A Statistical Analysis of Children's Health Inequality in Bangladesh

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Abstract

This study employs the Gini coefficient to assess health inequality among children of age 6-59 months using hemoglobin (Hb) levels data from the Bangladesh Demographic and Health Survey (BDHS) 2011. To undertake statistical inferences about the Gini coefficient, we calculate the statistic and its variance estimate using the stratified sampling design. Results suggest that the inequality in children's health using the Hb level is well above the naturally arising inequality that might be expected for a corresponding healthy population, and inequality in children under two years of age group is worse. In addition, our descriptive statistics analysis shows that more than half of the children suffer from low level of Hb or anemia, and the age of a child, place of residence and wealth status of the household are significant determinants of anemia.

JEL Classification: I14; C14; C40

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1. Introduction

This paper considers the hemoglobin (Hb) levels among children of age 6-59 months to assess the health inequality in Bangladeshi children by employing the Gini coefficient (G). The Gini coefficient is a widely known summary measure of inequality that has long been extensively used to measure inequality in income and consumption. Recently, the measure is often being used to measure inequality in other wellbeing indicators, such as education, health, and longevity; see, for example, Hoque and Clarke (2015), and citation therein.

The Hb level, measured in grams per deciliter (g/dl), is primarily used to define whether or not an individual is suffering from anemia; a global health problem affecting about two billion people or over 30% of the world's population, with the highest prevalence among preschool-age

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children (<5 years) of over 47% (e.g., Kotecha, 2011). Our adoption of this measure as an indicator of wellbeing recognizes its importance in understanding the health status of participants of the future labour market and contribution to economic and social outcomes in Bangladesh.

Health inequality may be defined as the variation in health status across individuals or groups in the population. The causes of such variations are, in most cases, complex and rooted in broader social injustice (Khan *et al*., 2011), which are not the focus of our study. We consider health inequality, above a naturally arising level of inequality in a healthy population, as a serious socioeconomic concern that may undermine developmental efforts. Therefore, measuring and monitoring the inequality is important as it feeds information to the policy makers.

A number of studies detailed the prevalence, causes and effects of low levels of Hb among pre-school age children in developing countries; e.g., Singh and Patra (2014), Ayoya *et al*. (2013), Zhao *et al*. (2012), Kotecha (2011), Chang *et al*. (2011), Lozoff *et al*. (2006), and Osório *et al*. (2001). There are also some studies that use descriptive statistics such as odd ratio or comparing trends in health and health care provisions of rich and poor to discuss health inequality in Bangladesh; e.g., Ahmed (2013), Uddin *et al*. (2012), Khan *et al*. (2011), Mahabub-ul-Anwar *et al*. (2008), Hong *et al*. (2006). Nevertheless, we are unaware of any study considered a standard inequality measure, such as the Gini coefficient, to assess children's health inequality using the Hb level.[1] Our study using Bangladesh Demographic and Health Survey (BDHS) 2011 data for Hb level is a unique contribution to the small literature that exists in this field.

[1]Wang (2013) estimates an economic-related inequality measure, the Concentration Index (CI), for anemia for Cambodian children aged 6-59 months. Although CI and G both are calculated in reference to the Lorenz curve (LC), for the CI calculation the data are arranged by a second variable, whereas G is calculated for a variable when sorted in ascending order. For example, Wang (2013) calculates CI using LC, where the x-axis represents the cumulative proportion of the sample, ranked by wealth status from poorest to richest categories; the y-axis represents the cumulative proportion of the Hb variable (for <11 g/dl level) corresponding to each wealth category. Using Cambodian DHS 2000, 2005 and 2010 data, he reports anemia inequality among children differs significantly by household wealth, with wealthy households having a lower proportion of children with anemia than for poor households.

The remainder of the paper is organized as follows: Section 2 provides a description of the data used and methodologies employed to estimate the Gini coefficient and its sampling variance using a stratified sampling design. Section 3 shows non-parametric observations regarding inequality on this subject matter. Section 4 examines the empirical distributions of Hb level and hypothesis tests. Section 5, which is the main contribution of this paper, uses the Gini coefficient and the variance estimates based on both stratified sampling design and the standard bootstrap methods to demonstrate inequality among the subgroups then section 6 concludes.

2. Data, sampling weights and methodology

2.1 Data and sampling weights

Data are taken from the Bangladesh Demographic and Health Survey (BDHS) 2011, a nationally representative sample survey that aims to detail statistics useful for assessing changes in key areas of development, including maternal and child health, education, and poverty eradication (e.g., NIPORT et al. 2013). The BDHS2011 is also the first survey that collected information on Hb levels as a measure of health of Bangladeshi children. The HemoCue system, a rapid testing technology for determining Hb levels in field survey recommended by the World Health Organization (WHO), was used for Hb testing among eligible children of age 6-59 months from every third of households that were selected for interviews.

