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Intergenerational influences on childhood anaemia

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Abstract

Prior research on assessing intergenerational influences on child anaemia has largely approached it from purely maternal perspective. Although there is much merit to focus on that, it is an extremely limited/reductionist view of understanding intergenerational influences. We expanded the intergenerational influences to include the fathers and overall of intergenerational household transfers. We analysed a sample of 19,619 mother–father-offspring trios from the 2015–2016 Indian National Fertility and Health Survey with available data on haemoglobin (Hb). Multinomial logistic regression models were used to establish associations between parent anaemia, household characteristics, and categories of offspring anaemia. Maternal and paternal Hb was measured as in children using a finger prick blood sample. The primary outcome was child's Hb level (in g/dl) and grades of anaemia defined as mild (10–10.9 g/dl), moderate (7–9.9 g/dl), and severe (<7 g/dl). Mean Hb was 10.1 g/dl for children, 14.2 g/dl among fathers, and 11.4 g/dl among mothers. Hb correlation was 0.1 between fathers and offspring and 0.2 between mothers and offspring ($P < 0.001$ for all correlations). Maternal–paternal Hb correlations were consistent across quintiles of wealth index. Maternal anaemia was associated with odds ratio of 1.3 (95% CI [1.1, 1.4]) and 1.6 (95% CI [1.4, 1.7]) for childhood mild and moderate/severe anaemia, respectively. Paternal mild anaemia was associated with an odds ratio of 1.1 (95% CI [0.9, 1.4]) and 1.4 (95% CI [1.2, 1.7]) for child moderate/severe anaemia. The clustering of poor circumstances suggests that public health strategies target social deprivation at the household level. A comprehensive perspective will provide holistic interventions to control childhood anaemia.

KEYWORDS

anaemia, childhood, intergenerational, paternal–maternal haemoglobin

1 | INTRODUCTION

Severe anaemia remains a major driver of high death rates in children in the developing countries (Kassebaum et al., 2014; Ngesa & Mwambi, 2014; Woldie, Kebede, & Tariku, 2015). The prevalence of childhood anaemia in developing countries is put at 40% and is classified as severe (Assefa, Mossie, & Hamza, 2014). In Southern Asia, anaemia affects 46–66% of children aged below 5 years (Lozoff & Georgieff, 2006; Woldie et al., 2015). Nutritional deficiency anaemia is the commonest form of childhood anaemia (Aikawa, Khan, Sasaki, & Binns, 2006; Woldie et al., 2015), linked to poor physical development, with implications for mental development (Aikawa et al., 2006; Armar-Klemesu, 2014; Birhane, Shiferaw, Hagos, & Mohindra, 2014; Cardoso, Scopel, Muniz, Villamor, & Ferreira, 2012; Desalegn, Mossie, & Gedefaw, 2014; Hurtado, Claussen, & Scott, 1999; Manning et al., 2012; McLean, Cogswell, Egli, Wojdyla, & de Benoist, 2009; Pasricha et al., 2010; Pollitt, 1999; Sachdev, Gera, & Nestel, 2005; Shafir, Angulo-Barroso, Calatroni, Jimenez, & Lozoff, 2006; Shet et al., 2009; Victoria et al., 2008; Villalpando, Shamah Levy, Ramirez Silva,

[Correction added on 24 September 2018, after first online publication: The data on final sample size and research year were incorrect and has now been updated in Abstract and Study Population sections.]

Mejia Rodriguez, & Rivera, 2003; Woldie et al., 2015). Severe anaemia can result in cardiovascular complications and, if not treated effectively, could result in hypoxemia and ultimately brain damage, multi-organ failure, and death. Although mild anaemia may not be associated with these deadly consequences, it flags early signs of serious illness for prompt attention.

