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Original Research

Reciprocal increase of malaria parasitaemia, *S.mansoni* egg count and anemia in malaria-Schistosomiasis mansoni co-infected individuals in Fincha Sugar Estate, western Ethiopia

Mebrate Dufera [1]*, Beyene Petros [2], Berhanu Erko [3] Nega Berhe [4]
[1]Department of Biology, Wollega University, Post Box No: 395, Nekemte, Ethiopia.
[2]Department of Microbial, Cellular and Molecular Biology, Addis Ababa University, Post Box No: 1176, Addis Ababa, Ethiopia.
[3]Aklilu Lemma Institute of Pathobiology, Addis Ababa University, Post Box No: 1176, Addis Ababa, Ethiopia.
[4]Aklilu Lemma Institute of Pathobiology, Addis Ababa University, Post Box No: 1176, Addis Ababa, Ethiopia; Centre for Imported and Tropical Diseases, Oslo University Hospital-Ulleval

Abstract

In Ethiopia where malaria parasite and *Schistosoma mansoni* infections are co endemic, the general population is quite vulnerable both to malaria and *Schistosoma mansoni* infections singly and concomitantly. However, data about the prevalence of malaria- *Schistosoma mansoni* co-infection and their reciprocal parasitological and clinical effects are lacking.The aim of this study was to assess the reciprocal effects of malaria- *Schistosoma mansoni* co-infections with emphasis on parasitological and clinical interactions. A community based cross sectional study was conducted in Finchaa Sugar Estate, western Ethiopia. Blood samples were collected by finger pricking and thick and thin smears stained with Giemsa. Fresh stool samples were collected and processed by the Kato-Katz method. Hemoglobin level was determined using a portable spectrophotometer.SPSS statistical software version 20 was used and P-value <0.05 was reported as statistically significant.The overall prevalence of parasite infections were: malaria (28.15%), *Schistosoma mansoni* (27.90%), malaria- *Schistosoma mansoni* co-infections (12.10%) and other intestinal helminths (11.85%).Among the total of 810 study participants, 452 (55.81%) harbored at least one parasitic infection and 358 (44.20%) had none of the investigated parasitic infections. Malaria parasite density increased with increasing infection intensity of *S. mansoni* and also *S. mansoni* parasite densities increased with increasing malaria parasite infection intensities. Increased risk of anemia (mean Hb=11.71) was significantly associated with malaria (P=0.001), *Schistosoma mansoni* (P=0.002) and malaria- *Schistosoma mansoni* co-infection (P=0.000).Malaria-*Schistosoma mansoni* co-infection was a significant factor for decrease in Hemoglobin levels when compared with mono-infections. The findings of this study showed that malaria-schistosomiasis mansoni co-infected individuals had an increased parasite density, infection intensities and anemia, reciprocally.

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***Corresponding Author:**

Mebrate Dufera [1]*,

E-mail:

mebratedufera@gmail.com

INTRODUCTION

Malaria, schistosomiasis and intestinal helminth infections are causes of high morbidity in most tropical parts of the world (Mazigo *et al.*, 2010). Helminths are among the most common chronic infections in the tropics and malaria parasite infections are the most deadly. These two groups of parasites have similar geographical distribution and co-infection is commonplace (Mwangi *et al*., 2006).

The implications of concomitant malaria and helminth infections have been mainly explored in animals under laboratory conditions (Yoshida *et al*., 2000). Studies on animal models have shown that concurrent infections by two or more parasite species could reciprocally affect the pathogenesis of each parasite (Christensen *et al*., 1988). Similar phenomenon in human parasites has also been suggested and in both cases, the possibility of antagonistic or synergistic interactions between parasites appears to be the mechanism in play (Whittle *et al*., 1969; Sokhna *et al*., 2004; Booth *et al*., 2004; Faye *et al*., 2008; Wilson *et al*., 2009).

Malaria-*Schistosoma mansoni* co-infection in Ethiopia

In Ethiopia some studies have been conducted to assess the prevalence and distribution of malaria-schistosomiasis co-infection. The prevalence of *S.mansoni* and *P. falciparum* infection as reported by Degarege *et al*. (2012) was 23.1%; by Mulu *et al*. (2013) the rate of *S. mansoni* co-infection among the malaria patients was 22.6% and recently by Getie *et al*. (2015) the prevalence of *S. mansoni* co-infection among malaria infected patients was 19.5%. According to Getie *et al.* (2015) the age group of 16–20 years old was significantly associated with co-infection. On the other hand, age group of 6–10 years old and moderate-heavy *S. mansoni* co-infection was significantly associated with severe malaria.

