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Household Responses to Information on Child Nutrition: Experimental Evidence from Malawi

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Abstract

Incorrect knowledge of the health production function may lead to inefficient household choices, and thereby to the production of suboptimal levels of health. This paper studies the effects of a randomised intervention in rural Malawi which, over a six-month period, provided mothers of young infants with information on child nutrition without supplying any monetary or in-kind resources. A simple model first investigates theoretically how nutrition and other household choices including labour supply may change in response to the improved health knowledge observed in the intervention areas. We then show empirically that, in line with this model, the intervention improved household consumption, child nutrition and consequently health. These increases are funded by an increase in the labour supply of fathers. We consider and rule out alternative explanations behind these findings. This paper is the first to establish that non-health choices, particularly parental labour supply, are affected by parents' knowledge of the child health production function.

Keywords: Infant Health, Health Information, Labour Supply, Cluster Randomised Control Trial
JEL classification: D10, I15, I18, O12, O15

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1. Introduction

Since Becker's (1965) seminal article, economists have long recognised that many goods are not directly bought in the market, but are produced at home using a combination of market and non-market goods. The home production framework has been particularly fruitful in studying the production of health, in particular child health (Grossman 1972, Rosenzweig and Schultz 1983, Gronau 1986 and 1997). An important implication of such models is that households make choices given their knowledge of the (child) health production function. Consequently, deficiencies in knowledge lead to suboptimal household choices and thereby distorted levels of child health. Establishing empirically the consequences of deficiencies in knowledge on household behaviour has, however, been challenging because knowledge is endogenous and is usually either unobserved or proxied by education which also affects child health through other channels including earnings.

In this paper, we overcome this challenge by exogenously improving mothers' knowledge of the production function through a cluster randomised trial in rural Malawi, which, in solely providing information on child nutrition to mothers, yields a clean source of identification. Our contribution is twofold. First we assess whether the intervention improved child nutrition and consequently health. Second, drawing on a simple theoretical model, we investigate how other household choices - in particular labour supply - change to accommodate the improved knowledge of the production function. In so doing, we establish empirically that non-health choices, particularly parental labour supply, are affected by parents' knowledge of the child health production function. This finding is a key contribution of the paper.

In the context we study, rural Malawi, mothers have many misconceptions about child nutrition. To take some examples, it is common practice to give porridge diluted with unsterilized water to infants as young as one week; the high nutritional value of groundnuts, which are widely available in the area, is not well-known; and widespread beliefs include that eggs are harmful for infants as old as 9 months, and that the broth of a soup contains more nutrients than the meat or vegetables therein. This evidence suggests that important changes can be expected if these misconceptions are corrected. Moreover, the fact that Malawi is a predominantly matrilineal society, where women have more power than in patrilineal societies, means that targeting mothers is likely to be an effective way of improving children's health.

The intervention that we study delivered information in an intense manner: trained local women visited mothers in their homes once before the birth of their child and four times afterwards, and provided information on early child nutrition on a one-to-one basis. It is important to stress that the fact that the intervention had been running for at least 3 years when outcome data were collected, allows for a sufficient time-frame for both, practices to change and for information to diffuse to the broader community. Consistent with this, we find not only that women's knowledge of child's nutrition improved, but also that child nutrition becomes more of a talking point with friends, suggesting that the salience of child nutrition also increased due to the intervention.

Consistent with the improvement in knowledge, as well as the increased salience of child nutrition, we find evidence of improvements in infants' diets and total household consumption, particularly of protein-rich foods and of fruit and vegetables. Further, we find strong evidence to suggest that these improvements are funded by increases in adult labour supply, specifically that of fathers. Overall, the findings are consistent with households learning that some relatively costly foods are more nutritious than they previously believed, and adjusting their labour supply so as to facilitate increases in their children's intake of them. Indeed, we show that households adjust their behaviour on several margins including child diet inputs and adult labour supply, making their response more complex than simply changing the composition of consumption while keeping total consumption constant.

We find that improving knowledge of child nutrition increases children's height, a widely used indicator of long-term nutritional status. This finding is particularly important for policy: malnutrition is a severe and prevalent problem in developing countries where around one third of children below the age of five are stunted in growth (de Onis *et al.* 2000) and almost half of all child mortality is associated with malnutrition (Pelletier *et al*. 1995). Moreover, malnutrition in infanthood not only decreases welfare, but is also linked to poor cognitive and educational performance and low productivity later on in life.[5]

[5]For long-term consequences of poor health or nutritional status in infanthood on long-term outcomes see, among others, Behrman 1996, Strauss and Thomas 1998, Glewwe *et al.* 2001, Alderman *et al*. 2001, Behrman and Rosenzweig 2004, Schultz 2005, Van den Berg *et al.* 2006, Hoddinott *et al.* 2008, Maluccio *et al.* 2009, Banerjee *et al.* 2010, Currie *et al.* 2009, Van den Berg *et al.* 2009, Maccini and Yang 2009, Currie 2010, Van den Berg *et al.* 2010, Lindeboom *et al.* 2010, Almond and Currie 2011, Barham 2012.

A further contribution of this paper is to consider spillover effects of the intervention on children not directly targeted by it, but who could have benefited from the new information diffusing in the community (Miguel and Kremer 2003, Angelucci and De Giorgi 2009, Janssens 2011). Investigating intervention effects on relatively older children who were born before the intervention began and thus not directly targeted by it, we find evidence of spillovers on food intake (in particular, increases in food diversity) but not on indicators of medium- or long-term health (specifically, weight and height). The latter is not all that surprising, as height is less malleable at older ages (Schroeder *et al.*, 1995).

Our work fits into the growing literature on the importance of information for health. A recent review by Dupas (2011a) suggests that the provision of health-related information can have significant impacts on health behaviour. For instance, providing specific information - such as the arsenic or fecal concentration of water (Madajewicz *et al.* 2007; Jalan and Somanathan 2008) - affects associated practices; Dupas (2011b) shows that teenage girls change their sexual behaviour in response to information on the risks of contracting HIV. There is also evidence that information campaigns about specific prevention practices can affect household behaviour - such as the promotion of oral rehydration therapy (Levine *et al.* 2004) and hand washing (Wilson and Chandler 1993).[6] Our work departs from these studies not only by considering a broader and more multifaceted type of information (ways to improve child nutrition), but also by studying the responses of households on a wider range of household margins - with a particular focus on labour supply - than those directly targeted by the intervention. In doing so, this is one of the first papers to investigate how individual and household behaviours not directly related to the topic of an information campaign adjust in response to it.

Our paper also contributes to the literature evaluating the effects of interventions that provide nutrition information on child health. Morrow *et al.* (1999) and Haider *et al.* (2000) have studied effects of similar interventions on feeding practices only (specifically exclusive breastfeeding) within small scale randomised controlled trials in Mexico and Bangladesh respectively. Further, a set of mostly non-experimental studies has investigated the effects of similar interventions on health outcomes, finding improvements in child weight-for-age, an

[6]However, it need not always be the case that providing information is sufficient to shift health behaviour. Kamali *et al.* (2003), Kremer and Miguel (2007) and Luo *et al.* (2012) find that health education does not change health behaviours relating to respectively HIV in Uganda, deworming in Kenya and anaemia in China.

indicator of medium-term health status (Alderman 2007, Linnemayr and Alderman 2011, Galasso and Umapathi 2009). This paper builds on this literature by considering the effects on child health, health practices, and other margins of household behaviour, all identified through a randomised controlled trial.

Finally, our paper relates to the literature investigating the causal effects of parental education on child health. Much of the literature relates to developed countries and provides mixed evidence. Currie and Moretti (2003) and McCrary and Royer (2011) find respectively, decreased incidence of low birthweight and modest effects on child health of increased maternal schooling in the US, while Lindeboom *et al.* (2009) find little evidence that parental schooling improves child health in the UK. For developing countries, we are only aware of Breierova and Duflo (2004) and Chou *et al.* (2010) who find that parental schooling decreases infant mortality in Indonesia and Taiwan respectively. However, it is difficult to disentangle whether the effect of education is working through changes in knowledge of the child production function, or through increased income and hence access to more and better quality care. Related to this, Thomas *et al.* (1991) and Glewwe (1999) finds that almost all of the impact of maternal education on child's height in Brazil and Morocco can be explained by indicators of access to information and health knowledge.

The rest of the paper is structured as follows. Section 2 provides some background information on rural Malawi and describes the experimental design, section 3 describes the theoretical framework, while section 4 sets out the empirical model and data. Our main results are presented in section 5, and section 6 contains an analysis of spillovers. Section 7 considers, and rules out, alternative potential explanations behind our findings, section 8 considers the internal validity of our results while section 9 concludes.

2. Background and Intervention

2.1 Background

Malnutrition in the early years (0-5) has important, potentially devastating, short- and long-run effects. It leaves children vulnerable to other illnesses and diseases, threatening their very survival (Bhutta *et al*. 2008) and affects longer term outcomes such as schooling, adult health and productivity (Glewwe *et al.* 2001, Maluccio *et al.* 2009). It is one of the major public health and development challenges facing Malawi, one of the poorest countries in Sub-

Saharan Africa. The Malawi Demographic and Health Survey (DHS) Report for 2004 indicates an under-five mortality rate of 133 per 1000, and under-nutrition is an important factor driving this: Pelletier *et al*. (1994) estimate that 34% of all deaths that occur before age 5 in Malawi are related to malnutrition (moderate or severe). Stunting, in other words being too short for one's age, is a primary manifestation of chronic malnutrition in early childhood. In Malawi, 48% of children younger than 5 are stunted, a rate that is the second highest in sub-Saharan Africa, and one of the highest in the world. It is 24 times the level expected in a healthy, well-nourished population. Further, 22% of children under the age of 5 are underweight for their age, which is 11 times the level expected in a healthy, well-nourished population.

Poor feeding practices are at least partly responsible for these extreme malnutrition indicators. Over half of all infants below 6 months of age are given food and/or unsterilized water (DHS, 2004), which can lead to gastrointestinal infections and growth faltering (Haider *et al.* 1996, Kalanda *et al.* 2006) and is contrary to World Health Organization (WHO) recommendations.[7] Furthermore, porridge diluted with unsterilized water is often given in large quantities to infants as young as one week (Kerr *et al.* 2007). In terms of nutrition for infants above 6 months of age, their diets frequently lack the necessary diversity of foods to provide sufficient amounts of energy, proteins, iron, calcium, zinc, vitamins and folate recommended by the WHO[8]: indeed in our sample, 25% of children aged 6-60 months did not consume any proteins over the three days prior to the survey, with a further 30% having consumed just one source of protein. Poor nutritional practices are likely to be related to a lack of knowledge: for instance, only 15% of mothers in our sample knew how to best cook fish combined with the local staple so as to maximise nutritional value.

It is against this background that, in 2002, a research and development project called MaiMwana (Chichewa for "Mother and Child") was set up in Mchinji District, in the Central region of Malawi.[9] Its aim was to design, implement and evaluate effective, sustainable and

[7]The WHO recommends exclusive intake of breastmilk for the first six months. Though there is a risk of vertical (mother to child) transmission of HIV through breastfeeding, reducing this risk by choosing not to breastfeed is not a viable option in rural Africa, where formula feeds are expensive and not commonly available and local water supplies are unlikely to be safe.

[8]Commonly eaten staples, such as maize flour, rarely have sufficient protein and micronutrients required for healthy growth and development (WHO, 2000).

[9]MaiMwana is a Malawian trust established as a collaboration between the Department of Paediatrics, Kamuzu Central Hospital, the Mchinji District Hospital and the UCL Centre for International Health and Development. See http://www.maimwana.malawi.net/MaiMwana/Home.html

scalable interventions to improve the health of mothers and infants. Mchinji is a primarily rural district, with subsistence agriculture being the main economic activity. The most commonly cultivated crops are maize, groundnuts and tobacco. The dominant ethnic group in the district is the Chewa (over 90% in our data). Socio-economic conditions are comparable to or poorer than the average for Malawi (in parentheses in what follows), with literacy rates of just over 60% (64%), with poor quality flooring materials used by 85% (78%) of households, piped water access for 10% (20%) of households, and electricity access for just 2% (7%) of households.[10]

2.2 The Intervention

In 2005, MaiMwana established an infant feeding counselling intervention in Mchinji District (still ongoing), to impart information and advice on infant feeding to the mothers of babies aged less than six months.[11] The intervention thus targets the very first years of life, a critical period for growth and development during which nutritional interventions are likely to be most beneficial (Shroeder *et al.* 1995, Shrimpton *et al*. 2001, Victora *et al*. 2010). The information is provided by trained female volunteers ("counsellors" hereon) nominated by local leaders. Each counsellor covers an average population of 1,000 individuals, identifying all pregnant women within this population and visiting them five times in their homes: once before giving birth (3rd trimester of pregnancy) and four times afterwards (baby's age 1 week, 1 month, 3 months, 5 months). Although all pregnant women are eligible for the intervention and participation is free[12], in practice around 60% of them are visited by the counsellors.[13]

In terms of the content of the visits, exclusive breastfeeding is strongly encouraged in all visits starting from the very first. Information on weaning is provided from when the baby is 1 month old (visits 3-5) and includes suggestions of suitable locally available nutritious foods, the importance of a varied diet (particularly, the inclusion of protein and micronutrient-rich foods, including eggs) and instructions on how to prepare foods so as to conserve nutrients and ease digestion (for instance to mash vegetables rather than liquidise them; to pound fish before cooking it). Counsellors were provided with a manual to remind

[10] Source: Malawi Population and Housing Census, 2008.
[11] Though the intervention is predominantly focused on nutrition, it also covers other issues relevant in this context such as birth preparedness, HIV testing and counselling, vaccinations, and family planning. See subsection 7.3 for a discussion of how these aspects of the intervention relate to our results.
[12] See Chassang *et al.* (2012) for a discussion on whether individuals should be charged or not to participate.
[13] Possible reasons why 40% of eligible mothers are not visited include constraints on the counsellors' time availability and mothers' availability and interest. Our analysis is an Intent to Treat one, as we discuss in section 4.

them of the content relevant for each visit, and simple picture books to aid in explaining concepts.