Prior assessments of intergenerational influences on anaemia have focused on the mother, stressing the impact of anaemia during pregnancy on the development of anaemia in the newborn, because maternal iron is positively correlated with ferritin from umbilical cord blood suggesting a causal relationship in maternal anaemia and childhood anaemia (Pasricha et al., 2010). Some have also argued that antenatal anaemia in mothers contributes to low birth weight and prematurity, both of which increase the risk of childhood anaemia (Pasricha et al., 2010). Furthermore, it is stressed that inability of the mother to breastfeed the child increases risk of offspring anaemia and that given the shared socio-economic environment, there is also shared dietary quality between mother and child (Kumar, Rai, Basu, Dash, & Singh, 2008; Pasricha et al., 2010; Sala et al., 2008; Wharton BA, 1999). Evidence for the genetic transmission of anaemia derives from heritability studies (Farley MA, Smith PD, 1987). For instance, the study on the genetic influences on F cells and other hematologic variables revealed that additive genetic effects account for 37% and 42% of haemoglobin (Hb) and RBC count, respectively. Other studies have also shown that normal Caucasians have higher Hb levels than Black individuals matched for age and sex (Farley MA, Smith PD, 1987).

There is thus a mechanism to link maternal Hb/anaemia with child anaemia. We however want to establish the strength of this maternal-offspring anaemia compared with paternal as a way to broaden the potential intervention to the household or even community level, instead of a maternal focus. Mothers do not exist in vacuum, and other factors may play a role because of social assortative mating due to phenotype and cultural factors (Farley MA, Smith PD, 1987) and similarity in socioe-conomic backgrounds. Mother/father/child share the same environment and the extent to which iron deficiency anaemia is driven by what they have and eat. Consequently, a maternal focus limits the scope of potential interventions. One strategy to examine the exclusivity of maternal-offspring associations would be to use information on paternal anaemia and compare the of the effect size of maternal-offspring and paternal-offspring associations. Some studies in the context of undernutrition and body mass index reveal that there appears to be no difference between maternal and paternal influences on offspring body mass index, suggesting the importance of shared environment and/or impossibility to tell if it is exclusively mother (Knight et al., 2005; Lawlor et al., 2008; Leary et al., 2006; Veena et al., 2004; Wilcox, Newton, & Johnson, 1995; Wills et al., 2010). But no such study has been done on child anaemia.

The knowledge of whether parent to child anaemia is causal or marker of some household disadvantages remains unclear (Subramanian, 2016). In this paper, we examined the associations of parental anaemia with offspring anaemia in a large sample of parent-offspring trios in India. The question is whether there is an independent effect of maternal Hb/anaemia on child Hb/anaemia which would be stronger and more consistent than that of the paternal Hb/anaemia effect on child Hb/anaemia.

Key messages

- Prior research on assessing intergenerational influences on child anaemia has largely approached it from purely maternal perspective.
- Although there is much merit to focus on that, it is an extremely limited/reductionist view of understanding intergenerational influences.
- Expanding the intergenerational influences to include the fathers and overall of intergenerational household transfers.
- The clustering of poor circumstances suggests that public health strategies target social deprivation at the household level.
- A comprehensive perspective will provide holistic intervention to control childhood anaemia.

2 | METHODS

Data for this study originated in the 2015–2016 Indian National Health Survey (NFHS). The NFHS is equivalent to the Demographic and Health Survey Program which operates in over 80 low/middle income countries (Corsi, Neuman, Finlay, & Subramanian, 2012). The NFHS used standardized questionnaires to collect information on maternal and child health, socio-economic conditions, and other indicators from nationally representative samples of women aged 15–49 years, their children (0–4 years), and partners (15–54 y). The 2015–2016 NFHS used a multistate, stratified cluster sampling procedure which has been described elsewhere (International Institute for Population Sciences and ICF, 2017). In this survey, anaemia testing was performed by measuring Hb concentration- from a drop of blood obtained from by finger prick. For this analysis, we selected a sample of 15,015 mother–father-offspring trios from the NFHS with available data on Hb.

2.1 | Study population

The study population constitutes a nationally representative cross-sectional sample of singleton children aged 0–59 months alive at the time of survey and born after January 2010 to mothers with data from the partner/father from all 36 States/Union Territories India (n = 30,874). From this sample, we removed n = 4,175 children with missing data on Hb (which is only available in children aged 6 months and older and an additional n = 571 with Hb missing on either mothers or fathers. An additional n = 6,509 cases with missing data on covariates were removed to yield a final analytic sample of 19,619 parent-offspring trios.

