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Implication of Lipid and Fasting Blood Glucose in Hypertensive Pregnant Women in Nigeria

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Authors' contributions

This work was carried out in collaboration among all authors. All authors read and approved the final manuscript.

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ABSTRACT

Background: Dyslipidemia in the early stages of pregnancy raises the risk of preeclampsia. When compared to women who had a normal pregnancy, those who have a history of preeclampsia have significantly different lipid profiles and are more vulnerable to lipoprotein oxidation. According to reports, a key contributing factor to preeclampsia-related hypertension is disorders in lipoprotein metabolism.
Aim: Therefore, the purpose of this study was to determine how fat and glucose may contribute to hypertensive disorders of pregnancy (HDP) in Nigerian women.
Methodology: The study's methodology used a prospective cohort design. Pregnant women receiving antenatal treatment at four different tertiary health centers in Nigeria comprised the participants. The study included 521 patients in all, of whom 34 acquired various forms of HDP. After an overnight fast, participants without HDP at baseline, in the second, third, or at the moment of developing hypertension had around 12 milliliters of venous blood sample taken aseptically from the antecubital vein. The lipid profile and fasting blood sugar levels were measured.
Results: Fasting plasma glucose and lipid profile mean values rose in the second and third trimesters, respectively, in hypertensive women. While fasting plasma glucose decreased significantly from the first to the third trimester in normotensive women, the mean values of triglycerides gradually increased. The median levels of total cholesterol, HDL-C, and LDL-C

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gradually increased beginning in the first trimester, peaked in the second, and then decreased in the third.

Conclusion: The findings of this study demonstrated that changes in lipid profiles and fasting blood sugar levels were related to hypertensive problems during pregnancy in Nigerian women. In women with systolic blood pressure 130 mmHg and diastolic blood pressure 80 mmHg at the first antenatal appointment, early estimate of fasting plasma glucose and lipid profile may be helpful in predicting the development of hypertensive problems in the future.

Keywords: Dyslipidemia; fasting blood glucose; hypertensive disorders in pregnancy; lipoprotein.

1. INTRODUCTION

Normal pregnancy is associated with predicted alterations in lipid metabolism and increases in lipid levels as the gestational period lengthens [1,2]. Maternal adipocytes display considerably increased deposition and hypertrophy during the first trimester to make sure there is enough glucose to meet the increasing fetus' metabolic needs [3]. Increased levels of maternal insulin and progesterone lead to increased lipogenesis, decreased lipolysis, and increased production of lipids. Following their transport across the placenta, these lipids are then digested, illuminating the critical function of lipids in normal embryonic development [2].

One of the reasons of perinatal disease and mortality may be the early-pregnancy maternal atherogenic lipid profile. Women have an increase in lipid levels throughout a typical pregnancy as their gestational age increases, including levels of triglycerides (TG) and total cholesterol (TC) [4]. The fact that TG and TC are both taken up by the placenta, digested, and given to the fetus in various ways shows how important they are for fetal development [5]. However, high levels of maternal TC and/or TG are associated with preterm birth (PTB), pregnancy-induced hypertension (PIH), preeclampsia, and large for gestational age [6,7]. On the other hand, PTB and a higher risk of small-for-gestational-age birth in the fetus are connected to decreased TC levels during pregnancy [2,8].

Obesity and pre-pregnancy overweight are significant risk factors for illnesses of hypertension associated with pregnancy [9–11]. Maternal obesity is associated with a complex interplay of metabolic problems, including hypertension, insulin resistance, dyslipidemia, hypercoagulability, reduced endothelial function, inflammatory up-regulation, and altered adipokine profiles [7,12].

The ability to predict pregnancies with a high risk of developing HDP [13] is an important issue. Because there are no early signs that suggest the pathophysiology underlying the disease, the diagnosis is solely based on the clinical presentation [14]. The pathophysiology of HDP has been connected to a number of factors. There are several of them, including maternal age, parity, obesity, metabolic syndrome, prior family history of HDP, prior diabetes, prior renal disease, prior antiphospholipid syndrome, poor glucose tolerance, and dyslipidemia with African American ancestry [15–18].