Parasite density in malaria, Schistosoma mansoni mono infection and malaria-Schistosoma mansoni co-infection

Studies conducted among humans by Florey *et al.* (2012) revealed the association between heavy malaria parasitemia and heavy intensity of *Schistosoma mansoni* in co-infected individuals. Malaria-schistosomiasis co-infection (without specifying schistosome spp.) also resulted in higher overall prevalence of malaria parasite with greater incidence and densities of gametocytes than *P. falciparum* single infected children (Sangweme *et al*., 2010).

An age effect was also observed, in which children under the age of five had higher levels of parasitaemia than older children (Faye *et al*., 2008). Increased egg load of *S. mansoni* (Sokhna *et al*., 2004) was also associated with co-infection.By comparing malaria prevalence in the presence and absence of *S. mansoni* co-endemicity, studies

In human populations, only few studies have been conducted, with contradictory results. The main finding is that there is a trend toward a worsening effect of hookworm and *S. mansoni* on the pathogenesis and incidence of malaria, respectively (Adegnika & Kremsner, 2012). Malaria and helminth co-infections may have considerable health consequences, leading to more severe clinical symptoms and pathology than for infection with single parasite species. Interactions of malaria and helminth infections increase the severity of anemia and organomegaly (Nacher *et al*., 2001).

conducted by Ndeffo-Mbah *et.al*. (2014) showed that the impact of schistosomiasis co-infection on increasing malaria prevalence was higher in areas of low malaria transmission than areas of high malaria transmission. Recently, studies conducted in Northwest Ethiopia by Getie *et al.* (2015) indicated that co-infected patients with a moderate-heavy egg burden of *S. mansoni* had significantly high mean *P.* parasitemia.

Malaria-*Schistosoma mansoni* co-infection and Hemoglobin

Like malaria, anemia due to schistosomes can additionally arise from destruction of red blood cells and/or dyserythropoiesis. It can be hypothesized that the combined presence of malaria and schistosomes might enhance the risk of anemia. Studies conducted by Koukounari *et al*.(2008) found evidence that malaria parasitaemia, heavy intensity of *S. mansoni* infection and being stunted were significantly associated with lower mean Hb, although only heavy intensity of *S. mansoni* infection was significantly associated with the risk of anemia among school children over 10 years of age.

Materials and methods

A cross-sectional community based study was undertaken in Finchaa Sugar Estate which is located in Finchaa valley, Oromia Regional state, western Ethiopia (Figure 1). It was ideally selected because of the co-endemicity of malaria parasite and *Schistosoma mansoni* infections, in which the general population is quite vulnerable both to malaria and *Schistosoma mansoni* infections singly and concomitantly.The area is about 325 km west of Addis Ababa and is situated between 9^0 30'N to 9^0 60' N latitudes and 37^0 10' to 37^0 30' E longitudes

and at an altitude of about 1,350-1,600 m above sea level with the average annual rainfall of 1,300 mm. The Sugar Estate is cultivating more than 18,000 hectares of irrigated land using sprinkle irrigation system with a production of 10,000 kg of sugar per day. The Sugar Estate has about seven camps in which each camp has one elementary school and one community health agent and also one health center in one of the villages (Agemsa).

