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ADIPOQ gene is linked to emotional eating behaviour in young Nigerian adults independent of psychological traits

Awoyemi Abayomi Awofala[1*], Olusegun Emmanuel Ogundele[1,2] and Khalid Olajide Adekoya[2]

Abstract

Background: A disturbance in eating behaviour (EB) is the hallmark of patients with eating disorders, and depicts a complex interaction of environmental, psychological and biological factors. In the present study, we propose a model of association of genetic susceptibility—represented by adiponectin (*ADIPOQ*) gene—with eating behavioural and psychological traits.

Results: Evaluation of the distribution of a polymorphism of the *ADIPOQ* (rs1501299 G>T) with respect to three EB factors involving cognitive restraint, uncontrolled eating and emotional eating revealed that *T*-allele in rs1501299 was associated with a decreased susceptibility to emotional EB in codominant (e.g., *GG* vs. *TT*) (beta-coefficient [β] = 2.39, 95% Confidence interval [CI] = − 4.02, − 0.76; *p* value [*p*] = 0.02), recessive (*GG* + *GT* vs. *TT*) (β = − 2.77, 95% CI = − 3.65, − 0.69; *p* = 0.005) and additive (*GG* = 0, *GT* = 1, *TT* = 2) (β = − 1.02, 95% CI = − 1.80, − 0.24; *p* = 0.01) models of inheritance. The presence of the *T*-allele was not significantly associated with psychological factors involving depression, anxiety and stress. Finally, none of the psychological traits significantly predicted any of the EB factors after controlling for age, body weight and gender.

Conclusions: Our data suggest that genetic variant in *ADIPOQ* locus may influence human emotional eating behaviour.

Keywords: Eating behaviour, Emotional eating, + 276G>T, *ADIPOQ* gene, Psychological traits

Introduction

The cognitive, behavioural and emotional aspects of eating habit encompass environmental, psychological and biological factors (LaCaille 2013). According to a gene—environmental approach, the different aspect of the eating behaviour can be influenced by genetic variability, explaining a part of the variance of the contribution to eating behaviours and different phenotypical features (Tholin et al. 2005). In this view, several genes have been investigated as possible candidates (Grimm and Steinle 2011). Among these, great interest has been recently devoted to genes encoding adiponectin pathway (Awofala et al. 2019; Christodoulou et al. 2020).

Adiponectin (*ADIPOQ*) is an adipocyte secreted 247—amino acid peptide that circulates in large amount in plasma (Ghadge et al. 2018; Martin 2014) and is involved in multiple functions such as insulin sensitization, cardioprotection, and anti-inflammatory processes (Berg et al. 2001; Ouchi et al. 2006; Yamauchi et al. 2007). Circulating adiponectin plays a potential role in the regulation of feeding behaviour and in energy homeostasis (Qi et al. 2004; Kubota et al. 2007), and appears to be related to psychological functioning. Indeed, altered levels of adiponectin have been found in eating disorders such as anorexia nervosa, binge eating disorder and bulimia nervosa (Khalil and Hachem 2014) and in several other psychiatric

*Correspondence: awofalaaa@tasued.edu.ng
[1] Department of Biological Sciences, Tai Solarin University of Education, Ijagun, P.M.B. 2118, Ijebu-Ode, Ogun State, Nigeria
Full list of author information is available at the end of the article

conditions including depression and anxiety disorders (Wędrychowicz et al. 2014; Carvalho et al. 2014).

Among *ADIPOQ* single nucleotide polymorphisms (SNPs), the SNP, rs1501299 (+276G/T in intron 2 on chromosome 3) is perhaps one of the most studied of the common *ADIPOQ* gene variant; studies have linked rs1501299 with circulating adiponectin and cardiometabolic consequences of obesity such as type 2 diabetes, insulin resistance and cardiovascular risk (Hara et al. 2002; Jang et al. 2005, 2006; de Luis et al. 2016). The *T*-allele in *ADIPOQ* rs1501299 was associated with increased insulin levels and decreased circulating adiponectin (de Luis et al. 2016). However, it has neither been investigated if the SNP is involved in human eating behaviour nor psychological traits, particularly in Nigerians.