It has been established that lipid buildup in artery intima cells causes endothelial dysfunction. A modified lipid profile also reduces the prostacyclin:thromboxane ratio, which is a crucial pathophysiological mechanism for pregnancy-induced hypertension [19]. A rise in triglycerides and small dense LDL may potentially contribute to impaired endothelial function [20]. Lipid profile changes during pregnancy are associated with preterm birth and poor perinatal outcomes [21].

Pregnant women may acquire insulin resistance throughout their pregnancy as a result of the normal pregnancy's progressive increase in the dose response of insulin to glucose. Early or mid-pregnancy hyperinsulinaemia and/or hyperglycemia have been recorded before the onset of preeclampsia, gestational hypertension, or both. In contrast to normotensive controls, third-trimester pregnant women with pregnancy-induced hypertension showed substantial hyperinsulinism in response to an oral glucose tolerance test (OGTT) [22].

Insulin resistance and hyperinsulinemia are signs of a healthy pregnancy [17]. Insulin resistance associated with pregnancy increases, reaches a peak in the third trimester, and then quickly returns to pre-pregnancy levels following delivery. The reason why women develop insulin resistance during healthy pregnancies is unknown. Cortisol, progesterone, estrogen, and

human placental lactogen variations have all been linked to pregnancy-related hormonal changes [23]. Insulin resistance is associated with hyperglycemia, hyperinsulinemia, and dyslipidemia [24]. It is known that the insulin resistance syndrome may also be accompanied by increased levels of leptin, tumor necrosis factor-a (TNF-a), and plasminogen activator inhibitor (PAI)-1 in addition to metabolic issues [24]. Despite the fact that many of these markers are proxies for insulin sensitivity, the observed relationships between many of these markers and the likelihood of pregnancy-induced hypertension further demonstrate that insulin resistance contributes to the development of pregnancy-induced hypertension [25]. Insulin resistance, glucose intolerance, and the emergence of pregnancy-associated hypertension, particularly the preeclampsia subtype, are all directly connected, according to Negrato et al. [23].

Indicators of the metabolic syndrome (insulin resistance, impaired glucose tolerance, and dyslipidemia) have been associated to the development of cardiovascular disease in pregnant women with hypertensive disorders [26, 27]. Insulin resistance and HDP are positively correlated [25]. In Nigeria, a portion of pregnant women with eclampsia had metabolic syndrome, according to Isezuo and Ekele [28]. They suggested that eclampsia screens for pre-eclamptic women would benefit from using metabolic syndrome indicators. The pathogenesis and incidence of pre-eclampsia-eclampsia have been associated to an increase in plasma antiphospholipid antibodies, despite this link being poorly understood [29,30].

The endothelial lesions seen in preeclampsia may also be caused by changes in lipid metabolism. Both the severity of proteinuria and the presence of hypertension seem to be related to the extent of endothelial damage [31]. Understanding the complex pathophysiology of preeclampsia may be made easier by considering a possible correlation between the severity of renal lesions as shown by proteinuria and the altered lipid profile [32].

It is still unknown whether hypertriglyceridemia becomes a risk factor for preeclampsia or whether there is any causal relationship between the two, but it does appear that high triglyceride levels increase the risk of placental vascular disorders, which result in endothelial dysfunction, atherosclerosis, and thrombosis [33]. High triglyceride levels may be contributing to preeclampsia based on the development of atherosclerosis in the placental spiral arteries of these women [34]. Preeclampsia patients' dyslipidemia, which is marked by high levels of triglycerides and VLDL, suggests that the condition and the endothelial lesions that cause atherosclerosis may share interfaces [32].

Early pregnancy dyslipidemia is associated with an elevated risk of preeclampsia [35]. Women with a history of preeclampsia display noticeable changes in lipid markers and a higher susceptibility to lipoprotein oxidation when compared to women who had a normal pregnancy [36]. Disorders of lipoprotein metabolism are reported to play a substantial role in the hypertension and proteinuria associated with preeclampsia [37]. Determining if cholesterol and glucose play a role in hypertensive problems in pregnant Nigerian women was the goal of this investigation.