2.2 | Outcome

The primary outcome was child's Hb level (in g/dl) and grades of anaemia defined as mild (10–10.9 g/dl), moderate (7–9.9 g/dl), and severe (<7 g/dl). For analyses purposes, the moderate and severe categories were combined. Blood testing was conducted by trained personnel, who were part of the survey team. The finger prick tests were carried out in the homes of the respondents, and blood samples were tested immediately using a portable HemoCue analyser (201+).

2.3 | Exposures

Maternal and paternal Hb was measured as in children using a finger prick blood sample. Grades of anaemia were defined as mild (12–12.9 g/dl in men, 10–11.9 g/dl in women, and 10–10.9 in pregnant women), moderate (9–11.9 g/dl in men and 7–9.9 g/dl in women), and severe (<7.0 g/dl in women and < 9.0 g/dl in men). Similar to children, the analysis of maternal and paternal anaemia combined the moderate and severe categories.

2.4 | Covariates

We included a number of child, parental (separately for mother and father), and household level socio-economic covariates which may be associated with child and maternal/paternal Hb levels. The sample frequency distribution, mean levels of child Hb, and frequency of grades of anaemia across covariates are given in Table 1.

2.5 | Analysis

We fitted multinomial regression models with a categorical outcome for child anaemia status: no anaemia, mild anaemia, or moderate/severe anaemia, treating no anaemia as the reference category. We fitted a series of four model specifications: (a) a model including maternal anaemia category with covariates, (b) a model including paternal anaemia category with covariates, (c) a model including maternal and paternal anaemia and covariates, and (d) a model with a categorical predictor indicating all combinations of maternal and paternal anaemia status with covariates. Additional sensitivity analyses were performed by stratifying the four models according to the following a priori specified covariates: quintile of wealth index, overall level of affluence (defined as highest wealth index, highest education, and urban residency, vs. others), and child age (6–7 months, 8–11 months, and 12–50 months) All analyses were weighted using survey weights provided in the NFHS and regression models account for clustering at the primary sampling unit and survey design and stratification characteristics.

3 | RESULTS

Among children in this sample, mean Hb was 10.5 g/dl (*SD* 1.5), 28% had mild anaemia, 30% had moderate anaemia, and 1% had severe anaemia. Among parents, mean levels of Hb were 14.1 g/dl (*SD* 1.8) among fathers (22% had any anaemia) and 11.5 g/dl (*SD* 1.6) among mothers (57% any anaemia). The Pearson correlation in Hb was 0.1 between mothers and fathers, 0.1 between fathers and offspring, and 0.2 between mothers and offspring ($P < 0.001$ for all correlations; Table 2). Maternal–paternal correlations in Hb were 0.14 in the poorest wealth quintile (Q1) and 0.08 in the richest (Q5, Table 2).

In multinomial logistic models examining the odds of a child having mild anaemia or moderate/severe anaemia (vs. no anaemia) as predicted by categories of maternal and paternal anaemia status indicated a generally stronger association between maternal anaemia categories and child anaemia compared with paternal anaemia categories and child anaemia (Table 3). In the first model including categories of maternal anaemia, mild anaemia in mothers was associated with an odds ratio of 1.3 (95% CI [1.1, 1.4]) for mild anaemia in children and 1.6 (95% CI [1.4, 1.7]) for moderate/severe anaemia in children. This increased to 1.4 (95% CI [1.2, 1.7]) and 2.4 (95% CI [2.1, 2.8]) for moderate/severe anaemia in mothers. In the following model, including only anaemia categories for the father, paternal mild anaemia was associated with an odds ratio of 1.3 (95% CI [1.1, 1.5]) for child mild anaemia and 1.6 (95% CI [1.4, 1.9]) for child moderate/severe anaemia. For moderate/severe paternal anaemia, it was 1.3 (95% CI [1.08, 1.6]) and 1.9 (95% CI [1.6, 2.2]).

