



# The Risk Factors for Child Anemia Are Consistent across 3 National Surveys in Nepal

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## ABSTRACT

**Background:** Anemia is an etiologically heterogeneous condition affecting over half of preschool-aged children in South Asia. An urgent need exists to elucidate context-specific causes of anemia to effectively address this issue.

**Objectives:** This study investigated national trends and stability in the prevalence of child anemia and associated risk factors from 2013 to 2016 in Nepal.

**Methods:** Same-season national surveys were administered in 2013, 2014, and 2016 in 63 sites across 21 districts, selected using multistage random sampling, representing the mountains, hills, and Tarai (plains). Among consenting households with children aged 6–59 mo, a random sample of capillary blood was selected each year for anemia assessment using an Hb 201+ hemoglobinometer, with  $n = 835, 807,$  and 881 children assessed, respectively. Prevalence of child anemia, defined as hemoglobin  $<11.0$  g/dL with adjustment for altitude, was estimated each year and disaggregated by region and child age. Prevalence ratios were estimated using log-binomial regression models with robust SE or robust Poisson regression when models failed to converge. Interaction terms between each risk factor and year were created to test for consistencies in associations over time.

**Results:** The national prevalence of child anemia decreased from 63.3% (95% CI: 59.0%, 67.5%) in 2013 to 51.9% (95% CI: 46.5%, 57.2%) in 2014 and increased to 59.3% (95% CI: 54.7%, 63.8%) in 2016. Across years, prevalence was highest in the Tarai (58.4–70.2%), followed by the mountains (53.0–61.1%) and hills (37.5–51.4%). Nationally and across time, child age and maternal anemia were significantly associated with child anemia. Child diarrhea and stunting, maternal thinness, and poor water and sanitation conditions also showed consistent trends toward higher anemia prevalence.

**Conclusions:** Anemia affects more than half of Nepalese children aged 6–59 mo. Although prevalence varies year to year, the stability of observed risk factors suggests the need to focus on reducing gastrointestinal infection, promoting adequate household sanitation, and improving maternal and child health. *Curr Dev Nutr* 2021;5:nzab079.

**Keywords:** children, anemia, Nepal, risk factors, nutrition

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Supplemental Tables 1–6 are available from the “Supplementary data” link in the online posting of the article and from the same link in the online table of contents at <https://academic.oup.com/cdn/>.

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Abbreviations used: APR, adjusted prevalence ratio; DHS, Demographic and Health Surveys; Hb, hemoglobin; LMIC, low- and middle-income countries; MDD-W, Minimum Dietary Diversity for Women; MUAC, midupper arm circumference; NNMS, Nepal National Micronutrient Survey; PoSHAN, Policy and Science for Health, Agriculture and Nutrition; VDC, village development community; WASH, water, sanitation, and hygiene.

## Introduction

Although the global prevalence of anemia has declined since the 1990s, it remains a major public health problem affecting 27% of the world's population (1). Moreover, progress made in resolving anemia has been uneven, with disparities persisting by region, sex, and age (2). Across all regions, prevalence is highest in children and women of

reproductive age (1, 3). In South Asia, where 58% of children aged  $<5$  y are anemic, anemia continues to be a barrier in achieving healthy child development and reducing morbidity and mortality (4). Anemia impairs the circulation of oxygen in the blood, resulting in weakness, fatigue, concentration difficulty, and decreased work productivity (5). In children, anemia is associated with an increased risk of infection as well as impaired cognitive and motor development and

poor school performance (6–9). These effects can have significant economic consequences in the long term. Reduced work capacity due to anemia is estimated to result in an annual productivity loss equivalent to \$4.2 billion USD in South Asia (10, 11).

Various environmental, social, and biological factors interact with one another to influence the prevalence and distribution of anemia within a population, making mitigation of the condition challenging. Iron deficiency often receives attention as the most important cause of anemia in low- and middle-income countries (LMICs). Infectious disease, genetic hemoglobin (Hb) disorders, and deficiencies in other nutrients like vitamins A and B-12 can also be important contributors (12, 13). Underlying factors, such as availability and access to a diverse diet, child caring behaviors, adequate health services, and clean water and sanitation, also influence anemia risk and can differ by sociocultural context and level of economic development.

Research assessing anemia prevalence is increasingly conducted through national surveys, which are often spaced out at 5-y intervals or longer. In Nepal, estimates produced by the Demographic and Health Surveys (DHS) reported that the prevalence of anemia in children aged <5 y decreased from 48% in 2006 to 46% in 2011 and then increased to 53% in 2016 (14). Although a lower prevalence (19%) was reported in 2016 by the Nepal National Micronutrient Survey (NNMS), these national estimates indicate that child anemia remains a public health problem (14, 15). The reasons behind this remain unclear, especially as Nepal has developed a national strategy and implemented programs for anemia control (16, 17).

Measuring variability in the prevalence of child anemia by year and assessing if context-specific, preventable determinants remain consistently associated with anemia across these years can provide a better understanding of trends in prevalence. Such evidence can also assist policymakers in determining which investments can help accelerate progress as Nepal aims to reduce child anemia prevalence to 10% by 2030, as well as provide evidence for other countries experiencing a high prevalence (18). Although risk factors for anemia are often examined in on-off surveys and studies, no studies to our knowledge have explored the consistency and variability of associations between multilevel risk factors and anemia in preschool-aged children over a period of a few years (19). Doing so would offer assurance of the veracity of such factors and a better understanding of their relative strength. The objective of this article was to examine year-to-year variability in the prevalence of preschool-aged child anemia at the national and regional level and investigate the stability of associated risk factors in Nepal in 2013, 2014, and 2016.

## Methods

### Study design

The current study was a component of the Policy and Science for Health, Agriculture and Nutrition (PoSHAN) Community Studies project that comprised 4 planned, annual, midyear population surveys conducted in the same sampled sites across 3 main agroecological zones—the mountains, hills, and Tarai (plains)—from 2013 to 2016 (20, 21). The goal of this observational study was to identify links between agriculture and nutrition to inform programs focused on improving food and nutrition security. In 2015, the survey was restricted to the Tarai due

to an earthquake of magnitude 7.8 in the hills and mountains in late April of that year, and thus no data from 2015 are presented in this analysis (22).

