

# Night Clubbing Habits and Co-Occurrence of Lifestyle-Related Behaviors, Clinical and Biochemical Risk Factors for Cardiovascular Disease



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## Abstract

**Background/Aim:** There is a global increase in life-style related risk factors for CVD. However, few studies have examined the prevalence of these factors in the general population at different social settings/gathering such as night clubs, drinking points/bars and discotheques where different lifestyle habits are freely displayed. Identifying such factors can lead to innovative strategies for improving cardiovascular health. The aim of this study was to determine the prevalence of lifestyle, clinical and biochemical risk factors for CVD among night clubbers in Southern Nigeria.

**Methods:** Anthropometrics, questionnaires, clinical and blood samples were collected from 255 participants who were classified as night clubbers (n=130) and non-night clubbers (n=125) and analyzed for body mass index (BMI), waist circumference (WC), socio-demographics, life style related behaviors, blood pressure indices, lipid profile and markers of insulin resistance using standard methods.

**Results:** Compared to non-clubbers, night clubbers had significant higher prevalence of CVD risk factors including habitual alcohol consumption (P<0.001), physical inactivity (P=0.026), poor dietary habits (P<0.001), short sleep duration (P<0.001), abnormal lipid sub fractions (high serum TG, T-chol and LDL levels) (P<0.001), raised blood pressure indices (SBP, DBP, MAP) (P<0.0001), raised serum uric acid level (P<0.0001) and a marker of insulin resistance (HOMA-IR) (P=0.060).

**Conclusion:** CVD risk factors are more prevalent among night clubbers than non-clubbers. Night club, discotheque, tavern, drinking point and bar patrons should be included among target populations in development of interventions necessary to reduce the high prevalence of CVD risk factors in the general population, because current risk factors become future diseases and public health burden. Knowledge of risk factors can be used to shift their population distribution.

**Keywords:** Night Clubbing; Drinking Point; Tavern; Discotheque; Lifestyle Habits; Heart Disease; Hypertension; Metabolic Disorders.

## Introduction

Over the years, it is a common practice to find people from various walks of life unwind daily or during weekends, irrespective of their social class and marital status at different night clubs/discotheques, drinking bars and taverns for various psychosocial reasons. On a typical Friday night, fun loving guys and ladies hang out fun. As a matter of fact, clubbing is as old as civilization itself. It is also a known fact that the human nature abhors vacuum and boredom, for something must spice up living or else life would not be worth living [1]. For instance, going back to the origin of night clubbing in the first two decades of 1900s, when the American working class get together at juke joints and honky funks and dance to the music played either in a juke or on a piano. Though later banned, clubbing was later revived in 1933 after which night clubbing spread throughout the world including

Asia, Europe, Africa as well as Australia [2]. In Nigeria, there is a rapid proliferation of club's houses/discotheques and drinking points/bars which offer entertainment for not only adolescents and young adults, but also across other age groups and gender [3,4] and those of legal drinking and smoking age of 18+ [5].

Many working-class clubbers patronize these entertainment houses for many psychosocial reasons including:

- i. To unwind, relax and rest after a bustling and hectic workday or week from stressful work environments and hard workmanship. It is seen as a popular way for physiological and psychological rejuvenation and increases productivity.
- ii. To socialize and meet new friends and potential marriage mates

iii. To escape marital stress especially for those who are married.

iv. To have unlimited fun and to satisfy other desires [2].

