

Impact of Polyparasitic Infections on Anemia and Undernutrition among Kenyan Children Living in a *Schistosoma haematobium*-Endemic Area

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Abstract. We measured prevalence of *Schistosoma haematobium*, *Wuchereria bancrofti*, *Plasmodium falciparum*, hookworm, and other geohelminths among school-aged children in four endemic villages in Kwale County, Kenya and explored the relationship between multiparasite burden, undernutrition, and anemia. In 2009–2010 surveys, cross-sectional data were obtained for 2,030 children 5–18 years old. Infections were most prevalent for *S. haematobium* (25–62%), hookworm (11–28%), and falciparum malaria (8–24%). Over one-half of children were anemic, with high rates of acute and chronic malnutrition. Associations with infection status showed significant age and sex differences. For boys, young age, low socioeconomic standing (SES), *S. haematobium*, and/or malaria infections were associated with greater odds of anemia, wasting, and/or stunting; for girls, heavy *S. haematobium* infection and age were the significant cofactors for anemia, whereas low SES and older age were linked to stunting. The broad overlap of infection-related causes for anemia and malnutrition and the high frequency of polyparasitic infections suggest that there will be significant advantages to integrated parasite control in this area.

INTRODUCTION

Coinfection with two or more parasitic infections is very common in resource-limited areas such as rural Kenya.¹ However, it was not until recently that the combined detrimental effects of polyparasitism on childhood growth and development have emerged as a research focus.^{2–6} The relationship between parasitic helminths and the subtle morbidities of undernutrition and anemia has been increasingly recognized in the past 20 years.^{4,6} Previous studies have examined the overlapping effects of infection by soil-transmitted helminths (STHs), including hookworm, *Ascaris lumbricoides*, and *Trichuris trichiura*, on these outcomes, and more recently, studies have examined the combined effects of STH with schistosomiasis.^{3,7,8} The public health importance of chronic malnutrition and anemia is in their intrinsically disabling effects. Related manifestations can often include reduced global functioning,⁹ decreased physical performance,^{10,11} and impaired cognition,^{2,12,13} resulting in decreased human capital among adults in affected populations^{14,15} with a related loss in years of healthy life.⁹ Past studies showing improvements in nutritional status and cognition after adequate antiparasitic treatment highlight the importance of effective control to prevent cognitive and growth impairment before they become irreversible.^{5,7,16} Catch-up growth (or increased linear growth velocity after growth insult resolves)¹⁷ can happen if inflammation is alleviated and chronic diseases are controlled before children mature. Likewise, anemia of inflammation, the leading cause of the anemia associated with schistosomiasis,¹⁸ can be significantly improved by curative therapy.¹⁹ The ensuing question is to define which parasites (or combination of parasites) are most clearly associated with growth impairment and anemia in areas with polyparasitism. In the present study, we present our findings from four villages in coastal Kenya

known to be coendemic for *Schistosoma haematobium*, STH, *Plasmodium falciparum*, and *Wuchereria bancrofti*.^{1,20–24}

MATERIALS AND METHODS

Study area and population. Cross-sectional data were collected from four villages, Nganja, Milalani, Vuga, and Jogo, in Coast Province, Kenya. All were known to be endemic for *S. haematobium* and other parasites (i.e., *P. falciparum* malaria, *W. bancrofti* lymphatic filariasis [LF], and STHs, including hookworms). This study, targeting children, was part of a larger community-based project studying the ecology of vector-borne and soil-transmitted parasitic infections. All children ages 5–18 years and resident of the area for more than 2 years were eligible to participate.

Subjects were enrolled at the time of the village demography survey in February, August, and November of 2009 for Nganja, Milalani, and Vuga, respectively, and in March of 2010 for Jogo. After an initial interview with the head of each household, in which general information about family structure and household living conditions were obtained, children were screened for the presence of endemic parasites, and their nutritional and fitness levels were assessed.

Ethics statement. Before study participation, written informed consent was obtained from each subject's parent or legal guardian, and individual verbal assent was obtained from participating children who were above the age of 7 years. Ethical clearance and oversight for this study were provided by the Institutional Review Board at the University Hospitals of Cleveland, Case Medical Center, and the Ethical Review Committee at the Kenya Medical Research Institute (KEMRI). Parasitic infections detected during the course of this survey were treated with antimalarials Artemisinin Combination Therapy (ACT), Diethylcarbamazine (DEC)/albendazole, albendazole, or praziquantel at age-appropriate doses, as indicated for each individual's testing outcomes.