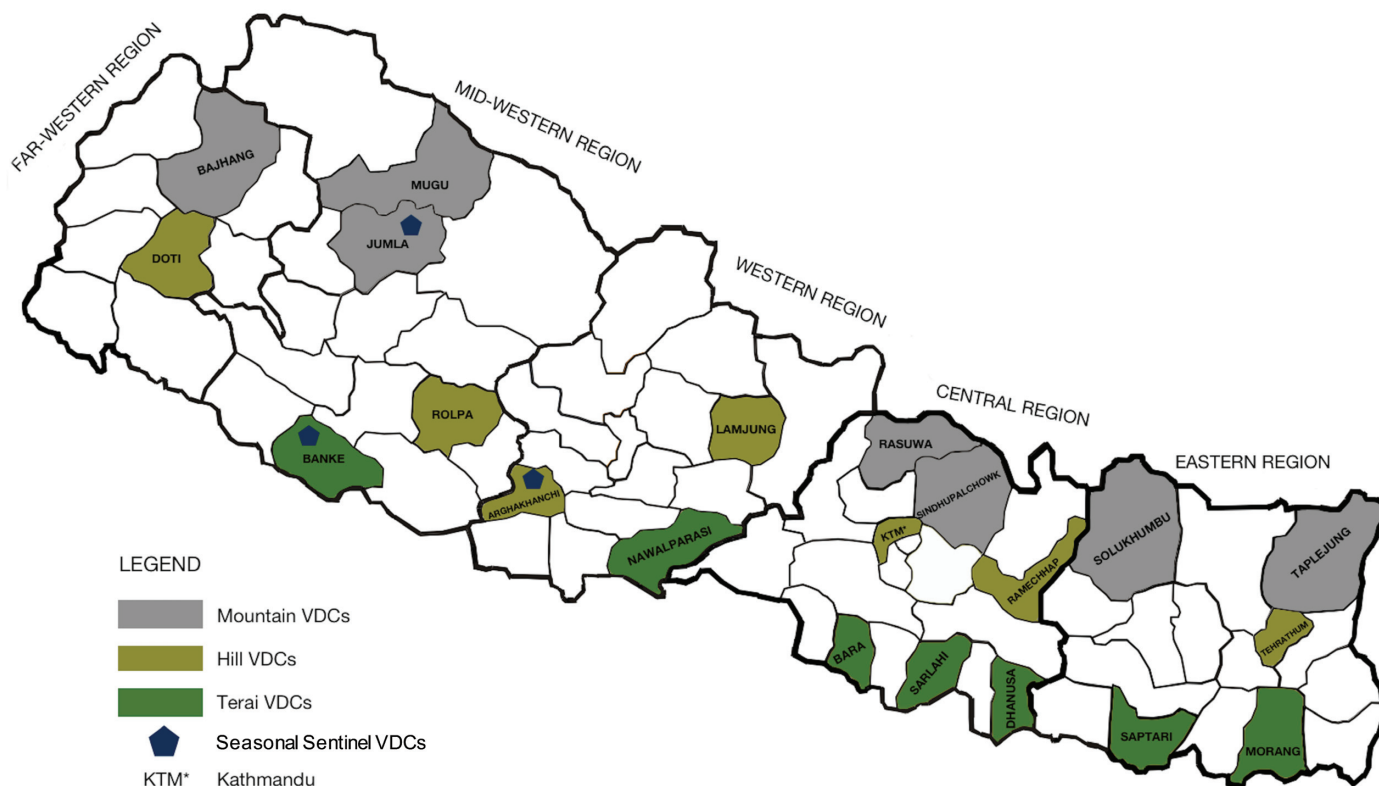
In early 2013, all districts in the country were stratified into the 3 main agroecological zones (Figure 1). Within each district, subdistricts, or village development communities (VDCs), were listed. From each zonal sampling frame, 7 VDCs were systematically selected following a random start, producing a sample of 21 VDCs that, because of the size of the sampling interval, were also located in 21 different districts geographically dispersed across each zone. Within each VDC, 9 wards were listed by population size, and 3 were systematically selected following a random start, resulting in a zonal population-weighted national sample of 63 wards (21 per zone). Within each ward, all households were visited and those with children aged  $\leq 60$  mo (or comprising childless, recently married couples) were eligible for enrollment. In subsequent years, within the same wards, the same households were visited and those with previously enrolled children were included until they reached  $>72$  mo of age, while previously unenrolled children  $\leq 60$  mo of age were added anew to the sample. Locations of households were mapped to facilitate relocation of households in subsequent surveys, as needed.

### Data collection

The surveys were carried out by 21 field teams (1 per VDC), each consisting of 3 interviewers and 1 supervisor, hired from a local research firm (New ERA Pvt. Ltd. and the Nepali Technical Assistance Group) and supervised by Johns Hopkins University and its Nutrition Innovation Lab Nepal team in Kathmandu. Each year, field teams were trained and standardized in obtaining informed consent, conducting interviews, and taking anthropometric and Hb measurements following the same basic protocol prior to the start of data collection. Each survey assessed individual- (child and maternal), household-, and community-level factors. Household interviews were conducted with heads of household, and mothers or female caretakers of children. Data were collected using paper forms that were sent to a central data management location. Data were checked by teams in the field for legibility, consistency, and completeness and data entry was subjected to strict quality control procedures (20, 21).

Each year, 1 in 4 consenting households were randomly sampled for Hb testing; the same households were not necessarily sampled each year for testing. If  $>1$  child of eligible age was present in the household, then 1 child was randomly selected for testing. A spot of whole blood was collected from the mother and 1 child using finger-sticks in children aged  $>6$  mo and their mothers, and heel-sticks in children aged  $\leq 6$  mo. Hb was assessed with an Hb 201+ hemoglobinometer (HemoCue AB). The HemoCue has been validated previously for measuring Hb and is recommended for use in field settings (23–25).

Study respondents received tokens for participation in the study. Tokens varied each year based on community engagement feedback and included items like soap, toothpaste, and mosquito nets. Caregivers were provided with their children's anthropometric and Hb test results, along with a brief explanation of the results. Children identified to be severely malnourished and/or anemic (Hb  $<7.0$  g/dL) were referred to the local health post (20).



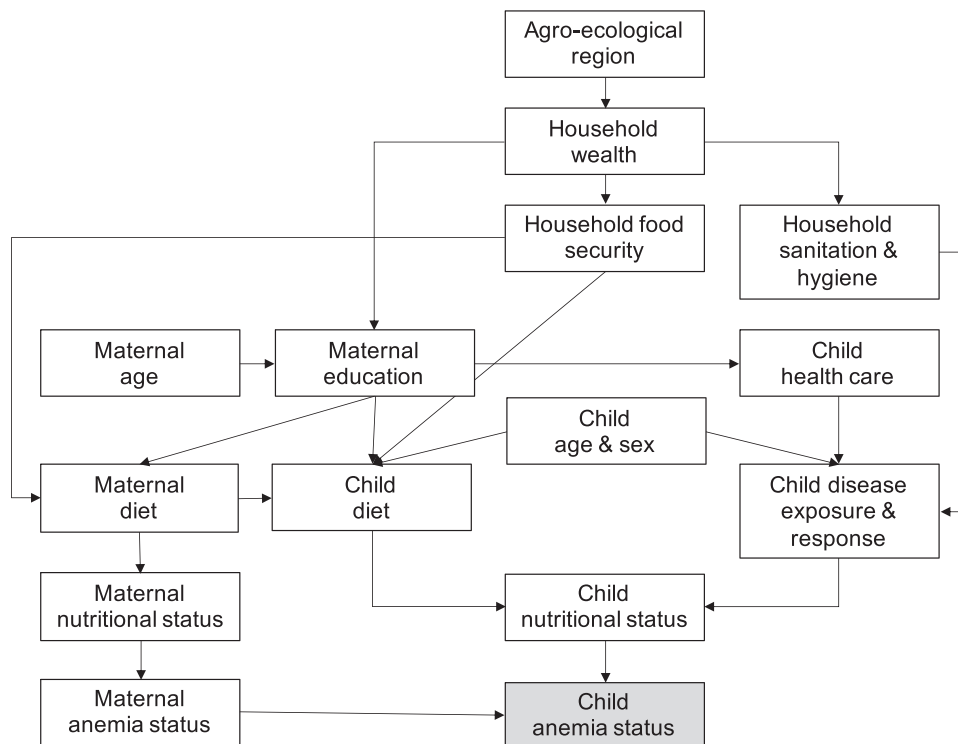
**FIGURE 1** Map of Policy and Science for Health, Agriculture and Nutrition (PoSHAN) Community Study nationally sampled survey sites. VDC, village development community.

### Risk factors

To guide selection of candidate variables representing potential risk factors for this analysis, we developed a conceptual framework based on a review of the literature of determinants of anemia in South Asia, informed primarily by observational studies (Figure 2). At the child level, these candidate variables included age; sex; stunting, wasting, and underweight status ( $z$ -scores of length-for-age, weight-for-length, and weight-for-age  $< -2$  below the WHO growth standards median, respectively); any reported diarrhea in the last 7 d; frequency of dietary intake of meat (poultry or red flesh meat, or organ meat) or snacks (instant noodles, beaten or puffed rice, sweets, biscuits, *dalmot*, or popcorn) in the last 7 d; any reported malaria in the last 30 d; and any receipt of vitamin A or iron supplementation or deworming tablets in the last 12 mo (26). Maternal-level candidate variables included maternal age, education, short stature (height  $< 145$  cm), low midupper arm circumference (MUAC  $< 22.5$  cm), dietary diversity in the last 7 d, and anemia status. Household-level candidate variables encompassed demographic and socioeconomic factors as well as those related to water, sanitation, and hygiene (WASH). Socioeconomic factors included caste, household food insecurity, and livestock ownership.