2.2.1 Experimental Design

The evaluation is based on a cluster randomised controlled trial designed as follows (see Lewycka *et al*. 2010, Lewycka 2011). Mchinji District was divided into 48 clusters by combining enumeration areas of the 1998 Malawi Population and Housing Census.[14] This was done in a systematic way, based on the contiguity of enumeration areas and respecting boundaries of Village Development Committees (VDCs)[15], such that each cluster contained approximately 8,000 individuals. Within each cluster, the 3,000 individuals (equating to 14 villages on average) living closest to the *geographical* centre of the cluster were chosen to be included in the study.[16] The study population therefore comprises of individuals living closest to the geographical centre of the clusters and was selected in this way in order to limit contamination between neighbouring clusters by creating a natural buffer area. 12 clusters were randomly selected to receive the infant feeding counselling intervention, with an average of three counsellors covering each cluster. A further 12 serve as controls.[17, 18]

2.2.2 Evaluation Sample Description

A census of women of reproductive age was conducted by MaiMwana in all of the clusters in 2004, before the intervention started ("baseline census" from hereon) in July 2005.[19] Approximately 3.5 years into the intervention, which is still in place, we drew a random sample from the baseline census in order to conduct the first follow-up survey.[20] Specifically,

[14] The District Administrative Centre was excluded because it is relatively more urbanised and hence less comparable to the rest of the District.
[15] This is an administrative area in Malawi, which groups together a number of villages, and is headed by a Group Village Headman (GVH).
[16] The geographic centre was chosen to be the most central village in the cluster as shown on a cartographic map from the National Statistical Office, Malawi, and whose existence was corroborated with the District Commissioner's records. See Lewycka (2011), pp. 122 for more details.
[17] MaiMwana Project also improved health facilities across the District, which benefitted both intervention and control clusters equally.
[18] Another 24 clusters were randomly assigned to receive another intervention - participatory women's groups - whereby women of reproductive age were encouraged to form groups to meet regularly and discuss and resolve issues relating to pregnancy, child birth and neo-natal health. Child nutrition was not a primary focus of this intervention and hence we exclude these clusters from this analysis (Rosato *et al.* 2006 and Rosato *et al.* 2009 contain a summary of issues covered by the groups).
[19] Further details on this baseline census can be found in Lewycka *et al.* (2010). We take the intervention start date to be July 2005. This is the date by which the first 6-month cycle had been fully completed.
[20] Data collection was carried out by MaiMwana in collaboration with the authors of this paper. The data were collected during Nov2008-March 2009 (Oct 2009-Jan 2010) in the first (second) follow-up. To ensure that results were not driven by seasonality, field teams collected data in both intervention and control clusters at the

in 2008 we drew a random sample of 104 women of reproductive age (17-43), regardless of their child bearing status[21], from each of the 24 clusters, leaving us with a target sample of 2,496 women. The baseline census contains some socio-economic and demographic characteristics of these women and their households, as shown in Table 1. Women are on average 24.5 years old, just over 61% of them are married, over 70% have some primary schooling but just 6% have some secondary schooling, and 66% reported agriculture as their main economic activity. Households are predominantly agricultural and poverty is high, as indicated by the housing materials and assets. The table also shows that the randomisation worked well with the sample well-balanced across intervention and control areas at baseline. A small imbalance is detected on only 1 out of 25 variables.[22]

[TABLE 1 HERE]

We succeeded in interviewing around two thirds of the sample drawn for the first follow-up survey: 65% in intervention clusters and 67% in control clusters. There are three main factors contributing to the attrition. First, the first follow-up was carried out 3.5 years after the baseline.[23] Second, the district of Mchinji is particularly challenging for the collection of panel data because respondents are known to report "ghost members" - fictitious household members - with the intention of increasing future official aid/transfers which may depend positively on household size (see Miller and Stoka 2012 for "ghost members" and Giné, Goldberg and Yang 2012 for problems related to personal identification in Malawi). Hence, it is possible that some women listed in the baseline census were in fact "ghost members" and so could not be found by the field team in 2008. Finally, an unexpected sharp drop of the British Pound against the Malawi Kwacha, resulted in fewer resources to track women who had moved.

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same time. All data were collected using Personal Digital Assistants (PDAs) with in-built consistency checks, which we believe resulted in improved accuracy relative to paper questionnaires. The data are available for download at http://www.esds.ac.uk/ (study 6996).

[21]This was done in order to avoid any possible bias arising from endogenous fertility decisions in response to the intervention. This turns out not to be an important concern, as we show in subsection 7.2. Note also that approximately two thirds of the collected sample contained a child born since the intervention started.

[22]Other welfare programmes were operating in the District at the same time as this intervention. The most important, to our knowledge, is the Mchinji Social Cash Transfer, which provided cash transfers to the poorest 10% of households in the district. At the time of the follow-up surveys, the intervention was in the pilot stage and only 2.5% of households in our sample (distributed evenly between intervention and control clusters) report having received it.

[23]This has the advantage of allowing us to measure the effect of the intervention 3.5 years after it started, which is likely to be more representative of its steady state impact rather than its short-term effect. See Banerjee *et al.* (2008) and Hanna *et al.* (2012) for examples of interventions that had a positive impact at the start but that fade away in the longer run.

The right hand panel of Table 1 shows that the balance on baseline characteristics is maintained in the sample of women who were found (non-attriters). Small imbalances are detected on just 1 variable at the 10% level, suggesting that attrition between baseline and the first follow-up was not significantly different between intervention and control clusters. While it is reassuring that attrition is not significantly different between intervention and control clusters in terms of observed variables, it could nonetheless be the case that there is differential attrition in terms of unobserved variables. To deal with such concerns, in section 8.1, we consider the attrition issue in detail, following a more formal approach which allows for differential attrition in both observed and unobserved variables. We will show that our conclusions are robust to the encountered attrition.

We conducted a second follow-up survey on these women one year later, in 2009-10, tracking and successfully interviewing 91% of the women interviewed at first follow-up: 92.5% and 90% in intervention and control areas respectively. Though not displayed, the balance for this sample is very similar to that displayed in the last three columns of Table 1, with the addition of a small imbalance in marital status.

The surveys contain detailed information on household consumption; consumption of liquids and solids for each child in the household (≤6 years); breastfeeding practices (≤2 years); self-reported health for all individuals in the household[24]; weights and heights of children (≤6 years)[25]; education (≥6 years) and labour supply (≥6 years); and the main respondent's knowledge about child nutrition. In addition to the household surveys, detailed information was collected on market level food prices, with repeat visits to the same markets in different months over the data collection period to attenuate any seasonality effects.

3. Conceptual Framework

In order to understand how information of the type provided by the intervention might affect household decisions, we present a simple theoretical model in which households care about their own consumption and leisure, and about the health of their child, which is a function of the child's consumption. For simplicity, our set-up is such that households have 1 adult and 1

[24] The main respondent reported on the health status of household members.
[25] In the second follow-up, the heights of children aged between 6 and 7 years were also measured. However, their weights were not measured because the weight of some children in this age range was likely to exceed the capacity of the scale.

child. The adult chooses simultaneously the amounts to spend on child consumption, C, adult consumption, *A*, and leisure *L* (or labour supply, *T-L*, since *T* is total time endowment of the adult). The household's optimisation problem is therefore:

$$\max_{\{C,A,L\}} \quad U(A,L) + G(H) \qquad (1)$$

$$st: \quad pA + C \leq w(T-L) \qquad (2)$$

$$H = h(\theta C) \qquad (3)$$

where *U(.,.)* captures the utility from adult consumption and leisure, *G(.)* captures the utility from child health, *p* is the price of adult consumption relative to child consumption, and *w* is the wage per unit of time. The child health production function, $h(\theta C)$, depends on the child's consumption, C, and θ, which is a parameter reflecting the household's efficiency in child health production: for a given amount of child consumption, C, a larger θ corresponds to better child health. [26]

In this framework, we think of intervention as raising the value of θ, either because it directly increases knowledge about child nutrition, and/or because it raises the salience of child nutrition in these communities, helping mothers to overcome problems of limited attention (Hausman 2008, Masatlioglu *et al.* 2012).

As standard, we assume that *U(.,.),G(.),* and *h(.)* are increasing and strictly concave in their arguments and that the second order condition to attain an interior maximum is satisfied.[27,28] This simple model allows us to derive two key results:

Proposition 1: *In a context where child consumption is low, providing information on child nutrition increases child consumption:* ***dC/dθ>0.***

To show this, we differentiate the first order conditions with respect to θ (see Appendix 2) and find that $\frac{dC}{d\theta}$ is positive if and only if:

$$\theta C[G''(h)^2 + h''G'] + G'h' > 0 \qquad (4)$$

[26] We use a static, unitary model to draw out the key behavioural responses to the intervention in the simplest way. See Chiappori (1997) and Blundell *et al.* (2005), among others, for work that incorporates labour supply, household production and/or children within a collective framework. See Grossman (1972) for dynamic considerations of a health production function.

[27] The assumption that U(.,.) and G(.) are separable allows us to abstract from the signs and magnitudes of the cross-partial derivatives of the household utility function with respect to A and H, as well as H and L. Given that the empirical literature has not shed light on these cross partial derivatives, allowing for such non-separabilities would complicate the model without improving its predictive power.

[28] We assume that the household cannot borrow, which is consistent with well-known credit constraints in developing countries, as discussed for instance in Dupas (2011a).

Consequently, $\frac{dC}{d\theta} > 0$, unless one of the following holds: (i) G'', which is negative, has a large magnitude, (ii) h'', which is negative, has a large magnitude, or (iii) θC is very large. None of these conditions seem likely to hold in our setting: very negative values of G'' (alternatively h'') imply that additional increases in child consumption decrease very rapidly the marginal utility from child health (alternatively the marginal productivity of child consumption); high values of θC are associated with high values of child health. Any of these would be plausible only in contexts where child health is already sufficiently high, unlike our setting where child health is poor, which indeed motivated the intervention.

Condition (4) is satisfied independently of the value of θC when $K(\theta C) \equiv G(h(\theta C))$ is not too concave, and in particular when the concavity of $K(\theta C)$, as measured by the elasticity of $K'(\theta C)$, is less than one.[29] A commonly used function that would satisfy this condition is $K(\theta C) = (\theta C)^{\alpha}$, with $\alpha<1$. However, it is worth stressing that for ***dC/dθ>0*** to hold we do not need that condition (4) holds for all values of θC but it is enough that it holds locally at the optimum.

Proposition 2. *If condition (4) is satisfied and leisure and adult consumption are complements, ($U_{LA} > 0$) or substitutes ($U_{LA} < 0$), but satisfying $wU_{LA} - pU_{LL} > 0$, then providing information leads to: (i) a decrease in leisure, L, (ii) an increase in household consumption, pA+C, (iii) a decrease in adult consumption, A.*

If condition (4) is satisfied, child consumption increases when θ increases (Proposition 1). It is optimal to accommodate this increase in child consumption along the other two margins available to the household: decreasing adult consumption and decreasing leisure. This is because the concavity of the utility function implies that utility decreases less by simultaneously reducing *L* and *A* than by reducing only one margin. Due to the decrease in leisure, total household consumption increases (the increase in child consumption more than offsets the decrease in adult consumption).[30]

[29]Note that condition (4) can be rewritten as $-\theta C\frac{K''(\theta C)}{K'(\theta C)} < 1$ in which the left hand side is the elasticity of $K'(\theta C)$. This type of condition normally arises in models with additive utility and hence it is natural that it appears. For instance, note that the condition would imply a restriction in the coefficient of relative risk aversion would K() be the Bernoulli utility function in a model with uncertainty.

[30] Our simple model abstracts from differential labour supply responses of the mother and the father. Indeed, in a two parent model, one could imagine that additional time devoted to the acquisition and preparation of more nutritious foods might be to the detriment of mother's labour supply and/or leisure. However, if male and female wages are the same, it would still be the case that total household labour supply increases with the father more

It is worth highlighting that complementarity between leisure and adult consumption ($U_{LA} > 0$) is sufficient but not necessary for this result to hold. The same result will be obtained when leisure and adult consumption are substitutes, as long as U_{LA} is not too large in absolute value (see Appendix 2).[31] Note that the literature has not reached a consensus on whether consumption and leisure are complements or substitutes; with early work by Heckman (1974) favouring the latter while Mortensen (1977) favours the former.