Urine examination. Egg burden for *S. haematobium* was assessed by Nuclepore urine filtration.²⁵ The presence of gross hematuria was also recorded. The subjects provided a

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single mid-morning urine specimen that was processed the same day. Three intensity categories were assigned as follows: negative for detectable eggs; light for 1–50 eggs/10 mL urine; or heavy for > 50 eggs/10 mL urine.

Stool examination. Eligible subjects were given a stool container by local community health workers the night before the parasitology survey. The following morning, stool samples were taken to the central facility and examined in duplicate by the quantitative Kato–Katz method for microscopic detection of eggs.²⁶ For each stool specimen, eggs per 1 g feces (epg) were determined to quantify intensity of hookworm infection. The stool samples were processed and scored within 10–20 minutes to provide optimal detection of hookworm ova. Other STH eggs, such as for *A. lumbricoides* or *T. trichiura*, were scored as present or absent.

Blood collection and processing. Finger prick blood collection was performed in all eligible children. None refused blood draw. The blood was used to measure hemoglobin (Hemocue, Ångelholm, Sweden) and perform rapid antigen testing for *Pf* malaria (ICT Diagnostics, Australia) and LF (Binax, Portland, ME). After hemoglobin determination, anemia and severe anemia were categorized according to World Health Organization (WHO) criteria for age and sex and scored as present or absent for each child.²⁷

Standardized anthropometric testing. Because growth is considered the best indicator of nutritional status in children, calibrated measurements of height and weight were used as study outcomes to assess developmental morbidity among study participants. Before the surveys, all technical staff performing anthropometric measurements received standardization training followed by independent reliability assessment. Supervised by a trained anthropometrist, trainees performed replicate measurements of height (until agreement within 0.5 cm) and weight (agreement within 1.0 kg) on 10 healthy volunteer children on the same day. The results were then compared for inter- and intraobserver reliability. The trainees' intra- and interexaminer technical errors of measurement fell within reference values^{28–30} and were, therefore, considered sufficiently accurate for individual morbidity assessment.

Eligible study children were measured according to procedures described by Jelliffe³¹ while wearing a kanga—a traditional light cloth—wrapped around their bodies. Weight was obtained by digital weight scale (model 803; SECA, Hanover, MD) and rounded to the nearest 0.1 kg. Height was measured with the use of a stadiometer (model 214; SECA, Hanover, MD), and measurements were read to the nearest 1.0 cm. Instruments were calibrated daily before use. Every measurement was performed two times, and the mean values were used for analysis. Reference population Z scores were calculated for each subject's height for age (HAZ) and body mass index for age (BAZ) using international reference standards for comparison taken from the WHO's Anthro-Plus program for ages 5–19 years (WHO, Geneva, Switzerland) with reference growth standards from the year 2006.^{32,33}

HAZ is considered an indicator of long-term linear growth, whereas BAZ variations better reflect acute changes in nutritional status. According to WHO standards,³² stunting was categorized as an observed HAZ that was 2 or more SDs below average (HAZ score ≤ -2). Children were categorized as clinically wasted if their BAZ was more than 2 SDs below average for their age (BAZ score \leq

-2). Children were further identified as severely wasted if their BAZ was ≤ -3 .

Data management and statistical analysis. Demographic data collected in the field were entered in handheld devices (Dell Axim, Round Rock, TX) using Visual CE 10 (Cambridge, MA) backed up on paper forms. Both sets of duplicate data were then transferred into ACCESS 2007 (Microsoft, Seattle, WA), and the databases were compared for errors. Parasitology and anthropometric data were similarly entered to complete the database. Exploratory analysis started with univariate distributions followed by bivariate analyses to explore the pair-wise relationships of individual outcomes. Hookworm egg counts were log-converted to adjust for their skewed distribution. Analysis of variance (ANOVA) or χ^2 testing was performed to assess the significance of differences detected among the study villages. Later, multivariable analysis was used to assess the significance of associations controlling for age, sex, infection, coinfection status, and a scale of household socioeconomic standing (SES) derived using principle component analysis (PCA) of combined asset scores.^{10,34} As dependent variables, the presence or absence of morbidity outcomes anemia, wasting, and stunting were modeled by applying binary logistic regression with generalized estimating equation (GEE) modeling to account for household-level clustering effects. A final model for each logistic regression was chosen after all variables in the model were either statistically significant or biologically plausible and marginally significant. This latter approach was based on previous published research on the relationship between different STH, schistosomiasis, and anemia³⁵ and the synergistic effects of malaria and schistosomiasis coinfection.³⁶ Two analytic approaches were taken: the first approach included village as a covariate in the model to account for climatic variations, whereas the second approach clustered villages by schistosomiasis risk. In this paper, only the latter is presented in detail, because of the two approaches, it provided the best-fit parsimonious models based on information criteria.