The BDHS program collects data using a stratified two-stage cluster sampling method. This particular Hb sample is comprised of 2,432 children, which was 92% of the eligible children selected for measuring Hb levels. These children were selected from 583 clusters across 20 strata, with a wide range in number of clusters across strata – from 5 to 60. Given that all households do not have eligible children under five years of age, and only one-third of the households were selected for Hb collection, the number of children in a cluster was small, ranging from only 1 to

16 children with an average of 4.16 children per cluster; see, Table 1. More than 10% of the clusters consist of just one child, almost 43% of the clusters contain 1 to 3 children, and less than three percent of the clusters have more than 10 children.

[Insert Table 1 here.]

When the average cluster size in the sample is unusually small, estimates, especially variance estimates, under the survey design assumption, may not be reliable (see, Clarke and Wheaton, 2007; Mass and Hox, 2005; UN, 2005, p. 51). The issue of small numbers of children in clusters deteriorates further when children are divided by gender, age group, place of residence or wealth category. For instance, as seen in Table 1, when children are classified by gender, we have, at most, nine children in a cluster, with more than 30% of the clusters containing only one child. As there is a large number of clusters containing one child, irrespective of which subgrouping we examine as well as for the full sample, makes cluster analysis almost impossible. Accordingly, we ignore clustering of the sample and proceed as if data have been obtained under a stratified sampling design.

To account for the stratified sampling design, sampling weights are calculated as follows. Let W_{hi} be a child's sampling weight, scaled so that $\sum_{h=1}^{H}\sum_{i=1}^{m} W_{hi} = M$, the number of sampled children. The weights are normalized as $w_{hi} = \frac{W_{hi}}{\sum_{h=1}^{H}\sum_{i=1}^{m} W_{hi}}$, such that $\sum_{h=1}^{H}\sum_{i=1}^{m} w_{hi} = 1$.

2.2 Methodology of estimation

The population Gini coefficient is $G = \frac{2}{\mu}\int_0^\infty yF(y)dF(y) - 1$, where $\mu = \int_0^\infty ydF(y)$ is the population mean of the wellbeing random variable $Y \in [0, \infty)$, and $F(y)$ is the cumulative

distribution function. To estimate the inequality in the wellbeing variable Hb, denoted by y_{hi}, we calculate a plug-in estimator for G, given by:

$$\hat{G} = \frac{2}{\hat{\mu}} \sum_{h=1}^{H} \sum_{i=1}^{m} w_{hi}\, y_{hi} \hat{F}(y_{hi}) - 1, \tag{1}$$

where $\hat{\mu} = \sum_{h=1}^{H} \sum_{i=1}^{m} w_{hi}\, y_{hi}$ and $\hat{F}(y_{hi}) = \sum_{r=1}^{H} \sum_{t=1}^{m} w_{rt} I\{y_{rt} \leq y_{hi}\}$.

To undertake inferences about the inequality in Hb, we estimate two variances for $\hat{G}$ under the stratified survey design. An analytical variance formula, modified for stratified sampling design provided by Binder and Kovačević (1995), given by:

$$\widehat{Var(\hat{G})} = \sum_{h=1}^{H} (u_h^* - \bar{u}^*)^2, \tag{2}$$

where $u_h^* = \sum_{i=1}^{m} w_{hi} \left\{ \frac{2}{\hat{\mu}} \left[y_{hi} \left(\hat{F}(y_{hi}) - \frac{(\hat{G}+1)}{2} \right) + B(y_{hi}) - \frac{\hat{\mu}}{2} \left(\hat{G} + 1 \right) \right] \right\}$ and

$B(y_{hi}) = \sum_{r=1}^{H} \sum_{t=1}^{m} w_{rt} y_{rt} I\{y_{rt} \geq y_{hi}\}$, and $\bar{u}_h^* = H^{-1} \sum_{h=1}^{H} u_h^*$. The second variance estimator for $\hat{G}$ is obtained using the standard bootstrap method following Wolter (2007, p. 215), given by:

$$\widehat{Var^*(\hat{G}^*)} = \frac{1}{B-1} \sum_{b=1}^{B} (\hat{G}_b^* - \bar{\hat{G}}^*)^2, \tag{3}$$

where B is the number of bootstrap replications, $\bar{\hat{G}}^* = \frac{1}{B} \sum_{b=1}^{B} \hat{G}_b^*$ and $\hat{G}_b^*$ is the bootstrap estimator of G calculated using the formula in expression (1) in bootstrap replication b. It would be useful if the bootstrap variance estimate was similar in magnitude to the asymptotic estimate, which are far easier to generate in practice. All estimations and simulations are undertaken using STATA 14 (StataCorp, 2016) and EViews 8 (Quantitative Micro Software, 2013). The number of bootstrap replications is $B = 799$.

