Does information on child health risk generate positive behavior change and improve child outcomes? A Regression Discontinuity Approach from Rural India

Marion Krämer[1], Santosh Kumar[2], Sebastian Vollmer[3]

Abstract

We test whether parents adjust consumption behavior in response to negative health information of their child and whether behavioral response lead to improvement in child health and cognition in rural India. As a part of the intervention, we shared the health status of the children with the mothers and provided information on effective dietary practices for the anemic children. Using experimental data and regression discontinuity design that exploits the exogenous cutoff of hemoglobin in identifying anemia, we do not find statistically significant treatment effects on any of the children's health and cognitive outcomes. It seems that information alone, even when combined with receiving the anemia status of a child, may not be effective in changing nutritional behavior in rural parts of India.

JEL codes: I12, I15, I18, O12

Keywords: Health information, Child health, Anemia, Cognition, Regression discontinuity, India

[1]Krämer: Department of Economics, University of Goettingen, Germany (marion.kraemer@wiwi.uni-goettingen.de); [2]Kumar: Department of Economics and International Business, Sam Houston State University, Huntsville TX, USA (skumar@shsu.edu); [3]Vollmer: Department of Economics & Centre for Modern Indian Studies, University of Goettingen, Germany (svollmer@uni-goettingen.de).

Ethical clearance: The Ethics committee of the University Medical Center Göttingen approved the study. Informed written consent of the survey, including the medical tests, was taken from the parents. Acknowledgments: We thank the Foundation *fiat panis* for providing funding for this study, as well as the German Research Foundation (DFG), which provided funding within the scope of the RTG 1666.

1. Introduction

Despite the recognition of the importance of nutrition for human capital formation, i.e. health and education, undernutrition and particularly micronutrient deficiencies are still widespread in the world and even more so in low- and middle-income countries. Nutritional disorders at any life stage, but particularly in childhood, translate into impaired cognitive and physical development and result in a high-risk of low productivity. Therefore, it might constrain economic development (Dasgupta & Ray, 1986 and Strauss & Thomas 1998).

Development economists have put emphasis on developing low-cost technologies to improve health and nutrition. These technologies range from oral rehydration solutions and dissemination of deworming pills to insecticide-treated bed nets, condoms to prevent HIV infections and chlorine stations at wells to purify the water. In the field of nutrition, these technologies include nutritional supplements, micronutrient-fortified products or biofortified seeds. Innovative mechanisms to distribute these technologies to the population in need have also partly been tested. Distribution channels include public facilities such as schools, health camps or agricultural extensions. Due to the high benefits and the low costs of these health technologies, a rational agent[1] would be expected to adopt these technologies from a neoclassical point of view. Though some progress has been made, adoption of these technologies is not as high as one would expect (Dupas 2011, Banerjee & Duflo 2012).

This led researchers to the hypothesis that the supply of or access to health and nutrition technologies, i.e. availability and monetary feasibility, might not be the only constraints poor households in low-income countries are facing. There is a growing strand of literature that investigates how far the lack of information constitutes an additional constraint, which limits the demand for and the proper use of these technologies (Dupas 2011b, Karlan, Ratan & Zinman 2014). The theory being that if the information constraint could be loosened by providing individuals with the required information, they would make better health and nutritional investments.

[1] Early neoclassical models define rationality in terms of utility maximization. Individuals maximize their utility given their preferences and the constraints they are facing. It is generally assumed that individual utility maximization is exclusively based on self-interest. A rational agent performs an action, e.g. makes an investment or buys a certain good, if the marginal utility is higher than the marginal costs. An individual behaving according to this pattern is called *homo oeconomicus* (Mankiw and Taylor 2011).

In this paper, we present new evidence on the impact of informing parents about the anemia status of their child and the need to feed their child more iron-rich food items. We investigate how this intervention affects changes in feeding practice, the children's hemoglobin levels as well as the cognitive ability and education outcomes. The conveyance of this information resulted from the ethical need to inform parents about the anemia status of their child in a randomized trial and the means to eliminate the nutritional disorder if their child's hemoglobin level was below a clinical threshold. By applying a regression discontinuity design (RDD), we are able to identify the causal effect of the information intervention on the mentioned outcomes.

Anemia refers to a situation where the level of hemoglobin in the blood is low. Though anemia can have different causes,[2] iron deficiency is the most common one (WHO 2001). Iron deficiency emerges from a diet that is low in iron or when iron cannot be properly absorbed from the diet (McLean et al. 2009). Anemia not only leads to low levels of physical activity (fatigue and loss of energy), but it also impairs cognitive development and work productivity. In economic terms, iron deficiency is considered to be the *costliest* micronutrient deficiency (Halterman et al. 2001, Bobonis et al. 2006). According to Horton & Ross (2003), who used data from 10 low-income countries, physical and cognitive impairment due to iron deficiency causes a median loss of 4.05% of a country's GDP. Globally, more than 20% of the world's population (about 1.62 billion people) are anemic (WHO 2008).[3] The low-income population is at a high-risk for iron deficiency due to a lack of dietary diversity. Moreover, their diet generally includes a large amount of rice and wheat, which inhibits the absorption of iron due to the high concentrations of phytate in these products, and the low consumption of meat from which iron can more easily be absorbed (FAO and WHO 2002). Depending on age and gender, anemia in India ranges from 23% to 58% (NFHS 2015b). In Jehanabad district (state of Bihar), where our study took place, the prevalence of anemia is even higher and ranges from 26% for adult males to 63% for children below the age of 5 (NFHS 2015a).

[2] E.g. excessive bleeding, hookworm infections or malaria (WHO 2001). Since all children in the data set used for the analysis are dewormed at school once a year, we are quite sure that most of the anemia observed in our study comes from iron deficiency.

[3] For the identification of an anemic individual (WHO 2008), the authors used the age and gender specific WHO hemoglobin cutoffs of mild anemia. This is for children under 5 years: < 11.0 g/dl; children 6–11 years: < 11.5 g/dl; children 12–14 years: < 12.0 g/dl; adult males: < 13.0 g/dl; adult females (non-pregnant): < 12.0 g/dl; adult females (pregnant): < 11.0 g/dl.

Rigorous impact evaluation of health and nutrition information on health-related behavioral change are still rare. With respect to general health information, the findings are generally positive. Jalan & Somanathan (2008) found that households in a suburb of New Delhi started purifying their water after they were informed that their drinking water was contaminated. Thornton (2008) studied changes in sexual practices after individuals were informed of their HIV status and finds that HIV-positive individuals are three times more likely to purchase condoms compared to HIV-positive individuals who were not informed of their HIV status. Dupas (2011a) also reported positive effects for providing teenagers in Kenya with information on the relative risk of HIV infection according to the relationship between their partner's relative age and their sexual risk behavior. In contrast, Kremer & Miguel (2007) found no improvement in worm prevention behavior after an intensive intervention on health-education at the school level. The literature on nutrition information in the context of anemia is less encouraging than the literature on general health information presented above. In an RCT conducted by Childs et al. (1997), existing doctor-parent contacts were used to convey information about breastfeeding and the link between iron and diet to parents of newborns in the UK. At the child's age of 18 months, they did not find any effect of the intervention on the prevalence of anemia in the sample. Using a relatively small sample of about 250 newborns in Brazil, Bortolini & Vitolo (2011) found the same results. They evaluated the effect of systematic dietary home counseling. However, they found a longer duration of breastfeeding and a higher consumption of iron-rich foods in the treatment group.

While much of the literature relates to enhancing preventive healthcare, this study addresses the adoption of remedial behavior. In our treatment, the anemia status of a child is revealed by a diagnostic test, which distinguishes our study from Childs et al. (1997) and Bortolini & Vitolo (2011). The hemoglobin testing makes the disorder explicit and the need for action immediate. In contrast, with preventive healthcare interventions, there is always the hope or assumption that oneself will not be affected. Hence, the need for action might not be perceived as acute and is likely to be further limited if the required preventive actions are costly. Related to this argument, people in low-income settings typically spend a large part of their budget on remedial healthcare but the adoption of preventive healthcare is limited (Dupas 2011b).

Our study adds to the limited literature that combines nutrition information with revealing an individual's health status. Thornton (2008) studied the effect of revealing an individual's HIV status; however, she examined the adoption of health behavior that reduces the transmission of the disease rather than individual treatment. She found that individuals buy more condoms when they are diagnosed as HIV positive compared to HIV positive individuals that did not learn their HIV status, indicating that individuals change their health behavior to adapt to their health conditions after learning about their status. Cohen et al. (2015) examined the effect of purchasing anti-malaria medicine after using a rapid diagnostic test for malaria. They found that some patients did indeed respond to revealing their own health status, however, half of the patients were buying anti-malaria medicine, despite having tested negative for malaria.[4] In an RCT, Luo et al. (2012) informed the parents of Chinese elementary school children about the anemia status of their child and present strategies in addressing their child's nutritional deficiency (eating balanced meals, including iron-rich products, counseling a doctor or taking iron supplements). The information was either conveyed by letter, by a single or by multiple face-to-face information session(s). The different information interventions did not have any impact on hemoglobin levels or anemia rates.[5] Using a sample of rural Indian households, we add external validity to the existing literature. An additional innovation of this study is that we do not only assess health and nutrition outcomes, but also look at productive outcomes such as cognition and education. To our knowledge, this is also the first time that RDD was applied in the context of anemia and nutrition information.

---

[4] The case of anemia evaluated in this article is different from HIV and malaria infections, as anemia is a non-communicable disease and hence not directly transmittable. This means that the adverse effects, though being potentially strong for the individual, do not involve externalities. Malnutrition only indirectly exhibits externalities, because it weakens the immune system, which is then less able to resist communicable diseases.

[5] The revealing of the individual health status also partly relates to the debate on the effectiveness of screening. Screening is the counseling and testing for certain diseases or body disorders of a large population before the individual notices any symptoms. The purpose of screening is the early diagnosis and treatment of a disease and hence the prevention of more severe health consequences (Wilken et al. 2012). Screening is commonly done for diabetes, hypertension, tuberculosis, cancer and HIV but more so in high-income countries. It is generally directly linked to the administration of medication plus the promotion of a change in lifestyle. In our study, the intervention after screening is limited to nutrition information, i.e. a change in lifestyle. We are not aware of any studies that test the effect of screening on behavioral changes towards the adoption of health technologies and better health behavior. The screening debate focuses more on the costs (financial, health psychological) of the screening process itself.

For a similar, but somewhat simpler information intervention and a similar age group, we can confirm the findings from Luo et al. (2012) that information alone does not seem to change nutrition-related behavior, even when combined with revealing a nutritional disorder of a child. Neither the hemoglobin levels nor the cognitive and education outcomes were affected by the treatment. The latter result is little surprising since any effect on these outcomes would be based on a change in feeding practices in the first place. Tough our study might be suffering from too little statistical power, with is a drawback; this finding might indicate that other forces than the lack of information, limit people in making rational health investments. We speculate that the unavailability and inaccessibility of iron-rich food items might be one reason for not detecting a treatment effect. Comparing our findings with the existing literature on human health behavior, other social and psychological factors are also likely to play a role. We, therefore, make a case for incorporating a complex set of factors that influence human behavior, including insights from sociology and psychology, into the design of policies that aim at changing health and nutrition behavior.

The remainder of the paper is structured as follows: in section 2, we describe the treatment, the dataset and the methodological approach. In section 3, we present the empirical specification and we describe the results in section 4. In section 5, we present robustness checks and we discuss our findings and conclude in section 6.

2. Treatment, Data and Methodological Approach

2.1 Treatment

In the scope of the data collection for a randomized controlled trial (for more details see Krämer et al., 2018), second-grade school-children were tested for their hemoglobin value. The testing was performed with an on-site hemoglobin measurement device directly in the village or at the children's homes. Irrespective of the medical results, parents were informed about the hemoglobin level of their child. In cases where a child's hemoglobin value was below a defined threshold, parents, in addition, received advice about adequate treatment. Figure 2 shows the timeline of the treatment and the surveys. The hemoglobin thresholds applied are the official WHO cutoffs for moderate and severe anemia for children aged between 5 and 11 years

(WHO 2011). Following the recommendation of a pediatrician, the advice about the adequate treatment given to parents was as follows:

> **8 ≥ Hemoglobin level ≤ 10.9 g/dl (moderate anemia):**[6] Recommendation of a more diverse diet for the child, especially the consumption of green leafy vegetables and meat if the household consumes meat.
>
> **Hemoglobin level < 8 g/dl (severe anemia):** In addition to the nutritional advice, it was highly recommended to bring the child to the next healthcare facility. If the hemoglobin level was below 6 g/dl, the interviewer team ensured that the child was quickly taken to the next healthcare facility.

We found very few observations (13 children, less than 1% of the sample) with a hemoglobin score below 8 g/dl and only one child with a hemoglobin score below 6 g/dl. That is why we limit the impact analysis to the information given for a hemoglobin value ≤ 10.9 g/dl.

Informing parents about their child's anemia status and recommending dietary changes were driven by the ethical need to share with the parents any findings from our study that might indicate a direct risk to the health of a child in our sample.[7] The evaluation of this treatment can thus be considered a *quasi-experiment*.[8] [9]

2.2 The Discontinuity in Treatment Assignment

The causal identification of the effects of revealing the presence of a nutritional disorder and of giving nutritional advice is complicated in most settings due to the potential presence of selection bias or confounding. To overcome potential bias in this

---

[6] The hemoglobin measurement is accurate to one decimal place. Similarly, the WHO cutoffs are also defined to one decimal place.

[7] When conducting research with human subjects and especially with vulnerable populations, such as children, it must be ensured that the benefits of the research outweigh the risks (Medical Research Council 2004). The treatment of providing information on nutrition and recommending dietary changes was implemented to maximize the benefits of the children involved in the survey.

