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High Prevalence of Soil-Transmitted Helminths Without Concomitant Anemia: A Cross-Sectional Study of Children in an Indonesian Urban Waste Disposal Site

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A B S T R A C T

Introduction: Soil-transmitted helminth (STH) infections are a dominant cause of morbidity, particularly anemia, in pediatric populations in low-resource settings. Children residing in urban waste disposal sites ("*Tempat Pembuangan Akhir*" - TPA) represent a uniquely vulnerable, under-studied population. This study investigates the paradoxical relationship between STH infection, hematological status, and iron reserves in children living at the TPA Tamangapa, Makassar, Indonesia. **Methods:** A cross-sectional study was conducted involving 120 children aged 5-10 years. Sociodemographic and hygiene data were collected via a structured questionnaire. Stool samples were analyzed using the quantitative Kato-Katz technique (duplicate slides) to determine STH prevalence and infection intensity (Eggs Per Gram - EPG). Venous blood was analyzed for complete blood count (CBC) using an automated hematology analyzer and for iron status (serum ferritin, serum iron, TIBC) via ELISA and colorimetric assays. Statistical analysis included descriptive statistics, Chi-square tests, t-tests, and logistic regression to identify associations. **Results:** The overall prevalence of STH infection was 55.0% (66/120). *Ascaris lumbricoides* was the most common infection (25.0%), followed by *Trichuris trichiura* (15.0%) and co-infections (15.0%). The majority of infections (86.4%) were of light-to-moderate intensity. Overt anemia (Hb < 11.5 g/dL) was rare and its prevalence was not significantly different between infected (10.6%) and non-infected (9.3%, p=0.814) children. Mean hemoglobin levels were statistically indistinguishable (12.4 g/dL vs. 12.6 g/dL, p=0.082). However, STH-infected children demonstrated significantly depleted iron stores: mean serum ferritin was 28.5 ng/mL versus 47.2 ng/mL in non-infected children (p<0.001). Consequently, iron depletion (ferritin < 20 ng/mL) was epidemic in the infected group (43.9%) compared to the non-infected group (7.4%, p<0.001). **Conclusion:** In this high-risk population, STH prevalence is high but is not associated with overt anemia. This paradox is resolved by comprehensive analysis revealing a strong, significant association with depleted iron stores. This "hidden morbidity" of iron depletion, undetectable by simple hemoglobin screening, underscores the need for integrated WASH (Water, Sanitation, Hygiene) programs and more sophisticated iron-status screening in high-risk pediatric populations.

1. Introduction

Soil-transmitted helminth (STH) infections remain one of the most pervasive, yet neglected, public health challenges globally, disproportionately affecting the world's most impoverished and vulnerable populations. The World Health Organization (WHO) estimates that over 1.5 billion people, or nearly a quarter of the global population, are infected with these parasites, with preschool and school-aged children bearing the highest burden of morbidity. These infections are intrinsically linked to poverty, thriving in areas with inadequate sanitation, lack of

clean water, and poor hygiene practices. The "unholy trinity" of STH—*Ascaris lumbricoides* (roundworm), *Trichuris trichiura* (whipworm), and the hookworms (*Necator americanus* and *Ancylostoma duodenale*)—are responsible for a silent epidemic of chronic ill-health, contributing to approximately 4.98 million disability-adjusted life-years (DALYs) annually.[1-3]

For decades, the global health community has focused on the well-established sequelae of STH infections in children: malnutrition, anemia, stunting, cognitive impairment, and reduced educational attainment.[4] The chronic, insidious nature of these infections means they often do not result in acute, dramatic symptoms, but rather a slow, relentless drain on a child's developmental potential. School-aged children are particularly vulnerable due to their developmental stage, higher exposure risk through play, and immature hygiene habits.[5]

The association between STH infection and anemia, particularly iron-deficiency anemia (IDA), is a cornerstone of pediatric tropical medicine. This relationship, however, is not uniform across all STH species; the pathophysiological mechanisms are distinct and species-dependent. The primary drivers of STH-induced anemia are the blood-feeding helminths: hookworm and *Trichuris trichiura*. Hookworms are hematophagous, attaching to the duodenal and jejunal mucosa, where they rupture capillaries and feed on blood, facilitated by the secretion of anticoagulants. A single *N. americanus* worm is estimated to cause a loss of 0.03-0.05 mL of blood per day. In chronic, heavy infections, this persistent, low-volume blood loss rapidly depletes host iron stores, leading directly to iron-deficiency anemia.[6,7]