3. Status of anemia among children aged 6 – 59 months in Bangladesh

According to the WHO, a person is considered to be anemic, when his or her Hb level is below two standard deviations (-2SD) of the mean of the Hb distribution of a normal population of people of the same gender and age, living at the same altitude. In a normal population, 2.5% of the individuals are expected to be below the 2SD threshold. When this proportion exceeds 5% of the population, anemia is considered as a public health problem (e.g., WHO 2001, p. 4). In addition, the WHO established cut-off points of Hb levels for different age groups residing at sea level, below which an individual is said to be anemic. For children of age 6-59 months, when the Hb level is less than 11.00 g/dl, a child is said to be anemic. A level of 10.00 to 10.9g/dl is termed as mild anemia, 7.00 to 9.00g/dl as moderate and below 7g/dl as severe anemia.

Most newborn infants up to four months old have abundant iron in their blood from their mothers (e.g., Kotecha 2011). In contrast, after about four months of age, a gradual decline occurs in the iron reserve due to continued rapid growth in infant body size, and from six months of age iron is derived mostly from external nutritional sources. While any deviation from the optimal Hb level can result in detrimental health consequences for any age group, children, 6-59 months old, are often regarded as being the most vulnerable (e.g., Singh and Patra, 2014; WHO, 2001). Thus, our attention is on children of age 6-59 months.

Anemia can be caused by many factors,[2] however, iron deficiency,[3] primarily due to an inadequate nutritional intake, is considered to be a major contributing factor of anemia. Globally, it accounts for over 50% of all anemia cases (e.g., Benoist *et al.*, 2008; WHO 2001, p. 3). This particular form of anemia is known as iron-deficiency anemia (IDA). IDA or anemia has some

[2]For example, nutritional deficiency (e.g., low intakes of folic acid and vitamins A, or B_{12}), acute and chronic inflammation, parasitic infections, sickle cell disease, malaria, and inherited or acquired disorders that affect hemoglobin synthesis (WHO, 2011).

[3]Iron deficiency is a condition in which the supply of mobilizable iron stores in the blood of an individual diminishes to zero and iron supply to the transport protein apotransferrin is compromised (e.g., WHO, 2001, p.3).

long-term deleterious consequences for children under five years of age, as well as for the wellbeing of the entire nation. Anemic children typically have poor defense against infections, lower physical growth, and poor motor and mental development (see, e.g., Zhao *et al*., 2012; Baker *et al*., 2010; Benoist *et al*., 2008; WHO, 2001). The economic implications of anemia are substantial, and the costs incurred for prevention, reduced productivity due to physical and mental deterioration and long-term effects on mental development and human capital formation can be enormous (e.g., Singh and Patra, 2014; WHO, 2001, p. 11-13). The current economic cost of poor Hb status in Bangladesh is estimated to be around 7.9% of the GDP (e.g., see USAID *et al*., 2011).

Figure 1 shows percentages of Bangladeshi children aged 6-59 months having different levels of anemia. More than half (52%) of the children suffer from some degree of anemia. This prevalence of anemia is well above the 40% benchmark rate considered as a severe public health problem.[4] By categories, 29% of children have mild anemia, over 21% have moderate anemia and less than 1% have severe anemia.

[Insert Figure 1 here.]

The prevalence of anemia in Bangladeshi children, when the children are classified by gender, age group and place of residence, are presented in Table 2. In addition, we report the relative risk (*RR*) factor whenever the sample is divided by a category that has two groups (e.g., gender: male and female). To determine which group (male or female) is at higher risk, the *RR* factor is calculated as the ratio of the proportion or percentage of one group to another, given by:

[4]WHO declares the situation as a severe public health problem when the anemia prevalence rate is ≥ 40%. For other cut-off points, see WHO (2001, p. 17).

$$RR = \frac{P_1}{P_2} = \frac{P_1(\text{anemic children}|\text{children in group1})}{P_2(\text{anemic children}|\text{children in group2})},$$

where P_1 and P_2 are probabilities or percentages of anemic children in group 1 and group 2 respectively. If $RR > 1$, the numerator group (group 1) has a higher risk, both groups are at the same risk when $RR = 1$, and for $RR < 1$, the denominator group is at higher risk of anemia.

[Insert Table 2 here.]

