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ARTICLE

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More evidence on the relationship between cash transfers and child height

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ABSTRACT

We examine the effect of the Zambia Child Grant Programme – an unconditional cash transfer (CT) targeted to rural households with children under age five – on height-for-age up to four years after programme initiation. The CT scheme had large positive effects on nutritional inputs like food expenditure and meal frequency, but no impact on child height-for-age. Production function estimates indicate that food carries little weight in the production of child height in the study sample. In settings with poor health infrastructure and harsh disease environments, a stand-alone CT is unlikely to address long-term chronic malnutrition unless accompanied by complementary interventions.

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1. Introduction

Over 700 million people in developing countries are currently reached by some type of cash transfer (CT) programme (World Bank 2015). In sub-Saharan Africa (SSA), the rise of such programmes has been nothing short of phenomenal – as of 2014, 40 countries offer unconditional CTs as part of their social protection system. The rapid expansion of cash as the primary instrument for poverty alleviation has been referred to as the 'quiet revolution' in development policy (Barrientos and Hulme 2008). Reviews of the evidence on the impacts of CT programmes have documented clear and positive effects in areas such as food security (Hidrobo et al. 2018), schooling (Baird et al. 2013; Fiszbein and Schady 2009) and productive activity (Daidone et al. 2016). However, aggregate evidence does not point to overwhelmingly positive effects on young child nutrition. This is concerning because CTs are often implemented under their assumed potential to break the inter-generational transfer of poverty, particularly CT programmes that condition cash receipt on specific behaviours around health and nutrition. Further, with an estimated 151 million children under age five throughout the world being stunted, chronic malnutrition continues to be one of the most important development challenges (WHO 2018).

Several recent articles have reviewed the existing state of evidence on the effects of CT programmes on child nutritional status and have found mixed results (Manley, Gitter, and Slavchevska 2013; de Groot et al. 2017; Manley and Slavchevska 2017; Owusu-Addo, Renzaho, and Smith 2018). For example, the systematic review by Manley, Gitter, and Slavchevska (2013) included 17 programmes and 21 studies that reported on height-for-age z-scores (HAZ). The meta-analysis revealed a slightly positive but not statistically significant effect of CT schemes (conditional and

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unconditional) on HAZ. The authors report larger effects among programmes where initial health conditions and infrastructure are worse and where households are poorer, suggesting some benefit of transfers for nutritional status among the most vulnerable. They report no systematic difference between conditional and unconditional programmes, although programmes where the conditions are unrelated to health (for example, work conditions) appear to be detrimental for nutritional status. Finally, they report a publication bias with results containing statistically significant positive CT impacts more likely to be published.

de Groot et al. (2017) lay out a conceptual framework to trace the potential pathways through which CTs could affect child nutritional status, and then summarise the evidence on these potential pathways focusing on SSA. Their conceptual framework is based on UNICEF's extended model of care as presented by Smith and Haddad (2002). The framework identifies three channels through which poverty or household economic status affects child nutrition: the environment, food intake and health behaviours. Specific components within each channel may interact with or moderate the effects of factors operating through other channels, making the overall aetiology complex. For example, the positive impact of food availability could be mitigated by actual feeding practices (the health behaviour channel) resulting in little to no overall impact on child nutritional status. The complexity inherent in this framework is insightful insofar as it highlights the difficulty in finding a direct link between CTs and nutritional status. In their review of the evidence from SSA, the authors find no systematic positive impact of CT programmes on child nutritional status, but do find positive effects on intermediate outcomes such as food security (typically measured at the household level) and use of health services. Both Manley, Gitter, and Slavchevska (2013) and de Groot et al. (2017) emphasise the relative dearth of evidence on the effects of CTs on the intermediate outcomes across the three channels, especially outcomes measured at the child level. This evidence would help our understanding of what CT programmes can and cannot do for child nutrition, as well as the integrated interventions necessary to enable social transfers to shape child nutrition.

The present article addresses these issues within the context of the Zambia Child Grant Programme (CGP), a government-run unconditional CT. The intervention consisted solely in a pure CT and the transfers amounted to about 27 percent of the average eligible household's expenditures at the start of the programme in 2011. The CGP operated in three districts with the highest child mortality and poverty rates in the country, targeted all households with a child under age five years, and paid transfers directly to female primary caregivers. A key programme objective beyond raising household food security was to improve young child nutritional status. A longitudinal cluster randomised control trial (RCT) was implemented between 2010 and 2014 to evaluate the impact of the CTs.

The CGP itself had a transformative effect on the lives of beneficiaries. Handa et al. (2018) summarise its impacts across domains ranging from consumption to agricultural activity and child material needs, and show that the programme led to an income multiplier of approximately 1.5 (each kwacha transferred generated an additional 0.50 kwacha in spending). Several studies have explored CGP's impacts on different nutrition and child health outcomes as part of their investigation into the manifold potential effects of the programme. For example, Handa et al. (2014), Seidenfeld et al. (2014) and Tiwari et al. (2016) determined that the programme enhanced food security among beneficiary households; Handa et al. (2014), Seidenfeld et al. (2014) and the American Institutes for Research (AIR) (2016) found, however, that there were no significant impacts on child anthropometry (as measured by weight-for-age z-scores (WAZ), weight-for-height z-scores (WHZ) and HAZ).

In this paper, we conduct a more thorough investigation into CGP's impacts on a specific outcome of child nutritional status – child height. Our choice of outcome is dictated by the fact that size or height in early life is reflective of nutritional conditions in the period between conception and a child's second birthday (the first 1,000 days of life), and is a strong predictor of later life outcomes ranging from educational attainment, cognitive performance, adult health and productivity (Black et al. 2013). We first probe how the CGP shaped young child height, as measured by HAZ and stunting. Next, using the health production function framework, we identify theoretically plausible inputs into child height that are captured in the evaluation surveys, and estimate input demand functions to assess programme effects on these inputs. Finally, we explore the link between child HAZ and these pathways during the evaluation period. While most previous studies examining CGP's impacts on child nutrition have used data only two years into the programme, in the current study, we also incorporate information from subsequent rounds of surveys (the last of which was conducted four years after the CGP was initiated), and thus look at both short- and medium-term consequences of the CT.

Our results underscore that the CGP had no effects on child height. In exploring potential mechanisms, we find strong positive effects of the programme on different food consumption measures. However, there are no impacts on health inputs such as morbidity, and mixed impacts on water and sanitation. The health production function itself is poorly estimated, explaining at most only 6 percent of the variation in HAZ. In addition, none of the inputs that are actually affected by the CGP are significant in the health production model, suggesting a weak correlation between HAZ and inputs such as food expenditures that the programme is able to affect. We also look at two subsamples that could be expected to benefit most from the transfers – children born at the start of the programme and those born into the programme. Children born at CGP's inception would have received transfers continuously for much of the vital first 1,000 days of life. Given the relatively long time-span of the evaluation, approximately 1,668 children were born into the study sample and might have benefited from improved maternal nutrition during the in utero period and from increased food consumption early in life. We do not, however, find programme effects even on these samples of children.

The evidence from the CGP is therefore consistent with the previous reviews on the effects of CTs on child nutrition. We find no impacts on child height even though this programme operated in remote districts with very poor baseline measures of nutritional status and where the scope for improvement was high; it was specifically targeted to households with very young children, many of whom would have benefitted from the transfers early in life; and the transfers were substantial – at more than a quarter of baseline average household expenditures.[1] The study sample is extremely impoverished. The evidence presented here suggests that despite the large gains in food consumption and diet diversity, the disease environment, access to health services and maternal health knowledge remain essentially the same. It is possible that for the outcomes that the CGP was able to improve, the realised effects were not substantial enough to ultimately impact child nutritional status. Alternatively, if these inputs are complements, it might be necessary to bring about sizeable improvements across *all* vital pathways (the environment, food intake and health behaviours) to achieve final effects. These synergistic impacts might be best achieved by combining cash transfers with complementary interventions – for example, interventions that provide nutritional information (such as behaviour change communication), or improve access to and/or the quality of services (such as health care, or water and sanitation). Several such 'cash plus' programmes are currently being implemented around the world, though the evidence base on the effects of these schemes is still scant (Barry, Maïdoka, and Premand 2017; Roelen et al. 2017; Roy et al. 2017).

The results of this analysis are a valuable addition to the evidence base on the impacts of cash transfers on child nutritional status. While most studies on this topic probe effects at most a couple of years after programme initiation (for example, see Table A2 in Manley, Gitter, and Slavchevska 2013), we track children in households that had been receiving CTs for up to four years. Our main sample, with 2,464 children at baseline, is also larger than that of similar studies (the review by Manley, Gitter, and Slavchevska (2013) covers studies with an average sample size of 1,195 observations). Finally, through detailed analysis, we show that the results are consistent across age groups and samples, thus rigorously establishing that there are no statistically significant positive changes to child height within the context of the Zambian CGP.

