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Differential response in lipid levels of type 2 diabetics and non-diabetic controls to *falciparum* malaria

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ABSTRACT

Objective: To investigate the effects of *falciparum* malaria on lipid profile and atherogenic indices of type 2 diabetics and non-diabetic adults in the Central Region of Ghana. **Methods:** Plasma lipid profile comprising total cholesterol (CHOL), high-density lipoprotein cholesterol (HDL), low-density lipoprotein cholesterol (LDL) and triglycerides (TG) were determined in 100 type 2 diabetics and 100 non-diabetic controls before and during *falciparum* malaria using the BT3000 autoanalyzer in a prospective case control study design. Atherogenic indices were computed. **Results:** At baseline, diabetics recorded significantly ($P < 0.05$) lower level of LDL but higher levels of CHOL/LDL and TG/HDL ratios than controls. LDL correlated ($P < 0.05$) positively but negatively with CHOL and HDL respectively in the two study groups. During malaria, diabetics exhibited higher ($P < 0.05$) levels of CHOL and TG but lower level of HDL. Non-diabetic controls had malaria-induced elevated level in TG only. The positive correlation between LDL and CHOL was maintained in the two study groups. The TG levels of diabetics correlated ($P < 0.05$) positively with LDL and HDL during malaria. In the case of controls, a positive ($P < 0.05$) correlation was found between LDL and HDL during *falciparum* malaria. **Conclusion:** *Falciparum* malaria modified the associations among the various components of lipid profile and elevated TG levels of diabetics and non-diabetic controls.

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Key words: *Falciparum* malaria, type 2 diabetes, lipid profile, atherogenic indices

INTRODUCTION

Changes in blood lipid levels have been reported in various disease conditions of infectious and chronic nature.[1,2] Such changes promote build-up of proatherogenic lipids with accelerated degradation of lipids with anti-atherogenic properties.[2] To this end, the levels of such lipids differ between healthy controls and their ill-health counterparts. The pattern of lipid changes may also differ with nature of disease making it necessary for the changes to be investigated under various disease conditions. Type 2 diabetes mellitus (T2DM) and malaria continue to affect millions of people globally. Whereas global reduction in malaria cases has been observed that of T2DM is on the increase with the contribution of the African continent to the two conditions hovering around 80%.[3,4] This suggests a high possibility of the two conditions interacting with each other in the same individual, implying that, resource-constrained African continent may continue to lag behind in economic growth compared to others since it has to overcome the double burden of malaria and T2DM on the health of her populace. Biochemically-relevant blood lipids routinely assayed in health facilities include total cholesterol (CHOL), low-density lipoprotein cholesterol (LDL), high-density lipoprotein cholesterol (HDL) and triglycerides, collectively called lipid profile. The various components of the lipid profile have been independently associated with varied disease conditions.[5] Indeed, markers of lipid metabolism have been associated with pathogenesis of T2DM and atherosclerosis with HDL being a major culprit.[6-8] It is

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therefore not surprising to find report of unfavorable lipid profile in diabetics compared with non-diabetic controls in various populations.[6,9] In parasitic infections, the essence of cholesterol has been acknowledged.[10] With respect to malaria, in general, reduced lipid profile has been found although elevated level is also reported.[11,12] None of these studies included T2DM patients, suggesting that, this group appears to be neglected in respect of malarial studies. Malaria-endemic Sub-Saharan African continent is predicted to experience the highest incidence of T2DM by 2030, probably, due to her already anticipated increased trend of diabesity.[13] To this end, investigations into malaria-associated lipid changes is long overdue and such studies may provide vital scientific information that can contribute to improved management of lipidemia in malaria-endemic regions of the globe. The current study therefore aimed at investigating the role of *falciparum* malarial disease in lipid metabolism of type 2 diabetics and their non-diabetic controls residing in the Central Region of Ghana by comparing their lipid profiles before and during *falciparum* malaria during a two-year follow-up.