At these clubs and discotheques several lifestyle habits are freely displayed including binge drinking of alcohol beverages, exposure to first and second hand tobacco smoking, indoor air pollutants, eat junk foods and meats dense with high cholesterol and low in fruits and vegetables, exposed to less sleep and inadequate water intake and noisy and promiscuous environments and other risk factors that predispose one to several metabolic aberrations and subsequent development of cardiovascular diseases (CVD) and other communicable and non-communicable diseases burdens alike [6-8]. These practices during their active service years take a cumulative toll on them after wards during the later years, when they are less active, retired and more exposed to physical inactivity as a risk factor for NCDs. Globally, more than 70% of cardiovascular mortality, 40% of chronic respiratory diseases, 34% of cancer mortality and about 50% of all chronic diseases deaths are attributed to a small number of known life style related modifiable risk factors [8,9]. With the increasing trend of night clubbing among young adults and the toll of NCDs in the general population, there is need to evaluate the prevalence of lifestyle risk factors and clinical as well as biochemical correlates of CVD among night clubbers in order to guide policies and preventive strategies to reduce the global increase prevalence of CVD in the general population.

### Subjects and Methods

#### Study Design and Study Population

This cross-sectional study was carried out between August 2016 and December 2018. Two hundred and fifty-five (255) subjects (130-night clubbers and 125 non-clubbers) aged 18 to 55 years actually participated in the survey of the 285 initially invited after applying the inclusion/exclusion criteria. Exclusion criteria were missing data, age outside the study age (>18 and < 55years), inappropriate completion of questionnaire, decline participation and poor mental state.

The invitation to participate in this study was distributed to target participants at several contact points including the primary health center where the study was conducted. Other points of recruitment of participants include fast food and drinking bars, club houses and other recreational centers. Written informed consent was obtained from participants and the study protocol was approved by the management of the health center and local Ethics Committee and the study protocols followed Helsinki Declaration [10] guidelines governing the conduct of human research. Participation was voluntary and participants were free to withdraw at any stage of the study. Those who qualified to participate were divided into 2-subgroups: night clubbers (those who regularly (2-4 times a week) attend night clubs (an entertainment house that is open from the evening until early morning) or involve in evening/night outdoor recreational

activities such as drinking, smoking, parties and eating junk foods/meats) at discotheques, bars and taverns, otherwise they were classified as non-clubbers.

#### Assessment Measures

Two survey instruments were used to assess the prevalence of CVD risk factors among participants. (1) socio-demographic/lifestyle assessment questionnaire adapted from previous studies [11] was used to assess the prevalence of indirect CVD risk factors (socio-demographic and lifestyle attributes), while the assessment of clinical and biochemical variables provided information on the direct CVD risk factors. The main outcome of this study was incident CVD risk factors defined as any socio-demographic/lifestyle, clinical or biochemical factor known to increase the odds for CVD among study participants, while CVD was defined as including several adverse cardiovascular endpoints such as coronary heart disease (CHD), ischemic heart disease (IHD), myocardial infarction, atrial fibrillation and cardiovascular death [12]. The questionnaire consisted of 2 sections. Section 1 contained information on the socio-demographic characteristics of participants including age, sex, life style habits (physical activity status, dietary habits, sleep duration, smoking habits, alcohol intake, occupation, educational level, and frequency of clubbing). Also, past medical history of CVD, or other related diseases was enquired about. These included type 2 diabetes mellitus (T2DM), hypertension, ischemic heart disease (IHD), myocardial infarction (MI), kidney disease, mental disorders, stroke and hematological disorders. All lifestyle variables and behavioral factors were measured on binary scale (i.e., yes/no).

Physical activity status of participants was determined based on the 2010 US healthy people physical activity guidelines standard which recommends 150 minutes of moderate to severe intensity of aerobic exercise per week in bouts of 10 minutes or more for physically active adults aged between 18 and 64 years.

Alcohol/beer consumption was established by asking participants whether they currently drink or not and those who drank up to 24hrs prior to the commencement of the study were regarded as current drinkers, those who had stopped drinking 6 months prior to study period were regarded as ex-drinkers while those who never drink was classified as non-drinkers. Cigarette smoking habit was enquired about and participants were stratified into three groups namely current smokers (those who currently smoke) ex-smokers (those who had stopped smoking 6months prior to the study period) and never smokers (those who never smoke). Sleep duration was determined by asking the participants to state the number of hours they normally sleep in a night and to know whether they usually wake up in the middle of the night or not. Based on the duration of sleep per night, they were categorized into three groups namely; short sleep duration ≤ 6hrs/night, normal sleep duration 7-8hrs/night and long sleep duration >8hrs/night.