RESULTS

Population characteristics. The demographic characteristics and parasitological findings for study participants are summarized in Table 1, with their hematologic and anthropometric outcomes summarized in Table 2. In all, 2,030 children, ages 5–18 years, were surveyed. Of these children, 2,013 had full parasitological and anthropometric data and were included in the final multivariable analysis; 76% of all eligible children in Nganja (235/309), 51% of all eligible children in Milalani (416/822), 74% of all eligible children in Vuga (726/983), and 74% of all eligible children in Jego (653/890) participated in the surveys.

Parasite burden and polyparasitism. There were significant differences among villages in terms of *S. haematobium* prevalence. Two of them (Nganja and Milalani) had significantly higher prevalence of active *Schistosoma* infection, with over 60% of school-aged children positive on urine filtration testing, whereas the other two villages (Vuga and Jego) had lower prevalence (25%) (Table 1). The high-prevalence villages also had the greatest prevalence of polyparasitism, with over 30% of children coinfecting with *S. haematobium* and one or more STH (Figure 1 and Table 1). Figure 1 shows the proportion of

TABLE 1
Demography and distribution of parasite infection among study children in four Kwale County villages, Kenya

| | Total (N = 2,030) | Nganja (N = 235) | Milalani (N = 416) | Vuga (N = 726) | Jego (N = 653) | P value* |
|---|----------------------|---------------------|-----------------------|-------------------|-------------------|----------|
| Demographic | | | | | | |
| Age mean in years (range) | 11.0 (5–19) | 11.2 (5–19.5) | 11.1 (5–19) | 11.6 (5–19) | 10.4 (5–18) | 0.0715 |
| Female | 48% | 45% | 51% | 51% | 46% | 0.0852 |
| Parasitology prevalence | | | | | | |
| <i>S. haematobium</i> | 37% | 62% | 62% | 25% | 25% | < 0.0001 |
| Heavy intensity (> 50 eggs/10 mL urine) | 20% | 40% | 32% | 12% | 14% | 0.0095 |
| Light intensity (1–50 eggs/10 mL urine) | 17% | 21% | 30% | 13% | 11% | < 0.0001 |
| Hookworm | 20% | 23% | 28% | 11% | 24% | < 0.0001 |
| <i>P. falciparum</i> (ICT card positivity) | 16.4% | 8.5% | 18% | 11% | 24% | < 0.0001 |
| <i>W. bancrofti</i> | 9.8% | 6.4% | 9% | 16% | 4.3% | < 0.0001 |
| <i>A. lumbricoides</i> | 0.5% | 0.4% | 0.7% | 0.2% | 0.3% | 0.8006 |
| <i>T. trichiura</i> | 18.1% | 37% | 37% | 8.9% | 10% | < 0.0001 |
| <i>S. haematobium</i> intensity mean eggs/10 mL | 109.4 | 195.6 | 138.3 | 52.3 | 51.4 | < 0.0001 |
| Hookworm intensity mean egg | 6.1 | 7.7 | 10.9 | 1.1 | 4.8 | < 0.0001 |
| Coinfection | | | | | | |
| <i>S. haematobium</i> – <i>Trichuris</i> | 9.6% | 25% | 23.5% | 2.6% | 3.2% | < 0.0001 |
| <i>S. haematobium</i> –hookworm | 9.3% | 15.3% | 17.5% | 4.2% | 7.6% | < 0.0001 |
| <i>S. haematobium</i> – <i>Pf</i> malaria | 6.9% | 6.4% | 14.9% | 2.9% | 6.7% | < 0.0001 |
| <i>S. haematobium</i> –filaria | 4.2% | 5.1% | 6.7% | 5.2% | 1.2% | < 0.0001 |
| Hookworm– <i>Trichuris</i> | 6% | 10.2% | 14.2% | 1.5% | 4.3% | < 0.0001 |
| Hookworm– <i>Pf</i> malaria | 4.7% | 3.4% | 6.9% | 1.2% | 7.6% | < 0.0001 |
| Hookworm–filaria | 1.3% | 1.3% | 2.1% | 1.2% | 1.1% | 0.4818 |

* P value refers to significance of differences among the villages by ANOVA or χ^2 testing.

