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Anemia, diet, and cognitive development: Impact of health information on diet quality and child nutrition in rural India

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Abstract

Lack of information about health risks may limit the adoption of improved nutritional and healthy behavior. This paper studies the effect of a nutrition information intervention on household dietary behavior, hemoglobin levels, and cognitive outcomes of children in rural India. Using experimental data and regression discontinuity design that exploits the exogenous cutoff of hemoglobin level for anemia, we find statistically insignificant treatment effects on dietary improvements, child health, and cognitive outcomes of children. Our findings suggest that light-touch nutrition information alone, even when parents are informed about the health risk of their children, may not promote healthy behavior and factors other than information might constrain households in making nutritional investments for their children.

JEL codes: I12, I15, I18, O12

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

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1. Introduction

Despite its importance as a determinant of human capital formation, micronutrient deficiencies - particularly anemia - are still widespread in many low- and middle-income countries (LMICs). Globally, anemia affects one-third of the world's population and about 50% of the anemic cases are due to iron deficiency, but this share is substantially higher in India (Anand et al., 2014; WHO, 2016). Iron deficiency caused due to the consumption of a nutritionally deficient diet is one of the important critical causes of anemia among Indian children (Tankachan et al., 2008, Pasrich et al., 2010).[1] Iron-deficient anemia (IDA) affects 42% of children under age five and its prevalence varies according to geographic regions (WHO, 2016). Countries in South Asia and Sub-Saharan Africa had the highest prevalence of IDA across all age groups. Despite substantial economic growth and several anemia-reduction programs, IDA is highly prevalent among children in India. Recent data from India shows that about 60% of the under-five children are anemic, making it an urgent public-health priority.

Iron deficiency is associated with increased physical and cognitive impairment in childhood, resulting in lower schooling, irreversible productivity loss in adulthood, and substantial economic burden in developing countries (Strauss and Thomas, 1998; Alderman, Behrman, Lavy, and Menon, 2001; Alderman, Hoddinott, and Kinsey 2006; Glewwe, Jacoby, and King, 2001). The estimated annual costs of IDA in early childhood amount to intangible costs of 8 million disability-adjusted life years and productivity loss equal to 1.3% of the gross domestic product in India (Plessow et al., 2015).

Several effective and inexpensive nutrition technologies, such as iron supplements, iron-fortified products, or bio-fortified seeds, are available to prevent iron deficiency; however, diffusion and adoption of these technologies have been limited in developing countries (Banerjee, Barnhardt, and Duflo, 2018). Similarly, consuming a more diverse and nutritious diet, an effective strategy to reduce iron deficiency, seems to be challenging to individuals despite significant potential health gains (Ogden et al., 2007; Oster, 2018). For example, despite such a high incidence of IDA, the average iron intake among Indian children is 2.4% of the recommended daily intake (Onyeneho et al., 2019).

[1] Since iron deficiency is the most common cause of anemia in India and globally, we use iron-deficient anemia (IDA) and anemia interchangeably in this paper.

The high prevalence of anemia and limited adoption of healthy behavior to prevent iron deficiency is puzzling as it can easily be tackled with nutrition-specific interventions which are simple and inexpensive. One reason for the limited adoption of health technology and health-promoting behavior such as consuming an iron-rich diet could be that households are unaware of the health risks caused due to iron deficiency and the potential solutions to reduce these risks. Lack of or limited access to knowledge about illness or preventive strategies may play an important role in explaining limited adoption of healthy behavior or improved dietary choices (Dupas, 2011a; Luo et al., 2012; Madajewicz et al., 2007). In a review paper, Dupas (2011a) suggest that the discrepancy between the actual and optimal health behavior is possibly due to information deficit and findings from several studies suggest that households are responsive to information to health risks.

This study examines the causal effect of an information intervention that informed parents of the anemic status of their children and preventive strategies to reduce anemia risk in their children. The intervention informed parents about the anemia status of their child and parents of anemic children were encouraged to provide diverse and iron-rich food to their children. The dietary advice was only given to the parents of anemic children and parents of non-anemic children were only informed of the anemic status of their child. We first investigate how this information intervention affects dietary behavior and consumption of iron-rich foods. We subsequently examine whether the information intervention had any effects on children's health status - measured by the children's hemoglobin levels - as well as its impacts on the cognitive ability and educational outcomes of the children.

Our study setting is the state of Bihar, one of the poorest states with low human capital in India. Bihar is an important setting to explore this question as depending on age and gender, anemia in India ranges from 32% to 63% (NFHS-4, 2017). Approximately, 8% of all children under age five in Bihar are stunted and 44% are underweight (NFHS-4, 2017).[2] Our study uses data from a randomized controlled trial conducted by the authors on the impacts of iron-fortified salt on anemia. In this experiment, grade two children were tested for anemia in 104 primary schools in Bihar. We use this data to roll out another experiment related to nutrition information and

[2] Per the 2015 Global Burden of Disease study, 10.6% of total years lived with disability were due to IDA in India.

anemia of the sampled children. Parents of anemic children (hemoglobin level ≤10.9 g/dl) were informed of their child's anemia status and were also advised to feed their child more iron-rich food items, namely green leafy vegetables and lean meats if the household consumes meat. Non-anemic children (hemoglobin level ≥ 11.0 g/dl) were not exposed to any information intervention. We exploit the discontinuity in the information intervention based on a hemoglobin level of 11.0g/dl and employ a regression discontinuity design (RDD) method to estimate the causal impacts of the information intervention on dietary behavior, hemoglobin, cognitive, and educational outcomes of the children. RDD addresses unobserved heterogeneity and omitted variable bias that might affect the knowledge of health risks and food dietary behavior.

Rigorous impact evaluation of health and nutrition information on health-related behavioral change is limited and has found mixed results. Several empirical studies have found positive effects of information treatment on health-promoting behaviors such as water purification (Jalan and Somnathan, 2008; Madajewicz et al., 2007), reduction in risky sexual behavior (Thornton, 2008; Dupas, 2011b), and consumption of fewer calories by diabetes patients (Oster, 2018).[3] These studies suggest households in developing countries face information constraint and their health behavior is often responsive to information and households do respond to learning about their health risks and health-specific information.

In contrast to the role of information on general health risks discussed before, the literature on nutrition information in the context of anemia is less encouraging. Wong et al. (2014) find modest effects of educating parents about nutrition and anemia on children's hemoglobin levels in north-western China. In an experimental study 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; however, no effects were found on anemia after 18 months. Using a relatively small sample of about 250 newborns in Brazil, Bortolini and Vitolo (2012) found that systematic dietary home counseling did not affect the prevalence of anemia, iron deficiency, or iron-deficient anemia. Resistance to dietary modification has also been noticed among both obese and obese-susceptible individuals in the USA (Ogden

[3] Jalan and Somanathan (2008) found that households in a suburb of New Delhi improve water purification after learning about the contaminated drinking water. Thornton (2008) found that individuals reduce risky sexual behavior after learning about their HIV status in Malawi. Dupas (2011a) reported teenager girls practiced safer behavior after learning age-specific relative risk of HIV infection.

et al., 2007).

The effectiveness of information treatment depends on the type and intensity of information, who received the information, type of illness, and gender and education of the information recipient (Dupas, 2011a). When health information was shared with children rather than parents in Kenya, the information intervention had no effects on children's behavior (Kremer and Miguel, 2007). Parents are expected to be more responsive to information about child's health than children. Whether the message is delivered to the mother or father or both also affects the effectiveness of information. For example, Dupas (2009) found women to be more responsive compared to men to the information as women were more likely to buy bednets than men in Kenya. The information campaign was more effective when a message was delivered to both compared with either of them receiving alone. The education level of the information recipient is another factor that modifies the effect of information intervention as more educated individuals have a higher propensity to change their behavior after receiving information.

While much of the literature relates to enhancing preventive healthcare, this study addresses the adoption of remedial behavior. In our treatment, the anemic status of a child is revealed by a diagnostic test. The hemoglobin testing makes the disorder explicit and the need for immediate action. In contrast, with preventive healthcare interventions, individuals believe they will not be affected, hence, the need for preventive action might not be perceived as acute. Furthermore, individuals procrastinate preventive care if the required preventive actions are costly. There are only a few studies that link information on healthy behavior with revealing an individual's health status in the context of HIV (Thornton, 2008), malaria (Cohen et al., 2015), and diabetes (Oster, 2018). 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. We add to this literature by using a sample of rural Indian households and a very simple information intervention. An additional innovation of this study is not only the assessment of health and nutrition but also of human capital 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.

Our main results confirm the findings in the previous literature that information alone is less effective in changing nutrition-related behavior, even when combined with the revelation of a nutritional disorder of a child (Luo et al., 2012). The RDD estimates show that parents who were informed of the anemic status of their children did not improve their dietary behavior and food consumption, particularly increased consumption of iron-rich food items. Consistent with insignificant effects on improvement in iron-rich diets, the information treatment effects on hemoglobin levels and cognitive outcomes are statistically insignificant and close to zero. Overall, our results indicate that informing parents about anemia and strategies to mitigate anemia risk for their school-age children appears to have no effect on children's health in rural Bihar, India.

Our study contributes to several strands of literature. First, this study contributes to the limited and mixed evidence on the effects of an information intervention on household health and nutritional behavior in developing countries (Luo et al., 2012; Zhao et al., 2013; Shimokawa, 2013). Luo et al. (2012) and Shimokawa (2013) found null effects, i.e., being informed of anemia and obesity risk and a balanced diet to reduce these health risks, respectively, does not necessarily change dietary behavior. In contrast, Zhao et al. (2013) found that information on hypertension reduced fat intake in China.[4] *Second*, this paper contributes to the literature on the effects of nutrition information on children's health (Wong et al., 2014; Fitzsimons et al., 2016). While Wong et al. (2014) show that educating parents about nutrition and anemia had a modest impact on anemia in China, Fitzsimons et al. (2016) find that intensive information intervention had significant improvements in the diet and physical growth of infants in rural Malawi. *Third*, this study relates to the broader literature on the association between child health and cognitive ability of children in developing countries (Almond, Currie, and Duque, 2018). Our study departs from the previous literature by not only considering health and nutrition outcomes but also looking at productive outcomes such as cognition and education. Previous studies show that iron supplementation reduced anemia and led to an improvement in cognitive outcomes in China (Luo et al., 2012)

[4] The effect of being informed of own health status on adoption of healthy behavior has been studied in the context of HIV (Thornton, 2008) and malaria (Cohen et al., 2015). The effects were positive in the HIV study, while no clear evidence emerged in the malaria study.

and Peru (Chong et al., 2016).