2. CGP

The Zambian Ministry of Community Development, Mother and Child Health (MCDMCH) began implementing the Child Grant unconditional CT programme (CGP) in three districts of the country – Kalabo and Shangombo in the west, and Kaputa in the north – in February 2011.[2] The programme aimed to alleviate poverty and the intergenerational transmission of poverty in these remote and impoverished districts characterised by high child mortality, morbidity, stunting, and wasting; another objective was to improve young child health and nutrition (AIR 2013).[3] The CGP targeted households with a child under the age of five years and provided the primary female caregiver of the child with roughly US$24 in cash once every two months (or roughly US$12 per month) irrespective of household size. At 27 percent of pre-intervention household expenditure, the transfer was expected to cover the cost of one meal a day for an average sized household for a month. The benefit size was adjusted routinely to keep pace with inflation. Households were expected to exit the CGP when the youngest child turned five years old.

A randomised control trial was implemented to estimate programme effects and evidence indicated that the programme had positive impacts on a range of outcomes including food security, consumption, and child material needs. The CGP also allowed households to accumulate livestock, invest in productive activities and build-up savings, and to thereby smooth consumption over time (Handa et al. 2016).

3. Conceptual framework

Our empirical approach in this paper is guided by the Becker (1965) household production function model as applied to child health and nutrition (Strauss and Thomas 1995). The model is well-known and distinguishes between three key relationships and associated empirical requirements that we wish to highlight here.

The first is the child nutrition production function, which relates child nutritional status to the physiological and behavioural inputs that have a direct effect on nutritional status. Examples of these inputs include caloric intake and features of the disease environment that directly relate to pathogen exposure such as faecal presence or the use of unclean water. Beyond accurate measurement (an issue that affects all aspects of the empirical specifications described below), the important econometric issue surrounding the estimation of the health production function is the idea that inputs are choices and are based on information known to the decision-maker (typically the parent) but not to the researcher, such as the child's innate health endowment.

The second is the input demand functions, which relate the inputs that enter into the nutrition production function to their main determinants, typically own prices and those of related inputs, plus other exogenous factors that might affect the full cost of using an input or shape preferences or tastes. A key input is time devoted to the production of health or nutrition, since virtually all inputs must be combined with time in order to be effective. The time cost of acquiring immunisations or curative and preventive health services can be quite prohibitive when access to services is limited, as is the case in our study sample, and can often swamp the direct cost of the services themselves. From an empirical perspective, the input demands, because they are reduced form 'solutions' to the utility maximisation problem, are functions of all exogenous variables in the model.

The third set of relationships are the final demand functions for goods and services that enter directly into the utility function, unlike input demands that only contribute to utility through their effect on nutrition. The most important final demand for our purposes is of course the demand for child nutrition, which is again a function of all the exogenous variables in the system, prices and factors that shape tastes and preferences. In this analysis, we model all three relationships: the nutrition production function, the final demand and the input demands.

4. Data and methodology

4.1. Study design

The Government of Zambia and UNICEF commissioned the CGP impact evaluation – a longitudinal cluster randomised control trial (RCT) with a baseline survey in 2010 and several follow-up surveys over 48-months. Due to resource constraints and the demonstration nature of the programme, the government did not scale-up the CGP throughout the initial districts, which allowed for the introduction of an experimental design. Thirty of about 100 community welfare assistance committees (CWACs or communities) in each of the three study districts were randomly chosen through a lottery to be included in the study. A list was created of all eligible households with a child under the age of three within these communities. While CGP was targeted to households with children under the age of five, a younger age limit was set for inclusion in the study sample so that these households would be eligible to receive transfers for at least two years. Subsequently, 28 households were randomly selected from each community for inclusion in the study sample. The final study sample comprised 2,519 households across 90 communities. After the 2010 baseline survey, coin flips were used to assign half the clusters per district to the treatment and half to the control group. The first transfer to the treatment group was made in February 2011; the control group – or delayed treatment group – was scheduled to receive transfers after the completion of the study. Figure A1 depicts the timeline of the study.

Data used in this analysis come from surveys conducted at baseline and 24-, 36- and 48-months after the CGP began. While a survey was also conducted at 30-months, it was shorter than the others and was oriented mainly towards assessing the impact of CGP on consumption smoothing. Since its survey instrument is less comparable to those used in the other survey rounds, we do not use its data for the current analysis. The survey instruments used in this paper collected a wealth of data on consumption, health, education, housing, agriculture and productive activities. Child anthropometric data (height and weight) were collected at every survey round – for children five years and under at baseline, seven years and under at 24-months and 30-months, eight years and under at 36-months, and nine years or under at 48-months. The age range of children measured at follow-up surveys was expanded to continue following children measured at baseline. Ethical review for the study was obtained at the American Institutes for Research (AIR) in Washington, D.C. and at the University of Zambia in Lusaka.[4]

As described below, our main study sample includes children who were aged 0–36 months at baseline and were measured for height at this time. We focus on these children since the study sample was specifically built around households with children in this age range and since for many of these children, any subsequent exposure to treatment would occur during the crucial early years of life. The anthropometric measures we focus on – HAZ and stunting – are constructed using World Health Organisation guidelines.

In Table 1, Column 1, we summarise characteristics of children 0–36 months at baseline. Mean baseline HAZ is −1.23 and 31 percent of children are stunted. Given the targeting criteria, the typical eligible household is still quite early in its life cycle – the mean age of mothers of the target children is 30 years, 77 percent are married and 57 percent of all household members are children aged 12 years or below. Households are ultra-poor – mean per capita consumption is close to US$0.30 - per day, of which 75 percent is devoted to food (Handa et al. 2016). Just 21 percent of the sample uses water from a protected source and less than half have access to a toilet, primarily a pit latrine.

Note that given baseline HAZ summary statistics for children in the age range of interest (mean of −1.23, standard deviation of 1.68 and intra-cluster correlation of 0.019) and treatment-control baseline balance (which we discuss in the next sub-section), ex post power calculations indicate that the evaluation would be able to detect programme effects on HAZ that were 0.24 standard deviations or greater with power 0.8 and 0.05 significance level. Thus, the study is powered to identify even 'small' effect sizes (Cohen 1988).

Table 1. Baseline summary statistics for main sample of children (aged 0–36 months at baseline).

	(1)	(2)	(3)	(4)
	All	Control	Treatment	P-value of difference
Age in months	19.99	20.20	19.77	0.22
Female	0.51	0.51	0.51	0.90
Height-for-age z-scores HAZ	−1.23	−1.23	−1.22	0.83
Stunted (< −2 HAZ)	0.31	0.31	0.30	0.66
Household characteristics				
Household size	5.82	5.73	5.92	0.34
# members aged 0–5 years	2.01	2.01	2.01	0.99
# members aged 6–12 years	1.28	1.27	1.29	0.77
Recipient-widowed	0.07	0.06	0.07	0.52
Recipient-never married	0.10	0.11	0.10	0.87
Recipient-divorced	0.06	0.07	0.06	0.21
Recipient-highest grade	3.96	3.70	4.22	0.08
Recipient age	29.80	29.44	30.17	0.31
Potential health inputs				
Has access to toilet facilities	0.48	0.49	0.47	0.81
Uses clean water source	0.21	0.20	0.22	0.81
Roof made of purchased material	0.05	0.06	0.05	0.24
Floor made of purchased material	0.03	0.03	0.03	0.72
Wall made of purchased material	0.32	0.32	0.31	0.88
Meal frequency: 3 or more	0.23	0.24	0.23	0.76
Household per capita food expenditure (ZMW)	29.21	28.27	30.15	0.42
Owns mosquito net	0.80	0.80	0.81	0.56
Sick-last 2 weeks	0.28	0.28	0.28	0.95
Child has health card	0.84	0.85	0.83	0.40
Taken to a well-baby/under-5 clinic-last 6 months	0.85	0.83	0.86	0.36
Received vitamin A	0.88	0.88	0.88	0.96
Received 1 BCG, 3 Polio, 3 DPT and 1 measles vaccine	0.67	0.65	0.69	0.20
Observations	2,464	1,234	1,230	

P-values are from Wald tests on the equality of treatment-control means. Standard errors are clustered at the community level.

