Soil-Transmitted Helminth Infection and Nutritional Status Among Urban Slum Children in Kenya

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Abstract. To evaluate the nutritional impact of soil-transmitted helminth (STH) infection, we conducted a cross-sectional survey of 205 pre-school (PSC) and 487 school-aged children (SAC) randomly selected from the surveillance registry of the Centers for Disease Control and Prevention of the Kibera slum in Kenya. Hemoglobin, iron deficiency (ID), vitamin A deficiency (VAD), inflammation, malaria, anthropometry, and STH ova were measured. Poisson regression models evaluated associations between STH and malnutrition outcomes and controlled for confounders. Approximately 40% of PSC and SAC had STH infection, primarily *Ascaris* and *Trichuris*; 2.9% of PSC and 1.1% of SAC had high-intensity infection. Malnutrition prevalence among PSC and SAC was anemia (38.3% and 14.0%, respectively), ID (23.0% and 5.0%, respectively), VAD (16.9% and 4.5%, respectively), and stunting (29.7% and 16.9%, respectively). In multivariate analysis, STH in PSC was associated with VAD (prevalence ratio [PR] = 2.2, 95% confidence interval = 1.1–4.6) and ID (PR = 3.3, 95% confidence interval = 1.6–6.6) but not anemia or stunting. No associations were significant in SAC. Integrated deworming and micronutrient supplementation strategies should be evaluated in this population.

INTRODUCTION

Soil-transmitted helminths (STHs) are parasitic intestinal worms that infect more than one-half of the world's population.[1] The most common species are *Ascaris lumbricoides*, *Trichuris trichiura*, and hookworm, which chronically infect children and are associated with significant physical, cognitive, and economic deficits.[2] Infection with STH causes more morbidity than death, contributing to as many as 405,000 deaths and 37 million disability-adjusted life years (DALYs) annually.[3]

World Health Organization (WHO) guidelines for STH control generally focus on routine mass drug administration for school-aged children, aiming primarily to reduce the prevalence of high-intensity infection.[4] Although school-based treatment is currently a priority, pre-school children also experience substantial morbidity and may benefit from deworming.[5,6] Another challenge for STH control is how to target interventions given the geographical heterogeneity of infection in countries, communities, and individuals, in which different species of STH often co-occur.[7] Guidance is also needed for urban slums, which house nearly one-third of the world's population and are less studied for STH interventions.[8]

The proven burden of chronic STH infection is primarily related to malnutrition and impaired growth.[1,9] For example, there are proven effects of STH infection on iron status, vitamin A status, anemia, and growth that are mediated through various mechanisms, including malabsorption, inflammation, and reduced food intake from reduced appetite.[1,5,9] However, few studies have explored the association of STH and malnutrition while accounting for multiple covariates and severity of STH infection.[9] Furthermore, the data on the burden of STH in the urban slum populations are limited. We, there-fore, aimed to evaluate the association of STH infection with anemia, iron deficiency, vitamin A deficiency, and growth among populations of pre-school– and school-aged children living in an urban slum in Kenya. We also aimed to evaluate the association of STH and inflammation to explore potential mechanisms by which STH infection may cause malnutrition.

METHODS

Study population and sample. The study was conducted in Kibera in southern Nairobi, Kenya, which is one of the largest contiguous urban slums in Africa.[10] The Centers for Disease Control and Prevention's International Emerging Infections Program (IEIP), in collaboration with the Kenya Medical Research Institute (KEMRI), has conducted community-based morbidity surveillance in Kibera since 2005.[11] IEIP performs active household surveillance for major infectious disease syndromes for approximately 28,000 participants living in 2 of Kibera's 13 villages. Field workers make home visits every 2 weeks to ask about recent illnesses; they also maintain an up-to-date registry by enrolling new arrivals and updating records to reflect outmigration.[10,11] Kibera is characterized by high population density, semipermanent housing, and lack of official city water or sewage services. Participants can access free healthcare at a centrally located clinic staffed by study-supported trained personnel and other clinics run by non-governmental organizations and government institutions bordering the slum.