Child age was reported by mothers and checked by immunization cards when available. Because we found that child age did not have a linear relation with anemia status, and for ease of interpretation, we categorized the variable as 6–11, 12–23, 24–35, or 36–59 mo. We categorized maternal age as  $< 25$ , 25–39, or  $\geq 40$  y, and maternal education as higher secondary education or more, secondary, primary, or none. Child and

maternal dietary intake were assessed using a 7-d FFQ of  $\sim 50$  frequently consumed foods that had been evaluated for validity through a previous study in Nepal (27). We focused on child dietary intake of flesh or organ meat, which are rich in bioavailable iron, and snack food intake, because child consumption of unhealthy snack foods has been found to be associated with inadequate nutrient intakes in Nepal (28). We categorized dietary intake of meat and snacks each as  $\geq 8$ , 4–7, or 1–3 times, or none in the last 7 d. Organ meat was not included in the FFQ of the 2013 and 2014 surveys but was included in the 2016 survey. Based on the assumption that a child was likely to consume organ meat if they had consumed flesh meat in the last week, we included organ meat in the generation of the meat intake categorical variable in 2016. However, organ meat was not included in any survey rounds when calculating maternal dietary diversity scores. We used the maternal dietary intake data from the 7-d FFQ to generate a variable using the Minimum Dietary Diversity for Women (MDD-W) food groups, with the cutoff for each food group set at any consumption of that group in the last 7 d (29). Finding that maternal dietary diversity did not have a linear relation with child anemia, we treated the variable as dichotomous around consumption of  $\geq 5$  food groups, similar to the MDD-W but based on a 7-d recall. Child and maternal anthropometric measurements were taken using standard equipment (21). Child stunting, wasting, and underweight status and maternal short stature and thinness were treated as dichotomous variables in the analysis, as were child sex, any reported occurrence of diarrhea in the last week, any reported malaria in the last month, and any receipt of micronutrient supplements or deworming tablets in the



**FIGURE 2** Conceptual framework guiding risk factor analysis.

last year. We used maternal Hb concentration to estimate maternal anemia status, with anemia defined as an Hb concentration  $<12.0$  g/dL for nonpregnant women and  $<11.0$  g/dL for pregnant women based on the WHO standard (23).

Caste was treated as categorical and classified as Brahmin/Chhetri, other Tarai caste, Dalit, Newar, Janajati, or other. Food insecurity was measured using the Household Food Insecurity Access Scale, and households were categorized as being food secure or experiencing mild, moderate, or severe food insecurity (30). We treated livestock ownership as a dichotomous variable, representing any livestock owned in the household. WASH factors included the presence of animal feces or rubbish around the house or compound (determined by observation), location of child defecation, and type of toilet and drinking water source; each was treated as dichotomous. We derived a socioeconomic status score independently for each survey using principal component analysis of household assets owned, and the quality and infrastructure of the dwelling (31). As such, our indicator of socioeconomic status was not conducive to measuring changes in household wealth, and therefore not included in this analysis; sensitivity analysis conducted using the socioeconomic status score as a covariate in adjusted models produced results similar to the ones presented. Agroecological zone was included as a community-level factor and categorized as the mountains, hills, or Tarai.

### Statistical analysis

The analysis was restricted to children aged 6–59 mo who had data collected on Hb concentration in 2013, 2014, or 2016. Infants aged  $<6$  mo

were not included because there is no standard to assist with interpretation of Hb data for this age group (23). Data from 2015 were excluded because data collection that year was primarily undertaken in the Tarai due to an earthquake that affected the rest of the country (20, 22). We treated child anemia as dichotomous based on the WHO standard of an Hb concentration  $<11.0$  g/dL. Because residing at high altitude increases Hb concentrations, we adjusted Hb concentrations for children and women living at elevations  $>1000$  m above sea level based on WHO recommendations (23). All children with biologically plausible Hb concentration data ( $\geq 4.0$  and  $<18.0$  g/dL) and completed interview forms who had mothers with completed interview forms were retained in the analysis. *P* values  $<0.05$  were considered statistically significant.

Prevalence of anemia and mean Hb concentration in preschool-aged children were estimated by year and disaggregated by region and child age. The national prevalence of mild (Hb 10.0–10.9 g/dL), moderate (Hb 7.0–9.9 g/dL), and severe (Hb  $<7.0$  g/dL) anemia were also estimated by year to provide further context (23). CIs of prevalence estimates were estimated using robust SEs to account for clustering. We took 2 steps in our analysis of potential risk factors for child anemia. In the first step, we developed unadjusted log-binomial models using robust SEs to account for clustering with each candidate risk factor as the exposure variable and child anemia status as the outcome variable separately by year. Risk factor variables that were nonsignificant in unadjusted models were not retained and their unadjusted prevalence ratios and 95% CIs were not reported on. As such, reported child malaria and receipt of micronutrient supplements in the last year were not reported on or included in the subsequent analysis step.



In the second step, we developed multivariable Poisson regression models with robust variance separately by year because log-binomial models failed to converge (32, 33). To isolate the effect of each risk factor on anemia risk and for comparability across years, each of these models included a candidate risk factor as the exposure variable and child anemia status as the outcome variable and was adjusted for a standard set of covariates that included child age and sex, maternal age, and agroecological zone. Interaction terms were created between each risk factor and year to test for consistencies in associations over time in unadjusted and adjusted analyses, with 2013 set as the reference year. Multicollinearity was tested for in the multivariable regression models. We assessed if children had been visited more than once over the 3 y of included analyses and ran sensitivity analyses of the associations between each risk factor and child anemia excluding children with >1 visit. Missing data on the outcome and candidate risk factors were examined and the analysis took a complete-case approach. All analyses were conducted using Stata version 14.2 statistical software (34).

Ethical approval for the study was provided by the Nepal Health Research Council, an autonomous body, under the Ministry of Health and Population, Government of Nepal, and the Institutional Review Board at the Johns Hopkins Bloomberg School of Public Health, Baltimore, MD.

## Results

A total of 835, 807, and 881 children 6–59 mo of age were assessed for anemia in 2013, 2014, and 2016, respectively. Across years, no children had biologically implausible Hb values, and <5% had incomplete data on risk factors, resulting in an analytic sample of 791, 777, and 851 children in 2013, 2014, and 2016, respectively. The majority of children (91.5%) were only visited once for Hb assessment, but some were visited 2 out of the 3 years (8.1%) or every year (0.4%). Prevalence estimates and risk factor associations with the outcome did not differ when restricting the data to children with 1 visit (Supplemental Tables 1–4).

### Sample characteristics

Sample characteristics are presented in Table 1. Many child, maternal, and household characteristics did not change over time, including child age and sex, stunting prevalence, maternal age, the prevalence of maternal short stature, caste, livestock ownership, and the proportion of households sampled from each zone. Each year, about half of children did not consume any meat in the previous 7 d, whereas the majority consumed snack foods at least once. Coverage of child deworming treatment was high (>87%) and increased each year. Improvements were evident over time for household sanitation, with increases in the proportion of households with improved toilets with each year of data collection, and declines in the proportion practicing open child defecation or having animal feces in or around the house. The majority of households had access to a protected drinking water source each year, and the proportion of food-secure households increased over time. Improvements were also evident in maternal education and maternal nutritional status measured using MUAC. The proportion of mothers who were anemic decreased from 2013 to 2014 and increased in 2016.

### Anemia prevalence

Nationally, the prevalence of anemia in children 6–59 mo of age significantly decreased from 63.3% (95% CI: 59.0%, 67.5%) in 2013 to 51.9% (95% CI: 46.5%, 57.2%) in 2014, but then increased to 59.3% (95% CI: 54.7%, 63.8%) in 2016 (Table 2). Mean child Hb followed a similar pattern (Figure 3). Anemia prevalence was highest in infants 6–11 mo of age each year (Table 3). Across years, most children who were anemic had either mild or moderate anemia (Supplemental Table 5). The proportion of children that were mildly or severely anemic did not differ over time, but the proportion classified as moderately anemic significantly decreased from 2013 to 2014.

Prevalence of anemia was highest in the Tarai (range 58.4–70.2%), followed by the mountains (53.0–61.1%) and then hills (37.5–51.4%) (Table 2). In each region, prevalence followed a similar trend as the national prevalence, decreasing from 2013 to 2014 and then increasing in 2016. Likewise, mean Hb increased from 2013 to 2014 and decreased in 2016 in all regions.

