High Prevalence of Gametocyte Carriage among Individuals with Asymptomatic Malaria: Implications for Sustaining Malaria Control and Elimination Efforts in Ethiopia

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ABSTRACT: Asymptomatic malaria often contributes to the reservoir of infection in disease transmission. This study reports the prevalence of gametocyte carriage among asymptomatic individuals and assesses the risk factors associated with asymptomatic malaria in the suburbs of Jimma town, southwestern Ethiopia. A cohort of 582 individuals residing in the suburbs of Jimma town was followed up for three months from September 2013 to November 2013. A total of 1,746 blood films were collected using active case detection. Sociodemographic profile of the study participants was collected using a pretested semistructured questionnaire. The incidence of asymptomatic *Plasmodium falciparum* malaria was 17.18/1,000 person-months at risk, and for *Plasmodium vivax*, it was 17.8/1,000 person-months at risk. The gametocyte carriage rates of *P. falciparum* and *P. vivax* were 66.7% and 12.9% among asymptomatic *P. falciparum*- and *P. vivax*-infected individuals, respectively. Geometric mean gametocyte and asexual parasite density among asymptomatic malaria cases were 412.66 (interquartile range [IQR], 300–660) and 1,866.61 (IQR, 900–6,540) parasites/μL, respectively. Age, place of residence, and distance from vector-breeding site were the main predictors of asymptomatic malaria. The high gametocyte carriage among individuals with asymptomatic malaria in the study area calls for screening and treatment of asymptomatic carriers to interrupt transmission and enhance malaria elimination efforts.

KEYWORDS: gametocyte carriage, asymptomatic malaria, Ethiopia

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Introduction

The successful transmission of malaria parasites from humans to the *Anopheles* mosquitoes depends on the availability of mature infectious gametocytes in human peripheral blood. Therefore, gametocyte carriage can be used as an estimate of transmission potential of malaria parasites from humans to *Anopheles* mosquitoes. Both asymptomatic and symptomatic *Plasmodium*-infected persons tend to harbor gametocytes.[1]

In malaria-endemic countries, a substantial proportion of parasite carriers are asymptomatic,[2] which may challenge malaria control and elimination efforts. Asymptomatic carriers do not usually seek treatment for their infection and therefore constitute a reservoir of parasites available for transmission by the *Anopheles* mosquitoes. It was believed that long-term asymptomatic carriage may represent a form of tolerance to the parasite in adults as well as children, building up their immune response, thereby protecting them from developing either a mild malarial attack or a more severe one by keeping their immunity effective.[3] On the other hand, asymptomatic carriage may represent a mode of entry to symptomatic malaria, as well as transmission, especially in young children.[4,5]

Malaria is a major public health problem in Ethiopia, where ~68% of the population lives in malaria-risk areas.[6] It is endemic in Ethiopia with differing intensity of transmission, with the exception of the central highlands, which are malaria free. The Federal Ministry of Health reported a total of 3,384,589 malaria cases from July 2011 to June 2012, 53.0% of which were laboratory confirmed, with 59.2% *P. falciparum* and 40.8% *P. vivax*.[6] Besides the individual suffering caused by the disease, malaria poses a significant economic burden in Ethiopia.[7]

In the last decade, massive scale-up of control interventions, including the distribution of long-lasting insecticidal nets and indoor residual spraying, together with the introduction of artemisinin-combination therapy, has led to substantial reductions in malaria prevalence and incidence in Ethiopia.[8–10] However, asymptomatic gametocytemia could sustain malaria transmission. Therefore, the aims of this study were to determine *Plasmodium* gametocyte carriage rate and parasitemia among asymptomatic individuals and to assess the risk factors associated with asymptomatic malaria in the suburbs of Jimma town, southwestern Ethiopia.

Methods

Study setting. A community-based cohort study was conducted from September 2013 to November 2013 in four selected villages of Jimma town (Barkume, Kito, Boye, and Cheshire; Fig. 1). Two of the villages (Barkume and Kito) are resettlement villages, while the remaining villages (Boye and Cheshire) are nonresettlement villages. Communities were resettled in the two resettlement villages in 2008. The resettlement was mainly due to displacement as a result of airport expansion, road construction, expansion of schools and health facilities, industrialization, and flooding problems at the center of the town.