In a model, which included the anaemia status of both parents, the magnitude of association with child outcomes was greater for mothers compared with fathers. For example, mild anaemia in mothers was associated with an OR of 1.3 (95% CI [1.1, 1.41]) for mild anaemia in children compared with fathers mild anaemia OR of 1.3 (95% CI [1.1, 1.5]), and a statistical test of the difference in these OR was not statistically significant ($P = 0.95$). Mild anaemia in mothers was associated with moderate/severe anaemia in children with an OR of 1.5 (95% CI [1.4, 1.7]) compared with paternal moderate/severe anaemia with an OR of 1.6 (95% CI [1.3, 1.8]; $P = 0.9$). Moderate/severe maternal anaemia was associated with moderate/severe anaemia in children with an OR of 2.4 (95% CI [2.0, 2.7]) compared with paternal moderate/severe anaemia with an OR of 1.8 (95% CI [1.5, 2.1]; $P = 0.02$).

Several sensitivity analyses were performed by examining the consistency of the odds ratios from the multinomial model by different strata of our study population according to wealth, affluence, and child age (Table 4). These analyses revealed that the greatest differences in the odds ratios of corresponding categories of maternal and paternal anaemia were found in the subgroups of the youngest ages and lowest wealth quintiles (Table 5).

4 | DISCUSSION

This study revealed significant associations of maternal and paternal Hb levels with offspring Hb. Maternal Hb level, family wealth, and food security have been reported important factors in iron status of children in India (Pasricha et al., 2010). According to Pasricha et al., the association between child's Hb level and anaemia status and maternal Hb level, as shown in this study, may come through manifold paths. For instance, it has been documented that anaemia resulting from iron deficiency during pregnancy contributes to low birth weight

TABLE 1 Distribution, mean levels of haemoglobin (Hb), and prevalence of anaemia among children according to study covariates

		Hb g/dl		Anaemia					
				No anaemia		Mild (10–10.9 g/dl)		Moderate/severe (≤9.9 g/dl)	
	No.	Mean	*SD*	No.	%	No.	%	No.	%
Maternal anaemia									
No anaemia	8,829	10.8	1.4	4,071	47.9	2,330	27.4	2,106	24.8
Mild anaemia	7,739	10.5	1.5	3,006	37.9	2,286	28.8	2,647	33.3
Moderate/severe anaemia	3,051	10.1	1.6	969	30.6	842	26.5	1,361	42.9
Paternal anaemia									
No anaemia	15,559	10.6	1.5	6,638	43.2	4,263	27.7	4,477	29.1
Mild anaemia	2,236	10.3	1.4	776	33.6	667	28.9	866	37.5
Moderate/severe anaemia	1,824	10.3	1.5	633	32.8	528	27.3	770	39.9
Child gender									
Girl	8,951	10.6	1.5	3,684	41.0	2,541	28.3	2,762	30.7
Boy	10,668	10.5	1.5	4,363	41.0	2,917	27.4	3,351	31.5
Child's completed age									
6–11 months	2,658	10.3	1.5	868	32.7	720	27.1	1,066	40.2
1 year	5,519	10.1	1.5	1,624	29.8	1,523	27.9	2,305	42.3
2 years	4,469	10.5	1.5	1,707	38.6	1,271	28.7	1,443	32.6
3 years	3,821	10.9	1.4	1,994	51.4	1,073	27.7	810	20.9
4 years	3,152	11.1	1.3	1,854	57.7	872	27.1	490	15.2
Months breastfed									
Not breastfed	1,516	10.7	1.4	654	43.8	428	28.7	409	27.4
1–6 months	1,804	10.6	1.6	861	42.5	577	28.5	588	29.0
7–12 months	4,250	10.5	1.5	1,759	39.1	1,184	26.3	1,553	34.5
13–24 months	7,279	10.4	1.5	2,642	36.9	2,035	28.4	2,484	34.7
25–36 months	3,302	10.7	1.4	1,401	45.6	873	28.4	799	26.0
37–59 months	1,468	10.9	1.4	732	53.3	361	26.3	280	20.4
HH wealth index									
Poorest	4,400	10.3	1.4	1,381	33.4	1,271	30.7	1,487	35.9
2nd	4,400	10.4	1.5	1,557	38.1	1,163	28.5	1,367	33.5
3rd	4,089	10.5	1.5	1,674	40.7	1,165	28.3	1,276	31.0
4th	3,511	10.7	1.5	1,693	44.9	938	24.9	1,139	30.2
Richest	3,219	10.8	1.4	1,741	49.7	922	26.3	844	24.1
Religion									
Hindu	14,225	10.5	1.5	6,448	41.1	4,337	27.7	4,888	31.2
Muslim	3,062	10.5	1.5	1,171	39.5	832	28.0	963	32.5
Christian	1,491	10.8	1.4	221	49.5	113	25.2	113	25.3
Sikh	327	10.4	1.4	94	36.2	86	33.0	80	30.8
Other/missing religion	514	10.6	1.5	113	41.2	91	33.3	70	25.5
Caste									
Scheduled Caste	3,589	10.5	1.4	1,551	38.7	1,133	28.3	1,321	33.0
Scheduled Tribe	3,984	10.4	1.5	775	36.6	577	27.2	768	36.2
Other Backward Class	7,435	10.5	1.5	3,556	40.9	2,395	27.5	2,748	31.6
General	4,611	10.7	1.5	2,165	45.1	1,354	28.2	1,276	26.6
Maternal education									
0 year	5,532	10.3	1.5	1,763	34.0	1,450	28.0	1,966	38.0
1–5 years	2,719	10.4	1.5	933	36.8	737	29.1	867	34.2
6–10 years	7,327	10.6	1.5	3,195	42.5	2,104	28.0	2,225	29.6
11–12 years	2,000	10.7	1.5	921	45.0	560	27.3	566	27.7
12+ years	2,041	10.9	1.4	1,235	53.0	607	26.1	489	21.0
Paternal education									
0 year	3,180	10.3	1.4	1,019	33.0	903	29.2	1,170	37.8