### Child-level factors

Anemia prevalence was significantly higher in younger children (6–35 mo of age) compared with older children (36–59 mo of age) in all models ( $P$  values <0.05) (Table 4). Interaction terms between each category of child age and year were not significant, indicating that the relation between child age and anemia was consistent over time (Supplemental Table 6). In all models, anemia prevalence was higher in children with any reported occurrence of diarrhea in the last week compared with those with no reported diarrhea [adjusted prevalence ratios (APR)s ranged from 1.12 to 1.20], with the association significant in adjusted analyses in 2014 and 2016 ( $P$  values <0.05). The interactions between diarrhea and 2014 ( $P$  value = 0.529) and that between diarrhea and 2016 ( $P$  value = 0.509) were nonsignificant in adjusted analyses. This suggests that the positive relation observed between child diarrhea and anemia in each of these years was similar to the relation in 2013. Each year, anemia prevalence was higher in stunted children in adjusted analyses (APRs ranged from 1.04 to 1.22), although this association was only significant in 2013 ( $P$  value <0.05). No interaction terms between stunting status and year were significant in adjusted analyses ( $P$  values = 0.349 and 0.068 for 2014 and 2016, respectively), suggesting a consistently positive relation between child stunting and anemia. The association between child dietary intake and anemia was less conclusive. In adjusted models, children who did not consume meat in the last week, compared with those who consumed it  $\geq 8$  times, had higher prevalence in adjusted analyses in 2013 and 2014 but lower prevalence in 2016. There were no significant associations between snack food consumption and anemia in adjusted models. Child sex, wasting, underweight, and lack of deworming treatment in the last 12 mo were not consistently associated with anemia over time.

### Maternal-level factors

Prevalence of anemia was significantly higher in children of anemic mothers compared with those with mothers who were not anemic (APRs ranged from 1.21 to 1.34;  $P$  values <0.05) (Table 5). In adjusted analyses, neither the interaction between maternal anemia and 2014 ( $P$  value = 0.919) nor that between maternal anemia and 2016 ( $P$  value = 0.380) were significant, and thus the relation between

**TABLE 1** Sample characteristics of children aged 6–59 mo by year, Nepal<sup>1</sup>

Risk factors	2013 (n = 791) n (%)	2014 (n = 777) n (%)	2016 (n = 851) n (%)
<i>Child-level</i>			
Age, mo			
6–11	94 (11.9)	89 (11.5)	89 (10.5)
12–23	196 (24.8)	173 (22.3)	185 (21.7)
24–35	158 (20.0)	168 (21.6)	189 (22.2)
36–59	343 (43.4)	347 (44.7)	388 (45.6)
Sex, M	415 (52.5)	407 (52.4)	462 (54.3)
Stunted (HAZ < -2)	268 (33.9)	291 (37.5)	292 (34.3)
Wasted (WLZ < -2)	129 (16.3)	100 (12.9)	96 (11.3)
Underweight (WAZ < -2)	263 (33.2)	261 (33.6)	247 (29.0)
Diarrhea, any in last 7 d	118 (14.9)	81 (10.3)	91 (10.7)
Meat intake, <sup>2</sup> last 7 d			
8+ times	17 (2.1)	17 (2.2)	25 (2.9)
4–7 times	78 (9.9)	54 (6.9)	92 (10.8)
1–3 times	318 (40.2)	303 (39.0)	361 (42.4)
None	378 (47.8)	403 (51.9)	373 (43.8)
Snack intake, <sup>3</sup> last 7 d			
8+ times	349 (44.1)	398 (51.2)	427 (50.2)
4–7 times	228 (28.8)	194 (25.0)	271 (31.8)
1–3 times	137 (17.3)	110 (14.2)	88 (10.3)
None	77 (9.7)	75 (9.7)	65 (7.6)
Received deworming treatment, last 12 mo	692 (87.5)	702 (90.2)	786 (92.4)
<i>Maternal-level</i>			
Age, y			
<25	293 (37.0)	291 (37.5)	327 (38.4)
25–39	479 (59.4)	468 (60.2)	504 (59.2)
≥40	28 (3.5)	18 (2.3)	20 (2.4)
Education			
Higher secondary or more	71 (9.0)	95 (12.2)	143 (16.8)
Secondary education	170 (21.5)	189 (24.3)	234 (27.5)
Primary education	98 (12.4)	113 (14.5)	134 (15.7)
None	452 (57.1)	380 (48.9)	340 (40.0)
Short stature (height <145 cm)	80 (10.1)	81 (10.4)	110 (12.9)
Thin (MUAC <22.5 cm)	205 (25.9)	170 (21.9)	151 (17.7)
Dietary diversity (≥5 food groups), last 7 d	645 (81.5)	638 (82.1)	748 (87.9)
Anemic (Hb <12 g/dL)	433 (54.7)	388 (49.9)	459 (53.9)
<i>Household-level</i>			
Caste			
Brahmin/Chhetri	201 (25.4)	194 (25.0)	201 (23.6)
Other Tarai caste	241 (30.5)	235 (30.2)	240 (28.2)
Dalit	134 (16.9)	130 (16.7)	144 (16.9)
Newar	22 (2.8)	15 (1.9)	24 (2.8)
Janajati	152 (19.2)	166 (21.4)	201 (23.6)
Others	41 (5.2)	37 (4.8)	41 (4.8)
Food insecurity <sup>4</sup>			
None	449 (56.8)	560 (72.1)	683 (80.3)
Mild	147 (18.6)	114 (14.7)	67 (8.0)
Moderate	144 (18.2)	75 (9.7)	73 (8.6)
Severe	51 (6.4)	28 (3.6)	27 (3.2)
Livestock ownership, any	569 (71.9)	567 (73.0)	592 (69.6)
Animal feces in or around house	420 (53.1)	343 (44.1)	310 (36.4)
Rubbish in or around house	395 (49.9)	219 (28.2)	265 (31.1)
Open child defecation	519 (65.6)	460 (59.2)	385 (45.4)
Improved toilet <sup>5</sup>	354 (44.8)	439 (56.5)	571 (67.1)
Protected drinking water source <sup>6</sup>	737 (93.2)	733 (94.3)	794 (93.3)
<i>Community-level</i>			
Agroecological zone			
Mountains	144 (18.2)	132 (17.0)	132 (15.5)
Hills	218 (27.6)	208 (26.8)	240 (28.2)
Tarai	429 (54.2)	437 (56.2)	479 (56.3)

<sup>1</sup>HAZ, height-for-age z-score; Hb, hemoglobin; MUAC, midupper arm circumference; WAZ, weight-for-age z-score; WLZ, weight-for-length z-score.<sup>2</sup>Includes consumption of chicken, duck, goat, buffalo, and pork.<sup>3</sup>Includes consumption of instant noodles, beaten rice, puffed rice, sweets, biscuits, *dalmot*, and popcorn.<sup>4</sup>Food insecurity assessed using the Household Food Insecurity Access Scale (30).<sup>5</sup>Defined as flush toilet, ventilated improved pit latrine, or pit latrine with slab.<sup>6</sup>Defined as protected ring well, bottled water, tubewell/borehole, or piped water.