4.2. Assessment of randomisation

Columns 2 to 4 of Table 1 assess baseline balance between children (aged 0–36 months) in the treatment and control groups. The mean HAZ of treatment group children is higher than that of the control group, but the difference is small in magnitude and not statistically significant. The only significant difference at the 10 percent level between treatment and control children at baseline is survey respondent's mean years of schooling. This is higher in the treatment group by about 0.5 years, though the overall mean is less than four years and very few respondents have completed primary school.

4.3. Sample composition

Not all of the children aged 0–36 months who were measured for height at baseline could be measured at subsequent waves. Table 2 checks for balance on baseline characteristics for those who were lost to follow-up. The estimates reported in the table are derived from models that regress each indicator separately on a treatment indicator variable. The coefficients suggest that the treatment group children who left were similar to their counterparts in the control group. There are a few marginally significant differences and one, distance to a market, that is significant at the 95 percent level of significance. In the case of the latter, the negative coefficient indicates that those in the treatment group who were lost to follow-up at 36-months were less likely to live in more remote areas than the children from the control group who were untracked. We include this variable in the list of control variables that we use in the subsequent empirical exercises.

In Table 3, we examine the treatment-control differences in characteristics of all the children who were measured at follow-up survey rounds but not at baseline (children new to the study sample). Most of these children were born after the programme was initiated and we look at this specific group of children

Table 2. Examining attritors – balance on baseline characteristics for children who are aged 0–36 months at baseline and have height-for-age z-scores (HAZ) measurements at this time, but who are not measured at follow-up surveys.

	Differences in treatment-control means		
	Sample		
	(1)	(2)	(3)
Variable	In baseline, not in 24-month survey	In baseline, not in 36-month survey	In baseline, not in 48-month survey
Height-for-age z-scores HAZ	0.0151	0.122	0.00262
	(0.161)	(0.207)	(0.191)
Age in months	0.688	−0.00369	−0.717
	(0.922)	(0.943)	(0.881)
Female	−0.0384	−0.0949*	−0.00235
	(0.0524)	(0.0564)	(0.0490)
Log household size	0.0472	−0.000108	−0.00552
	(0.0485)	(0.0421)	(0.0462)
Recipient-widowed	0.0564	0.0480	0.0502
	(0.0449)	(0.0393)	(0.0373)
Recipient-never married	0.0148	−0.000477	0.00970
	(0.0330)	(0.0340)	(0.0408)
Recipient-divorced	0.0101	−0.0285	−0.0560*
	(0.0278)	(0.0262)	(0.0309)
Recipient-highest grade	0.517	0.516	0.447
	(0.387)	(0.437)	(0.476)
Recipient age	1.978	0.575	1.173
	(1.348)	(1.640)	(1.300)
Residence in Shangombo	−0.00664	−0.0424	0.00782
	(0.0814)	(0.0987)	(0.0915)
Residence in Kaputa	−0.0420	0.0176	−0.0156
	(0.122)	(0.124)	(0.128)
Log distance to nearest food market (in kilometres)	−0.557	−0.616**	−0.392
	(0.354)	(0.303)	(0.341)
Household expenditure per capita (ZMW)	2.736	5.638	7.662*
	(4.230)	(4.144)	(4.411)
Observations	437	355	393

Each coefficient in this table is from a separate OLS regression of the concerned variable on the treatment indicator. Robust standard errors presented in parentheses are adjusted for clustering at the community level. *** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$.

separately in our analysis. While 'new' children in treatment and control groups are similar, it seems like the treatment group children who were measured in the last two survey rounds are more likely to be female than those in the control group.

4.4. Methodology

In order to identify the impact of CGP on HAZ and health inputs, we estimate a difference-in-differences (DiD) model using the following specification:

$$Y_{it} = B_0 + B_1 X_{it} + \delta_t + B_2 CGP_i + \gamma(\delta_t {}^* CGP_i) + \varepsilon_{it} \quad (1)$$

In this framework, Y_{it} is the outcome of interest for child i at time t (HAZ, health inputs), X_{it} is a vector of covariates that include: child age, sex and baseline characteristics including district of residence (the stratification indicator), log household size, recipient age, education and marital status, distance to food market, household demographic composition and a vector of community-level prices. δ_t are survey round fixed effects, CGP_i is an indicator for treatment group and $\delta_t{}^*CGP_i$ is the vector of terms representing the interaction between the treatment variable and each of the time fixed effects; its coefficients represent the DiD estimators for programme impacts at different survey rounds. ε_{it} is the error term for child i at time t.

In addition to Equation (1), we also estimate fixed effects specifications for the child nutrition production function:

Table 3. Examining joiners – balance on characteristics for children who would have been aged 0–36 months at baseline, and were measured for height-for-age a-scores (HAZ) at follow-up surveys but not at baseline.

	Differences in treatment-control means		
	Sample		
	(1)	(2)	(3)
Variable	In 24-month survey, not in baseline sample (62 months old or younger	In 36-month survey, not in baseline sample (74 months old or younger	In 48-month survey, not in baseline sample (86 months old or younger
Height-for-age z-scores HAZ	−0.0132	0.0231	−0.0342
	(0.104)	(0.111)	(0.0956)
New member of household	−0.0427	−0.00902	−0.0116
	(0.0270)	(0.0112)	(0.0128)
Age in months	1.552*	1.150	0.467
	(0.805)	(0.775)	(0.913)
Female	0.0372	0.0576**	0.0638***
	(0.0308)	(0.0239)	(0.0210)
Log household size	0.0187	0.0167	0.0206
	(0.0320)	(0.0303)	(0.0307)
Recipient-widowed	−0.00583	−0.00164	−0.00849
	(0.0206)	(0.0204)	(0.0171)
Recipient-never married	0.00895	0.00120	−0.00237
	(0.0294)	(0.0268)	(0.0277)
Recipient-divorced	−0.0232	−0.0123	−0.0220*
	(0.0147)	(0.0132)	(0.0127)
Recipient-highest grade	0.403	0.324	0.370
	(0.316)	(0.286)	(0.287)
Recipient age	0.712	0.928	0.517
	(0.817)	(0.766)	(0.753)
Residence in Shangombo	−0.0433	0.00841	0.0212
	(0.102)	(0.0987)	(0.100)
Residence in Kaputa	0.0185	−0.0244	−0.0393
	(0.108)	(0.109)	(0.107)
Log distance to nearest food market (in km)	−0.149	−0.282	−0.253
	(0.256)	(0.245)	(0.243)
Household expenditure per capita (ZMW)	1.371	−0.131	0.0876
	(2.739)	(2.714)	(2.571)
Observations	1,147	1,876	2,237

Each coefficient in this table is from a separate OLS regression of the concerned variable on the treatment indicator. Robust standard errors presented in parentheses are adjusted for clustering at the community level. *** p < 0.01, ** p < 0.05, * p < 0.1. Baseline values used for all household characteristics.

$$HAZ_{it} = \alpha_i + B_1 X_{it} + \delta_t + B_2 TREAT_{it} + \theta(I_{it}) + \varepsilon_{it} \quad (2)$$

Where α_i is the fixed effect for child i, X_{it} are indicators for different age categories, δ_t are survey round fixed effects, $TREAT_{it}$ is an indicator variable equal to 0 for everyone at baseline, 0 for control children in follow-up surveys and 1 for treatment children at follow-up and I_{it} is a vector of different health inputs (such as clean water and food consumption) for child i in time t.

In all specifications, standard errors are clustered at the level of randomisation – the community (or CWAC). While there was no differential attrition in follow-up survey rounds, inverse probability weights are applied in (1) to account for general household attrition (AIR 2013).

4.5. Study samples

We use several samples for this analysis. We first present impacts on HAZ for the pooled cross-sectional sample and the cohort sample, and then do the same for the children born at the start of the programme and for children born into the programme. The pooled cross-sectional sample includes children aged 0–36 months at each survey round – there are 6,801 children in this sample.

The cohort sample includes the 10,566 children who were aged 0–36 months at baseline. During the 24-month, 36-month and 48-month surveys, these children were between ages 24 and 60 months, 36 and 72 months, and 48 and 84 months respectively. Children born at the onset of the CGP (those aged 0-11 months at baseline) are defined as the cohort sample – this sample comprises 3,784 children. The impacts on the sample of 1,668 children born into the programme are examined using data from the 48-month survey for children younger than 48-months at the time.

We present impacts on the input demands for the cohort sample, but results are essentially unchanged when we estimate these models for the other samples (results available on request). Finally, when estimating the health production functions (HAZ fixed effect models), we use an unbalanced panel sample. This comprises a subset of children in the cohort sample – 2,204 children who have data at baseline and who also appear in at least one of the last two survey rounds, as these three survey rounds have the richest set of inputs available in the data. We have estimated the fixed-effects models on the balanced panel with no change in results.