A 24hr dietary recall questionnaire was used to obtain information on the actual food intake of participants. Questions

were asked on the frequency and quantity of consumption of specific food types including carbohydrates, fats, protein, fruits and vegetables. The extent to which a particular food type was consumed was studied on a three-point continuum as always, sometimes and never. Good dietary habit was defined as moderate consumption of balanced diet 2-3 times a day with a substantial amount of fruits, vegetables, fish and whole grains, while poor dietary habit was defined as excessive consumption of unbalanced diets with little or no fruits and vegetables, frequent consumption of meal outside home, consumption of readymade foods, late night eating and high intake of animal based foods and low intake of water.

**Measurements**

Before the commencement of the study, participants were informed about the aims and procedures for the study. Assessment of anthropometric indices including height and weight were performed. Height was measured to the nearest 0.1cm using height meter and weight was measured to the nearest 0.1kg using a weighing scale (Seca Model, Germany) after observing all necessary precautions to maintain accuracy as described previously [13]. Body mass index (BMI) was calculated as the ratio of weight in kg divided by the square of body height in meters (Weight kg/Height<sup>2</sup> m<sup>2</sup>). BMI < 18.5kg/m<sup>2</sup> was considered as underweight, BMI= 18.5-24.9kg/m<sup>2</sup> normal weight and BMI≥ 30kg/m<sup>2</sup> was regarded as obese [14].

Waist circumference (WC) was measured at the iliac crest to the nearest mm with a soft non-elastic measurement tape. WC >94cm and >80cm for men and women respectively were considered abnormal (indicative of obesity) [15]. The blood pressure (BP) of each participant was measured with a sphygmomanometer (Acosin 300 Dekamet Ltd. England) with appropriate curve size and after sitting quietly for 15 minutes. The measurement was taken twice and the average of the two was calculated and used. Normotension was defined as systolic BP < 140mmHg and diastolic BP <90mmHg. Greater than these values, BP was considered to be in hypertensive range [16].

**Biochemical Analyses**

Venous blood samples were collected before breakfast for measurement of fasting blood sugar (FBS), serum uric acid (SUA) level and lipid sub-fractions (high density lipoprotein-cholesterol (HDL-C), low density lipoprotein-cholesterol (LDL-C), triglyceride-cholesterol (TG-C), (total cholesterol (T-chol), (very low density lipoprotein-cholesterol (VLDL-C)) and insulin level. Fasting serum insulin level was measured by ELISA immunoassay (Sigma chemical Co. USA) insulin resistance was estimated by homeostasis model assessment [homeostasis model assessment-insulin resistance (HOMA-IR)] score calculated as FBS (mmol/L) x fasting serum insulin (mμ/L) HOMA-IR [FBG (mmol/L) x insulin (μU/mol)]/22.5

When FBG is <140mg/dl

HOMA-IR ≥ 1.8 was diagnostic of IR [17].

FBS and lipid sub-fractions were measured using a multi-channel Automated System: Lipid pro TM Model KM-0014; info Pia Co. Ltd. South Korea. Serum uric acid was measured by enzyme-linked immunosorbent (ELISA) method using commercially available kit sigma chemical Co. USA. Two FBS measurements >7.1mmol/L was regarded as abnormal (diabetes mellitus) LDL-C was calculated using the modified Friedewald formula. Reference ranges for the lipid sub-fractions used in this study were as follows;

TG-C<150mg/dl, HDL-C =40-59mg/dl, T-chol-C<200mg/dl and LDL-C<100mg/dl.