Gender: We find that female children have a slightly lower prevalence of anemia (50.4%) than male children (52.7%). The calculated *RR* factors suggest that male children are at higher risk for all types of anemia except for mild anemia. However, at the 5% nominal significance level, we fail to reject the null hypothesis that the *RR* factor is equal to one, suggesting that the anemia prevalence across gender are the same. That gender is not a significant issue in determining anemia prevalence among children aged 6-59 months is consistent with recent suggestions that Bangladeshi girls are as well cared for as young boys within their households up to their fifth birthday (UNICEF, 2011). Similar findings reported for some other developing countries, for example, Borbor *et al*. (2014) and *Osório et al*. (2001), accord with our results.

Age Group: When children are classified by two age groups, results in Table 2 shows that children aged 6-23 months are at a much higher risk of anemia (72% higher chance to be anemic) compared to the older children (24-59 months), with the estimated *RR* factor significantly greater than 1 for each form of anemia (mild, moderate and severe). Such vulnerability of younger children of age group 6-23 months to anemia has often been reported for developing countries. For instance, see Ewusie *et al*. (2014), Ayoya *et al*. (2013) and Osório *et al*. (2001) for children in Ghana, Haiti and Southern Brazil, respectively. This high occurrence of anemia among children less than two years

of age is likely due to widespread poverty and the infants' vulnerability to diseases and infections, which further decreases their body's capacity to ingest and absorb iron (see, e.g., Ewusie *et al.*, 2014; Osório *et al*., 2001). The same reason may apply to malnourished mothers with low levels of iron in breast milk, resulting in anemic children.

Place of residence: Almost three-quarters of the population of Bangladesh live in the rural areas, and the incidence of poverty among people living in rural areas is higher than people living in the urban areas.[5] It follows that children living in rural areas are less well-nourished than those living in urban areas (see, e.g., Borbor *et al.*, 2014; Fosto, 2007; Smith *et al*., 2005) and thus are more likely to be anemic. The sample data is consistent with the above. The proportion of rural anemic children is significantly higher than that in the urban areas (χ^2 =4.84, p-value 0.014), and the *RR* factor for anemia is statistically significantly higher than 1 at the nominal 5% significance level, suggesting that rural children are more vulnerable to the risk of anemia than are urban children. However, for severe and moderate anemia, we fail to reject the hypothesis that the *RR* factor is 1, at least at the 10% level of significance. A similar prevalence of severe and moderate anemia for rural and urban children may result from chronic malnutrition, which could be linked to the general level of poverty in Bangladesh.[6]

Wealth: To ascertain whether anemia prevalence varies for children from different household wealth levels, we grouped children by the BDHS wealth category. This variable in BDHS is an indication of the relative socioeconomic status of the household, which is measured using an asset-

[5]The proportions of rural and urban people in Bangladesh living under the upper poverty line are 35.2% and 21.3% respectively (BPC, 2014). The poor health outcomes of children related to poverty are higher in rural areas; e.g., 39% of rural children under age five are underweight compared to that of 28% urban children (BDHS, 2011, p. 166)
[6]Over 17% of the population of the country lives under the *lower* poverty line, where the lower poverty line corresponds to the per capita expenditure level at which the household could just buy enough food with no money is left over to buy anything else (Haughton and Khandker, 2009, p. 53). This statistic is used to measure the undernourishment status of a population as it does not include non-food items.

based index (see, e.g., NIPORT *et al.* 2013, p. 176). It makes sense to assume that children in wealthy households receive better nutritional intake and avoid anemia, given the likely positive relationship between feeding practices and household wealth status. As reported in Table 2, a child living in the poorest household is 32% more likely to be anemic than a child living in the richest household – this further supports our claim that poverty is a major cause of anemia in children. We also notice that the percentage of children with various levels of anemia (except for severe anemia) decline with increased wealth, at least at the 5% nominal significance level. A small portion of the children in each wealth category suffer from severe anemia, and the portions of children with severe anemia across wealth categories are statistically the same (χ^2=2.842, p-value 0.585). Such findings may suggest that chronic anemia may be related to a range of factors, such as acute and chronic inflammation, parasitic infections, sickle cell disease, malaria, or even lack of identifying anemia, rather than simply nutritional deficiency due to the child's household economic standing (e.g., Zhao et al 2012).