2. MATERIALS AND METHODS

2.1 Study Design

A prospective cohort design was used for the investigation. Pregnant women who were receiving antenatal care at four different tertiary healthcare facilities in Nigeria, including the Ado-Ekiti campus of the Ekiti State University Teaching Hospital, the Federal Medical Centre, the University College Hospital, and the Adeoyo Maternity Hospital, comprised the participants. Due to their importance as important referral centers, hospitals draw visitors from all around the region. Women without hypertension who were in their first or second trimester of pregnancy were recruited as participants between June 2011 and October 2012. Participants were tracked through delivery.

2.2 Inclusion Criteria

Women first visited in the first or second trimester (20 weeks at booking) with systolic and diastolic blood pressures below 140 mm/Hg and participants who granted written informed consent are included in the study.

2.3 Exclusion Criteria

Women who are already hypertensive at study admission, pregnant women first observed at or before 20 weeks of pregnancy, and participants

with proteinuria measured by a dipstick at or above 300 mg/L (1+) are all excluded.

2.4 Study Population

The study included 521 patients in all, of whom 34 acquired various forms of HDP. Of the remaining 487 participants, 50 were lost for follow-up whose pregnancy outcomes were unknown. These participants were those who did not develop HDP until the end of the study period, those whose pregnancy outcome was unknown until the end of the study period, those who were lost for follow-up, and those who dropped out of the study for reasons unrelated to the study. Until the end of the research period, the remaining 437 individuals had normotension. Table 1 displays the various follow-up trimesters for both normotensive and hypertensive women.

Age, place of residence, marital status, educational background, occupation, ethnic group, diet history and social history, family history, past medical history/medication, and gynecological/obstetrical history were the socio-demographic characteristics of the study population that were gathered from each participant through a semi-pretest questionnaire.

2.5 Sample Collection

Approximately 10 milliliters of venous blood were taken aseptically from the antecubital vein of each participant after an overnight fast (10–12 hours), depending on whether they had HDP at the time of the development of hypertension or not. Reminder calls were placed to each participant before to their second and third trimester appointments. Following centrifugation at 4000 rpm for 5 minutes, blood samples were distributed into fluoride oxalate bottles and EDTA-containing sample bottles to extract plasma. Small aliquots of the plasma were kept at -20°C until analysis.

2.6 Determination of Fasting Plasma Glucose

Fasting plasma glucose was determined by glucose oxidase method, as described by Barham and Trinder [38] (Dialab, Austria).

2.7 Determination of Lipid Profile

According to Owoade et al. [39]'s description, the enzymatic approach was used to measure the plasma triglyceride concentration (Randox, United Kingdom). The Trinder-described enzymatic approach was used to measure the plasma cholesterol levels [39]. (Randox, United Kingdom). The enzymatic approach published by Frieldwald et al. [40] was used to determine the plasma high density lipoprotein cholesterol (Randox, United Kingdom). The concentration of low density lipoprotein was estimated using Friedewald's formula [40].

2.8 Statistical Analysis

For the analysis of the study population's data, the Statistical Package of Social Sciences (SPSS) software version 22.0 (SPP, Inc., Richmond, CA) was used. To determine whether the difference between the mean values was significant, paired student t-tests were utilized. Analysis of variance (ANOVA) was used to determine whether differences in group means were significant. Multiple variable comparisons

Table 1. Summary of participant's recruitment

Event	Normotensive n=487	Hypertensive n=34	Total n=521
Yes	0	34 (100.0%)	34
No (Normotensive)	437 (89.7%)	0	437
Lost for follow-up	50 (10.3%)	0	50
Trimester			
1, 2 & 3	69 (14.2%)	9 (26.5%)	78
2 & 3	64 (13.1%)	8 (23.5%)	72
1 & 2	26 (5.3%)	3 (8.8%)	29
1 & 3	69 (14.2%)	4 (11.8%)	73
1	158 (32.4%)	0	158
2	101 (20.7%)	10 (29.4%)	111

Values are in number of participants with percentage in parenthesis, % = percent, n= number of participants, HDP = hypertensive disorders, 1= first trimester, 2= second trimester, 3= third trimester

were made using Post-Hoc. The Pearson correlation coefficient was used to evaluate the link between each variable. For the purpose of comparing the means of qualitative (non-quantitative) variables, chi square analysis was performed. Cox proportional hazard regression model analysis was used as the technique to quantify the survival and hazard function in the survival analysis (time to event analysis). Statistical significance was set at p0.05 for two-sided probabilities. The appropriate standard deviation or standard error of the mean is used to report values.