**Statistical Analysis**

Categorical variables were analyzed using frequencies and percentages while means ± standard deviations were computed for quantitative variables. Univariate associations between categorical variables were examined using chi-square while differences in quantitative variables between night clubbers and non-clubbers were tested using independent t-test. P<0.05 was considered statistically significant. All data analyses were carried out using the Statistical Package for Social Sciences (SPSS version 22.0)

**Results**

The socio-demographic variables that showed significant (P<0.05) differences between night clubbers and non-clubbers were age (P=0.0002), gender (P=0.004) and educational attainment (P=0.036). Other socio-demographic variables were not significantly different between clubbers and non-clubbers (Table 1). Table 2 shows that the prevalence of alcohol intake (P<0.020), poor dietary habits (P<0.001), and short sleep duration (P<0.001) were significantly higher in clubbers than non-clubbers.

**Table 1:** Socio-demographic Characteristics of Study Participants.

Variable	Night clubbers	Non-night clubbers	P-value
	(n=130)	(n=125)	
Age (mean)	35.02 ± 3.03	33.60 ± 3.07	0.0002
<b>Gender</b>			
Male	101 (77.7)	75 (60.0)	0.004**
Female	29 (22.3)	50 (40.0)	
<b>Marital Status</b>			
Married	59 (45.4)	46 (36.8)	0.104
Single	65 (50.0)	77 (61.6)	
Divorced	06 (4.6)	02 (1.6)	
<b>Educational Status</b>			
Secondary	48 (36.9)	29 (23.2)	0.036**
OND/NCE	57 (43.8)	60 (48.0)	
HND/BSC	25 (19.2)	36 (28.8)	
<b>Employment Status</b>			

Employed	82 (63.1)	89 (65.9)	0.722
Unemployed	48 (36.9)	46 (34.1)	
<b>Ethnicity</b>			
Ibibio	85 (65.4)	79 (63.2)	0.329
Anang	43 (33.1)	46 (36.8)	
Others	02 (1.5)	0 (0.0)	

OND = Ordinary National Diploma; NCE = National Certificate of Education; HND = Higher National Diploma  
 BSc = Bachelor of Science ; \*\* = Significant at 5% (P < 0.05)

**Table 2:** Lifestyle-related Risk Factors for CVD among Study Participants.

Variables	Night Clubbers (n=130)	Non-clubbers (n=125)	P-value
<b>Smoking Habits</b>			
Smokers (current)	42 (32.3)	27 (21.6)	0.075
Non-smokers	88 (67.7)	98 (78.4)	
<b>Alcohol Drinking</b>			
Drinkers (current)	75 (57.7)	38 (30.4)	<0.001*
Non-drinkers	55 (42.3)	87 (69.6)	
<b>Physical Activity</b>			
Active	62 (47.7)	78 (62.4)	0.026**
Inactive	68 (52.3)	47 (37.6)	
<b>Dietary Habits</b>			
Good	32 (24.6)	71 (56.8)	<0.001*
Poor	98 (75.4)	54 (43.2)	
<b>Sleep Duration</b>			
≥8 hrs	19 (14.6)	22 (17.6)	<0.001*
5-8hrs	33 (25.4)	73 (58.4)	
<5hrs	78 (60.0)	30 (24.0)	

\*\* = Significant at 5% (P < 0.05); \* = Significant at 1% (P < 0.01)

**Table 3:** Clinical Risk Factors for CVD among Study Participants.

Variables	Clubbers (130)	Non-clubbers (125)	P-value
BMI	28.45 ± 8.23	26.0 ± 7.44	0.0134*
WC	92.23 ± 10.44	84.41 ± 9.36	<0.0001*
SBP	129.52 ± 14.56	120.34 ± 15.09	<0.0001*
DBP	88.21 ± 5.29	90.34 ± 7.44	<0.0092*
PP	39.18 ± 5.86	32.13 ± 6.9	< 0.0001*
MAP	103.42 ± 7.95	98.92 ± 6.28	< 0.0001*

\*= Significant at 1% (P < 0.01); BMI= Body Mass Index (Kg/h<sup>2</sup> m<sup>2</sup>); WC = Waist Circumference (cm); SBP= Systolic Blood Pressure (mmHg); DBP= Diastolic Blood Pressure (mmHg); PP= Pulse Pressure (mmHg); MAP= Mean Arterial Pressure (mmHg).