Studies assessing genetic and environmental influences in EB using the original Three Factor Eating Questionnaire (TFEQ) (Stunkard and Messick 1985), a widely used self-assessment instrument that measures 3 domains: cognitive restraint, hunger, and disinhibition have shown that genetics has important effects on EB (Steinle et al. 2002; Rohde et al. 2015). An earlier US family study which included 624 Amish men and women reported 28%, 40%, and 23% as heritability estimates of restraint, disinhibition, and hunger (Steinle et al. 2002). A more recent German study, which included 548 Sorbs and 350 replication German cohort found that 3 variants in adiponectin gene were nominally significantly associated with hunger (rs2036373) and disinhibition (rs822396, rs864264) based on additive model of inheritance (Rohde et al. 2015). Interestingly, study has recently shown that elevated blood adiponectin level is causally related to eating disinhibition in central European population (Awofala et al. 2019). Of note, as the original factor structure of TFEQ could not be replicated in several studies (Karlsson et al. 2000; Mazzeo et al. 2003), a refined 18—item version (TFEQ-R81) (Khalil and El Hachem 2014) was used in the present study.

Investigation of the genetic influence of EB and the associated psychobehavioural traits is important for extending our understanding of food intake regulation and energy balance as well as in the pathophysiology of eating disorders. Our aim was to evaluate the role of adiponectin gene on EB in a Nigerian cohort of young adults.

Methods

Study design

This was a cross-sectional study at a public university.

Study population

The sample cohort included 560 healthy young adults of Nigerian descent. Participants were extensively phenotyped for a wide range of anthropometric, eating behavioural and psychological traits including weight, height, depression, anxiety, stress, and cognitive, behavioural and emotional aspects of eating habits using clinical instruments and standardized questionnaires. The anthropometric assessment is described more in depth from a previous publication (Ogundele et al. 2018). Genetic information was available for 78 of these participants.

Assessments

A total of 555 participants with mean age of 21.5 ± 2.9 years and mean BMI 20.8 ± 5.4 kg/m^2 completed the revised Three-Factor Eating Questionnaire (TFEQ-R18) scale (Karlssson et al. 2000). The TFEQ-R18 covers 3 EB domains: the 9-item uncontrolled eating scale that was constructed from the hunger (6 items) and disinhibition (3 items) scales of the original TFEQ and assesses the tendency to lose control over eating when feeling hungry or when exposed to external stimuli; the 6-item cognitive restraint scale that assesses control over food intake to influence body weight and body shape; and, the 3-item emotional eating scale that was constructed from items included in the disinhibition scale, measures the propensity to eat in response to a range of negative emotions such as anxiety, depression and anger. Although constructed using data from obese adults, TFEQ-R18 has been shown to be applicable to other populations (De Lauzon et al. 2004; Anglé et al. 2009), and was satisfactorily replicated in the present study sample (unpublished data).

Participants also completed the short version of the Depression Anxiety Stress Scale (DASS-21; Lovibond and Lovibond 1996). The scale has been translated and validated in many languages with excellent values of reliability, and strong internal consistency in several ethnic groups for both adults and adolescents (Szabó 2010; Oei et al. 2013; Tonsing 2014), and covers 3 broad spectrums of psychological symptoms involving depression, anxiety and stress, and with each containing a 7-item sub-scales that measure the frequency and severity of each symptom or trait in clinical and non-clinical sample. In our study, the Cronbach α internal-consistency reliability for each of the 7-item scores was satisfactorily high in depression ($\alpha = 0.78$), anxiety ($\alpha = 0.81$) and stress ($\alpha = 0.73$) scales.

Genetic analysis

Genotyping of *ADIPOQ* rs1501299 *G*/*T* (+276G > T) was performed as described previously (Kubota et al. 2007). Genomic deoxyribonucleic acid (DNA) was isolated

from dried blood spot (DBS) samples of 78 participant using Zymo Research (ZR) DNA Card Extraction Kits according to the manufacturer's protocol. The genotyping reaction was performed with SNP-specific polymerase chain reaction using single-base primer extension technology (Sequenom MassARRAY Genotyping System (Sequenom, San Diego, CA, USA) based on the method described by Yue and colleagues (Yu et al. 2015). The genotyping efficiency was > 92%.