Among households in the IEIP participant registry, approximately 25% of households were designated as potential sources for enrollment of pre-school children (PSC; ages 6–59 months), and the other 75% of households were designated as potential sources for enrollment of school-aged children (SAC; ages 5–14 years); this designation ensured that PSC and SAC were not from the same household. Households were then selected with probability proportional to size from each target group, and one child was randomly chosen from

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each selected household. Sampling weights were calculated to account for non-response and post-stratified to the IEIP cluster size populations. Sample size was capped by the laboratory capacity for daily processing of fresh stools for STH. At 80% power and accounting for 20% non-response, the achievable target sample size of 293 PSC and 899 SAC was powered to detect an odds ratio of 2.0 for STH infection associated with having an infected sibling.

Written informed consent was obtained from all participating households. Subjects with anemia, positive malaria test, wasting, or STH infection were referred for free care at the nearby IEIP-run clinic. Institutional review boards of KEMRI and the Centers for Disease Control and Prevention (CDC) approved this study.

Data collection. Fieldwork took place from April to June of 2012. Trained fieldworkers administered a mobile device-based questionnaire to obtain demographic and socioeconomic data and child breastfeeding, deworming, and nutrition supplementation history. Anthropometric measurements of height and length were taken using a wooden measuring board accurate to 0.1 cm (Irwin Shorr Productions, Olney, MD). Weight was measured to the nearest 0.1 kg using a digital scale (Seca Corp, Hanover, MD). Trained fieldworkers completed the measurements using standard techniques. Capillary blood samples were collected for hemoglobin (Hb) measurements, malaria testing, and analysis of micronutrient and inflammatory status.

A goal of three stool samples was collected from selected children. Stools were collected over 3 consecutive days when possible, accepted only if produced after midnight the previous night, and maintained in cool boxes before delivery to the laboratory for analysis before 14:00 hours each day. Stool samples were processed at KEMRI using the Kato–Katz technique. Two slides were prepared per sample; approximately 7% of all slides were reread for quality control.

Details of the nutrition laboratory analysis are described elsewhere.[12] In brief, Hb was determined using a HemoCue Hb301 machine (Ängelholm, Sweden). Anemia was defined by age and corrected for altitude according to WHO guidelines[13]: Hb < 11.0 g/dL in children 6–59 months of age, Hb < 11.5 g/dL in children 5–11 years of age, and Hb < 12 g/dL in children 12–14 years of age; 0.6 g/dL was added to cutoffs to account for an altitude of 1,800 m. A rapid diagnostic test kit (SD BIOLINE Malaria Ag P.f/Pan, Hagal-dong, Korea) was used to test for malaria. Blood samples were centrifuged, and frozen plasma samples were transported to VitA-Iron Tech (Willstaett, Germany), where levels of ferritin, retinol binding protein (RBP), C-reactive protein (CRP), and α-1-acid glycoprotein (AGP) were measured by sandwich enzyme-linked immunosorbent assay (ELISA).[14] The following thresholds were used to define abnormal values for these biochemical indicators: ferritin < 12 µg/L in PSC and ferritin < 15 µg/L in SAC[15]; RBP < 0.7 µmol/L; CRP > 5 mg/L; AGP > 1 g/L.[16] Because biomarkers of nutrition are influenced by inflammation, iron deficiency, vitamin A deficiency, and anemia were defined using a correction factor approach to adjust ferritin, RBP, and Hb values for the presence of inflammation, which was described previously.[16,17] Because the prevalence of inflammation varied by age, separate correction factors were calculated for PSC and SAC. Among PSC, correction factors for ferritin, RBP, and Hb were as follows: 0.87, 1.0, and 0.91 for early inflammation (elevated CRP and normal AGP);

0.36, 1.51, and 1.08 for early convalescent inflammation (elevated CRP and AGP); 0.78, 1.1, and 1.0 for late convalescent inflammation (elevated AGP and normal CRP). Among SAC, correction factors for ferritin, RBP, and Hb were as follows: 0.70, 1.36, and 1.01 for early inflammation; 0.66, 1.36, and 1.09 for early convalescent inflammation; 0.99, 1.21, and 1.08 for late convalescent inflammation.