[8] Natural experiments are categorized as a subgroup of quasi-experiments and are defined as situations where individuals are assigned to the treatment and control groups because of a natural event. The researcher does not influence the assignment to the treatment or control groups, as is the case in a RCT, but merely functions as an observer (DiNardo 2008).

[9] For our main analysis, we include both the control and treatment groups of the RCT conducted by Krämer (2018), since we were expecting to lose too much power if we restrict our analysis to the control group alone. It might be the case that our estimates are downward biased due to the inclusion of the treatment group of the RCT. We therefore perform the analysis with the control group only in a robustness check in chapter 3.5.

study we make use of the discontinuity in the conveyance of the nutrition information and of the anemia status that is a result of the data generating process described above.

The sharp cutoff point for moderate anemia, which clearly determines if the information treatment was given, creates room for the application of a *regression discontinuity design* (RDD).[10] If the cutoff point is indeed arbitrary, was not manipulated by the entity who assigns the treatment and the forcing variable (also called *assignment* or *running* variable), i.e. the hemoglobin score in this study, was not manipulated by the target individuals – assumptions that are investigated in the next section – RDD is a valid estimation method which will causally identify the treatment effect at the cutoff points.[11] If the assumptions are fulfilled, the stochastic error component is continuously distributed over the forcing variable, such that around the thresholds the assignment to the treatment is as good as random. Observations further away from the cutoff are, however, likely to be different from each other and hence do not constitute a valid comparison group. This is because socio-economic characteristics and the general health status of children are possibly correlated with their hemoglobin level. Unhealthy and poorly nourished children in poor households are more likely to have low hemoglobin levels, whereas healthier and better-nourished children tend to be systematically better off and have higher hemoglobin levels. However, individuals close to the threshold only differ in treatment status but not in other characteristics including their underlying health. Thus, average outcomes just above the cutoff can be used as valid counterfactuals for average outcomes just below the cutoff (Lee and Lemieux 2010).

2.4 Sampling and Data

Data collection - From November 2014 until January 2015, a health survey, which included a diagnostic test for hemoglobin, was carried out among 2000 school-aged children in the two blocks Modanganj and Kako of the district Jehanabad located in the Indian state of Bihar. In 2014, we found a prevalence of anemia among second grade students of about 45%. From a list of 228 government-funded schools that exist in the two blocks, a simple-random sample of 108 schools was drawn and on average,

[10] The idea of the RDD method dates back to Thistlethwaite & Campbell (1960).
[11] The identifying assumptions have been formalized by Hahn et al. (2001).

20 children per school from the second grade were chosen for anemia testing. The sample is therefore representative of second grade students in government-funded schools in the two blocks. We specifically sampled children from the second grade because they are at the beginning of a phase of rapid brain development, since the frontal lobes experience spurts of development between the ages of 7 and 9 (Anderson 2002, Hudspeth & Pribram 1990, Thatcher 1991).

After acquiring parental consent, children were interviewed on child health and feeding practices and data on their household socioeconomic characteristics was collected, trained medical personnel performed medical checks with the children, including a diagnostic test for hemoglobin values.[12] By taking a small drop of blood from a child's finger, hemoglobin levels were assessed using an on-site hemoglobin measurement device called HemoCue® Hb 301 (AB Leo Diagnostics, Helsinborg, Sweden). Additionally, cognitive and education tests were performed with the children at the school level. From August until October 2016, i.e. about two years after the intervention, a follow-up survey was conducted that collected the same data for the same children. With this setup we can only investigate medium-term changes in response to the intervention but no immediate reactions.

Outcome variables – As direct outcomes, data on *feeding practices* were collected by a food frequency table. The information in the food frequency table is self-reported by parents. Three different indicators for feeding practices were developed from this table. First, a dietary diversity score (DDS), which was calculated by summing up the number of food groups represented in the child's diet. Similar to Torheim et al. (2003) and Kennedy et al. (2010) and based on data availability, the following food groups were included: Legumes, fruits, vegetables/green leafy vegetables, eggs, meat/poultry/fish, milk/dairy products.[13] If parents reported that their child consumes an item from one of these food groups, at least several times per month, the food group was assigned the value of one. Values for all food groups were summed up, such that the DDS ranges from zero (no item from any food group is consumed) to six (at least one item from each food group is consumed). FAO (2007)

[12] There were a negligible number of six children (less than 1%) that refused the hemoglobin test. In contrast, there was a high demand for the medical check, also from the parents of children that were not in our sample, as many households perceived the health survey as a free healthcare service.

[13] There exists no international consent on which food groups should be included in a DDS and how these food groups are defined (FAO 2007).

reviews studies that show that DDSs are valid indicators for the adequacy of micronutrient (and macronutrient) intake. Since the hemoglobin level was always measured and the nutrition information was always given after the parents were interviewed for their feeding practices, the possibility that the feeding practices were reported biasedly is minimized. Second, feeding practices are measured by the frequency of the consumption of food items that are available in the study region and that are supposed to contain a relatively high level of iron. These food items are green leafy vegetables (one indicator) and meat (second indicator). We create categorical variables for the frequency of the consumption of these two food groups that range from 1 (*the child never consumes an item from this food group or less than once per month)* to 5 (*the child consumes an item from this food group daily).*

As a more indirect outcome, *cognitive ability* was measured by five different cognitive tests (forward digit-span, backward digit-span, block design, Stroop-like day-and-night test and progressive matrices). For a detailed description of the cognitive tests, please refer to Krämer et al. (2018). These tests specifically assess *executive functions*, which are needed for purposeful goal-directed activities including inhibition, planning and organizing and working memory. Executive functions are supposed to be mediated in the frontal lobes, the brain region that, as stated above, experiences spurts of development at the age of the sampled children (Lezak 1995, Anderson 2001, Salimpoor & Desrocher 2006).

Education outcomes are also categorized as more indirect outcomes. They were assessed by math and reading tests as well as the child's school attendance (Krämer et al. 2018). Reading skills were tested on a scale from 0 to 4, ranging from *child does not recognize letters* to *child fluently reads a short story*. For the reading assessment, the materials from the Annual Status of Education Report (ASER 2014), developed by the Indian non-governmental Organization *Pratham*, were used. For the math assessment, the material from ASER (2014) was used as basis, but extended to 13 different exercises at the baseline and 15 at the endline, ranging from *child does not recognizes one-digit numbers* to *child is able to solve advanced division problems*. Finally, the school attendance of the child for the year before the follow-up survey was recorded from the official school attendance register.

To ensure similar test conditions for all children, cognitive and education assessments were performed one by one by prospective female teachers. In order to minimize disturbance, they were performed in a separate room whenever possible.

We standardized the test scores of these outcomes by subtracting the mean from the score at the baseline and dividing by the standard deviation at the baseline for the whole sample for each test. Hence, a standardized cognition score of 0.5 would mean that the student scored 0.5 standard deviations higher than the mean in 2014.

3. Empirical Framework

3.1. The regression discontinuity design (RDD)

The study uses a regression discontinuity design to estimate the causal impacts of information intervention on feeding practice, anemia, cognitive, and educational outcomes of children by comparing children below the 11.0 g/dl cutoff to those who are just above the cutoff. The anemic status is a deterministic function of hemoglobin level as children are categorized as anemic if the hemoglobin level is less than 11.0g/dl and children with more than 11.0g/dl hemoglobin level are non-anemic. The sharp threshold of 11.0g/dl hemoglobin level determines the assignment of anemic status among children, thus appropriate for the application of sharp regression discontinuity (RD) design. In the "sharp RD design", the treatment is based on a deterministic function of the cutoff. We use pooled normalized local linear regression (LLR) estimation approach with triangular kernel weights (Lee & Lemieux, 2010); Imbens & Lemieux 2008); Cattaneo et al. 2016).

The normalized pooled regression function is as follows[14]

$$\Delta Y_i = \alpha_i + \tau D_i + \beta_1(X_i - c) + \beta_2 D_i(X_i - c) + \varepsilon_i \quad \text{where } c - h \leq X \leq c + h \quad (1).$$

ΔY represents the change from 2014 to 2016 of the different outcome variables (feeding practice, hemoglobin, cognitive ability and education outcomes). We use the

[14] In a local linear regression, a straight line is fitted to the data within a predefined window with bandwidth h (i.e. locally) around the cutoff point. The choice of the window width – h – is described below. The treatment effect is modeled by a jump in the function at the cutoff point. We allow the regression function to differ at both sides of the cutoff by including an interaction term between X, the forcing variable, and *D*, the treatment dummy, but estimate both regression lines simultaneously, i.e. pooled. For convenience in the interpretation, we subtract the values of the forcing variable from the value of the cutoff point – c – (i.e. we normalize the forcing variable), thereby the treatment dummy, *D*, yields the treatment effect. We impose a triangular kernel, which gives more weight to the observations close to the cutoff.

change in the outcome to control for the initial level of the outcome variable and to increase the precision of our estimates. α_i is the intercept of the function on the right side of the cutoff. β_1 is the slope of the function on the right side and β_2 is the difference between the slopes on the left and right side of the cutoff. ε_i represents the error term. D_i is a dummy that takes on the value of one if a child's hemoglobin level was $\leq$ 10.9 g/dl in 2014 and 0 otherwise and indicates the treatment. Hence D_i is defined as

$$D_i = \begin{cases} 0 & \text{if } X_i > 10.9. \\ 1 & \text{if } X_i \leq 10.9. \end{cases}$$

τ represents the treatment effect, e.g. the size of the discontinuity at the cutoff point and hence the main coefficient of interest. We estimate equation (1) within a narrow bandwidth (h) around the cutoff point. We apply robust standard errors clustered at the school level. This method estimates the local average treatment effect around the cutoff.

The bandwidth (h) is selected use the data driven method that minimizes the mean squared error (MSE) for the local linear regression point estimator for independent and identically distributed data (Imbens & Kalyanaraman, 2012). This method was further developed by Calonico et al. (2014) and Calonico et al. (2016) for clustered data (henceforth CCT). Because our data is clustered at the school level, we apply the CCT approach in the local liner regression estimation approach. We conduct robustness checks to various bandwidths, rectangular kernel weights, and inclusion of a vector of control variables described in Table 1. Furthermore, we impose a polynomial of order two on all data points within the bandwidth selected by the CCT procedure. Finally, we show results for different order polynomials for all data point, i.e. globally and not only for a small bandwidth around the cutoff.

3.2. Validity of the regression discontinuity design

A regression discontinuity design is valid only if following assumptions are satisfied. First, the forcing variable (the hemoglobin level) should evolve smoothly across the cutoff. Second, neither the individual who assigns the treatment nor the targeted individual should be able to precisely manipulate the forcing variable (Hahn et al. 2001, Lee & Lemieux 2010, Imbens & Lemieux 2008). It appears that both assumptions are likely to be true in our setting as the cutoffs for anemia were set in terms of standard deviations from the mean of a hemoglobin distribution of a reference population (WHO 2001). This implies that a child is not exposed to a sharp health risk increase between a hemoglobin value of 11 g/dl and 10.9 g/dl.

The second assumption is that neither the individuals being studied nor the people who assign treatment (i.e. medical staff) are able to manipulate assignment to the treatment. Manipulation of assignment variable (i.e. hemoglobin level in our case) could likely bias the estimates. The cutoff level and also the knowledge about the information treatment were unknown to households and they did not have any incentive to manipulate the hemoglobin value. Furthermore, it is impossible to adjust feeding practices in a way that hemoglobin levels can be precisely determined. Since no benefits for the medical staff were involved in the conveyance of the treatment and the required effort for the communication of the nutrition information was very minimal, we also do not see any incentive for the medical staff to have manipulated a child's assignment to the treatment.

Though randomness around the threshold cannot be fully tested, there are some empirical tests that can provide suggestive evidence. First, the non-manipulation of the hemoglobin level is supported by the histogram (Figure 1). If individuals had precisely manipulated the forcing variable, one would see a discontinuity in frequencies around the cutoff (marked with a vertical red line in figure 1). We would have observed very few children with a hemoglobin level of 10.9 but many with a level of 11 g/dl. There is no such gap in frequencies in figure 1, indicating that manipulation around the cutoff is not an issue in our setting.

Second, if the treatment was indeed as good as randomly assigned around the threshold, baseline covariates should be equally distributed just above and below the cutoff (Lee and Lemieux 2010). [15] This balancing test presented in Table 1 shows that these variables are uniformly distributed around the cutoff point of 10.9g/dl. The last

[15] Hahn et al. (2001) show that continuity in the assignment variable is sufficient to obtain unbiased estimates. Therefore, the equality in means of individuals above and below the threshold is not required, however, it is likely to be the case within a small bandwidth around the cutoff point.

column of the table provides the p-values for the t-test of equality of the means, clustered at the school level. Except for the hemoglobin value, which by construction is lower below and higher above the threshold, and the share of mothers that help their child with their homework, which given the large number of t-tests might differ by chance, all means are similar and none of the other p-values of the t-test for differences in means above and below the cutoff are statistically significant, indicating randomness around the cutoff.

4. Results

4.1 Graphical Illustration

Figure 3 illustrates the potential discontinuities by plotting the change in our outcome variables from 2014 to 2016, against the normalized hemoglobin values in 2014. Due to the normalization of the forcing variable, point 0 at the x-axis is equal to a hemoglobin value of 10.9 g/dl. Section A of figure 3 shows discontinuity graphs for the feeding practice indicators (the dietary diversity score, the frequency of meat consumption and the frequency of consuming green leafy vegetables), section B for anemia outcomes (hemoglobin levels) and section C for cognitive and education outcomes (5 different cognitive tests, math and reading test scores and school attendance rate). For illustrative reasons, changes in outcomes are averaged over each discrete value of the forcing variables and plotted against the respective discrete values of the normalized hemoglobin values from 2014. To represent the density of the observations, the size of the dots in the graphs represents the number of observations within each discrete hemoglobin value. A linear regression line is fitted to the data points and the grey line shows the confidence intervals. In panel A, we show graphs for all data points (globally) and in Panel B for observations within the bandwidth that is selected by the CCT procedure.