Therefore, under assumptions which we believe to be very reasonable in this setting, receiving information on child nutrition increases both child and household consumption, decreases adult consumption and increases adult labour supply. We now turn to testing these propositions using the data and experimental set-up described in Section 2.

4. Empirical Framework

4.1 Estimation and Inference

The randomised experiment provides us with a clean and credible source of identification to test the propositions emerging from the theoretical framework above. To do so, we estimate OLS regressions of the form

$$Y_{ict} = \alpha + \beta_1 T_c + X_{ict}\beta_2 + Z_{c0}\beta_3 + \mu_t + u_{ict}, \quad \text{t=1,2} \qquad (5)$$

where Y_{ict} includes outcomes for unit *i* (household or individual, depending on the outcome of interest) living in cluster *c* at time *t* (=1, 2 for first and second follow-ups, 2008-09 and 2009-10, respectively).[32] In line with the model, the particular dimensions of household behaviour likely to be affected include household and child consumption, labour supply, and child health[33]; T_c is a dummy variable which equals 1 if the main respondent of our survey was, at the time of the baseline in 2004, living in a cluster that later received the intervention; $\mathrm{X_{ict}}$ is a vector of household/individual-level variables measured at time t including a quadratic polynomial in child age, child gender, maternal education and marital status, and distance of household to the closest trading centre; Z_{c0} is a vector of cluster-level variables measured at baseline such as proportions of women with Chewa ethnicity, and with primary or secondary schooling. μ_t is a vector of month-survey year dummies indicating the month of

than offsetting any potential reduction in mother's labour supply. If male wages are higher than female wages, the results would hold in terms of earnings rather than labour supply.

[31] $U_{LA} > 0$ is also sufficient for the second order conditions to hold.

[32] A number of the outcomes are binary measures. We re-ran these using Probit models; results are very similar and not reported.

[33] Adult consumption also may be affected but, unfortunately, no good measure of adult specific goods is available in our data.

the interview, and u_{ict} is an error term which is uncorrelated with the error term of others living in other clusters ($E[u_{ict}u_{jwq}] = 0$ for $i \neq j, c \neq w$), but which may be correlated in an unrestricted way with that of others living in the same cluster, independently of the time period ($E[u_{ict}u_{jcq}] \neq 0$). Note that this correlation structure allows for first, the error term for individuals/households in the same cluster to be correlated over time, and second, for the presence of spillovers within but not across clusters, consistent with the presence of large buffer areas in place between study areas in adjacent clusters, as discussed in section 2.2.1.

The treatment indicator, T_c, is defined on the basis of the cluster of residence of the main respondent in the 2004 baseline census, regardless of whether she received the counsellor's visit. Therefore, we identify an intention-to-treat parameter. Defining T_c on the basis of baseline (2004) residence avoids 2 biases: the first potentially arising from counsellors choosing to visit some mothers and not others (and *vice versa*, with some mothers choosing not to receive the visits), which would render actual participation endogenous; the second bias might occur if women have migrated to intervention clusters from control clusters so as to benefit from the intervention. An additional reason for defining T_c on the basis of baseline residence rather than actual treatment is that the intervention may generate spillovers within the cluster to households ineligible for the intervention. Focusing solely on those who were eligible to receive counsellor visits would not give a full picture of the intervention effects, a point which we come back to in section 6 when we measure the extent of spillovers.

In terms of inference, standard statistical formulae for clustered standard errors based on asymptotic theory (cluster-correlated Huber-White estimator) have been shown to provide downward biased standard error estimates if the number of clusters is small (less than 30) thus over-rejecting the null hypothesis of no effect (Donald and Lang 2001, Wooldrige 2004, Duflo *et al.* 2004, and Cameron *et al.* 2008).[34] This is a potential issue here, as there are just 24 clusters. Cameron *et al.* (2008) recommend instead a wild cluster bootstrap-t procedure to estimate the correct p-value for hypotheses tests of significance. Their Monte Carlo simulations suggest that this method performs relatively well compared to the cluster-correlated Huber-White estimator. We utilise this bootstrap procedure for inference in this paper. In all estimation tables, we report the clustered standard error computed using the

[34] Cameron *et al.* (2008) indicate 30 as a rule of thumb for when the number of clusters can be considered small, but they indicate that in general the size of the bias will depend on the level of intra-cluster correlation (of both the regressor and error) and the number of observations per cluster.

cluster correlated Huber-White estimator, as well as the p-value of a t-test of the null that the coefficient is zero computed using the wild-bootstrap cluster-t procedure.

4.2 Internal Validity

Although the identification of the treatment effect relies on the randomisation, one potential source of bias arises from the fact that the intervention may have reduced infant mortality in intervention areas. However, this is only likely to be relevant for outcomes relating to children's health, where this differential mortality might alter the (unobserved) distribution of health endowments of children in our sample. Under the assumption that weaker children are the ones more likely to survive as a result of the intervention (an intuitive and common assumption known as "the selection effect" - see Deaton (2007), Bozzoli *et al.* (2009) among others), this would imply that the average child health endowment is relatively poorer in intervention areas. Consequently, we may be *underestimating* the effect of the intervention on children's health. Another potential source of bias is that if the intervention affected fertility, this could alter the composition of children in intervention and control clusters.[35] However, as we show in section 7.2, the intervention does not appear to have affected either fertility or family planning, suggesting that this is not an issue in our context.

A third important potential source of bias in our sample arises from the attrition encountered between the baseline and first follow-up surveys, which was greater than initially expected. In section 8.1, we provide several pieces of evidence which alleviate concerns that our results are subject to attrition bias. Finally, though Table 1 has shown that the randomisation worked well in terms of balancing observable characteristics, it could be the case that some unobserved variables were not balanced, particularly as just 24 clusters were randomised. In Section 8.2, we provide evidence to show that this is unlikely to be driving our results.

4.3 Outcome Measures

In line with the theoretical model, our outcomes of interest include household and child consumption, labour supply, and child health and morbidity, which are detailed below. We pool data from the 2008-09 and 2009-10 follow-up surveys for the analysis. Statistics pertaining to all of the outcomes described in this section are provided in the discussion of results (section 5).

[35]This is not a problem when we compare household or adult level outcomes since the sample is drawn on from a census of women of reproductive age, independent of their fertility.

4.3.1 Household Consumption

We have information at the household level on the quantities consumed and purchased of over 25 different food items in the week preceding the survey, and the amounts spent on them. Data was also collected on expenditures on items such as fuel and transport (over the past month), and clothing, health and education (all over the past year).[36] In 2009-10, information was also collected on conversion factors from the most-frequented markets and trading centres, which are used to convert non-standard measurement units (such as a heap of tomatoes) into standard measurement units (such as kilograms).[37]

Food consumption aggregates are computed by summing up food expenditures and adding on the values of non-purchased food. To impute the latter, we first use conversion factors to convert quantities measured in non-standard units to standard units, and then use median unit values to impute their value.[38] Total household monthly non-durable consumption is then computed as the sum of food consumption and the non-food expenditures outlined above (all converted to monthly terms). Finally, we obtain per-capita consumption values by dividing the relevant value by household size.

4.3.2 Child Consumption

We collected information on child-specific intake of liquids and solid foods, focusing on diet variety. These are reported by the main respondent, who is the mother in a majority (92%) of cases. For children under the age of 2, there are three measures of liquid intake - whether or not (s)he had maternal milk, other milk, or water in the 3 days prior to the survey. In the second follow-up survey, there are also data on whether or not certain foods were consumed in the 3 days prior to the survey by all children aged less than 6 years. We use these data to create three categories of solid food intake: the number of types of cereal (porridge and

[36] The recall period for these items in the 2009-10 survey was modified to only record expenditures since the 2008-09 survey. This was done so as to avoid double-counting of purchases, since the gap between the two surveys was less than a year (between 9 and 11 months).
[37] These conversion factors were applied to data from both waves.
[38] Median unit values are computed by dividing expenditure on a certain good by the quantity purchased, and taking the median at the cluster level. In the small number of cases where there were insufficient observations within a cluster to reliably compute the median, it was taken at the district level instead. This method of imputation is similar to that used by Attanasio *et al.* (forthcoming). As a robustness check, we also valued consumption using the market prices rather than the median unit values. This is not our preferred method, since most households rarely purchase the foods they commonly consume from the markets. Reassuringly, though, both methods yield a food consumption share of total non-durable consumption of 0.86.

nsima, thus taking integer values 0, 1 or 2)[39], the number of types of protein-rich foods (meat, fish, eggs and beans, thus taking integer values between 0 and 4), and whether either fruit or vegetables, or both, were consumed (taking values 0,1 or 2).[40]

4.3.3 Adult Labour Supply

Labour supply is measured in three ways: whether or not an individual is engaged in an income-generating activity; whether or not an individual has a secondary income-generating activity; and the total number of hours worked in the week preceding the survey (number of days worked in the week preceding the survey multiplied by the number of hours worked per day; set to zero for those not working). We distinguish between all adults (aged 15 and over), and adults with dependent children (where, in line with ILO Conventions, a dependent child is an individual under the age of 15), as the latter are more likely to be directly affected by the intervention.

4.3.4 Child Health

Both physical growth and morbidity are taken as indicators of child health. Physical growth is measured by height and weight: we compute standardized height-for-age z-scores, weight-for-age z-scores, and weight-for-height z-scores.[41] Height and weight are robust indicators of a child's growth and development, and a child's height has been shown to be correlated with outcomes later on in life, thus making it a measure of long-term health. The second measure of child health, morbidity, is maternal-reported and includes the prevalence of diarrhoea, fast breathing, fever, chills, and vomiting in the 15 days prior to the survey.

[39] Nsima is a thick paste made from maize flour and is a staple food in Malawi. Apart from being difficult to digest for infants, nsima does not contain all of the nutrients required by infants. MaiMwana recommends giving porridge to infants, ideally mixed with vegetables or protein, rather than nsima.

[40] So for instance, the measure for number of proteins consumed is the sum of the four dummy variables indicating whether a child consumed meat, eggs, fish and beans. It takes a value of 4 if all four foods are consumed, 3 if only 3 of them are consumed, and so on.

[41] Height was measured using a SECA Leicester Height Measure for children aged 2 through 6, and using a SECA Measuring Mat for children less than 2. Weight was measured using a Salter weighing scale. Z-scores are created using STATA macros supplied by the World Health Organisation (WHO). A stunted (underweight) child has a height-(weight-) for-age z-score that is below -2 SD based on the 2006 WHO reference population. A wasted child has a weight-for-height z-score that is below -2 SD based on the WHO reference population. The height-for-age z-score is missing for 9% of children in intervention areas and 12% in control areas. These missing values are either because the child could not be located or refused to be measured (72%), or because the values are outliers according to the WHO subroutine (28%).

5. Results

5.1 Knowledge and Salience of Child Nutrition

The key rationale underlying the intervention is that households are inefficient producers of child nutrition because they do not have the correct knowledge. In other words, the nutrition production function that households optimise over is "distorted". We begin by providing evidence that the intervention improved women's knowledge of child nutrition (captured in the model in section 3 by an increase in the parameter θ).

In Table 2, we report the effects of the intervention on nutrition knowledge, as measured by an index that combines responses to individual knowledge questions (reproduced in Appendix 3).[42] Column (1) relates to "directly exposed" respondents (i.e. those with a child born since July 2005, 68% of the sample) and Column (2) to "indirectly exposed" respondents (no child born since July 2005, 32% of the sample). The Table shows that the intervention was successful at increasing nutritional knowledge for both types of respondents, though the coefficient for the indirectly exposed is not statistically significant from 0. Note that identification of these effects is complicated by the very large intra-cluster correlation associated with this outcome, as can be seen from the Table.

[TABLE 2 HERE]

At the time of data collection, the intervention had been in place for roughly 3.5 to 4.5 years, and was still on-going. Therefore, mothers were not only exposed to it when they were visited by the counsellor (so when they were pregnant or child was less than 6 months) but also when the counsellor visited other local women/friends as they had children. This "roughly constant" presence of the intervention could have contributed to making child nutrition a more salient topic throughout villages in the cluster, thereby increasing the likelihood that households acted on the improved knowledge on child nutrition and that this knowledge was not easily forgotten. In line with this argument, columns (3) and (4) of Table

[42] More precisely, the index is computed as follows (as in Anderson 2008): first, scores for individual questions are defined as 0 if incorrect and 1 if correct. So a higher value of the index indicates better knowledge. Second, individual question scores are normalised by demeaning each score by its sample mean and dividing the demeaned score by the control group standard deviation. Finally, the index score is computed by taking a weighted average of these normalised scores, where the weights are computed from the inverted covariance matrix (exact formula in Appendix A of Anderson, 2008). This weighting procedure is analogous to GLS (Generalised Least Squares) and helps increase efficiency by assigning low weights to highly correlated questions and high weights to uncorrelated questions (which can be thought of as providing "new" information).