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 describe the results in section 4. In section 5, we present robustness checks, and discuss our findings and conclude in section 6.

2. Background, Intervention, and Data

2.1. Background

Anemia refers to a condition where the level of hemoglobin in the blood is low. Though anemia can have different causes,[5] iron deficiency is the most common (WHO, 2001). Anemia can be caused by a variety of factors such as iron deficiency, acute or chronic infection, blood loss, or genetic hemoglobin disorders like Beta Thalassemia Trait (BTT). Our intervention only addresses iron deficiency anemia, which unfortunately we cannot directly distinguish from other causes of anemia but based on existing evidence we can be certain that iron deficiency is the predominant cause of anemia in our setting. The average prevalence of BTT is small (3.4%) in the eastern regions of Bihar, Chattisgarh, Jharkhand, and eastern Uttar Pradesh (Nagar et al., 2015). Another study in the adjoining district in Bihar found a hookworm infection rate of 2.3% (Banerjee, Barnhardt, and Duflo, 2018), indicating that hookworm infection is not very prevalent in Bihar. Banerjee et al. (2018) also report that deworming is very common in schools in Bihar. This is corroborated in our school survey which found that our sample children were dewormed at least once a year. Further, malaria is not endemic in Bihar with only 0.1 cases reported for every 1000 persons (Caravotta, 2009). Thus, it is unlikely that genetic factors, malaria, or hookworm infections explain a large share of anemia cases in our study sample. Additionally, several small-scale, as well as the national level studies, found that mean iron intake is quite low in India- 11.2% (Pasricha et al., 2010) and 2.4% of the recommended daily intake (NFHS, 2017). The low level of iron intake indicates that poor diet is an important cause of iron deficiency among Indian children.

[5] 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.

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 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 and Ross (2003), who used data from 10 low-income countries, physical and cognitive impairment due to iron deficiency causes a median loss of 4.1% of a country's GDP. Globally, more than 20% of the world's population (about 1.62 billion people) are anemic (WHO, 2008).[6] 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). In the Jehanabad district (state of Bihar, India), where our study took place, the prevalence of anemia ranges from 26% for adult males to 63% for children below the age of 5 (NFHS-4, 2017).

2.2. Intervention & theory of change

The analysis is based on data collected from a randomized controlled trial that evaluated the effect of the use of double fortified salt in school-lunch preparation on anemia and cognitive outcomes. The DFS intervention was done at the school level (for more details see Krämer, Kumar, and Vollmer, 2020). The study measured the hemoglobin level of second-grade children in government-funded rural primary schools. The testing was performed with an on-site hemoglobin measurement device directly in the village or at the children's homes. We exploit this existing study to implement an information treatment at the household level. All parents were informed about the anemic status of their children and parents of anemic children were further provided nutrition information on how to mitigate the anemia risk by modifying their dietary behavior. Parents of non-anemic children received no information and thus

[6] 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.

formed the control group. 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). The following information was shared with the parents of the anemic children (treatment group):

> **8 ≥ Hemoglobin level ≤ 10.9 g/dl (moderate anemia):** Parents were advised to provide a more diverse and iron-rich diet for the child, especially the consumption of green leafy vegetables and meat if the household consumes meat (iron-rich food).
>
> **Hemoglobin level < 8 g/dl (severe anemia):** In addition to the nutritional information, parents were advised to consult a doctor at the nearest health facility. If the hemoglobin level was below 6 g/dl, the interview team ensured that the child was quickly taken to the nearest health facility for medical advice.
>
> **Hemoglobin level > 10.9 g/dl (Non-anemic):** Parents of non-anemic children were not exposed to information intervention and served as a control group.

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 analysis to the information given for a hemoglobin value ≤ 10.9 g/dl.[7]

Our information intervention was designed to benefit vegetarian as well as non-vegetarian populations. The government surveys estimate that about 20-30% of the Indian population follow a vegetarian diet, however, the rate of vegetarianism in Bihar is quite low (8%) (SRS baseline survey, 2014). Therefore, increasing the consumption of animal sources of iron could be quite effective in increasing the iron intake among children. Since our information treatment specifically advised parents to include iron-rich vegetables as well as meat products, we believe that the treatment is likely to be helpful for the vegetarian as well as non-vegetarian population. In addition to the consumption of iron-rich diets, the nutrition literature highlights the importance of vitamin C in how well iron is absorbed into our body. Vitamin C has been shown to

[7] For our main analysis, we include both the control and treatment groups of the RCT conducted by Krämer, Kumar, and Vollmer (2020), 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 the appendix.

help iron absorption. Foods high in vitamin C include dark leafy vegetables such as spinach, cauliflower, cabbage; tomato, potato, peas, and citrus fruits such as oranges, mango, lychee, papaya, etc.

Our information treatment did not specifically advise parents to include consumption of foods high in vitamin C but we believe consumption of leafy vegetables and peas which are a good source of vitamin C would have aided iron absorption. Our study lacks the data on the consumption of specific food items high in vitamin C but prior government report suggests that the monthly per capita consumption of cauliflower, potato, eggplant, and cabbage which are a good source of vitamin C was higher in Bihar than the national average consumption of these vegetables (NSS, 2011-12). Furthermore, mango, papaya, guava, lychee, etc. are seasonally available in the study regions and most of the rural households consume these home-grown fruits. For example, Bihar is the largest producer of lychee (high in vitamin C) and the third-largest producer of guava (rich in iron and vitamin C both). Furthermore, we also note that studies in economics found positive impacts of iron pills alone on anemia in Peru (Chong et al., 2014). The adolescents in this study were not provided vitamin C to aid iron absorption. Furthermore, the global flagship program "the weekly iron-folic acid supplementation" implemented in many countries provides iron pills only and does not include vitamin C supplementation to combat anemia.

One concern could be that Indian households often consume rice which is high in phytic acid and affects iron absorption adversely. This has limited relevance in our study as consumption of leafy vegetables would counteract the adverse effect of phytic acid in iron absorption. In one study, preschoolers with IDA were given vitamin C supplements twice a day - 100 mg at each of two phytate-rich meals. After two months, most of the kids were no longer anemic (Seshahdri et al., 1985). Additionally, in another paper, we find that the use of iron-fortified salt in school lunches which included rice reduced anemia by 20% in our study region (Krämer, Kumar, and Vollmer, 2020). Results from these two studies are comforting as they show that even if children continue to eat food high in phytates such as rice, and an additional iron intake either through salt or dietary modification is likely to have positive impacts on anemia reduction.

While designing our field experiments, the study team relied on local knowledge about the availability of iron-rich foods in the local market or the sampled villages. The field staff found that iron-rich vegetables, such as cabbage, legumes, lentils, gram, were locally produced and were available seasonally in the rural areas of Bihar, so the availability of fresh and iron-rich vegetables is not a major constraint as long as they are in season. The information treatment did not include food items that were not grown locally or not available. Since these vegetables were locally produced and traded within the village when they are in season, we believe that the availability of these food items is unlikely to be a constraint. especially for the non-haem (plant-based) source of iron. Furthermore, Bihar is the fourth-largest producer of vegetables in India and commonly grown crops are cauliflower, potato, cabbage, eggplant, tomato, etc. (GOI, 2018). The widely grown fruits in Bihar are guava, banana, mango, pineapple, and lychee. Since our information treatment specifically advised parents to include iron-rich vegetables, as well as meat products (sometimes banana was also mentioned), we believe that the treatment is likely to be helpful for the vegetarian as well as non-vegetarian population. We would like to note that seasonal production of some of these crops may have occasionally affected the availability or affordability especially for the poorer households who may lack the financial resources to buy expensive food products in the off-season.

Our conceptual framework posits that lack of information about nutritional deficiencies of children and strategies to overcome this contributes to malnourishment in early childhood in developing countries. The theory of change for the information intervention in our study setting rests on the assumption that many parents are unaware of their children's anemic status because there is a lack of access to blood testing and demand for diagnostics is generally low unless there are severe symptoms. Anemia symptoms are generally not visible and are non-fatal. This information intervention will encourage caretakers to adjust the diets of their children to include more iron-rich food items. This will improve the hemoglobin levels for most children, particularly those with iron deficiency anemia and to a lesser extent also for others. If the improvement in hemoglobin levels is strong enough, this will also support cognitive development and performance in school.

Cognitive outcomes could also be affected by the information treatment even if there are no first-order effects on diet and hemoglobin concentration in blood. This is possible if parents reacted to the information in the short-term, immediately after the treatment, and adjusted diet to include more iron-rich foods, meat, and green leafy vegetables. The improved diet in the short run could have improved hemoglobin levels and could have led to permanent impacts on the cognitive development of children. The short-term diet and health-related behavior change may not sustain for longer periods and may dissipate after some time but the lasting impacts on cognition may persist in the long run, and therefore, measuring the impacts on cognitive outcomes is important regardless of first-order effects on the proximate outcomes.

Our baseline survey found that only 50% of the parents were aware of iron-rich food and none knew the anemic status of their children. The low iron intake as mentioned before suggests the food-based intervention of consumption of iron-rich foods could be an effective strategy to address anemia problems among children in India. And the low awareness about the iron-rich food in the baseline survey indicates that information failure, indeed, exists in our study setting. Parents' lack of knowledge about anemia was a major cause of the high prevalence of anemia in rural China (Wong et al., 2014.) Thus, our information intervention assumes that educating parents about anemia and diet could help parents adjust children's diet and in turn improve both children's health and cognitive outcomes.

Previous studies show that the average intake of iron is low among young children in India and, therefore, the food-based intervention of consumption of iron-rich foods could be an effective strategy to address anemia problems among children in India. The low iron intake and low awareness about the iron-rich food in the baseline survey indicate that information failure is an important problem and therefore, relaxing the information constraint should be the correct intervention in these settings.

2.3. Data and variables

2.3.1. 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) within the Indian state of Bihar. The baseline prevalence of anemia among second-grade students was 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, 20 children per school from the second grade were chosen for anemia testing. The sample is, therefore, representative of second-grade students in public schools in the two blocks.