If the information treatment were effective in improving the tested outcomes, one would see a jump at point 0 of the x-axis. For outcomes where we expect an increase due to the treatment, the regression line to the left of the cutoff would then be above the regression line to the right of the cutoff. In panel A in none of the graphs can a discontinuity at the cutoff be detected, instead all data points evolve smoothly at the cutoff, indicating that the information treatment did not affect any of the tested outcomes. When focusing on observations close to the cutoff point (Panel B), no jump can be detected for most outcomes. There might be discontinuity for some of the cognitive and education outcomes such as in the backward digit-span test, the block design test, and the cognitive index test and school attendance. Furthermore, there is quite some variability in the data in that the confidence intervals are relatively large and the observed discontinuities in cognitive outcomes are in an unexpected direction.

4.2 Main Results

Estimation results for regression (1) are presented in table 2 for feeding practices and anemia and in table 3 for cognitive and education outcomes. In Panel A, estimation results are presented for the data driven bandwidth selected by the method proposed by CCT. Panel B shows estimates for different arbitrarily chosen bandwidths (0.3, 0.5, 1.0, 1.5, 2.0 and 2.5) and Panel C for estimates with a rectangular kernel. In panel D, we include a set of control variables, and in panel E, we show results for the application of a polynomial of order two on observations with the CCT bandwidth. In Panel F, results for global estimates for different higher order polynomials are presented.[16]

The results from the discontinuity graphs can broadly be confirmed by the regression analysis. For the feeding practice outcomes (columns 1-3, table 2), none of the estimated coefficients are statistically significant and for the frequency of meat and green vegetable consumption, the coefficients display the unexpected sign. Regarding the anemia outcome (columns 4, table 2) there is a statistically significant effect of the nutrition information intervention on hemoglobin. Using CCT bandwidth, the estimate predicts that the information treatment on average led to a negative change in hemoglobin scores by the size of 0.469 g/dl (P-value: 0.034, SE: 0.218). However, the effect is only statistically significant for very small bandwidths (0.3, 0.4 and 0.5) and does not stay robust across specifications.[17] The coefficient also displays the unexpected sign.

For the cognitive measures, a few point estimates are statistically significant

[16] Regression underlying panel G are described and discussed in the robustness checks.

[17] One would expect standard errors to get larger with smaller bandwidths, as estimates get more imprecise, and coefficients might change because of the bias inherent in a larger bandwidth. This pattern is, however, not observed in table 2.

but most are not (columns 1-6, table 3). Based on the estimates using the CCT bandwidth (Panel A), the revealing of a child's anemia status and the provision of information on better feeding practices, on average, decreased the change in the *block design* test score by 0.480 standard deviations, compared to the mean in 2014 (P-value: 0.041, SE: 0.232). Since the cognitive index is a composite index of all five cognitive tests the statistically significant and qualitatively large point estimate for the *block design* tests is also reflected in a decrease of the cognitive index by 0.310 standard deviations (significant at the 10% level, P-value: 0.093, SE: 0.183). The coefficient for *block design* remains statistically significant for most specifications and the cognitive index is statistically significant only for some of the other specifications (bandwidth of 0.5, rectangular kernel, inclusion of control variables and local polynomial of 2^{nd} order). However, coefficients show an unexpected negative sign. Since estimates for the different cognitive tests are not consistent, i.e. only one cognitive test shows robust statistically significant estimates (*block design*) and the direction of the coefficients for the different cognitive tests are also not uniform; we cannot draw a general conclusion regarding cognitive ability. If anything, we find an undesirable negative effect of the nutrition information intervention. Finding an effect on cognition but not on feeding practices and hemoglobin would also be counterintuitive, as cognitive outcomes could only be affected through a change in feeding practices and an increase in hemoglobin values. For the education outcomes (columns 7-9, table 3), none of the coefficients are statistically significant and in some specifications they have the unexpected, opposite sign.

Overall, even though our treatment combines nutrition information with revealing the health risk of the child, we do not find a positive treatment effect that is robust across specifications and consistent across indicators.

5. Robustness checks

5.1 Power

As RDD estimates are generally considered more reliable within a small bandwidth around the cutoff point, many observations are normally excluded from the analysis. Furthermore, the correlation between the RDD forcing variable and the treatment status reduces the power of the estimates.

We calculate the minimal detectable effect (MDE) for different bandwidths taking the correlation between the treatment and the forcing variable into account (Table 7).[18] In the last column of table 7, we compare these MDE to those found in Luo et al. (2012) and the effect sizes found in the intervention evaluated by the RCT in Krämer, Kumar, & Vollmer (2018). The sample size of the survey was not determined to be able to conduct the RDD analysis but to be able to detect statistically significant treatment effects in the randomized trial. Power for this analysis is therefore somewhat low and the absence of statistical significance alone cannot be interpreted as evidence for a zero effect of the nutrition information. But given that effect sizes are low in general and that we find a similar picture across many different outcomes, we believe that it is still worthwhile to report these results and that it is unlikely that the nutrition information had strong effects.

To increase power, we run regression (1) without taking differences between the values in 2014 and 2016 for the outcome variables, i.e. instead of ΔY we include Y, the value of the outcome variable in 2016. The smallest MDE that can be found without taking differences in outcomes are presented in table A.3 in the appendix. Without taking differences, the MDE only get slightly smaller. Estimates without taking differences in outcomes are very similar to the estimates from the previous specification (regression output not shown).

5.2 Irregularities in the Conveyance of the Nutrition Information (Imperfect Compliance)

There are two sources of potential irregularities in the conveyance of the nutrition information, which might lead to an attenuation bias, i.e. bias estimates towards

[18] The MDE is calculated using the following formula:
$MDE = (t_{(1-\kappa)}+t_{\alpha})/(1/P(1-P)J)^{1/2} * (\rho + (1-\rho)/n\ \sigma)^{1/2} * RDDE$
where $RDDE = 1/(1-r^2)$ and r is the correlation between treatment status and the continuous assignment variable, i.e. RDDE is the RDD design effect. For a normal distribution and a position of the cutoff at 25% of the distribution – the conditions that apply to the data set used in this analysis - Schochet (2009) calculate a RDDE of 2.17 for a linear functional form. The remaining part of the formula is the standard formula for calculating MDE in RCTs. We hence multiply the MDE that could be detected in a RCT setting by the factor 2.17. We assume a power of $\kappa = 80\%$ and a significance level of $\alpha = 5\%$. Standard deviation σ, number of clusters J, fraction of treated individuals P, n the average number of individual in each cluster and ρ the intra-cluster correlation is taken from the dataset itself for observations within the respective bandwidth and for the respective outcome. We do not know the take-up of the nutrition information and we assume 100% take-up by parents of children with a hemoglobin value ≤ 10.9 g/dl and 0% take-up of the nutrition information by parents of children with a hemoglobin value > 10.9 g/dl.

zero.[19] The first potential source is that the information did not reach the parents or did not reach the person that is responsible for child feeding. We consider this risk to be rather low since the hemoglobin testing drew a lot of attention in the village and most of the times many people gathered together during the testing. Thus, if the information was not taken up directly by the parents it was very likely taken up by a neighbor, a sibling or grandparent and possibly shared with the parents later. However, we assume that there might be stronger treatment effects for the conveyance of the nutrition information directly to the mother, i.e. when it was ensured that the information reached the mother. We came up with this assumption because some empirical evidence shows that decisions on child health and nutrition are mostly made by mothers and grandmother (Thomas 2011, Thomas 1993). From another household survey that one of the authors conducted in another district of Bihar, we also know that mothers mostly make decisions about what to cook and what children eat in our study area.[20] Our dataset allows us to distinguish between observations where mothers were present during the hemoglobin testing from those where other caretakers of the children were present. We therefore run regression (1) for the subgroup of children for which we collected maternal anthropometrics. Results of this regression are shown in table 6 for the feeding practice and anemia outcomes and in table 6 for the cognitive and education outcomes. The picture looks very much the same as in the regressions before. There is no statistically significant effect for any of the specifications on feeding practices and the number of anemia symptoms. For the smaller bandwidths, hemoglobin levels appear to be statistically significant; however, the estimates are not robust at the larger bandwidths and different functional forms. Furthermore, coefficients have a negative sign. For the cognitive outcomes, some coefficients are statistically significant; however, except for the *block design* estimates, they do not seem to be robust across the different specifications and the signs of the coefficients are also not consistent across cognitive tests. Estimates for the education outcomes are insignificant across the different specifications and mostly have the expected sign. Taking this evidence together, same conclusions hold for the

[19] Before we were essentially assuming a situation of perfect compliance: Everybody who was earmarked to receive the nutrition information also received it, and every parent to whom the nutrition information was not earmarked, did not receive it. Now we are discussing the implications of this assumption and we relax the assumption to some extent.

[20] In the other household survey, it was found that in 68% of the households, females between 18-49 years of age decided what is cooked in the household (which would be mostly the mothers in our survey), and in 22% of households, grandmothers decide.

subgroup of children where mothers were present during the hemoglobin testing: We find no robust impact of revealing a child's anemia status and providing nutrition information to parents of anemic children.

Finally, as previously stated, we cannot rule out the possibility that our medical staff sometimes forgot to inform parents or that the parents of the control children took-up the treatment and subsequently changed feeding practices, when the nutrition information was addressed to parents of anemic children (in the evaluation framework those parents would be called crossovers). Even when we assume that the irregularities in the conveyance of the treatment were not systematic but occurred randomly, this circumstance would cause an attenuation bias and might hence be the reason why we do not find any significant robust treatment effect. A similar concern, with the same implication, might be that the information was not conveyed very precisely, particularly at the cutoff, i.e. even though being instructed differently it might have been the case that the medical personnel did not take the cutoff too seriously and sometimes advised parents of children with a hemoglobin of 11 g/dl to change their feeding practice or did not advice parents of children with a hemoglobin of 10.9 g/dl. During the data collection we had a very effective structure of supervision and quality control in place and believe that, if any, such cases only occurred in very few cases and thus cannot explain our findings. We further address this potential concern by performing a regression where we exclude observations directly at the cutoff (10.9 and 11 g/dl). Results of the *donut* regression are shown in the last panel (panel G) of table 4 and 5 for the main results and in table 6 for the subgroup of hemoglobin tests where the nutrition information was directly conveyed to the mother. Results are in line with the previous estimates, which further indicate that such type of imprecision was not an issue. Since it might be the case that the quality and carefulness of the medical staff differed, we test if estimates change when excluding each of the six medical staff once. Again, the results do not change much.

5.3 Attenuation Bias

As the data used for this evaluation comes from a randomized trial conducted by Krämer et al. (2018), which tested the effects of using iron-fortified iodized salt in the Indian school-feeding program (henceforth: *school intervention*), there might be another source of attenuation bias. In general, the circumstance that another nutrition intervention was evaluated with the same dataset does not bias the results since the *school intervention* was randomized at the school level and hence children that were treated by the iron-fortified salt intervention were equally distributed across the different hemoglobin values found in 2014. Table 1 shows the share of children that belonged to the treatment group in the *school intervention*, just above and below the threshold (first variable in the covariates section). To the left of the cutoff, 52% of the children were treated by the *school intervention* and to the right of the cutoff, this is true for 55% of the children, showing that belonging to the treatment group of the *school intervention* is quite balanced above and below the cutoff. Nevertheless, we included the information if the child was treated by the *school intervention* in the set of control variables for one of our specifications. The inclusion of covariates did not make much of a difference in estimation results (tables 2, 3, 5, 6 panel D). Since the *school intervention* looks at similar outcomes (hemoglobin, cognition and education) and at least the hemoglobin levels and also the math and reading scores were weakly positively affected by the *school intervention*, there might be the risk that a saturation effect had occurred. The saturation might again bias our estimates towards zero. As a robustness check to the potential presence of a saturation effect, we run regression (1) exclusively for the control group of the *school intervention*. Doing this, we lose about half of our observations and hence a lot of power, which is the reason why we did not restrict our main analysis to the control group of the *school intervention.* Results for this subsample are shown in table A.1 in the appendix for the feeding practice and anemia outcomes and in table A.2 for the cognition and education outcomes. The pattern of the estimates remains similar to the full sample; however, the coefficients for hemoglobin are not statistically significant for any of the specifications, whereas the coefficients for the *backward digit-span* and the *day and night tests* turn out to be statistically significant. Again, the estimates for none of the outcomes are robust across specifications and they often have the unexpected sign. These estimates indicate that an attenuation bias due to saturation does not explain our null finding.

6. Discussion

In this article, we report results from a RDD analysis, studying the impact of revealing the anemia status of school-aged children and advising parents to feed their child iron-rich food items in rural India. As outcome variables, we looked at feeding practices, hemoglobin levels, cognitive ability and education outcomes. At least for the medium term (two years after the intervention), we did not find any robust impact on any of the measured outcomes. The non-detection of any treatment effect for many of the outcomes is in line with the findings from Luo et al. (2012), who also did not find any effect of a very similar intervention in China. It is, however, in contrast to other health information treatments that also included the revealing of individual health status and where individuals – at least partly – reacted to the treatment (Thornton 2008, Cohen et al. 2015).

One potential reason for our null finding might be that positive treatment effects, if any, only occurred immediately and had diminished after two years. Another explanation might be that parents were not able to adjust nutritional behavior due to unavailability, accessibility or affordability of iron-rich food items. We also discussed limitations of our study with respect to statistical power.