Anthropometric indices including BMI and WC showed significant (P<0.0134 and P<0.001 respectively) higher values in night clubbers than non-clubbers. Likewise, the mean values

of blood pressure indices were significantly higher in clubbers than non-clubbers (SBP (P<0.001), DBP (P<0.0092), PP (P<0.0001), and MAP (P<0.0001) (Table 3). Table 4 shows that serum (UA<0.0001), T-chol (P<0.0001), TG-C (P<0.0001), LDL-C (P<0.0001), insulin level (P<0.046), HOMA-IR (P<0.0160) and atherogenic index of plasma( AIP) (P<0.0001) were significantly higher in night clubbers than non-clubbers.

**Table 4:** Biochemical Risk Factors for CVD among Study Participants.

Variables	Clubbers (n=130)	Non-clubbers (n=125)	P-value
Uric acid	6.82 ± 2.30	4.22 ± 1.99	<0.0001*
HDL-C (mg/dL)	49.02 ± 9.88	52.01 ± 9.67	0.0153*
T-chol (mg/dL)	208.3 ± 15.99	165.4 ± 21.56	<0.0001*
TG-C (mg/dL)	166.5 ± 19.22	144.2 ± 21.86	<0.0001*
LDL-C (mg/dL)	125.5 ± 20.32	115.3 ± 27.88	<0.0001*
Insulin level (µU/mL)	8.1 ± 2.10	7.6 ± 1.87	0.046*
HOMA-IR	1.96 ± 0.99	1.7 ± 0.690	0.0160*
USG	1.028 ± 0.73	1.017 ± 0.68	0.9011
AIP log (TG/HDL)	0.53 ± 0.04	0.44 ± 0.3	< 0.0001

\*= Significant at 1% (P < 0.01); HDL-C = High Density Lipoprotein-cholesterol; T-chol =Total Cholesterol

TG-C = Triglyceride-cholesterol; HOMA-IR = Homeostasis Model Assessment Insulin Resistant ; USG = Urinary Specific Gravity; AIP = Atherogenic Index of Plasma

### Discussion

Findings of the present study showed that, night clubbing habit is a significant risk factor for higher prevalence of CVD risk factors such as poor life-style related behaviors (physical inactivity, poor dietary habits, short sleep duration and alcohol consumption), abnormal clinical indices (elevated adiposity and raised blood pressure) and adverse biochemical endpoints (high serum UA, T-chol, TG-C, LDL-C, insulin level, HOMA-IR and AIP). Convincing evidences show that these factors can act in isolation or in synergy to cause adverse cardiovascular endpoints. For instance, preponderance of research evidence shows inverse association between physical activity level and CVD risk in all age categories regardless of the level of activity [18]. Physical inactive populations are more prone to developing CVD. According to Sesso et al., [19] physical inactivity is associated with a 19% increased risk for CHD. Rojas and his colleagues [20], in their clinical based study observed that physical inactivity was the most prevalent risk factor among participants with CVD. Their findings were similar to many other studies that yielded similar results which prompted the inclusion of adequate physical activity as an important element in CVD preventive policies across all ages and populations [21-23].