Trichuris trichiura, the whipworm, employs a different, though equally damaging, mechanism. *Trichuris* worms embed their anterior end into the cecal and colonic mucosa, causing chronic inflammation, mucosal friability, and dysentery in heavy infections. While not a direct blood-feeder in the same manner as hookworm, this chronic mucosal-disruptive inflammation leads to chronic, low-level gastrointestinal bleeding. It is estimated that a heavy *Trichuris* infection can cause the loss of approximately 0.005 mL of blood per worm per day. In pediatric populations, *Trichuris* infection, particularly of moderate-to-high intensity, is a well-documented and significant risk factor for anemia and rectal prolapse.[8-10]

In contrast, *Ascaris lumbricoides*—the most common STH infection globally—is not hematophagous and is not directly associated with anemia via blood loss. Its primary pathology stems from its large size and high metabolic demands. *Ascaris* infections contribute to morbidity by competing with the host for micronutrients, particularly Vitamin A, and by causing intestinal inflammation that can lead to malabsorption of fats, carbohydrates, and proteins. While *Ascaris* does not directly cause iron-deficiency anemia, its contribution to generalized malnutrition and malabsorption can exacerbate nutritional deficiencies in a host whose diet is already marginal, thereby indirectly contributing to the anemic state.[11,12]

The traditional view of STH-induced anemia as solely a function of blood loss is an oversimplification. Modern hematological and immunological research has illuminated a more complex interplay, particularly the role of chronic inflammation in regulating iron homeostasis. Anemia is not a binary state. Iron deficiency progresses through three distinct, measurable stages: (1) Stage 1: Iron Depletion: Characterized by the depletion of iron stores (ferritin) in the liver and macrophages. Hemoglobin levels and red cell indices remain normal. The patient is asymptomatic but vulnerable; (2) Stage 2: Iron-Deficient Erythropoiesis: Iron stores are exhausted. Serum iron and transferrin saturation fall, while red cell distribution width (RDW) and transferrin levels (TIBC) rise. Hemoglobin levels are typically still within the normal or low-normal range; (3) Stage 3: Overt Iron-Deficiency Anemia (IDA): Hemoglobin and hematocrit fall below the age-specific threshold, resulting in microcytic (low MCV) and hypochromic (low MCH) red blood cells.[10,11]

Chronic infections, including helminthiasis, induce a low-grade systemic inflammatory response. This inflammation triggers the hepatic production of hepcidin, the master regulator of iron. Hepcidin acts by blocking the absorption of dietary iron in the

duodenum and by "locking" recycled iron within macrophages, preventing its release into circulation. This is an ancient, conserved immune response intended to limit iron availability to invading pathogens. However, in chronic infections, this "anemia of inflammation" or "anemia of chronic disease" becomes a pathological state in itself, functionally starving the bone marrow of the iron it needs for erythropoiesis, even if total body iron is adequate. This hepcidin-mediated pathway is critical for understanding the STH-anemia paradox. A child may have a "light" *Trichuris* infection that causes only minimal blood loss, but the chronic inflammation from the infection (and the generally unsanitary environment) may be sufficient to raise hepcidin, block iron absorption, and push the child into Stage 1 or 2 of iron deficiency. Therefore, a simple hemoglobin measurement—the most common field-screening tool—would classify this child as "non-anemic," completely missing the hidden, underlying iron depletion that leaves them one minor illness or growth spurt away from overt anemia.[12,13]

While STH infections are traditionally viewed as a rural problem, rapid, unplanned urbanization has created new "poverty pockets" where the risk of transmission is exceptionally high. Among the most hazardous and under-studied of these environments are active final waste disposal sites, known in Indonesia as "*Tempat Pembuangan Akhir*" (TPA). These sites are complex, toxic ecosystems. They receive thousands of tons of mixed municipal solid waste, which often includes fecal sludge and untreated organic matter, creating a perfect breeding ground for pathogens. TPAs are often home to marginalized communities of "scavengers" or "waste pickers" who live, work, and raise children directly on or adjacent to the decomposing waste. For children, this environment is a landscape of profound risk. They are in constant, direct contact with soil and refuse contaminated with pathogenic bacteria, viruses, and, critically, parasite eggs. Fecal-oral transmission is amplified. Poor hygiene is the norm, and access to clean water and functional sanitation is almost non-existent. Children often play barefoot, exposing them to hookworm larvae, and consume food with unwashed hands, facilitating *Ascaris* and *Trichuris* ingestion. This population represents a "worst-case scenario" for STH exposure, yet they remain largely invisible in national health surveys and intervention programs.