Statistical analysis

Variables were reported as means ± SD. The independent sample *t* test was used to compare gender groups on the whole sample ($N = 555$). Relationships of BMI with EB and psychological traits and between psychological components and eating behaviour were established using the Pearson's correlation coefficient test. The overall associations of *ADIPOQ* genotypes (*GG*, *GT*, *TT*) with EB phenotypes and psychological traits were tested. To comprehensively analyse the association between the exposure and the outcomes, genotype analyses were performed assuming codominant (*GG* vs. *GT* vs. *TT*), recessive ($GG + GT$ vs. *TT*), and multiplicative or additive ($GG = 0$, $GT = 1$, $TT = 2$) models by means of linear regression adjusting for gender. As *ADIPOQ* rs1501299 *G*/*T* genotype was available for 78 participants in this study, a complete case genotype analysis was carried out on the sample. Independent sample *t* test was performed to evaluate BMI, EB scores, and psychological trait scores differences in the participants with different *ADIPOQ* genotypes. General Linear Model (GLM) was also adopted to test the relationship between psychological trait scores and the three eating behavioural phenotypes adjusting for age, gender and BMI. All statistical analyses were implemented in statistical analysis system (SAS) version 9.2.1.

Results

The demographic and clinical characteristics of male and female participants are reported in Table 1. Female participants were more likely to be younger (21.4 ± 2.6 vs. 22.1 ± 3.0; $t = 2.66$, $p = 0.008$) and had higher BMI (21.5 ± 4.5 vs. 18.1 ± 5.2; $t = 2.63$, $p = 0.01$) than their male counterparts. Other comparisons between gender groups were not significant.

Correlational analysis of BMI with eating behavioural phenotypes and psychological traits revealed uncontrolled eating as the only TFEQ-R18 scale that was significantly correlated with BMI ($r = -0.27$, $p = 0.02$). None of the psychological traits was significantly correlated with BMI. Of note however, significant positive correlations of emotional eating scale ($r = 0.33$–0.42, $p < 0.001$) and uncontrolled eating scale ($r = 0.35$–0.36, $p < 0.001$) with depression, anxiety and stress psychological trait scales were observed.

The distributions of genotypes of rs1501299 with respect to the 3 eating behavioural phenotypes are presented in Table 2. In the codominant models, participants with less frequent *TT* genotypes were less likely to develop emotional EB when compared with those with more frequent *GG* genotypes ($\beta = -2.39$, 95% CI = −4.01, −0.76; $p = 0.02$). This observation was recapitulated in the recessive ($GG + GT$ vs. *TT*) ($\beta = -2.77$, 95% CI = −3.65, −0.69; $p = 0.005$) and additive ($GG = 0$, $GT = 1$, $TT = 2$) ($\beta = -2.77$, 95% CI = −3.65, −0.69; $p = 0.005$) models of inheritance. Notably, none of these genotypes was significantly associated with uncontrolled or cognitive restraint scales of TFEQ-R18.

The comparisons of clinical variables, in relations to rs1501299 polymorphism, are reported in Table 3. *G*-allele carriers, as compared to *TT* genotype carriers, reported significantly higher emotional eating scores (5.9 ± 2.4 vs. 3.8 ± 2.4; $t = 2.87$, $p < 0.01$).

Table 1 General, psychological and eating behavioural characteristics of participants

Variable	Total ($n = 555$)	Male ($n = 121$)	Female ($n = 434$)	Independent sample *t* test
Age (years)	21.5 ± 2.9	22.1 ± 3.0	21.4 ± 2.6	2.66*
BMI (kg/m^2)	20.8 ± 5.4	18.1 ± 5.4	21.5 ± 4.5	− 2.63*
Cognitive restraint	16.0 ± 4.3	16.0 ± 4.0	16.1 ± 4.2	− 0.26
Uncontrolled eating	19.7 ± 5.6	19.9 ± 5.2	19.6 ± 5.5	0.52
Emotional eating	5.8 ± 2.7	5.8 ± 2.2	5.7 ± 2.8	− 0.10
Depression	4.7 ± 4.0	5.3 ± 3.9	4.5 ± 4.1	0.79
Anxiety	6.0 ± 4.5	5.7 ± 4.4	6.0 ± 4.6	− 0.27
Stress	7.2 ± 4.0	7.7 ± 3.6	7.1 ± 4.1	0.55

Statistics: data are reported as numbers (mean ± SD)

Comparisons between male and female participants—*$P < 0.05$

BMI body mass index, *n* sample size

Table 2 Distribution of rs1501299 genotypes according to the 3 eating behavioural scales