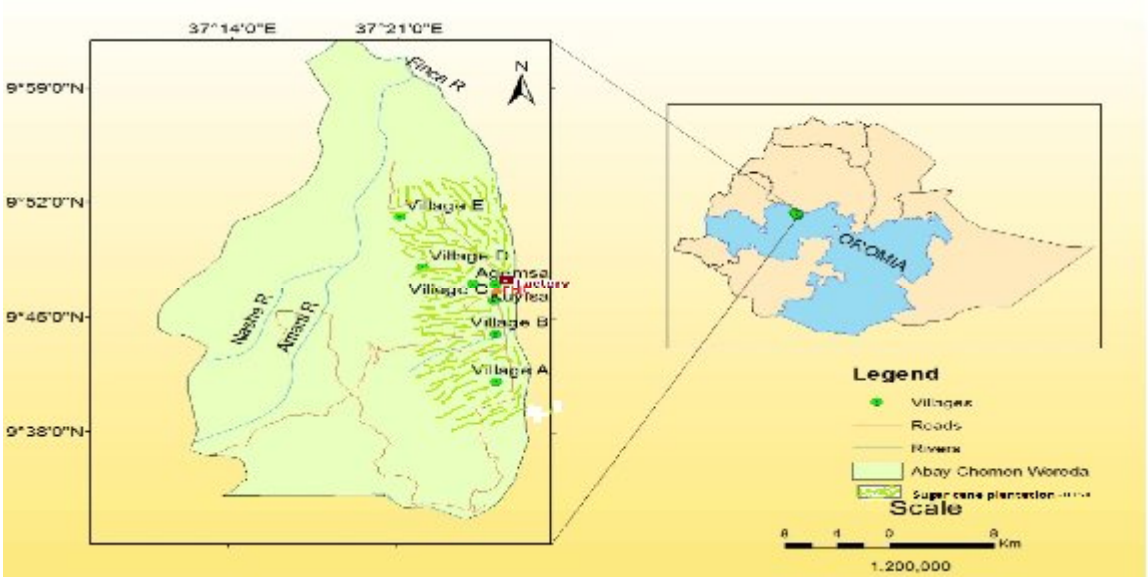


Fig 1: Sketch-map of the study area -Finchaa Sugar Estate, western Ethiopia (Source: Garmin 72 GPS)

The study populations were residents of the three purposively selected villages Camp 7, Kuyissa and Agemsa. Study participants who fulfill the inclusion criteria were selected from the community lists using stratified random sampling methods from the three villages after informed consent/assent.The three villages were selected based on previous study conducted by Dufera et.al. (2014) in which among the 7 camps, village A (camp 7) is the most schistosomiasis affected area (37.5%) followed by Kuyisa (25%) and Agemsa.

Inclusion criteria to participate in the study were (1) >5 years of age of both sexes; (2) Adult populations, parents or guardians gave written informed consent; (3) in addition to the written consent from caretakers, children also were supposed to agree and provide informed assent; (4) Individuals lived in the study area for more than two years were included in the study.While, exclusion criteria were (1) Individuals with history of anti-malarial/anti-helminthic medication use within 2 weeks prior to enrollment and (2) pregnant women and children under 5 years were excluded from the study.

Sample size was estimated for each study villages using Daniel's formula $n=Z^2 P (1-P)/d^2$,Then using proportional allocations 364, 273 and 182 individuals were included in the study from Camp 7,Kuyissa and Agemsa respectively with a sum of 819 study subjects.

Diagnostic techniques

Blood sample collection and parasitological examination for malaria parasites

A finger prick blood sample was collected after cleaning the finger surface using sterile cotton wool. Both thick and thin smears were prepared in a single slide labeled with identification number and stained with 10% Giemsa (Sigma, Aldrich, Nairobi) in phosphate buffer (WHO, 1993). Species specific and parasite densities were estimated under a light microscope at high magnification power by counting the number of parasites per 200 white blood cells (WBC). Parasitaemia was calculated per 200 white blood cells assuming as standard a WBC count of 8 000 WBC/µL of blood (Cheesebrough, 1998).

Determination of Hemoglobin (Hb) Concentration and Anemia

After rubbing the finger tip using sterile cotton wool, finger-prick blood samples were collected and used to fill the microcuvette by touching the cuvette tip on the middle until completely filled with the drop of blood. The loaded microcuvette was then inserted into the holder of a portable, digital counter and/or battery-operated Hemo Cue Hb 201 analyzer

(Hemo Cue AB, Angel Holm, Sweden) Hemoglobin was read from the digital counter in g/dL.

Stool sample collection and parasitological examination for *S. mansoni*

Malaria patients and their counterpart controls were informed to bring sizable stool sample of their own. Those who volunteered to deliver the specimen were given a clean, dry, leak-proof plastic container and clean wooden applicator stick. Stool containers were then collected at the health stations and labeled with identification numbers. These samples were brought to the health center on the same day, and duplicate Kato-Katz cellophane thick smears were prepared from each specimen (WHO, 1993).

Coarse quantification of eggs was obtained by counting the number of eggs on a smear of 41.7mg of stool (Martin & Beaver, 1968). The mean number of eggs from each Kato Katz thick smear was multiplied by a factor of 24 in order to express infection intensities as the number of eggs per gram of faeces (Epg) (WHO, 1993) following the standard procedure used by World Health Organization (WHO, 2002).