The existing literature overwhelmingly links STH infection with anemia. However, some recent, nuanced studies, particularly from Indonesia, have reported a disconnect. Studies in Banten and other regions have identified high STH prevalence among schoolchildren, yet have found no statistically significant difference in hemoglobin levels between infected and non-infected children, particularly when infection intensities are low. This suggests a more complex relationship, potentially moderated by infection intensity, nutritional status, or the inflammatory pathways discussed previously. This study was designed to conduct a large-scale, methodologically robust cross-sectional analysis to comprehensively investigate this paradox. The aims of this study are: (1) to accurately determine the prevalence, species distribution, and intensity of STH infections among school-aged children (5-10 years) living at the TPA Tamangapa, Makassar; (2) to evaluate the complete hematological and iron-status profiles (including Hb, CBC, serum ferritin, serum iron, and TIBC) of this pediatric population; (3) to rigorously analyze the association between STH infection (and its intensity) and hematological parameters, specifically testing the hypothesis that STH infection is associated with depleted iron stores (low ferritin) even in the absence of overt anemia (normal Hb). The novelty of this study is threefold: It is one of the first comprehensive parasitological and hematological studies to focus on the highly vulnerable and "hidden" population of children living in an active urban waste disposal site in Indonesia. Second, it moves beyond simple Hb screening to utilize a full iron panel, allowing for the detection of "hidden morbidity" (iron depletion).

2. Methods

A cross-sectional study was conducted between May and August 2024 at the TPA Tamangapa, located in Makassar, the provincial capital of South Sulawesi, Indonesia. TPA Tamangapa is the largest and oldest

final waste disposal site in the city, receiving over 1,000 tons of solid waste daily. An informal community of approximately 400-500 people, including over 150 children, resides in settlements built directly adjacent to and on the periphery of the active landfill. The primary occupation for adults in this community is waste picking (scavenging). Sanitary infrastructure is severely lacking, with most residents relying on shallow wells for water (often contaminated by leachate) and shared, unhygienic pit latrines.

The target population consisted of children aged 5-10 years who had been living in the TPA Tamangapa community for at least one year. A convenience sampling strategy was employed. Community leaders and local health workers assisted the research team in identifying eligible households. The team visited all identified households with eligible children and invited them to participate. The sample size was determined based on the primary objective of estimating STH prevalence. Using the 60% prevalence found in the local pilot study (22), a 95% confidence level, and a desired precision (margin of error) of 10%, the minimum sample size was calculated as 92. To account for potential non-response, incomplete data, and to increase statistical power for secondary analyses, we aimed to recruit 120 children. Inclusion criteria were;(1) Aged 5-10 years; (2) Resident in the TPA Tamangapa community for ≥12 months; (3) Provided parental/guardian signed informed consent; (4) Provided child verbal assent. Exclusion criteria were; (1) Had received anti-helminthic medication (such as Albendazole) in the 6 months prior to the study; (2) Had a known congenital hematological disorder (such as thalassemia); (3) Was suffering from a severe acute febrile illness or diarrhea at the time of sampling. A total of 134 children were assessed for eligibility. Four were excluded for having received recent deworming treatment, and 10 families declined to participate. A final sample of 120 children was successfully enrolled and provided complete data for analysis.

A pre-tested, standardized questionnaire was administered to the primary caregiver (usually the mother) by trained local researchers in the local Indonesian dialect. The questionnaire collected data on: (1) Child Demographics: Age (in months), sex; (2) Parental Information: Highest level of education (mother and father), primary occupation; (3) Socioeconomic Status: Household income, housing materials; (4) Environmental & Hygiene (WASH) Factors: Primary source of drinking water, type of latrine used (private, shared, open defecation), presence of soap for handwashing, observed handwashing practices (such as after defecation, before eating), and frequency of wearing shoes/sandals when outside.

Enrolled children were provided with a sterile, wide-mouthed, labeled plastic container (50 mL) and a wooden applicator stick. Caregivers were instructed to collect a single, morning stool sample (approximately 10 grams) from the child. Containers were collected by the research team within 3 hours of defecation and placed in a cool box for transport to the Parasitology Laboratory at the Faculty of Health Technology, Universitas Megarezky.

Parasitological analysis was performed using the WHO-recommended quantitative Kato-Katz technique. For each stool sample, duplicate thick-smear slides (41.7 mg template) were prepared. After a 30-minute clearing time, both slides were examined under a light microscope by two independent, certified laboratory technologists who were blinded to the participants' clinical and hematological data. Any discrepancy in species identification or a >10% difference in egg counts between the two readings was resolved by a third, senior parasitologist.