Jimma town is located 350 km southwest of Addis Ababa. The geographical coordinates of the town are ~7°41′N latitude and 36°50′E longitude. The town is characterized by warm climate, with mean annual minimum and maximum temperatures of 14°C and 30°C, respectively. The mean annual rainfall ranges from 1,138 mm to 1,690 mm. Maximum precipitation occurs during the three months from June to August, and minimum precipitation occurs in December and January (National Meteorological Agency, unpublished data). Malaria transmission in Jimma is seasonal, which peaks in October and November following the long rainy season (June to September). Minor malaria transmission also occurs from April to May following the short rains from February to March.[11] Almost all malaria cases are due to *P. vivax* and *P. falciparum*, with *Anopheles arabiensis* being the primary malaria vector.[12–15]

Study participants and data collection. A cohort of 604 study participants residing in 202 households was initially recruited for the three-month follow-up study. A total of 22 individuals were lost to follow up, and thus, 582 study participants were considered for this study. The sample size determination and sampling techniques are described elsewhere.[12] Baseline sociodemographic and socioeconomic characteristics of each household were collected using a semistructured questionnaire. A unique identification card was given to each household member at baseline. Average distance from vector-breeding sites was recorded monthly. Artificial vector-breeding sites in both locations include vehicle ruts, ditches, and pits dug for plastering houses and brickmaking, while natural breeding sites in the study areas include intermittent streams, swamps, ponds, stream beds, and marshes. Parasitological data were collected once every month by active case detection. Each study participant was visited monthly. During the monthly house-to-house visit, finger-prick blood samples were collected from each study participant following a standard protocol.[16] Meanwhile, axillary temperature of each of the study participants was recorded during each visit using a digital thermometer to identify asymptomatic individuals