The selection of second grade was guided by the evidence from the biomedical and economic literature. Previous studies in biomedical and economic science show that primary school children could benefit from increased iron intake. The biomedical literature on the effect of iron supplementation on cognitive development among preschool children found positive impacts on cognitive development in primary school children aged 5-12 years (Low et al. 2013; Larson et al., 2017). The literature in economics also focuses on primary school children and found positive effects of iron supplementation on cognitive outcomes. Luo et al. (2012) found a significant increase in math test scores among 4th-grade students in China. Similarly, Chong et al. (2016) found a 21% increase in cognition test scores among 11-19 years old adolescents in Peru. The age group that we study is 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 and Pribram, 1990; Thatcher, 1991). Furthermore, a reduction in anemia is also likely to lead to more effective instruction in school because children have more energy and are better able to concentrate and pay attention.

With the help of local enumerators and trained medical personnel, we collected socioeconomic information about the children and their households. The survey contains detailed information on feeding practices; socioeconomic and sociodemographic characteristics; access to health care; health insurance; class size; student-teacher ratio; and calories and iron content of the children's school lunches. Trained medical personnel measured hemoglobin level using blood samples collected through capillary blood at home.[8] Additionally, children were administered cognitive and education tests at the school. Approximately two years after the start of the intervention (August-October 2016), we conducted a follow-up survey to investigate the impacts of the information treatment on health and cognitive outcomes. The follow-up sample of about 1,700 children constitutes our main analytical sample.

2.3.2. Outcome variables

The survey collected information on the child's food consumption using a food frequency table, reported by the child's caretaker. Caretakers were asked whether children consumed the different types of food items never, several times in a month, once a week, several times in a week, and daily. The response to the frequency of the

[8] Hemoglobin levels were assessed using an on-site hemoglobin measurement device called HemoCue® Hb 301 (AB Leo Diagnostics, Helsinborg, Sweden).

consumption of food items ranges from 1 (*the child never consumes)* to 5 (*the child consumes daily)*. Using the consumption frequency information from the diet table, three different indicators for feeding practices were developed: a dietary diversity score (DDS), frequency of meat consumption (haem source of iron), and frequency of green vegetable consumption (non-haem source of iron). Similar to Torheim et al. (2003) and Kennedy et al. (2010), and based on data availability, the following six food groups were included in the DDS: (i) Legumes, (ii) fruits, (iii) vegetables/green leafy vegetables, (iv) eggs, (v) meat/poultry/fish, and (vi) milk/dairy products.[9] The DDS was calculated by summing up the number of food groups represented in the child's diet. If parents reported that their child consumes an item from one of the six food groups at least several times in a 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). Other indicators of dietary behavior were measured by the consumption of food items that are available in the study region and are a rich source of iron. These food items are broadly grouped as animal source foods (haem iron) and plant source foods (non-haem iron). The consumption of green leafy vegetables and meat products are the other two outcome variables. 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.

The Food and Agricultural Organization of the United Nations (FAO) reviews studies that show that DDSs are valid indicators for the adequacy of micronutrient (and macronutrient) intake and have significant predictive power to assess nutritional adequacy (FAO, 2007). Studies in different age groups have shown that an increase in individual dietary diversity scores is related to increased nutrient adequacy of the diet. Using the FAO-developed DDS, several studies conducted in developing countries found a positive relationship between DDS and nutrient adequacy among children in Mali (Hatloy et al., 1998), among Filipino children (Kennedy et al., 2007), and in China (Zhao et al., 2017). Dietary diversity has also been shown to be associated with the

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

nutritional status of children such as height-for-age (HAZ) in several resource-poor countries (Arimond et al., 2004).

We used a 30-day recall period to collect data on the household's diet. The DDS is sometimes measured based on a 24-hour recall period because it is possible to ask for specific food items and quantities. The major disadvantage of this approach is that it does not capture food items that are not consumed every day, for instance, animal source products. Thus, the 24-hour dietary information is less likely to describe the typical diet of the households and capture seasonal variation in the diet. For our purpose, the disadvantage of a 24-hour recall outweighs the advantage and we thus opted for a 30-day recall, which is also a commonly used recall period for food consumption. It is not based on individual food items or exact quantities but rather on food groups as described above. The DDS is based on the consumption of food items from a specific food group, and therefore, 30 days recall period works well for the DDS.

Following the WHO recommendation, we measure iron-deficient anemia by hemoglobin levels. Hemoglobin levels were measured using blood samples collected through the finger-prick method. We intend to measure iron-deficient anemia through hemoglobin concentration because iron deficiency is the most common cause of anemia. From a policy perspective, hemoglobin is a relevant measure of anemia because it is more suitable for a scale-up of a screening and information intervention (if it works) and it also measures the impact at the population level regardless of the causes of anemia.

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). *Education outcomes* were assessed by math and reading test scores as well as the child's school attendance. Reading skills were tested on a scale from 0 to 4, ranging from a *child who does not recognize letters* to a *child who 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 the basis, but extended to 13 different exercises at the baseline and 15 at the endline, ranging from a *child does not recognize one-digit numbers* to a *child can solve advanced division problems*. See Krämer, Kumar, and Vollmer (2020) for a detailed description of the cognitive as well as math and reading tests. Finally, the school attendance of the child for the year before the follow-up survey was recorded from the official school attendance register.

We standardized the test scores 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.

2.3.3. Sample description

Table 1 presents the baseline summary of the key characteristics of the sample. The average DDS score and the blood hemoglobin level are 3.9 and 11.03 g/dl, respectively. The cognitive outcomes and test scores are low; for example, the average math score is 4.3 on a scale of 0 to 13, whereas the reading test score is 0.74 on a scale of 0 to 4. Similarly, the block design score and digit forward span score are less than 4. Most children are Hindu in rural areas (98%). Approximately 32 % of the sampled children belong to socially disadvantaged caste groups- Scheduled Caste (SC) or Scheduled Tribe (ST). The average family size is close to 8 members and 43% of the children are female. Coverage by health insurance (38%) and the rate of institutional births (34%) are low compared to the national average. Less than 10% of the household have access to an improved source of sanitation. For school-level variables, the average enrollment rate is about 258 children and on average a typical class has 34 students. Approximately 86% of the sampled children were accompanied by their mothers at the time of the information treatment.

3. Empirical Framework

3.1. The Regression Discontinuity Design (RDD)

The study uses a regression discontinuity design to estimate the causal impacts of the information intervention on dietary behavior, hemoglobin level, cognition, and educational outcomes of children by comparing children who are just below and above the 11.0 g/dl cutoff.

The anemia 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 a hemoglobin level of at least 11.0g/dl are non-anemic. Around the thresholds, the assignment to the intervention is as good as random, since the stochastic error component is continuously distributed over the baseline hemoglobin variable, i.e. the forcing variable. Individuals close to the threshold only differ in treatment status but not in other characteristics.

We use pooled normalized local linear regression (LLR) estimation approach with triangular kernel weights (Lee and Lemieux, 2010). The normalized pooled regression function is as follows[10]

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

ΔY represents the change from 2014 to 2016 of the different outcome variables (diet, hemoglobin, cognitive ability, and education outcomes). We use the 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 ≤ 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 (LATE) around the cutoff.

The bandwidth (h) is selected using 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 and Kalyanaraman, 2012). This method was further developed by Calonico et al. (2019) for clustered data (henceforth

[10] 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.

CCT). Because our data is clustered at the school level, we apply the CCT approach in the local linear regression estimation approach. Since only data points close to the cutoff are included in the analysis, estimates only apply to individuals with hemoglobin values in 2014 that are close to the 10.9 g/dl cutoff. Hence, we measure the LATE for the population close to the cutoff. Instead of LATE, the overall average treatment effect (ATE) can be estimated only with the strong assumption of homogenous treatment effects. In our study, we believe that our information intervention may have heterogenous impacts across subpopulations. For example, the treatment effects may vary by (i) who received the information treatment (any household member versus caretaker); (ii) household characteristics (educated or wealthy households may be more likely to act upon the information treatment); and (iii) the hemoglobin level of the children (severely anemia children may benefit more than mildly anemic children). These factors violate the assumption of homogenous treatment effects and therefore, our estimates should be interpreted as LATE and not as overall ATE.

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 points, i.e. globally and not only for a small bandwidth around the cutoff.

3.2. The Validity of the regression discontinuity design

The RDD is a valid method for causal interpretation only if the following two assumptions are met. First, the forcing variable (the baseline 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, Todd, and Klaauw, 2001; Lee and Lemieux, 2010; Imbens and Lemieux, 2008). It appears that both assumptions seem to be true in our setting. The cutoffs for anemia were set in terms of standard deviations from the mean of a hemoglobin distribution of a reference population (WHO, 2001).

The second assumption is that neither the individuals being studied nor the people who assign treatment (i.e., medical staff) can manipulate assignment to the treatment group. The cutoff level and the knowledge about the information treatment were unknown to households and they did not have any incentive to manipulate the hemoglobin value. Moreover, it is impossible to adjust feeding practices in a way that hemoglobin levels can be precisely determined. Furthermore, another important concern could be that the enumerators may have manipulated the treatment assignment, i.e., they did not provide information when they should have, or they provided information when they should not have. We do not see any incentive for the medical staff to have manipulated the assignment to either of the experimental groups since they received no monetary benefits for the conveyance of the treatment, and also the required effort to implement the nutrition information was very minimal and low-cost. Medical personnel was additionally supervised through spot checks and no irregularities were observed. We believe our rigorous training and supervision protocol is likely to minimize this concern. Since most of the enumerators were medically trained staff, they understood the intervention well, and given their background we believe they are unlikely to manipulate the treatment assignment. In case, control groups (parents of non-anemic children) somehow received the treatment, our estimated effects would be underestimated and biased to zero. We perform several tests to check these two conditions. First, the non-manipulation of the assignment variable 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.

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). [11] Column (7) in Table 2 provides the p-values for the t-test of equality of the means. 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 a large number of t-tests might differ by chance, no statistically significant differences appear above and below the cutoff, indicating randomness around the cutoff.

4. Results

4.1. Graphical Illustration of the results

Figure 3 illustrates the potential discontinuities by plotting the change in our outcome variables from 2014 to 2016, against the normalized hemoglobin values in

[11] 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.