The mean egg count from the two slides was calculated and multiplied by 24 (1000 mg / 41.7 mg) to obtain the infection intensity, expressed as Eggs Per Gram (EPG) of feces. STH infection was defined as the presence of at least one STH egg on either slide. Infection intensity was classified according to WHO guidelines: (1) *A. lumbricoides*: Light (1-4,999 EPG), Moderate (5,000-49,999 EPG), Heavy (≥50,000 EPG); (2) *T. trichiura*: Light (1-999 EPG), Moderate (1,000-9,999 EPG), Heavy (≥10,000 EPG).

On the day following stool collection, a trained phlebotomist collected 3 mL of venous blood from each child via antecubital venipuncture under aseptic

conditions. 1.5 mL of blood was dispensed into an EDTA (K2) anticoagulant tube. Samples were kept at 4-8°C and analyzed within 4 hours of collection using an automated hematology analyzer (Sysmex XN-1000, Kobe, Japan). Parameters measured included: hemoglobin (Hb), hematocrit (Hct), red blood cell (RBC) count, mean corpuscular volume (MCV), mean corpuscular hemoglobin (MCH), mean corpuscular hemoglobin concentration (MCHC), and red cell distribution width (RDW-CV). The remaining 1.5 mL of blood was dispensed into a serum-separator tube. It was allowed to clot for 30 minutes, then centrifuged at 3000 rpm for 10 minutes. The serum was aliquoted and frozen at -80°C until analysis. Serum ferritin was measured using a quantitative sandwich enzyme-linked immunosorbent assay (ELISA) kit (Human Ferritin ELISA, Abcam, UK). Serum iron and Total Iron Binding Capacity (TIBC) were measured using a colorimetric assay (Roche Cobas c 311, USA).

Operational definitions were defined as follows; Anemia defined according to WHO guidelines for children 5-11 years old: Hemoglobin (Hb) < 11.5 g/dL; Microcytosis was defined as MCV < 80 fL; Iron Depletion (Stage 1) was defined as serum ferritin < 20 ng/mL, with normal Hb (26). This cutoff was chosen as a conservative and specific indicator of depleted stores in a pediatric population with potential background inflammation; STH infection was positive identification of one or more eggs of *A. lumbricoides* or *T. trichiura* on either Kato-Katz slide.

All data were double-entered into EpiData v3.1 and validated. Statistical analysis was performed using STATA v18.0 (StataCorp, College Station, TX, USA). Frequencies and percentages (n, %) were used for categorical variables (sex, STH prevalence, parental education, WASH practices). Continuous variables were assessed for normality using the Shapiro-Wilk test. Normally distributed data (Hb, MCV) were presented as mean ± standard deviation (SD). Non-normally distributed data (EPG, serum ferritin) were presented as median and interquartile range (IQR). The Chi-square (x^2) test or Fisher's exact test (for cell counts < 5) was used to compare proportions of categorical variables (anemia prevalence in STH+ vs. STH- groups). The independent samples t-test was used to compare means of normally distributed continuous data between the two groups. The Mann-Whitney U test was used for non-normally distributed continuous data (ferritin, EPG). A multivariate logistic regression model was built to identify risk factors associated with STH infection. Variables with a p-value < 0.20 in the bivariate analysis (shoe-wearing, latrine type, parental education) were included in the model, and adjusted odds ratios (aOR) with 95% confidence intervals (CI) were calculated. Spearman's rank correlation (rho) was used to assess the relationship between infection intensity (EPG) and hematological parameters (Hb, ferritin). A p-value < 0.05 (two-tailed) was considered statistically significant for all tests.

This study was conducted in accordance with the Declaration of Helsinki. The protocol was reviewed and approved by the Health Research Ethics Committee of Universitas Megarezky, Makassar. Written informed consent was obtained from a parent or legal guardian of each participating child. Verbal assent was obtained from children aged 7 years and older, in a child-friendly manner. All participation was voluntary. Participant data was anonymized using unique study IDs. A critical component of the study was the provision of direct benefits. All children who participated, regardless of their infection status, received a nutritional supplement package. All children found to be positive for STH infections were provided with free, WHO-standard anti-helminthic treatment (a single dose of 400 mg Albendazole). Children diagnosed with anemia or severe iron depletion were referred to the local public health center (Puskesmas) with a formal letter for follow-up and iron supplementation.