2 show that respondents were more likely to report having a one-to-one chat about child nutrition with friends. The magnitude of this effect is quite large: almost 20 percentage points for the directly exposed and almost 10 percentage points for the indirectly exposed (in both cases roughly a 100% increase over the control mean).[43]

5.2 Consumption Responses

The theoretical model suggests that increases in household consumption can be expected to occur as a result of the improved child nutrition knowledge due to the intervention. This is in spite of the fact that the intervention does not provide any monetary or in-kind resources. Table 3 reports the effects on per capita monthly consumption of all households in our sample, irrespective of whether or not they have children born since the intervention started in July 2005. This is first to avoid the potential endogenous fertility bias mentioned in section 4.2.1 (which we discard in any case in section 7.2) and second, because all households within a cluster could benefit indirectly from the intervention due to spillovers (which are likely given the increased salience of nutrition).

The table shows that the intervention increased per-capita non-durable consumption substantially - by 500 MK (USD 3.56), corresponding to 23% of the control group mean. Food consumption, which comprises 83% of total non-durable consumption, accounts for the majority of this increase at 408 MK. Within food consumption, the bulk of this increase is concentrated among proteins and fruit and vegetables, each of which increased by 1/3 compared to control areas.

A number of factors are likely to explain this substantial increase in consumption: first, the time span of the intervention is sufficiently long (it had been already up and running for over 3 years by the time consumption was first measured), second, that there was scope to substantially increase labour supply to fund the increased consumption (as our results below attest to), and third, nutrition seems to have become more salient in intervention areas.

[TABLE 3 HERE]

[43] However, despite this large magnitude, the p-value is just below 0.10 for the directly exposed, owing to the large intra-cluster correlation (0.20).

We next consider the effects of the intervention on food intake at the child-level. Here we focus on the outcomes of children born since the intervention started ("directly exposed" children), whose mothers were thus eligible to receive visits from the infant counsellor, putting them at around 0 - 4.5 years of age.[44] Later on in section 6, we focus on older children born before the intervention began, in order to measure spillovers from the intervention.

Virtually all children aged less than 6 months (99.4%) are breastfed, making it unsurprising to find no intervention effect on this outcome. In terms of other liquid intake, there is a reduction in the probability that an infant aged less than 6 months consumes water or non-maternal milk. These results, shown in Table 4, suggest that exclusive breastfeeding most likely increased, in line with the information provided by the counsellors. The Table also shows that the intervention did not lead to an increase in the intake of breast milk for children aged 6 months to 2 years, suggesting that any improvements in child health or nutritional status (considered further on) for children older than 6 months are unlikely to be due to an increased intake of breast milk after the first six months of life.

[TABLE 4 HERE]

Table 5 shows the effects of the intervention on children's food intake. It shows that children older than six months living in intervention areas consume a greater variety of protein-rich foods, which are crucial for the healthy growth and development of children.[45] On average, children in intervention areas consume 1.5 different sources of protein compared to just over 1 in control areas. The table also shows that overall diet variety improves (column (1)), though the coefficient is not statistically significant from 0 (p-value of 0.118).

[TABLE 5 HERE]

[44] The eldest child born since the intervention started is 53 months of age in our sample.

[45] Because the intervention promotes exclusive breastfeeding for children below 6 months, one would expect that the intake of solid food would decrease for this group. Indeed, we find negative coefficients but the sample is very small (151) and hence the estimates are not statistically significant. Note that information on solid food intake for this age range was only collected in the second survey.

5.3 How is the increased consumption funded?

The large increases in consumption just observed are particularly striking when considered against the fact that the intervention did not provide any resources whatsoever, neither monetary nor in-kind: it purely provided information, albeit on a one-to-one basis over a six-month period. This raises the natural question as to how this observed increase in consumption, which is accompanied by an improvement in children's diets, is funded. The model in section 3, particularly Proposition 2, shows that labour supply plays a potentially important role. To investigate this empirically, we consider the effects of the intervention on three margins of adult labour supply: whether or not an individual works at all (i.e. has an income generating activity), whether (s)he also has a second job, and the total number of hours worked per week.

We first consider the results for all adult males, displayed in the upper panel of Table 6.[46] It shows that males are 6.6 percentage points more likely to take on a second job, a very large increase given that only 12% of males have more than one job (in control clusters). If the underlying factor driving the increases in labour supply is to fund better diets for children, then one would expect observed increases to be concentrated among parents. To investigate this, we split males into those with and without dependent children. When we do this we find that fathers have much larger labour supply adjustments (top-central panel of the Table): they are 8 percentage points more likely to have a second job in intervention than in control clusters, and work over 5 hours more per week. In contrast, non-fathers are 3 percentage points more likely to have a second job, which is not significantly different from 0 and have virtually no difference in hours worked (top-right panel of the Table). It is also worth stressing that the fact that fathers but not non-fathers are affected is consistent with the intervention being at play and alleviates concerns that the labour supply responses are driven by unobserved factors such as differential labour market conditions across intervention and control clusters.

[TABLE 6 HERE]

[46] Wherever possible, we take a very conservative approach and consider samples that are not a dictated by fertility choices (in this case, all males, regardless of whether or not they have children). This is to rule out concerns regarding biases that might arise due to the intervention affecting fertility. However, we will show in section 7.2 that the intervention did not affect fertility. Given this, and as the more interesting margin to consider is fathers and non-fathers (as we expect labour supply responses to be concentrated amongst the former), here we further split adult males into those with and without dependent children.

The lower panel of Table 6 displays the results for adult females, and also separates these into those with and without dependent children. There is no evidence of any significant impact of the intervention on any of the three measures of labour supply of females. This holds even when we split the sample into mothers and non-mothers.[47]

The finding that the intervention increases the likelihood of males engaging in secondary jobs is consistent with it being a margin with clear scope for increase as only 12% of males in control clusters have a second job. According to our data, most of these second jobs are non-agricultural self-employment activities.[48] Moreover, there is considerable entry into and exit from secondary jobs: among those with (without) a secondary job at first follow-up, 33% (7%) have one by the time of the second follow-up, a year later. While an extensive literature has documented increases in labour supply in response to increases in uncertainty and income shocks in developing countries (Saha 1994, Kochar 1999, Rose 2001, Lamb 2003, Kijima 2006, Ito and Takashi 2009), we are the first to document that labour supply responds to changes in the perceived child health production function.

The increase in labour supply was a clear prediction of the theoretical model. However, beyond the model, there are important features of Malawian society that are likely to be contributing to the finding that male labour supply increases. In particular, the main ethnic group in the Mchinji District - the Chewa - is a matrilocal and matrilineal group, meaning that men usually move to their wives' villages on marriage, and that wealth (predominantly land) is often held by women and passed on through the matriline (Phiri 1983, Sear 2008). As a consequence, women have more power and authority than in patrilineal societies common across most of Africa and South Asia (Reniers 2003). Indicative of this empowerment, we saw that all three measures of labour supply - work participation, the likelihood of having two jobs and hours worked - are strikingly similar for males and females (last rows of the top and bottom panels of Table 6).[49] Thus, the finding that male labour supply increases in response to mother receiving information on child nutrition is in line with the cultural background in this setting (mothers having enough power so as to persuade the father to work more).

[47] Note though that while the coefficient on whether or not a mother works is negative and fairly sizeable, the intra-cluster correlation is also large and hence the estimates are imprecise.

[48] Over half of these second jobs involve employment in an own/family business, a further quarter involve work on the family farm, and the rest involve work as an employee in public/private sector (~20%) or on someone else's farm (<5%).

[49] This has been documented by others for the Malawian context including Goldberg (2011) and 2004 DHS (pages 34-36, Malawi DHS 2004 Report). In the also matrilineal Khasi society (India), women and men have similar labour supply profiles (Gneezy, Leonard, and List 2009).

5.4 Has children's health improved?

A key question of policy interest is whether the observed adjustments on various margins of household behaviour (increased consumption and labour supply) feed through to improvements in child health, which is what we look at in this section. We consider effects on children's physical growth (Table 7) and morbidity (Table 8) for children "directly exposed" to the intervention. We re-iterate here that the intervention may have reduced infant mortality, and under the assumption that weaker children are the ones more likely to survive as a result of the intervention, the effects displayed below likely *under-estimate* the true intervention effect on child health.

Starting from having very low nutritional status - 56% of children are stunted, and 16% are underweight in control clusters - we find that the intervention increases children's height by 20% of a standard deviation of the WHO reference population.[50] While non-negligible, this increase in height is around half of that obtained with more intensive interventions, particularly those providing food directly: a recent meta-analysis concluded that the provision of complementary food in food-insecure populations resulted in an average increase of 0.41 standard deviations of age-adjusted height (Bhutta *et al.* 2008).

The poor nutritional status of our sample is apparent not only in that children are too short for their age, but also that they are too heavy for their stature (usually a sign that their diet contains too much carbohydrates and too few proteins). More precisely, in control clusters children's weight-for-height z-score is 66% of a standard deviation above that of the WHO reference population. Table 7 shows that the intervention decreases significantly the weight-for-height z-score by 18% of a standard deviation, which means that the intervention is resulting in children becoming closer to the WHO norm (and hence putting them in a healthier path).[51]

[TABLE 7 HERE]

[50] As is common with anthropometric data from developing countries, the SD of the height-for-age z-score in our sample is larger than in the WHO Reference Population (in our case the SD is 1.5 instead of 1), and so this increase corresponds with a 13% of a SD increase using the SD for our sample.

[51] According to the medical literature, height and weight do not necessarily evolve in parallel. Victora *et al.* (2010) indicate that the faltering patterns of height-for-age are very different from those of weight-for-age. Victora (1992) shows that in Africa and Latin America, the relationship between malnutrition due to low height is only very weakly related to malnutrition caused by low weight, unlike in Asia where the relationship is much stronger.

In Table 8, we also detect a large and significant drop in the percentage of maternal reported cases of diarrhoea (in the 15 days prior to the survey) among children less than 6 months (a 5 percentage point drop from a base of almost 13%). This is consistent with the reduction in water intake (usually unsterilized) amongst this age group as a result of the intervention, shown earlier in section 5.2.

[TABLE 8 HERE]

Clearly, we cannot disentangle whether the improvement in physical growth is due to the reduction in the intake of liquids other than breast milk when the child was less than 6 months, or to the improvement in child food intake after age 6 months, or a combination of the two. However, what is of primary interest in this paper is that households responded to the information provided by increasing consumption and working more, to improve child health.

6. Spillovers on older children

The analysis on child-level outcomes has so far considered effects on children "directly exposed" to the intervention, i.e. those born after the intervention started (July 2005) and consequently whose mothers were eligible to receive the full cycle of five visits from the counsellor. However, the evidence in section 5.1, which showed increased salience of nutrition in the community (as measured by one-to-one chats about child nutrition), suggests that the intervention could also have impacted the diets and health of older children living in intervention clusters (children born before July 2005 when the intervention began). There are two types of such children: the first are the older siblings of "directly exposed" children ("siblings of the directly exposed"), thus potentially generating a within (intra) household externality; the second are those living in households without a child born since the intervention ("indirectly exposed children"), thus generating an across (inter) household externality. The direction of a within household externality is unclear: on one hand, the intervention could lead mothers to improve nutritional inputs for all her children, to the benefit of siblings of the directly exposed child. Conversely, mothers could substitute resources (monetary, time, etc) away from siblings of the directly exposed child in order to comply with the provided information, to the detriment of these siblings. Ignoring such spillovers would lead us to potentially under- or over-estimate the effects of the intervention.

Here we investigate whether the increased awareness of nutrition-related issues affected child outcomes for other children in these areas. In order to estimate the extent of spillovers on older children, we estimate equation (5) on the sample of children born before the intervention started. This analysis therefore pertains to children aged around 3.5 through 7 years and pools the "siblings of the directly exposed" and the "indirectly exposed children".[52] We first estimate the effect on the intake of foods, shown in Table 9 below. We observe an increase, significant at the 10% level, in the intake of protein-rich and staple foods.

[TABLE 9 HERE]

In order to distinguish within and across household spillovers, we disaggregate this sample further into "siblings of the directly exposed" and the "indirectly exposed children". We find evidence of positive within household spillovers in the intake of protein-rich foods by older siblings (left hand column). The identification of across household spillovers is complicated somewhat by the smaller sample size. Nonetheless, the point estimates (in the right hand column for each type of food) are all positive and the increase in the intake of staples is statistically significant at 10%, providing some weak evidence of across household spillovers.

[TABLE 10 HERE]

We next consider the extent of spillovers on physical growth and morbidity, and find no evidence that the improvements in children's food intake yielded any improvement in their physical growth, or reductions in morbidity, as can be seen from Tables 11 and 12. This is in some ways not surprising: the increased intake of nutritious foods is likely to have started a few months after the birth of the "directly exposed" children (when women in the cluster received visits). Consequently, the older children were unlikely to have been exposed to the intervention during the 'critical growth period' (first two years) when nutrition is most effective at improving physical growth (Shrimpton *et al.* 2001, Victora 2010). This result is also consistent with Schroeder *et al* 1995, who find that a nutritional supplementary feeding programme in Guatemala had large impacts on annual height and weight gain only among infants aged less than 3 years, but no impacts for height and weight gain between ages 3 and

[52] The analysis for food consumption focuses on children aged around 4.5 through 7 (as this outcome is only observed in the second follow up); the health analysis is for those aged around 3.5 through 6 (as it is observed in both surveys, and the youngest child born before the intervention began would have been around 3.5 years of age at the time of the first survey). Notes to specific tables contain details on the exact samples.