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 we show observations within the bandwidth that is selected by the CCT procedure.

If the information treatment were effective in improving the outcomes, one would see a jump at point 0 of the x-axis. In panel A none of the graphs show a discontinuity at the cutoff, instead, all data points evolve smoothly at the cutoff, indicating that the information intervention had no impacts on any of the outcomes related to feeding practices, hemoglobin, cognition, and education. Similar results emerge when we focus on observations close to the cutoff point (Panel B). There might be slight discontinuity for some of the cognitive and educational outcomes, such as in the backward digit-span test, the block design test, and the cognitive index and school attendance. Furthermore, in some cases, we also note the wide confidence intervals due to large data variability and for a few cognitive outcomes, the observed discontinuities are in an unexpected direction.

4.2. Main Results

Estimation results for Equation (1) are presented in Table 3 for feeding practices and anemia and in Table 4 for cognitive and educational outcomes. In Panel A, estimation results are presented for the data-driven bandwidth selected by the method proposed by CCT, and Panel B shows estimates for arbitrarily chosen bandwidths (0.5, 1.0, 2.0, and 2.5).

The results from the discontinuity graphs can broadly be confirmed by the regression analysis. For the feeding practice outcomes (columns 1-3, Table 3), 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 3), 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. However, the effect is only statistically significant for very small bandwidths (0.4 and 0.5) and does not stay robust across specifications in Panel B when bandwidths range from 1 to 2.5.[12]

For the cognitive measures, a few point estimates are statistically significant, but most are not (columns 1-6, Table 4). Based on the estimates using the CCT bandwidth (Panel A), informing the parents of their 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 0.48 standard deviations, compared to the mean in 2014. 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 0.31 standard deviations (significant at the 10% level). The coefficient for *block design* remains statistically significant for the bandwidths of 0.5, 2.0, and 2.5. The cognitive index is statistically significant only for the 0.5 bandwidth in Panel b of Table 4. However, for other indicators of cognitive outcomes, most of the coefficients show an unexpected negative sign. Since estimates for the different cognitive tests are not consistent, i.e. only one cognitive test shows 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. Finding an effect on cognition but not on feeding practices and hemoglobin is puzzling, 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 4), the information intervention had a statistically insignificant impact.

[12] 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 3.

Together, these results indicate that information intervention about anemia risk and how to combat it had no statistically significant effects on the health and cognitive outcomes of the children. However, we would caution that some of the coefficients are imprecisely estimated and have large standard errors. This could be due to low power, and therefore, these results should be interpreted with caution. The power calculation in Table 8 does indicate that our study was powered to detect relatively large effects on the DDS and hemoglobin levels. Nonetheless, it is reassuring though that increase in the bandwidth does not only reduce standard errors as they should in almost all cases also reduce point estimates quite close to zero. While this is no proof, it gives us some confidence in our null result. We believe that this finding is important. It is entirely possible that this intervention has zero effect for the reasons that we have discussed in the last section and statistical analysis supports this conclusion because our analysis does not reject the null hypothesis of no effect.

4.3. Additional specifications

We examine the sensitivity of the results in Tables 3 and 4 to different specifications. Tables 5 and 6 show estimates of additional specifications of equation (1); Panel A shows estimates with a rectangular kernel. In Panel B we include a set of control variables and Panel C shows results for the application of a polynomial of order two on observations with the CCT bandwidth, Panel D shows results for global estimates for different higher-order polynomials, and finally, Panel E excludes hemoglobin value of 10.9 and 11g/dl.[13] In general, the results are very similar to the findings in Tables 3-4; none of the coefficients in Table 5 are statistically significant, indicating that the nutrition information intervention had no impacts on feeding practices and hemoglobin levels. Results in Table 6 confirm the findings of Table 5; the information intervention had no effects on cognitive and educational outcomes in the modified model of equation (1).

4.4. Power

The RDD estimates are generally considered more reliable within a small bandwidth around the cutoff point but the bandwidth restriction leads to exclusion of many observations and, in turn, to less power to the analysis. Furthermore, the

[13] Regression underlying Panel E are described and discussed in the robustness checks.

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 8).[14] In the last column of Table 8, we compare these MDE to effect sizes found in Luo et al. (2012) and Krämer, Kumar, & Vollmer (2020). At least for some of the outcomes, which we consider to be comparable (hemoglobin, math, and reading scores), the MDE that can be found in this study is 2 to 3 times larger. With a bandwidth of 2.5 in this study, this study is powered to detect a change in hemoglobin levels of 0.551 g/dl. For comparison, we found a statistically significant effect size of 0.185 g/dl in the companion paper and Luo et al. (2012) found a statistically insignificant effect size of 0.275 g/dl in one of their information interventions. In contrast, in a multivitamin supplementation intervention that was used by Luo et al. (2012) as a benchmark, effect sizes ranged from 0.202 to 0.416 g/dl. Estimates for math and reading score in the companion paper ranged from 0.2-0.4 standard deviations. This indicates that our study is powered to detect a large effect on test scores: an effect in the range of 0.6 standard deviations. The power analysis indicates that insufficient power might be a reason for the non-detection of an effect of the intervention and we are only able to detect a relatively large effect. Power for this analysis is somewhat low, however, the absence of statistical significance alone cannot be interpreted as evidence for a zero effect of the nutrition information.

5. Robustness and heterogeneity

5.1. Robustness checks

First, the model in equation (1) includes ΔY as the outcome variable to control for the baseline value of the outcome variable. However, as discussed before, this may suffer from low power. Therefore, to increase power, we reestimate equation (1) and use the endline value of Y as the outcome instead of ΔY. The smallest MDE that can be found without taking differences in outcomes are presented in Table A3. The MDE in Table A3 is slightly smaller than the MDE in Table 8. The estimated effect from this analysis is similar to the findings in Tables 3 and 4 (results are shown in Appendix Tables A4 and A5.).

[14] The online appendix shows the method used to calculate MDE.

Second, our results would be biased if the enumerators made an error in implementing the treatment around the cutoff. Although enumerators were strictly advised during the training to adhere to the treatment cutoff protocol, one can imagine a situation wherein enumerators delivered the information treatment to non-anemic children who were close to the cut-off. This is highly unlikely, but we 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 excluding the children who were near the cutoff are shown in Panel E of Tables 5 and 6 and are in line with the previous estimates.

Third, the data used for this evaluation comes from a randomized trial conducted by Krämer, Kumar, and Vollmer (2020), which evaluated the effects of using iron-fortified iodized salt in the Indian school-feeding program (henceforth: *school intervention*). In general, the fact that another nutrition intervention was evaluated with the same dataset does not bias the findings of this study since the *school intervention* was randomized at the school level and the children that were treated by the iron-fortified salt intervention were equally distributed across the hemoglobin cutoff.[15] Table 1 shows the share of children that belonged to the treatment group in the *school intervention*, just above and below the threshold. 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 this covariate did not make much of a difference in estimation results (results available upon request).

5.2. Heterogeneity: Did mothers receive the information?

It is also likely that the treatment effects depend on whether the nutritional information was directly shared with the mother versus other members of the household. The previous study shows that decisions on child health and nutrition are

[15] The school intervention provided double fortified salt for the preparation of school lunch. It started in August 2015 and continued for a year. The year-long school intervention increased hemoglobin levels and reduced anemia prevalence by 20%. However, the improved anemia status of school children did not lead to an improvement in the cognitive abilities or test scores of children. While exploring the heterogeneity in effects, we found significant treatment effects on test scores for children with higher school attendance (Krämer, Kumar, Vollmer, 2020).

mostly made either by mothers or grandmothers (Thomas, 2011). Our medical survey collected mothers' anthropometric data as well while drawing the blood sample from the children. Mothers for whom we have the anthropometric data suggest that mothers were present at the time of blood draw of their children. We use this information to conduct the heterogenous analysis by whether the information treatment was received by mothers or other household members. Based on mothers' anthropometric data, we estimate the fraction of mothers who were given the information treatment to be about 86% in our sample.

The heterogeneity analysis estimates equation (1) for the subgroup of children that were potentially accompanied by their mothers. Results of this regression are shown in Table 7 for the feeding practice, hemoglobin, and cognitive and educational outcomes. The results are similar to previous findings. There is no statistically significant effect for any of the specifications on feeding practices. For the smaller bandwidths (CCT and 0.5), hemoglobin levels appear to be statistically significant at the 10% level of significance; however, the estimates are not robust to the larger bandwidths and different functional forms. 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. We do not find any evidence of heterogeneous effects. These findings indicate that the treatment effects do not vary according to whether the mother or the other members of the household received the nutrition information.

5.3. Attenuation bias

Several factors may attenuate the estimated effects in this study. In case, the treatment compliance was not perfect, i.e., if the parents of the anemic children did not receive the treatment because enumerators failed to deliver information, then this might lead to an attenuation bias. Nonetheless, we consider this risk to be rather low since the hemoglobin testing drew a lot of attention in the village and quite often villagers gathered at the testing site out of curiosity. This would have ensured that enumerators relayed information to the parents of anemic children. According to our estimate, about 86% of the mothers of anemic children were given the information treatment directly from the enumerators and for the remaining 14%, other members of the households were informed of the dietary suggestions. This suggests that failure to deliver the information to parents of anemic children is not a major concern. The main reason for mothers not receiving the information treatment was their unavailability at home at the time of testing and information sharing. To avoid revisit and reduce survey expenses, the enumerators shared the information with other adult members of the household.

The drawback of crowd gathering is that it may have led to contamination of the control groups. The contamination concern could be that parents of non-anemic children sometimes might have received or taken up the treatment. This is particularly relevant for non-anemic children close to the cut-off. It may happen if enumerators deliberately delivered information to the parents of non-anemic children or due to spillovers. Parents of non-anemic children may have learned about the information from the treated households in the villages (spillovers). As mentioned before, enumerators were trained to deliver information to caregivers of officially classified anemic children only. This would have ensured that enumerators were very unlikely to manipulate the treatment. As stated above, results from the donut regressions that exclude the children near the cut-off are qualitatively similar to our main findings, indicating a minimal chance of contamination. However, the spillover of information from treatment to control groups could happen. These circumstances imply that assignment to the treatment might not be a deterministic function of the forcing variable but instead, the probability of assignment to the treatment increases at the cutoff. In such a setting, a fuzzy RDD can potentially be applied. Unfortunately, we do not know which individuals were affected by this potential imprecision in the conveyance of the treatment, such that we are not able to perform a fuzzy RDD and conduct a sharp RDD analysis. Regardless, in all cases when parents of anemic children did not receive information or when parents of non-anemic children received information or information spillover, the estimated effects would be underestimated.