**TABLE 2** Prevalence of anemia and mean hemoglobin (Hb) in children aged 6–59 mo by year and region, Nepal<sup>1</sup>

	2013	2014	2016
National			
Children, <i>n</i>	791	777	851
Prevalence, % (95% CI)	63.3 (59.0, 67.5)	51.9 (46.5, 57.2)	59.3 (54.7, 63.8)
Mean Hb ± SD, g/dL	10.5 ± 1.3	10.8 ± 1.3	10.6 ± 1.3
Mountains			
Children, <i>n</i>	144	132	132
Prevalence, % (95% CI)	61.1 (53.4, 68.3)	53.0 (43.4, 62.5)	58.3 (45.9, 69.8)
Mean Hb ± SD, g/dL	10.5 ± 1.7	10.6 ± 1.5	10.6 ± 1.5
Hills			
Children, <i>n</i>	218	208	240
Prevalence, % (95% CI)	51.4 (43.6, 59.1)	37.5 (29.0, 46.8)	46.3 (37.8, 54.9)
Mean Hb ± SD, g/dL	10.9 ± 1.3	11.2 ± 1.2	11.0 ± 1.2
Tarai			
Children, <i>n</i>	429	437	479
Prevalence, % (95% CI)	70.2 (64.2, 75.6)	58.4 (52.0, 64.5)	66.2 (60.2, 71.7)
Mean Hb ± SD, g/dL	10.2 ± 1.2	10.6 ± 1.2	10.3 ± 1.3

<sup>1</sup> CIs estimated using robust SEs to account for clustering.

maternal and child anemia status was stable comparing each of these years with 2013 (Supplemental Table 6). Children of mothers who were thin showed a trend toward higher anemia prevalence over time (APRs ranged from 1.04 to 1.18). Although the adjusted association between maternal MUAC and child anemia was only significant in 2014 ( $P$  value <0.05), the interaction terms between MUAC and 2014 ( $P$  value = 0.414) and between MUAC and 2016 ( $P$  value = 0.548) were nonsignificant. Other maternal factors were less stably associated with child anemia. Across years, maternal age did not appear to be associated with child anemia in adjusted analyses. Children of women with no education had higher prevalence compared with those with more than secondary education in 2013 and 2014, but the direction of the relation between maternal education and child anemia reversed in 2016 in adjusted analyses. Children with mothers who consumed <5 food groups in the last 7 d had significantly higher anemia prevalence only in 2014 in adjusted analyses ( $P$  value <0.001) and the interaction term between maternal dietary diversity and 2014 was significant ( $P$  value = 0.001). Likewise, maternal stature did not show a consistent relation with child anemia.

### Household- and community-level factors

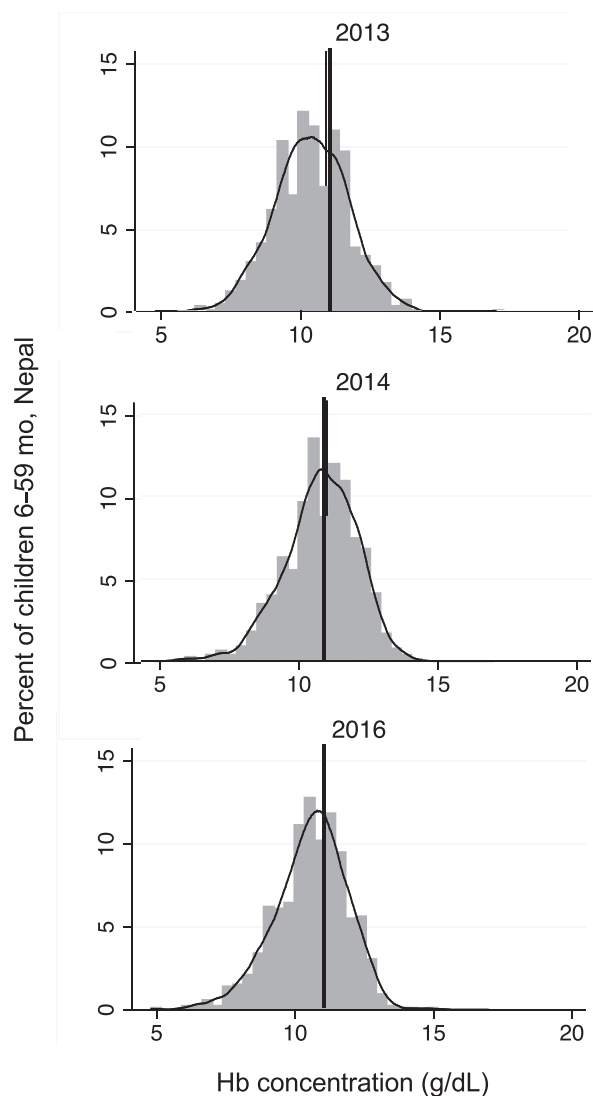
Each factor of poor hygiene and sanitation was positively associated with anemia prevalence over time, but to differing degrees of strength and significance (Table 6). Children living in households without a protected drinking water source had a higher prevalence of anemia than those with access to a protected source every year. The relation strengthened in adjusted analyses across years (APRs ranged from 1.23 to 1.49), although it was only significant in 2013 ( $P$  value <0.001). Interaction terms representing lack of access to a protected water source in 2014 ( $P$  value = 0.509) and in 2016 ( $P$  value = 0.139) were nonsignificant, suggesting that the relation between water access and anemia in each of these years was similar to that of 2013 (Supplemental Table 6). In unadjusted analyses, children living in households without an improved toilet or that practiced open child defecation had significantly higher anemia prevalence regardless of year ( $P$  value <0.05),

but for each, the strength and statistical significance of these relations decreased in adjusted analyses. The presence of animal feces or rubbish in or around the household was positively associated with anemia prevalence each year, but these associations were not statistically significant in unadjusted or adjusted analyses. None of the interaction terms generated for these WASH factors and year were significant, indicating consistency in their associations with child anemia over time.

Children belonging to the Dalit caste had higher prevalence compared with the upper-caste Brahmin/Chhetri group across years, although this association was only significant in 2014 in adjusted analyses ( $P$  value <0.05). Overall, household food insecurity was not strongly associated with anemia; anemia prevalence was higher in children living in severely food-insecure households compared with those living in food-secure homes each year, but this relation was not statistically significant. Livestock ownership was not associated with anemia at any timepoint. Residing in the hills was consistently protective against anemia in all models (APRs ranged from 0.69 to 0.81), whereas children living in the Tarai had a higher anemia prevalence than those living in the mountains (APRs ranged from 1.07 to 1.13).

### Discussion

This study estimated national and regional trends in anemia prevalence and assessed the stability of risk factors for anemia in children 6–59 mo of age in Nepal using 3 panel surveys from the PoSHAN Community Studies. The national prevalence of anemia varied over time (51.9–63.3%), decreasing from 2013 to 2014 but then increasing in 2016, and is in line with the prevalence of child anemia in South Asia (58%) (4). Despite observing variability in anemia prevalence, we found that child age and maternal anemia were significantly associated with child anemia from 2013 to 2016. Child diarrhea and stunting, low maternal MUAC, and multiple indicators of inadequate WASH also



**FIGURE 3** Distribution of adjusted hemoglobin (Hb) by year in children aged 6–59 mo, Nepal. Vertical line indicates 11.0-g/dL cutoff used to define anemia status.

showed consistent trends toward higher child anemia prevalence over time.

Our finding that anemia prevalence varied between 2013 and 2016 is curious given that many factors that likely contribute to anemia risk, including child nutrition and health status, maternal education, food security, and hygiene and sanitation, improved over the same period of time. Although our estimate of prevalence in 2016 (59.3%) is higher than that of the 2016 DHS Report (53%), both analyses observed sequential variability in prevalence in recent years. The 2016 NNMS estimated a lower prevalence (19%), but this difference could be attributable to seasonal differences in data collection periods among the surveys, because seasonality can affect anemia risk (35). Nonetheless, based on the aggregate of these results, child anemia continues to be a major public health problem in Nepal. It is possible that recovery from the 2015 earthquake influenced the increase in prevalence observed in 2016 in our study. However, as mentioned, indicators of health, food security,

and sanitation continued to improve in 2016. This is in line with an assessment of the nutrition and food security situation before and after the earthquake using data from the PoSHAN study, which found conditions were comparable or improved a year after the disaster. A possible explanation, which requires additional exploration, is that interventions and/or household resilience to shocks might have helped mitigate the negative impact of the earthquake (22).

Nepal has prioritized reducing anemia in national public health strategies, including the Intensification of Maternal and Neonatal Micronutrient Program, which implemented national distribution of iron and folic acid supplementation and deworming treatment (16). The 2018–2022 Multi-Sector Nutrition Plan-II outlines various interventions in the health, education, WASH, and agriculture sectors aimed at reducing child anemia prevalence. This approach of targeting iron deficiency and helminth infection while also addressing the sectors that influence the underlying causes of anemia is in line with our findings, which suggest the need to scale up interventions in maternal and child health care, dietary quality, and household sanitation to reduce the anemia burden. Regional disparities in anemia prevalence in Nepal were evident in this and other studies, with the Tarai bearing the highest proportion of anemic children (36, 37). This stresses the need to identify contextual factors specific to the Tarai that consistently place children residing there at greater risk (21). For example, arsenic contamination of water in the Tarai is an infrequent but intermittent problem that could contribute to the higher anemia prevalence (38). Prioritizing anemia prevention efforts in this region would support the government's overall investments in child health and development.