In the introduction, we provided motivation for the nutrition information intervention by pointing out that the availability of commodities or technologies is not the only requirement for their adoption (supply side interventions), but that also the demand for these technologies must be created by nutrition information. The opposite is obviously equally true: For the information intervention to materialize in a change in nutrition behavior, the availability and the (financial) accessibility of the technology (in our case, the availability of iron-rich food products) are also required. The WHO (2001) states that improvements in nutrition build on these three pillars: the availability of micronutrient-rich food, the financial accessibility to those food items as well as a change in feeding practices (i.e. utilization). However, the tested intervention only addresses the latter pillar. In the follow up survey in 2016, we included two questions that give an idea about the availability and financial accessibility of iron-rich food items. In the follow-up survey, nearly 86% of parents that were given the nutrition information reported that they were often or sometimes not able to afford feeding their child a balanced meal and 77% reported that they were often or sometimes not able to feed their child a balanced meal, because only a limited variety of food was available in their surroundings. Both answers suggest that both availability and accessibility might have constrained parents from providing a more diverse diet for their children, even though they would have been willing to do so.

Evidence from other studies also supports the notion that even when availability and accessibility is ensured, people do not necessarily respond to health information or at least not as much as one would expect. E.g. children who were educated about the adverse effects of worm infections and means to prevent infection did not adapt their health behavior (i.e. wear shoes and adopt more hygienic behavior), even though adoption should have been cheap and feasible (Kremer and Miguel 2007). Fernandes et al. (2014) conduct a meta-analysis on financial education and find that financial information explains very little of the savings and borrowing behavior, despite the existence of financial services. In the context of nutrition, Banerjee et al. (2015) found that making iron-fortified iodized salt (DFS) available in Indian villages and informing households with a flyer of the product's availability and its benefits, did not encourage take-up. In contrast, an education movie had a positive impact on adoption of the fortified salt compared to the control group; however, the overall take-up was only about 10%. Furthermore, Childs et al. (1997) argue that parents are confronted with many different views, information and ideas on how a child should be fed. They get advice from their extended family, their community, advertisements and the media. The nutrition information given in the scope of this study must therefore be seen as a complex set of information that parents encounter in their decision-making process.

Childs et al. (1997), as well as Banerjee & Duflo (2012), also argue that beliefs, social norms, habits and culture play a crucial role when people make decisions about food intake. In case new information contrasts with deep-seated beliefs and habits they will hardly be taken-up. Insights from psychology, also point to certain mental tendencies that prevent human beings from converting their intentions into actions, including limited attention[21] and present bias (Laibson 1997,

[21] Limited attention refers to the idea that given the wealth of information a human being is exposed to, one can hardly consider all of the dimensions and options (Mullainathan & Sharif, 2013).

O'Donoghue & Rabin 1999, Banerjee & Mullainathan 2010, Dupas 2011, Karlan et al. 2006). The present bias, i.e. the fact that individuals give stronger weight to rewards and discomforts that are in the present, might also play a crucial role in explaining why revealing that a child is moderately anemic is less effective compared to revealing the presence of other diseases (HIV, malaria). While the degree of suffering from a nutritional disorder might not be perceived as very severe, mostly because the cause and symptoms are rather salient, the costs of changing nutritional habits are high. Moreover, the benefits of a nutritional change need time to materialize and occur in the future.

Given what we already know about what limits and what motivates human behavior from a neoclassical perspective, as well as from insights from sociology and psychology, it becomes clear that any policy or intervention that aims at changing nutritional behavior needs to consider a complex array of different factors[22]. Certainly

> *"…we should recognize – indeed assume – that information alone will not do the trick. This is just how things are, for the poor, as for us…"* (Banerjee & Duflo 2012, p. 70).

This seems to be true even when the presence of a nutritional disorder is revealed.

[22] Some innovative interventions have been developed and successfully tested in the field. They range from commitment opportunities, nudging (i.e. small incentives), reminders to enforced mandatory policies (Dupas 2011b).

5. References

Anderson, Peter. 2002. "Assessment and Development of Executive Function (EF) During Childhood." *Child Neuropsychology* 8 (2): 71–82. doi:10.1076/chin.8.2.71.8724.

Anderson, Vicki. 2001. "Assessing Executive Functions in Children: Biological, Psychological, and Developmental Considerations." *Pediatric Rehabilitation* 4 (3): 119–36. doi:10.1080/13638490110091347.

ASER. 2014. "Aser Tools & Testing." http://www.asercentre.org.

Banerjee, Abhijit, Esther Duflo, and Sharon Barnhardt. 2015. "Movies, Margins and Marketing: Encouraging the Adoption of Iron-Fortified Salt." *NBER Working Paper Series*, 26. doi:10.3386/w21616.

Banerjee, Abhijit, and Sendhil Mullainathan. 2010. "The Shape of Temptation: Implications for the Economic Lives of the Poor." *National Bureau of Economic Research Working Paper Series* 0 (1990): 15973+. doi:10.2139/ssrn.1598547.

Banerjee, Abhijit V., and Esther Duflo. 2012. "Poor Economics." *Poor Economics*, 303. doi:10.1007/s13398-014-0173-7.2.

Bobonis, Gustavo J, Edward Miguel, and Charu Puri-Sharma. 2006. "Anemia and School Participation." *The Journal of Human Resources* 41 (4): 692–721.

Bortolini, Gisele Ane, and Márcia Regina Vitolo. 2011. "The Impact of Systematic Dietary Counseling during the First Year of Life on Prevalence Rates of Anemia and Iron Deficiency at 12-16 Months." *Jornal de Pediatria* 88 (1): 33–39. doi:10.2223/JPED.2156.

Calonico, S., M. D. Cattaneo, M. H. Farrell, and R Titiunik. 2016. "Regression Discon- Tinuity Designs Using Covariates." *Working Paper. University of Michigan.*

Calonico, Sebastian, Matias D. Cattaneo, and Rocio Titiunik. 2014. "Robust Nonparametric Confidence Intervals for Regression-Discontinuity Designs." *Econometrica* 82 (6): 2295–2326. doi:10.3982/ECTA11757.

Cattaneo, Matias D, Rocío Titiunik, and Gonzalo Vazquez-Bare. 2016. "Comparing Inference Approaches for RD Designs: A Reexamination of the Effect of Head Start on Child Mortality."

Cattaneo, Matias D, and Gonzalo Vazquez-Bare. 2016. "The Choice of Neighborhood in Regression Discontinuity Designs." *Observational Studies* 2: 134–46.

Childs, F, A Aukett, P Darbyshire, S Ilett, and L N Livera. 1997. "Dietary Education and Iron Deficiency Anaemia in the Inner City." *Archives of Disease in Childhood* 76 (2): 144–47. doi:10.1136/adc.76.2.144.

Cohen, Jessica, Pascaline Dupas, and Simone Schaner. 2015. "Price Subsidies, Diagnostic Tests, and Targeting of Malaria Treatment: Evidence from a Randomized Controlled Trial." *American Economic Review* 105 (2): 609–45. doi:10.1257/aer.20130267.

DiNardo, John. 2008. "Natural Experiments and Quasi-Natural Experiments." In *New Palgrave Dictionary of Economics*, 1–16. doi:10.1057/b.9780631218234.2008.X.

Dupas, Pascaline. 2011a. "Do Teenagers Respond to HIV Risk Information? Evidence from a Field Experiment in Kenya." *American Economic Journal: Applied Economics* 3 (1): 1–34. doi:10.1257/app.3.1.1.

———. 2011b. "Health Behavior in Developing Countries." *Annual Review of Economics* 3 (1): 425–49. doi:10.1146/annurev-economics-111809-125029.

FAO. 2007. "Nutrition and Consumer Protection Division. Guidelines for Measuring

Household and Individual Dietary Diversity.” Rome. http://www.fao.org/3/a-i1983e.pdf.
FAO, and WHO. 2002. “Human Vitamin and Mineral Requirements. Report of a Joint FAO/WHO Expert Consultation, Bangkok, Thailand.” Rome. http://www.fao.org/3/a-y2809e.pdf.
Fernandes, D., Lynch Jr, J. G., & Netemeyer, R. G. 2014. “Financial Literacy, Financial Education, and Downstream Financial Behaviors.” .*Management Science* 60(8): 1861–83. doi:10.1287.
Hahn, Jinyong, Petra Todd, and Wilbert Klaauw. 2001. “Identification and Estimation of Treatment Effects with a Regression-Discontinuity Design.” *Econometrica* 69 (1): 201–9. doi:10.1111/1468-0262.00183.
Halterman, Jill S, Jeffrey M Kaczorowski, C Andrew Aligne, Peggy Auinger, and Peter G Szilagyi. 2001. “Iron Deficiency and Cognitive Achievement among School-Aged Children and Adolescents in the United States.” *Pediatrics* 107 (6): 1381–86. http://www.ncbi.nlm.nih.gov/pubmed/11389261.
Horton, Susan, and J. Ross. 2003. “The Economics of Iron Deficiency.” *Food Policy* 28 (1): 51–75. doi:10.1016/S0306-9192(02)00070-2.
Hudspeth, William J., and Karl H. Pribram. 1990. “Stages of Brain and Cognitive Maturation.” *Journal of Educational Psychology* 82 (4): 881–84. doi:10.1037/0022-0663.82.4.881.
Imbens, Guido W, and Karthik Kalyanaraman. 2012. “Optimal Bandwidth Choice for the Regression Discontinuity Estimator.” *The Review of Economic Studies* 79 (3): 933–59. doi:10.1093/restud/rdr043.
Imbens, Guido W, and Thomas Lemieux. 2008. “Regression Discontinuity Designs: A Guide to Practice.” *Journal of Econometrics* 142: 615–35.
Jalan, Jyotsna, and E Somanathan. 2008. “The Importance of Being Informed: Experimental Evidence on Demand for Environmental Quality.” *Journal of Development Economics* 87 (1): 14–28.
Karlan, Dean, Nava Ashaf, Wesley Yin, and Nava Ashraf. 2006. “Tying Odysseus to the Mast: Evidence from a Commitment Savings Product in the Philippines.” *The Quarterly Journal of Economics* 121 (2): 635–72. doi:10.1162/qjec.2006.121.2.635.
Karlan, Dean, Aishwarya Lakshmi Ratan, and Jonathan Zinman. 2014. “Savings by and for the Poor: A Research Review and Agenda.” *Review of Income and Wealth* 60 (1): 36–78. doi:10.1111/roiw.12101.
Kennedy, Gina, Maylis Razes, Terri Ballard, and Marie Claude Dop. 2010. “Measurement of Dietary Diversity for Monitoring the Impact of Food Based Approaches 1 , 2 Gina Kennedy, Maylis Razes, Terri Ballard and Marie Claude Dop.” *International Symposium on Food and Nutrition Security: Food-Based Approaches for Improving Diets and Raising Levels of Nutrition*.
Krämer, Marion, Santosh Kumar, and Sebastian Vollmer. 2018. “"Improving Child Health
and Cognition: Evidence from a School-Based Nutrition Intervention in India," Courant
Research Centre: Poverty, Equity and Growth - Discussion Papers 247, University of
Goettingen, Gottingen, Germany.
Kremer, Michael, and Edward Miguel. 2007. “The Illusion of Sustainability.” *The Quarterly Journal of Economics* 122 (3): 1007–65. doi:10.1162/qjec.122.3.1007.
Laibson, David. 1997. “Golden Eggs and Hyperbolic Discounting.” *Quarterly*

Journal of Economics 112 (2): 443–77. doi:10.1162/003355397555253.
Lee, David S, and Thomas Lemieux. 2010. “Regression Discontinuity Designs in Economics.” *Journal of Economic Literature* 48 (2): 281–355. doi:10.1257/jel.48.2.281.
Lezak, Muriel Deutsch. 1995. *Neuropsychological Assessment*. 3rd ed. New York: Oxford University Press.
Ludwig, Jens, and Douglas L. Miller. 2007. “Does Head Start Improve Children’s Life Chances? Evidence from a Regression Discontinuity Design.” *The Quarterly Journal of Economics* 122 (1): 159–208. doi:10.1162/qjec.122.1.159.
Luo, Renfu, Yaojiang Shi, Linxiu Zhang, Huiping Zhang, Grant Miller, Alexis Medina, and Scott Rozelle. 2012. “The Limits of Health and Nutrition Education: Evidence from Three Randomized-Controlled Trials in Rural China.” *CESifo Economic Studies* 58 (2): 385–404. doi:10.1093/cesifo/ifs023.
Mankiw, N Gregory, and Mark P. Taylor. 2011. *Principles of Economics, Second Edition*. *Book*. Vol. 328. doi:10.1017/CBO9780511511455.
McLean, Erin, Mary Cogswell, Ines Egli, Daniel Wojdyla, and Bruno de Benoist. 2009. “Worldwide Prevalence of Anaemia, WHO Vitamin and Mineral Nutrition Information System, 1993-2005.” *Public Health Nutrition* 12 (4): 444–54. doi:10.1017/S1368980008002401.
Medical Research Council. 2004. “MRC Ethics Guide: Medical Research Involving Children.” London. https://www.mrc.ac.uk/documents/pdf/medical-research-involving-children/.
Mullainathan, Sendhil, and Eldar Sharif. 2013. *Scarcity: Why Having Too Little Means So Much*. Picador.
NFHS. 2015a. “National Family Health Survey 4. District Fact Sheet Jehanabad Bihar.” Mumbai.
———. 2015b. “National Family Health Survey 4. India Fact Sheet.” Mumbai.
O’Donoghue, Ted, and Matthew Rabin. 1999. “Doing It Now or Later.” *American Economic Review* 89 (1): 103–24. doi:10.2307/116981.
Ray, Debraj. 2007. “Development Economics.” *New Palgrave Dictionary of Economics*, no. March: 1–31. doi:10.1057/9780230226203.0385.
Rippmann, Christian E, Philipp C Nett, Draguljub Popovic, Burkhardt Seifert, Thomas Pasch, and Donat R Spahn. 1997. “Hemocue, an Accurate Bedside Method of Hemoglobin Measurement?” *Journal of Clinical Monitoring* 13 (6): 373–77. http://www.ncbi.nlm.nih.gov/pubmed/9495289.
Salimpoor, Valorie N, and Mary Desrocher. 2006. “Increasing the Utility of EF Assessment of Executive Function in Children.” *Developmental Disabilities Bulletin* 34 (1-2): 15–42.
Strauss, John, and Duncan Thomas. 1998. “Health, Nutrition, and Economic Development.” *Journal of Economic Literature* 36 (2): 766–817. doi:http://www.aeaweb.org/jel/index.php.
Thatcher, Robert W. 1991. “Maturation of the Human Frontal Lobes: Physiological Evidence for Staging.” *Developmental Neuropsychology* 7 (3): 397–419. doi:10.1080/87565649109540500.
Thistlethwaite, Donald L, and Donald T Campbell. 1960. “Regression-Discontinuity Analysis: An Alternative to the Ex Post Facto Experiment.” *Journal of Educational Psychology* 51 (6): 309–17. doi:10.1037/h0044319.
Thomas, Duncan. 1993. “The Distribution of Income and Expenditure within the Household.” *Annales d’Économie et de Statistique*, no. 29: 109–35. doi:10.2307/20075898.