6. Discussion

Using the RDD method, this study investigates the impact of a nutrition information intervention on feeding practices, hemoglobin, and cognitive and educational outcomes of young children in rural Bihar, India. After two years of intervention, we find little evidence of statistically significant effects on any of the measured outcomes, indicating that households are resistant to adjust their dietary behavior after acquiring new information about their children's health. Our findings are consistent with previous findings that light-touch one-time intervention that educates parents about children's health risk may not affect health behavior especially in the context of micronutrient deficiencies which are mostly non-fatal and symptoms are not visible to caretakers (Luo et al., 2012; Wong et al., 2014).

These null findings should be interpreted with caution as some of the coefficients are imprecisely estimated. The large standard errors could be due to low power as our study was powered to estimate large effects on the outcome variables. It is reasonable to assume that our information intervention has zero effect for the reasons outlined below and statistical analysis supports this conclusion because our analysis does not reject the null hypothesis of no effect. Given the large standard error in this study, we recommend that future studies use large samples to reduce the uncertainty in the results.

These findings help us draw several important conclusions in the context of the effectiveness of parental health education programs on children's health in developing countries. *First*, in addition to the low power alluded to before, another potential reason for our findings might be the low intensity of the intervention. Parents were educated about anemia and were provided the dietary knowledge only once in our study. This information intervention was not a sustained and ongoing activity. For the parental education program to be successful, the intervention has to be intensive, interactive, and should occur multiple times (Wong et al., 2014). When parents were informed about their children's anemic status through a letter in China, the health education program had no impact on hemoglobin levels (Luo et al., 2012). Therefore, for the health and dietary education program to be effective, the intensity, frequency, and interactivity of the program are crucial (Wong et al., 2014). A one-time light-touch information campaign might not be sufficient to nudge households to change their behavior. *Second*, it is possible that positive treatment effects, if any, only occurred immediately and had diminished after two years. Parents may have adjusted the diet and adopted healthy behavior immediately after the intervention but may not have continued with these practices until the follow-up survey. Thus, a crucial disadvantage of the light-touch parental education program such as ours is that it may reduce the information hurdle in the short run but does not provide comprehensive and lasting information needed for long-term behavior change. We speculate that a continuous reminder on a sustained basis may have nudged the parents to modify their behavior.

Third, the effectiveness of information on parental behavior also depends on the degree of intrahousehold concordance in preferences and women's role in household decision-making (Dupas, 2011). If mothers' preferences differ from fathers regarding the production of their child's health, then educating one parent may not be effective. Information treatment could also have limited impacts if one parent decides purchases for daily household needs. There is mixed evidence on the role of women in decision-making about visiting markets or purchase of daily needs in India. For example, only 22.5% of females alone participated in taking decisions about purchases for daily needs in rural Odisha, India (Routray et al., 2017). Concerning women's freedom of movement, about 50% of women were allowed to go to the market alone or places outside their village (IIPS, 2017). From another household survey that one of the authors conducted in another district of Bihar, the study found that mothers mostly make decisions about what to cook and what children eat in our study area.[16] In case, the anemia-related information was not shared with the male member of the family or there is discordance in preferences between mother and father, the information intervention may not work. For example, the information campaign was more effective when a health message was delivered to both parents compared with either of them receiving it alone in Kenya (Dupas, 2011).

Fourth, another important consideration could be that anemia symptoms are non-fatal and cannot be easily discerned by parents. When symptoms are non-fatal, parents may procrastinate taking any immediate remedial action because they do not see the benefits immediately, but the cost is incurred now. While the degree of suffering from a nutritional disorder might not be perceived as very severe, mostly because the cause and symptoms are more subtle and salient, the costs of changing nutritional habits are high.

Fifth, we also speculate that second-grade children experience a spurt of physical growth and require more energy and micronutrients including iron in their growing years. This is likely to attenuate the effect of the treatment on hemoglobin if any. Lack of education of the message recipient may further worsen the processing of information.

Sixth, our local knowledge and secondary data from government sources show that the study region is a major producer of iron-rich vegetables and fruits, and therefore, these food items should be easily available. The WHO (2001) states that improvements in nutrition build on these three pillars: the availability of micronutrient-rich food, the

[16] 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.

financial accessibility to those food items as well as a change in feeding practices (i.e. utilization). However, our intervention only addresses the latter pillar. Our household surveys did not collect data on the actual availability and affordability of iron-rich vegetables and fruits. However, the follow-up survey included two questions that indirectly allude to the availability and financial accessibility of food items in the study regions. In the follow-up survey, nearly 48% of parents reported that they were *often* not able to afford to feed their child a balanced meal in the last 12 months and 35% reported that they were *often* not able to feed their child a balanced meal because only a limited variety of food was available in their regions. These data suggest that we cannot rule out with certainty that seasonal or occasional lack of availability and affordability especially for poorer households could not have constrained parents to adjust the dietary behaviors. However, these numbers should be interpreted with caution as the survey questions were about balanced meals and not about iron-rich vegetables per se, and hence, the reliability of these responses might be limited. This is a subjective measure and at best provides suggestive evidence that affordability could have been an issue at least in a few months in a year.

Finally, deep-rooted beliefs, social norms, inertia, habits, and culture make individuals very reluctant about changing their dietary behavior, and changing behaviors require major changes in thinking and action (Banerjee and Duflo, 2012). Evidence from other studies, however, shows that even when health products are easily available and accessible, individuals do not necessarily respond to health information or at least not as much as one would expect. For example, 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). In the context of nutrition, Banerjee, Barnhardt, and Duflo (2018) 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. This indicates that changing dietary patterns is complex and difficult and - even if availability and affordability are ensured - that information alone especially the light-touch, even when a nutritional disorder is revealed, might not be sufficient to ensure adoption of healthy behavior.

In summary, the effectiveness of information treatment depends on the type and intensity of information, frequency, who received the information, type of illness, and gender and education of the information recipient (Miguel and Kremer, 2007; Dupas, 2011a). Unfortunately, we are not able to disentangle the true underlying mechanism for the null findings of the information treatment in our experiment. 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, 2011). The success of social norms-based intervention implemented at the community level holds promise. The Reduction in Anemia through Normative Innovations (RANI) is one such intervention currently underway in India to reduce anemia among women of reproductive age. Future studies should consider these factors while designing health information campaigns.

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Figure 1: Distribution of baseline hemoglobin values

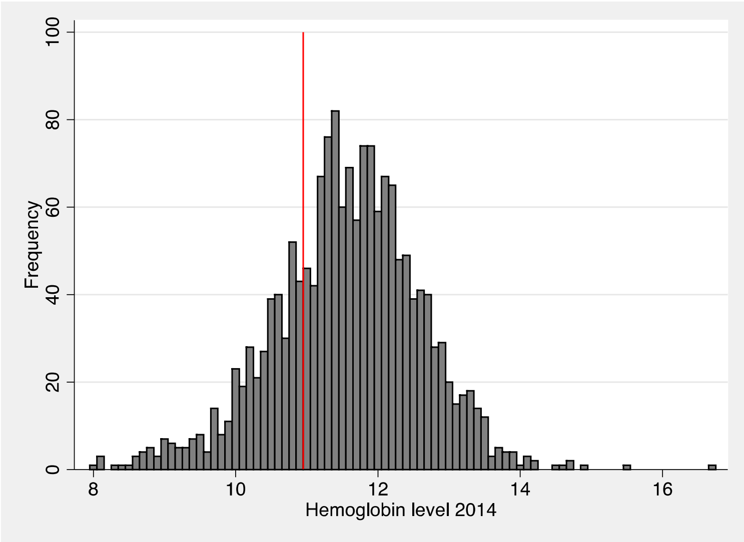


Figure 2: Timeline of data collection and the treatment

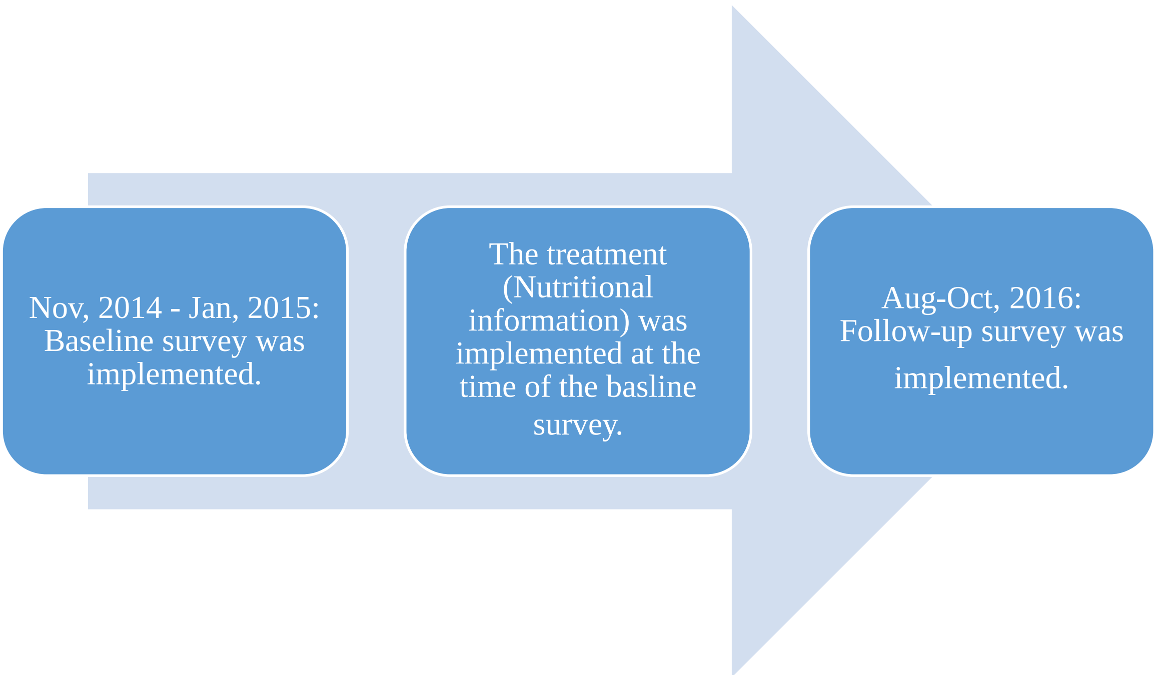
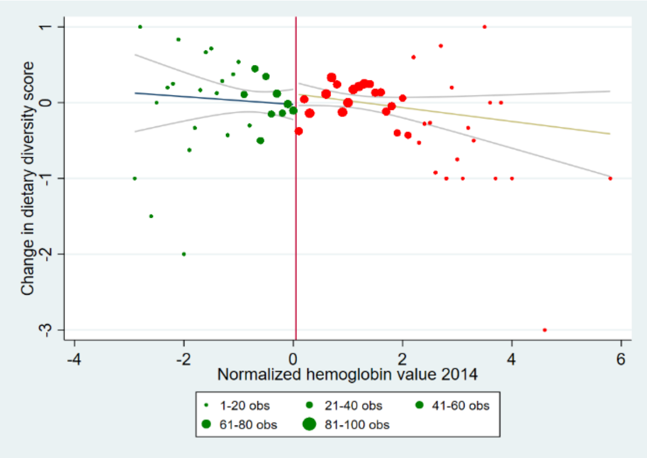