Scales	Eating behaviour (SE)			Genetic models					
	GG ($n=29$)	GT ($n=37$)	TT ($n=12$)	Codominant β (95% CI)[a]	*P*	Recessive β (95% CI)	*P*	Additive β (95% CI)	*P*
Cognitive restraint	17.5 ± 0.7	17.0 ± 0.8	15.5 ± 1.8	− 2.02 (− 5.28, 1.25)	0.48	− 1.71 (− 4.68, 1.26)	0.26	− 0.91 (− 2.46, 0.64)	0.25
Emotional eating	6.1 ± 0.5	5.8 ± 0.4	3.8 ± 0.7	− 2.39 (− 4.02, − 0.76)	**0.02**	− 2.17 (− 3.66, − 0.69)	**0.005**	− 1.02 (− 1.80, − 0.24)	**0.01**
Uncontrolled eating	20.4 ± 1.1	20.4 ± 1.0	19.0 ± 1.7	− 1.45 (− 5.28, 2.37)	0.75	− 1.32 (− 4.82, 2.17)	0.46	− 0.54 (− 2.41, 1.32)	0.51

Eating behaviour phenotypes include cognitive restraint, emotional eating and uncontrolled eating.

β beta-coefficient, *SE* standard error, *n* sample size

[a] Only comparison of *GG* (reference) versus *TT* genotypes

Table 3 Comparison of clinical variables according to rs1501299 genotypes

Variable	G-allele carriers (GG and GT genotypes; $n=66$)	TT genotype ($n=12$)	Independent sample *t* test
BMI (kg/m^2)	20.5 ± 5.5	21.1 ± 3.2	− 0.39
Cognitive restraint	17.2 ± 4.6	15.5 ± 6.2	1.13
Uncontrolled eating	20.4 ± 5.7	19.0 ± 5.7	0.76
Emotional eating	5.9 ± 2.4	3.8 ± 2.4	2.87*
Depression	4.3 ± 3.7	4.0 ± 3.5	0.24
Anxiety	5.3 ± 3.9	5.5 ± 5.5	− 0.18
Stress	6.6 ± 3.7	6.6 ± 4.0	− 0.00

Statistics: data are reported as numbers (mean ± SD)

BMI body mass index, *n* sample size

* $P<0.05$

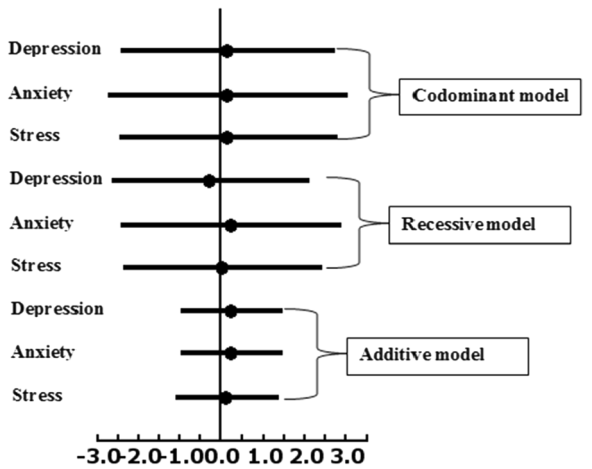

Fig. 1 Forest plots of mean differences in psychological components (depression, anxiety, and stress) with rs1501299 allele in the codominant, recessive and additive models

The genetic association between rs1501299 and psychological traits is shown in Fig. 1. None of the psychological traits was significantly associated to rs1501299 polymorphism.

Finally, General Linear Model showed that none of the psychological traits significantly predicted any of the TFEQ-R18 subscales after controlling for age, BMI and gender (Table 4).

Discussion

In the present study, we evaluated *ADIPOQ* rs1501299 variant as a risk factor for eating behavioural phenotypes and their associated psychobehavioural traits. The main findings are that the minor allele carrier in *ADIPOQ* rs1501299 ($G>T$) variant is significantly associated with a decreased emotional EB but not cognitive and uncontrolled EB factors. This significantly decreased in emotional EB, which indicate individuals' lower eating responses, was not influenced by psychobehavioural factors, as no significant association between *ADIPOQ* rs1501299 SNP and psychological traits was found. Moreover, none of the psychological traits significantly predicted EB factors after controlling for age, sex and body weight.

The emotional eating scale of the TFEQ-R18 depicts a measure of overeating induced by negative mood and emotions, and indicates individuals' tendencies to overeat as opposed to appetite loss when experiencing negative emotions (e.g., fear, anxiety, depression or anger).