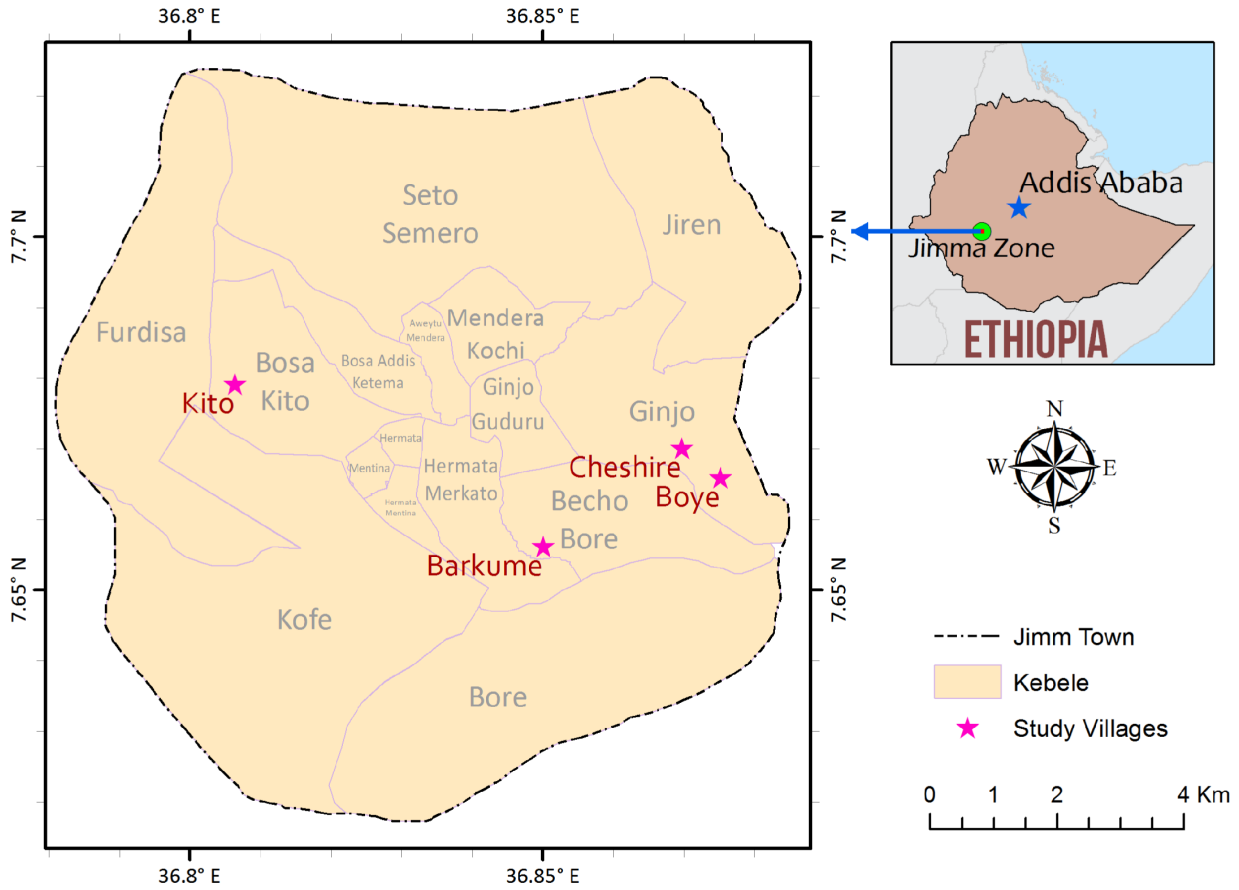

Figure 1. Map of the study sites.

from febrile cases. Asymptomatic malaria was defined as individuals without fever (axillary temperature < 37.5°C) in which sexual and/or asexual stages of *Plasmodium* were detected in the thick blood smear.

Thick and thin blood films were prepared, stained with 10% Giemsa stain, and examined microscopically by an experienced laboratory technologist at Medical Parasitology Laboratory, Jimma University. Blood film slides were considered negative if no parasite was detected after examining 100 high power fields. Gametocyte and asexual malaria parasite densities were quantified against 500 and 200 leukocytes, respectively. This was converted to the number of parasites per microliter of blood, assuming a standard approximation of leukocyte count of 8,000/μL. Quality control slides (all the positive slides and 10% of the negative slides) were reexamined by an independent blinded laboratory technologist. The agreement of the two slide readers was 100% in detection and *Plasmodium* species identification. A third reader was involved when the difference between the two readers exceeded 30% during parasite count, in which case the median result was used. Study participants tested positive for malaria during the study were treated according to the *National Malaria Diagnosis and Treatment Guidelines*: artemether–lumefantrine for uncomplicated falciparum malaria and chloroquine for *P. vivax*-infected individuals.

Data analysis. Data were checked for completeness, coded, and entered into a computer. The association of age and gender with overall *Plasmodium* infection among the study community was tested using Pearson's chi-square test. Asymptomatic malaria incidence was estimated as the total number of malaria-positive cases without fever per 1,000 person-months at risk (calculated by multiplying the total number of new asymptomatic malaria cases by 1,000 person-months at risk divided by the total number of person-months at risk). Similarly, symptomatic malaria incidence was estimated as the total number of malaria-positive cases with fever per 1,000 person-months at risk.

Generalized linear mixed effect model (GLMM) was used to determine the main predictors of asymptomatic and symptomatic *Plasmodium* infections and gametocyte carriage rates among asymptomatic and symptomatic malaria cases. Linear mixed model (LME) was employed to assess the influence of age, gender, and month on log-transformed gametocyte and asexual malaria parasite density. A nested random effect was included in both GLMM and LME models to allow for correlations of observation within individuals and between individuals within the same household. Analysis of variance was used to compare the mean gametocyte and asexual malaria parasite density between asymptomatic and symptomatic cases. Data were analyzed using SPSS version 20.0 (SPSS) and SAS version 9.3 (SAS) statistical software packages. $P < 0.05$ was considered to the statistically significant during the analyses.