———. 2011. “Intra-Household Resource Allocation: An Inferential Approach.” *Journal of Human Resources* 25 (4): 635–64. doi:10.2307/145670.

Thornton, Rebecca L. 2008. “The Demand for Learning HIV Status and the Impact on Sexual Behavior: Evidence from a Field Experiment.” *American Economic Review* 98 (5): 1829–63. doi:10.1257/aer.98.5.1829.

Torheim, Liv Elin, Ingrid Barikmo, Christine L Parr, Anne Hatløy, F Ouattara, and Arne Oshaug. 2003. “Validation of Food Variety as an Indicator of Diet Quality Assessed with a Food Frequency Questionnaire for Western Mali.” *European Journal of Clinical Nutrition* 57 (10): 1283–91. doi:10.1038/sj.ejcn.1601686.

WHO. 1959. “Iron Deficiency Aneamia. Report of a Study Group.” Geneva. http://apps.who.int/iris/bitstream/10665/40447/1/WHO_TRS_182.pdf?ua=1&ua=1.

———. 2001. “Iron Deficiency Anaemia: Assessment, Prevention and Control.” Geneva. http://www.who.int/nutrition/publications/micronutrients/anaemia_iron_deficiency/WHO_NHD_01.3/en/index.html.

———. 2008. “Worldwide Prevalence of Anaemia 1993–2005. WHO Global Database on Anaemia.” Geneva. http://apps.who.int/iris/bitstream/10665/43894/1/9789241596657_eng.pdf.

———. 2011. “Haemoglobin Concentrations for the Diagnosis of Anaemia and Assessment of Severity.” Geneva. http://www.who.int/vmnis/indicators/haemoglobin.pdf.

Wilken, Dennis, Xaver Baur, Lioubov Barbinova, Alexandra Preisser, Evert Meijer, Jos Rooyackers, and Dick Heederik. 2012. “What Are the Benefits of Medical Screening and Surveillance?” *European Respiratory Review*. doi:10.1183/09059180.00005011.

Figure 1: Distribution of baseline hemoglobin values

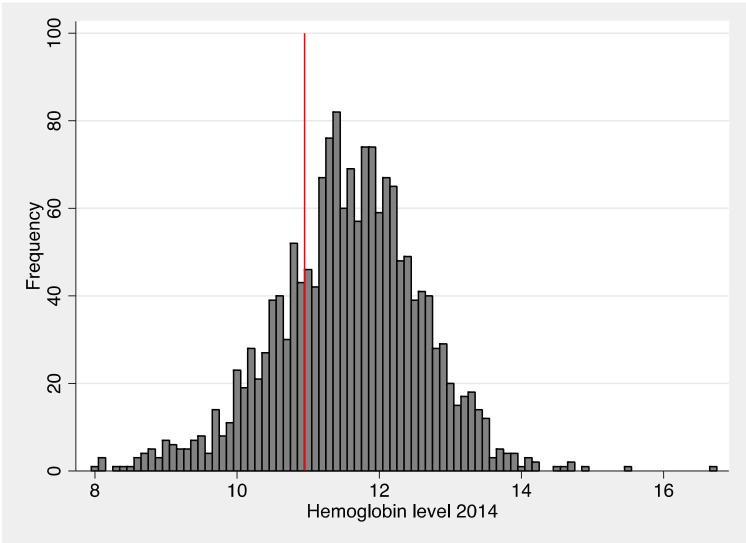


Figure 2: Timeline of data collection and the tretament

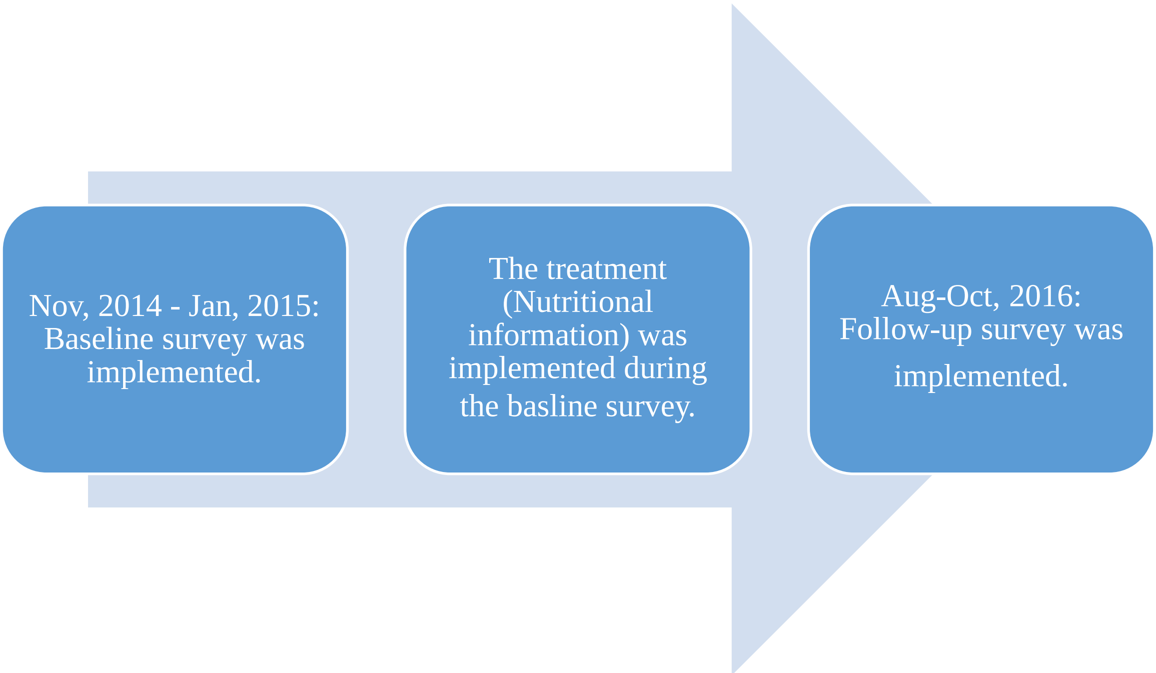
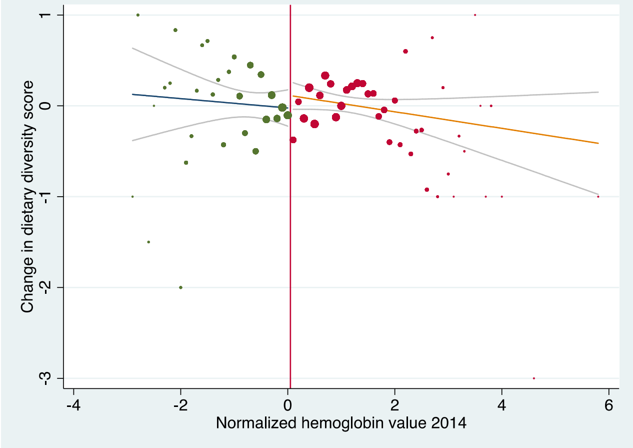
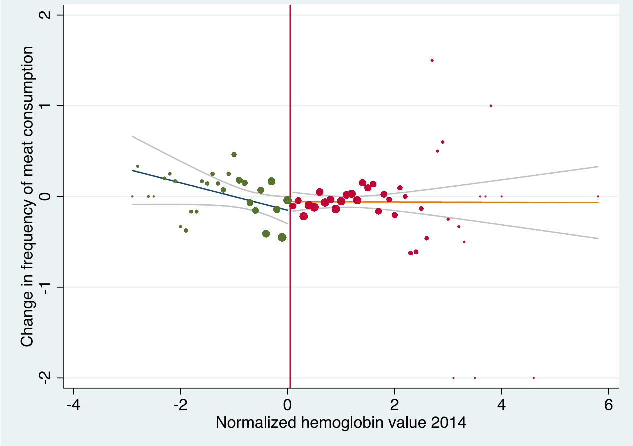