3. RESULTS

The lipid profile and fasting blood glucose of hypertensive women during the three trimesters are displayed in Table 2. When all the parameters were examined using ANOVA, significant differences were found in each one. Comparing the first and second trimesters revealed no statistically significant differences in any of the variables. In the second and third trimesters, respectively, the mean levels of fasting plasma glucose and lipids (triglycerides, total cholesterol, HDL-C, and LDL-C) peaked. The lipid profile and fasting blood glucose of normotensive women during each of the three trimesters are shown in Table 3. Using ANOVA, significant differences were found in each parameter. While fasting plasma glucose gradually decreased from the first, second, and third trimesters, there was a gradual increase in the mean triglyceride values. The median levels of total cholesterol, HDL-C, and LDL-C gradually increased beginning in the first trimester, peaked in the second, and then decreased in the third.

In women with HDP during the first trimester, the lipid profile and fasting blood glucose are adjusted cox regression results in Table 4. There was no discernible variation in the lipid profile or fasting blood sugar. In the women with HDP during the second trimester, the lipid profile and fasting blood glucose are adjusted cox regression plotted in Table 5. In the second trimester of pregnancy, an increase in LDL-C of 1.0 will be linked to a 1.3 times rise in HDP development. Development of HDP is 1.3 times more likely for every additional unit of LDLC (HR = 1.284, B coefficient = 0.217). For each additional unit of triglycerides or total cholesterol, the production of HDP is inhibited in lipids

Table 2. Lipid profile and fasting blood glucose in women with hypertensive disorders in pregnancy

Index	1st trimester n=10	2nd trimester n=10	3rd trimester n=10	P1	P2	P3	P4
TG (mg/dL)	70.4±8.7	86.6±16.7	80.4±13.0	0.000*	0.425	0.774	0.629
TC (mg/dL)	155.8±10.4	191.2±17.0	174.4±12.0	0.000*	0.128	0.468	0.347
HDL (mg/dL)	52.2±5.6	65.0±6.2	59.2±10.3	0.000*	0.119	0.685	0.570
LDL (mg/dL)	89.5±10.6	108.9±11.8	99.3±13.6	0.000*	0.326	0.577	0.538
FPG (mg/dL)	65.8±5.5	76.1±4.1	74.5±6.7	0.000*	0.149	0.809	0.332

*Values are reported as means ± standard error of mean, P1 =values obtained from ANOVA, P2=values compared between 1st and 2nd trimester, P3=values compared between 2nd and 3rd trimester, P4=values compared between 1st and 3rd trimester, TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, FPG = Fasting plasma glucose, n = number of participants, * = significant at p < 0.05 (2-tailed), p = significant level*

Table 3. Lipid profile and fasting blood glucose in normotensive pregnant women

Index	N	1st trimester	2nd trimester	3rd trimester	P1	P2	P3	P4
TG (mg/dL)	58	73.6±4.3	79.8±6.5	100.6±6.5	0.000*	0.422	0.017*	0.001*
TC (mg/dL)	58	157.1±4.1	184.4±6.7	178.5±6.8	0.000*	0.000*	0.482	0.006*
HDL (mg/dL)	58	46.9±2.3	70.6±4.7	70.0±5.3	0.000*	0.000*	0.933	0.000*
LDL (mg/dL)	58	95.1±3.9	98.0±6.8	89.2±5.6	0.000*	0.694	0.311	0.360
FPG (mg/dL)	82	78.1±2.6	73.0±1.4	71.4±1.5	0.000*	0.081	0.472	0.016*

*Values are reported as means ± standard error of mean, P1 =values obtained from ANOVA, P2=values compared between 1st and 2nd trimester, P3=values compared between 2nd and 3rd trimester, P4=values compared between 1st and 3rd trimester, TG = Triglyceride, TC = Total cholesterol, HDL = High density lipoprotein, LDL = Low density lipoprotein, FPG = Fasting plasma glucose, n=number of participants, * = significant at p < 0.05 (2-tailed), p = significant level*

Table 4. Adjusted cox regression of lipid profile and fasting blood glucose in women with hypertensive disorders in pregnancy during 1st trimester