Ethical clearance. Ethical clearance was obtained from the Ethical Review Committee of Jimma University. Permission was sought from Jimma Town Health Office. Written informed consent was obtained from the heads of the households prior to data collection. During the parasitological survey, all participants infected with malaria parasites were treated according to the *National Malaria Diagnosis and Treatment Guidelines*. The research was conducted in accordance with the principles of the Declaration of Helsinki.

Results

Sociodemographic characteristics of the study participants. Table 1 shows the sociodemographic characteristics of the study participants. Of the 604 study participants initially enrolled, 582 (96.4%) completed the three-month follow-up, from which a total of 1,746 blood films were collected. Females comprised 57% of the study participants. Age of the study participants ranged from 6 months to 85 years (median age, 19 years). More than half (56.5%) of them were >14 years of age. Of the total blood films collected during the follow-up period, 94% (1,640/1,746) were collected from afebrile individuals (body temperature < 37.5°C). The remaining

Table 1. Sociodemographic characteristics of the study participants.

CHARACTERISTICS	FREQUENCY n (%)	NUMBER OF BF COLLECTED
Gender		
Male	251 (43.13)	753
Female	331 (56.87)	993
Age group in years		
<5	74 (12.71)	222
5–14	179 (30.76)	537
>14	329 (56.53)	987
Place of residence		
Resettlement villages	292 (50.17)	876
Non-resettlement villages	290 (49.83)	870

Notes: Resettlement villages: Barkume and Kito; nonresettlement villages: Boye and Cheshire.
Abbreviation: BF, blood film.

106 (6.0%) blood smears were collected from febrile study participants.

Incidence of asymptomatic and symptomatic malaria. A total of 89 (15.29%) individuals experienced at least one asymptomatic or symptomatic malaria episode during the three-month follow-up. Of the 89 individuals, 69 (11.86%), 17 (2.92%), and 3 (0.52%) individuals had one, two, and three malaria episodes, respectively. Overall, 112 (6.4%) malaria cases were recorded during the follow-up. *P. falciparum*, *P. vivax*, and mixed infections accounted for 58 (51.8%), 52 (46.4%), and 2 (1.8%) of the total cases, respectively. Of the total malaria cases, 54% (60/112) were asymptomatic. Overall, the incidence of *P. falciparum* malaria was 34.4/1,000 person-months at risk, and for *P. vivax*, it was 30.9/1,000 person-months at risk.

Of the 582 study subjects, 50 (8.6%) experienced at least one asymptomatic malaria episode during the follow-up, and 40 (6.9%) and 10 (1.7%) individuals experienced one and two asymptomatic malaria episodes, respectively. No asymptomatic individual with all the three malaria episodes was found during the follow-up. *P. vivax*, *P. falciparum*, and mixed infections accounted for 50.0% (30/60), 48.3% (29/60), and 1.7% (1/60) of the total asymptomatic infections, respectively (Fig. 2). The incidence of asymptomatic *P. falciparum* was 17.18/1,000 person-months at risk. Asymptomatic *P. vivax* malaria incidence was 17.8/1,000 person-months at risk. Of the 52 symptomatic malaria cases recorded, *P. falciparum*, *P. vivax*, and mixed infections accounted for 55.8% (n = 29), 42.3% (n = 22), and 1.9% (n = 1), respectively (Fig. 2). The incidence rates of symptomatic *P. falciparum* and *P. vivax* malaria were 17.18 and 16.6 per 1,000 person-months at risk, respectively.

Risk factors for asymptomatic and symptomatic malaria. Table 2 shows the results of GLMM for predictors of asymptomatic and symptomatic *Plasmodium* infections. Age, place of residence, and distance from vector-breeding site were the main predictors of asymptomatic malaria in the study community. Asymptomatic *Plasmodium* infection was found in 5% (12/222) of children below five years of age. The number of cases with *P. falciparum* and the number of cases with *P. vivax* were equal (six cases each) among the asymptomatic children below five years of age. Children below five years of age were 2.5 times more likely to have asymptomatic *Plasmodium* infection as compared to those >14 years of age (odds ratio [OR] = 2.46, 95% confidence interval [CI]: 1.02–5.96). Individuals who lived in the resettlement villages were three times at higher risk of asymptomatic *Plasmodium* infection as compared to those who lived in the nonresettlement villages (OR = 3.34, 95% CI: 1.51–7.37). The risk of asymptomatic *Plasmodium* infections among those who lived <1 km from vector-breeding site was two times higher as compared to those who lived >1 km away from the vector-breeding site (OR = 2.13, 95% CI: 1.06–4.27). Other covariates assessed did not show a significant effect on asymptomatic *Plasmodium* infection ($P > 0.05$).