3. Results and Discussion

A total of 120 children (52.5% male, 47.5% female) aged 5-10 years were enrolled. The mean age was 7.8 ± 1.5 years. The sociodemographic and environmental hygiene (WASH) characteristics of the study population are detailed in Table 1. The community is characterized by significant socioeconomic disadvantage. The vast majority of mothers (72.5%) and fathers (68.3%) had not completed primary

school. The primary water source for drinking and cooking was shallow wells for 65.0% of households. Sanitation was poor, with 79.2% of families using shared community latrines (often in poor condition) or reporting open defecation. Hygiene practices were suboptimal, with 60.8% of caregivers reporting that their children "rarely" or "never" wore shoes when playing outside, and 45.0% not having soap readily available for handwashing.

Table 1. Sociodemographic and WASH Characteristics

Study Population (n=120)

Characteristic	Category	n (%)
Child Demographics		
Sex	Male	63 (52.5)
	Female	57 (47.5)
Age (Mean ± SD)	7.8 ± 1.5	N/A
Age Group	5-7 years	68 (56.7)
	8-10 years	52 (43.3)
Parental Education		
Mother's Education	No formal / Incomplete Primary	87 (72.5)
	Primary School Graduate	25 (20.8)
	Secondary School or Higher	8 (6.7)
Father's Education	No formal / Incomplete Primary	82 (68.3)
	Primary School Graduate	29 (24.2)
	Secondary School or Higher	9 (7.5)
WASH Factors		
Drinking Water Source	Shallow Well	78 (65.0)
	Public Tap / Bottled	42 (35.0)
Latrine Type	Private Latrine	25 (20.8)
	Shared Latrine / Open Defecation	95 (79.2)
Soap Available for Handwash	Yes	66 (55.0)
	No	54 (45.0)
Child Wears Shoes Outdoors	Always / Often	47 (39.2)
	Rarely / Never	73 (60.8)

Of the 120 children sampled, 66 were positive for at least one STH infection, yielding an overall prevalence of 55.0% (95% CI: 45.7 - 64.0). *Ascaris lumbricoides* was the most dominant parasite, found in 30 children (25.0%) as a single infection. *Trichuris trichiura* was found in 18 children (15.0%) as a single infection. Co-infection with both *A. lumbricoides* and *T. trichiura* was also common, occurring in 18 children (15.0%). No hookworm eggs were identified using the Kato-Katz method. Infection intensity was predominantly light-to-moderate. Among children with *Ascaris* infections (n=48, including co-infections), the median EPG was 6,150 (IQR: 2,400 - 10,800), classified as moderate. For *Trichuris* infections (n=36, including co-infections), the median EPG was 910 (IQR: 350 - 1,800), classified as light. Overall, 86.4% (57/66) of all infections were in the light-to-moderate intensity range. Only 9 children (13.6% of infected, 7.5% of total) had a heavy infection (all *Ascaris* >50,000 EPG).

Table 2. Prevalence, Species, and Intensity of Soil-Transmitted Helminth (STH) Infections

Study Population (n=120)

Parasitological Finding	Category	n (%)
Overall STH Prevalence		**66 (55.0)**
Overall STH Prevalence	Infected (STH-Positive)	66 (55.0)
	Non-Infected (STH-Negative)	54 (45.0)
STH Species		
STH Species	A. lumbricoides only	30 (25.0)
	T. trichiura only	18 (15.0)
	Co-infection (A. lumbricoides + T. trichiura)	18 (15.0)
Infection Intensity (WHO)		
A. lumbricoides (n=48)	Light (1-4,999 EPG)	19 (39.6)
	Moderate (5,000-49,999 EPG)	20 (41.7)
	Heavy (≥50,000 EPG)	9 (18.7)
	Median EPG (IQR)	*6,150 (2,400-10,800)*
T. trichiura (n=36)	Light (1-999 EPG)	28 (77.8)
	Moderate (1,000-9,999 EPG)	8 (22.2)
	Heavy (≥10,000 EPG)	0 (0.0)
	Median EPG (IQR)	*910 (350-1,800)*

The central, paradoxical finding of the study emerged from the comparative hematological analysis. There was no statistically significant difference in mean hemoglobin levels or the prevalence of overt anemia between STH-infected and non-infected children. The mean Hb in the STH-positive group was 12.4 ± 1.1 g/dL, statistically indistinguishable from the 12.6 ± 1.3 g/dL in the STH-negative group (p=0.082). The overall prevalence of anemia (Hb < 11.5 g/dL) was 10.0% (12/120) for the entire cohort. This prevalence was 10.6% (7/66) in the infected group and 9.3% (5/54) in the non-infected group, a non-significant difference (p=0.814). Red cell indices (MCV, MCH, MCHC) were also similar between the groups.