ICT = rapid immuno-chromatography test for malaria. High- and low-risk villages refer to high and low *S. haematobium* prevalence, respectively.

children infected with one, two, three, or four or more parasites in the different study villages.

The age distribution of the different parasite infections is shown in Figure 2. *S. haematobium* was the most common infection in all age groups followed by hookworm and *Trichuris*. *P. falciparum* malaria was common in all ages, but most prevalent among 11- to 12-year-old children. LF was most common among older children, particularly 17- to 18-year-old children, perhaps reflecting the impact of LF elimination campaigns that have been active in the area since 2003.

Morbidity outcomes: anemia, stunting, and wasting. Table 2 summarizes the hematologic and anthropometric findings of the children surveyed. Over one-half (50.8%) of the children studied were anemic, and 1.1% were severely anemic. Hemoglobin levels increased with age but varied inversely with intensity of *S. haematobium* infection. Overall, the nutritional

status of the children was poor, with a high prevalence of both acute and chronic undernutrition reflected as wasting (BAZ ≤ -2) and stunting (HAZ ≤ -2), respectively. The prevalence of wasting and anemia was significantly higher among boys than girls in all villages (see below). In multivariable analysis, the significant interaction of sex with other covariates of our morbidity outcomes led us to stratify all subsequent analysis by sex.

Anemia. For both boys and girls, significant bivariate associations were found between anemia and age, single infections (*Pf*malaria, *S. haematobium*, and hookworm), and polyparasitic infections (*S. haematobium*–hookworm, *S. haematobium*–*Pf* malaria, and hookworm–*Pf* malaria). Results are summarized in Figure 4 and Supplemental Table 1. Multivariable logistic regression modeling, accounting for household clustering, indicated that younger boys (5–6 years) were significantly more

TABLE 2
Hematologic and anthropometric characteristics of children surveyed in Kwale County, Kenya

| | Total (N = 2,030) | Nganja (N = 235) | Milalani (N = 416) | Vuga (N = 726) | Jego (N = 653) | P value* |
|------------------------------|----------------------|---------------------|-----------------------|-------------------|-------------------|----------|
| Hematology | | | | | | |
| Percent anemic† | 50.8% | 47.5% | 50.7% | 45.2% | 58.4% | 0.0567 |
| Severely anemic‡ | 1.1% | 2.1% | 1.9% | 0.8% | 0.6% | 0.0961 |
| Mean hemoglobin (range) | 11.7 (3.4, 15.8) | 11.9 (4.8, 17) | 11.8 (6.3, 15.7) | 11.9 (5.2, 15.9) | 11.9 (3.4, 15.8) | 0.0217 |
| Anthropometrics | | | | | | |
| Mean HAZ (range) | –1.4 (–6.6, 7) | –1.9 (–5.7, 1.9) | –0.98 (–3, 1.9) | –1.37 (–6, 7) | –1.4 (–6.6, 2.6) | 0.0008 |
| Mean BAZ (range) | 0.99 (–6, 4.1) | –1.2 (–3.7, 0.9) | –0.83 (–4.2, 2.8) | –1.15 (–4.8, 4.1) | –0.8 (–6, 4.6) | 0.0019 |
| Wasting§ (%) | | | | | | |
| WHO reference standards | 19.2% | 18.7% | 12.9% | 30.8% | 10.4% | < 0.0001 |
| Stunting¶ (%) | | | | | | |
| WHO reference standards | 36% | 45.4% | 35.3% | 43.0% | 25.5% | < 0.0001 |
| Severely wasted** (%) | | | | | | |
| WHO reference standards | 6.4% | 3.3% | 2.8% | 13.6% | 2.0% | < 0.0001 |

* P value refers to significance of differences in prevalence among the villages by ANOVA or χ^2 testing.

† Anemia: hemoglobin (Hb) for age < 12 years, Hb < 11.5 g/dL; for age > 12 years, Hb < 12 g/dL, except for males > 15 years, Hb < 13 g/dL.

‡ Severely anemic: Hb < 8 g/dL.

§ Wasting: BAZ ≤ -2 .

|| WHO 2007 growth reference charts.

¶ Stunting: HAZ ≤ -2 .