Similarly, age, place of residence, and distance from the vector-breeding site were the main predictors of symptomatic malaria (Table 2). Children below five years of age were three times more likely to have symptomatic malaria as compared to those >14 years of age (OR = 3.32, 95% CI: 1.26–8.76). Moreover, children between 5 and 14 years of age were two times more likely to have symptomatic malaria as compared to those >14 years of age (OR = 2.31, 95% CI: 1.07–4.99). Individuals who lived in the resettlement villages were three times at higher risk of symptomatic malaria as compared to those who lived in nonresettlement villages (OR = 3.25, 95% CI: 1.52–6.95). The risk of symptomatic malaria among those who lived <1 km from the vector-breeding site was 8.7 times higher as compared to those who lived >1 km away from the vector-breeding site (OR = 8.74, 95% CI: 3.08–24.8). The incidence of symptomatic malaria was higher during September (OR = 2.81, 95% CI: 2.13–3.72) and October (OR = 2.47, 95% CI: 1.87–3.26) compared to November. Other covariates assessed did not show a significant effect on symptomatic malaria ($P > 0.05$).

Gametocyte carriage rate and parasite density. Of the total microscopically confirmed malaria cases, 40.17% (45/112) had gametocyte stages. *P. falciparum* gametocyte was detected in 60% (n = 36) of the total *P. falciparum*-positive cases. *P. vivax* gametocyte was detected in 17% (n = 9) of the total *P. vivax*-positive cases. Table 3 shows the gametocyte carriage rate among asymptomatic and symptomatic malaria cases. Overall, the gametocyte carriage rate among asymptomatic malaria cases was 40% (24/60). At the species level, *P. falciparum* gametocyte carriage rate among asymptomatic *P. falciparum*-positive cases was 66.7% (20/30), while *P. vivax* gametocyte carriage rate among asymptomatic *P. vivax*-positive cases was 12.9% (4/31). There was no significant difference in the gametocyte carriage rate between sexes and among age groups ($P > 0.05$).

Geometric mean gametocyte densities were 412.66/µL (interquartile range [IQR], 300–660) and 307.66/µL (IQR,

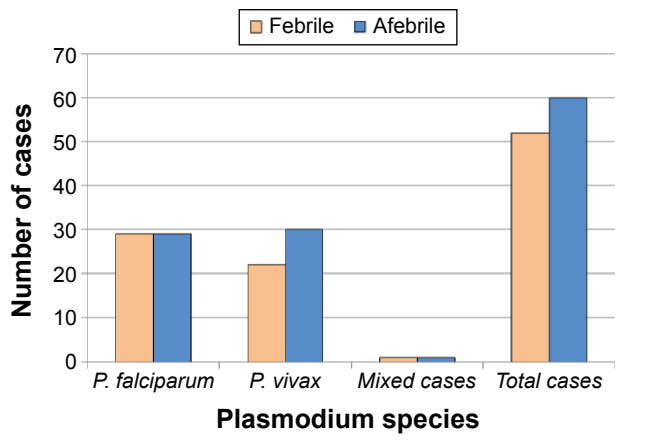

Figure 2. Asymptomatic and symptomatic *Plasmodium* infections among the study communities in the suburbs of Jimma town, southwest Ethiopia.

Table 2. GLMM for the assessment of risk factors of asymptomatic and symptomatic malaria among the study participants in the suburbs of Jimma town, southwest Ethiopia.