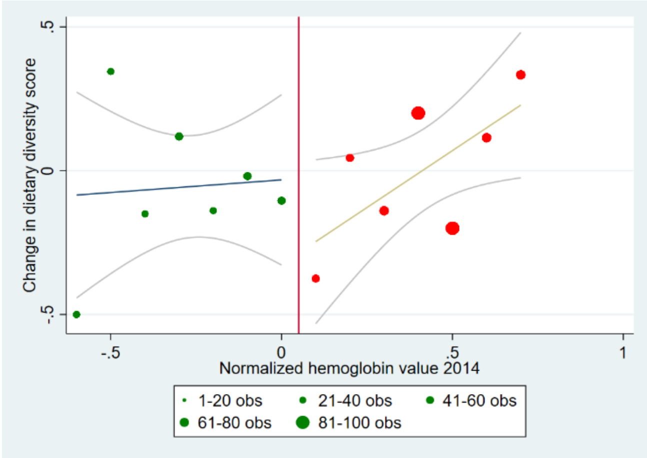
Figure 3: Discontinuity graphs

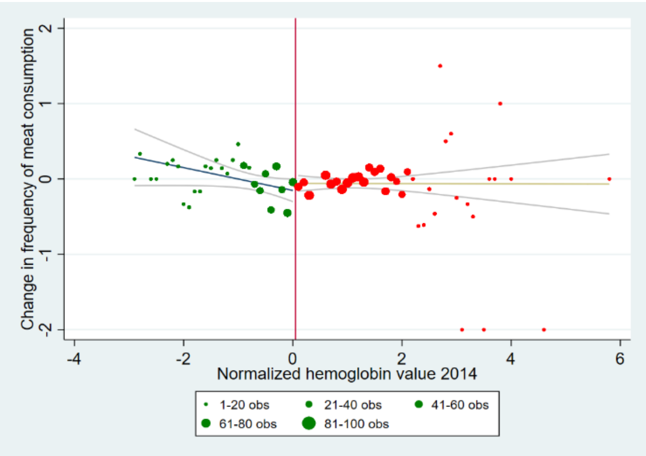
A. Feeding practices

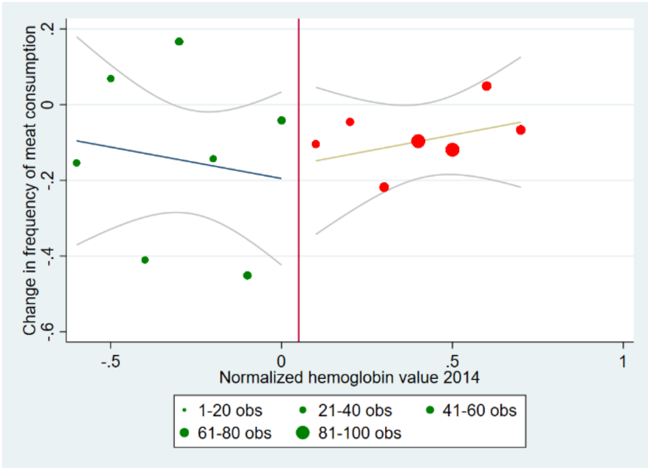
Panel A: Globally

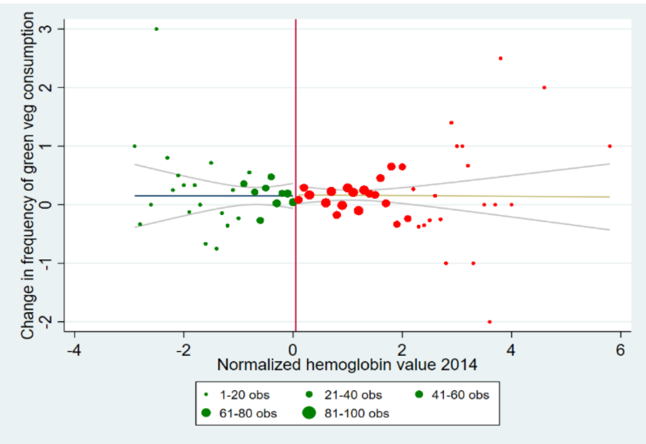
Panel B: Close to the cutoff

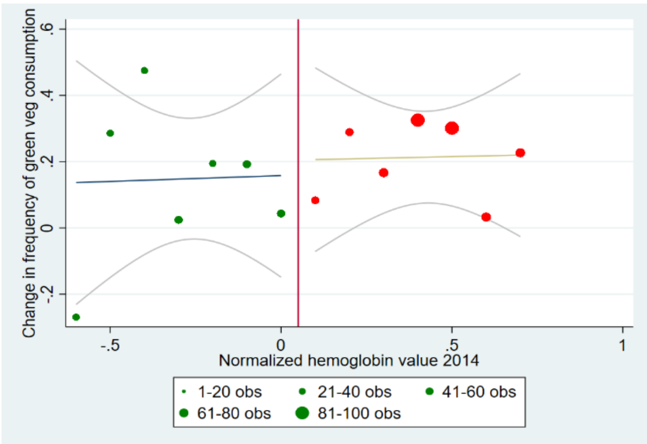








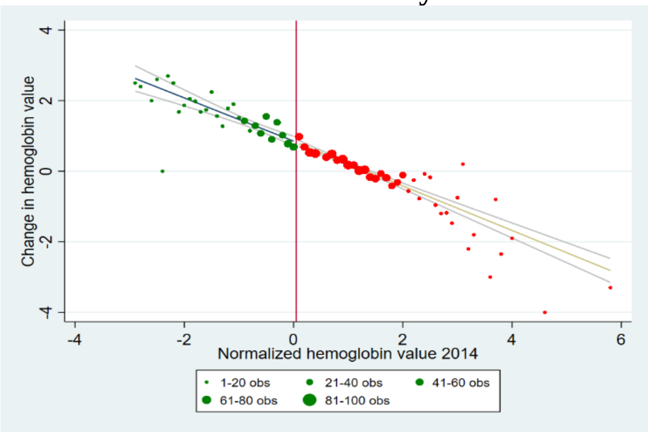




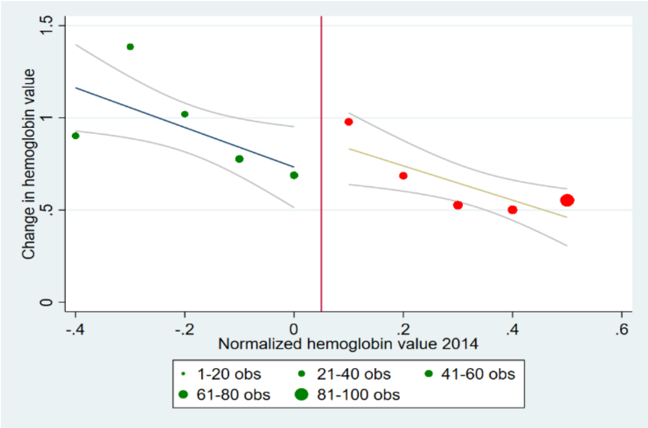
Note: Change in frequency outcome variables is averaged over each discrete value and plotted against the respective discrete values of the normalized hemoglobin values from 2014. The size of the markers represents the no. of observations within each discrete hemoglobin value. A regression line is fitted to the data points and the grey lines show the confidence interval