4. Descriptive statistics and distributions of Hb level

The mean Hb level in the *all children* sample is 10.79 g/dl which is lower than the anemia threshold level of 11.00 g/dl. We reported earlier that 51.6% of the children in this sample are anemic. While exploring the distribution of the Hb data, we see the empirical distribution of the Hb in *all children* sample is slightly negatively skewed (see, Figure 2), and not normally distributed.[7] This shape of the distribution implies the presence of some severe anemic children, with very low levels of Hb, some as low as 3.1 g/dl. We see similar pattern in the shape of distributions, when the Hb sample is classified by children's background characteristics, such as

[7]A Jarque-Bera (Bera and Jarque, 1981) test strongly rejects the null hypothesis that Hb level is normally distributed (JB=340.9, p-value 0.000).

gender, age group, place of residence and household's wealth status, implying the presence of some moderate to severe anemic children in every category.

[Insert Figure 2 here.]

Table 3 presents hypothesis test results of subgroups separated by children's gender, age, place of residence and household's wealth status. Results suggest, consistent with the previous anemia analysis, that although the female children are slightly better off with higher mean Hb than the male children, the difference in mean Hb levels is not statistically significant, providing further evidence that gender of a child under five years of age does not affect the health outcome in Bangladesh.

[Insert Table 3 here.]

However, the age of a child, place of residence, and household's wealth status have statistically significant impacts on the mean level of Hb, at 5% level of significance. The mean Hb levels for children across wealth categories vary markedly, with the lowest (10.56 g/dl) and the highest (10.99 g/dl) mean Hb levels for children from the *poorer* and the *richest* wealth category households respectively. Overall, there is a significant positive association between the mean Hb and wealth (when sorted from poorest to richest order), at the 5% nominal significance level. A child from a wealthier household is more likely to receive adequate dietary intake of nutrients necessary for the synthesis of hemoglobin than one from a less wealthy household.

In addition, we find that children under two, irrespective of their households' economic background and place of residence, are more vulnerable in terms of Hb synthesis compared to

older children.[8] On average, a child under two years of age, living in rural areas or belonging to a less wealthy household would have a lower level of Hb and is more likely to suffer from the incidence of anemia. As such, to improve the children's health in Bangladesh in terms of Hb level, necessary interventions, e.g., distribution of iron supplements and deworming medication, should focus heavily on children under two.

5. Gini coefficient estimates and sampling variances in children aged 6-59 months

We now report Gini coefficient estimates $(\hat{G})$ and the sampling variances (using expressions (2) and (3) in section 2.2) of Hb data for all children and by gender, age group, place of residence and wealth category, and corresponding hypothesis test results in Table 4. The estimated Gini coefficient for the *all children* sample is 0.064. As there will always be variation in children's Hb levels, even in a healthy children population, we estimated G for the non-anemic children to ascertain a base G with which we could compare the Gini for the full sample of children. This sample statistic is 0.031, suggesting that the inequality in Hb levels among children of age 6-59 months is more than double the natural variation in Hb level we would observe if all the children were healthy. Our hypothesis test results about G using both analytical and bootstrap standard errors affirm that at the 5% significance level, inequality in Hb among children is statistically significantly different from zero.

[Insert Table 4 here.]

As reported in Table 4, variance estimates for $\hat{G}$ using analytical and bootstrap methods are highly comparable in the full sample as well as across categories. In some cases, where the

[8]Statistics and hypothesis test results are available up on request.

number of children in samples are lower, the bootstrap variance estimates are marginally smaller, which may imply better performance of bootstrap method for smaller samples. However, as variance estimates are fairly small, it does not seem to affect either the qualitative or the quantitative outcome in hypothesis testing irrespective of the variance estimate being adopted. However, obtaining the analytical variance estimates are far easier and computationally timesaving compared to those using the bootstrap method.

When children are divided by gender, the inequality among the male children is 4.8% higher than that for female children. However, we fail to reject the hypothesis that the inequalities are the same (at the nominal 5% significance level), no matter which standard error we used to calculate the test statistic. This implies that gender is not an issue in determining children's health inequality using Hb levels in Bangladesh.

The estimated Gini coefficient of the Hb level among the children of age 6-23 months is 0.07, which is 22.81% higher than that for the 24-59 months old children group. The difference in inequalities between the two age groups is statistically different from zero. We find that, in addition to higher anemia prevalence and variability in the Hb level distribution measured by the standard deviation for children under two, the inequality is also remarkably larger for this group. As mentioned earlier, children under two grow quickly and need adequate iron-rich food to keep their Hb levels within the normal range. However, households' economic condition and imbalanced child feeding practices[9] may affect their nutritional needs more severely than for children aged 24-59 months, leading to higher inequality in Hb level.

[9] In Bangladesh, while nearly all children under two years of age are breastfed, only 21% of them are fed appropriately, following the recommended infant and young child feeding guidelines (see NIPORT et al., 2013, p. 176).