Table 4 Relationship between psychological traits and the 3 eating behavioural phenotypes

DV = cognitive restraint			DV = emotional eating			DV = uncontrolled eating		
IV	β	*p*	IV	β	*p*	IV	β	*p*
Age	− 0.02	0.91	Age	0.09	0.39	Age	0.20	0.40
BMI	0.18	0.08	BMI	− 0.02	0.79	BMI	− 0.18	0.16
Gender	− 0.67	0.59	Gender	− 0.81	0.24	Gender	− 4.11	0.01
Stress	0.34	0.08	Stress	0.18	0.10	Stress	0.07	0.76
Anxiety	− 0.19	0.28	Anxiety	0.03	0.74	Anxiety	0.33	0.12
Depression	− 0.07	0.69	Depression	0.03	0.73	Depression	0.08	0.71

Statistics: Tables report General Linear Models of eating behavioural traits by psychological factors

DV dependent variable, *IV* independent variable, *BMI* body mass index, *β* beta-coefficient

Thus, emotional eaters have difficulties in distinguishing between the psychological states of negative emotions on one hand and hunger-satiety on the other. Interestingly, our results indicate that emotional eating as measured by the TFEQ-R18 is influenced by the *T*- allele in *ADIPOQ* rs1501299 (*G* > *T*). This suggests that persons with less frequent *TT* genotypes exhibit lower emotionally induced eating than do those with more frequent *GG* genotypes. Importantly, that this study showed genetic association with only emotional EB but not others may indicate genetic dissociation among the 3 eating behavioural traits. In support of this, two earlier female twin studies found genetic influences on only the disinhibition scale of the original TFEQ (Mazzeo et al. 2003; Neale et al. 2003), which may be comparable with our results for emotional eating. In addition, an observational study of the role of adiponectin in EB (Rohde et al. 2015) showed that genetic polymorphisms in *ADIPOQ* were related to disinhibition and hunger scales of the original TFEQ. A more recent study from our group indicated that the observational association between the effect allele carriers in the *ADIPOQ* SNPs showing elevated adiponectin serum levels along with eating disinhibition was causal (Awofala et al. 2019). Of note, the disinhibition scale evaluates impulsive eating in response to emotional, cognitive and social cues. Indeed, items of the TFEQ-R18 emotional eating scale were derived from the original TFEQ disinhibition scale.

It is particularly noteworthy that the direction of association between adiponectin and eating behaviours reported in the studies reviewed above differs in our present study. The opposite relationship can in part be explained by the use of *ADIPOQ* SNPs other than the one in the present study. In addition, it appears that factors that make up the disinhibition scale (i.e., emotional, cognitive and social factors) need to be disentangled to found out if they have specific relationship to adiponectin gene. Nevertheless, the observed association between *ADIPOQ* rs1501299 (*G* > *T*) variant and decreased emotional EB seems to correlate well with known roles of the minor *T* allele at *ADIPOQ* rs1501299 *G*/*T* polymorphism in adiponectin levels and insulin resistance: *T* allele was associated with high levels of adiponectin and insulin sensitivity in non-diabetic men (Hivert et al. 2008). However, subjects with the *T* allele at rs1501299 showed significantly lower high-density lipoprotein (HDL)-cholesterol and higher brachial-ankle pulse wave velocity (baPWV) leading to an increase in arterial stiffness in essential hypertensive patients (Kawai et al. 2013). The latter discrepancy among the study results may be due to several factors such as differences in study design and subject characteristics as well as diversities in basic metabolic health status and genetic properties. Our study subjects were young adults without any history of disease or medication.

We have previously shown that *ADIPOQ* rs1501299 (*G* > *T*) variant was not significantly correlated to several measures of obesity in young Nigerian adults (Kubota et al. 2007). However, on the basis of the results presented above, it is clear that EB is influenced at least in part by adiponectin gene. It thus appears that some genetic variants affecting EB may to a certain extent not be involved in the development of obesity. Indeed, several variants of *ADIPOQ* studied in relation to obesity have produced inconsistent results across different populations (Ukkola et al. 2005; Hivert et al. 2008; Cohen et al. 2011; Kaur et al. 2018). Notably however, several studies in which TFEQ-RI8 was used as a measure of EB have reported close associations of emotional eating and cognitive restraint scales with BMI (Elfhag and Linné 2005; de Lauzon-Guillain et al. 2006; Anglé et al. 2009). In the present study, only scores on the uncontrolled eating scale were significantly associated with BMI in the univariate analysis; this association failed to reach statistical significance in the adjusted model. Indeed, it appears higher body weight make people eat differently (Anglé et al. 2009). A 2-year follow-up French study by de Lauzon-Guillain et al. (2006) indicated that initial scores of