MATERIALS AND METHODS

Study site

The study was carried out at the Diabetes Clinic of Cape Coast Teaching Hospital (CCTH) in the Cape Coast Metropolis, the capital of the Central Region of Ghana. CCTH serves as the referral hospital for the other health facilities in the Central Region, which has a population of more than two million. It has a well-structured and well-attended Diabetes Clinic with patients from various parts of the region. The region has mainly tropical climate with an economy largely relying on small-scale farming and fishing in the informal sector, supported by a small proportion of her working population in the formal sector. CCTH was chosen because the characteristics of users of the facility reflect those of the entire region.

Selection of participants and laboratory analyses

The study compared the lipid profiles of 100 randomly selected out-patients type 2 diabetics aged 40-80 years with 100 non-diabetic controls who were followed over a 2-year period for symptomatic *Plasmodium falciparum* infection. The control group, recruited from the general population of the Cape Coast metropolis, was age-matched with the diabetic group. At baseline and during *falciparum* malaria, 10 ml of venous blood sample was obtained from respondents after overnight fast for laboratory analyses. Blood samples were separated into plasma and serum. Serum samples were aliquoted and stored at -80°C for later analyses but plasma lipid determination was conducted same day. Plasma levels of total cholesterol, triglycerides and HDL cholesterol were measured by enzymatic methods using the BT3000 autoanalyzer manufactured by JAS diagnostics (JAS Diagnostics Inc., USA) with reagents for the tests provided by the same manufacturer. LDL cholesterol level was estimated by the Friedewald formula.[14]

Malaria diagnosis was done by the CareStart™ Malaria HRP2Pf rapid diagnostic test (RDT) kit manufactured by Access Bio (Access Bio Inc., USA), an RDT that has been extensively evaluated to be highly sensitive and specific for *P. falciparum* detection with strong correlation to microscopy.[15] The randomly selected diabetics were already diagnosed type 2 diabetic patients who were attending the Diabetes Clinic of CCTH for appropriate treatment.

ETHICAL APPROVAL

The study was approved by the Committee on Human Research, Publications and Ethics of the Kwame Nkrumah University of Science and Technology, Kumasi. All protocols followed were in accordance with the ethical requirements of the CCTH, Ghana Health Service and the World Medical Association declaration of Helsinki. Above all, written informed consent was obtained from each study participant.

Statistical analysis

Data obtained were analyzed by Statistical Package for Social Sciences (SPSS) software version 17. Data are presented as mean ± standard deviation. Independent sample t-test was used to compare the mean levels of lipid indices between study groups and between genders at baseline. Mean levels of lipid parameters before and during malaria were compared across groups by one-way ANOVA with Tukey's post hoc HSD test. Pearson correlation, simple and stepwise linear regression analyses were performed. A p-value < 0.05 was considered statistically significant.

RESULTS

Baseline lipid profile and atherogenic indices of respondents in the two study groups is depicted in Table 1. With the exception of mean LDL cholesterol level that was significantly ($P = 0.029$) lower in diabetics, the mean levels of the remaining components of the lipid profile did not differ ($P > 0.05$) between the defined study groups. With respect to atherogenic indices, diabetics had significantly ($P < 0.05$) higher mean levels of CHOL/LDL and TG/HDL than their control counterpart. The mean levels of the remaining indices were comparable ($P > 0.05$; Table 1) between study groups.

Table 1: Baseline lipid profile and atherogenic indices of respondents by study group