5. Results

5.1. Programme impacts on HAZ

Table 4 presents mean HAZ by treatment status and survey round. On average, children in both groups are more than one standard deviation below the reference mean and this trend persists over time. None of the differences between the treatment and control means are statistically significant. Given that assignment to treatment is random and there is baseline balance, these differences essentially provide causal estimates of the impact of the CGP on height.

The null effect of CGP on child HAZ can be seen visually in Figure 1 – the distribution of HAZ for treatment and control groups almost entirely overlap at each survey round.

In Table 5, Panel A, we present the results of the impact of the CGP on HAZ for the entire pooled cross-sectional sample and several sub-groups using Equation 1. None of the effects attain statistical significance. Panel B of the same table shows that there are also no significant impacts on child stunting. The results for the cohort sample (Table 6) are similar. The effect sizes in Tables 5 and 6 are tiny, and would be of no practical significance even if they were statistically significant. Recall that we have power to detect an effect of 0.24 SD, which itself is a small effect.

Table 4. Mean height-for-age z-scores (HAZ) and proportion stunted in treatment and control group at different survey waves.

	HAZ			Proportion stunted		
	Treatment	Control	T-statistic	Treatment	Control	T-statistic
Panel A: Pooled cross-sectional sample (children aged 0–36 months at each survey wave)						
Baseline *T = 1,230; C = 1,234*	−1.217	−1.235	−0.268	0.301	0.312	0.602
24-month survey *T = 808; C = 834*	−1.456	−1.427	0.387	0.375	0.362	−0.541
36-month survey *T = 717; C = 745*	−1.117	−1.121	−0.043	0.308	0.285	−0.990
48-month survey *T = 608; C = 625*	−1.291	−1.271	0.183	0.352	0.368	0.586
Panel B: Cohort sample (children aged 0–36 months at baseline)						
Baseline *T = 1,230; C = 1,234*	−1.217	−1.235	−0.268	0.301	0.312	0.602
24-month survey *T = 1,300; C = 1,284*	−1.385	−1.428	−0.785	0.321	0.333	0.681
36-month survey *T = 1,406; C = 1,365*	−1.160	−1.061	1.846	0.245	0.245	0.003
48-month survey *T = 1,341; C = 1,406*	−1.184	−1.153	0.559	0.258	0.252	−0.375

The t-statistic tests for difference in treatment-control means.

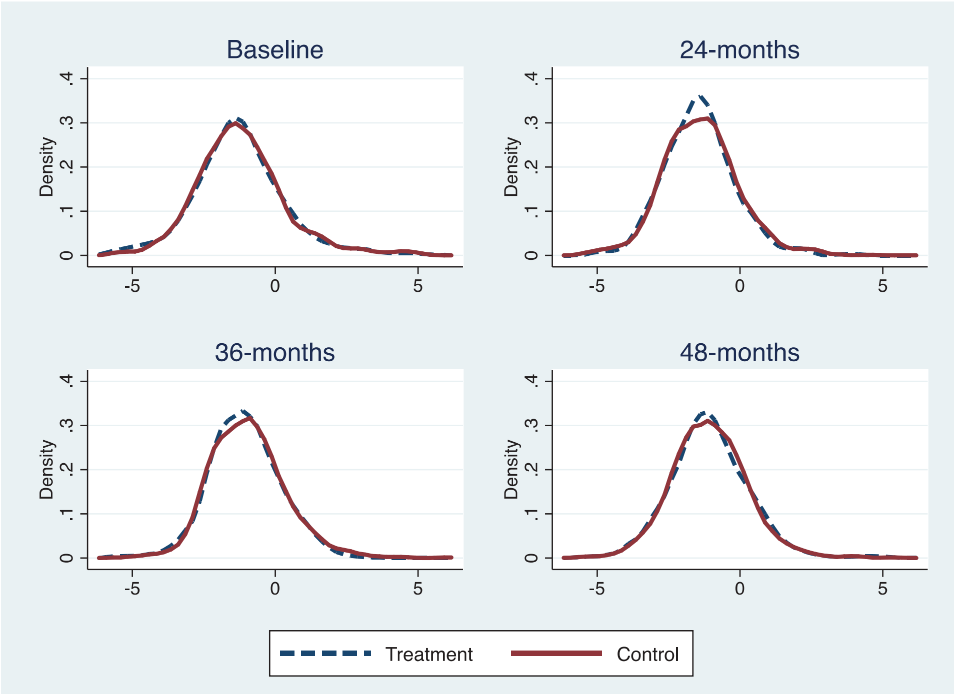

Figure 1. Height-for-age z-scores (HAZ) of children (aged 0–36 months at baseline) measured at all four surveys, Treatment-Control differences.

These graphs are presented for the balanced panel of 1,616 children aged 0–36 months at baseline – 815 from the control group and 801 from the treatment group.

Table 5. Impacts on height-for-age z-scores (HAZ) and stunting across survey waves, Pooled cross-sectional sample.

	Samples (age at each wave)					
	(1)	(2)	(3)	(4)	(5)	(6)
	Main sample Age: 0–36 months	Boys Age: 0–36 months	Girls Age: 0–36 months	Age: 6–36 months	Age: 0–24 months	Age: 0–60 months
Panel A: Impacts on HAZ						
24-Month Impact	−0.0234	0.0514	−0.103	−0.0317	−0.00903	0.00869
	(0.102)	(0.150)	(0.110)	(0.105)	(0.140)	(0.0700)
36-Month Impact	−0.0313	−0.0134	−0.0528	−0.0464	0.0249	−0.0869
	(0.132)	(0.175)	(0.148)	(0.133)	(0.153)	(0.101)
48-Month Impact	−0.0722	−0.0365	−0.0971	−0.0495	0.000428	−0.0717
	(0.144)	(0.211)	(0.186)	(0.151)	(0.190)	(0.109)
Baseline mean	−1.226	−1.335	−1.121	−1.289	−1.140	−1.324
R-squared	0.058	0.049	0.063	0.046	0.077	0.040
Panel B: Impacts on stunting						
24-Month Impact	0.0197	−0.0127	0.0581	0.0156	0.0449	0.00337
	(0.0299)	(0.0421)	(0.0388)	(0.0303)	(0.0389)	(0.0240)
36-Month Impact	0.0386	0.0266	0.0551	0.0370	0.0462	0.0224
	(0.0341)	(0.0515)	(0.0399)	(0.0344)	(0.0388)	(0.0245)
48-Month Impact	0.00540	0.0208	−0.0150	0.00240	−0.0119	0.0197
	(0.0363)	(0.0514)	(0.0447)	(0.0379)	(0.0440)	(0.0279)
Baseline mean	0.306	0.340	0.274	0.318	0.282	0.324
R-squared	0.050	0.048	0.044	0.046	0.060	0.040
Observations	6,801	3,441	3,360	6,434	4,068	12,404

Robust standard errors presented in parentheses are adjusted for clustering at the community level. ** p < 0.01, ** p < 0.05, * p < 0.1. All estimation models include controls for child age and gender, districts, and baseline values of the following variables: log household size, respondent age, education and marital status, household demographic composition, log distance to the nearest food market and a vector of community-level prices.

Table 6. Impacts on height-for-age z-scores (HAZ) and stunting across survey waves, Cohort sample.

	Samples (baseline age)					
	(1)	(2)	(3)	(4)	(5)	(6)
	Main sample Age: 0–36 months	Boys Age: 0–36 months	Girls Age: 0–36 months	Age: 6–36 months	Age: 0–24 months	Age: 0–60 months
Panel A: Impacts on HAZ						
24-Month Impact	0.0307	0.0536	0.00606	0.0572	0.0270	0.0201
	(0.0728)	(0.104)	(0.0965)	(0.0701)	(0.0903)	(0.0658)
36-Month Impact	−0.119	−0.160	−0.0824	−0.101	−0.109	−0.0755
	(0.0887)	(0.124)	(0.110)	(0.0905)	(0.110)	(0.0871)
48-Month Impact	−0.0613	−0.0383	−0.0857	−0.0468	−0.0415	−0.0183
	(0.0973)	(0.127)	(0.123)	(0.0978)	(0.120)	(0.0906)
Baseline mean	−1.226	−1.335	−1.121	−1.289	−1.140	−1.324
R-squared	0.055	0.067	0.043	0.058	0.062	0.057
Panel B: Impacts on stunting						
24-Month Impact	−0.00313	−0.0208	0.0148	−0.0186	0.0123	−0.0127
	(0.0261)	(0.0342)	(0.0334)	(0.0262)	(0.0305)	(0.0223)
36-Month Impact	0.0116	0.0263	−0.00323	0.00204	0.0194	0.00194
	(0.0229)	(0.0353)	(0.0280)	(0.0246)	(0.0291)	(0.0226)
48-Month Impact	0.0221	0.0154	0.0266	0.00964	0.0326	0.00466
	(0.0263)	(0.0365)	(0.0316)	(0.0280)	(0.0299)	(0.0254)
Baseline mean	0.306	0.340	0.274	0.318	0.282	0.324
R-squared	0.062	0.079	0.045	0.064	0.061	0.059
Observations	10,566	5,143	5,423	9,276	7,614	14,339

See notes to Table 5.