Statistical analysis. We used the WHO Child Growth Standards (WHO Anthro, Geneva, Switzerland) to calculate z-scores and categorized underweight as a weight-for-age z-score of < −2, stunting as a height/length-for-age z-score of < −2, and wasting as a body mass index (BMI)-for-age z-score of < −2 to allow for comparisons between PSC and SAC. We used the weekly expenditure on food by the household to classify respondents by socioeconomic status. Children were classified as having received or not received vitamin A supplementation in the past 1 year based on written documentation when available or otherwise, respondent recollection.

Statistical analyses were performed using R version 2.15.2 (R Core Team, 2012). The survey package was used to incorporate survey weights. Comparisons between PSC and SAC were performed with χ^2 test. Means and standard deviations (SDs) were computed for continuous variables, and t test was used to compare means. Multivariable analyses used Poisson regression to provide prevalence ratios (PR).[18] All analyses were run separately for PSC and SAC.

Five nutritional outcomes were investigated: anemia, vitamin A deficiency (VAD), iron deficiency (ID), iron deficiency anemia (IDA), and stunting. Inflammation was also investigated as an outcome (e.g., elevated CRP and/or elevated AGP). These outcomes were dichotomized according to previously described cutoffs. Several alternative measures of the primary exposure (STH infection) were used: presence/absence of any STH infection; presence/absence of species-specific infection with *Ascaris* or *Trichuris*; presence/absence of coinfection with both *Ascaris* and *Trichuris*; and intensity of infection (none, light, medium/moderate, or high according to standard egg per 1 gram-based WHO cutoffs[19] using the highest-intensity species found in the case of coinfection, with eggs per 1 gram determined as an average among all the subjects' stools). Demographic and clinical confounders were determined independently for each outcome *a priori* based on knowledge from published literature that they were potential predictors of the various nutritional outcomes. Demographic factors considered were age, sex, maternal education, ethnicity, socioeconomic status, and breastfeeding. Clinical factors included were malaria, stunting, wasting, ID, and VAD. To test the robustness of the model, we also performed a sensitivity analysis for the VAD outcome, which included history of recent vitamin A supplementation.

Unadjusted prevalence ratios and 95% confidence intervals were computed for association between a specific outcome and primary exposure. Age, sex, and socioeconomic status were forced in the models as potential confounders. When the primary exposure was *Ascaris* infection, presence of *Trichuris* infection was also forced in the models. The decision to include the other demographic variables and clinical variables in the models was based on at least a 10% change in the prevalence ratio of the primary exposure. Variables that were originally continuous were treated as both continuous and dichotomized variables in separate models. The results of these models were compared to assess residual confounding and

whether associations were linear. The best models selected were those models with confidence intervals that were more precise.

RESULTS

In total, 1,192 children were targeted as potential study participants, and 844 children were enrolled. The primary reasons for non-enrollment were either household members were absent ($N = 216$) or the individuals refused to participate ($N = 132$). An additional 132 children were excluded because of missing laboratory or stool data, and 20 children withdrew, which led to a total of 692 children (205 PSC and 487 SAC) in the final analysis (net response rate of 58%). Age, sex, and other demographics were similar among enrolled and non-enrolled children (data not shown).

The demographic and health characteristics of enrolled PSC and SAC are shown in Table 1. There were no significant differences between the two groups in most demographic characteristics. Most children were of Luo ethnicity, and over one-half were, by self-report, dewormed in the last year. Mean weekly per capita expenditure on food was USD $5.40 in PSC households compared with USD $4.90 in SAC households ($P = 0.03$). Approximately 40% of PSC and SAC had any STH infection, primarily consisting of *Ascaris* and *Trichuris* species. There was only one hookworm infection. The overall prevalence of high-intensity STH infection was low, with no greater high-intensity infection with *Ascaris* in PSC compared with SAC (2.9% versus 1.1%, $P = 0.09$). There were no cases of high-intensity infection with *Trichuris*, and of the *Trichuris* infections, 97% were light intensity infections (data not shown).