(Continues)

TABLE 1 (Continued)

		Hb g/dl		Anaemia					
				No anaemia		Mild (10–10.9 g/dl)		Moderate/severe (≤9.9 g/dl)	
	No.	Mean	*SD*	No.	%	No.	%	No.	%
1–5 years	2,999	10.4	1.5	1,105	37.3	826	27.9	1,033	34.9
6–10 years	8,408	10.6	1.5	3,491	41.2	2,363	27.9	2,610	30.8
11–12 years	2,338	10.7	1.5	1,073	46.5	581	25.2	652	28.3
12+ years	2,694	10.8	1.4	1,359	48.7	785	28.1	649	23.2
Paternal smoking	3,477	10.7	1.5	1,340	45.7	754	25.7	837	28.6
Maternal smoking	66	10.3	2.2	5	39.2	3	21.1	5	39.6
Mother's age									
15–24 years	5,508	10.4	1.5	2,214	35.9	1,833	29.7	2,126	34.4
25–29 years	7,505	10.6	1.5	3,267	42.9	2,049	26.9	2,298	30.2
30–34 years	4,213	10.6	1.5	1,734	44.4	1,046	26.8	1,125	28.8
35–49 years	2,393	10.6	1.5	831	43.2	531	27.6	563	29.3
Father's age									
15–29 years	6,766	10.4	1.5	2,429	35.2	1,984	28.7	2,489	36.1
30–39 years	10,195	10.6	1.5	4,564	44.1	2,824	27.3	2,960	28.6
40–54 years	2,658	10.6	1.5	1,054	44.5	651	27.5	664	28.0

TABLE 2 Pearson correlations and intra-class correlations (ICC) for different pairwise comparisons of haemoglobin (Hb) levels and by wealth (Q1 and Q5)

	Overall	Q1	Q5
Sibling pair (ICC)	0.40	0.38	0.33
Mother–child	0.17	0.17	0.11
Father–child	0.11	0.10	0.08
Mother–father	0.12	0.14	0.08
Two unrelated individuals in a village (ICC)	0.15	0.13	0.16
Two unrelated individuals in a state (ICC)	0.10	0.13	0.08

Note. Pearson correlations account for sampling weights; ICC from multilevel models of child haemoglobin with random effect for mother; three-level model of child nested in villages/communities and states.