Why did the programme have no impact on child height and stunting? The results above can be interpreted as the reduced form or final demand for nutrition. The theoretical framework indicates that nutritional status is fundamentally determined by the nutrition production function, which in turn depends on the level and efficient application of health and nutrition inputs. In other words, for the programme to have had an impact on nutrition, it must have either affected the level of inputs in the production function or their efficient application. Consequently, to understand why the programme had no effect on child nutrition we look at programme effects on the input demands themselves as well as the health production function. Variable definitions and availability by wave are provided in Table A1.

5.2. Programme impacts on potential inputs

Tables 7–10 show programme effects on nutrition inputs that we group into three categories: the environment, food intake and health behaviours. These represent the three main pathways through which the CT may have affected child nutrition. Table 7 shows results for inputs that represent the disease environment and exposure to pathogens – note that these were collected at baseline and at 36- and 48-months only. Results show significant treatment effects on access to toilets, clean water and durable

Table 7. Impacts on environmental inputs across survey waves, Cohort sample (children 0–36 months at baseline).

	(1)	(2)	(3)	(4)	(5)
Dependent variables:	Has access to toilet facilities	Uses clean water source	Roof made of purchased material	Floor made of purchased material	Wall made of purchased material
36-Month Impact	0.105**	0.0913**	0.0262*	0.0436**	0.00393
	(0.0420)	(0.0362)	(0.0149)	(0.0124)	(0.0188)
48-Month Impact	0.0714	0.0569	0.0170	0.0225*	0.0135
	(0.0510)	(0.0372)	(0.0182)	(0.0133)	(0.0197)
Baseline mean	0.484	0.212	0.055	0.029	0.317
Observations	7,961	7,975	7,978	7,955	7,980
R-squared	0.370	0.196	0.093	0.071	0.785

See notes to Table 5. Data on household characteristics were not collected during the 24-month survey. Estimation conducted only for the households of children with valid height-for-age z-scores (HAZ) measures.

floors (with p-values below 0.05), but these are concentrated only at 36-months. Point estimates for toilet (column 1) and clean water (column 2) are sizeable at about 10 percentage points, representing increases of 22 and 43 percent over the baseline means respectively.

The programme also had significant impacts on the food security pathway; children in the CGP programme consume more meals (column 1 in Table 8) and reside in households that spend more on food per capita (column 2) than children in control household, and these effects are statistically significant at every follow-up wave. Table 9 indicates that treatment group children also consume more protein-rich food (column 2) and dairy products (column 3) than their control group counterparts (data available only from the 48-month survey).

In contrast to the effects on the previous two categories of inputs, none of the CGP impacts on the health inputs and behaviours (Table 10) are statistically significant (with the exception of one estimate in column 4).[5] Note though that many of these estimates are signed as expected – for example, treatment group children are less likely to have been sick in the two weeks prior to the surveys (column 2), presumably because their households are better able to improve diets and take preventive steps.

Results from the input demand analysis suggest that the CGP has affected the levels of some potentially important inputs. Food consumption as well as use of clean water and sanitation seems to have improved significantly as a result of the CGP. From a theoretical perspective, these would be important inputs into the production of child nutrition, making it somewhat puzzling that programme effects on child height cannot be observed. However, if the inputs analysed are complements, it might be necessary to bring about sizeable improvements across *all* vital pathways (the environment, food intake and health behaviours) to achieve final effects.

5.3. Health production function estimates

We now turn to examine which of the inputs are empirically strong determinants of HAZ. As the full set of inputs is only available at baseline and the last two survey rounds, we focus our estimates on

Table 8. Impacts on food intake across survey waves, Cohort sample (children 0–36 months at baseline).

	(1)	(2)
Dependent variables:	Meal frequency: 3 or more (0–60 months)	Log food expenditure per capita in household
24-Month Impact	0.321**	0.277**
	(0.0401)	(0.0763)
36-Month Impact	0.282**	0.159**
	(0.0445)	(0.0572)
48-Month Impact	0.190**	0.163**
	(0.0527)	(0.0697)
Baseline mean	0.231	3.125
Observations	7,349	10,564
R-squared	0.156	0.269

See notes to Table 5. Data on meal frequency were collected only for children aged 0–60 months. Estimation conducted only for children with valid height-for-age z-scores (HAZ) measures.

Table 9. Impacts on diet diversity across survey waves, Cohort sample (children 0–36 months at baseline).

	(1)	(2)	(3)
Dependent variables:	Children who receive food from 4 or more food groups	Children who receive protein rich foods	Children who receive dairy products
48-Month Impact	0.0498	0.133**	0.0973**
	(0.0408)	(0.0535)	(0.0407)
Control group mean at 48 months	0.208	0.605	0.155
Observations	851	853	854
R-squared	0.098	0.077	0.201

See notes to Table 5. Data on these outcomes were collected only at the 48-month survey for children aged 0–60 months. Estimation conducted only for children with valid height-for-age z-scores (HAZ) measures.

Table 10. Impacts on health inputs and behaviour across survey waves, Cohort sample (children 0–36 months at baseline).

Dependent variables:	(1) Household owns a mosquito net	(2) Sick during last two weeks	(3) Has health card	(4) Taken to a well-baby or under-five clinic in last six months	(5) Received vitamin A dose[a]	(6) Received 1 BCG, 3 Polio, 3 DPT and 1 measles vaccines[a,b]
24-Month Impact	0.0313	−0.00643	0.0312	−0.000577	−0.00809	0.0382
	(0.0346)	(0.0301)	(0.0361)	(0.0392)	(0.0185)	(0.0253)
36-Month Impact	0.0515	−0.0266	0.00256	0.0493		
	(0.0385)	(0.0267)	(0.0344)	(0.0414)		
48-Month Impact	0.0272	−0.00381	0.0575	0.104**		
	(0.0314)	(0.0288)	(0.0392)	(0.0493)		
Baseline/Control mean	0.805	0.282	0.845	0.846	0.879	0.855
Observations	10,565	10,531	7,602	7,524	4,833	2,379
R-squared	0.029	0.037	0.088	0.073	0.080	0.056

See notes to Table 5. Estimation conducted only for children with valid height-for-age z-scores (HAZ) measures.
[a]Data on these outcomes were collected only in the first two surveys for children aged 0–60 months.
[b]We examine this outcome using only 24-month data since by this wave all the children in the main cohort sample would have reached the age by which they should have obtained the examined vaccines.

data from these waves and pool both follow-up rounds to generate the average programme impact across the two rounds.

Column 1 of Table 11 begins with an OLS specification using only the control group so as not to contaminate the production relationship with any potential effects of CTs. These suggest a few anomalous results, notably large negative coefficients for improved walls and food expenditure. In

Table 11. Estimation of health production function – Examining height-for-age z-scores (HAZ) with child fixed effect models, Unbalanced panel sample of children (0–36 years at baseline).

Dependent variable: HAZ	(1) OLS, only control group	(2) FE, only control group	(3) FE, only treatment group	(4) FE, full sample[a]
Treatment				−0.0978
				(0.0961)
Has access to toilet facilities	0.00242	0.0325	0.0282	0.0313
	(0.0731)	(0.0638)	(0.0699)	(0.0480)
Uses clean water source	−0.101	−0.129	−0.194**	−0.173**
	(0.0814)	(0.0901)	(0.0964)	(0.0689)
Roof made of purchased material	0.148	0.0983	0.00525	0.0599
	(0.128)	(0.169)	(0.117)	(0.104)
Floor made of purchased material	0.248	−0.130	−0.108	−0.112
	(0.186)	(0.234)	(0.123)	(0.115)
Wall made of purchased material	−0.521**	−0.241	−0.184	−0.209*
	(0.106)	(0.163)	(0.131)	(0.115)
Log food expenditure per capita in household	−0.129**	−0.0628	−0.0337	−0.0481
	(0.0490)	(0.0604)	(0.0582)	(0.0423)
Owns a mosquito net	0.0213	0.00219	−0.0404	−0.0250
	(0.0698)	(0.0783)	(0.0910)	(0.0594)
Sick in last two weeks	−0.0883	−0.181**	0.0777	−0.0568
	(0.0659)	(0.0578)	(0.0810)	(0.0505)
Observations	3,068	3,068	3,066	6,134
R-squared	0.057	0.047	0.037	0.038
F-statistic of inputs (p-value)	6.24 (0.000)	1.81 (0.101)	1.92 (0.081)	1.91 (0.067)