For nutritional status, compared with SAC, PSC had a significantly higher prevalence of anemia, ID, VAD, inflammation, and stunting (Table 1). Although the prevalence of wasting was overall low (6.8%), the prevalence was higher in SAC compared with PSC ($P < 0.0001$). The prevalence of any inflammation (either elevated CRP or AGP) was higher in PSC at 48.9% (compared with 15.8% in SAC; $P < 0.0001$).

TABLE 1
Demographic and health characteristics of enrolled PSC and SAC in the Kibera slum, Kenya, 2012

Characteristics	PSC ($N = 205$)		SAC ($N = 487$)		
	Number*	Percent or mean†	Number	Percent or mean†	P value‡
Age, years (mean)	205	3.2 ± 0.08	487	11.4 ± 0.13	< 0.001
Boys (%)	98	47.4	228	46.8	0.89
Weekly per capita food spending per household, USD (mean)	205	5.4 ± 0.21	487	4.9 ± 0.12	0.03
Less than complete maternal primary school education (%)	52	25.3	138	28.5	0.38
Ethnicity (%)					
Luo	137	67.3	345	71.6	0.52
Luhya	42	21.0	84	17.0	0.52
Kisii	14	6.1	26	4.9	0.52
Other	12	5.6	32	6.5	0.52
Currently breastfeeding (%)	38	19.9	–	–	NC
Dewormed in the past 1 year (%)	122	59.5	268	54.6	0.24
Positive malaria (%)	23	10.8	22	4.6	0.003
STH indicators (exposures)					
Presence of any STH infection (%)	82	40.5	193	40.7	0.97
Ascaris infection	50	24.1	109	22.7	0.70
Trichuris infection	47	24.0	135	28.8	0.21
Hookworm infection	0	0.0	1	0.2	NC
Intensity of *Ascaris* infection (%)					
None	155	75.9	378	77.3	0.09
Light	11	5.4	44	9.2	0.09
Moderate	33	15.7	60	12.4	0.09
High	6	2.9	5	1.1	0.09
Presence of *Ascaris*/*Trichuris* coinfection (%)	15	7.5	51	10.8	0.20
Malnutrition indicators (outcomes)					
Anemia§ (%)	78	38.3	68	14.0	< 0.0001
Anemia (corrected for inflammation; %)¶	72	35.3	50	10.2	< 0.0001
IDA (%)	22	10.7	17	3.5	0.0002
Elevated AGP (> 1 g/L; %)	97	48.3	66	13.6	< 0.0001
Elevated CRP (> 5 mg/L; %)	39	20.6	34	7.2	< 0.0001
Any inflammation (elevated CRP or AGP; %)	98	48.9	76	15.8	< 0.0001
Low ferritin (< 12 or < 15 µg/L; %)	34	16.6	24	4.8	< 0.0001
ID (exclusion method; %)‖	33	20.2	19	4.0	< 0.0001
ID (corrected for inflammation; %)¶	46	23.0	25	5.0	< 0.0001
Low RBP (< 0.7 µg/L; %)	53	26.8	45	9.2	< 0.0001
VAD (exclusion method, %)‖	26	16.8	26	5.6	< 0.0001
VAD (corrected for inflammation; %)¶	34	16.9	23	4.5	< 0.0001
Stunted (height-for-age z-score < −2; %)	59	29.7	82	16.9	0.0002
Underweight (weight-for-age z-score < −2; %)	16	7.8	17	11.4	0.26
Wasted (BMI-for-age z-score < −2; %)	3	1.5	44	9.0	< 0.0001

NC = not calculated.
* Unweighted frequencies.
† Values are percent or mean ± SD weighted for complex survey design.
‡ P values were computed from χ^2 tests for differences in proportions and t tests for differences in means.
§ Corrected for altitude: Hb < 11.6 g/dL for 6–59 months; Hb < 12.1 for 5–11 years; Hb < 12.6 for 12–14 years.
¶ Hb, ferritin, and RBP values were adjusted for the presence of inflammation using correction factors.
‖ Excluding children with inflammation (CRP > 5 mg/L or AGP > 1 g/L).