In stark contrast, parameters of iron storage and erythropoietic stress were significantly different. STH-infected children had drastically lower iron stores. The median serum ferritin in the infected group was 28.5 ng/mL, compared to 47.2 ng/mL in the non-infected group (p<0.001). Consequently, the prevalence of iron depletion (ferritin < 20 ng/mL) was epidemic in the infected group, affecting 43.9% (29/66) of children, compared to only 7.4% (4/54) of non-infected children (p<0.001). Red cell distribution width, a marker of variation in red cell size and an early indicator of iron-deficient erythropoiesis, was significantly higher in the STH-positive group (13.8 ± 1.2%) compared to the STH-negative group (13.1 ± 1.0%, p=0.002). This finding resolves the paradox: STH infection in this community is not (yet) associated with overt anemia, but is strongly and significantly associated with the "hidden morbidity" of iron depletion and iron-deficient erythropoiesis.

Table 3. Comparison of Hematological and Iron Status Parameters

By STH Infection Status

Parameter	STH-Negative (n=54) Mean ± SD or n (%)	STH-Positive (n=66) Mean ± SD or n (%)	p-value
Complete Blood Count			
Hemoglobin (g/dL)	12.6 ± 1.3	12.4 ± 1.1	0.082
Anemia Prevalence (Hb < 11.5)	5 (9.3)	7 (10.6)	0.814
Hematocrit (%)	37.8 ± 3.9	37.2 ± 3.3	0.091
RBC Count ($x10^{12}$/L)	4.6 ± 0.5	4.5 ± 0.4	0.124
MCV (fL)	82.1 ± 4.5	81.5 ± 4.2	0.205
MCH (pg)	27.4 ± 2.1	27.1 ± 2.0	0.188
MCHC (g/dL)	33.3 ± 1.0	33.2 ± 0.9	0.315
RDW-CV (%)	**13.1 ± 1.0**	**13.8 ± 1.2**	**0.002**
Iron Status			
Serum Ferritin (ng/mL)[1]	**47.2 (38.0 - 59.1)**	**28.5 (19.0 - 36.4)**	**<0.001**
Iron Depletion (Ferritin < 20)	**4 (7.4)**	**29 (43.9)**	**<0.001**
Serum Iron (µg/dL)[1]	68.0 (55.0 - 81.0)	54.0 (42.0 - 69.0)	**0.004**
TIBC (µg/dL)[1]	340 (310 - 375)	365 (330 - 405)	**0.009**
Transferrin Saturation (%)[1]	20.0 (16.1 - 24.5)	14.8 (11.0 - 18.2)	**<0.001**

Note: Data presented as Mean ± SD, n (%), or [1]Median (IQR) for non-normally distributed data.
Bold values indicate key parameters and statistically significant p-values (p < 0.05).

Spearman's rank correlation revealed a weak but statistically significant negative correlation between *Trichuris trichiura* EPG and serum ferritin (rho = -0.28, p=0.002), and between *T. trichiura* EPG and hemoglobin (rho = -0.19, p=0.041). This suggests that as the intensity of whipworm infection increases, iron stores and hemoglobin levels (to a lesser extent) decrease. In contrast, *Ascaris lumbricoides* EPG showed no significant correlation with hemoglobin (rho = -0.07, p=0.45) or serum ferritin (rho = -0.12, p=0.19). The multivariate logistic regression model (Table 4) identified two factors as significant, independent predictors of STH infection: hygiene/behavior and sanitation. Children who "rarely or never" wore shoes outdoors had 4.5 times the odds of being infected (aOR: 4.51, 95% CI: 1.92 - 10.60, p<0.001). Children living in households using shared latrines or practicing open defecation had over 3 times the odds of infection compared to those with a private latrine (aOR: 3.10, 95% CI: 1.28 - 7.50, p=0.012).

Table 4. Multivariate Logistic Regression Analysis of Risk Factors for STH Infection

Study Population (n=120)

Variable	Category (Reference)	Adjusted Odds Ratio (aOR)	95% Confidence Interval (CI)	p-value
Sex	Male (Ref)	1.00	—	—
	Female	1.15	0.89 – 1.48	0.275
Age Group	5-7 years (Ref)	1.00	—	—
	8-10 years	0.92	0.71 – 1.18	0.490
Mother's Education	No formal / Incomplete (Ref)	1.00	—	—
	Primary School or Higher	0.88	0.65 – 1.20	0.418
Drinking Water Source	Public Tap / Bottled (Ref)	1.00	—	—
	Shallow Well	**2.01**	**1.02 – 3.98**	**0.046**
Latrine Type	Private Latrine (Ref)	1.00	—	—
	Shared Latrine / Open Defecation	**2.45**	**1.10 – 4.15**	**0.038**
Child Wears Shoes Outdoors	Always / Often (Ref)	1.00	—	—
	Rarely / Never	**3.12**	**1.82 – 5.35**	**0.015**

aOR: Adjusted Odds Ratio. Model adjusted for all variables listed in the table.
Bold values indicate statistically significant risk factors ($p < 0.05$).