Treatment

All participants who were positive for malaria were treated with anti-malarial drugs.Also individuals found positive for *S. mansoni* and STH infections were treated with a single dose of praziquantel 40 mg/kg and mebendazole 500 mg, respectively. All treatments were given free of charge under the supervision of a physicians at the health center according to the national treatment protocols (FMoH, 2004).

Data Analysis

Data were entered into a computer then screened and filtered in Microsoft Excel 2007 spreadsheets, and transferred into SPSS version 20.0 software for statistical analysis. Descriptive statistics was used to provide a clear picture of background variables. The frequency distribution of both dependent and independent variables was determined. Hb levels, intensity of *S. mansoni* infection (Epg), and malaria parasite intensity (parasites/µL) were expressed as means among study participants. All graphs were drawn using MS-Excel and all box-plots were drawn using SPSS version 20.0.P-value <0.05 was reported as statistically significant. To assure the quality of data, 10% each of the total slides were randomly selected for malaria parasite and *S. mansoni* infection and re-examined by an experienced laboratory technologist, who was blinded for the previous results.

Ethical Considerations

The study was approved by the Research Ethics Review Committee of Collage of Natural Sciences, Addis Ababa University and by the National Research Ethics Review Committee. To participate in the research project and written consent/assent was obtained from the study participants.

RESULTS

Socio-demographic characteristics

A total of 810 study participants, male 415 (51.23%) and female 395 (48.77%), were included in the study. Their mean age, Hemoglobin, *S. mansoni* Epg and malaria parasites/µL were 23, 13.84g/dL, 241 and 574, respectively.

Among the total of 810 study participants, 452 (55.81 %) harbored at least one parasitic infection and 358 (44.20%) have none of the investigated parasitic infections. Among mono-infections, the most prevalent parasitic infection was *S. mansoni* 117 (14.44%), followed by malaria 104 (12.84%), malaria-*Schistosomes mansoni* co-infection 98 (12.10%) and other intestinal helminth parasites such as, Hookworm, *T.trichiura*, *A. lumbricoides*, *S. stercoralis* and Taenia spp. 96 (11.85%).Males were more infected (32.72%) than females (23.09%). As age increased infection

prevalence was decreased and individuals within 5-9 and 10-14 age ranges were more affected than other age groups (Table 1).

Table 1: Prevalence of investigated parasitic diseases stratified by sex and age among study participants (n=810) in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

Sex	Jninfected	Parasite							
		Malaria n (%)	mal +Sm n (%)	Mal+OIHP n (%)	Sm n (%)	Sm+ OIHP n(%)	OIHP n(%)	Total Infected n(%)	**Over all total n (%)**
Male	164(20.25)	55 (6.79)	71(8.77)	17(2.10)	73(9.01)	5(0.62)	44(5.43)	265(32.72)	415(51.23)
Female	194(23.95)	49(6.05)	27(3.33)	9(1.11)	44(5.43)	6(0.74)	52(6.42)	187 (23.09)	395(48.77)
Total	358(44.20)	104(12.84)	98(12.10)	26(3.21)	117(14.44)	11(1.36)	96(11.85)	452(55.81)	810(100)
Age(Years)									
5-9	6(0.74)	3(0.37)	14(1.73)	5(0.62)	48(5.93)	8(0.99)	49(6.05)	127(15.68)	133(16.42)
10-14	31(3.83)	7(0.86)	19(2.35)	9(1.11)	35(4.32)	3(0.37)	37(4.57)	110(12.35)	141(17.41)
15-19	22(2.72)	18(2.22)	14(1.73)	8(0.99)	5(0.62)	0(0)	2(0.25)	47(5.80)	69(8.52)
20-24	56(6.91)	25(3.09)	23(2.84)	2(0.25)	12(1.48)	0(0)	4(0.49)	66(8.15)	122(15.06)
25-29	86(10.62)	15(1.85)	10(1.23)	2(0.25)	5(0.62)	0(0)	2(0.25)	34(4.20)	120(14.81)
≥30	157(19.38)	36(4.44)	18(2.22)	0(0)	12(1.48)	0(0)	2(0.25)	68(8.40)	225(27.78)

mal=malaria, Sm=*S. mansoni*, OIHP-Other intestinal helminth parasites (Hookworm, *T.trichiura*, *A.lumbricoides*, *S.stercoralis* andTaenia spp).