Improvements were observed in multiple indicators of hygiene and sanitation over time, likely reflecting the efforts of national strategies and programs (18, 39, 40). All indicators of poor WASH were associated with higher anemia prevalence each year in this analysis, consistent with previous studies linking poor sanitation to anemia in Nepal, although the strength and statistical significance of each factor's association with anemia differed (36, 41). It is possible that the improvements in WASH contributed to the decline in anemia prevalence between 2013 and 2014. However, even though WASH conditions improved over time,  $\geq 1$  indicator of inadequate sanitation and hygiene was observed in more than one-third of households by 2016 and the prevalence of anemia remained high. These findings suggest that reducing anemia through WASH will require an intensification of current interventions, with wider coverage and concentration on the behaviors and conditions most strongly associated with anemia risk. The need for more effective WASH strategies was stressed in a trial in Zimbabwe, which showed household-level interventions had no effect on diarrhea prevalence, Hb concentration, or child anemia prevalence (42). Living in poor WASH conditions also contributes to diarrhea through increased fecal-oral exposure (43–45). Even as diarrhea prevalence decreased in this population, children with recent occurrence of diarrhea were more likely to be anemic than those without diarrhea in each year assessed. Similarly, in an analysis of predictors for anemia over time in India, diarrhea was found to be significantly and negatively associated with child Hb (46). Intensifying efforts to prevent and treat child exposure to gastrointestinal infection therefore appears to be a clear pathway to reducing anemia prevalence.

Child age was stably associated with anemia, with prevalence decreasing with age and highest in infants 6–11 mo of age. This is



**TABLE 3** Prevalence of anemia and mean hemoglobin (Hb) in children aged 6–59 mo by year and child age, Nepal<sup>1</sup>

Age, mo	2013 (n = 791)	2014 (n = 777)	2016 (n = 851)
6–11			
Children, n	94	89	89
Prevalence, % (95% CI)	80.9 (71.2, 87.8)	77.5 (69.8, 83.8)	76.4 (63.4, 85.8)
Mean Hb ± SD, g/dL	9.8 ± 1.1	9.9 ± 1.2	9.8 ± 1.4
12–23			
Children, n	196	173	185
Prevalence, % (95% CI)	80.6 (73.5, 86.2)	64.2 (54.8, 72.5)	73.0 (66.4, 78.7)
Mean Hb ± SD, g/dL	9.9 ± 1.3	10.4 ± 1.2	10.0 ± 1.4
24–36			
Children, n	158	168	189
Prevalence, % (95% CI)	60.8 (52.9, 68.1)	50.6 (41.3, 59.8)	59.8 (52.7, 66.5)
Mean Hb ± SD, g/dL	10.5 ± 1.3	10.8 ± 1.3	10.6 ± 1.1
36–59			
Children, n	343	347	388
Prevalence, % (95% CI)	49.9 (43.2, 56.5)	39.8 (33.6, 46.2)	48.7 (42.5, 55.0)
Mean Hb ± SD, g/dL	10.9 ± 1.3	11.2 ± 1.2	11.0 ± 1.2

<sup>1</sup>CIs estimated using robust SEs to account for clustering.

consistent with other nationally representative studies that have assessed risk factors for anemia in children aged <5 y—in Nepal, Pakistan, Bhutan, and Bangladesh—and could be caused by the failure to meet demands for iron required during this period of rapid growth and development (4, 36, 47, 48). It is possible that the higher prevalence observed in young infants was influenced by the status of iron stores at birth and thus by maternal status, because numerous studies have shown that higher maternal iron stores are protective against iron deficiency in infants (49–51). Additionally, maternal anemia status is recognized as one of the strongest predictors of child anemia because low maternal Hb is associated with numerous adverse child health outcomes, including low birthweight and preterm birth, which in turn can increase a child's risk of anemia (12, 52). Feeding practices during infancy and childhood can also influence the risk of anemia. Inadequate dietary intake, particularly during the complementary feeding period from 6 to 23 mo of age when non-breast milk foods are introduced into the diet, results in nutrient gaps that can increase the risk of iron deficiency and anemia (12).

A common strategy to address child anemia has been to promote intake of foods rich in iron during the complementary feeding period (53, 54). Although consumption of iron-rich meat was not significantly associated with child anemia in our adjusted analyses, anemia prevalence was higher in those not consuming meat in the last week in 2013 and 2014, suggesting the need to further explore this relation. Studies in the region have produced mixed results regarding the association between child diet and anemia. Dietary patterns were not associated with Hb concentration in preschool-aged children in a cross-sectional analysis in Bhutan, and an analysis of DHS data in Bangladesh found no associations between anemia and reported minimum acceptable diet or consumption of iron-rich foods in 6–23-month-olds (47, 55). In contrast, a study analyzing data from a recent DHS in Myanmar found child consumption of iron-rich foods to be significantly associated with anemia, and an analysis of drivers of anemia in India concluded that dietary intake of iron, folate, and vitamin B-12 predicted changes in anemia prevalence in pregnant women (56,

57). It is possible that the lack of a consistent association found in our analysis and other cross-sectional, observational studies was because the dietary intake assessments utilized did not adequately capture usual dietary intakes and how such intakes can relate to the risk of iron deficiency. Overall, these findings reiterate the complexity of anemia as well as the need for accurate and precise assessments of dietary intake in children.

Other studies conducted in the region have found poor child nutritional status to be associated with anemia (36, 47, 48). In our study, anemia prevalence was consistently higher in stunted children compared with nonstunted children over time, but the relation was only statistically significant in one of the years assessed. Stunting, like anemia, has a complex etiology influenced by many biological and environmental factors. The relation between child anemia and stunting and other indicators of nutritional status such as wasting and underweight in our study requires further exploration. Prevalence of anemia was higher in children belonging to the Dalit caste compared with upper-caste children across years, although this relation was only statistically significant at 1 time point. Children living in severely food-insecure households had higher anemia prevalence than those living in food-secure homes, but this association was not significant at any point. Although these indicators of socioeconomic status were not significantly associated with anemia every year assessed, the higher prevalence in those with lower status might reflect the influence of socioeconomic factors on increasing a child's exposure to more immediate risk factors, like suboptimal WASH conditions and inadequate diet, as indicated in the conceptual framework developed for this analysis (58, 59). For example, in a pooled analysis of LMICs, children living in households in the lowest wealth quintile were 21% more likely to be anemic than those in the highest wealth quintile (12). In India, socioeconomic gains explained 17% of the improvement in Hb concentration in pregnant women in an analysis of anemia determinants (46).

This is the first study, to our knowledge, to repeatedly assess the prevalence and risk factors of preschool-aged child anemia over time, in