TABLE 2

Bivariate analysis of four measures of STH infection and several nutritional status outcomes† among PSC in the Kibera slum, Kenya, 2012

STH infection	Anemia* n (%)	ID‡ n (%)	VAD§ n (%)	IDA n (%)	Any inflammation¶ n (%)	Stunting‖ n (%)
Any STH infection						
Yes	31 (37.9)	21 (24.9)	**19 (23.7)**	9 (10.7)	34 (41.5)	27 (32.8)
No	41 (33.6)	25 (21.6)	**15 (12.3)**	13 (10.8)	64 (53.9)	32 (27.5)
P value**	0.53	0.59	**0.04**	0.98	0.09	0.43
Ascaris infection						
Yes	18 (35.6)	**17 (33.5)**	**13 (27.0)**	7 (14.0)	19 (38.0)	18 (35.2)
No	54 (35.2)	**29 (19.6)**	**21 (13.7)**	15 (9.7)	79 (52.4)	41 (27.9)
P value**	0.97	**0.048**	**0.03**	0.40	0.08	0.34
Trichuris infection						
Yes	18 (38.0)	13 (26.1)	11 (23.5)	7 (13.6)	23 (48.7)	13 (28.5)
No	54 (34.5)	33 (22.0)	23 (14.8)	15 (9.8)	75 (49.0)	46 (30.0)
P value**	0.67	0.56	0.17	0.46	0.97	0.85
Ascaris/Trichuris coinfection						
Yes	5 (30.6)	**9 (56.3)**	5 (33.9)	**5 (30.6)**	8 (53.2)	4 (26.8)
No	67 (35.7)	**37 (20.2)**	29 (15.5)	**17 (9.1)**	90 (48.6)	55 (29.9)
P value**	0.69	**0.002**	0.07	**0.01**	0.74	0.80

* Corrected for altitude (Hb < 11.6 g/dL adjusted for inflammation using correction factors).
† Percentages are weighted for complex survey design.
‡ Ferritin < 12 μg/L adjusted for inflammation using correction factors.
§ RBP < 0.7 μg/L adjusted for inflammation using correction factors.
¶ Elevated AGP (> 1 g/L) or CRP (> 5 mg/L).
‖ Height-for-age z-score < −2.
** P value computed from χ^2 test for differences in proportions.

When correcting for the presence of inflammation using the correction factor approach, prevalences of anemia and VAD were lower compared with uncorrected values, and the prevalence of ID was higher compared with uncorrected values.

The bivariate associations between nutrient outcomes and four different measures of STH infection among PSC are shown in Table 2. There were no differences in the prevalence of anemia or IDA between those children with and without any STH infection or species-specific infection with *Ascaris* or *Trichuris*. However, the prevalences of ID and IDA were significantly higher among those children coinfected with both *Ascaris* and *Trichuris* compared with those children not coinfected. Compared with those children not infected, there was a higher prevalence of ID among those children infected with *Ascaris* ($P = 0.048$) and a higher prevalence of VAD among those children infected with *Ascaris* or any STH infection ($P = 0.03$ or $P = 0.04$, respectively). There was a lower prevalence of any inflammation among those children with any STH infection ($P = 0.09$) and those children with *Ascaris* infection ($P = 0.08$), although these findings were not statistically significant in the analysis. Also, when we compared the stages of inflammation, there were no significant associations between STH infection and either elevated CRP or elevated AGP (data not shown). Among SAC, there were no significant bivariate associations between STH infection and any of the measured nutrition outcomes, except a higher prevalence of VAD among those children infected with *Ascaris* (8.0% versus 3.5%, $P = 0.0496$).