Malaria-Schistosoma mansoni co- infection prevalence and parasite density

Mean parasites/μL in malaria, and malaria-*Schistosoma mansoni* co-infected individuals were 574 and 1059, respectively.On the other hand, mean egg count in *Schistosoma mansoni*, and malaria-*Schistosomes mansoni* co-infected individuals were 241 and 389, respectively.Higher means of parasites/μL and Epg were detected in malaria-*Schistosoma mansoni* co-infected individuals than mono infected ones (Table 2).

Table 2: Prevalence of mean parasite density of malaria and schistosomes parasites (n=552), among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

Infection type	Parasite density		
	Minimum	Maximum	Mean±Std.
Malaria (parasites /μL) (n=228)	40	5320	574±1024.21
Schistosoma mansoni (Epg) (n=226)	24	960	241±250.75
Malaria + *Schistosoma mansoni* (n=98):			
Parasites /μL	160	5440	1059±1410.69
Egg count (Epg)	70	1080	389±300.98

Intensity of *S.mansoni* infection (Mean Epg) in malaria-*Schistosoma mansoni* co-infected females was slightly greater than males with mean Hb of 399 and 385 respectively. Regarding age, mean Epg was high among age groups between 5-9 (665) and 10-14 (455) years than other age groups (Table 3).

Table 3: Intensity of *S.mansoni* infection in malaria-Schistosoma mansoni co-infected individuals, stratified by sex and age (n=98) among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

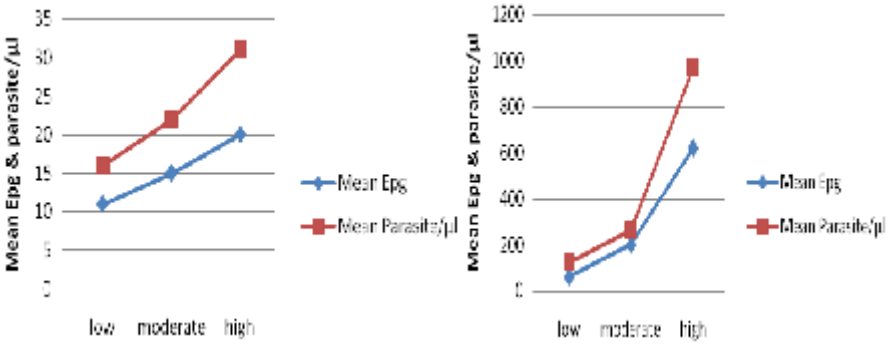
Sex	Intensity of *S.mansoni* infection (Epg) in malaria-*Schistosoma mansoni* co-infected individuals			Total n (%)	Mean Epg
	Light (1-100) n (%)	Moderate (101-400) n (%)	Heavy (>400) n (%)		
Male	18(18.38)	21(21.43)	32(32.65)	71(72.45)	399
Female	4(4.08)	8(8.16)	15(15.31)	27(27.55)	385
Age (year)					
5-9	1(1.02)	3(3.06)	11(11.22)	15(15.31)	665
10-14	3(3.06)	5(5.10)	11(11.22)	19(19.39)	455
15-19	4(4.08)	2(2.04)	7(7.14)	13(12.27)	317
20-24	8(8.16)	10(10.20)	5(5.10)	23(23.47)	300
25-29	3(3.06)	5(5.10)	2(2.04)	10(10.20)	235
≥30	5(5.10)	9(1.11)	4(4.08)	18(18.34)	184

About 29 (29.59 %) of malaria-*Schistosoma mansoni* co-infected individuals were within 501-5000 parasites/μL and with heavy intensity of *S.mansoni* infection (> 400 Epg). Mean (parasites/μL) had been also increased as intensity of *S.mansoni* infection increased (Table 4).

Table 4: Intensity of S.mansoni and malaria parasite infection in malaria-Schistosoma mansoni **co-**infected individuals (n=98) among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

Intensity of malaria parasite infection (parasites/μL)	**Intensity of *S.mansoni* infection (Epg)**			**Total n (%)**	**Mean (parasites/μL)**
	Light (1-100) n (%)	Moderate (101-400)n (%)	Heavy (>400) n %		
1-50	1(1.02)	0(0)	1(1.02)	2(2.04)	80
51-500	21(21.43)	16(16.33)	10(10.20)	47(47.96)	200
501-5000	0(0)	10(10.20)	29(29.59)	39(39.80)	1342
>5000	0(0)	0(0)	10(10.20)	10(10.20)	4180