7 years. Similarly, it is perhaps not too surprising that there are no effects on reported cases of diarrhoea or vomiting, since older children are less vulnerable to such illnesses.

[TABLES 11 AND 12 HERE]

7. Alternative Explanations

The theoretical framework suggests that consumption and labour supply increase because the productivity of consumption (in terms of child health) increased as a result of the intervention. However, here we consider three alternative explanations for our findings: First that increases in adult labour supply are driven by improvements in adult health that are somehow generated by the intervention; second, that the intervention decreased fertility in intervention clusters, potentially yielding an increase in child quality and thus health and nutrition; and third, that information provided on issues other than child nutrition within this multi-faceted intervention could have generated the observed improvements in child health. We discuss each in turn and provide evidence to rule them out as explanations for the observed findings.

7.1 Adult Health

Whilst it is possible that increases in adult labour supply are driven by improvements in adult health that are somehow generated by the intervention, we believe this to be unlikely since the advice provided is targeted specifically at children's nutrition, which is unlikely to yield similar improvements in adult health. Nonetheless, to address this concern more directly, in Table 13, we test whether the intervention affects adult health, separately for males and females, across a range of self-reported measures capturing both morbidity and physical condition. We find no evidence that it does.

[TABLE 13 HERE]

7.2 Fertility and Family Planning

A second alternative explanation for the findings of increased parental investment into child nutrition and improved child health is that the intervention decreased fertility in intervention clusters, potentially yielding an increase in child quality (Becker and Tomes, 1976). A

reduction in fertility could be generated through two channels: first, indirectly, by reducing infant mortality and as a result inducing households to reduce their demand for children; or second, directly, through the family planning component of the intervention.

To investigate these potential fertility effects, we examine the effect of the intervention on the use of modern family planning methods (which are much more effective at preventing pregnancy than commonly used traditional methods), as well as the number of children born to women in our sample since the intervention started as reported in the MaiMwana Health Surveillance System.[53] Results are displayed in Table 14. We note that the coefficients are small and far from significant at conventional levels, despite the low levels of intra-cluster correlation. The lack of effects on family planning is consistent with conversations with programme officials, who indicated that this component was not effective because counsellors were uncomfortable discussing this issue. Moreover, it would be hard to reconcile reductions in fertility with increases in paternal labour supply.

[TABLE 14 HERE]

7.3 Other aspects of the intervention

As is usually the case with public health interventions, the intervention was multi-faceted and provided information on issues other than infant feeding practices which could also have influenced child health: encouragement of vaccination of children, encouragement of HIV testing, and information on hygiene practices. Though these additional aspects of the intervention could certainly improve child health, it is much more difficult to believe that they could increase household consumption and labour supply, which are the key results of this paper.

Moreover, some of these aspects probably had limited impact even on the behaviours that they were trying to promote. According to UNICEF (2008), the vaccination rate for the majority of the 9 recommended vaccine doses in childhood was above 90% in Mchinji in

[53] The MaiMwana Health Surveillance System measures the physical growth of all children born in the 24 clusters since 2005 at age 1 month and 7 months. This source therefore provides a more complete picture of births in the study areas than cross-sectional surveys. Nevertheless, there may still be selection from differential mortality of infants in the first month life as a result of the intervention.

2006.[54] Consequently, the scope to improve vaccination rates, and thereby child health, was limited. Further, the finding of no reductions in the prevalence of diarrhoea for adults (Table 13) and children aged above 6 months (directly exposed and indirectly exposed; Tables 7 and 12 respectively) suggests that the component on hygiene information probably had limited success. Our conversations with the intervention managers indicated that counsellors felt uncomfortable talking to mothers about HIV so we would not expect a strong effect of the intervention on the probability that individuals got tested. Moreover, the prevalence of HIV in Mchinji is relatively low (6.4%), and around 2/3 of those that get tested do not collect the results (Thornton, 2008). As we discussed in the previous section, the intervention did not have an effect on family planning either.

For these reasons, we believe that the components related to infant feeding are most likely to be driving the results reported in this paper.

8. Potential Sources of Bias

In this section we discuss two potential sources of bias in our findings. The first relates to attrition from the sample between the baseline and the follow-ups, and the second relates to the relatively small number of clusters. We discuss each of these in turn.

8.1 Attrition

One concern is that our results may be biased due to attrition between the baseline census (2004) and the two follow-up surveys (2008-09, 2009-10). Whilst we showed in Section 2 that both the sample drawn and the sample successfully interviewed are well-balanced along observed characteristics (Table 1), a concern remains that attrition induced differences in unobserved variables, potentially biasing our findings.

Two particular concerns are the following. First, our estimates of Table 7 could be biased upwards if households with *worse* health endowments were more likely to attrit from intervention than from control clusters. However, Table 11 (cols 1 and 3) showed that the health status of older siblings is worse (though not significantly so) in intervention than in

[54] Exceptions are 3rd dose of Pentavelent (83.2%), Polio at birth (33.6%), second (88.7%) and third (69%) dose of Polio, as well as Measles (83.2%).

control clusters. This provides suggestive evidence that those who attrited from intervention clusters are, if anything, relatively healthier than those attriting from control clusters.[55]

Second, our male labour supply estimates of Table 6 could be biased upwards if industrious households were less likely to attrit from intervention than control clusters. However, if this were the case, then we would have expected to see the labour supply of non-fathers, and not just fathers, to be higher in intervention than in control clusters (assuming the process driving attrition is the same for households with just fathers and those with just non-fathers). Table 6 did not show this to be the case.

We also address this issue directly using a Heckman selection model (Heckman, 1979). A first stage Probit model estimates the probability that a sampled woman (and therefore her household) was successfully interviewed as a function of the intervention and characteristics of the assigned interviewer at first follow-up (given that the majority of attrition occurred between baseline and first follow up). Estimates from the first stage yield an inverse-Mills Ratio, which enters as an additional regressor in the second stage - equation (5) augmented with the inverse Mills Ratio - thereby correcting for selection due to attrition.[56]

The interviewer characteristics provide a source of exogenous variation in the first stage (see for instance Zabel 1998, Fitzgerald *et al.* 1998). Specifically, we use the number of children aged 0-3 in the interviewer's household and the size of the interviewer's plot of land, both of which proxy for the ease and intensity with which interviewers were able to track respondents.[57] A key identification assumption is that interviewer characteristics are uncorrelated with respondents' characteristics and outcomes. We believe this assumption to be reasonable in this context.[58]

[55] This argument assumes that (a) the process driving attrition is independent of whether or not the household contains an older sibling, and (b) that households are not substituting away resources from the older child to the younger child who benefits directly from the intervention. We believe that the former is reasonable given that both types of household have a child that would potentially gain from the intervention. Moreover, regarding (b), results from Table 9, which shows increased protein intake for older siblings (and no reduction in other foods) suggests that parents are not substituting across children in this manner.

[56] Table A1 in Appendix 1 shows that attrition was not random, but the important issue is whether there was differential attrition between intervention and control.

[57] In particular, individuals with young children may be more intrinsically motivated for a study on child health, or they may know many other community members with young children. Conversely, interviewers with a larger plot of land have a higher opportunity cost of time. Both of these factors turn out to be jointly strong predictors of whether or not a woman (and hence her household) is interviewed (p-value of joint significance <0.01).

[58] A concern noted by Thomas *et al.* (2012) is that good interviewers may be assigned to the most difficult clusters. However, in our case this concern is not relevant due to the process through which interviewers were

Table 15 and 16 report the estimates of the programme effects for two outcomes, household consumption and main respondent's labour supply.[59] As can be seen from the tables, the selection corrected estimates (middle panel) are very close in magnitude to the OLS estimates reported earlier (top panel)[60], thereby providing additional evidence that our results are not driven by attrition bias.

In conclusion, note that the validity of the above three pieces of evidence rests on three different assumptions: (1) the process driving attrition is similar for households with and without an "older sibling", (2) the process driving attrition is similar both for women from households in which all adult males are fathers and for those from households containing adult males who are not fathers, (3) interviewer characteristics (number of children and size of land plot) are orthogonal to the respondent's characteristics. The three pieces of evidence, taken together, provide a strong argument that our conclusions are not driven by differential attrition between intervention and control clusters.

8.2 Number of clusters

The fact that 24 clusters were randomised into intervention and control poses two challenges. The first, which we dealt with throughout the empirical analysis, is that the standard errors estimated using the cluster-correlated Huber-White estimator might be too small, leading to over-rejection of the null hypothesis of no effect. As discussed in subsection 4.2, we estimate the correct p-values using wild cluster bootstrap-t as recommended by Cameron *et al*. (2008).

The second challenge is that the randomisation might not have succeeded in balancing some unobservable variables. For instance, it could have been that "healthier" clusters were allocated to intervention clusters, which could explain our results. However, this would not be consistent with the results in Table 11 which show that, if anything, the nutritional status

allocated to clusters. Clusters were paired in such a way as to include, subject to logistical constraints, an intervention and a control cluster in the pairing. Among potential interviewers residing in either of the two clusters, the best was selected as an interviewer to cover the pair of clusters (and hence the interviewer was not allocated to the area from a central pool). That there was just 1 interviewer per pair of clusters makes it highly unlikely that chosen interviewers were representative of the population of the cluster.

[59]The limited amount of information collected in the 2004 baseline restricts the extent to which we can use this approach to correct for attrition bias in our main estimates. In particular, we have no information on the number or characteristics of adult males in households that attrited, and thus are unable to apply this method to verify the labour supply effects for adult males. A similar caveat applies to child level outcomes.

[60] We report standard errors computed using standard block pair bootstrap because we are not aware of a t-wild procedure for non-linear models. However, our focus in this section is not so much on the statistical significance of the estimates robust to attrition, but on how close they are to our main specification.

of children born before the intervention started is worse in intervention than control clusters (though not significantly so). Similarly, if our labour supply findings were driven by the intervention clusters exhibiting better labour market possibilities than control clusters, we would have expected to find the labour supply of non-fathers in intervention clusters to also be significantly higher than in control clusters, contrary to our findings in Table 6. These pieces of evidence suggest that imbalance between the clusters is unlikely to be driving our findings.

9. Conclusion

In this paper, we use exogenous variation in mothers' knowledge of the child health production function induced by a cluster randomised intervention in Malawi, to establish empirically that improving knowledge of the child health production function influences a broad range of household behaviours.

We first establish that the intervention improved mothers' knowledge on nutrition. Using a simple theoretical model, we show that households should react to this improved knowledge by increasing consumption (both child and household) and increasing adult labour supply. In line with the predictions of the model, our empirical results show that households act on improved nutrition-related information not only by changing the composition of consumption but also by increasing total consumption - for both children and the household. The magnitude of the increase - at 23% of control household consumption - is large. This is particularly startling given that the intervention did not provide any monetary or in-kind resources. The increased consumption, which yielded improvements in children's height by 0.2 standard deviations of the WHO norm, is funded by increases in fathers' labour supply, at both the extensive and intensive margins. This finding of a non-health outcome, labour supply, being linked to how parents perceive the child health production function, is a new finding and a key contribution to this literature.

We hypothesize that two issues might have contributed to the success of the intervention. First, the provision of information was not merely a one-off event in the intervention areas, but a sustained activity, still in place, that serves to spread information and to remind households of the importance of child nutrition on an ongoing basis. Indeed, the intervention generated interest on child nutrition within the village, beyond just households directly affected, making child health and nutrition related issues more salient in these communities.

This, in turn, generated positive spillovers in food consumption of older children, particularly within the household.

Second, the main ethnic group in rural Malawi, the Chewa, is a matrilineal one, in which women are likely to have more bargaining power and authority within the household than women in patrilineal societies common in much of the rest of Africa and South Asia. This higher female empowerment might indicate that women are in a good position to implement the recommendations given by the counsellors as well as to encourage fathers to work more. It is not clear whether such responses may emerge in other settings and we see this as an area worthy of further investigation.