(Desalegn et al., 2014), which increases the risk of childhood anaemia (Subramanian, 2016). Kumar et al. (2008) have also reported that severe maternal anaemia may also reduce breast milk iron content. Again, the mother and child share socio-economic circumstances, which may ultimately affect the dietary quality of the child similar to the mothers (Pasricha et al., 2010). In this study, maternal severe anaemia was associated with 11.2% of the cases of severe anaemia in offspring and 51.1% of moderate anaemia reported in offspring. Severe anaemia in fathers was also found to be associated with severe anaemia in 8.8% of the offspring with anaemia. Other factors tested but which were weak for predicting severe anaemia in children included the gender of the child, which was 2.7% and 3.3% for boys and girls, respectively; breast feeding, and household wealth. The younger children and children who were not breastfed contributed to more of the cases of severe anaemia in children with 5.0% and 6.6%, respectively.

Previous studies have suggested an association between growth and the development of anaemia in children. However, other studies (Chen et al., 2009; Pasricha et al., 2010) did not. Just like the other studies, our data confirmed previously reported associations between lower Hb levels and male gender (Chen et al., 2009; Pasricha et al., 2010), findings that were possibly related to greater absolute longitudinal growth among boys (Leary et al., 2006).

The risk of iron-deficiency anaemia in offspring may thus depend on complex interactions between a number of maternal/household dietary-iron content (type of diet), iron bioavailability (duration of breastfeeding and appropriate complementary feeding practices), increased iron use (growth velocity and erythroid mass expansion), and inappropriate iron losses (Leary et al., 2006). Other biological factors associated with childhood Hb levels included serum folate level, presence of inflammation, and hemoglobinopathy status. Studies have shown associations between Hb and C-reactive protein levels when ferritin levels are factored into the equation. Such studies hold that higher C-reactive protein levels decreased the coefficient of the relationship between Hb and ferritin levels, a result related to ferritin being an acute-phase protein (Pasricha et al., 2010).

As shown in other studies, the results of our study highlight important associations of household wealth and offspring with anaemia that have been previously reported (Pasricha et al., 2010; Skalicky et al., 2006). These results point to a direct link between broader socio-economic conditions and Hb levels in children and their mothers, attributable to shared malnutrition (Smith & Spivak, 1985), deficiencies in other micronutrients (Russell & Suter, 2015), exposure to biofuel smoke (Mishra & Retherford, 2007), and other possible mechanisms associated with lower socio-economic status (Pasricha et al., 2010).

In the light of the foregoing, it could be argued that household financial crises may threaten the health status of children low and middle-income countries (Horton, 2009) and may play a role in maternal and offspring anaemia through its effect on food insecurity. This will worsen if the stressors listed above undermine socio-

TABLE 3 Odds ratios and 95% CI from a multinomial model of categories of child anaemia according to categories of maternal/paternal anaemia

	Child mild anaemia						Child moderate/severe anaemia					
	Maternal			Paternal			Maternal			Paternal		
Maternal/paternal Anaemia	OR	95% CI		OR	95% CI		OR	95% CI		OR	95% CI	
None	1.00						1.00					
Mild	1.27	1.14	1.42				1.56	1.40	1.74			
Moderate/severe	1.43	1.23	1.66				2.43	2.10	2.81			
None				1.00						1.00		
Mild				1.29	1.10	1.51				1.59	1.36	1.86
Moderate/severe				1.29	1.08	1.55				1.87	1.57	2.23
None	1.00			1.00			1.00			1.00		
Mild	1.26	1.13	1.41	1.27	1.08	1.49	1.54	1.38	1.72	1.56	1.33	1.83
Moderate/severe	1.41	1.21	1.64	1.26	1.05	1.52	2.35	2.12	2.72	1.77	1.48	2.11

Note. "No anaemia" category treated as the outcome reference category in multinomial model. Adjusted for child sex, birth order, duration of breastfeeding, child age, wealth index, religion, caste, maternal/paternal education, maternal/paternal smoking, maternal/paternal age, and fixed effects for state.