As infection intensity of malaria parasite increased, there was also an increase in intensity of *S.mansoni* infection.However, infection intensity was higher in *P.falciparum-S.mansoni* co-infected individuals compared to *P.vivax-S.mansoni* co-infected ones *(*Figure 2a, b.)



a) Pv-Sm Infection Intensity ***b)*** Pf-Sm Infection Intensity

Figure 2: Synergistic effects of malaria parasites *and* S.mansoni *infection intensity* among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

In co-infection, more intensity of infection 72 (73.45%) was due to *P.falciparum*. High mean Epg (752) was due to mixed infection followed by *P.falciparum* with a significant difference between malaria parasite species (P=0.000).

Table 5: Intensity of S.mansoni infection in malaria parasite infected individuals (n=98) stratified by malaria parasite species among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

Malaria parasite	Intensity of *S.mansoni* infection (Epg)					
	Light (1-100) n (%)	Moderate (101-400) n (%)	Heavy (>400) n (%)	Total n (%)	Mean Epg	P
P.vivax	11(11.22)	6(6.12)	3(3.06)	20(20.41)	189	
P.falciparum	7(7.14)	24(24.49)	41(41.84)	72(73.47)	414	0.000
Mixed (Pf+Pv)	0(0)	0(0)	6(6.12)	6(6.12)	752	

Hemoglobin levels in malaria-*Schistosoma mansoni* co-infected individuals

The independent sample t-test mean comparision showed that except for malaria positive and *Schistosomiasi mansoni* positive cases (P=0.093),there were significant differences in Hb level between both singly and co-infected and the negative cases (p<0.05) (Table 6).

Table 6: Gender and infection status based comparison of hemoglobin levels among malaria-*Schistosoma mansoni* co-infection study participants (n=810) in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

			t-test for Equality of Means			
				95% Confidence Interval of the Difference		
Independent variables	N	Mean Hb	t	Lower	Upper	P
Sex						
Male	415	14.06	3.189	0.175	0.737	0.001
Female	395	13.61				
Infection status						
Schistosoma mansoni positive	226	12.57	-9.462	-2.126	-1.394	0.000
Schistosoma mansoni negative	584	14.33				
Malaria positive	228	13.00	-5.865	-1.371	-0.683	0.000
Malaria negative	383	14.03				
Malaria positive	228	12.57	1.683	-0.073	0.936	0.093
Schistosoma mansoni positive	226	13.00				
Malaria + *Schistosoma mansoni*	98	11.71	-7.190	-3.100	-1.760	0.000
Uninfected	712	14.14				
Schistosoma mansoni positive	226	12.57	2.309	0.125	1.609	0.022
Malaria + *Schistosoma mansoni*	98	11.71				
Malaria positive	228	13.00	3.608	0.591	2.007	0.000
Malaria + *Schistosoma mansoni*	98	11.71				

Multiple linear regression analysis of hemoglobin level as dependent variable and infections and co-infections as Independent variables showed that malaria-*Schistosoma mansoni* co-infection was a strong significant factor for decrease in Hemoglobin levels when compared to mono-infections. Malaria parasites /µL and S.mansoni Epg were significant negative predictors of hemoglobin levels.

Table 7: Hemoglobin levels in relation to sex, age and infection with malaria, Schistosoma mansoni and malaria-Schistosoma mansoni co-infections, among malaria-Schistosoma mansoni co-infection study participants (n=611) in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

		95.0% Confidence Interval for B		
Independent variable	B*	Lower Bound	Upper Bound	P value
Sex	0.540	0.270	0.809	0.000
Age	0.345	0.273	0.418	0.000
Malaria	0.611	-0.961	-0.260	0.001
Schistosoma mansoni	0.931	-1.513	-0.348	0.002
Malaria + *Schistosoma mansoni*	1.214	-1.648	-0.780	0.000
Malaria parasite /µL	-2.069	-2.393	-1.744	0.000
Schistosoma mansoni EPG	-1.282	-1.575	-0.989	0.000

*Coefficient of regression

Univariate logistic regression analysis of anemia status as dependent variable and infections and co-infections as risk factors showed that *Schistosoma mansoni was a miled (P=0.041) risk factor whereas malaria alone (P=0.016) and* malaria-*Schistosoma mansoni* co-infections were strongly *(P=0.000)* associated with increased risk for anemia.