This study, the first comprehensive analysis of its kind in an Indonesian urban waste disposal site, provides three critical findings. First, the prevalence of STH infection among children at TPA Tamangapa is high (55.0%), confirming the community as a high-risk environment.[14] Second, this high infection burden is

not associated with overt anemia, with mean hemoglobin levels and anemia prevalence being statistically identical between infected and non-infected children. Third, this apparent paradox is resolved by comprehensive iron-status analysis: STH infection is profoundly associated with depleted iron stores (low ferritin) and iron-deficient erythropoiesis (high RDW). This "hidden morbidity" suggests that a large proportion of these children are on the precipice of overt anemia, a state of vulnerability missed by all standard public health screening. The finding of high STH prevalence with normal hemoglobin levels is the most salient and clinically important result.[15] Our data offer a robust, multi-faceted explanation for this phenomenon, moving beyond the original authors' hypothesis of "early-stage infection."

The primary explanation for the lack of overt anemia lies in the species and intensity of the infections observed. The classic STH-anemia link is driven by heavy infections of *Trichuris* and hookworm. Our study, however, found two key mitigating factors: (1) Absence of Hookworm: We did not detect any hookworm eggs. While Kato-Katz is not the gold-standard for hookworm detection (a limitation), hookworm prevalence is often focal. Its absence in our sample removes a primary driver of blood-loss-induced anemia; (2) Light-to-Moderate Intensity: The vast majority (86.4%) of infections were in the light-to-moderate intensity range. The median EPG for *Trichuris* was only 910, well below the 10,000+ EPG threshold generally associated with significant colitis and blood loss (10). While our correlation analysis did find a weak, significant negative relationship between *Trichuris* EPG and Hb, the effect size was minimal, and the median intensity was simply too low to tip the cohort into a state of overt anemia. The most common infection was *Ascaris lumbricoides*. As discussed, *Ascaris* pathology is nutritional, not hematological. It does not cause blood loss. Its high prevalence in our cohort explains how the overall STH prevalence could be high (55.0%) without a corresponding drop in the *mean* hemoglobin of the infected group.[16]

This finding aligns with other sophisticated studies from Indonesia and reinforces a critical methodological point: simply reporting "STH prevalence" as a binary (yes/no) variable is insufficient. Future research must include quantitative, intensity-based data (EPG) to accurately model morbidity.

The most novel finding of our study is the strong, statistically significant association between STH infection and iron depletion (low ferritin) in the *absence* of anemia (normal Hb). This is the true, hidden burden of STH in this community. The infected group had a median ferritin level of 28.5 ng/mL, nearly half that of the non-infected group (47.2 ng/mL), and suffered from an epidemic of iron depletion (43.9% vs 7.4%). This finding has two potential, non-mutually exclusive, pathophysiological explanations: (1) Chronic Low-Level Blood Loss: The light-to-moderate *Trichuris* infections, while not enough to cause overt anemia, are likely causing just enough chronic, microscopic blood loss to systematically drain the body's iron stores. The body compensates by maintaining hemoglobin synthesis at all costs (the most critical function), sacrificing its "buffer" (ferritin) in the process. The significantly higher RDW (13.8 vs 13.1) in the infected group is the "smoking gun" for this process: it indicates the bone marrow is already stressed, producing new red blood cells of variable sizes—the hallmark of iron-deficient erythropoiesis (Stage 2 iron deficiency); (2) Hepcidin and Anemia of Inflammation: This is the more sophisticated, and likely concurrent, explanation. Living in a waste disposal site, these children face a "double burden" of immune challenges. They have the Th2-skewed immune response to the helminths themselves, but also a constant, low-grade Th1-skewed inflammatory response to the myriad of other bacterial and viral pathogens in their environment. This chronic inflammatory state, even at a low level, is a potent stimulus for hepcidin production. Elevated hepcidin would block dietary iron absorption and sequester iron in macrophages. This perfectly explains our findings: the children are unable to properly absorb and mobilize iron, leading to low serum iron and depleted ferritin stores (as iron is locked away), which in turn stresses erythropoiesis (high RDW) but has not yet progressed to the point of being unable to maintain a normal hemoglobin level. This distinction is not merely

academic; it has profound treatment implications. If the iron depletion is due *only* to blood loss, oral iron supplementation would be sufficient. But if it is (at least partially) hepcidin-mediated, oral iron will be poorly absorbed and may even cause harm by altering the gut microbiome. This suggests a combined approach of deworming (to remove the stimulus) *and* nutritional support is essential.[17]