cognitive restraint were not connected with subsequent adiposity changes in either adults or adolescents; however, in all age groups studied, higher values of initial adiposity including BMI predicted a larger increase in cognitive restraint score over the two year period. Moreover, findings from a Swedish young male twin study indicated that obese individuals exhibited more emotionally induced eating than do non-obese ones (Tholin et al. 2005).

Several studies have reported the correlation between circulating adiponectin and psychological factors, particularly depression, anxiety and stress (Carvalho et al. 2014; Diniz et al. 2012; Unsal et al. 2012). Low levels of adiponectin were found in several psychiatric conditions, including severe depression (Carvalho et al. 2014; Diniz et al. 2012) and anxiety disorders (Unsal et al. 2012). In addition, higher levels of adiponectin were significantly related to lower self-ratings of depression and anxiety but stress in patients with anorexia nervosa during inpatient treatment (Buckert et al. 2017). In this cross-sectional study in a young Nigerian population, we did not see any significant association between *ADIPOQ* rs1501299 and psychological factors. In addition, our study observed relationship between psychological traits and EB that failed to reach statistical significance in the adjusted model. Though adiponectin gene is a known predictor of adiponectin concentrations in several populations (Hivert et al. 2008; Cohen et al. 2011; Tanimura et al. 2011; de Luis et al. 2016), we have no serum adiponectin levels available in the present study to confirm this in the young Nigerian population. Further studies are thus warranted to shed more light on the complexity of our results.

This study is limited in several respects. First, we are aware that the significant results presented could prove to be false positives because of the small sample size, and as such further studies using larger sample are warranted. It should be noted that due to our small sample size, we were unable to carry out rigorous stratify analysis to further shed light on the veracity of our results. However, with larger sample, the functional and biological relevance of the described variant would require further study. In addition, our results cannot be easily generalized to older adults and other ethnic groups in Nigeria. Further, we did not run a replication study neither did have available serum adiponectin levels that could add validity to our results. Notwithstanding these limitations, the earlier interpretations of our results should enable the reader to reach a reasonable conclusion.

In conclusion, the present study suggests that the *T* allele at *ADIPOQ* rs1501299 G/T polymorphism represents a putative protective factor for young people to reduce their emotional EB. Hence, the present results may prove to be useful for personal-based early prevention and management of pathological EB factors such as binge eating.

Abbreviations

ADIPOQ: Adiponectin gene; baPWV: Brachial-ankle pulse wave velocity; BMI: Body mass index; CI: Confidence interval; DASS: Depression anxiety scale; DBS: Dried blood spot; DNA: Deoxyribonucleic acid; EB: Eating behaviour; GLM: General linear model; GWAS: Genome-wide association studies; HDL: High-density lipoprotein cholesterol; SAS: Statistical analysis system; SE: Standard error; SNP: Single-nucleotide polymorphisms; TFEQ: Three-factor eating questionnaire; ZR: Zymo research.

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Authors' contributions

AAA conceived the study, conducted the analysis and drafted the manuscript. OEO took part in the study design, data generation and analysis plan. KOA provided technical and material support and assisted in the analysis plan. All authors read and approved the final manuscript.

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Availability of data and materials

Not applicable.

Ethics approval and consent to participate

The present study was conducted in compliance with the Declaration of Helsinki. Study procedures were fully explained to participants prior to collection of data; after which participants were asked to sign written informed consent for participation in the study and for genetic analysis. This study protocol was approved by the Health Research Ethics Committee (HREC) of Lagos University Teaching Hospital (LUTH) in the University of Lagos (protocol number: ADM/DCST/HREC/APP/80).

Consent for publication

Not applicable.

Competing interests

The authors declare that they have no competing interests.

Author details

[1] Department of Biological Sciences, Tai Solarin University of Education, Ijagun, P.M.B. 2118, Ijebu-Ode, Ogun State, Nigeria. [2] Department of Cell Biology and Genetics, University of Lagos, Akoka, Lagos State, Nigeria.

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