Results of multivariate Poisson regression analyses assessing the association of STH infection and ID, VAD, and IDA among PSC are shown in Table 3. Both any STH infection and *Ascaris* infection were significantly associated with VAD (PR = 2.2, 95% confidence interval [95% CI] = 1.1–4.6; PR = 2.2, 95% CI = 1.04–4.6, respectively). This relationship was unchanged in the sensitivity analysis, which corrected for recent vitamin A supplementation. There was some indication of a dose–response relationship, with children with moderate-intensity *Ascaris* infection more likely to have VAD than those children without *Ascaris* infection (PR = 2.4, 95% CI = 1.01–5.6). When we added reported history of vitamin A supplementation to the models, the association between any STH, *Ascaris*, or infection intensity and VAD did not change

TABLE 3

Multivariate analysis assessing the association of STH infection and nutritional outcomes among PSC in the Kibera slum, Kenya, 2012

Primary exposure	Outcomes	Unadjusted prevalence ratio	95% CI	Adjusted prevalence ratio	95% CI
Any STH infection	VAD*	1.92	1.03–3.58	2.23†	1.11–4.58
Ascaris infection	VAD*	1.97	1.06–3.65	2.20‡	1.04–4.57
Intensity of *Ascaris* infection					
None	VAD*	Reference	–	Reference	–
Light	VAD*	1.52	0.53–4.36	2.36§	0.57–7.12
Moderate	VAD*	2.04	1.05–3.94	2.43	1.01–5.58
High	VAD*	1.00	0.16–6.14	1.09	0.07–5.30
Ascaris/Trichuris coinfection	ID¶	2.78	1.61–4.79	3.28‖	1.54–6.60
Ascaris/Trichuris coinfection	IDA	3.36	1.39–8.16	3.10**	1.15–7.65

* RBP < 0.7 μg/L adjusted for the presence of inflammation using correction factors.
† Adjusted for sex, age, socioeconomic status, ID, and stunting.
‡ Adjusted for *Trichuris*, sex, age, socioeconomic status, ID, and stunting.
§ Adjusted for *Trichuris*, sex, age, socioeconomic status, ID, and stunting.
¶ Ferritin < 12 μg/L adjusted for the presence of inflammation using correction factors.
‖ Adjusted for sex, age, socioeconomic status, VAD, stunting, malaria, and wasting.
** Adjusted for sex, age, socioeconomic status, VAD, AGP, stunting, malaria, and wasting.

considerably (data not shown). Coinfection with *Ascaris* and *Trichuris* was significantly associated with both ID and IDA (PR = 3.3, 95% CI = 1.5–6.6; PR = 3.1, 95% CI = 1.2–7.7, respectively). There was no significant association of STH infection in PSC and anemia, stunting, or inflammation.

Among SAC, multivariate regression analysis showed no significant association between any STH and *Ascaris*/*Trichuris* coinfection and any of the nutritional outcomes (data not shown). There was, however, a significant positive association for anemia comparing high-intensity infection with *Ascaris* with no infection (PR = 5.3, 95% CI = 1.2–16.5). The presence of any STH was negatively associated with elevated AGP (PR = 0.55, 95% CI = 0.33–0.89).

DISCUSSION

In this urban slum environment, we report a high prevalence of *Ascaris* and *Trichuris* infections among both PSC and SAC, despite exposure to frequent informal deworming. To our knowledge, this study was one of the first studies to describe the association of STH and malnutrition in both PSC and SAC and account for multiple known risk factors, including inflammation and malaria, as well as severity of STH infection. We found that STH infection in PSC, even at low to moderate intensity, was associated with important micronutrient deficiencies, namely VAD and ID. STH infection in SAC, however, was not associated with measures of poor nutritional status.

The 40.7% prevalence of STH infection in SAC that we reported in Kibera was higher than the 12.9% prevalence reported among primary school children in surrounding Nairobi[20] as well as the 16.2% prevalence reported among children ages 10–18 years living in urban slum settings in western Kenya.[21] These findings suggest the need to consider urban slums as separate ecological zones from their parent cities for planning deworming interventions.