Figure 3: Discontinuity graphs

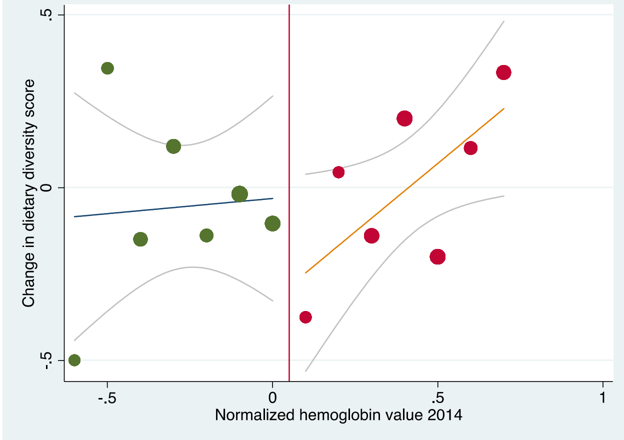
A. Feeding practices

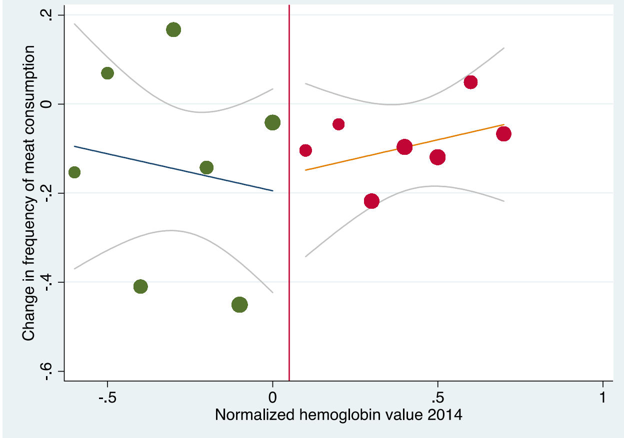
Panel A: Globally

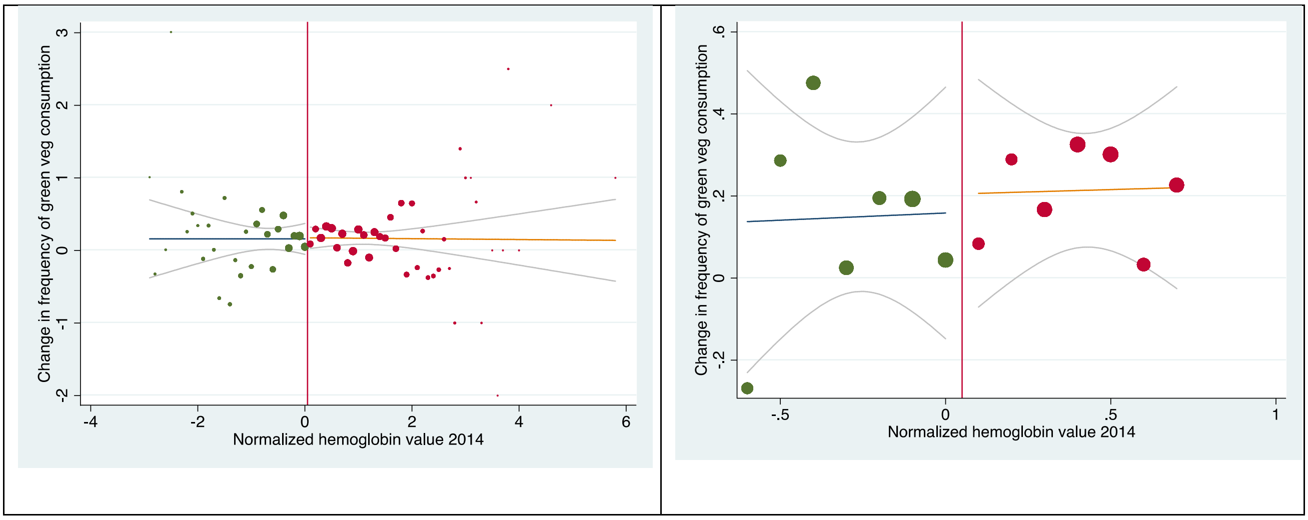




Panel B: Close to the cutoff

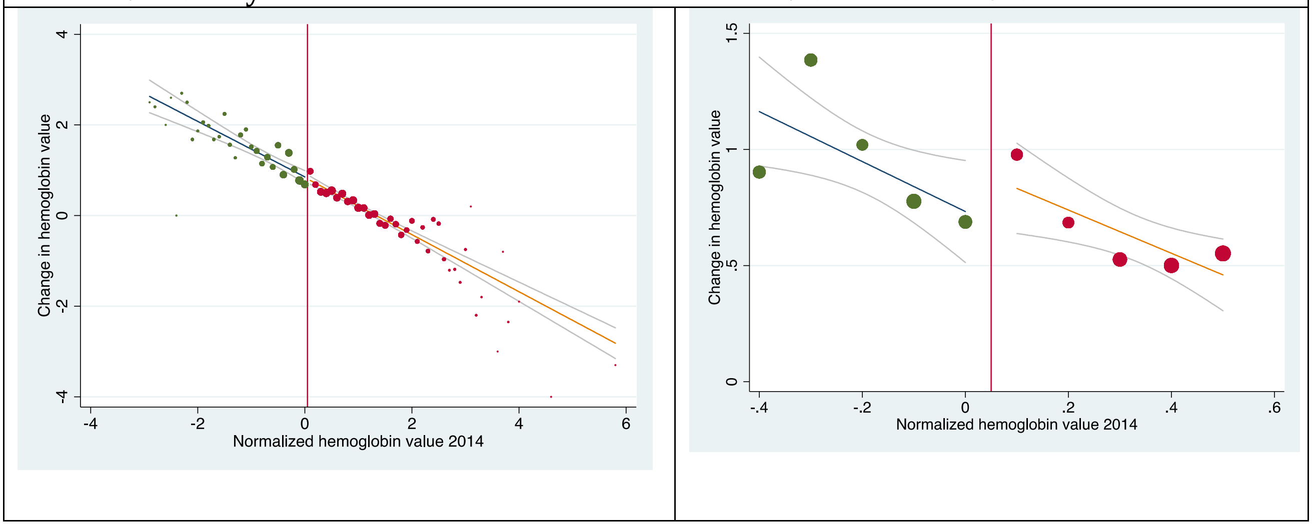






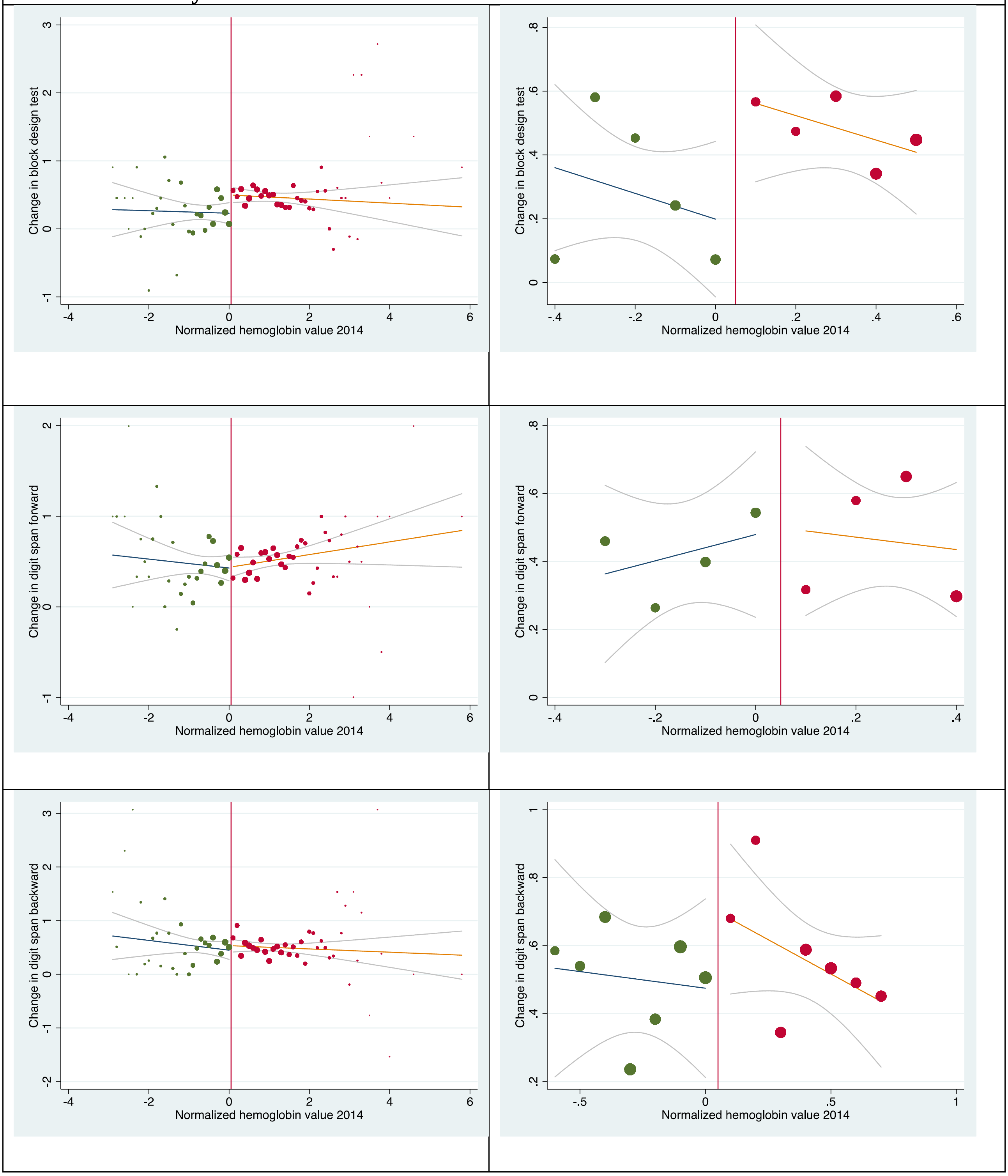
B. Anemia outcomes

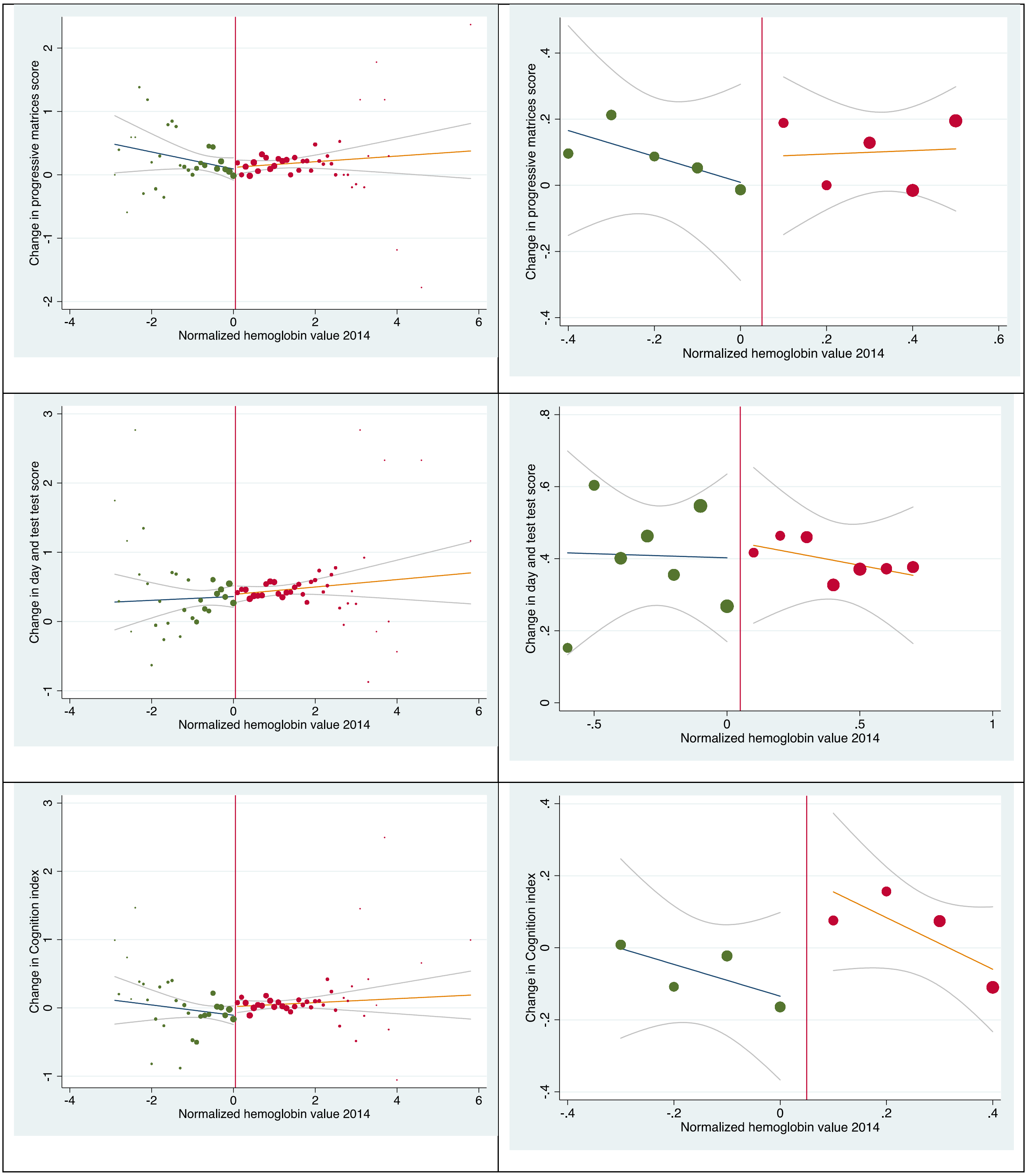
Panel A: Globally **Panel B: Close to the cutoff**



C. Cognitive and education outcomes

Panel A: Globally	**Panel B: Close to the cutoff**




Change in progressive matrices score
Normalized hemoglobin value 2014
Change in progressive matrices score
Normalized hemoglobin value 2014
Change in day and test test score
Normalized hemoglobin value 2014
Change in day and test test score
Normalized hemoglobin value 2014
Change in Cognition index
Normalized hemoglobin value 2014
Change in Cognition index
Normalized hemoglobin value 2014

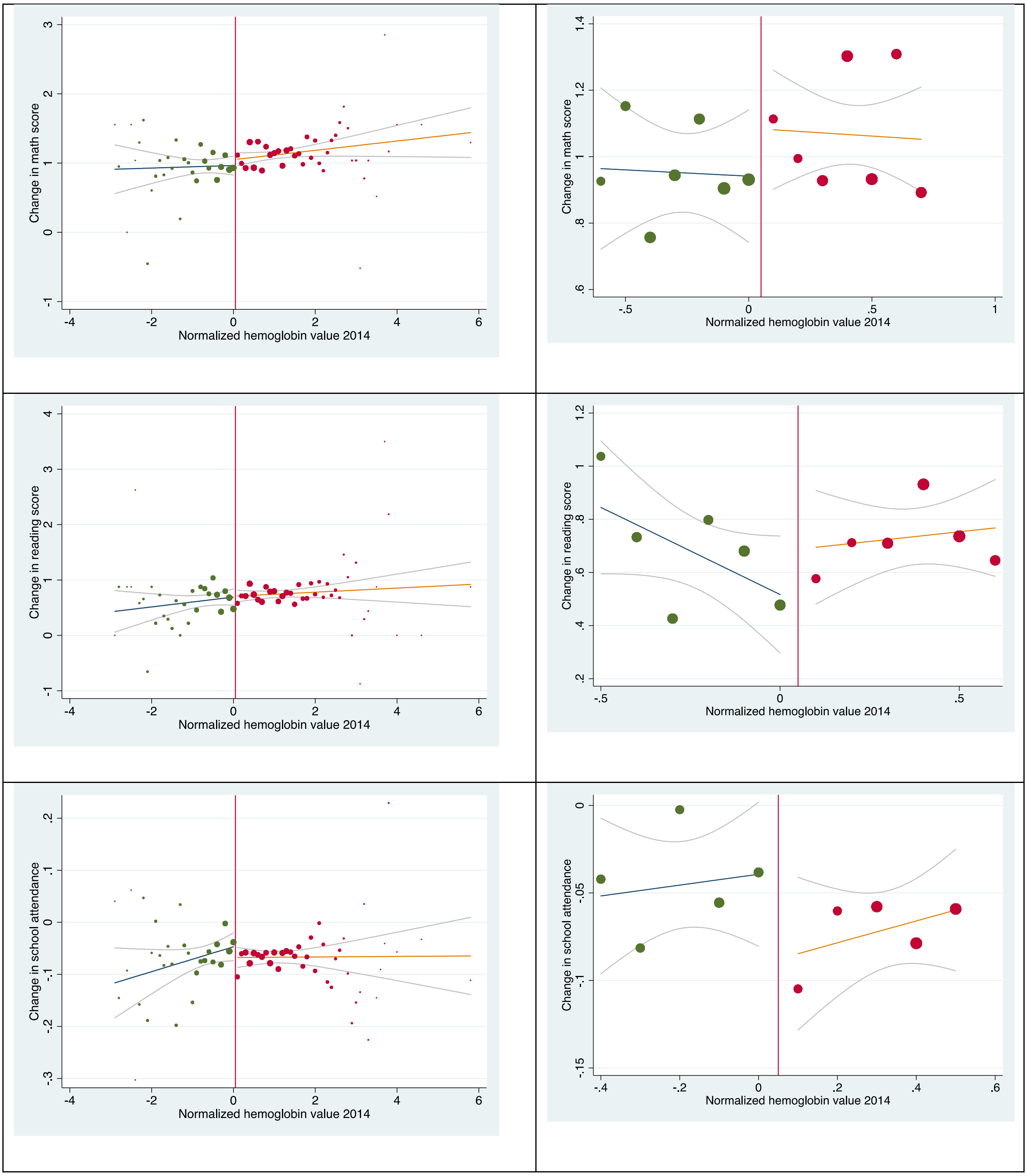

Change in math score
Normalized hemoglobin value 2014
Change in math score
Normalized hemoglobin value 2014
Change in reading score
Normalized hemoglobin value 2014
Change in reading score
Normalized hemoglobin value 2014
Change in school attendance
Normalized hemoglobin value 2014
Change in school attendance
Normalized hemoglobin value 2014