**TABLE 4** Child-level risk factors associated with anaemia in children aged 6–59 mo by year, Nepal<sup>1</sup>

Risk factors	2013 (n = 791)		2014 (n = 777)		2016 (n = 851)	
	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)
Age, mo						
6–11	1.62 (1.36, 1.93)**	1.60 (1.34, 1.91)**	1.95 (1.66, 2.29)**	1.90 (1.61, 2.25)**	1.57 (1.29, 1.90)**	1.57 (1.29, 1.91)**
12–23	1.62 (1.42, 1.84)**	1.59 (1.39, 1.82)**	1.61 (1.35, 1.93)**	1.64 (1.35, 2.00)**	1.50 (1.33, 1.69)**	1.49 (1.31, 1.69)**
24–35	1.22 (1.04, 1.43)**	1.20 (1.34, 1.91)**	1.27 (1.04, 1.56)**	1.29 (1.06, 1.57)**	1.23 (1.08, 1.40)**	1.21 (1.06, 1.38)**
36–59	1.00	1.00	1.00	1.00	1.00	1.00
Sex						
Male	1.00	1.00	1.00	1.00	1.00	1.00
Female	1.10 (1.01, 1.19)**	1.07 (0.99, 1.16)*	1.04 (0.92, 1.18)	1.01 (0.90, 1.14)	0.98 (0.87, 1.10)	0.98 (0.87, 1.09)
Stunting status						
Not stunted (HAZ $\geq -2$ )	1.00	1.00	1.00	1.00	1.00	1.00
Stunted (HAZ $< -2$ )	1.14 (1.01, 1.29)**	1.22 (1.08, 1.38)**	1.04 (0.91, 1.20)	1.13 (0.99, 1.28)*	0.97 (0.86, 1.10)	1.04 (0.93, 1.16)
Wasting status						
Not wasted (WLZ $\geq -2$ )	1.00	1.00	1.00	1.00	1.00	1.00
Wasted (WLZ $< -2$ )	1.15 (1.03, 1.29)**	1.00 (0.88, 1.12)	0.96 (0.79, 1.17)	0.80 (0.67, 0.96)**	1.12 (1.00, 1.25)**	0.97 (0.87, 1.07)
Underweight status						
Not underweight (WAZ $\geq -2$ )	1.00	1.00	1.00	1.00	1.00	1.00
Underweight (WAZ $< -2$ )	1.19 (1.07, 1.32)**	1.18 (1.07, 1.31)**	1.04 (0.91, 1.19)	1.00 (0.89, 1.13)	0.99 (0.88, 1.13)	0.98 (0.88, 1.09)
Diarrhea, any in last 7 d	1.00	1.00	1.00	1.00	1.00	1.00
No	1.20 (1.05, 1.36)**	1.12 (0.99, 1.26)*	1.32 (1.13, 1.55)**	1.20 (1.03, 1.39)**	1.30 (1.13, 1.49)**	1.18 (1.03, 1.36)**
Yes	1.00	1.00	1.00	1.00	1.00	1.00
Meat intake, <sup>2</sup> last 7 d						
8+ times	1.21 (0.71, 2.07)	1.15 (0.73, 1.80)	1.05 (0.53, 2.08)	1.00 (0.53, 1.88)	1.00	1.00
4–7 times	1.44 (0.91, 2.28)	1.29 (0.88, 1.88)	1.28 (0.74, 2.22)	1.24 (0.74, 2.08)	0.91 (0.63, 1.33)	0.85 (0.60, 1.20)
1–3 times	1.72 (1.08, 2.72)**	1.45 (0.98, 2.13)*	1.69 (0.98, 2.91)*	1.42 (0.86, 2.34)	1.12 (0.81, 1.54)	0.97 (0.71, 1.31)
None	0.81 (0.67, 0.99)**	0.92 (0.75, 1.12)	0.73 (0.60, 0.90)**	0.89 (0.74, 1.08)	0.92 (0.76, 1.13)	1.05 (0.88, 1.27)
Snack intake, <sup>3</sup> last 7 d						
8+ times	0.87 (0.73, 1.05)	0.98 (0.82, 1.18)	0.82 (0.66, 1.02)*	0.98 (0.80, 1.20)	0.83 (0.69, 0.99)**	0.91 (0.76, 1.08)
4–7 times	0.94 (0.78, 1.14)	1.00 (0.84, 1.20)	0.94 (0.75, 1.18)	1.14 (0.94, 1.39)	0.91 (0.76, 1.13)	1.05 (0.81, 1.36)
1–3 times	1.00	1.00	1.00	1.00	1.00	1.00
None	1.00	1.00	1.00	1.00	1.00	1.00
Received deworming treatment, last						
12 mo	1.00	1.00	1.00	1.00	1.00	1.00
Yes	1.01 (0.86, 1.19)	0.99 (0.85, 1.16)	1.24 (0.99, 1.55)*	1.14 (0.90, 1.44)	1.26 (1.00, 1.61)*	1.27 (1.00, 1.62)*
No	1.00	1.00	1.00	1.00	1.00	1.00

<sup>1</sup>Log-binomial regression models with robust SEs were used to estimate prevalence ratios. Robust Poisson models were used when models failed to converge. Each adjusted model included 1 risk factor as the exposure variable and controlled for the same set of covariates: child age and sex, maternal age, and region. \* $P < 0.10$ , \*\* $P < 0.05$ , \*\*\* $P < 0.001$ . APR, adjusted prevalence ratio; HAZ, height-for-age z-score; PR, prevalence ratio; WAZ, weight-for-age z-score; WLZ, weight-for-length z-score.

<sup>2</sup>Includes consumption of chicken, duck, goat, buffalo, and pork.

<sup>3</sup>Includes consumption of instant noodles, beaten rice, puffed rice, sweets, biscuits, *daimot*, and popcorn.

**TABLE 5** Maternal-level risk factors associated with anemia in children aged 6–59 mo by year, Nepal<sup>1</sup>

Risk factors	2013 (n = 791)		2014 (n = 777)		2016 (n = 851)	
	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)
Age, y						
<25	1.40 (0.96, 2.05)*	1.04 (0.72, 1.50)	1.28 (0.73, 2.26)	0.99 (0.58, 1.66)	1.25 (0.82, 1.91)	1.07 (0.66, 1.74)
25–39	1.20 (0.82, 1.75)	1.00 (0.70, 1.44)	1.10 (0.64, 1.89)	0.99 (0.61, 1.63)	1.15 (0.75, 1.77)	1.07 (0.65, 1.76)
≥40	1.00	1.00	1.00	1.00	1.00	1.00
Education						
Higher secondary or more	1.00	1.00	1.00	1.00	1.00	1.00
Secondary	1.01 (0.76, 1.35)	1.07 (0.83, 1.39)	1.22 (0.91, 1.65)	1.27 (0.98, 1.65)*	1.03 (0.83, 1.28)	0.98 (0.80, 1.19)
Primary	1.05 (0.79, 1.40)	1.10 (0.86, 1.40)	1.23 (0.91, 1.67)	1.30 (1.01, 1.68)**	1.00 (0.77, 1.27)	0.91 (0.73, 1.15)
None	1.11 (0.85, 1.45)	1.19 (0.94, 1.52)	1.36 (1.00, 1.85)*	1.41 (1.08, 1.84)**	1.04 (0.86, 1.25)	0.94 (0.80, 1.11)
Stature						
Not short (≥145 cm)	1.00	1.00	1.00	1.00	1.00	1.00
Short (<145 cm)	0.90 (0.75, 1.07)	0.89 (0.75, 1.05)	1.11 (0.93, 1.32)	1.12 (0.96, 1.32)	1.14 (1.03, 1.25)**	1.10 (1.00, 1.20)**
MUAC						
Not thin (≥22.5 cm)	1.00	1.00	1.00	1.00	1.00	1.00
Thin (<22.5 cm)	1.15 (1.04, 1.28)**	1.10 (0.99, 1.22)*	1.27 (1.11, 1.46)**	1.18 (1.04, 1.33)**	1.16 (1.02, 1.32)**	1.04 (0.93, 1.17)
Dietary diversity, last 7 d						
≥5 food groups	1.00	1.00	1.00	1.00	1.00	1.00
<5 food groups	0.97 (0.83, 1.13)	1.09 (0.94, 1.26)	1.28 (1.08, 1.52)**	1.45 (1.25, 1.69)**	1.07 (0.94, 1.23)	1.05 (0.92, 1.19)
Anemia status <sup>2</sup>						
Not anemic	1.00	1.00	1.00	1.00	1.00	1.00
Anemic	1.44 (1.29, 1.61)**	1.33 (1.18, 1.49)**	1.46 (1.24, 1.72)**	1.34 (1.18, 1.52)**	1.28 (1.13, 1.45)**	1.21 (1.04, 1.41)**

<sup>1</sup>Log-binomial regression models with robust SEs were used to estimate prevalence ratios. Robust Poisson models were used when models failed to converge. Each adjusted model included 1 risk factor as the exposure variable and controlled for the same set of covariates: child age and sex, maternal age, and region. \*P < 0.10, \*\*P < 0.05, \*\*\*P < 0.001. APR, adjusted prevalence ratio; MUAC, midupper arm circumference; PR, prevalence ratio.

<sup>2</sup>Anemic defined as having a hemoglobin concentration <12.0 g/dL for nonpregnant women and <11.0 g/dL for pregnant women.