Data used from baseline and the 36-month and 48-month survey waves. Unbalanced panel sample comprises of children who were measured in at least two of these three survey waves. Robust standard errors presented in parentheses are adjusted for clustering at the community level. ** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. All estimation models control for survey round fixed effects and child age (indicators for different age categories). Data on household characteristics were not collected during the 24-month survey. Estimation conducted only for children with valid HAZ measures.
[a]The treatment indicator = 0 for control group, = 0 for treatment group during baseline, = 1 for treatment group in follow-up waves.

addition, neither clean water nor toilet facilities show a significant relationship with child nutrition. Of course, these estimates are biased because we cannot control for all relevant factors (the omitted variable problem) and the inputs are not exogenous, but rather are choices taken by parents based on factors unobserved to the researcher, such as the child's health endowment and the general level of sanitation and cleanliness in the vicinity. Column 2 employs child-level fixed effects (FE) on the control group sample to purge the regression of time invariant sources of endogeneity. In this specification, the effects of improved walls and food become statistically insignificant, and while still negatively signed, their magnitudes are roughly halved. In addition, the coefficient on morbidity increases in absolute value and becomes statistically significant, while water and sanitation continue to have no effect on child nutrition. Note, however, that there is no programme impact on morbidity (column 2 in Table 10). Results in these two columns help us understand the lack of programme effects on child nutrition – the inputs that are significantly affected by the CGP (food consumption, floor, sanitation) do not appear statistically significant in the empirical version of the child nutrition production function, and one important variable (water) actually has a negative coefficient.

Beyond changing the levels of the inputs, the programme might also affect their efficiency due to the way they are applied or combined with other inputs. Column 3 presents FE results on the treatment group only to see if the coefficients of the inputs are different from the control group, and indeed some differences do emerge.

The effect of morbidity is not significant and in fact, it is positively signed. Clean water continues to take a negative coefficient and is now statistically significant. Column 4 pools the treatment and control groups and adds a treatment indicator which turns on for the treatment group only at follow-up rounds. These coefficients show persistent negative effects of clean water and improved walls on child nutrition.[6]

We conducted additional analyses to understand the unexpected negative effect of clean water on HAZ in Column 3. Among the treatment group, only 318 of the 1,964 follow-up observations lived in households that switched from an unclean to a clean water source after baseline; the majority of the treatment group with unclean baseline water did not switch (1,226 follow-up observations). Among this latter group, HAZ actually improved by 0.10 z-scores, while HAZ declined by 0.15 z-scores in the group switching from unclean to clean water. The majority of the switching households (those changing from unclean to clean water) reside in 14 communities in Kaputa district. This suggests that the negative effect of clean water likely represents the fact that clean water infrastructure became available in only a handful of treatment communities. We also discovered that these same communities had much higher levels of improved sanitation than communities where non-switching households reside. The non-random placement of infrastructure, plus the fact that treatment effects on water and sanitation only emerge at 36-months, could explain the negative coefficient of clean water in the production function. This underscores the point that both demand and supply-side factors play an important role in ensuring that appropriate health inputs exist that can influence child nutrition and links to the potential of integrated social protection (or cash plus) programmes.

5.4. Children born at the start of the programme and children born into the programme

In this section, we present programme impacts on HAZ and stunting for two sub-samples that could be expected to have benefitted the most from the transfers.

First, we explore impacts for the cohort sample of children born right at the inception of the programme – children aged 0–11 months at baseline. During the 24-month, 36-month and 48-month surveys, these children were between ages 24 to 35 months, 36 to 47 months, and 48 to 59 months respectively.[7] This sample would have continuously benefitted from transfers early in their life – that is, during much of the first 1,000 days window. Panel A in Table A3 looks at effects on HAZ and Panel B at stunting for this sample. There are no significant programme effects across the board. Results are unchanged if we focus instead on children aged 0–5 months at baseline.

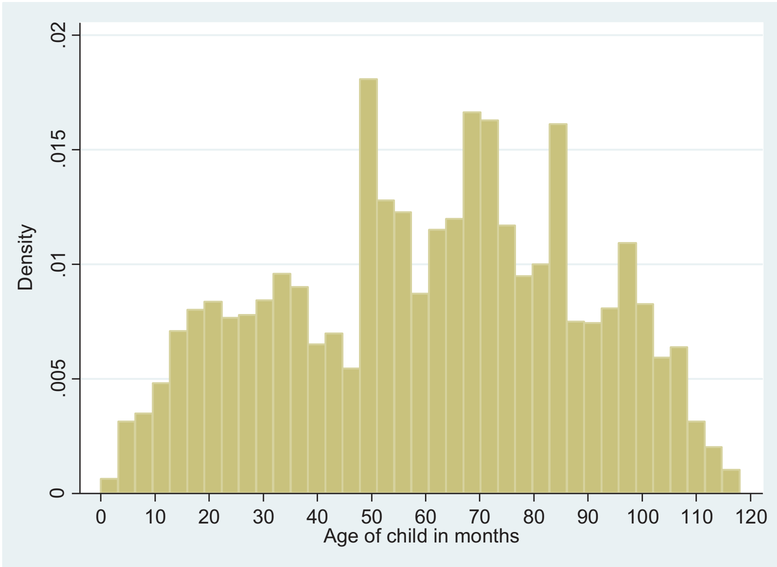

Figure 2. Age in months of children with height measured at the 48-month survey.
Observations = 5,389.

Given the length of the evaluation period and the characteristics of the sample, 1,668 children born during the period of the study were measured at the 48-month survey. Figure 2 shows the age distribution of children in our sample at 48-months. There is a break in the histogram at 48-months – children younger than 48-months would not have been alive at baseline. In principle, these children are fully treated, in the sense that their mothers were receiving cash support from the time they were in utero. This leads to the question – are there impacts on the height of these children?[8]

Table A5 shows single-difference impact estimates (between treatment and control at 48-months) on HAZ and stunting for children born into the programme (Panels A and B respectively). None of the estimated treatment effects are statistically significant. Additional pathways for the CGP to affect the nutritional status of these children is through maternal nutrition (which we did not measure) and ante- and peri-natal care and birth-weight. Table A6 reports impact estimates for these outcomes and none are statistically significant with the exception of one marginally significant positive impact on the receipt of quality antenatal care receipt (column 4), a variable that captures whether women were counselled and tested for AIDS, and given tetanus injection and malaria drugs while pregnant. These results are consistent with Handa et al. (2016) who explore the impacts of the CGP on the use of maternal health care services 24-months after the initiation of the programme and find no effects for the entire sample.

6. Discussion

We confirm the findings of several recent review articles, which conclude that there is a weak demonstrated relationship between unconditional CT programmes and child nutrition. Our in-depth study is particularly well-suited to exploring this question because the target population comprised young households with a child under age three and because the study period was four years – longer than most impact evaluations of cash transfers in SSA. In fact, close to 1,700 children were actually born during the study period, and so were exposed to the programme from the in utero period.

The CGP did affect some seemingly important intermediate outcomes, such as household food consumption and access to improved sanitation. However, these inputs do not appear to be significant in the nutrition production function. One explanation is that the realised effects were not large enough to shape child nutritional status. Alternatively, it might be necessary to bring about improvements across *all* vital pathways (the environment, food intake and health behaviours) to affect nutritional outcomes. Of course, there are other important inputs to nutrition not captured in our production function. One is caring practices, themselves a function of knowledge about nutrition. The 36- and 48-month follow-up surveys of the evaluation contained questions to gauge the health knowledge of female respondents. Specifically, women were asked to name food sources of iron and Vitamin A, strategies for treating diarrhoea, and the time when solid foods should be introduced to young children. Of all caregivers, 33 and 29 percent were unable to name a single food source of iron and vitamin A respectively. Knowledge about when to introduce solid foods and responses to diarrhoea was above 70 percent. However, the correlation in responses to these two questions across the surveys is extremely low – 0.10 and 0.07 respectively – which is consistent with the hypothesis that respondents may have simply been guessing at the responses. These data suggest that health knowledge is very low among study households. Thus, while treatment households increase food consumption after receiving the CTs, they are not necessarily making nutritional choices that might move child anthropometric measures. Overall, evidence seems to suggest that complementary inputs such as nutritional training and/or preventive health products and care may be needed.