Table 8: *Malaria*, *Schistosoma mansoni* and *malaria-Schistosoma mansoni* co-infections as risk factors for anemia *among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.*

Risk factors			95% C.I.for EXP(B)		P value
	B*	EXP(B)**	Lower	Upper	
Malaria	0.775	0.461	0.245	0.864	0.016
Schistosoma mansoni	0.523	0.593	0.359	0.979	0.041
Malaria + *Schistosoma mansoni*	2.243	0.106	0.052	0.217	0.000

*Coefficient of regression

**Odds Ratio

Individuals co-infected with *P.falciparum* and *S.mansoni* had lower mean Hb concentration (11.29) compared to mono- infected individuals.

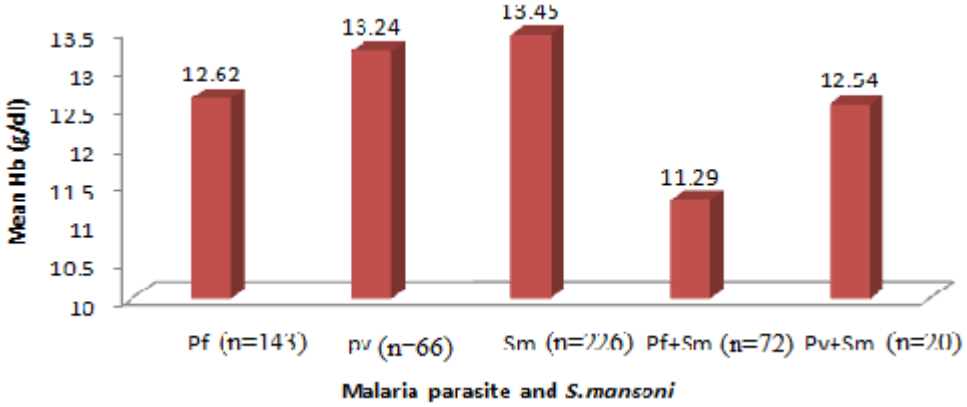
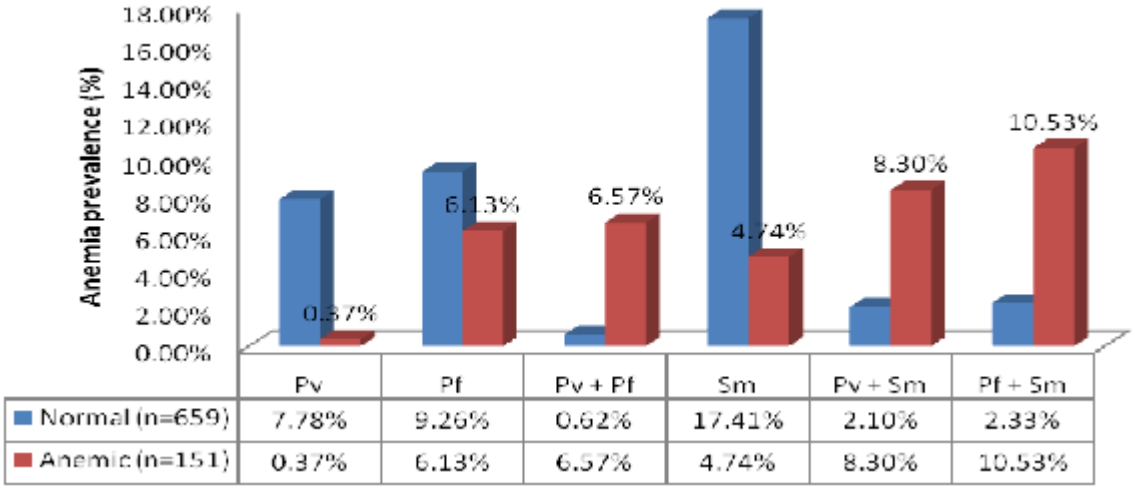


Figure 3: Mean Hb levels in S.mansoni alone, S.mansoni and *P.falciparum / P. vivax* co-infection among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

The highest percentage of anemic cases was due to *P.falciparum- S.mansoni* co-infection (10.53%) followed by *P.vivax-S.mansoni* co-infection (8.30%) compared to malaria or *S.mansoni* mono infections.