TABLE 4 Odds ratios and 95% CI from a multinomial model of categories of child anaemia according to categories of maternal/paternal anaemia for different population subgroups

	Child mild anaemia						Child moderate/severe anaemia					
	Maternal			Paternal			Maternal			Paternal		
Maternal/paternal Anaemia	OR	95% CI		OR	95% CI		OR	95% CI		OR	95% CI	
Wealth												
Q1												
Mild	1.12	0.90	1.40	1.19	0.88	1.59	1.85	1.41	2.44	1.56	1.13	2.13
Mod/severe	1.48	1.10	1.98	1.15	0.85	1.56	3.63	2.57	5.12	1.98	1.38	2.85
Q2												
Mild	1.27	1.01	1.61	1.31	0.97	1.77	1.70	1.30	2.22	1.55	1.04	2.29
Mod/severe	1.57	1.15	2.15	1.37	0.93	2.02	2.01	1.43	2.84	1.66	1.11	2.47
Q3												
Mild	1.18	0.92	1.52	1.04	0.73	1.50	1.71	1.31	2.22	1.40	0.97	2.03
Mod/severe	1.45	1.05	2.02	1.33	0.88	2.01	2.60	1.92	3.53	1.45	0.95	2.23
Q4												
Mild	1.52	1.17	1.97	1.16	0.79	1.69	1.57	1.21	2.03	1.06	0.70	1.63
Mod/severe	1.25	0.84	1.86	1.05	0.65	1.69	2.38	1.67	3.40	1.80	1.07	3.04
Q5												
Mild	1.33	1.01	1.74	1.70	1.10	2.63	1.57	1.18	2.08	1.28	0.79	2.07
Mod/severe	1.45	0.95	2.22	1.30	0.74	2.30	2.47	1.66	3.66	1.08	0.57	2.03
Less affluent												
Mild	1.25	1.12	1.40	1.31	1.11	1.54	1.69	1.50	1.91	1.52	1.28	1.80
Mod/severe	1.46	1.26	1.70	1.32	1.10	1.58	2.58	2.22	3.00	1.86	1.54	2.25
Most affluent												
Mild	1.89	1.11	3.21	1.34	0.61	2.93	1.27	0.69	2.35	0.95	0.24	3.81
Mod/severe	1.29	0.52	3.20	0.95	0.26	3.43	4.12	1.30	13.08	0.58	0.15	2.20
Child age 6–7 months												
Mild	2.21	1.23	3.96	1.84	0.80	4.26	1.52	0.79	2.94	1.02	0.39	2.69
Mod/severe	0.85	0.38	1.93	3.87	1.39	10.80	1.69	0.72	3.98	0.69	0.24	1.98
8–11 months												
Mild	1.24	0.88	1.75	1.45	0.83	2.53	1.91	1.19	3.07	1.81	0.83	3.96
Mod/severe	0.99	0.60	1.63	1.28	0.72	2.29	3.02	1.56	5.83	1.50	0.59	3.82
12–59 months												
Mild	1.27	1.12	1.43	1.30	1.09	1.54	1.67	1.48	1.89	1.46	1.23	1.73
Mod/severe	1.53	1.30	1.80	1.25	1.03	1.52	2.60	2.23	3.02	1.76	1.46	2.12

Note. Models adjusted for birth order, months of breastfeeding, religion, caste, maternal, and paternal age.