Another important input not directly affected by the CT programme is health infrastructure and the availability of key services. We conducted a health facilities survey at baseline to understand the context under which households are making health-related decisions. Just 41 health facilities service the three study districts, of which four are dispensaries that provide drugs but not skilled care. Less than 10 percent have a protected water source and just six percent have electricity. While almost all facilities offer a well-baby clinic, actual laboratory testing is limited, with just 36 percent offering a malaria test and 16 percent providing a pregnancy test. An inventory of drugs available on the day of the interview showed that under half the facilities had oral rehydration salts, 39 percent had Fansidar and 23 percent had Cotrimoxazole. These supply-side factors are important to understanding the potential for a demand-side intervention to affect child nutrition. Indeed, to further highlight this issue, at the 48-month survey, we asked mothers about the challenges facing their children. Challenges included household-level factors (food, clothing) and external factors (availability and quality of health services and schools). Women in treatment households rated household-level factors as significantly less challenging relative to the control group – factors that can be directly resolved by the CTs. However, there were no significant differences in perceptions of challenges relating to external factors, and some of the highest rated challenges were 'availability of health services', 'drugs and medication' and 'quality of health services'. This evidence speaks directly to the health infrastructure available in these districts, which further explains the lack of effects on child nutrition.

In conclusion, results from the RCT of an unconditional CT programme targeted to households with young children show no effect on child HAZ after four years. While the intervention did affect several plausible intermediate outcomes on the causal pathway, such as food consumption, these impacts were perhaps not large enough to generate effects on nutritional status. The determinants of nutrition are complex, and include not only food but also caring practices and the disease environment. Two key complementary inputs, nutrition knowledge and health infrastructure, are very low in the study setting, and are plausible explanations for the lack of impact of this demand-side intervention. It may be the case that in other settings where the level of these complementary inputs is higher, an unconditional CT programme can deliver impacts on child nutrition. However, in a setting such as the one studied here, cash alone is not enough to address long-term chronic malnutrition, even though it might lead to substantial improvements in other dimensions of household wellbeing such as food security. Attempts are underway in many parts of the world to enhance the effectiveness of CTs on child nutrition and other outcomes by combining them with complementary interventions such as the provision of information and the improvement of access to and/or the quality of supply-side services. While the evidence-base on such 'cash plus' initiatives or integrated social cash transfer programming is still fairly limited, there are some promising results (Barry, Maïdoka, and Premand 2017; Roelen et al. 2017; Roy et al. 2017), which warrants their consideration in the effort to address child nutritional deficiencies through cash transfers in the Zambian context.

Notes

1. The CGP is more generous than most of the CTs that are evaluated by the studies included in the Manley, Gitter, and Slavchevska 2013) review and for which this information is available; of the 12 programmes with this data, only one provided transfers that were a considerably larger proportion of pre-programme income.
2. The MCDMCH was later renamed as the Ministry of Community Development and Social Services (MCDSS).
3. Those who are short for their age are classified as being stunted, and those who are light for their height are considered to be wasted (WHO 2018).
4. CGP questionnaires and reports are available on the Transfer Project website (http://www.cpc.unc.edu/projects/transfer).
5. Information on vitamin A dosage and vaccines was collected for children aged 0–60 months only during the first two surveys. It is recommended that the vaccines we examine be received within the first year of life (Rutstein and Rojas 2006). All the children in the main cohort sample would have passed this age by the 24-month survey and so we examine vaccine receipt using only 24-month data.
6. Results presented in Table 11 are unchanged when we add district-specific time trends to the estimation model to allow child HAZ in all communities in a district to experience a common linear trend.
7. Table A2 presents descriptive statistics for children born at the start of CGP.
8. Descriptive statistics for children born into the sample are provided in Table A4.

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Appendix

Table A1. Definition and availability across surveys of indicators used as outcome variables.

Domain	Indicator	Definition	Level	Survey wave			
				Baseline	24-months	36-months	48-months
Child anthropometry	Height-for-age z-score (HAZ)	The number of standard deviations a child's height is from the average age- and sex-specific height in the reference population	Child (0–5 years at baseline)	X	X	X	X
	Stunted	= 1 if HAZ<-2 = 0 otherwise	Child (0–5 years at baseline)	X	X	X	X
Environmental characteristics	Has access to toilet facilities	= 1 if the main type of toilet facility for the household is an own, community or neighbour's flush toilet or pit latrine; = 0 if it is a bucket/other container, acqua privy or if the household uses no facility	Household	X		X	X
	Uses clean water source	= 1 if the main source of water supply for the household is a protected well, borehole, tap, water kiosk or purchased from other vendors; = 0 if the household uses water from a river, lake, stream, dam, spring or unprotected well, or rainwater		X		X	X
	Roof made of purchased material	= 1 if the roof of the household dwelling is made of asbestos sheets or tiles, other tiles, iron sheets or concrete; = 0 if roof is made of grass, straw or thatch		X		X	X
	Floor made of purchased material	= 1 if the walls of the household dwelling are made of bricks or iron sheets; = 0 if floor is made of pole, dagga, mud, grass or straw		X		X	X
	Wall made of purchased material	= 1 if the floor of the household dwelling is made of concrete or wood; = 0 if walls are made of mud or bare earth		X		X	X
Food intake	Meal frequency (three or more)	= 1 if child is given solid foods three or more times a day = 0 otherwise (defined only for children who have been started on solid food)	Child (0–5 years)	X	X	X	X
	Log food expenditure per capita in household	Logarithm of total household per capita food expenditure	Household (per capita)	X	X	X	X
	Consumed food from four or more food groups	= 1 if child consumed at least four of the following food groups on the previous day: grains/roots/tubers, legumes/nuts, dairy, meats/poultry/fish, eggs, vitamin A foods, fruits/vegetables; = 0 if consumed fewer than four food groups	Child (0–5 years)				X
	Consumed protein rich foods	= 1 if child consumed protein on the previous day, that is at least one of the following foods: legumes/nuts, dairy, meats/poultry/fish, eggs; = 0 if did not consume protein					X
	Consumed dairy products	= 1 if child consumed dairy on the previous day; = 0 if did not consume dairy					X

(*Continued*)

Table A1. (Continued).

Domain	Indicator	Definition	Level	Survey wave: Baseline	24-months	36-months	48-months
Health inputs and behaviour	Household owns a mosquito net	= 1 if household owns a mosquito net; = 0 otherwise	Household		X	X	X
	Sick during last two weeks	= 1 if has been sick during the last two weeks; = 0 if has not been sick	Child		X	X	X
	Has health card	= 1 if child has a health card and it was seen by the interviewer; = 0 if child does not have a health card, or the child has a health card but it was not seen by the interviewer	Child (0–5 years)		X	X	X
	Taken to well-baby or under-five clinic in last six months	= 1 if child has been taken to a well-baby or under-five clinic for a check-up in the last six months; = 0 if child was not taken to a clinic		X	X	X	X
	Received vitamin A dose	= 1 if child has ever received a Vitamin A dose; = 0 if child has not received Vitamin A		X	X		
	Received one BCG, three Polio, three DPT and one measles vaccines	= 1 if child has received at least one dose of both BCG and measles, and at least three doses of both the oral polio vaccine and DPT; = 0 otherwise		X	X		
Birth outcomes	Sought antenatal care from doctor or nurse	= 1 if child's mother sought antenatal care for this pregnancy from a doctor or nurse; = 0 if she did not seek antenatal care, or if antenatal care was sought from other individuals: midwives, clinical officers or traditional birth attendants	Child (0–5 years)	X	X	X	X
	Month of pregnancy at which first antenatal care was received	The month of pregnancy when mother received antenatal care for the first time for this pregnancy		X	X	X	X
	Received antenatal care at least four times during pregnancy	The number of times mother received antenatal care during this pregnancy		X	X	X	X
	Received quality antenatal care	= 1 if during this pregnancy, the mother received all of the following three services: counselling and testing for AIDS, a tetanus injection, and anti-malaria drugs; = 0 otherwise		X	X	X	X
	Child born smaller than average or very small	= 1 if at birth, the child was smaller than average or very small; = 0 if the child was very large, larger than average or average		X	X	X	X
	Received assistance from doctor or nurse during delivery	= 1 if a nurse or doctor assisted with the delivery of the child; = 0 if assistance was received from other individuals: midwives, clinical officers, traditional birth attendants, relatives or friends		X	X	X	X

The baseline survey was conducted in 2010. The 24-, 36-, and 48-month surveys took place in 2012, 2013 and 2014 respectively.

Table A2. Baseline summary statistics for children born at the start of the programme (age≤11 months at baseline).