B. Anemia outcomes

Panel A: Globally



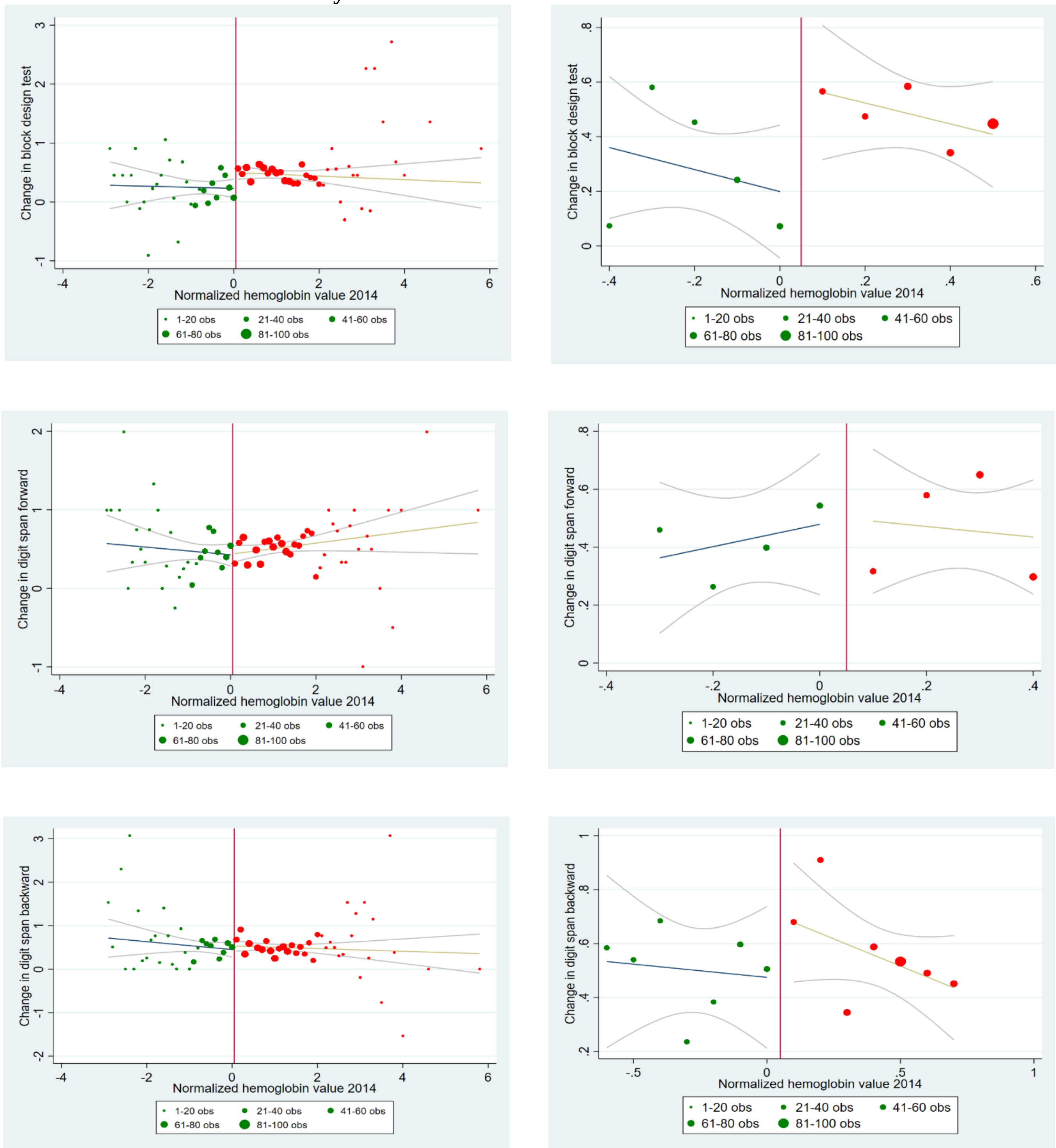
Panel B: Close to the cutoff



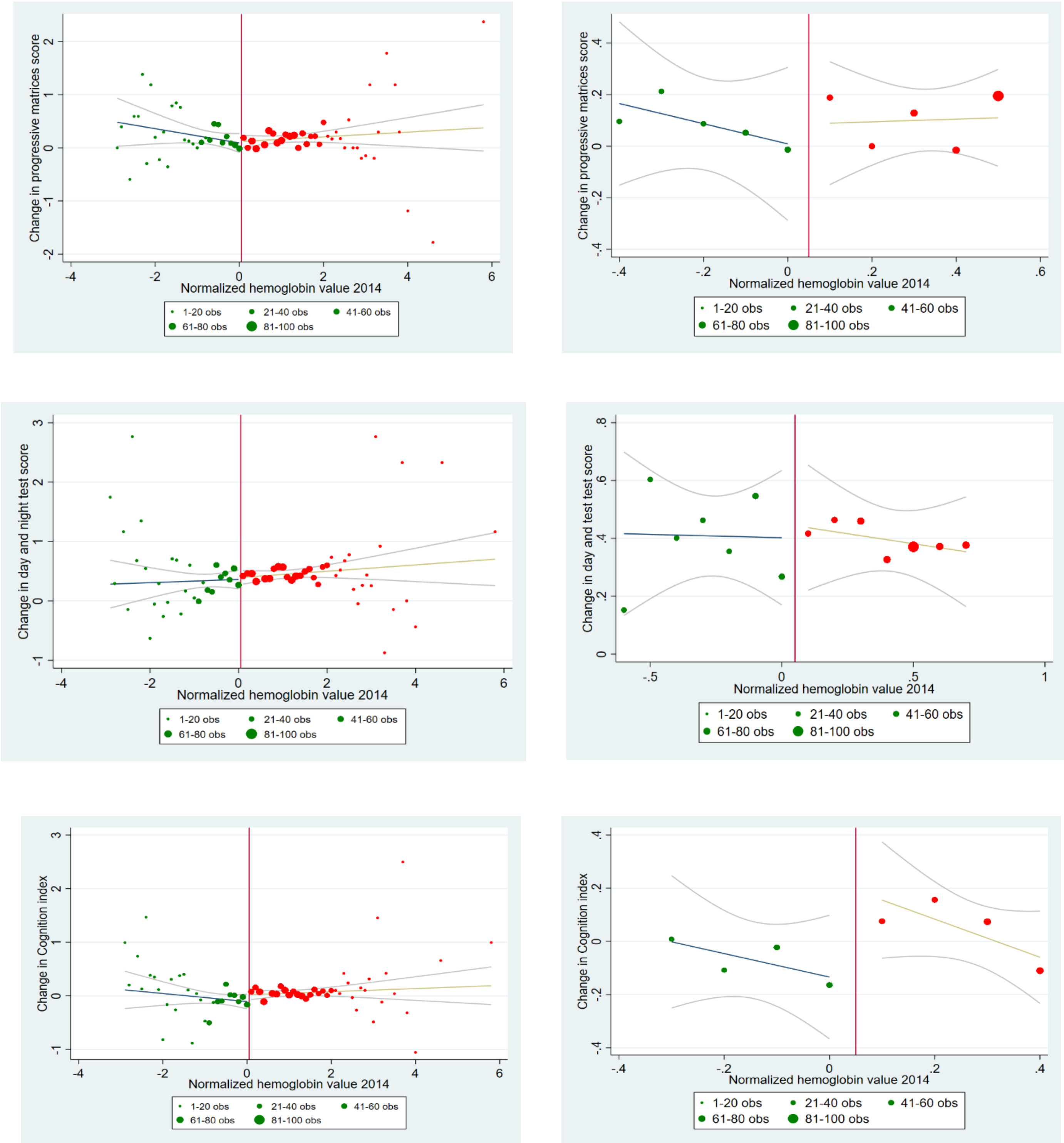
Note: Change in hemoglobin value is averaged over each discrete value and plotted against the respective discrete values of the normalized hemoglobin values from 2014. The size of the markers represents the no. of observations within each discrete hemoglobin value. A regression line is fitted to the data points and the grey lines show the confidence interval.

C. Cognitive and education outcomes

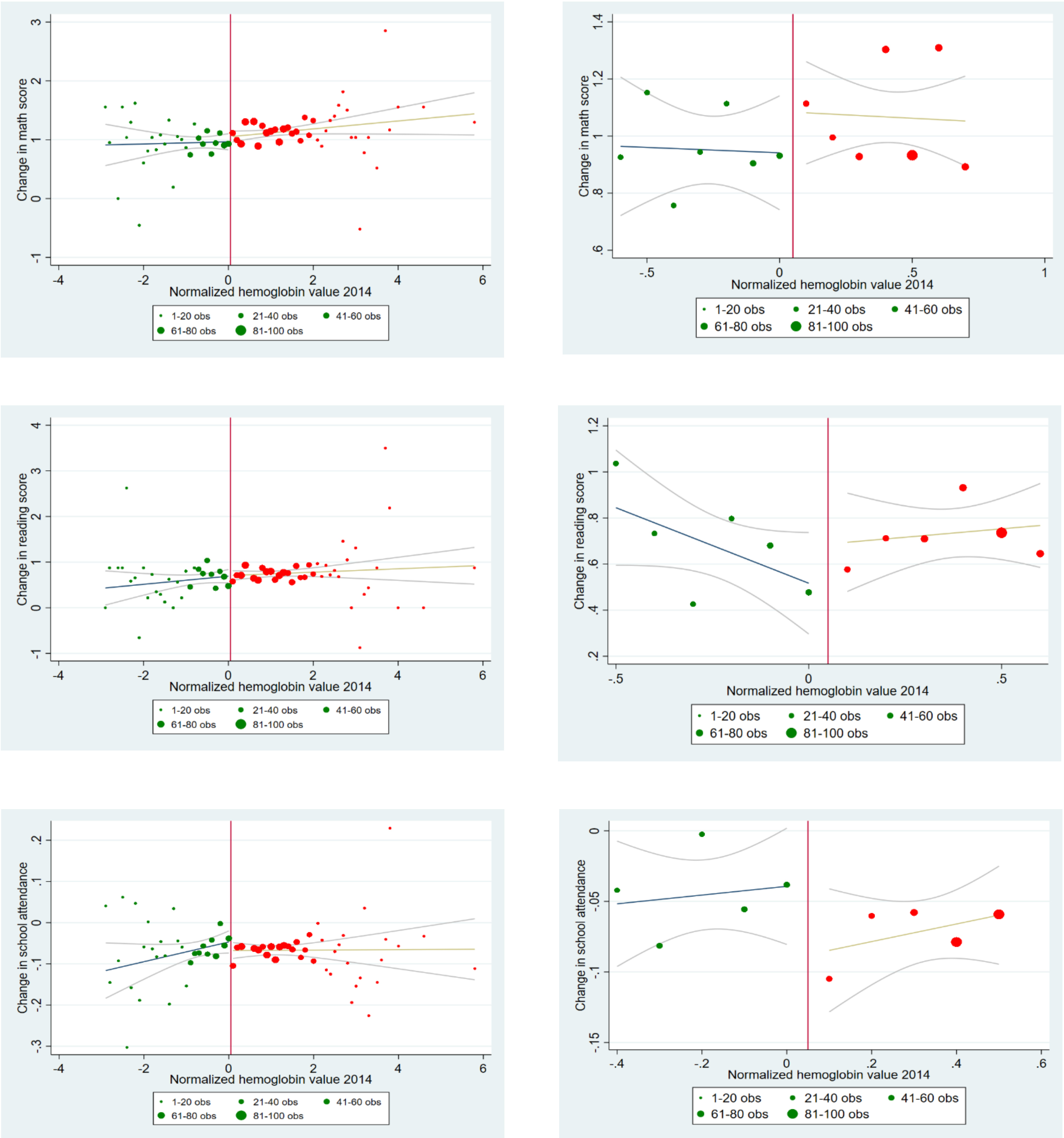
Panel A: Globally | **Panel B: Close to the cutoff**



Note: Change in outcome variables performance is averaged over each discrete value and plotted against the respective discrete values of the normalized hemoglobin values from 2014. The size of the markers represents the no. of observations within each discrete hemoglobin value. A regression line is fitted to the data points and the grey lines show the confidence interval.