	Pv	Pf	Pv + Pf	Sm	Pv + Sm	Pf + Sm
Normal (n=659)	7.78%	9.26%	0.62%	17.41%	2.10%	2.33%
Anemic (n=151)	0.37%	6.13%	6.57%	4.74%	8.30%	10.53%

Figure 4: Anemia prevalence in Pv/Pf/Sm mono and co-infected individuals among malaria-Schistosoma mansoni co-infection study participants in Finchaa Sugar Estate, Western Ethiopia, 2012-2014.

DISCUSSION

In Ethiopia, several studies have been conducted to assess the prevalence and risk factors of malaria and schistosomiasis mansoni in different parts of the country. However, concerning malaria-*Schistosoma mansoni* co-infection, limited studies have been conducted focusing mainly on the study of co-prevalence and co-distribution. In the present study the overall prevalence of malaria co-infection with *Schistosoma mansoni* was 12.10% an addition to very few malaria and *Schistosoma mansoni* co-infection studies in Ethiopia.However, the co-prevalence determined was lower compared to the report from different parts of the Country: southern Ethiopia, 22.6 % (Mulu *et al*., 2013) and 23.1% (Degarege *et al*., 2012); and northwestern Ethiopia, 19.5% (Getie *et al*., 2015). The low co-infection prevalence in the present study could be attributed to chemotherapy and the repeated mass PZQ administrations taken in the community twice a year as it is an Agro-industrial enterprise.

The present study has shown that co-infection due to malaria and *Schistosoma mansoni* infections reciprocally leads to an increase in parasite density. This is similar to the report of Getie *et al*. (2015), a study conducted in northwest Ethiopia that indicated co-infected patients with a moderate to heavy egg burden of *S. mansoni* also had significantly higher mean malaria parasitemia.Also higher parasitaemia and schistosome Epg were detected in malaria-*Schistosoma mansoni* co-infected individuals compared to the mono- infected ones. This is in agreement with reports from Oswald *et al*. (1992) who showed that chronic schistosomiasis may lead to the increase in parasitemia, suggesting during chronic schistosomiasis infection aTh2 cytokine, IL-10, is produced and then suppresses macrophages that kill pRBC, hence indirectly increases malaria parasitaemia.

In the current work an age effect was observed in malaria-*Schistosoma mansoni* co-infection, in which lower age groups had higher levels of mean Epg than older age groups, showing that school-aged children are the most affected group due to high unsafe water-contact behavior such as bathing and

swimming. This is similar to the report of Brooker *et al*. (2006) and Mboera *et al*. (2006). The present study also indicated that in malaria-*Schistosoma mansoni* co-infected study participants malaria parasitaemia and heavy intensity of *S. mansoni* infection were significantly associated with lower mean Hb, which is in line with the findings of Koukounari *et al*. (2008).The observed increase in malaria parasite density in *P.falciparum-S.mansoni* co-infected individuals compared to *P.vivax-S.mansoni* co-infected ones can be explained by the fact that *P.falciparum* infect both young and old RBCs.

Comparisons of the hemoglobin levels among the five categories of infections (*P.falciparum*, *P. vivax, S.mansoni, P.falciparum -S.mansoni* and *P.vivax-S.mansoni*), the mean Hb concentration in *P.falciparum-S.mansoni* co-infection to be greatly lower than the other categories which further provided clear evidence that concurrent *P. falciparum* and *S. mansoni* infection highly enhances the risk of lower Hb levels and anemia. Similar findings have been reported by other studies (Nacher, 2002; Basavaraju & Schwantz, 2006). These findings demonstrate a synergistic interaction of *P. falciparum* and *S. mansoni* infections as the etiology of anemia with increased *P. falciparum* parasite density being responsible for a more severe outcome. Similar to what was reported by Lalloo *et al.* (2007) the present work has presented evidence that, *S. mansoni* infection could aggravate malaria parasitemia in *P.falciparum* co-infection leading to malaria related anemia and likewise *P.falciparum* infection in *Schistosoma mansoni* patients would enhance infection intensity and anemia.

CONCLUSION AND RECOMMENDATIONS

The findings of this study showed that malaria and *S. mansoni* co-infection reciprocally increase parasite densities, infection intensities and anemia.Thus, such understanding of the interactions of malaria and *Schistosoma mansoni* and their effect on parasitaemia, EPG and anemia is a necessary factor in planninig community-based interventions in endemic areas where co-infection with the two parasites is common. Health education, mass deforming programs proper disposal of wastes, avoiding unsafe water-contact behavior and control of snail vectors should be implemented by health agents and local communities.

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