PARAMETERS	NUMBER EXAMINED	ASYMPTOMATIC MALARIA				SYMPTOMATIC MALARIA			
		POSITIVE n (%)	OR	95% CI	*P*-VALUE	POSITIVE n (%)	OR	95% CI	*P*-VALUE
Gender									
Male	753	19 (2.52)	0.61	0.33–1.13	0.114	29 (3.85)	0.65	0.01–1.34	0.06
Female	993	41 (4.13)	Ref			23 (2.32)	Ref		
Age group in years									
<5	222	12 (5.41)	2.46	1.02–5.96	0.046*	12 (5.41)	3.32	1.26–8.76	0.02*
5–14	537	20 (3.72)	1.57	0.78–3.16	0.211	21 (3.91)	2.31	1.07–4.99	0.03*
>14	987	28 (2.84)	Ref			19 (1.93)	Ref		
Month									
September	582	20 (3.44)	1.05	0.81–1.35	0.714	21 (3.61)	2.81	2.13–3.72	0.00*
October	582	21 (3.61)	1.17	0.91–1.51	0.209	20 (3.44)	2.47	1.87–3.26	0.00*
November	582	19 (3.26)	Ref			11 (1.89)	Ref		
Estimated monthly income (ETB)									
<500	252	7 (2.78)	0.73	0.20–2.59	0.620	5 (1.98)	0.92	0.22–3.90	0.91
500–1000	1203	41 (3.41)	0.77	0.30–1.96	0.578	40 (3.32)	1.63	0.56–4.72	0.37
>1000	291	12 (4.1)	Ref			7 (2.4)	Ref		
Place of residence									
Resettlement villages	876	44 (5.53)	3.34	1.51–7.37	0.003*	39 (4.45)	3.25	1.52–6.95	0.00*
Non-resettlement villages	870	16 (1.89)	Ref			13 (1.49)	Ref		
Distance from vector-breeding site									
<1 km	1074	47 (4.38)	2.13	1.06–4.27	0.033*	48 (4.47)	8.74	3.08–24.8	0.00*
>1 km	672	13 (1.93)	Ref			4 (0.6)	Ref		
House type									
Holes in the walls	135	5 (3.70)	1.31	0.37–4.69	0.676	6 (4.4)	1.96	0.64–6.06	0.22
No holes in the walls	1611	55 (3.41)	Ref			46 (2.86)	Ref		
Overall	1746	60 (3.66)							

Notes: *Significant at $P < 0.05$. Resettlement villages: Barkume and Kito.
Abbreviations: ETB, Ethiopian Birr; OR, odds ratio; CI, confidence interval; Ref, reference.

Table 3. GLMM for gametocyte carriage among asymptomatic and symptomatic *Plasmodium* infections in the suburbs of Jimma town, southwest Ethiopia.

CHARACTERISTICS	ASYMPTOMATIC CASES					SYMPTOMATIC CASES				
	NUMBER POSITIVE	GAMETOCYTE CARRIAGE n (%)	OR	95% CI	*P*-VALUE	NUMBER POSITIVE	GAMETOCYTE CARRIAGE n (%)	OR	95% CI	*P*-VALUE
Gender										
Male	19	8 (42.11)	1.23	0.37–3.95	0.725	29	11 (37.93)	0.33	0.05–2.21	0.23
Female	41	16 (39.02)	Ref			23	10 (43.48)	Ref		
Age										
<5	12	5 (41.67)	0.743	0.13–4.38	0.726	12	5 (41.67)	0.34	0.02–5.83	0.41
5–14	20	5 (25.0)	0.161	0.01–2.00	0.150	21	10 (47.62)	1.48	0.16–14.0	0.70
>14	38	14 (36.8)	Ref			19	6 (31.58)	Ref		
Month										
September	20	10 (50.0)	3.06	0.78–13.83	0.119	21	9 (42.86)	1.85	0.10–33.1	0.56
October	21	9 (42.9)	1.87	0.46–8.49	0.388	20	10 (50.00)	2.25	0.11–44.9	0.46
November	19	5 (26.3)	Ref			11	2 (18.18)	Ref		
Overall	60	24 (40.0)				52	21 (40.4)			

Notes: Numbers in parenthesis indicate percentage values. Resettlement villages: Barkume and Kito.
Abbreviation: Ref, reference.