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Table 1: Baseline Sample Balance

	Full Sample			Interviewed Sample		
	Control Group	Difference: Treatment - Control	p-value	Control Group	Difference: Treatment - Control	p-value
Woman's Characteristics						
Married (dv = 1)	0.615	-0.021	0.386	0.661	-0.034	0.184
Some Primary Schooling or Higher	0.707	0.033	0.402	0.682	0.040	0.340
Some Secondary Schooling or Higher	0.066	0.010	0.535	0.060	-0.007	0.545
Age (years)	24.571	-0.180	0.637	25.492	-0.429	0.376
Chewa	0.948	-0.044	0.330	0.957	-0.050	0.246
Christian	0.977	0.006	0.476	0.979	0.008	0.336
Farmer	0.661	-0.075	0.108	0.688	-0.060	0.128
Student	0.236	0.015	0.438	0.204	0.022	0.274
Small Business/Rural Artisan	0.036	0.030	0.129	0.037	0.024	0.220
Household Characteristics						
Agricultural household	0.995	-0.005	0.471	0.995	0.002	0.591
Main Flooring Material: Dirt, sand or dung	0.913	-0.041	0.232	0.916	-0.027	0.474
Main roofing Material: Natural Material	0.853	-0.018	0.697	0.857	-0.004	0.891
HH Members Work on Own Agricultural Land	0.942	-0.057	0.124	0.950	-0.056	0.120
Piped water	0.011	0.040	0.314	0.009	0.032	0.340
Traditional pit toilet (dv = 1)	0.772	0.054	0.218	0.791	0.054	0.182
# of hh members	5.771	0.066	0.817	5.848	0.132	0.863
# of sleeping rooms	2.116	0.199	0.038*	2.152	0.166	0.128
HH has electricity	0.002	0.007	0.166	0.002	0.004	0.338
HH has radio	0.630	0.030	0.408	0.641	0.015	0.709
HH has bicycle	0.509	0.015	0.643	0.512	0.008	0.843
HH has motorcycle	0.008	0.001	0.925	0.007	0.002	0.779
HH has car	0.006	-0.002	0.612	0.007	-0.003	0.298
HH has paraffin lamp	0.925	0.032	0.262	0.926	0.036	0.178
HH has oxcart	0.058	-0.015	0.204	0.059	-0.022	0.090+
N	1248	1248		846	814	

Notes to Table: + indicates significant at the 10% level, * indicates significant at the 5% level. p-values reported here are computed using the wild cluster bootstrap-t procedure as in Cameron *et al.* 2008, explained in section 4.1. Full Sample includes all women (and their households) originally drawn to be part of the 2008-09 survey. Interviewed Sample includes women (and their households) actually interviewed in 2008-09 (and used in the analysis).

Table 2. Women's Nutrition Knowledge and Chats on Child Nutrition

	Knowledge score		Chat with a Friend	
	Directly Exposed	**Indirectly Exposed**	**Directly Exposed**	**Indirectly Exposed**
T_z	0.175+	0.160	0.193	0.098+
Standard Error	[0.089]	[0.091]	[0.098]	[0.044]
Wild Cluster Bootstrap p-value	{0.098}	{0.162}	{0.118}	{0.084}
Observations	1081	431	2007	818
R-squared	0.091	0.119	0.048	0.026
IntraCluster Correlation	0.189	0.113	0.209	0.072
Mean, Control	0.001	-0.153	0.2	0.121

Notes to Table: All regressions include controls for age, quadratic in age, education and chewa ethinicity at zone level in 2004, and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Sample includes all female main respondents. The sample for the knowledge score includes only households present in both waves of the survey, and covers women aged 15-63 years. Knowledge score is computed as follows: Each question was scored 1 if the respondent gave the correct answer and 0 if she didn't. A total of 7 questions on nutrition were asked, 3 in first follow up and 4 in second follow up. Individual scores were normalised by subtracting the sample mean and dividing by the control group standard deviation. The score was then computed as a weighted mean of these normalised scores, with weights computed from the covariance matrix of the individual scores. See Anderson 2008 for more details. Chat with a friend is an indicator for whether the respondent spoke with a friend on a one-to-one basis about any child nutrition issues in the week preceding the survey. "Directly Exposed" indicates households with at least one child born after July 2005. "Indirectly Exposed" households are those without any child born after July 2005, but who may potentially be indirectly exposed to the intervention. For Chats, sample includes women aged 17-43 years old (when available, responses from both follow-up surveys are included).

Table 3: Household Consumption

	[1]	[2]	[3]	[4]	[5]	[6]	[7]
	Per Capita Monthly Food Consumption for:						
	Total Non-durable	**Food**	**Health**	**Cereals**	**Proteins**	**Fruit and Vegetables**	**Other Foods**
T_z	502.889**	408.037*	6.053+	2.780	113.671*	224.985+	64.440*
Standard Error	[165.785]	[144.746]	[2.949]	[46.617]	[40.631]	[97.743]	[24.633]
Wild Cluster Bootstrap p-value	{0.016}	{0.030}	{0.082}	{0.887}	{0.022}	{0.054}	{0.036}
Observations	3190	3200	3199	3205	3202	3204	3204
R-squared	0.05	0.06	0.01	0.11	0.02	0.17	0.02
IntraCluster Correlation	0.095	0.111	0.023	0.074	0.042	0.172	0.053
Mean Control Areas	2146.00	1784.00	17.11	606.00	349.80	679.70	149.70

Notes to Table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Regression includes month-year dummies to control for seasonality. All coefficients in terms of Malawi Kwacha. (The average exchange rate to the US Dollar was approx. 140MK = 1 US$ at the time of the surveys). "Total Non-Durable" is the sum of food consumption and expenditures on items such as transport, education, health, etc, "Food" is food consumption (including food which is not bought), "Health" is the per-capita expenditure (in MK) on health care, "Cereals" includes consumption of rice, maize flour and bread, "Proteins" includes consumption of milk, eggs, meat, fish and pulses "Fruit and Vegetables" includes consumption of green maize, cassava, green leaves, tomatoes, onions, pumpkins, potatoes, bananas, masuku, mango, ground nuts and other fruits and vegetables, "Other Foods" includes cooking oil, sugar, salt, alcohol and other foods.

Table 4: Intake of Liquids by Children Aged < 24 months.

	[1]	[2]	[3]	[4]	[5]	[6]
	Water		**Milk other than maternal**		**Breastmilk**	
	< 6 months	**6-24 months**	**< 6 months**	**6-24 months**	**< 6 months**	**6-24 months**
T_z	-0.127+	0.011	-0.066+	-0.040	-0.004	-0.049*
Standard Error	[0.066]	[0.016]	[0.037]	[0.040]	[0.011]	[0.020]
Wild Cluster Bootstrap p-value	{0.066}	{0.547}	{0.084}	{0.356}	{0.755}	{0.012}
Observations	359	950	151	510	361	999
R-squared	0.24	0.04	0.08	0.02	0.02	0.11
IntraCluster Correlation	0.024	0.024	0.060	0.059	0.000	0.012
Mean, Control	0.488	0.953	0.101	0.203	0.994	0.925

Notes to Table: All regressions include controls for age, age-squared, gender and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Samples pooled for both waves. Samples for columns 1, 3 and 5 includes children aged less than 6 months and whose mothers were potentially undergoing the intervention at the time of the survey. Samples in columns 2, 4 and 6 includes children born after July 2005, and aged 6 to 53 months at time of survey. "Water" is an indicator for whether the child had any water in the 3 days prior to the survey, "Milk other than maternal" is an indicator (measured in second follow up only) for whether the child had milk other than breastmilk in the 3 days prior to the survey; "Breastmilk" is an indicator for whether the child was being breastfed at the time of the survey.

Table 5: Effects on Child Solid Food Intake

	Number of Foods	Number of Protein-Rich	Number of Staples	Number of Fruit and Veg
	[1]	[2]	[3]	[4]
T_z	0.436	0.316+	0.106	0.009
Standard Error	[0.241]	[0.151]	[0.058]	[0.064]
Wild Cluster Bootstrap p-value	{0.118}	{0.064}	{0.104}	{0.923}
Observations	1276	1282	1285	1284
R-squared	0.14	0.07	0.05	0.20
IntraCluster Correlation	0.103	0.093	0.074	0.086
Mean, Control	5.109	1.175	1.729	1.659
Total	8	4	2	2

Notes to Table: All regressions include controls for age, age-squared, gender, wealth at baseline, education of the main respondent and median zone distance to closest trading centre and dummies for the month of interview.Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Sample contains all children born after July 2005, and who were aged between 6 and 53 months at time of survey. Data on child solid intake collected in the second follow up only. "Number of Foods" is the number of foods (between 1 and 8) taken by the child during the 3 days prior to the survey, "Number of Protein-Rich" takes integer values between 0 and 4 depending on the intake of meat, fish, eggs and beans, "Number of Staples" takes integer values between 0 and 2 depending on intake of nsima and porridge, "Number of Fruit and Veg" takes integer values between 0 and 2.

Table 6: Effects on Labour Supply

	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]
	Works	**Has at least 2 jobs**	**Weekly Hours Worked**	**Works**	**Has at least 2 jobs**	**Weekly Hours Worked**	**Works**	**Has at least 2 jobs**	**Weekly Hours Worked**
		All Males			**Fathers**			**Non-Fathers**	
T_z	0.055	0.061*	3.757	0.071	0.080*	5.370+	0.023	0.030	0.836
Standard Error	[0.066]	[0.028]	[2.508]	[0.061]	[0.035]	[3.033]	[0.099]	[0.022]	[2.636]
Wild Cluster Bootstrap p-value	{0.567}	{0.050}	{0.214}	{0.288}	{0.038}	{0.078}	{0.839}	{0.252}	{0.743}
Observations	3956	3953	3637	2380	2378	2160	1602	1601	1501
R-squared	0.15	0.06	0.18	0.05	0.03	0.05	0.10	0.06	0.20
IntraCluster Correlation	0.208	0.036	0.100	0.408	0.046	0.142	0.291	0.041	0.139
Mean, Control	0.836	0.122	25.740	0.913	0.166	30.260	0.717	0.052	18.860
		All Females			**Mothers**			**Non-Mothers**	
T_z	-0.035	0.032	-0.801	-0.064	0.038	-1.024	0.021	0.017	-0.690
Standard Error	[0.071]	[0.023]	[2.684]	[0.071]	[0.029]	[3.013]	[0.090]	[0.015]	[2.557]
Wild Cluster Bootstrap p-value	(0.667}	{0.244}	{0.803}	{0.450}	{0.212}	{0.743}	{0.803}	{0.270}	{0.855}
Observations	4445	4443	4134	3015	3013	2787	1440	1440	1356
R-squared	0.12	0.05	0.14	0.05	0.03	0.05	0.09	0.05	0.18
IntraCluster Correlation	0.214	0.025	0.144	0.312	0.031	0.187	0.229	0.013	0.129
Mean, Control	0.861	0.108	24.540	0.938	0.135	27.640	0.687	0.048	17.730

Notes to Table: All regressions include controls for age, age-squared, marital status, education and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the the cluster (at which treatment was assigned; wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. The sample in the top-left panel ("All Males") includes all males aged 15-65 years; that in the bottom-left ("All Females") includes all females aged 15-65 years; that in the top-centre panel ("Fathers") includes all males aged 15-65 years with a child aged <15 years; that in the bottom-centre panel ("Mothers") includes all females aged 15-65 years with a child aged < 15 years; that in the top-right ("Non-Fathers") includes all males aged 15-65 years without a child aged < 15 years, while that in the bottom-right panel ("Non-Mothers") includes all females aged 15-65 years without a child aged < 15 years. "Works" in an indicator of whether individual had an income-generating activity at the time of the survey, "Has at least 2 jobs" is an indicator for whether individual has 2 income generating activities, "Weekly Hours worked" give the total hours worked in the week prior to the survey on both income generating activities.

Table 7: Intervention Effects on Child Physical Growth

	[1]	[2]	[3]	[4]	[5]	[6]
	Height For Age		**Weight for Age**		**Weight for Height**	
Age at measurement -->	**<6 months**	**> 6 months**	**<6 months**	**> 6 months**	**<6 months**	**> 6 months**
T_z	0.136	0.204+	-0.133	0.004	-0.369	-0.181+
Standard Error	[0.278]	[0.107]	[0.172]	[0.102]	[0.333]	[0.088]
Wild Cluster Bootstrap p-value	{0.691}	{0.060}	{0.491}	{0.985}	{0.386}	{0.058}
Observations	324	2192	339	2265	319	2217
R-squared	0.05	0.04	0.03	0.02	0.07	0.01
IntraCluster Correlation	0.048	0.022	0.048	0.030	0.197	0.027
Z-Scores, Control	-0.560	-2.343	0.008	-0.841	0.633	0.659

Notes to Table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. All regressions include controls for age, age-squared, gender and dummies for the month of interview. Sample in columns 1, 3 and 5 includes children born after June 2005 and who were < 6 months and whose mothers were potentially undergoing the intervention at the time of measurement. Sample in columns 2, 4 and 6 includes children born after July 2005 and who were aged between 6 and 53 months at time of measurement. "Height-for-Age", "Weight-for-Age" and "Weight-for-Height" are standardised z-scores relative to the WHO reference population.

Table 8: Intervention Effects on Child Morbidity

	[1]	[2]	[3]	[4]	[5]
	Suffered Diarrhoea	**Suffered from Vomiting**	**Suffered from Fast Breathing**	**Suffered Fever**	**Suffered from Chills**
			< 6 months		
T_z	-0.049+	-0.055	0.035	0.010	-0.001
Standard Error	[0.027]	[0.040]	[0.052]	[0.073]	[0.050]
Wild Cluster Bootstrap p-value	{0.096}	{0.216}	{0.531}	{0.877}	{0.993}
Observations	376	376	376	376	376
R-squared	0.06	0.06	0.06	0.08	0.03
IntraCluster Correlation	0.000	0.026	0.037	0.066	0.075
Mean, Control	0.129	0.169	0.124	0.421	0.101
			> 6 months		
T_z	0.014	-0.012	0.018	0.022	0.016
Standard Error	[0.037]	[0.052]	[0.053]	[0.064]	[0.053]
Wild Cluster Bootstrap p-value	{0.721}	{0.861}	{0.761}	{0.709}	{0.749}
Observations	2362	2366	2363	2371	2370
R-squared	0.11	0.01	0.02	0.01	0.01
IntraCluster Correlation	0.034	0.081	0.139	0.080	0.112
Mean, Control	0.251	0.207	0.101	0.507	0.149

Notes to Table: Notes to table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. All regressions include controls for age, quadratic in age, gender and dummies for the month of interview. Sample in columns 1 and 3 includes children born after June 2005 and who were < 6 months and whose mothers were potentially undergoing the intervention at the time of survey. Sample in columns 2 and 4 includes children born after July 2005 and who were aged between 6 and 53 months at time of survey. Each column represents a different dependent variable which takes value 1 if the the child has suffered the condition specified in the column heading in the 15 days previous to the survey as reported by the main respondent, 0 otherwise.