Lipid index	Diabetic (N=100)	Non-diabetic (N=100)	P-value
CHOL	5.30±1.30	5.21±1.20	0.595
LDL	3.06±1.01	3.36±1.03	0.029*
HDL	1.26±0.11	1.26±0.13	0.822
TG	1.12±0.23	1.02±0.20	0.141
CHOL/LDL	1.78±0.60	1.57±0.30	0.002*
CHOL/HDL	4.07±1.10	4.22±1.01	0.33
TG/HDL	2.14±1.20	2.00±0.90	0.39
TG/LDL	0.99±0.60	0.81±0.50	0.033*
LDL/HDL	2.65±0.90	2.83±0.90	0.20
	Male respondents		
	N=32	**N=26**	
CHOL	5.24±1.30	4.93±1.20	0.254
LDL	3.19±1.40	2.96±0.13	0.345
HDL	1.23±0.10	1.27±0.10	0.228
TG	1.14±0.20	1.18±0.20	0.759
CHOL/LDL	1.68±0.30	1.66±0.20	0.782
CHOL/HDL	3.83±0.80	3.7±0.60	0.335
TG/HDL	2.34±1.30	1.93±1.00	0.197
TG/LDL	1.09±0.8	0.95±0.70	0.491
LDL/HDL	2.37±0.73	2.26±0.60	0.559
	Female respondents		
	N=68	**N=74**	
CHOL	5.32±1.30	5.32±1.20	0.979
LDL	3.00±1.30	3.50±0.13	0.002*
HDL	1.28±0.11	1.26±0.10	0.522
TG	1.12±0.20	0.97±0.20	0.056
CHOL/LDL	1.83±0.70	1.54±0.30	0.001*
CHOL/HDL	4.24±1.20	4.43±1.01	0.348
TG/HDL	2.00±1.20	2.03±0.8	0.89
TG/LDL	0.94±0.6	0.76±0.40	0.034*
LDL/HDL	2.84±1.00	3.04±1.01	0.285

Figures represent mean±standard deviation in mmol/L except the ratios; LDL=low-density lipoprotein cholesterol; HDL=high-density lipoprotein cholesterol; TG=triglycerides; CHOL=total cholesterol; * = significant P value; N=number of respondents; mean values were compared by independent t-test

Stratifying data by gender in each study group revealed that apart from the mean LDL/HDL ratio that was significantly ($P = 0.026$) higher in diabetic males than their female counterpart, all the remaining lipid indices were similar ($P > 0.05$) between the genders. In the non-diabetic control group, mean LDL cholesterol and CHO/HDL levels were significantly ($P < 0.05$) higher in females than males. However, mean levels of triglyceride and CHOL/LDL were elevated ($P < 0.05$) in the males.

Comparison of baseline mean lipid indices of male diabetics with their non-diabetic counterpart did not show any significant ($P > 0.05$) difference between them (Table 1).

Similar comparison between diabetic and non-diabetic females revealed that the diabetics had significantly ($P < 0.05$) lower mean level of LDL, but higher levels of CHOL/LDL and TG/LDL than the control group (Table 1).

In the presence of *falciparum* malaria, mean levels of CHOL/LDL and CHOL/HDL were significantly ($P < 0.05$) elevated in diabetics compared with their non-diabetic group. With the exception of mean level of LDL/HDL that did not change ($P > 0.05$) due to *falciparum* malaria, the levels of all the other lipid indices differed significantly ($P < 0.05$; Table 2) from their mean levels before malaria.

A Tukey's posthoc HSD test showed significant ($P < 0.05$) elevation of mean levels of CHOL, TG, CHOL/HDL and TG/HDL but reduced level of HDL in diabetics with malaria compared to their mean baseline levels (Table 2).

In the non-diabetic control group, malaria-related significant ($P = 0.041$) elevation was observed for TG only, with the malaria-induced mean levels of all the other lipid indices remaining virtually ($P > 0.05$) unchanged, compared to baseline values (Table 2).

In spite of the seeming variation between diabetics and non-diabetic controls with respect to changes in levels of specific lipid indices before and during *falciparum* malaria, the two groups of respondents exhibited similar pattern in proportion of respondents who had various forms of dyslipidemia with or without malaria (Figures 1 and 2). In both study groups, higher proportion of respondents had hypertriglyceridemia and hypercholesterolemia in malaria.

At baseline, CHOL level of non-diabetics correlated positively with LDL ($R = 0.89$; $P < 0.001$) and TG ($R = 0.202$; $P = 0.037$) but negatively with TG/LDL ($R = 0.206$; $P = 0.033$). In addition, HDL correlated negatively with LDL ($R = -0.246$; $P = 0.01$) but positively with TG/LDL ($R = 0.212$; $P = 0.028$), with CHOL/HDL correlating negatively with CHOL/LDL ($R = -0.545$; $P < 0.001$). Above all, TG/LDL associated positively with TG/HDL ($R = 0.648$; $P < 0.001$) but negatively with LDL/HDL ($R = -0.533$; $P < 0.001$).