160–640) for asymptomatic and symptomatic malaria cases, respectively (Table 4). The difference between asymptomatic and symptomatic malaria cases in terms of mean gametocyte density was not significant ($F = 0.405$, $P = 0.528$). There was no significant difference in the geometric mean gametocyte density between sexes and among age groups in both asymptomatic and symptomatic malaria cases ($P > 0.05$).

Geometric mean asexual malaria parasite densities were 3,468.41/μL (IQR, 1,660–8,200) and 1,866.61/μL (IQR, 900–6,540) in symptomatic and asymptomatic malaria cases, respectively. The asexual parasite density was significantly higher in symptomatic malaria cases compared to asymptomatic malaria cases ($F = 7.55$, $P = 0.007$). There was no significant difference in the geometric mean asexual parasite density between males and females and among age groups ($P > 0.05$). The highest asexual malaria parasite density was recorded during the month of November in both asymptomatic and symptomatic malaria cases, with geometric mean densities of 2,815.76/μL (IQR, 1,040–7,600) and 4,796.36/μL (IQR, 2,080–7,080), respectively. The difference in the geometric mean asexual parasite density between November and September was significant for asymptomatic malaria cases ($P = 0.013$).

Discussion

Data on gametocyte carriage among people with asymptomatic malaria in malaria-endemic settings are essential to estimate the reservoirs of infection. Asymptomatic gametocyte carriers maintain malaria transmission in malaria-endemic areas, regardless of the effective treatment of clinical malaria patients. Thus, the present study aimed at determining the gametocyte carriage rate among asymptomatic *Plasmodium*-infected individuals and assessing the risk factors associated with asymptomatic malaria in the suburbs of Jimma town, southwestern Ethiopia. Accordingly, *P. falciparum* gametocyte carriage rate among the study participants with asymptomatic *Plasmodium* infections in the study area was high (66.7%). This was higher than the *P. falciparum* gametocyte carriage rates documented among asymptomatic *Plasmodium*-infected individuals in Ethiopia,[17] Gambia,[18] Bangladesh,[19] and Tanzania,[20] in which *P. falciparum* gametocyte carriage rates were 41.2% (7/17), 10.5%, 2.5%, and 30.5%, respectively. The high gametocyte prevalence observed among the study participants would suggest that asymptomatic *Plasmodium*-infected individuals could serve as a reservoir of infection in the study setting. This finding should be seen in the light of treatment of the study participants with a positive malaria blood film, which could reduce the possibility of subsequent infections. On the other hand, as the study was carried out during high malaria transmission season, the overall prevalence of gametocyte carriage in the study area could be overestimated.

P. vivax gametocyte carriage rate among asymptomatic *P. vivax*-infected individuals documented in this study was lower (12.9%) compared to *P. falciparum* gametocyte carriage

Table 4. Linear mixed effect model for log-transformed gametocyte and asexual malaria parasite density among asymptomatic and symptomatic *Plasmodium* infections in the suburbs of Jimma town, southwest Ethiopia.

CHARACTERISTICS	ASYMPTOMATIC MALARIA				SYMPTOMATIC MALARIA			
	GAMETOCYTE DENSITY		ASEXUAL PARASITE DENSITY		GAMETOCYTE DENSITY		ASEXUAL PARASITE DENSITY	
	GM (IQR)	*P*-VALUE	GM (IQR)	*P*-VALUE	GM (IQR)	*P*-VALUE	GM (IQR)	*P*-VALUE
Gender								
Female	426.1 (280–1200)	0.37	1969.30 (900–6320)	0.515	328.16 (180–700)	0.77	3584.91 (1920–7400)	0.87
Male	388.89 (240–720)		1662.90 (520–6720)		286.12 (140–640)		3378.71 (1460–8540)	
Age								
<5	473.7 (220–2180)	0.46	1824.38 (1120–6260)	0.847	329.84 (160–680)	0.907	4236.98 (1940–8540)	0.472
5–14	452.54 (320–640)	0.81	2016.32 (780–5960)	0.985	460.26 (240–1360)	0.24	3516.81 (1520–9160)	0.702
>14	385.84 (240–800)		1783.94 (640–6600)		164.28 (120–200)		3010.08 (1080–6760)	
Month								
September	251.96 (120–440)	0.964	981.79 (380–2460)	0.013*	241.36 (60–420)	0.78	2641.55 (1260–8540)	0.21
October	732.6 (400–1260)	0.276	2372.82 (1280–6780)	0.965	435.71 (240–780)	0.75	3862.66 (3430–8480)	0.65
November	458.61 (420–610)		2815.76 (1040–7600)		178.89 (160–200)		4796.36 (2080–7080)	
Overall	412.66 (300–660)		1866.61 (900–6540)		307.66 (160–640)		3468.41 (1660–8200)	