Note: Change in outcome variables is averaged over each discrete value and plotted against the respective discrete values of the normalized hemoglobin values from 2014. The size of the markers represents the no. of observations within each discrete hemoglobin value. A regression line is fitted to the data points and the grey lines show the confidence interval.



Note: Change in outcome variables is averaged over each discrete value and plotted against the respective discrete values of the normalized hemoglobin values from 2014. The size of the markers represents the no. of observations within each discrete hemoglobin value. A regression line is fitted to the data points and the grey lines show the confidence interval.

Table 1: Summary statistics of the full sample

	Mean	SD	N
	(1)	(2)	(3)
Outcome variables			
Feeding practices			
Dietary Diversity Score	3.85	1.18	517
Frequency of meat consumption	1.79	0.85	516
Frequency of green veg consumption	3.41	1.03	509
Hemoglobin			
Hemoglobin	11.02	0.29	517
Cognition			
Block design	3.58	2.2	506
Digit span forward	3.99	0.97	507
Digit span backward	0.97	1.24	507
Progressive matrices	4.85	1.56	507
Day and night	5.06	3.3	507
Cognitive index	-0.09	0.93	506
Education			
Math	4.34	3.65	507
Reading	0.74	1.02	507
School attendance	0.79	0.16	490
Covariates			
Treatment group from school intervention	0.54	0.50	517
Religion (Muslim HH)	0.02	0.15	517
SC/ST	0.32	0.47	517
Rural HH	0.98	0.14	517
HH size	7.47	3.14	517
Years schooling father	5.01	4.77	508
Years schooling mother	1.45	2.91	514
Asset index	-0.12	0.93	509
Institutional delivery	0.34	0.47	514
Health insurance	0.38	0.49	512
Diarrhea	0.05	0.21	517
Improved sanitation	0.07	0.26	517
Male child	0.43	0.50	517
Help with homework	0.15	0.36	512
Time physical care	45.78	25.29	517
School meetings	0.65	0.47	516
Father at home	0.89	0.32	516
Distance to school	10.21	6.12	517
Number of meals	3.07	1.03	517
Cut meals	0.82	0.38	517
Iron supplementation	0.19	0.39	510
Maternal health knowledge	0.37	0.48	517
Total enrollment	258.91	153.5	517
Class size	33.61	17.06	517
Student teacher ratio	38.62	11.6	517
Calories of MDM per child	68.22	21.82	517
Iron in MDM per child	0.77	0.29	517

Notes: This table presents baseline summary statistics as well as p-values for the 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: Sample statistics and covariates balance (Hemoglobin sample)

	Left side 10.5-10.9 g/dl hemoglobin			Right side 11.0-11.4 g/dl hemoglobin			
	Mean	SD	N	Mean	SD	N	P-value
	(1)	(2)	(3)	(4)	(5)	(6)	(7)
Outcome variables							
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***
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
Religion (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
Rural HH	0.98	0.16	204	0.98	0.13	313	0.492
HH size	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	153.7	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 the 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 3: 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 4: 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 5: 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: 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: 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: 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 7: 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	-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	-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. Approximately 86% of the anemic children were accompanied by their mothers at the time of the treatment.

Table 8: Minimal detectable effects for different bandwidth

	Bandwidths						Effect size in other nutrition interventions
	Bw 0.3	Bw 0.5	Bw 1.0	Bw 1.5	Bw 2.0	Bw 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, Kumar, and Vollmer (2020).
2 Effect size from the evaluation of the school intervention by Krämer, Kumar, and Vollmer (2020) 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.

Appendix

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) calculates an 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 an RCT setting by the factor 2.17. We assume power of $\kappa = 80\%$ and a significance level of $\alpha = 5\%$. Standard deviation σ, number of clusters J, the fraction of treated individuals P, n the average number of individuals 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.

Table A1: 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 Hb value 10.9 and 11)	-0.014	0.054	-0.562	0.073
	(0.526)	(0.306)	(0.765)	(0.236)

Notes: N denotes the 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 A2: 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)	(0.041)
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 A3: Minimal detectable effects for different bandwidth (no difference in outcome variable)

	Bandwidths						**Effect size in other nutrition interventions**
	Bw 0.3	Bw 0.5	Bw 1.0	Bw 1.5	Bw 2.0	Bw 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, Kumar, and Vollmer (2020).
2 Effect size from the evaluation of the school intervention by Krämer, Kumar, and Vollmer (2020) 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.

Table A4: The average treatment effect of nutrition information on child health and feeding practices (Endline outcome)

	Dietary diversity score	Frequency of meat consumption	Frequency of green veg consumption	Hemoglobin
	(1)	(2)	(3)	(4)
A. Main results				
Optimal Bandwidth (CCT)	0.676**	0.241	-0.118	-0.469**
	(0.224)	(0.163)	(0.196)	(0.218)
Bandwidth	0.3	0.6	0.7	0.4
N	422	632	733	517
B. Alternative bandwidths				
Bandwidth 0.5	0.505**	0.262	-0.123	-0.335**
	(0.203)	(0.174)	(0.243)	(0.200)
N	543	543	543	517
Bandwidth 1.0	0.185	0.137	-0.112	-0.105
	(0.167)	(0.126)	(0.161)	(0.145)
N	1,022	1,022	1,022	969
Bandwidth 2.0	0.020	0.045	-0.001	-0.046
	(0.125)	(0.088)	(0.125)	(0.109)
N	1,606	1,606	1,606	1,509
Bandwidth 2.5	0.012	0.042	0.017	-0.015
	(0.113)	(0.079)	(0.116)	(0.102)
N	1,708	1,708	1,708	1,609
C. Rectangular Kernel	0.719	0.207	-0.051	-0.193
	(0.213)	(0.157)	(0.180)	(0.193)
D. With controls	0.676	0.241	-0.118	-0.469
	(0.224)	(0.163)	(0.196)	(0.218)
E. Local polynomial (2nd order)	0.452	0.199	-0.150	-0.320
	(0.205)	(0.145)	(0.212)	(0.205)
F. Global polynomial regressions				
Polynomial 1st order	-0.078	0.006	-0.007	0.011
	(0.095)	(0.069)	(0.103)	(0.093)
Polynomial 2nd order	0.079	0.055	0.023	0.004
	(0.144)	(0.100)	(0.1400	(0.115)
G. Donut (excluding Hb value 10.9 and 11)	0.103	0.034	-0.075	-0.052
	(0.328)	(0.163)	(0.240)	(0.276)

Notes: Dependent variable: Outcome variables from the follow-up survey. 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. Column 4 has results similar to Table 3 because hemoglobin is the forcing variable. *, **, *** denote significance at the 10%, 5% and 1% level, respectively.

Table A5: Average treatment effect on cognition and education (Endline outcome)

	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	-0.037	-0.099	-0.137	0.059	-0.038	-0.094	0.057	0.101	0.024
	(0.157)	(0.207)	(0.189)	(0.134)	(0.211)	(0.193)	(0.217)	(0.250)	0.042)
Bandwidth	0.8	0.5	0.7	0.8	0.6	0.6	0.7	0.6	0.5
N	775	514	691	775	602	602	691	602	504
B Alternative bandwidth									
Bandwidth 0.5	-0.071	-0.099	-0.061	0.011	-0.035	-0.076	0.125	0.176	0.024
	(0.191	(0.207)	(0.228)	(0.167)	(0.231)	(0.212)	(0.282)	(0.282)	(0.042)
N	514	514	514	514	514	514	514	514	504
Bandwidth 1.0	-0.017	-0.011	-0.110	0.073	0.034	-0.011	0.053	-0.003	0.032
	(0.143	(0.131)	(0.153)	(0.118)	(0.146)	(0.141)	(0.176)	(0.174)	(0.028)
N	955	955	955	955	955	955	955	955	940
Bandwidth 2.0	-0.046	0.083	-0.051	0.064	0.039	0.025	0.015	0.009	0.012
	(0.113)	(0.094)	(0.114)	(0.088)	(0.110)	(0.106)	(0.130)	(0.135)	(0.021)
N	1,488	1,488	1,488	1,488	1,488	1,488	1,487	1,488	1,468
Bandwidth 2.5	-0.062	0.070	-0.047	0.057	0.031	0.013	0.006	0.036	0.007
	(0.107)	(0.086)	(0.106)	(0.082)	(0.102)	(0.098)	(0.121)	(0.125)	(0.019)
N	1,584	1,584	1,584	1,584	1,584	1,584	1,583	1,584	1,564
C Rectangular kernel	-0.018	-0.212	-0.176	0.094	-0.046	-0.127	-0.028	-0.021	0.033
	(0.150)	(0.190)	(0.171)	(0.121)	(0.191)	(0.178)	(0.181)	(0.219)	(0.040)
D With controls	-0.037	-0.099	-0.137	0.059	-0.038	-0.094	0.057	0.101	0.024
	(0.157)	(0.207)	(0.189)	(0.134)	(0.211)	(0.193)	(0.217)	(0.250)	(0.042)
E Local polynomial									
2nd order	-0.067	-0.043	-0.067	0.061	0.019	-0.005	0.091	0.075	0.032
	(0.182)	(0.161)	(0.196)	(0.139)	(0.176)	(0.166)	(0.230)	(0.245)	(0.032)
F Global polynomial regressions									
Polynomial 1st order	-0.105	0.027	-0.070	0.016	-0.016	-0.045	-0.044	0.012	-0.016
	(0.094)	(0.077)	(0.093)	(0.074)	(0.091)	(0.090)	(0.111)	(0.117)	(0.016)
Polynomial 2nd order	-0.058	0.122	0.039	0.068	0.079	0.072	0.058	0.071	0.019
	(0.120)	(0.100)	(0.128)	(0.096)	(0.120)	(0.114)	(0.138)	(0.145)	(0.024)
G Donut (excluding	-0.060	-0.270	-0.311	0.120	0.026	-0.046	0.039	-0.029	0.046
Hb value 10.9 and 11)	(0.232)	(0.181)	(0.220)	(0.156)	(0.191)	(0.181)	(0.241)	(0.297)	(0.040)

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.