In the case of diabetics, baseline CHOL correlated positively with LDL ($R = 0.494$; $P < 0.001$) and CHOL/HDL ($R = 0.205$; $P = 0.032$), with LDL associating negatively with HDL ($R = -0.261$; $P = 0.006$). The other indices of atherogenesis exhibited similar associations as seen in the control group.

In the presence of *falciparum* malaria, non-diabetic LDL exhibited positive associations with CHOL ($R = 0.605$; $P < 0.001$) and HDL ($R = 0.481$; $P = 0.006$), with their HDL correlating negatively with CHOL/HDL ($R = -0.636$; $P < 0.001$). In addition, TG/HDL and TG/LDL correlated positively ($R = 0.685$; $P > 0.001$) in this study group.

Table 2: ANOVA with Tukey's posthoc hsd comparison of lipid profile and atherogenic indices of respondents before and during malaria

ANOVA Lipid index	Diabetic		Non-diabetic		F-value	P-value
	DM (N=70)	BM (N=100)	DM (N=30)	BM (N=100)		
CHOL	5.77±0.30	5.30±1.30	5.36±0.10	5.21±1.20	5.21	0.002*
LDL	3.05±0.31	3.06±1.01	3.26±1.05	3.36±1.03	3.66	0.013*
HDL	1.21±0.30	1.26±0.11	1.24±0.31	1.26±0.13	3.21	0.023*
TG	1.45±0.11	1.12±0.23	1.22±0.10	1.02±0.20	9.40	<0.001*
CHOL/LDL	1.96±0.70	1.78±0.61	1.70±0.70	1.57±0.30	6.00	0.001*
CHOL/HDL	4.9±1.50	4.07±1.10	4.36±1.40	4.22±1.01	260	<0.001*
TG/HDL	2.86±1.30	2.14±1.20	2.51±0.16	2.00±0.90	9.57	<0.001*
TG/LDL	1.15±0.60	0.99±0.60	1.02±0.70	0.81±0.50	5.42	0.001*
LDL/HDL	2.65±1.01	2.65±0.90	2.69±1.10	2.83±0.90	1.92	0.127
TUKEY'S HSD Lipid index	**DM**	**BM**	**P-value**	**DM**	**BM**	**P-value**
CHOL	5.77±0.30	5.30±1.30	0.004*	5.36±0.10	5.21±1.20	0.78
LDL	3.05±0.31	3.06±1.01	0.952	3.26±1.05	3.36±1.03	0.452
HDL	1.21±0.30	1.26±0.11	0.029*	1.24±0.31	1.26±0.13	0.483
TG	1.45±0.11	1.12±0.23	0.005*	1.22±0.10	1.02±0.20	0.041*
CHOL/LDL	1.96±0.70	1.78±0.61	0.094	1.7±0.70	1.57±0.30	0.19
CHOL/HDL	4.9±1.50	4.07±1.10	0.002*	4.36±1.40	4.22±1.01	0.621
TG/HDL	2.86±1.30	2.14±1.20	<0.001*	2.51±0.16	2.00±0.90	0.136
TG/LDL	1.15±0.60	0.99±0.60	0.525	1.02±0.70	0.81±0.50	0.34
LDL/HDL	2.65±1.01	2.65±0.90	0.514	2.69±1.10	2.83±0.90	0.85

Figures represent mean±standard deviation in mmol/L except the ratios; LDL=low-density lipoprotein cholesterol; HDL=high-density lipoprotein cholesterol; TG=triglycerides; CHOL=total cholesterol; *=significant P value; DM=during malaria; BM=before malaria; N=number of respondents