** Severely wasted: BAZ ≤ -3 .

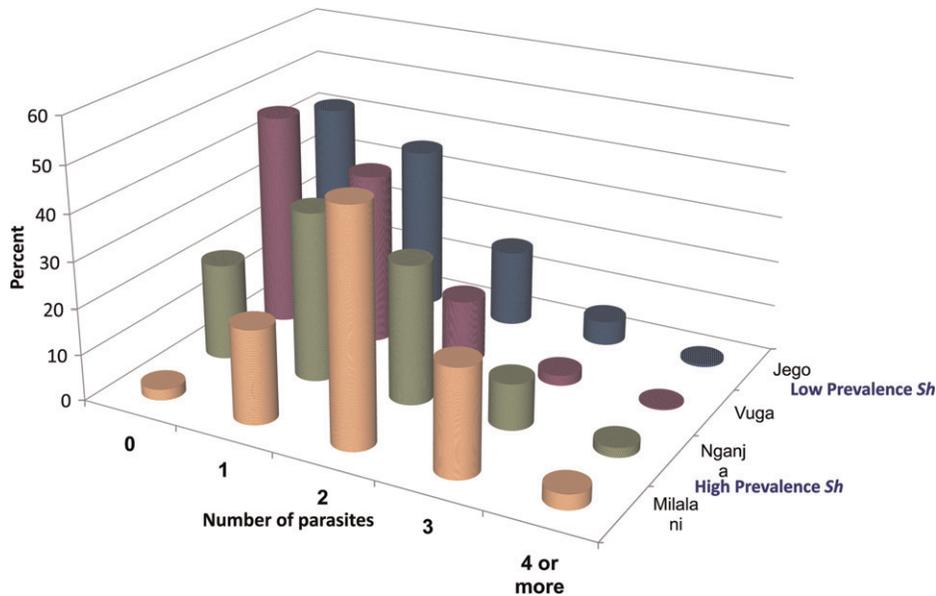


FIGURE 1. Percentage of children per village coinfected with different parasites.

anemic than the reference group of 17- to 18-year-old children ($P = 0.026$). In addition, both heavy- and light-intensity *S. haematobium* infection and *Pf* malaria were independently associated with anemia among boys. With respect to household resources, boys belonging to the fourth poorest stratum of households were also significantly more anemic. Although hookworm infection and combined infections with *S. haematobium*–hookworm, *S. haematobium*–*Pf* malaria, and hookworm–*Pf* malaria were significantly associated with anemia among boys in the unadjusted analysis, this significance was not retained after multiple adjustment in the multivariable model.

Among girls, odds of anemia were significantly greater in older girls (17–18 years) compared with 7- to 8-year-old children. Like among boys, heavy-intensity *Schistosoma* infection was independently associated with girls’ anemia. Different from what was found among boys, for girls, light *S. haematobium* infection was not a significant covariate for anemia. In bivariate analysis, coinfection with *S. haematobium*–hookworm or *S. haematobium*–*Pf* malaria was linked to anemia in girls, but

this relationship was no longer significant when adjusted for other covariates.

Severe anemia. Bivariate analysis indicated significant associations between *S. haematobium* infection (either heavy or light intensity) and severe anemia. A significant link was seen for hookworm infection as well. Among boys, a strong interaction was seen between hookworm and *Pf* malaria as predictors of severe anemia (odds ratio [OR] = 5.7, 95% confidence interval [CI] = 1.6, 20.1, $P = 0.0007$) that was not found among girls. Being resident of a high-risk village was a significant predictor of severe anemia among girls (OR = 3.7, 95% CI = 1.4, 9.6, $P < 0.0001$). However, after adjusting for other single and multiple infections, these associations were no longer significant. Figure 4 summarizes the findings.

Acute malnutrition (wasting). In exploratory bivariate analysis, both boys and girls with *S. haematobium* infection, *Pf* malaria, filaria, or *Trichuris* (data not shown) and boys infected with *Pf* malaria and coinfecting with *S. haematobium*–filaria were likely to be wasted. Overall, there were increased levels of malnutrition in older children, which