We found that the place of residence had an impact on children's Hb levels; the rural children are at higher risk of anemia with significantly lower mean Hb level than the urban children. However, with an estimated 7.35% lower Gini coefficient, rural children's Hb levels are more equally distributed than that of the urban children. Despite these differences, we fail to reject the null hypothesis that the Hb level Gini coefficients for rural and urban children are significantly different. Unlike the anemia prevalence and mean Hb level, this suggests that inequality in Hb level is unaffected by the place of residence of a child, statistically speaking.

Table 4 also shows that while there is a marginally positive relationship between the mean Hb level and wealth category, the inequality in Hb level does not follow a similar relationship with the wealth category. The inequality of the Hb level is the highest for the *richest* households, and is followed by the inequality amongst children from the *poorer* households – not by the children in *richer* or *middle* category households. The least inequality was found among children from households in the *richer* category. This fails our null hypothesis that the Gini coefficients across five wealth categories are the same at 10% level of significance. It is perhaps due to that fact that even though the mean level of Hb in children increases with the improvement in their households' wealth status, in each wealth category there are children with all forms of anemia,[10] leading to more even distributions across wealth categories.

The above discussion on the empirical Gini coefficients from Hb data suggests that although health inequality amongst children of age 6-59 month is predominantly a national concern, the age of a child plays an important role in inequality; under two years of age shows the highest inequality. Other factors, such as gender, place of residence and wealth category are also

[10]We earlier related the causes of severe to moderate type of anemia among children from *rich* households to various diseases and infections, rather than just nutritional consequences linked to the household's wealth status.

worthy of consideration, but statistically not very important in determining inequality, as least for the current sample data.

6. Concluding remarks

Although the use of the Gini coefficient is common in inequality analysis, especially with income variables, most practitioners avoid reporting standard errors with the Gini coefficient estimates assuming cumbersome computations. We reported both analytical standard errors based on Binder and Kovačević's (1995) formula and conventional bootstrap standard errors under the stratified sampling design for all of our Gini coefficient estimates, and conducted hypothesis tests for inequalities in children's Hb levels.

We also explored children's Hb status using non-parametric analyses, such as the traditional chi-square and the relative risk factor. We found that the age of a child, place of residence and wealth status of a household are important factors in causing disparities in children's health. However, when the Gini coefficient is used, we find that only the age of a child is the significant factor contributes to the inequality. If policy makers only use the non-parametric test results, they would have to consider three factors to improve the children's health. Whereas, if a formal inequality measure, the Gini coefficient, is considered, policy interventions to fight such an inequality can put a priority on the age of child – children under two. Based on every statistical analysis in this study, we conclude that the children of age 6-23 months are most vulnerable to health quality in regards to anemia and inequality of their hemoglobin levels. As such, this group of children should be heavily focused in any effort to eradicate childhood anemia, and to improve the overall health equality of children aged 6-59 months in Bangladesh.

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Table 1. Number of strata, cluster, and observations, average size of a cluster and proportions of clusters with different numbers of observations in Hb among Bangladeshi children age 6-59 months, BDHS2011: by gender, age group, place of residence, and wealth categories.

	Number of Strata	Number of clusters	Number of obs.	Average size of a cluster	Min. number of obs. in a cluster	Max. number of obs. in a cluster	% of clusters with 1 obs.	% of clusters with ≤3 obs.	% of clusters with ≥10 obs.
All children	20	583	2432	4.172	1	16	10.292	42.71	2.715
Gender									
Male	20	503	1244	2.473	1	9	30.020	78.53	0
Female	20	495	1188	2.400	1	9	30.909	80.61	0
Age Group					1				
6-23 Months	20	425	783	1.842	1	7	48.176	94.35	0
24-59	20	556	1649	2.966	1	11	19.065	65.83	0.180
Place of Residence									
Rural	7	385	1711	4.444	1	16	9.910	37.66	2.597
Urban	13	198	721	3.641	1	9	11.610	52.52	0
Wealth Category									
Poorest	17	261	559	2.142	1	7	41.379	83.91	0
Poorer	17	255	476	1.867	1	8	50.587	90.98	0
Middle	18	269	443	1.647	1	9	57.249	90.80	0
Richer	20	260	460	1.769	1	6	52.308	92.69	0
Richest	20	230	494	2.148	1	14	45.217	85.65	0.434

Table 2. Percentage of Bangladeshi children aged 6-59 months with anemia by gender, age group, place of residence, and wealth category, and hypothesis tests, BDHS 2011.