	All	Control	Treatment	P-value of diff.
Age in months	7.25	7.47	7.03	0.14
Female	0.50	0.47	0.54	0.08
Height-for-age z-score (HAZ)	−0.64	−0.61	−0.67	0.73
Stunted (% < −2 HAZ)	0.18	0.17	0.18	0.92
Household characteristics				
Household size	5.87	5.75	6.00	0.31
# members aged 0–5 years	2.11	2.11	2.11	1.00
# members aged 6–12 years	1.21	1.16	1.25	0.38
Recipient-widowed	0.05	0.04	0.05	0.50
Recipient-never married	0.13	0.15	0.12	0.36
Recipient-divorced	0.05	0.05	0.04	0.42
Recipient-highest grade	3.84	3.71	3.96	0.50
Recipient age	28.45	28.33	28.58	0.74
Potential health inputs				
Has access to toilet facilities	0.45	0.47	0.44	0.71
Uses clean water source	0.18	0.16	0.20	0.57
Roof made of purchased material	0.06	0.08	0.03	0.05
Floor made of purchased material	0.02	0.02	0.02	0.64
Wall made of purchased material	0.31	0.31	0.31	0.97
Meal frequency: 3 or more (0–60 months)	0.16	0.18	0.15	0.47
Household per capita food expenditure (ZMW)	27.70	27.46	27.95	0.87
Owns mosquito net	0.81	0.82	0.81	0.85
Sick-last 2 weeks	0.30	0.28	0.31	0.39
Child has health card	0.92	0.95	0.89	0.03
Taken to a well-baby/under 5 clinic-last 6 months	0.90	0.91	0.90	0.52
Received vitamin A dose	0.69	0.71	0.67	0.38
Received 1 BCG, 3 Polio, 3 DPT and 1 measles vaccines	0.28	0.27	0.28	0.89
Observations	572	286	286	

P-values are from Wald tests on the equality of treatment-control means. Standard errors are clustered at the community level.

Table A3. Impacts on height-for-age z-scores (HAZ) and stunting across survey waves, Children born at the start of the programme (age≤11 months at baseline), Cohort sample.

	Cohort samples		
	(1)	(2)	(3)
Dependent variable: HAZ	Total sample	Boys	Girls
Panel A: Impacts on HAZ			
24-Month Impact	0.0102	0.0272	−0.0703
	(0.183)	(0.238)	(0.223)
36-Month Impact	−0.100	−0.107	−0.147
	(0.203)	(0.267)	(0.240)
48-Month Impact	−0.0122	0.0896	−0.136
	(0.215)	(0.270)	(0.258)
Control group mean at 48-months	−0.643	−0.738	−0.550
R-squared	0.090	0.103	0.091
Panel B: Impacts on stunting			
24-Month Impact	0.00738	0.0249	0.0114
	(0.0474)	(0.0550)	(0.0622)
36-Month Impact	0.0128	0.0310	0.00142
	(0.0488)	(0.0623)	(0.0580)
48-Month Impact	0.0192	0.000977	0.0393
	(0.0453)	(0.0627)	(0.0552)
Control group mean at 48-months	0.177	0.197	0.156
R-squared	0.087	0.109	0.072
Observations	3,784	1,834	1,950

Robust standard errors presented in parentheses are adjusted for clustering at the community level. ** p < 0.01, ** p < 0.05, * p < 0.1. All estimation models include controls for child age and gender, districts, and baseline values of the following variables: log household size, respondent age, education and marital status, household demographic composition, log distance to the nearest food market and a vector of community-level prices.

Table A4. Summary statistics for children born during the programme (age≤48 months at final follow-up survey).

	All	Control	Treatment	P-value of diff.
Age in months	27.13	27.48	26.75	0.17
Female	0.48	0.46	0.50	0.12
Height-for-age z-scores (HAZ)	−1.37	−1.35	−1.38	0.76
Stunted (% < −2 HAZ)	0.36	0.36	0.36	0.98
Baseline household characteristics				
Household size	5.62	5.55	5.68	0.49
# household members aged 0-5	1.92	1.92	1.91	0.84
# household members aged 6-12	1.25	1.24	1.26	0.83
Recipient-widowed	0.05	0.05	0.05	0.87
Recipient-never married	0.09	0.09	0.08	0.66
Recipient-divorced	0.05	0.06	0.04	0.16
Recipient–highest grade	3.98	3.85	4.11	0.35
Recipient age	28.21	27.89	28.54	0.31
Birth outcomes				
Sought antenatal care from doctor or nurse	0.74	0.73	0.76	0.57
Received first antenatal care at which month of pregnancy	4.24	4.29	4.18	0.28
Received antenatal care at least four times during pregnancy	0.61	0.64	0.58	0.13
Received quality antenatal care	0.90	0.88	0.92	0.10
Child born smaller than average or very small	0.10	0.09	0.11	0.23
Received assistance from doctor or nurse during delivery	0.42	0.43	0.40	0.50
Observations	1,668	854	814	

P-values are reported from Wald tests on the equality of Treatment-Control means for each variable. Standard errors are clustered at the community level. Birth outcomes data for each child are pulled from the survey in which this information was first reported for the child.

Table A5. Impacts on height-for-age z-scores (HAZ) and stunting at 48 months, children born during the programme (age≤48 months at final follow-up survey).

	Samples		
	(1)	(2)	(3)
Dependent variable: HAZ	Total sample	Boys	Girls
Panel A: Impacts on HAZ			
48-Month Impact	−0.111	−0.119	−0.0766
	(0.0990)	(0.137)	(0.140)
Control group mean at 48 months	−1.350	−1.470	−1.209
R-squared	0.066	0.075	0.086
Panel B: Impacts on stunting			
48-Month Impact	0.0155	0.0281	−0.00173
	(0.0293)	(0.0382)	(0.0369)
Control group mean at 48 months	0.362	0.405	0.312
R-squared	0.036	0.044	0.040
Observations	1,668	867	801

Robust standard errors presented in parentheses are adjusted for clustering at the community level. ** p < 0.01, ** p < 0.05, * p < 0.1. All estimation models include controls for child age and gender, districts, and baseline values of the following variables: log household size, respondent age, education and marital status, household demographic composition, log distance to the nearest food market and a vector of community-level prices.

Table A6. Impacts on birth outcomes, children born during the programme (age≤48 months at final follow-up survey).

	(1)	(2)	(3)	(4)	(5)	(6)
Dependent variables:	Sought antenatal care from doctor or nurse	Received first antenatal care at which month of pregnancy	Received antenatal care at least four times during pregnancy	Received quality antenatal care[a]	Child born smaller than average or very small	Received assistance from doctor or nurse during delivery
Programme Impact	0.0515	−0.108	−0.0575	0.0277*	0.0179	−0.0407
	(0.0434)	(0.101)	(0.0425)	(0.0164)	(0.0189)	(0.0426)
Control group mean at 48- months	0.732	4.291	0.645	0.881	0.092	0.436
Observations	1,127	1,109	1,107	1,105	1,121	1,119
R-squared	0.089	0.068	0.065	0.055	0.037	0.131

Robust standard errors presented in parentheses are adjusted for clustering at the community level. ** $p < 0.01$, ** $p < 0.05$, * $p < 0.1$. Birth outcomes data for each child are pulled from the survey in which this information was first reported for the child. All estimation models include controls for child age and gender, districts, and baseline values of the following variables: log household size, respondent age, education and marital status, household demographic composition, log distance to the nearest food market and a vector of community-level prices. Estimation conducted only for children with valid HAZ measures.

[a]Whether women were counselled and tested for AIDS, and given tetanus injection and malaria drugs while pregnant.

June 2010
Random selection of clusters to enter study.
Selection of first 30 in each of the three districts (out of 90 overall)

June-September 2010
Targeting and selection of households in 30 clusters in each district.
From the eligibility lists, 28 households per cluster selected for the study sample.

October-November 2010: Baseline survey (2,519 households)
Treatment: 1,260 households surveyed
Control: 1,259 households surveyed

December 2010
Coin toss by Ministry to assign households to treatment or control/delayed treatment status.

February 2011
First transfer in treatment clusters

October-November 2012: 24-month follow-up (2,298 households)
Treatment: 1,153/1,260 households surveyed
Control: 1,145/1,259 households surveyed

June-July 2013 30-month follow-up (2,423 households)
Treatment: 1,179/1260 households surveyed
Control: 1,185/1259 households surveyed

October-November 2013 36-month follow-up (2,459 households)
Treatment: 1,221/1,260 households surveyed
Control: 1,238/1,259 households surveyed

September-October 2014 48-month follow-up (2,423 households)
Treatment: 1,197/1,260 households surveyed
Control: 1,226/1,259 households surveyed

Figure A1. Timeline for CGP impact evaluation.
Adapted from Natali et al. (2016).