Index	B coefficient	Hazard ratio	Confidence interval: Lower	Upper	p-value
Glucose (mg/dl)	-0.007	1.009	0.994	0.967	1.021
Triglyceride (mg/dl)	-0.031	0.840	0.969	0.612	1.534
Total cholesterol (mg/dl)	0.271	2.593	1.311	0.132	13.033
HDL (mg/dl)	-0.270	0.388	0.763	0.077	7.560
LDL (mg/dl)	-0.257	0.387	0.773	0.078	7.683

**= significant at p<0.05, p= significant level, HDL = High density lipoprotein, LDL = Low density lipoprotein*

Table 5. Adjusted cox regression of lipid profile and fasting blood glucose in women with hypertensive disorders in pregnancy during 2nd Trimester (Without HDL)

Index	B coefficient	Hazard ratio	Confidence interval:		p-value
			Lower	Upper	
Glucose (mg/dl)	0.069	1.072	0.997	1.152	0.060
Triglyceride (mg/dl)	-0.024	0.976	0.955	0.998	0.031*
Total cholesterol (mg/dl)	-0.262	0.770	0.624	0.950	0.015*
LDL (mg/dl)	0.250	1.284	1.044	1.579	0.018*

**= significant at p<0.05, p= significant level, LDL = Low density lipoprotein*

Table 6. Adjusted cox regression of lipid profile and fasting blood glucose in women with hypertensive disorders in pregnancy during 2nd trimester

Index	B coefficient	Hazard ratio	Confidence interval		p-value
			Lower	Upper	
Glucose (mg/dl)	0.052	1.053	1.002	1.108	0.043*
Triglyceride (mg/dl)	-0.008	0.992	0.830	1.186	0.933
Total cholesterol (mg/dl)	-0.002	0.998	0.409	2.436	0.997
HDL (mg/dl)	-0.019	0.981	0.404	2.380	0.966
LDL (mg/dl)	-0.005	0.995	0.406	2.436	0.991

**= significant at p<0.05, p= significant level, HDL = High density lipoprotein, LDL = Low density lipoprotein*

Table 7. Un-adjusted cox regression of lipid profile and fasting blood glucose in women with HDP

Index	B coefficient	Hazard ratio	Confidence interval		p-value
			Lower	Upper	
Glucose 1	-0.010	0.990	0.971	1.009	0.305
Glucose 2	0.011	1.011	0.994	1.028	0.210
Triglyceride 1	0.005	1.005	0.993	1.016	0.417
Triglyceride 2	-0.002	0.998	0.989	1.008	0.730
Total cholesterol 1	-0.003	0.997	0.986	1.008	0.616
Total cholesterol 2	-0.003	0.997	0.988	1.006	0.545
High density lipoprotein1	0.009	1.009	0.987	1.031	0.434
High density lipoprotein 2	-0.001	0.999	0.986	1.012	0.904
Low density lipoprotein 1	-0.005	0.995	0.984	1.007	0.406
Low density lipoprotein 2	-0.003	0.997	0.988	1.005	0.452

**= significant at p<0.05, p= significant level, 1= first trimester, 2= second trimester, 3= third trimester*

by 2.4 percent [100% - (100% X 0.976)] and 33.0 percent [100% - (100% X 0.770), respectively. (HR =0.976 and 0.770). According to the negative B coefficient (B coefficient = -0.024 and -0.262), greater triglyceride and total cholesterol levels in HDP women will be linked to a decreased risk of HDP development in the second trimester of pregnancy.

The lipid profile and fasting blood glucose levels of women with HDP during the second trimester are illustrated in Table 6 using adjusted Cox regression. Fasting plasma glucose was statistically significant after correcting or controlling for all the biochemical markers (p=0.043). In the second trimester of pregnancy, HDP will develop at a rate that is 1.1 times higher for every 1.0 increase in fasting plasma glucose. This means that the development of HDP is 1.053 times more likely for every additional unit of fasting plasma glucose (HR = 1.053, B coefficient = 0.639). Unadjusted cox regression of the lipid profile and fasting blood glucose in HDP-affected females is shown in Table 7.