	Anemia Status by Hb Level				
	Severe (<7.0	Moderate (7.0-9.9	Mild (10-10.9	Any anemia (<11 g/dl)	Number of
Gender					
Male	0.80%	23.15%	28.70%	52.65%	1244
Female	0.76%	20.03%	29.63%	50.42%	1188
Relative Risk, *RR*	1.05	1.16	0.97	1.04	
$\chi^2(1)$ [a]	0.017	3.484	0.256	1.212	
p-value	0.449	0.062	0.307	0.136	
Age Group					
6-23 months	1.53%	37.55%	32.95%	72.03%	783
24-59 months	0.42%	14.07%	27.35%	41.84%	1649
Relative Risk, *RR*	3.64	2.67	1.21	1.72	
$\chi^2(1)$ [a]	8.409	172.658	8.062	193.707	
p-value	0.002	0.000	0.002	0.000	
Place of residence					
Rural	0.76%	22.21%	30.04%	53.01%	1711
Urban	0.83%	20.25%	27.05%	48.13%	721
Relative risk, *RR*	0.916	1.097	1.111	1.101	
$\chi^2(1)$ [a]	0.034	1.149	2.203	4.841	
p-value	0.427	0.142	0.069	0.014	
Wealth category					
Poorest	0.89%	25.40%	32.20%	58.60%	559
Poorer	1.26%	26.68%	29.41%	57.35%	476
Middle	0.45%	19.86%	31.60%	51.92%	443
Richer	0.43%	16.96%	27.17%	44.57%	460
Richest	0.81%	18.42%	25.10%	44.33%	494
$\chi^2(4)$ [b]	2.842	21.600	8.615	26.133	
p-value	0.585	0.000	0.071	0.000	
Anemia across Wealth category					
Slope	-0.001	-0.023	-0.016	-0.034	
z-score [c]	0.770	4.046	2.592	4.943	
p-value	0.221	0.000	0.005	0.000	

Notes: Hemoglobin is measured in g/dl. [a] H_0: Relative risk factor, RR = 1, or anemia prevalence between the two groups of children is the same and H_1: RR≠1. [b] H_0: Percentage of anemic children is the same for all age groups and. [c] H_0: Association between anemia prevalence and wealth category is zero and H_1: Association between anemia prevalence and wealth is negative.

Table 3. Descriptive statistics of Hb among Bangladeshi children aged 6-59 months, BDHS 2011: by gender, age group, place of residence and wealth category, and hypothesis tests.

		Mean Hb level, g/dl	Standard deviation	Hb distribution		Number of children
				Min., g/dl	Max., g/dl	
Gender	Male	10.767 (0.043)	1.275	3.1	14.4	1244
	Female	10.824 (0.042)	1.220	4.7	14.7	1188
Age Group	6-23 months	10.188 (0.056)	1.242	4.7	14.7	783
	24-59 months	11.086 (0.035)	1.149	3.1	14.7	1649
Place of Residence	Rural	10.761 (0.037)	1.231	3.1	14.7	1711
	Urban	10.923 (0.061)	1.286	6	14.4	721
Wealth Category	Poorest	10.663 (0.066)	1.195	6.2	14.1	559
	Poorer	10.555 (0.061)	1.264	4.7	13.8	476
	Middle	10.874 (0.071)	1.235	6.6	14.7	443
	Richer	10.980 (0.061)	1.168	3.1	14.1	460
	Richest	10.985 (0.073)	1.324	6.2	14.4	494

Hypothesis Tests

Null hypothesis*	**Wald test**	
	χ^2	p-value
$\mu_{\text{male}} = \mu_{\text{female}}$	1.030	0.311
$\mu_{6-23} = \mu_{24-59}$	200.50	0.00
$\mu_{\text{rural}} = \mu_{\text{urban}}$	5.130	0.024
$\mu_{\text{poorest}} = \mu_{\text{poorer}} = \mu_{\text{middle}} = \mu_{\text{richer}} = \mu_{\text{richest}}$	37.2	0.000

Notes: Figures in parentheses are survey design adjusted (stratified sampling design) standard error of mean. *Two-sided alternative hypothesis. In survey design adjusted mean comparison hypothesis testing, adjusted Wald statistic has F distribution with finite sample. Chi-square statistics are calculated as $\chi^2(\text{df1}) = \text{df1} \times F_{(\text{df1},\text{df2})}$ for asymptotic approximation.

Table 4. Gini coefficient estimates, sampling variances, and hypothesis tests for Hb levels in Bangladeshi children aged 6-59 months and children by gender, age group, place of residence, and wealth category, BDHS 2011.