The association that we observed between STH infection and ID and VAD is not surprising, because mechanisms to explain the adverse effects of helminth infection on nutrition have been proposed and include decreased appetite and nutrient intake, impaired absorption, and increased nutrient loss or altered metabolism.[9] In addition, *Ascaris* worms express a range of retinol binding proteins, retinol dehydrogenases, and retinoic acid receptors, which use retinol for growth and development,[22] although a study in Sri Lanka among adolescent girls found that *Trichuris* infection but not *Ascaris* infection was associated with lower serum vitamin A concentration compared with those girls not infected.[23] WHO and United Nations Children's Fund (UNICEF) have recommended that deworming be included in vitamin A supplementation programs,[24] and combined vitamin A supplementation and deworming have been shown to reduce *Ascaris* reinfection rates among PSC in Panama.[25] Although our results support an integrated approach, additional research is needed to elucidate the timing and duration of combined interventions with vitamin A and deworming.

We did not find an association between STH infection and anemia, which may be because of the low prevalence of high-intensity infections and hookworm infection as well as other known risk factors for anemia that were prevalent in this population (e.g., vitamin deficiencies, malaria, inflammation, and inherited blood disorders).[26] Studies in Zanzibar, similarly, found no association between helminth infection early in life and anemia or malnutrition, which is hypothesized to be secondary to increased environmental exploration and thus, increased exposure to STH by better-nourished children.[27] However, in a 12-month deworming trial of children 6–71 months of age, mebendazole improved anemia only in children < 24 months of age, suggesting that young children may still benefit from deworming, because they are at higher risk of anemia, although at lower intensity of helminth infection.[5]

We found no evidence of helminth-induced inflammation and in fact, a negative association between any STH infection and AGP among SAC. Helminth infections stimulate inflammation during the initial acute phase of the primary infection or during reinfection, whereas stable, low-intensity infections may not be associated with elevated levels of acute-phase proteins.[28] Furthermore, helminths induce a Th2 cytokine response, characterized by the production of the cytokines interleukin-4 (IL-4), IL-5, IL-9, and IL-13, the antibodies immunoglobulin G1 (IgG1) and IgE, and eosinophilia, which have an anti-inflammatory effect on the host and may explain the negative association that we observed of STH with chronic inflammation.[29]

Although the WHO currently recommends routine mass drug administration, there is mixed evidence on the benefits of routine deworming. For example, a recent Cochrane review of 41 trials showed that, although treatment of infected children can improve growth, routine deworming alone has no effect on growth, cognitive ability, or school attendance.[30] Furthermore, the recently published de-worming and vitamin A (DEVTA) study, which dewormed 1 million PSC every 6 months in northern India for 5 years, showed no impact on hemoglobin, weight, or mortality.[31] These studies have led some to question the value of deworming interventions[32]; however, these trials did not specifically evaluate the effects of deworming on micronutrient deficiencies, such as iron or vitamin A, which according to our study's findings, may be prevalent even in populations with light to moderate STH infection. Our results from a lower-intensity STH infection setting also challenge the current emphasis on driving down the prevalence of high-intensity STH infection, which is presumed to be the major cause of morbidity.[33]

This study had several limitations. First, the study was conducted in two villages of the Kibera urban slum, and therefore, the findings may not be representative of the rest of Kibera or other urban slums. Second, this study was a cross-sectional study, and therefore, causality cannot be determined. The study was also underpowered to adequately assess the associations of STH infection intensity and nutrition outcomes as well as the effects of hookworm infection, which were uncommon, likely because of low evening temperatures and the urban environment. However, the statistical approach was robust, because multiple covariates were assessed, continuous variables were used to reduce residual confounding, and prevalence ratios were used as a conservative and interpretable measure of association. Finally, the low response rate was not unexpected given the challenge of reaching households in this highly mobile urban slum setting and collecting adequate and timely stools. Although the response rate was low, analyzed and non-analyzed children were similar on assessable demographic characteristics.

In conclusion, PSC are an important target group for deworming interventions, because they have a similar STH

burden to SAC, and even low- to moderate-intensity STH infection in this group is associated with significant micronutrient deficiencies. PSC may be reached through integrated programs, such as vitamin A campaigns, vaccination programs, and Child Health Days.[24] Furthermore, in high population-density urban slums, the feasibility of reaching young children through community-based distribution is likely to be higher than in rural areas. This setting provides an opportunity to evaluate integrated deworming and micronutrient supplementation strategies in young children.

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