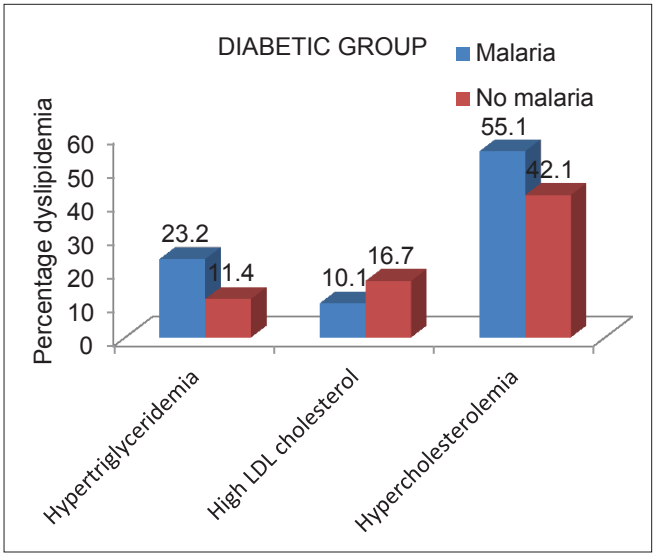

Figure 1: Percentage dyslipidemia in diabetics with and without malaria

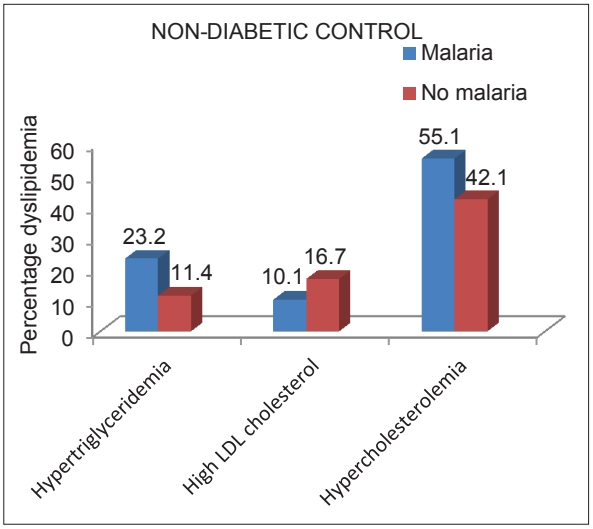

Figure 2: Percentage dyslipidaemia in non-diabetics with and without malaria

With respect to diabetics with malaria, LDL associated positively with CHOL (R = 0.357; P = 0.003) and TG (R = 0.247; P = 0.04), with TG associating positively with HDL (R = 0.264; P = 0.028). Also, CHOL/LDL correlated positively with CHOL/HDL (R = 0.775; P < 0.001). Similarly, positive associations were observed between TG/HDL and TG/LDL (R = 0.339; P = 0.005) and between TG/HDL and LDL/HDL (R = 0.318; P = 0.008).

At baseline, a stepwise linear regression model using CHOL as dependent variable revealed that LDL, TG and HDL were significant (R^2 = 0.847; adjusted R^2 = 0.843 P < 0.001) independent predictors of the observed CHOL level in the non-diabetic group. The model accounted for more than 84% of the observed variation in baseline CHOL level.

Similar analyses of baseline diabetic lipid indices showed that their CHOL level could be predicted independently (R^2 = 0.228; adjusted R^2 = 0.22; P < 0.001) by LDL only, with the model explaining just 22% of the observed variation.

In the presence of malaria, stepwise linear regression model revealed LDL as the only independent predictor (R^2 = 0.366; adjusted R^2 = 0.344; P < 0.001) of malaria-associated CHOL level, explaining about 34% of the observed variation in the control group. With respect to diabetics who had malaria, the stepwise regression model showed that LDL and TG/HDL could independently (R^2 = 0.245; adjusted R^2 = 0.221; P < 0.001) predict malaria-induced CHOL level except that the model explained just about 22% of the observed variation.