Table 1: Balancing table (Hemoglobin sample)

	Left side 10.5-10.9 g/dl hemoglobin			Right side 11.0-11.4 g/dl hemoglobin			
	(1) Mean	SD	N	(2) Mean	SD	N	(3) P-value
Feeding practices							
Dietary Diversity Score	3.80	1.20	204	3.88	1.17	313	0.446
Frequency of meat consumption	1.83	0.91	203	1.77	0.81	313	0.393
Frequency of green veg consumption	3.38	1.00	200	3.44	1.06	309	0.558
Hemoglobin							
Hemoglobin	10.71	0.14	204	11.23	0.14	313	0.000***
Number of anemia symptoms	1.01	1.05	204	1.03	1.09	313	0.858
Cognition							
Block design	3.76	2.21	199	3.46	2.19	307	0.133
Digit span forward	3.98	1.00	200	4.01	0.95	307	0.666
Digit span backward	0.96	1.29	200	0.98	1.22	307	0.879
Progressive matrices	4.87	1.73	200	4.84	1.46	307	0.881
Day and night	5.04	3.28	200	5.09	3.33	307	0.865
Cognitive index	-0.07	0.96	199	-0.10	0.92	307	0.721
Education							
Math	4.30	3.78	200	4.38	3.57	307	0.807
Reading	0.73	0.99	200	0.75	1.04	307	0.809
School attendance	0.78	0.16	195	0.80	0.16	295	0.196
Covariates							
Treatment group from school intervention	0.52	0.50	204	0.55	0.50	313	0.552
Muslim HH	0.03	0.17	204	0.02	0.14	313	0.451
Sc/st	0.32	0.47	204	0.31	0.46	313	0.804
Block	0.71	0.45	204	0.66	0.47	313	0.270
Rural HH	0.98	0.16	204	0.98	0.13	313	0.492
N of HH members	7.39	3.04	204	7.53	3.22	313	0.618
Years schooling father	4.84	4.86	201	5.13	4.73	307	0.510
Years schooling mother	1.44	2.89	203	1.46	2.93	311	0.940
Asset index	-0.15	0.97	200	-0.10	0.89	309	0.558
Institutional delivery	0.37	0.48	201	0.33	0.47	313	0.272
Health insurance	0.42	0.49	202	0.35	0.48	310	0.143
Diarrhea	0.04	0.21	204	0.05	0.21	313	0.841
Improved sanitation	0.07	0.26	204	0.07	0.26	313	0.998
Male child	0.40	0.49	204	0.45	0.50	313	0.260
Help with homework	0.10	0.30	201	0.19	0.39	311	0.006***
Time physical care	47.25	28.92	204	44.83	22.62	313	0.287
School meetings	0.66	0.48	203	0.65	0.48	313	0.878
Father at home	0.88	0.33	203	0.89	0.31	313	0.613
Distance to school	10.03	6.28	204	10.33	6.02	313	0.583
Number of meals	3.09	0.92	204	3.07	1.10	313	0.874
Cut meals	0.82	0.38	204	0.82	0.38	313	0.944
Iron supplementation	0.16	0.37	201	0.21	0.41	309	0.151
Maternal health knowledge	0.36	0.48	204	0.37	0.48	313	0.914
Total enrollment	252.83	153.70	204	262.88	153.56	313	0.467
Class size	32.62	16.37	204	34.26	17.50	313	0.286
Student teacher ratio	38.89	11.76	204	38.46	11.52	313	0.679
Calories of MDM per child	66.96	20.15	204	69.05	22.85	313	0.288
Iron in MDM per child	0.75	0.29	204	0.78	0.30	313	0.297

Notes: This table presents baseline summary statistics as well as p-values for difference in means t-tests between children just above and just below the cutoff of 10.9 g/dl. All variables shown are child level variables from the baseline. Standard errors are clustered at the school level. SD: Standard deviation, N: Number of observations, MDM: Midday Meal.

Table 2: The average treatment effect of nutrition information on child health and feeding practices (different bandwidths)

	Dietary diversity score	Frequency of meat consumption	Frequency of green veg consumption	Hemoglobin
	(1)	(2)	(3)	(4)
A. Main results				
Optimal Bandwidth (CCT)	0.229	-0.062	-0.052	-0.469**
	(0.249)	(0.188)	(0.211)	(0.218)
Bandwidth	0.7	0.7	0.8	0.4
N	733	733	818	517
B. Alternative bandwidths				
Bandwidth 0.5	0.263	-0.106	0.051	-0.335*
	(0.294)	(0.226)	(0.284)	(0.20)
N	543	543	543	517
Bandwidth 1.0	0.126	-0.079	-0.098	-0.105
	(0.197)	(0.150)	(0.190)	(0.145)
N	1,022	1,022	1,022	969
Bandwidth 2.0	-0.010	-0.077	0.016	-0.046
	(0.145)	(0.120)	(0.150)	(0.109)
N	1,606	1,606	1,606	1,509
Bandwidth 2.5	-0.044	-0.076	0.016	-0.015
	(0.130)	(0.112)	(0.140)	(0.102)
N	1,708	1,708	1,708	1,609

Notes: N: Number of observations. Each cell represents a different regression. The RD coefficients are estimated by fitting a local linear regression using a triangular kernel to the right and the left of the cutoff without including the baseline covariates. All specifications allow for different slopes to the left and the right of the cutoff and standard errors clustered at the school level are in parentheses. Panel A corresponds to the optimal bandwidth while Panel B corresponds to alternative bandwidth. *, **, *** denote significance at the 10%, 5% and 1% level, respectively.

Table 3: The average treatment effect of nutrition information on cognition (different bandwidths)

	Cognitive outcomes						Educational outcomes		
	Block design	Digit span forward	Digit span backward	Progressive matrices	Day and night	Cognitive index	Math	Reading	Attendance
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
A. Main results									
Optimal Bandwidth	-0.480**	0.137	-0.246	-0.149	-0.096	-0.310*	-0.104	-0.054	0.056
(CCT)	(0.232)	(0.234)	(0.187)	(0.185)	(0.183)	(0.183)	(0.163)	(0.195)	(0.036)
Bandwidth	0.5	0.4	0.7	0.5	0.7	0.4	0.7	0.6	0.6
N	514	395	691	514	691	395	691	602	563
B. Alternative bandwidth									
Bandwidth 0.5	-0.480**	0.015	-0.262	-0.149	-0.121	-0.300*	-0.107	0.017	0.058
	(0.232)	(0.193)	(0.216)	(0.185)	(0.222)	(0.156)	(0.200)	(0.221)	0.039
N	514	514	514	514	514	514	514	514	482
Bandwidth 1.0	-0.260	0.028	-0.212	-0.043	0.025	-0.140	-0.108	-0.120	0.044
	(0.165)	(0.127)	(0.157)	(0.151)	(0.148)	(0.116)	(0.132)	(0.133)	(0.028)
N	955	955	955	955	955	955	955	955	899
Bandwidth 2.0	-0.294**	0.092	-0.124	-0.032	0.030	-0.101	-0.104	-0.069	0.024
	(0.118)	(0.101)	(0.118)	(0.126)	(0.124)	(0.095)	(0.101)	(0.098)	(0.022)
N	1,488	1,488	1,488	1,488	1,488	1,488	1,487	1,487	1,405
Bandwidth 2.5	-0.290***	0.064	-0.103	-0.029	0.026	-0.101	-0.096	-0.040	0.019
	(0.109)	(0.091)	(0.111)	(0.118)	(0.113)	(0.088)	(0.094)	(0.094)	(0.020)
N	1,584	1,584	1,584	1,584	1,584	1,584	1,583	1,583	1,493

Notes: N: Number of observations. Each cell represents a different regression. The RD coefficients are estimated by fitting a local linear regression using a triangular kernel to the right and the left of the cutoff without including the baseline covariates. All specifications allow for different slopes to the left and the right of the cutoff and standard errors clustered at the school level are in parentheses. Panel A corresponds to the optimal bandwidth while Panel B corresponds to alternative bandwidth. *, **, *** denote significance at the 10%, 5% and 1% level, respectively

Table 4: Additional specifications: Treatment effects on child health and feeding practices

	Dietary diversity score	Frequency of meat consumption	Frequency of green veg consumption	Hemoglobin
	(1)	(2)	(3)	(4)
A. Rectangular kernel	0.274	-0.037	-0.126	-0.193
	(0.235)	(0.172)	(0.198)	(0.193)
B. With controls	0.071	-0.079	-0.039	-0.434
	(0.259)	(0.185)	(0.224)	(0.235)
C. Local polynomial	0.152	-0.040	-0.073	-0.320
2nd order	(0.236)	(0.211)	(0.265)	(0.205)
D. Global polynomial regressions				
Polynomial 1st order	-0.170	-0.095	0.000	0.011
	(0.109)	(0.100)	(0.126)	(0.093)
Polynomial 2nd order	-0.033	-0.104	-0.014	0.004
	(0.166)	(0.134)	(0.172)	(0.115)
E. Donut (excluding Hb	-0.001	-0.014	-0.137	-0.047
value 10.9 and 11)	(0.251)	(0.202)	(0.295)	(0.241)

Notes: N: Number of observations. Each cell represents a different regression. The RD coefficients are estimated by fitting a local linear regression using a triangular kernel to the right and the left of the cutoff without including the baseline covariates. All specifications allow for different slopes to the left and the right of the cutoff and standard errors clustered at the school level are in parentheses. Panel A corresponds to the optimal bandwidth while Panel B corresponds to alternative bandwidth. *, **, *** denote significance at the 10%, 5% and 1% level, respectively.

Table 5: Additional specifications: Treatment effects on cognitive and educational outcomes

	Cognitive outcomes						Educational outcomes		
	Block design	Digit span forward	Digit span backward	Progressive matrices	Day and night	Cognitive index	Math	Reading	Attendance
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
A. Rectangular Kernel	-0.401*	-0.029	-0.244	-0.075	-0.048	-0.361**	-0.145	-0.163	0.053
	(0.208)	(0.219)	(0.184)	(0.183)	(0.166)	(0.173)	(0.142)	(0.171)	(0.034)
B. With controls	-0.436*	0.016	-0.264	-0.122	-0.122	-0.357**	-0.029	0.025	0.066
	(0.241)	(0.228)	(0.212)	(0.194)	(0.200)	(0.173)	(0.168)	(0.198)	(0.034)
C. Local polynomial	-0.509**	0.024	-0.170	-0.075	-0.059	-0.266*	-0.104	-0.115	0.049
2nd order	(0.231)	(0.172)	(0.179)	(0.171)	(0.164)	(0.138)	(0.146)	(0.184)	(0.033)
D. Global polynomial regressions									
Polynomial 1st order	-0.268***	-0.005	-0.083	-0.032	-0.030	-0.126	-0.081	-0.011	0.011
	(0.096)	(0.074)	(0.100)	(0.105)	(0.096)	(0.080)	(0.086)	(0.093)	(0.017)
Polynomial 2nd order	-0.322**	0.115	-0.052	-0.071	0.069	-0.080	-0.094	-0.053	0.021
	(0.126)	(0.102)	(0.127)	(0.140)	(0.140)	(0.103)	(0.108)	(0.109)	(0.023)
E. Donut (excluding Hb	-0.193	-0.468**	-0.230	0.072	0.186	-0.234	-0.056	-0.153	0.027
value 10.9 and 11)	(0.266)	(0.221)	(0.210)	(0.251)	(0.214)	(0.184)	(0.168)	(0.229)	(0.040)

Notes: N: Number of observations. Each cell represents a different regression. The RD coefficients are estimated by fitting a local linear regression using a triangular kernel to the right and the left of the cutoff without including the baseline covariates. All specifications allow for different slopes to the left and the right of the cutoff and standard errors clustered at the school level are in parentheses. Panel A corresponds to the optimal bandwidth while Panel B corresponds to alternative bandwidth. *, **, *** denote significance at the 10%, 5% and 1% level, respectively

Table 6: Heterogeneous effects: Whether mothers received the information treatment

	CCT Bw	Bw 0.5	Bw 1.0	Bw 2.0	Bw 2.5	Rectangular kernel	With controls	Local polynomial
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)
Panel A : Child health and feeding practices								
Dietary diversity score	0.279	0.355	0.175	0.009	-0.022	0.154	0.131	0.295
	(0.292)	(0.319)	(0.216)	(0.161)	(0.145)	(0.267)	(0.309)	(0.274)
Frequency of meat consumption	-0.072	-0.098	-0.114	-0.104	-0.093	-0.072	-0.038	-0.037
	(0.206)	(0.247)	(0.168)	(0.129)	(0.120)	(0.187)	(0.210)	(0.235)
Frequency of green veg consumption	0.010	0.010	-0.158	-0.045	-0.033	-0.049	-0.043	-0.094
	(0.275)	(0.275)	(0.195)	(0.160)	(0.151)	(0.253)	(0.274)	(0.277)
Hemoglobin	-0.388*	-0.439*	-0.093	-0.049	-0.016	-0.359	-0.346	-0.314
	(0.225)	(0.252)	(0.147)	(0.111)	(0.104)	(0.222)	(0.245)	(0.226)
Panel B: Cognitive outcomes								
Block design	-0.440*	-0.440*	-0.256	-0.265**	-0.260**	-0.355	-0.444	-0.451*
	(0.243)	(0.243)	(0.171)	(0.128)	(0.118)	(0.217)	(0.260)	(0.246)
Digit span forward	0.067	-0.052	-0.004	0.077	0.050	-0.133	-0.104	-0. 9
	(0.241)	(0.203)	(0.141)	(0.110)	(0.098)	(0.226)	(0.243)	(0.178)
Digit span backward	-0.266	-0.267	-0.246	-0.142	-0.116	-0.272	-0.292	-0.180
	(0.225)	(0.239)	(0.175)	(0.130)	(0.123)	(0.220)	(0.240)	(0.220)
Progressive matrices	-0.147	-0.147	-0.052	-0.041	-0.034	-0.099	-0.183	-0.035
	(0.196)	(0.196)	(0.165)	(0.134)	(0.124)	(0.196)	(0.187)	(0.183)
Day and night	-0.115	-0.141	0.047	0.046	0.033	-0.073	-0.164	-0.125
	(0.206)	(0.221)	(0.155)	(0.128)	(0.115)	(0.201)	(0.220)	(0.195)
Cognitive index	-0.318*	-0.314*	-0.154	-0.100	-0.100	-0.419	-0.430	-0.264
	(0.188)	(0.164)	(0.127)	(0.103)	(0.095)	(0.183)	(0.179)	(0.159)
Panel C : Educational outcomes								
Math	-0.159	-0.153	-0.152	-0.147	-0.139	-0.150	-0.065	-0.146
	(0.146)	(0.193)	(0.126)	(0.097)	(0.088)	(0.136)	(0.151)	(0.154)
Reading	-0.045	-0.045	-0.177	-0.115	-0.078	-0.106	-0.028	-0.094
	(0.222)	(0.222)	(0.133)	(0.098)	(0.095)	(0.209)	(0.213)	(0.205)
Attendance	0.056	0.058	0.043	0.027	0.021	0.053	0.076*	0.043
	(0.039)	(0.042)	(0.029)	(0.022)	(0.021)	(0.037)	(0.037)	(0.035)

Notes: Each cell represents a different regression. Unless otherwise indicated in Panels A-C, the RD coefficients are estimated by fitting a local linear regression using a triangular kernel to the right and left of the cutoff. All specifications allow for different slopes to the left and the right of the cutoff and standard errors are clustered at the school level. *, **, *** denote significance at the 10%, 5% and 1% level, respectively.