Category	n	$\hat{G}$	$\widehat{Var(\hat{G})}$	$\widehat{Var^*(\hat{G}^*)}$
All children	2432	0.064	1.609	1.611
Gender				
Male	1244	0.066	2.928	2.928
Female	1188	0.063	3.542	3.345
Age Group				
6-23 months	783	0.070	5.816	5.836
24-59 months	1649	0.057	1.719	1.695
Place of residence				
Rural	1711	0.063	2.001	2.015
Urban	721	0.068	8.257	8.033
Wealth Category				
Poorest	556	0.065	5.700	5.687
Poorer	474	0.067	10.120	9.988
Middle	443	0.065	7.431	7.671
Richer	457	0.060	5.861	5.804
Richest	494	0.070	11.510	10.410

Hypothesis Tests

Null Hypothesis	Change in $\hat{G}$	Using $\widehat{Var(\hat{G})}$		Using $\widehat{Var^*(\hat{G}^*)}$	
		Wald Statistic	p-value	Wald Statistic	p-value
$\mu_{\text{male}} = \mu_{\text{female}}$	0.003	13.391	0.238	1.435	0.231
$\mu_{6-23\text{ months}} = \mu_{24-59\text{ months}}$	0.013	22.429	0.000	22.441	0.000
$\mu_{\text{rural}} = \mu_{\text{urban}}$	-0.005	2.437	0.118	2.488	0.115
G's across five wealth groups are the same	n.a.	6.759	0.149	7.060	0.133

Notes: Variances have been scaled by 10^6. All coefficients are estimated based on stratified sampling design. $\widehat{Var(\hat{G})}$ and $\widehat{Var^*(\hat{G}^*)}$ are analytical and bootstrap variance estimators in expression (2) and (3), respectively. Bootstrap estimators are calculated for 799 replications.

Figure 1. Prevalence of anemia in children aged 6-59 months in Bangladesh, BDHS 2011.

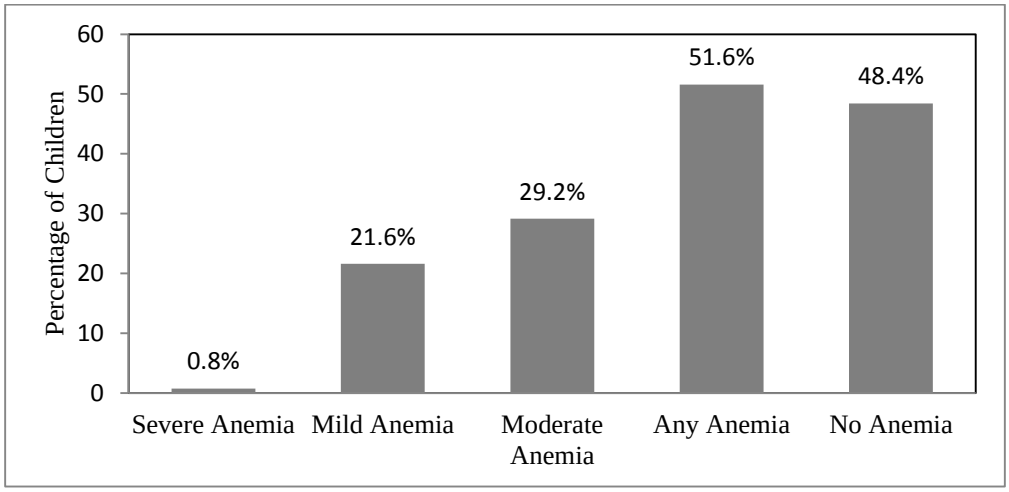
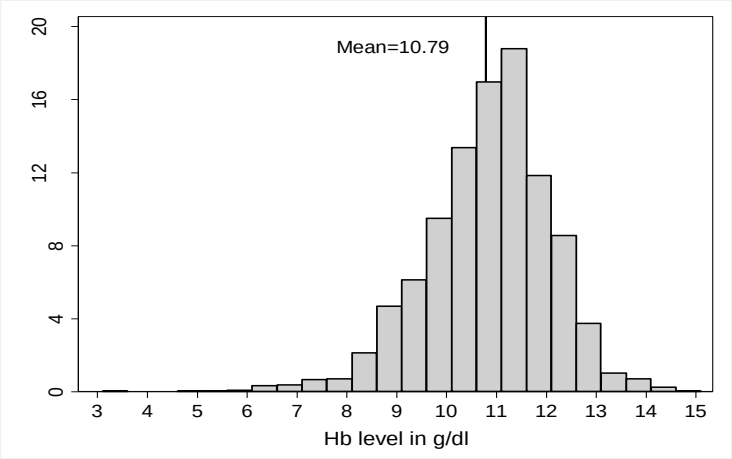


Figure 2. Empirical distribution of Hb for Bangladeshi children aged 6-59 months, BDHS 2011



Notes: Skewness and kurtosis estimates of the distribution are -0.593 and 4.399 respectively.