DISCUSSION

Cross-sectional design has mostly been used to investigate effect of *falciparum* malaria on lipid profiles.[11,12] The current report applied a prospective case control study design to assess how malaria influenced lipid and lipoprotein levels in type 2 diabetics compared with non-diabetic adults residing in the Central region of Ghana. The relatively higher mean serum LDL cholesterol level found in non-diabetics without malaria compared to their diabetic counterpart disagrees with an earlier study that reported higher LDL levels for diabetics than controls.[9] The difference between the current observation and the previous report could be ascribed to differences in characteristics of respondents, diabetic duration and treatment effect.[9] In fact, meformin, a widely used antidiabetic drug in Ghanaian health facilities, is known to improve lipid profile.[16] Therefore, the comparable and normal baseline levels of the other components of lipid profile and their ratios between diabetics and controls found in the present study could be due to the acknowledged lipid-lowering effect of metformin in diabetics since majority of them were on that medication.[16] On the other hand, the comparable lipid profiles between diabetics and non-diabetic controls in the present study could reflect elevation of lipid levels in sera of non-diabetic respondents due probably, to increased sedentary lifestyle.

Gender-specific variation in lipid profile has been acknowledged with males found to have lower HDL but higher TG than females.[17] In the current study, comparable baseline lipid profile between genders was observed in the diabetic group in support of an earlier finding in diabetics but not the non-diabetic study group.[18] Whereas the gender-related pattern of baseline TG level observed for controls in the present study corroborates earlier report in Europe, the observed mean baseline HDL, LDL, CHOL and their corresponding ratios in the present report is at variance with the Euoropean study but seems to support other findings in Africa.[17,19] The seeming continental difference in lipid profile of non-diabetic respondents could be ascribed to variations in sample size, race and other lifestyle characteristics of study participants. Overall, the mean baseline levels of the various components of lipid profile and their ratios in the current study were lower than those of previous studies, signifying a relatively reduced baseline lipid-profile-dependent CVD risk of respondents in the present study.[18,19]

In the presence of *falciprum* malaria, the suppression effect of metformin on diabetic LDL level was maintained with the two study groups exhibiting similar malaria-induced mean LDL levels. In addition, mean baseline LDL levels did not differ from malaria-induced LDL levels in both diabetics and controls. This finding is at variance with a study in Korea that reported significantly decreased and another in India that observed increased mean LDL level in malaria patients compared with uninfected controls.[20,21] Compared to the present study that includes diabetics and non-diabetics infected with *falciprum* malaria, previous studies either excluded diabetics or included malaria caused by other species of *Plasmodium*.[20,21] Apart from the specific species of the infecting parasite, variations in characteristics of respondents with respect to lifestyle changes and degree of infection could partly explain the varied observations.

Triglyceride level has long been reported to be linked to risk of cardiovascular event, myocardial infarction, coronary heart disease and death in adult irrespective of the other components of lipid profile.[22,23] Triglyceride level increased appreciably in *P. falciparum* infection compared with baseline level in both study groups but malaria-induced increased CHOL and decreased HDL levels were confined to only the diabetic group. The increased triglyceride due to malaria in the current study is in support of a number of earlier reports and has been postulated as a means adopted by the *plasmodium* parasite to induce its infectivity.[24,25] However, the pattern of changes observed in the other components of lipid profile differed between diabetics and non-diabetic controls, suggesting that malaria employs different mechanisms in inducing its cardiovascular disease risk in the two study groups. This calls for different approaches to manage the malaria-induced lipid abnormalities in the two study groups.

Of the various lipid ratios, CHOL/HDL ratio which has recently been observed to better predict cardiovascular event increased only in diabetics due to malaria, pointing to a heightened risk in this study group compared to controls.[20]

CONCLUSION

Falciparum malaria elevated TG levels in diabetic and non-diabetic controls but exhibited varied effects on other

components of lipid profile in the two study groups. This calls for different approaches to management of malaria-associated dyslipidemia in the two groups of respondents.

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Authors Contribution:
SA – Contributed to the design, conduct, data collection, analysis, interpretation, writing of manuscript and critical review of manuscript, **SB –** Was involved in data collection, analysis, interpretation and writing of manuscript, **BAE and JNB –** Contributed to the design, analysis, interpretation, critical review and writing of manuscript.

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