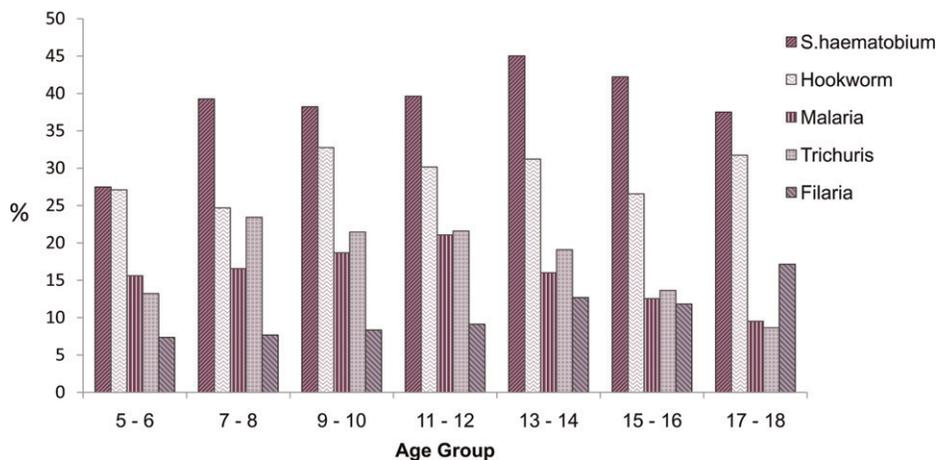


FIGURE 2. Parasite prevalence by age groups.

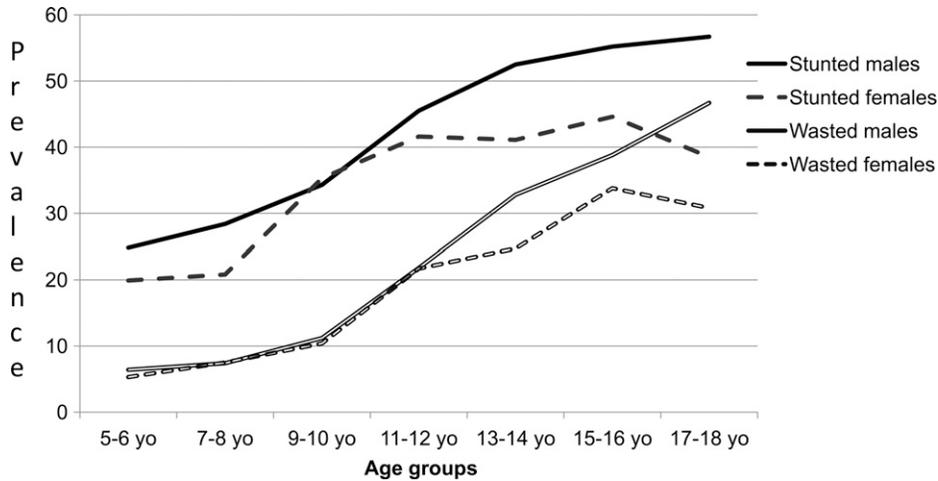


FIGURE 3. Malnutrition distribution by age and sex.

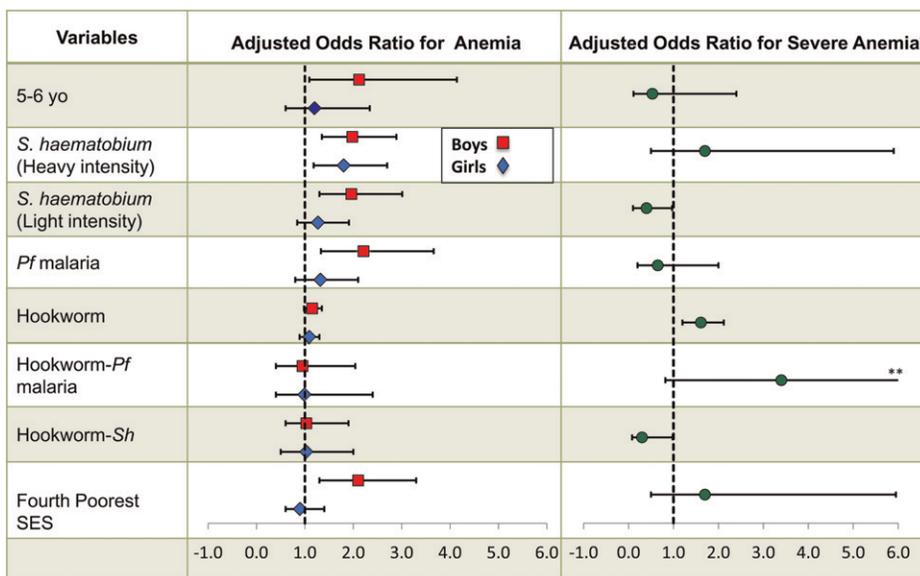
was more evident in boys than girls (Figure 3). As shown in Figure 5 and Supplemental Table 2, on multivariable adjustment and stratification by sex, only boys had greater odds of wasting in the presence of light-intensity *Schistosoma* infection. An independent additive effect was significant when boys were coinfecting with *S. haematobium* and *Pf* malaria ($P = 0.015$). Older age (> 10 years) was a significant correlate of wasting in both boys and girls. Girls who were (1) older than 10 years, (2) residents of a low-risk village, or (3) or residents of the poorest stratum of households were significantly more likely to be wasted.