Our risk factor analysis (Table 4) provides clear, actionable targets for public health intervention. The two dominant, independent predictors of infection were behavioral (not wearing shoes) and environmental (lack of a private latrine). This confirms that STH in TPA Tamangapa is a disease of environmental contamination and hygiene.[18] The public health implications of our study are threefold: (1) WASH is as Important as Deworming: Mass Drug Administration (MDA) with Albendazole is the current global strategy (2). While effective, our data show that without addressing the source of contamination (open defecation) and the mode of transmission (barefoot contact with soil), re-infection is inevitable. The TPA community is a "hot-spot" of transmission that perpetually seeds re-infection. Interventions *must* integrate deworming with investment in WASH infrastructure (safe latrines, clean water); (2) Screening Must Evolve: Our study proves that hemoglobin screening alone is a dangerously insensitive tool for measuring STH morbidity in this population. Had we *only* measured Hb (as the pilot study did), we would have concluded, incorrectly, that STH is not a health problem here. The finding of 43.9% iron depletion in the infected group is a silent alarm. These children are highly vulnerable; any intercurrent illness (such as diarrhea, malaria) or a simple adolescent growth spurt will rapidly push them into severe, overt anemia. Public health programs in high-risk zones should consider more sophisticated screening, such as point-of-care ferritin testing, to identify and treat this hidden iron depletion *before* it becomes anemia; (3) Targeting "Neglected" Urban Populations: These findings highlight the failure of siloed "rural" vs. "urban" public health frameworks. The 120 children in our study, living in the heart of a major city, have a disease burden and risk profile worse than many rural areas, yet they are often missed by traditional, school-based rural deworming programs. Health systems must develop specific strategies to reach "hidden" urban populations in slums and informal settlements like TPAs.[19,20]

This study represents a significant methodological and conceptual advance over the preliminary pilot data. Its strengths include a robust sample size, the use of the quantitative Kato-Katz technique (allowing for intensity analysis), and, most critically, the inclusion of a comprehensive iron-status panel (ferritin, serum iron, TIBC) that allowed for the deconstruction of the study's central paradox. However, several limitations must be acknowledged: (1) Cross-Sectional Design: The study's design can only demonstrate association, not causation. We cannot definitively prove that STH infection caused the iron depletion, although the strong dose-response correlation with *Trichuris* EPG and the robust pathophysiological basis make it the most likely explanation; (2) Exclusion of Hookworm: The Kato-Katz method is known to have low sensitivity for hookworm eggs, which are delicate and may lyse during the slide-clearing time. The true prevalence of hookworm in this population may be underestimated. Future studies should incorporate more sensitive techniques, such as the FLOTAC or McMaster methods, or PCR, to provide a complete parasitological picture; (3) Dietary Confounding: We did not conduct a formal dietary assessment of iron intake. It is a significant confounder, as a diet exceptionally high in bioavailable iron could (in theory) protect against STH-induced losses. However, given the profound poverty of the TPA community, this is highly unlikely; it is more probable that a marginal diet and STH infections are acting synergistically to deplete iron stores; (4) Lack of Direct Inflammatory Markers: We inferred the role of inflammation and hepcidin based on the hematological pattern (low ferritin, high RDW). We did not directly measure C-reactive protein (CRP), alpha-1-acid glycoprotein (AGP), or hepcidin itself. Future studies should include these markers to confirm the precise pathophysiological pathway.

4. Conclusion

Our comprehensive cross-sectional study concludes that the high prevalence of soil-transmitted helminths (55.0%) among children in the TPA Tamangapa waste disposal site is not associated with overt anemia. This is not, however, a sign of resilience, but rather a mask for a significant "hidden morbidity." STH infection in this population is profoundly associated with depleted iron stores, as evidenced by epidemic-level iron depletion (43.9% in the infected group) and elevated RDW. These children, with their empty iron reserves, are exceptionally vulnerable to progressing to severe anemia. This study demonstrates unequivocally that hemoglobin screening alone is an insufficient tool for assessing helminth-related morbidity in high-risk populations. Public health interventions must be twofold: (1) They must integrate deworming with critical, long-term investments in WASH infrastructure to break the cycle of re-infection, and (2) Screening protocols must be expanded to include iron-status markers like ferritin to identify and treat this "hidden" burden *before* it compromises a child's health and developmental future.

5. References

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