.9)	(.021)
Snack	-136	0.017	2.99	0.87	-3.13	-0.013
	(457)	(.024)	(18.2)	(7.50)	(41.3)	(.024)
# Obs	275	275	275	275	275	275
R-squared	0.021	0.046	0.042	0.048	0.032	0.067
Panel B: Year 3						
Meal	89.1	-0.004	-18.2	-4.02	-18.9	-0.048
	(446.5)	(.021)	(14.82)	(6.64)	(34.0)	$(.023)^{**}$
Snack	534	-0.018	-1.82	1.18	56.7	0.028
	(548)	(.028)	(18.5)	(8.13)	(42.4)	(.027)
Observations	264	264	264	264	264	264
R-squared	0.013	0.027	0.024	0.026	.019	0.069

Table 11: Incentivized Supermarket Choice Adults - Years 2 and 3

Note: Each column in each panel represents a separate linear regression controlling for age and gender of the decision maker. Standard errors in parentheses. *** p<0.01. Unhealthy items are classified as foods with 4 more points and drinks with 1 or more points as determined by the UK's Food Standard Agency (FSA) nutrient profiling technique. For full details of how the points are calculated please see https://assets.publishing.service.gov.uk/government/uploads/system/uploads/attachment_data/file/216094/dh_123492.pdf

Number of unhealthy items cho				
Meal	0.048	0.061	0.085	
	(.164)	(.168)	(.170)	
Snack	-0.184	-0.15	-0.132	
	(.192)	(.196)	(.197)	
Risk preference		0.006	0.003	
		(.031)	(.031)	
Controls for gender and age	Ν	Ν	Y	
Observations	212	212	212	
R-squared	0.006	0.007	0.0165	

Table 12: Children's incentivized task in Year 3

Note: Each column in each panel represents a separate linear regression.

Table 13: Number of low-calories choices, incentivized

	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

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

Table 14: Baseline health biomarkers (based on fasted blood samples) - Levels

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

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

Table 15: The impact of the Meal and Snack treatment on blood biomarkers

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