Table 7: Minimal detectable effects for different bandwidth

	Bandwidths						Effect size in other nutrition interventions
	0.3	0.5	1.0	1.5	2.0	2.5	
	(1)	**(2)**	**(3)**	**(4)**	**(5)**	**(6)**	**(7)**
Panel A: Child health and feeding practices							
Dietary diversity	1.776	1.361	0.97	0.906	0.876	0.843	
Frequency of meat consumption	1.409	0.991	0.667	0.613	0.605	0.579	
Frequency of green vegetable consumption	2.081	1.66	1.318	1.186	1.109	1.051	
Hemoglobin	0.871	0.724	0.601	0.545	0.563	0.551	0.136*[1] 0.151[2] 0.275[3] 0.202**[4] 0.416**[4]
Panel B: Cognitive outcomes							
Block design	1.202	0.922	0.698	0.699	0.667	0.639	0.012[1] 0.045[2]
Digit span forwards	1.246	0.99	0.655	0.599	0.585	0.555	-0.105[1] -0.135[2]
Digit span backwards	1.278	0.996	0.788	0.715	0.691	0.668	0.009[1] -0.23[2]
Progressive matrices	1.123	1.211	0.849	0.76	0.713	0.692	0.070[1] 0.112[2]
Day and night	1.32	1.134	0.862	0.754	0.723	0.683	0.116[1] 0.210[2]
Cognitive index	0.919	0.91	0.69	0.634	0.601	0.58	0.028[1] 0.058[2]
Panel C: Educational outcomes							
Math	1.273	1.054	0.79	0.729	0.701	0.663	0.112[1] 0.197*[2]
Reading	1.253	1.039	0.711	0.664	0.635	0.603	0.129[1] 0.182*[2]
Attendance	0.265	0.19	0.156	0.147	0.143	0.136	-0.005[1]

1 Effect size from the evaluation of the school intervention by Krämer et al. (2018).
2 Effect size from the evaluation of the school intervention by Krämer et al. (2018) at 90% school attendance.
3 Effect size in Luo et al. (2012), Information experiment 2.
4 Effect size in Luo et al (2012), Experiments 1 and 2, multivitamin supplement treatment arm.
*, **, *** denote significance at the 10%, 5% and 1% level, respectively.

A. Appendix

Text A.1 Control Variables

We use a set of control variables for the three different outcome categories. For the feeding practice and anemia outcomes we include socioeconomic characteristics (rural or urban, block, a wealth index, the father's and mother's years of schooling, caste, religion, the number of household members and if the child's father lives in the household), nutritional factors (an indicator for household food security, the number of meals the child eats every day, if the child took iron supplements in the last year, the average intake of calories and iron from the school meal, an indicator for maternal health knowledge), access to healthcare (dummy for institutional delivery of the child and if any household member is covered by a health insurance), morbidity indicators (if the child suffered from diarrhea in the last 30 days and if the household possesses an improved sanitation facility) as well as one biological factor (sex of the child).

For the cognitive outcomes, we include the same covariates. In addition, we use indicators for psychosocial stimuli (a dummy if the mother helps the child with its homework, the time the mother spends on giving physical care to the child and if parents participate in parent-teacher meetings at school, a dummy if the father lives in the household) and a dummy for the test administrator was included. Further indicators for quality of schooling (total school enrollment, the student-teacher ratio, the number of children that attended second grade at the baseline and the fourth grade at the endline on the day of the interview and the distance to the school) were included as controls for the education outcomes. Since it might be the case that the quality of information conveyed differs between the six medical people that conducted the medical tests, we further control for who conducted the blood test for anemia.

Table A.1: Average treatment effect on the sample excluding the DFS treatment group

	Dietary diversity score	Frequency of meat consumption	Frequency of green veg consumption	Hemoglobin
	(1)	(2)	(3)	(4)
A CCT Bandwidth	0.444	-0.127	-0.040	-0.298
	(0.562)	(0.546)	(0.841)	(0.258)
Bandwidth	0.4	0.4	0.3	0.6
N	172	172	122	266
B Alternative bandwidth				
Bandwidth 0.5	0.413	-0.098	-0.038	-0.340
	(0.520)	(0.469)	(0.667)	(0.285)
N	225	225	225	227
Bandwidth 1.0	0.176	-0.034	-0.118	-0.190
	(0.349)	(0.285)	(0.443)	(0.210)
N	421	421	421	420
Bandwidth 2.0	0.103	-0.075	0.097	-0.109
	(0.256)	(0.206)	(0.292)	(0.168)
N	692	692	692	682
Bandwidth 2.5	0.090	-0.087	0.093	-0.072
	(0.226)	(0.190)	(0.257)	(0.154)
N	743	743	743	735
C Rectangular Kernel	0.482	-0.089	-0.189	-0.238
	(0.569)	(0.515)	(0.778)	(0.242)
D With controls	0.451	-0.068	0.208	-0.237
	(0.698)	(0.515)	(0.808)	(0.220)
E Local polynomial				
2nd order	0.195	0.008	-0.121	-0.201
	(0.434)	(0.368)	(0.698)	(0.226)
F Global polynomial regressions				
Polynomial 1st order	-0.095	-0.152	0.096	-0.075
	(0.185)	(0.176)	(0.196)	(0.133)
Polynomial 3rd order	0.169	0.106	-0.049	-0.152
	(0.345)	(0.287)	(0.468)	(0.225)
G Donut (excluding	-0.014	0.054	-0.562	0.073
Hb value 10.9 and 11)	(0.526)	(0.306)	(0.765)	(0.236)

*Note*s: N denotes number of observations. Each cell represents a different regression. Unless otherwise indicated in panels A-E, the RD coefficients are estimated by fitting a local linear regression separately using a triangular kernel. All specifications allow for different slopes to the left and the right of the cutoff and standard errors are clustered at the school level. *, **, *** denote significance at the 10%, 5%, 1% level respective.

Table A.2: Average treatment effect on cognition and education for sample excluding the DFS treatment

	Cognitive outcomes						Educational outcomes		
	Block design	Digit span forward	Digit span backward	Progressive matrices	Day and night	Cognitive index	Math	Reading	Attendance
	(1)	(2)	(3)	(4)	(5)	(6)	(7)	(8)	(9)
A CCT Bandwidth	-0.202	0.047	-0.541**	0.069	0.474*	-0.204	0.287	0.422	0.055
	(0.418)	(0.443)	(0.258)	(0.314)	(0.242)	(0.289)	(0.307)	(0.262)	(0.0410
Bandwidth	0.5	0.3	0.7	0.5	0.6	0.4	0.5	0.5	0.7
N	243	131	328	243	284	187	243	243	307
B Alternative bandwidth									
Bandwidth 0.5	-0.202	-0.363	-0.607*	0.069	0.510*	-0.178	0.287	0.422	0.069
	(0.418)	(0.298)	(0.324)	(0.314)	(0.278)	(0.233)	(0.307)	(0.262)	(0.054)
N	243	243	243	243	243	243	243	243	229
Bandwidth 1.0	-0.189	-0.145	-0.378*	0.117	0.467**	-0.041	0.161	0.194	0.042
	(0.263)	(0.193)	(0.207)	(0.239)	(0.191)	(0.165)	(0.185)	(0.156)	(0.036)
N	448	448	448	448	448	448	448	448	423
Bandwidth 2.0	-0.432**	-0.083	-0.193	0.108	0.203	-0.126	-0.104	0.036	0.023
	(0.174)	(0.131)	(0.154)	(0.178)	(0.161)	(0.123)	(0.120)	(0.116)	(0.026)
N	777	777	777	777	777	777	776	777	731
Bandwidth 2.5	-0.319	-0.036	-0.223	0.139	0.336*	-0.038	0.039	0.120	0.036
	(0.207)	(0.173)	(0.181)	(0.203)	(0.189)	(0.147)	(0.153)	(0.131)	(0.032)
N	614	614	614	614	614	614	613	614	577
C Rectangular kernel	-0.115	-0.124	-0.456*	0.166	0.417*	-0.298	0.210	0.332	0.046
	(0.344)	(0.444)	(0.236)	(0.284)	(0.223)	(0.273)	(0.294)	(0.252)	(0.039)
D With controls	-0.318	-0.210	-0.495*	0.014	0.442	-0.119	0.421	0.681**	0.046
	(0.445)	(0.495)	(0.294)	(0.280)	(0.291)	(0.346)	(0.398)	(0.268)	(0.048)
E Local polynomial									
2nd order	-0.298	-0.239	-0.658*	0.129	0.406*	-0.153	0.280	0.236	0.049
	(0.403)	(0.294)	(0.340)	(0.279)	(0.224)	(0.202)	(0.265)	(0.217)	(0.044)
F Global polynomial regressions									
Polynomial 1st order	-0.403**	-0.138	-0.098	0.072	0.132	-0.135	-0.155	0.018	0.011
	(0.158)	(0.098)	(0.136)	(0.163)	(0.141)	(0.112)	(0.103)	(0.125)	(0.024)

Polynomial 2nd order	-0.459**	-0.009	-0.127	0.080	0.269	-0.080	-0.075	0.013	0.032
	(0.198)	(0.136)	(0.199)	(0.199)	(0.189)	(0.142)	(0.146)	(0.132)	(0.030)
G Donut (excluding	-0.007	-1.292***	-0.415	0.194	0.384	-0.146	0.192	0.186	0.034
Hb value 10.9 and 11)	(0.342)	(0.392)	(0.439)	(0.436)	(0.313)	(0.217)	(0.302)	(0.262)	(0.049)

Notes: N: Number of observations. Each cell represents a different regression. Unless otherwise indicated in Panels A-E the RD coefficients are estimated by fitting a local linear regression separately using a triangular kernel. All specifications allow for different slopes to the left and the right of the cutoff and standard errors are clustered at the school level. *, **, *** denote significance at the 10%, 5% and 1% level, respectively.

Table A.3: Minimal detectable effects for different bandwidth (no difference in outcome variable)

	Bandwidths						**Effect size in other nutrition interventions**
	0.3	**0.5**	**1.0**	**1.5**	**2.0**	**2.5**	
Panel A: Child health and feeding practices							
Dietary diversity	1.308	1.028	0.822	0.76	0.741	0.712	
Frequency of meat consumption	0.842	0.653	0.522	0.488	0.477	0.459	
Frequency of green vegetable consumption	1.627	1.37	0.997	0.888	0.848	0.809	
Hemoglobin	0.87	0.732	0.547	0.51	0.513	0.495	0.136*[1] 0.151[2] 0.275[3] 0.202**[4] 0.416**[4]
Panel B: Cognitive outcomes							
Block design	1.254	0.829	0.65	0.62	0.581	0.552	0.012[1] 0.045[2]
Digit span forwards	1.205	0.894	0.592	0.574	0.541	0.518	-0.105[1] -0.135[2]
Digit span backwards	1.429	1.057	0.831	0.728	0.682	0.66	0.009[1] -0.23[2]
Progressive matrices	0.779	0.582	0.499	0.462	0.423	0.41	0.070[1] 0.112[2]
Day and night	1.457	1.121	0.861	0.749	0.71	0.676	0.116[1] 0.210[2]
Cognitive index	0.919	0.91	0.69	0.634	0.601	0.58	0.028[1] 0.058[2]
Panel C: Educational outcomes							
Math	1.273	1.054	0.79	0.729	0.701	0.663	0.112 0.197*[2]
Reading	1.253	1.039	0.711	0.664	0.635	0.603	0.129[1] 0.182*[2]
Attendance	0.265	0.19	0.156	0.147	0.143	0.136	-0.005[1]

1 Effect size from the evaluation of the school intervention by Krämer et al. (2018).
2 Effect size from the evaluation of the school intervention by Krämer et al. (2018) at 90% school attendance.
3 Effect size in Luo et al. (2012), Information experiment 2.
4 Effect size in Luo et al (2012), Experiments 1 and 2, multivitamin supplement treatment arm.
*, **, *** denote significance at the 10%, 5% and 1% level, respectively.