Chronic malnutrition (stunting). In bivariate analysis, older age and *S. haematobium* infection were found to be associated with chronic undernutrition. Low SES and village were predictors of stunting in both boys and girls in unadjusted analysis. After sex-stratified multivariable analysis (Figure 5 and Supplemental Table 3), age over 8 years and belonging to a poor

(lowest SES) household were significant predictors of stunting for both boys and girls. Only girls (but not boys) coinfecting with *S. haematobium*-*Pf* malaria were more likely to be stunted. However, this effect was marginally significant in multivariable analysis ($P = 0.06$). In assessing the impact of individual infections, analysis of the effects of infection by *S. haematobium* yielded opposite results for boys and girls: among boys, light-intensity *Schistosoma* infection was associated with stunting, whereas uninfected girls were more likely to be stunted. We also found that boys who resided in high-risk villages were more likely to be chronically undernourished.

DISCUSSION

Among children, anemia and undernutrition can occur through many possible pathways. Although much of impaired



Anemia Logistic Regression final model was stratified by gender and included age, *Sh* heavy and light intensity infection, hookworm, *Pf* malaria, co-infection *Sh*-hookworm, *Sh*-*Pf* malaria, Hookworm-*Pf* malaria and SES.

*Severe anemia logistic regression model (Hb < 8 gr/dl) combined both genders.

** For Hookworm-*Pf* malaria co-infection 95 % CI was truncated in the figure; OR: 3.39, 95 % CI (0.8, 13.9), $P=0.09$.

FIGURE 4. Adjusted ORs for anemia and severe anemia.

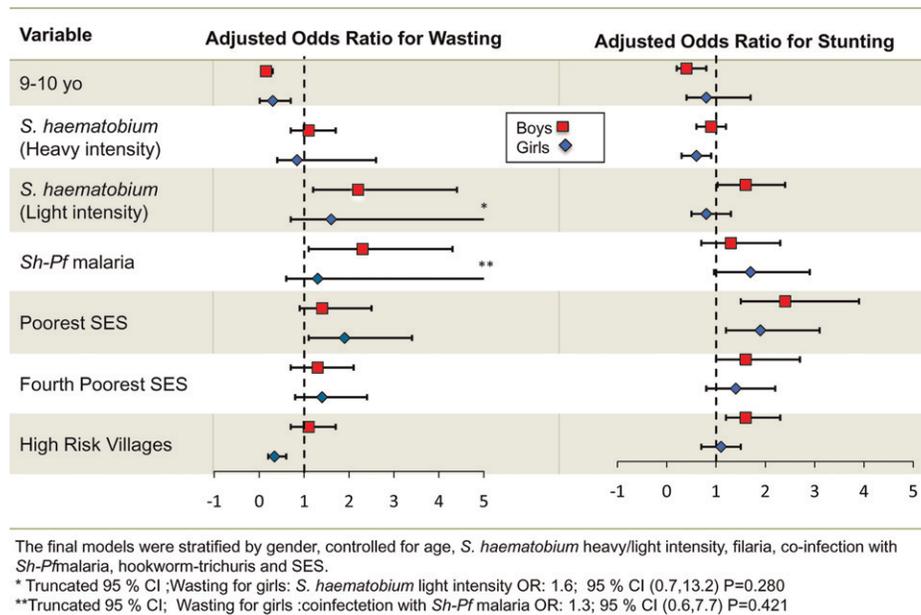


FIGURE 5. Adjusted ORs for acute (wasting) and chronic malnutrition (stunting).

early childhood development in sub-Saharan Africa has been ascribed to protein, calorie, and micronutrient deficiencies, it is becoming increasingly apparent that the process of chronic parasitic infection is associated with continuing inflammation that can limit childhood growth^{6,7,37} and cause persistent or recurrent anemia of chronic inflammation.¹⁸ Despite the difficulties in isolating individual causes of undernutrition and anemia in developing areas, we were able to show a clear association between infection and both anemia and undernutrition after adjusting for sex-specific effects and possible SES and environmental confounders.