Notes: *Significant at $P < 0.05$. Resettlement villages: Barkume and Kito.
Abbreviations: IQR, interquartile range; GM, geometric.

rate recorded in this study (66.7%). The lower *P. vivax* gametocyte carriage rate among asymptomatic *P. vivax*-infected individuals is in agreement with the study conducted in Ethiopia that showed a lower *P. vivax* gametocyte carriage rate (13.6%) among asymptomatic *P. vivax*-infected individuals as compared to the *P. falciparum* gametocyte carriage rate (41.2%). This might be due to the shorter duration of *P. vivax* gametocyte carriage than that of *P. falciparum*. *P. vivax* gametocytes circulate for a maximum of three days,[1] which might increase the chance of missing the gametocytes during microscopic examination.

The mean gametocyte density among asymptomatic *Plasmodium* infections in this study was higher than that reported by studies conducted in Tanzania,[20] Western Kenya,[21] and Cameroon.[22] The high gametocyte density recorded in this study might be due to the effect of season. The current study was conducted during peak malaria transmission season (from September to November). Increased gametocyte carriage during high malaria transmission season was also documented in some epidemiological studies[23,24] and was hypothesized to be a result of the induction of gametocyte production by an increase in bites from uninfected mosquitoes.[25]

The prevalence and density of *Plasmodium* gametocyte were similar in all the age groups. In contrast, other studies reported significantly higher prevalence of gametocyte in younger children.[20–22] Acquired sexual stage-specific immunity has previously been reported to influence gametocyte prevalence,[26] but the mechanism remains unclear.

Age, place of residence, and distance from the vector-breeding site were the main predictors of asymptomatic and symptomatic *Plasmodium* infections. Children below five years of age were at higher risk of asymptomatic *Plasmodium* infections than other age groups. This finding is in line with studies conducted in Cameroon[22] and Ethiopia.[17] The decrease in asymptomatic malaria incidence with increasing age might be due to increase in acquired immunity with age. Individuals who lived in the resettlement villages in this study were at higher risk of asymptomatic and symptomatic *Plasmodium* infection as compared to those who lived in the nonresettlement villages. This might be due to the higher risk of exposure to infectious mosquito bites in the resettlement villages.[12] The presence of vector-breeding sites such as pits dug for plastering and brickmaking made by new settlers during house construction could be prolific mosquito breeding habitats, which in turn may result in higher mosquito abundance in the resettlement villages.

The limitation of this study is that the prevalence of sexual and asexual parasites was determined solely by microscopic examination of thick and thin blood films. This may underestimate the prevalence of sexual and asexual parasites reported in this study.

In conclusion, *P. falciparum* gametocyte carriage rate was high among people with asymptomatic malaria. Age, place of residence, and distance of the household from the vector-breeding site are the main predictors of incidence of asymptomatic and symptomatic malaria in the study area. Systematic screening and treatment of asymptomatic carriers are needed to reduce the reservoirs of infection and, hence, to reduce malaria transmission in this area. Further study is also needed to determine the factors associated with *Plasmodium* gametocyte carriage among individuals with asymptomatic malaria.

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Author Contributions

Conceived the study: TD and DY. Designed the study protocol: TD, DY, and AZ. Involved in data acquisition and data entry: TD and EZ. Involved in data analysis: TD and DE. Drafted the manuscript: TD and EZ. Critically reviewed the manuscript: DY. All the authors read and approved the final manuscript.

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