Table 9: Spillovers in Food Intake of Children Born Before Intervention

	[1]	[2]	[3]	[4]
	Number of Foods	**Number of Protein-Rich**	**Number of Staples**	**Number of Fruit and Veg**
T_z	0.441	0.281+	0.135+	0.003
Standard Error	[0.254]	[0.143]	[0.079]	[0.077]
Wild Cluster Bootstrap p-value	{0.148}	{0.072}	{0.086}	{0.969}
Observations	841	843	846	841
R-squared	0.11	0.09	0.07	0.08
IntraCluster Correlation	0.173	0.103	0.198	0.184
Mean, Control	5.355	1.252	1.744	1.793
Maximum	8	4	2	2

Notes to Table: All regressions include controls for age, quadratic in age, gender, wealth at baseline, education of the main respondent and median zone distance to closest trading centre and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Sample includes children born before July 2005 and aged between 41 and 84 months. Data on solid food intake collected in second follow up only. "Number of Foods" is the number of foods (between 1 and 8) taken by the child during the 3 days prior to the survey, "Number of Protein-Rich" takes integer values between 0 and 4 depending on the intake of meat, fish, eggs and beans, "Number of Staples" takes integer values between 0 and 2 depending on intake of nsima and porridge, "Number of Fruit and Veg" takes integer values between 0 and 2.

Table 10: Spillovers in Food Intake of Children Born Before Intervention With and Without Younger Siblings

	Number of Foods		Number of Protein-Rich		Number of Staples		Number of Fruit and Veg	
	Born before the intervention started							
	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed
T_z	0.485	0.347	0.333+	0.142	0.143	0.129+	-0.012	0.054
Standard Error	[0.276]	[0.297]	[0.156]	[0.200]	[0.089]	[0.066]	[0.077]	[0.107]
Wild Cluster Bootstrap p-value	{0.130}	{0.357}	{0.050}	{0.509}	{0.124}	{0.072}	{0.867}	{0.709}
Observations	640	201	642	201	644	202	640	201
R-squared	0.12	0.13	0.10	0.13	0.09	0.06	0.08	0.13
IntraCluster Correlation	0.197	0.142	0.113	0.108	0.251	0.036	0.168	0.224
Mean, Control	5.313	5.505	1.185	1.495	1.746	1.737	1.811	1.726

Notes to Table: All regressions include controls for age, quadratic in age, gender, wealth at baseline, education of the main respondent and median zone distance to closest trading centre and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Sample in columns 1, 3, 5 and 7 includes children born before July 2005 aged between 41 and 84 months and have a younger sibling born after July 2005 ("sibling of directly exposed"); sample in columns 2, 4, 6 and 8 includes children born before July 2005, aged between 41 and 84 months who have no younger sibling born after July 2005 (indirectly exposed). Data on solid food intake collected in second follow up only. "Number of Foods" is the number of foods (between 1 and 8) taken by the child during the 3 days prior to the survey, "Number of Protein-Rich" takes integer values between 0 and 4 depending on the intake of meat, fish, eggs and beans, "Number of Staples" takes integer values between 0 and 2 depending on intake of nsima and porridge, "Number of Fruit and Veg" takes integer values between 0 and 2.

Table 11: Spillovers in Physical Growth of Children Born Before Intervention

	[1]	[2]	[3]	[4]	[5]	[6]
	Height For Age		Weight for Age		Weight for Height	
	Born before the intervention started					
	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed
T_z	-0.272	-0.331	-0.100	-0.308	-0.002	-0.139
Standard Error	[0.160]	[0.245]	[0.163]	[0.296]	[0.146]	[0.231]
Wild Cluster Bootstrap p-value	{0.132}	{0.222}	{0.585}	{0.330}	{0.993}	{0.591}
Observations	398	190	401	195	393	189
R-squared	0.05	0.11	0.01	0.12	0.03	0.13
IntraCluster Correlation	0.048	0.104	0.029	0.213	0.023	0.089
Z-Scores, Control	-2.044	-2.068	-1.029	-0.938	0.341	0.448

Notes to Table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. All regressions include controls for age, age-squared, gender and dummies for the month of interview. Sample in columns 1, 3 and 5 includes children born before July 2005 and aged between 41 and 59 months at time of measurement and have a younger sibling born after July 2005 ("sibling of directly exposed"); sample in columns 2, 4 and 6 includes children born before July 2005 and aged between 41 and 59 months at time of measurement and who have no younger sibling born after July 2005 ("indirectly exposed"). "Height-for-Age", "Weight-for-Age" and "Weight-for-Height" are standardised z-scores relative to the WHO reference population.

Table 12: Spillovers in Morbidity of Children Born Before Intervention

	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]	[10]
	Suffered Diarrhoea		Suffered from Vomiting		Suffered from Fast Breathing		Suffered Fever		Suffered from Chills	
	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed	Sibling of Directly Exposed	Indirectly Exposed
T_z	-0.007	0.042	-0.031	-0.028	0.006	-0.03	-0.018	0.025	-0.017	-0.054
Standard Error	[0.036]	[0.046]	[0.054]	[0.071]	[0.049]	[0.059]	[0.062]	[0.076]	[0.074]	[0.073]
Wild Cluster Bootstrap p-value	{0.903}	{0.376}	{0.613}	{0.675}	{0.899]	{0.621}	{0.777}	{0.759}	{0.889}	{0.478}
Observations	474	239	473	240	472	239	474	240	474	240
R-squared	0.032	0.091	0.06	0.063	0.077	0.047	0.039	0.068	0.04	0.092
IntraCluster Correlation	0.0209	0.0692	0.0743	0.0734	0.103	0.123	0.0556	0.0382	0.231	0.219
Mean, Control	0.113	0.0636	0.19	0.2	0.0985	0.118	0.473	0.509	0.167	0.182

Notes to Table: Notes to table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. All regressions include controls for age, quadratic in age, gender and dummies for the month of interview. Sample in columns 1, 3, 5, 7 and 9 includes children born before July 2005 and aged between 41 and 59 months at time of measurement and have a younger sibling born after July 2005 ("sibling of directly exposed"); sample in columns 2, 4, 6, 8 and 10 includes children born before July 2005 and aged between 41 and 59 months at time of measurement and who have no younger sibling born after July 2005 ("indirectly exposed"). Each column represents a different dependent variable which takes value 1 if the the child has suffered the condition specified in the column heading in the 15 days previous to the survey as reported by the main respondent, 0 otherwise.

Table 13: Effects on Adult Health

	[1]	[2]	[3]	[4]	[5]	[6]	[7]	[8]	[9]
	Walk 5 kms Easily	**Carry a 20 kg Load Easily**	**Unable to Carry Out Daily Activities**	**Suffered Diarrhoea**	**Suffered Fever**	**Suffered from Cough**	**Suffered from Chills**	**Suffered from Vomiting**	**Suffered from any Illness symptom**
					Males				
T_z	-0.066	-0.004	0.073+	-0.002	0.060	0.009	0.022	0.011	0.054
Standard Error	[0.051]	[0.031]	[0.038]	[0.012]	[0.045]	[0.055]	[0.030]	[0.017]	[0.060]
Wild Cluster Bootstrap p-value	{0.228}	{0.945}	{0.068}	{0.863}	{0.188}	{0.919}	{0.492}	{0.603}	{0.380}
Observations	3809	3809	3816	3751	3752	3758	3748	3760	3744
R-squared	0.088	0.086	0.015	0.001	0.01	0.004	0.005	0.005	0.01
IntraCluster Correlation	0.109	0.052	0.039	0.008	0.059	0.077	0.053	0.016	0.085
Mean, Control	0.870	0.893	0.350	0.065	0.285	0.275	0.102	0.121	0.501
					Females				
T_z	-0.078	0.001	0.056	-0.006	0.071	-0.004	0.014	0.015	0.050
Standard Error	[0.052]	[0.033]	[0.042]	[0.014]	[0.043]	[0.055]	[0.040]	[0.035]	[0.054]
Wild Cluster Bootstrap p-value	{0.180}	{0.947}	{0.208}	{0.671}	{0.104}	{0.887}	{0.781}	{0.703}	{0.363}
Observations	4296	4295	4295	4252	4252	4256	4246	4241	4241
R-squared	0.122	0.153	0.021	0.009	0.015	0.009	0.01	0.008	0.018
IntraCluster Correlation	0.102	0.058	0.041	0.010	0.048	0.080	0.076	0.047	0.072
Mean, Control	0.870	0.893	0.350	0.065	0.285	0.275	0.102	0.121	0.501

Notes to Table: All regressions include controls for age, age-squared, gender, and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. Each column represents a different dependent variable which takes value 1 if the column heading is correct according to the main respondent and 0 otherwise. In Columns 1 and 2, the dependent variable takes value 1 if the adult member can do what is specified in the column heading, 0 otherwise. In columns 3-9, the dependent variable takes value 1 if the the adult member has suffered the condition specified in the column heading in the 15 days previous to the survey as reported by the main respondent, 0 otherwise.

Table 14: Intervention Effects on Family Planning and Fertility

	[1]	[2]	[3]	[4]
	Use of any modern family planning method	**Number of children since July 2005**	**Had at least one child since July 2005**	**Had at least two children since July 2005**
T_z	0.016	-0.047	-0.034	-0.012
Standard Error	[0.041]	[0.046]	[0.030]	[0.022]
Wild Cluster Bootstrap p-value	{0.693}	{0.354}	{0.280}	{0.563}
Observations	2809	1657	1657	1657
R-squared	0.055	0.07	0.08	0.02
IntraCluster Correlation	0.036	0.014	0.011	0.017
Mean, Control	0.379	0.583	0.474	0.107

Notes to Table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. ** p<0.01, * p<0.05, + p<0.1. All regressions includes controls for age, quadratic in age, and (family planning regression only) for dummies for the month of interview. "Number of children since July 2005" is the number of children born to the main respondent and surveyed at age 1 month since July 2005; "Had at least one (two) child(ren) since 2005" is an indicator which equals 1 if main respondent has had at least 1(2) child(ren) since July 2005. Column 1 sample includes women 17-43 years old (when available, both waves responses are included). Sample in columns 3 and 4 includes all women surveyed as main respondents in the 2008 survey, and comes from the Mai Mwana Health Surveillance System, which measures at age 1 month all children born to these women since the start of the intervention

Table 15: Heckman Selection Model for Attrition, Consumption

	[1]	[2]	[3]	[4]	[5]	[6]	[7]
	Per Capita Monthly Food Consumption for:						
	Total Non-durable	**Food**	**Health**	**Cereals**	**Proteins**	**Fruit and Vegetables**	**Other Foods**
Ordinary Least Squares							
T_z	502.889**	408.037*	6.053+	2.780	113.671*	224.985+	64.440*
Standard Error	[165.785]	[144.746]	[2.949]	[46.617]	[40.631]	[97.743]	[24.633]
Wild Cluster Bootstrap p-value	{0.016}	{0.030}	{0.082}	{0.887}	{0.022}	{0.054}	{0.036}
Observations	3190	3200	3199	3205	3202	3204	3204
R-squared	0.05	0.06	0.01	0.11	0.02	0.17	0.02
IntraCluster Correlation	0.095	0.111	0.023	0.074	0.042	0.172	0.053
Mean Control Areas	2146.00	1784.00	17.11	606.00	349.80	679.70	149.70
Heckman Selection Model for Attrition							
T_z	495.150**	409.823**	5.286	-2.710	108.387*	235.514**	66.209*
Standard Error	[153.511]	[141.214]	[3.568]	[50.674]	[43.729]	[85.447]	[26.558]
Inverse Mills ratio	-1,326.323*	-1,372.899*	7.494	-136.71	-259.041	-798.571+	-178.536
	[610.709]	[646.707]	[16.708]	[176.344]	[169.515]	[423.768]	[146.157]
Selection Equation (coefficients)							
T_z	-0.081	-0.080	-0.080	-0.080	-0.08	-0.079	-0.080
	[0.152]	[0.141]	[0.148]	[0.146]	[0.144]	[0.137]	[0.148]
# children 0-3	0.221*	0.22*	0.220*	0.220*	0.220*	0.220*	0.221*
	[0.093]	[0.092]	[0.093]	[0.094]	[0.092]	[0.096]	[0.090]
land size (acres)	-0.017	-0.017	-0.017	-0.018	-0.018	-0.018	-0.018
	[0.015]	[0.014]	[0.016]	[0.015]	[0.015]	[0.015]	[0.014]
Observations	4975	4986	4984	4991	4988	4990	4990

Notes to Table: Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. Standard errors for Heckman Selection model computed using a block bootstrap method. ** p<0.01, * p<0.05, + p<0.1. Regression includes month-year dummies to control for seasonality. Sample in top panel includes only households successfully interviewed in the follow-up surveys, that in bottom panel includes all households drawn into the follow-up samples. All coefficients in terms of Malawi Kwacha. (The average exchange rate to the US Dollar was approx. 140MK = 1 US$ at the time of the surveys). "Total Non-Durable" is the sum of food consumption and expenditures on items such as transport, education, health, etc, "Food" is food consumption (including food which is not bought), "Health" is the per-capita expenditure (in MK) on health care, "Cereals" includes consumption of rice, maize flour and bread, "Proteins" includes consumption of milk, eggs, meat, fish and pulses "Fruit and Vegetables" includes consumption of green maize, cassava, green leaves, tomatoes, onions, pumpkins, potatoes, bananas, masuku, mango, ground nuts and other fruits and vegetables, "Other Foods" includes cooking oil, sugar, salt, alcohol and other foods. Excluded variables in the second stage of the Heckman Selection Model are "# children 0-3" (number of children aged 0-3 of first follow-up survey interviewer) and "land size(acres)" (land size in acres of first follow-up survey interviewer).