4. DISCUSSION

One of the reasons of perinatal morbidity and mortality may be the maternal atherogenic lipid profile early in pregnancy [41]. Women have an increase in lipid levels throughout a typical pregnancy as their gestational age increases, including levels of triglycerides (TG) and total cholesterol (TC) [2,41]. Both TG and TC are taken up by the placenta, which then processes them and gives them to the fetus in diverse ways. This proves that both lipids are essential for the development of the fetus [2, 5]. However, high levels of maternal TC and/or TG are associated with HDP and premature birth [2,36].
Only triglycerides significantly increased during the second and third trimesters of pregnancy in this study's normotensive women. Total cholesterol, high density lipoprotein cholesterol (HDLC), and low density lipoprotein cholesterol (LDLC) considerably increased during the second trimester and sharply dropped during the third trimester in both hypertensive and normotensive women. In normotensive women, there were appreciable increases in mean total cholesterol, HDLC, and triglycerides between the first and second trimesters as well as between the second and third trimesters. Similar to this, during the first and third trimesters, the triglycerides, total cholesterol, and HDLC levels of the normotensive women considerably increased. There was no observable mean difference between the first, second, and third trimesters in the hypertensive women's triglycerides, total cholesterol, HDLC, and LDLC.

In this study, it was discovered that triglycerides, total cholesterol, and low-density lipoprotein cholesterol (LDLC) are biomarkers and predictors of hypertensive disorders of pregnancy (HDP) in the second trimester. Although they are risk factors for the development of HDP in the second trimester, triglycerides and total cholesterol also correlate well with LDLC. HDP formation was decreased by 2.4 and 33.0 percent for every additional unit of triglycerides or total cholesterol, respectively. Higher triglyceride and total cholesterol levels will reduce the likelihood of developing HDP during the second trimester of pregnancy in HDP women (B coefficient = -0.024 and -0.262). However, with every additional unit of LDLC during the second trimester of pregnancy, HDP development is 1.284 times more likely (HR = 1.284, B coefficient = 0.217). The second trimester saw considerably higher mean values across all lipid components in hypertensive women (p 0.05).

The findings of this analysis are in line with past research that discovered a positive correlation between the outcome of the pregnancy and the maternal lipid profile [42,43]. The maternal lipid profile and pregnancy success, however, were reported to have a poor association in some prior studies [2,19,36]. These conflicting results may be partially explained by different research strategies, such as case-control studies [42,43] versus cohort studies [7,44], small sample sizes [7,43], insufficient adjustment for confounders [43,44], or different study populations [42,43,45].

Insulin, an anabolic hormone, regulates glucose, lipid homeostasis, and energy storage through its metabolic effects on the usual insulin-responsive tissues [46]. In particular, insulin promotes the storage of glucose as glycogen in the liver and skeletal muscles and facilitates the deposition of fatty acids in the form of triglycerides in adipose tissue [47]. Insulin-mediated anabolic metabolic effects are reduced during insulin resistance in the conventionally insulin-responsive tissues. In a healthy state, insulin boosts endothelial nitric oxide production, which has a relaxing and anti-inflammatory effect [24]. In contrast, insulin resistance selectively impairs the insulin-stimulated nitric oxide pathway, and compensatory hyperinsulinemia may activate the Nitrogen-Activated Protein Kinase (MAPK) pathway, which enhances vasoconstriction, promotes inflammation, increases sodium and water retention, and increases blood pressure [24].

5. CONCLUSION

According to the findings of the current investigation, hypertensive problems during

pregnancy in Nigerian women were associated with changes in lipid profiles and fasting blood glucose. In women with systolic blood pressure 130 mmHg and diastolic blood pressure 80 mmHg at the first antenatal appointment, early estimate of fasting plasma glucose and lipid profile may be helpful in predicting the development of hypertensive diseases in pregnancy.

ETHICAL APPROVAL AND CONSENT

The ethical approval for the study was obtained from the University of Ibadan/University College Hospital (UI/UCH) Joint Ethics Committee Ibadan, Oyo State, Nigeria. (UI/UCH EC Registration Number: NHREC/05/01/2008a; UI/UCH Ethics Committee assigned number: UI/EC/10/0195). A written informed consent was obtained from each participant before recruitment into the study.

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COMPETING INTERESTS

Authors have declared that no competing interests exist.

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