**TABLE 6** Household- and community-level risk factors associated with anemia in children aged 6–59 mo by year, Nepal<sup>1</sup>

Risk factors	2013 (n = 791)		2014 (n = 777)		2016 (n = 851)	
	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)	PR (95% CI)	APR (95% CI)
Caste						
Brahmin/Chhetri	1.00	1.00	1.00	1.00	1.00	1.00
Other Tarai caste	1.24 (1.08, 1.43)**	1.09 (0.91, 1.30)	1.15 (0.90, 1.47)	0.96 (0.75, 1.23)	1.11 (0.94, 1.31)	0.89 (0.73, 1.10)
Dalit	1.13 (0.94, 1.34)	1.08 (0.91, 1.28)	1.33 (1.07, 1.65)**	1.24 (1.01, 1.53)**	1.13 (0.89, 1.45)	1.02 (0.82, 1.28)
Newar	0.87 (0.59, 1.28)	0.80 (0.55, 1.18)	0.71 (0.41, 1.24)	0.76 (0.40, 1.42)	0.60 (0.36, 1.01)*	0.62 (0.40, 0.96)**
Janajati	0.92 (0.73, 1.17)	0.91 (0.71, 1.17)	0.91 (0.70, 1.20)	0.90 (0.69, 1.16)	1.07 (0.89, 1.29)	1.02 (0.87, 1.20)
Others	1.39 (1.16, 1.67)**	1.23 (1.01, 1.49)**	1.61 (1.22, 2.14)**	1.39 (1.06, 1.82)**	1.32 (1.03, 1.70)**	1.05 (0.79, 1.40)
Food insecurity <sup>2</sup>						
None	1.00	1.00	1.00	1.00	1.00	1.00
Mild	1.17 (1.05, 1.31)**	1.16 (1.06, 1.27)**	0.98 (0.80, 1.19)	1.01 (0.83, 1.21)	1.00 (0.80, 1.26)	1.00 (0.81, 1.23)
Moderate	1.01 (0.88, 1.15)	1.03 (0.91, 1.17)	1.09 (0.89, 1.34)	1.13 (0.93, 1.37)	1.10 (0.91, 1.33)	1.08 (0.90, 1.29)
Severe	1.13 (0.92, 1.38)	1.10 (0.91, 1.35)	1.18 (0.88, 1.60)	1.20 (0.92, 1.58)	1.14 (0.90, 1.45)	1.06 (0.85, 1.32)
Livestock ownership						
Any	1.00	1.00	1.00	1.00	1.00	1.00
None	0.98 (0.86, 1.12)	1.02 (0.91, 1.14)	1.00 (0.82, 1.22)	1.02 (0.89, 1.18)	0.94 (0.83, 1.06)	0.96 (0.85, 1.09)
Animal feces in or around house						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes	1.10 (0.98, 1.24)	1.08 (0.97, 1.21)	1.10 (0.92, 1.31)	1.07 (0.93, 1.24)	1.04 (0.92, 1.18)	1.00 (0.90, 1.14)
Rubbish in or around house						
No	1.00	1.00	1.00	1.00	1.00	1.00
Yes	1.08 (0.98, 1.19)	1.08 (0.97, 1.19)	1.08 (0.91, 1.28)	1.07 (0.91, 1.25)	1.04 (0.90, 1.21)	1.03 (0.92, 1.15)
Location of child defecation						
Not open	1.00	1.00	1.00	1.00	1.00	1.00
Open	1.23 (1.08, 1.40)**	1.16 (0.99, 1.25)*	1.35 (1.14, 1.60)**	1.11 (0.95, 1.30)	1.23 (1.10, 1.37)**	1.10 (0.98, 1.24)*
Improved toilet <sup>3</sup>						
Yes	1.00	1.00	1.00	1.00	1.00	1.00
No	1.19 (1.06, 1.33)**	1.13 (1.01, 1.27)**	1.27 (1.08, 1.49)**	1.12 (1.00, 1.27)*	1.19 (1.03, 1.38)**	1.07 (0.94, 1.21)
Main drinking water source <sup>4</sup>						
Protected	1.00	1.00	1.00	1.00	1.00	1.00
Not protected	1.09 (0.96, 1.23)	1.49 (1.25, 1.77)**	1.01 (0.72, 1.41)	1.33 (0.98, 1.80)*	1.01 (0.80, 1.26)	1.23 (0.99, 1.53)*
Agroecological zone						
Mountains	1.00	1.00	1.00	1.00	1.00	1.00
Hills	0.84 (0.70, 1.01)*	0.81 (0.68, 0.97)**	0.71 (0.54, 0.93)**	0.69 (0.53, 0.91)**	0.79 (0.61, 1.02)*	0.80 (0.62, 1.03)*
Tarai	1.15 (1.00, 1.31)**	1.10 (0.96, 1.27)	1.10 (0.91, 1.34)	1.07 (0.87, 1.31)	1.13 (0.92, 1.39)	1.13 (0.28, 0.75)

<sup>1</sup>Log-binomial regression models with robust SEs were used to estimate prevalence ratios. Robust Poisson models were used when models failed to converge. Each adjusted model included 1 risk factor as the exposure variable and controlled for the same set of covariates: child age and sex, maternal age, and region. \*P < 0.10, \*\*P < 0.05, \*\*\*P < 0.001. APR, adjusted prevalence ratio; PR, prevalence ratio.

<sup>2</sup>Food insecurity assessed using the Household Food Insecurity Access Scale (30).

<sup>3</sup>Defined as flush toilet, ventilated improved pit latrine, or pit latrine with slab.

<sup>4</sup>Defined as protected ring well, bottled water, tubewell/borehole, or piped water.

this case 3 measurements over 4 y, controlling for extraneous influences such as geographical and household variation in sampling, season, and protocol and training differences. As a result, the findings robustly reflect the persistence of this public health problem and the strength and direction of risk factors. They also provide insights into the extended value of single-study estimates and the chronicity of anemia, as well as the importance for policy to maintain focus on risk factor mitigation. For example, although our findings on prevalence and associations between anemia and child age, diarrhea, stunting, dietary intake, maternal anemia status, and household WASH concur with studies elsewhere in South Asia, the current design has revealed factors that are consistently or variably associated with early childhood anemia. We were also able to distinguish areas, such as the WASH sector, that might require increased investment and better integration with traditional strategies for anemia control (60–62). Furthermore, our findings continue to support the importance of advancing socioeconomic factors that influence household access to diverse diets, quality health care, and clean water and sanitation.

Our study raises questions that require further research, such as why prevalence varied over the years assessed and why certain risk factors were significantly and/or consistently associated with child anemia whereas others were not, particularly modifiable factors like child and maternal diet and nutritional status and receipt of deworming treatment, which we would have expected to have strong associations with anemia over time. As our conceptual framework depicts, the interaction between multiple factors contributes to the etiology of anemia, making it challenging to identify and address determinants of this public health problem. Although questions remain, our identification of factors that were consistently associated with anemia provides strength to the prioritization of addressing certain risk factors when operating in low-resource communities. Our findings could also provide lessons for future assessments of child health and anemia status. For example, although we measured deworming coverage, we did not assess the prevalence of helminth infections. It has been estimated that ~12% of children 6–59 mo of age have a helminth infection in Nepal, with roundworm the most prevalent (11%), followed by hookworm (1%) and whipworm (<1%) (15). Assessing both prevalence and treatment of such infections could better elucidate how they relate to anemia risk.

Limitations of our study include the observational nature of the study design, which restricted our ability to determine causal relations, and the use of only 1 biomarker (Hb), which limited inference about the contribution of different causes to the observed anemia prevalence. We used a HemoCue device to assess Hb concentrations, which is commonly used in field settings because it is portable and relatively inexpensive. However, we recognize that there is evidence that the HemoCue results in higher Hb concentrations in children than automated hematology analyzers that are typically used in clinical settings (63). We were unable to include data from 2015, but data collected before and after the earthquake open the door for additional research exploring the effects of a natural disaster and subsequent recovery efforts on a population's health and nutrition conditions. We also did not collect data on other potential risk factors, such as genetic disorders and inflammation, in this setting. However, the sequential assessments conducted under our study have revealed new insights about changes in anemia prevalence over time, as well as the stability or variability of

associated risk factors. These insights will be key in informing policymakers as they decide where investments and scale-up efforts should focus to effectively and sustainably address the child anemia burden in Nepal.

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