Table 16: Heckman Selection Model for Attrition, Main Respondent (Female) Labor Supply

	[1]	[2]	[3]
	Works	**Has at least 2 jobs**	**Weekly Hours Worked**
Ordinary Least Squares			
T_z	-0.077	0.036	-1.665
Standard Error	[0.073]	[0.030]	[3.095]
Wild Cluster Bootstrap p-value	{0.346}	{0.214}	{0.657}
Observations	3298	3297	3040
R-squared	0.093	0.039	0.075
IntraCluster Correlation	0.287	0.033	0.188
Mean, Control	0.929	0.136	27.540
Heckman Selection Model for Attrition			
T_z	-0.073	0.035	-1.683
	[0.085]	[0.034]	[3.352]
Inverse-Mills Ratio	-0.454	-0.238	-3.201
	[0.439]	[0.152]	[9.770]
Selection Equation (coefficients)			
T_z	-0.075	-0.075	-0.061
	[0.140]	[0.149]	[0.156]
# children 0-3	0.219*	0.219**	0.243**
	[0.085]	[0.082]	[0.087]
land size (acres)	-0.017	-0.017	-0.015
	[0.014]	[0.016]	[0.017]
Observations	4992	4991	4742

Notes to Table: All regressions include controls for age, age-squared, marital status, education and dummies for the month of interview. Standard errors computed using the cluster-correlated Huber-White estimator are reported in brackets, with clustering at the level of the cluster (at which treatment was assigned); wild cluster bootstrap-t p-values in curly brackets. Standard errors for Heckman Selection model computed using a block bootstrap method. ** p<0.01, * p<0.05, + p<0.1. The sample in the top panel includes all female main respondents aged 15-65 at time of the follow-up surveys who were interviewed, the sample in the bottom panel includes all women drawn in the sample for the follow-up surveys. "Works" in an indicator of whether individual had an income-generating activity at the time of the survey, "Has at least 2 jobs" is an indicator for whether individual has 2 income generating activities, "Weekly Hours worked" give the total hours worked in the week prior to the survey on both income generating activities. Excluded variables in the second stage of the Heckman Selection Model are "# children 0-3" (number of children aged 0-3 of first follow-up survey interviewer) and "land size(acres)" (land size in acres of first follow-up survey interviewer).

Appendix 1

Table A1. Differences in characterisitcs between those that attrited and those who did not

	Non-attrited	Difference Attrited - Not Attrited	p-value
Woman's Characteristics in 2004			
Married (dv = 1)	0.646	-0.112	0.004**
Some Primary Schooling or Higher	0.704	0.053	0.068+
Some Secondary Schooling or Higher	0.055	0.042	0.001**
Age (years)	25.169	-1.904	0.002**
Chewa	0.934	-0.021	0.118
Christian	0.982	-0.008	0.184
Farmer	0.661	-0.104	0.002**
Student	0.213	0.087	0.002**
Small Business/Rural Artisan	0.050	0.005	0.555
Age less than 16 in 2004	0.142	0.068	0.000**
Household Characteristics in 2004			
Agricultural household	0.996	-0.010	0.088+
Main Flooring Material: Dirt, sand or dung	0.910	-0.046	0.001**
Main roofing Material: Natural Material	0.859	-0.044	0.062+
HH Members Work on Own Agricultural Land	0.925	-0.032	0.048+
Piped water	0.026	0.014	0.106
Traditional pit toilet (dv = 1)	0.818	-0.053	0.046*
# of hh members	5.837	-0.090	0.468
# of sleeping rooms	2.215	0.002	0.943
HH has electricity	0.004	0.002	0.651
HH has radio	0.646	-0.003	0.833
HH has bicycle	0.511	0.014	0.583
HH has motorcycle	0.006	0.006	0.210
HH has car	0.006	-0.002	0.330
HH has paraffin lamp	0.947	-0.016	0.044**
HH has oxcart	0.048	0.007	0.472
N	1594	902	

Notes to Table: + indicates significant at the 10% level, * indicates significant at the 5% level. p-values reported here are computed using the wild cluster bootstrap-t procedure as in Cameron *et al.* 2008, explained in section 4.1. Non-attrited refers to women (and their households) actually interviewed in 2008-09 (and used in the analysis). Attrited refers to women (and their households) drawn to be part of the sample in 2008-09, but who were not interviewed.

Appendix 2: Proofs

Proof of Proposition 1

We assume that the solution is an interior one so that the budget constraint is binding at the optimum. Substituting in the health production function and budget constraint for H and A in the objective function, we can express the optimisation problem as:

$$\underset{\{C,L\}}{Max} F(C, L;\, \theta)$$

$$\text{where } F(C, L;\, \theta) = U(\tfrac{w(T-L)-C}{p}, L) + G(h(\theta C))$$

The first order conditions are:

$$F_C(C, L;\, \theta) = -\frac{1}{p}U_A'(\tfrac{w(T-L)-C}{p}, L) + G'(h(\theta C))h'(\theta C)\theta = 0$$

$$F_L(C, L;\, \theta) = -\frac{w}{p}U_A'(\tfrac{w(T-L)-C}{p}, L) + U_L'(\tfrac{w(T-L)-C}{p}, L) = 0$$

Differentiating the two first order conditions, we get

$$\begin{bmatrix} F_{CC} & F_{CL} \\ F_{CL} & F_{LL} \end{bmatrix} \begin{bmatrix} dC \\ dL \end{bmatrix} = \begin{bmatrix} -F_{C\theta} \\ -F_{L\theta} \end{bmatrix} d\theta \quad \text{(A1)}$$

Noting that $F_{L\theta} = 0$ due to additive separability, we get that

$$\frac{dC}{d\theta} = \frac{\begin{vmatrix} -F_{C\theta} & F_{CL} \\ -F_{L\theta} & F_{LL} \end{vmatrix}}{\begin{vmatrix} F_{CC} & F_{CL} \\ F_{CL} & F_{LL} \end{vmatrix}} = -\frac{F_{C\theta}F_{LL}}{|SOC_2|} \quad \text{(A2)}$$

where

$$|SOC_2| = \begin{vmatrix} F_{CC} & F_{CL} \\ F_{CL} & F_{LL} \end{vmatrix}$$

Since $U(.)$ is concave in L and $G(.)$ and $h(.)$ are concave in their arguments, $F_{LL} < 0$ and $F_{CC} < 0$ at the optimum, and so $|SOC_2| > 0$. Consequently, the sign on $\frac{dC}{d\theta}$ is the same as the sign on $F_{C\theta} = \theta C[G''(h')^2 + h''G'] + G'h'$. **QED**

Proof of Proposition 2

To prove that leisure, L decreases when child consumption, C, increases due to an increase in θ, note that from (A1), we obtain that

$$\frac{dL}{d\theta} = -\frac{F_{CL}}{F_{LL}}\frac{dC}{d\theta} \quad \text{(A3)}$$

where:

$$F_{CL} = \frac{1}{p}\left(\frac{w}{p}U_{AA} - U_{LA}\right) \quad \text{(A4)}$$

$$F_{LL} = \left(\frac{w}{p}\right)^2 U_{AA} - 2\frac{w}{p}U_{LA} + U_{LL} \quad \text{(A5)}$$

Note that if $U_{LA} > 0$ or if $wU_{LA} - pU_{LL} > 0$, then both $F_{CL} < 0$ and $F_{LL} < 0$. Then, by (A3),

$$sign\left(\frac{dL}{d\theta}\right) = -sign\left(\frac{dC}{d\theta}\right) \quad \text{(A6)}$$

which proves the first part of the proposition.

The proof of the second part of the proposition (that household consumption increases) then follows immediately. According to the budget constraint, household consumption $(pA + C) = w(T - L)$. Therefore, if L decreases, household consumption increases necessarily, which proves the second part of the proposition.

To prove the third part of the proposition (that adult consumption decreases), write the budget constraint as

$$A = \frac{1}{p}[w(T - L) - C] \quad \text{(A7)}$$

Differentiating (A7) with respect to θ and using (A3), we obtain that:

$$\frac{dA}{d\theta} = \left(\frac{1}{pF_{LL}}\right)(wF_{CL} - F_{LL})\left(\frac{dC}{d\theta}\right) \quad \text{(A8)}$$

Substituting (A4) and (A5) into (A8), we obtain:

$$\frac{dA}{d\theta} = \left(\frac{1}{pF_{LL}}\right)\left(\frac{w}{p}U_{LA} - U_{LL}\right)\left(\frac{dC}{d\theta}\right) \quad \text{(A9)}$$

which implies that adult consumption decreases when child consumption increases $\left(\frac{dC}{d\theta}\right) > 0$ because $\left(\frac{1}{pF_{LL}}\right) < 0$ (by the second order conditions), and we have assumed that either $U_{LA} > 0$ following Mortensen 1977 among others or $wU_{LA} - pU_{LL} > 0$ holds. **QED**

Proof that $U_{LA} > 0$ is sufficient for the Second Order Conditions to hold

The Lagrangian function associated with the optimization problem is

$L = U(A, L) + G(h(\theta C)) + \mu(pA + C - w(T - L))$

The relevant bordered Hessian is $D = \begin{bmatrix} 0 & p & w & 1 \\ p & U_{AA} & U_{AL} & 0 \\ w & U_{AL} & U_{LL} & 0 \\ 1 & 0 & 0 & \frac{d^2L}{dC^2} \end{bmatrix}$,

and the second principal minor is $D_2 = \begin{bmatrix} 0 & p & w \\ p & U_{AA} & U_{AL} \\ w & U_{AL} & U_{LL} \end{bmatrix}$.

The sufficient conditions for optimality are that $|D_2| > 0$ and $|D| < 0$.

$|D_2| = w(pU_{AL} - wU_{AA}) + p(wU_{AL} - pU_{LL})$. If $U_{AL} > 0$, then $|D_2| > 0$.

$|D| = \frac{d^2L}{dc^2}|D_2| - \begin{vmatrix} p & w & 1 \\ U_{AA} & U_{AL} & 0 \\ U_{AL} & U_{LL} & 0 \end{vmatrix} = \frac{d^2L}{dC^2}|D_2| - U_{AA}U_{LL} + (U_{AL})^2$. From above, we know that $|D_2| > 0$. Then, given concavity of $U(A, L)$, $U_{AA}U_{LL} - (U_{AL})^2 > 0$, and given that $\frac{d^2L}{dC^2} = \theta^2 G''(h')^2 + \theta^2 G' h'' < 0$, the condition $|D| < 0$ is verified. **QED**

Appendix 3: Questions on Nutrition Knowledge

If an infant is being breastfed and suffers from diarrhoea, should the breastfeeding :

1 Continue as usual
2 Increase
3 Decrease
4 Stop and replace with another type of milk or liquid
5 Don't Know

Which of the following is most nutritious for infants between 6 months and 3 year ?

1 Biscuits
2 Groundnuts or soya
3 They both have the same nutritional value
4 Don't Know

When should you start to give some solid foods to the baby?

1 From birth
2 After 1 month old
3 After 3 months old
4 After 6 months old
5 Don't Know

If a woman is HIV positive, how should she feed her baby?

1 Exclusive breast feeding for 6 months, followed by early cessation
2 Exclusive breast feeding for 6 months, followed by complementary feeding
3 Complementary feeding from birth
4 Don’t know

What is more nutritious for a child older than 6 months:

1 Nsima
2 Phala (porridge)
3 Both are the same

Can you explain to me how best to cook fish with phala for a child older than 6 months (tick all those mentioned).

1 Pound the fish
2 Sieve the powder
3 Add powder to flower/phala
4 Use powder + flour to prepare phala
5 None of the above
6 Don't Know

Should eggs be given to an infant aged 9 months and above?

1 Yes
2 No
3 Don't know