Results of our surveys show an alarmingly high prevalence of anemia in an area where malaria, a major contributor to childhood anemia, is now decreasing in frequency.³⁸ Anemia has been acknowledged as a major public health threat,²⁷ with multiple measurable downstream effects on physical and cognitive function in children as well as risk for low-birth weight pregnancies and increased prematurity. Iron deficiency is typically considered the most common etiology of acquired anemia. However, in rural areas such as Kwale County, other mechanisms must be considered, particularly those mechanisms related to inflammation caused by endemic parasite infections.¹⁸ Boys surveyed in our study showed a consistent association between light-intensity *Schistosoma* infection, anemia, and both acute and chronic undernutrition. These findings suggest that a proinflammatory state, occurring even with low-level parasite burden, can lead to chronic morbidity.¹⁹ This process could undoubtedly lead to development of irreversible pathology beginning in early life. The underlying mechanisms for this process are not fully explored.³⁹ Parasite migration, persistence in the circulation, egg retention, and consequent inflammatory response by the host is a plausible mechanistic pathway; however, more research is needed in this area.

We found important age and sex differences in the association between infection, environmental effects, and anemia. Younger boys (5–6 years) and older girls (17–18 years) were more likely to be anemic. Post-menarchal girls presumably have a higher blood loss during their monthly cycle, which

could exacerbate the mixed anemia caused by *Schistosoma* infection.⁴⁰ If anemia is present in young school-aged boys (5–6 years), it is more than likely caused by events occurring at a younger age. The reality of early childhood helminth infection is gaining better recognition, because recent surveys in Uganda, Kenya, Niger, and Ghana show a *Schistosoma* egg shedding prevalence of over 50% in pre-school-aged children in high-risk areas.^{41–44} Of note, this population has not been reliably included in schistosomiasis control programs.⁴⁵ It is also important to note that adolescents require attention in this respect as well,⁴⁶ which is highlighted in our results for older girls. In our study, we noted that severely anemic boys were more likely to be coinfecting with hookworm and *Pf* malaria, indicating the need for comprehensive intervention to prevent severe pathologic outcomes.

Polyparasitic interactions have been previously shown to negatively affect the growth of children,⁴⁷ although the relationship with chronic growth failure (stunting) has not been as clear.^{47,48} This gap in the association with chronic parasitic infection is probably caused by the prolonged lag (time elapsed) between initial infection and its effect on linear growth and the detection of growth failure (HAZ < -2), which takes months to years to become manifest.³⁷ Causality is, therefore, difficult to establish because of a multiplicity of potential confounders, particularly those confounders associated with poor diet, poverty, and low SES.⁴⁹ In our study, unmeasured variation in cofactors among the different villages and lower participation in Milalani could have added undetected bias. Seasonal variation in malaria transmission risk could have affected the observed outcomes. We had previously found no predictable seasonal pattern in 2009–2010 malaria transmission based on monthly or quarterly rainfall.³⁸ Other location-specific climatic variations are expected to be reflected embedded in the village-level factors included in the analysis.

Despite the above limitations, our results clearly show (1) a marked sex difference, with boys being more undernourished than girls, which is in agreement with other studies,⁸

and (2) that boys and girls with polyparasitic infections (*Pf* malaria–*S. haematobium*) had higher odds of being wasted and stunted, respectively. The impact of SES in all of our study's morbidity outcomes points to a social stratification in an already impoverished area. These effects were better seen in acute undernutrition rather than stunting, likely because of the shorter time from infection to disease for the wasting outcome.

One limitation of our surveys is the lack of data regarding enteric infections. The relationship between diarrheal diseases and stunting is well-known^{50,51} and could potentially have served as additional causes of stunting in our study communities. Also, single urine and stool sampling is likely to miss a variable proportion of each helminth infection, leading to some misclassification bias. Causality cannot be established with cross-sectional data, which was presented in our study. However, there is a strong suggestion that parasitic infections in childhood have a consistent association with growth and hemoglobin status in the school-aged child. Our previous report has detailed the impact of anemia and stunting on the physical fitness of Kwale County children.¹⁰ We believe that these effects can be reversed if adequate treatment is in place, particularly if it is associated with decreased risk for reinfection,³⁷ because children have a limited window for growth rebound (catch-up growth) before reaching puberty that would allow them to achieve normal or nearly normal adult physical growth parameters if they remain free from infection.^{17,37,52}

The high prevalence of anemia and malnutrition in our villages and the associations found between single or multiparasite infections and childhood morbidity suggest a serious need for integrated parasite control efforts. The reversibility of the morbidity outcomes presented here will only be determined with adequate antiparasitic treatment delivered through committed, integrated control programs that are, to date, still absent from this area.

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