TABLE 5 Sensitivity analysis of child haemoglobin anaemia according to categories of maternal/paternal haemoglobin and for different population subgroups

	Child haemoglobin						
	Maternal haemoglobin			Paternal haemoglobin			
Model	Beta	95% CI		Beta	95% CI		*P* value
Unadjusted	0.146	0.127	0.164	0.073	0.055	0.090	<0.001
Adjusted	0.116	0.099	0.133	0.068	0.051	0.085	0.0004
Mother-daughter	0.132	0.106	0.158				
Father-son				0.085	0.061	0.108	
Wealth 1	0.121	0.087	0.154	0.060	0.027	0.0938	0.024
Wealth 2	0.132	0.100	0.164	0.085	0.050	0.1194	0.073
Wealth 3	0.123	0.087	0.158	0.054	0.019	0.0883	0.011
Wealth 4	0.139	0.093	0.184	0.064	0.026	0.1022	0.022
Wealth 5	0.091	0.043	0.140	0.059	0.014	0.1038	0.371
Less affluent	0.131	0.113	0.149	0.072	0.055	0.0892	<0.001
Most affluent	0.111	0.013	0.209	0.058	−0.043	0.1596	0.471
6–7 months	0.081	0.004	0.157	0.118	0.054	0.1832	0.458
8–11 months	0.111	0.040	0.181	0.037	−0.019	0.0920	0.141
12–59 months	0.133	0.113	0.152	0.069	0.050	0.0874	<0.001

economic advancement or worsen food insecurity in India. Interventions that support nutrition and address socio-economic conditions may help mitigate these phenomena.

A study in urban slums of New Delhi investigated 90 anaemic children and identified important contributions from iron and vitamin B12 deficiency (Pasricha et al., 2010; Subramanian, 2016). Another study of young Mexican children revealed iron-deficiency anaemia was less common than anaemia from other causes (Duque, Flores-Hernandez, Flores-Huerta, 2007; Gomber et al., 1998). In Malawi, infectious diseases and vitamin B12 and folate deficiencies, but not iron deficiency, were important factors associated with severe childhood anaemia (Calis et al., 2008; Pasricha et al., 2010). Results of studies in Thailand and the United States also indicated that iron deficiency was less common than other cause of childhood anaemia (Wright, 2005; Thurlow, Winichagoon, Green, 2005). Our data provide major insights into household, and economic factors associated with anaemia in children in India.

The results should, however, be taken with the following limitations in mind. First, although this is a cross-sectional study, for which we report association rather than causation, we have used the maternal–paternal comparative design to strengthen the inferences which can be drawn from these data. Second, as stated above, the measurement of growth trajectory may have put more light on our understanding of growth and the influence of maternal/paternal influences on offspring anaemia. Despite these limitations, we have identified been able to ascertain a robust association between maternal and paternal anaemia with anaemia in children.

In the developing countries, iron-deficiency anaemia remains a leading risk factor for anaemia (Ezzati, Lopez, Rodgers, Vander Hoorn, & Murray, 2002). It is linked with impaired cognitive development and potentially restricts economic development (Cardoso et al., 2012; Hunt, 2002; Manning et al., 2012; McLean et al., 2009; Shafir et al., 2006; Victoria et al., 2008; Woldie et al., 2015). Policy makers should put in place interventions that include iron supplementation, food fortification, and dietary diversification in the household level (Zimmermann & Hurrell, 2007) (Allen, 2000).

5 | CONCLUSIONS

This study revealed that in addition to maternal influence on childhood anaemia, paternal and overall household influences must be considered for a more comprehensive policy framework for intervention at the household level. In this study, both maternal and paternal influences correlate significantly with childhood anaemia. Thus, the practice of previous assessments of intergenerational influences on child anaemia that focused mainly on a maternal perspective had not only been reductionist in nature but limited in their views of the factors that influence childhood anaemia in households. Beyond the pregnancy-related pathways to childhood nutrition and anaemia, there are other pathways such as the environment, household income, and parental nutritional practices, including the father's. All of these must be considered for an effective intervention.

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CONFLICTS OF INTEREST

The authors declare that they have no conflicts of interest.

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CONTRIBUTIONS

NGO produced the initial draft of the manuscript; DJC performed the analysis, and AK helped to evaluate the manuscript. SVS supervised the development of the work, helped in data interpretation and manuscript evaluation, and serve as the corresponding author. All authors reviewed and approved the final version of the manuscript.

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