One study showed that even lower intensity physical activity is associated with significant reduction in CVD risk (51% and 70%) in the older individuals [24]. According to the Physical



Activity Guidelines Advisory Committee Report, men and women with high level of physical activity could have a 30% to 35% CVD risk reduction [25]. The patho-mechanistic pathways underlying the cardio-protective effect of physical activity is explained largely in part by its anti-inflammatory effect [26], and indirectly through reduction in the risk of developing medical conditions that are known risk factors for CVD such as hypertension, dyslipidemia, obesity, type 2 diabetes mellitus (T2DM), renal disease, oxidative stress and high serum homocysteine level [27]. In many studies, improvements in markers of inflammation and metabolic aberrations following moderate to severe exercise have been reported, including improvement in serum levels of C-reactive protein, amyloid A, interleukin 6 and inter cellular adhesion molecule 1 [26-28]. Also, improvement in blood sugar level and lipid profile, as well as BMI and BP indices have been reported following moderate to severe exercise. This may partly explain the significant differences in some clinical and biochemical risk factors for CVD between night clubbers and non-clubbers in the present study.

A significant higher prevalence of poor dietary habits was found among night clubbers than non-clubbers. For instance, higher prevalence of meals outside home, consumption of readymade foods, higher intake of alcohol beverages, animal-based foods, late night eating and low intake of fruits, vegetables and water were more prevalent among clubbers than non-clubbers. These dietary habits are similar to those previously shown to increase the risk of CVD and determinants (obesity, T2DM, hypertension and dyslipidemia) [29,30], but contradict the healthy/prudent eating recommendations of Dietary Approaches to Stop Hypertension (DASH) diet [31] and the Mediterranean diet [32-35] which consist of diet rich in fruits, vegetables, fish and whole grains [36,37]. Several epidemiological and clinical based studies indicate inverse association between healthy diet and CVD risk and determinant likely due to the effect of varied bio-constituents and their antioxidant, anti-inflammatory and immune system modulating activities. Healthy diet protects against overweight/obesity, high BP, T2DM, IR and dyslipidemia and vice versa [38].

More night clubbers reported shorter sleep duration at night than non-clubbers which may in part, be ascribed to the disruption in timing of circadian rhythms, consequent upon irregular sleeping time. Short sleep duration has not only been implicated in poor social, emotional, lifestyle, behavioral, and personal choices but has also been linked to higher incidence of CVD and determinants. A link between insufficient sleep and increased risk of MI was reported by Liu et al., [39]. Bertisch et al. [40] reported 29% higher risk of CVD development in participants who had poor sleep compared to a reference group. Sleeping 5 or fewer hrs per night increased the risk of CHD by 39%, while 6hrs of sleep per night increased the CHD risk by 18% compared with normal sleep duration (8hrs per night) [41]. Indeed, adequate sleep promotes optimum cardiovascular and metabolic homeostasis [42]. The

mechanism underlying a potential association of short sleep duration with incident CVD include induction of inflammation [43] and development of established risk factors for CVD such as T2DM [44], high BP [45], dyslipidemia, Obesity [46], chronic kidney disease [47] and hyperuricemia [48].

The prevalence of alcohol intake was also significantly higher among night-clubbers than no clubbers. This suggests a higher likelihood of alcohol-induced CVD in the former than the latter, given the fact that alcohol abuse is a powerful risk factor for CVD [49]. Although the cardio-protective effects of mild to moderate alcohol consumption has been extensively reported [50], evidence is emerging that some disorders of the cardiovascular system could occur even at the lowest level of alcohol intake and increasing in a dose-dependent manner to the highest levels of alcohol intake [51]. This is consistent with a study that showed a dose-response effect of alcohol consumption, and the diverse relationships (direct, inverse, U-shaped and J-shaped) of alcohol intake with cardiovascular health [52]. However, it should be noted that the detrimental effect of low alcohol consumption could be due to the co-occurrence of multiple CVD risk factors, as observed among night clubbers in the present study.

Similarly, night clubbers had higher prevalence of smoking than non-clubbers, suggesting a higher risk of smoking-induced CVD in the former than the latter as reported previously. Evidence has been garnered that smoking increases the risk of CVD such as CHD, stroke, sudden death, peripheral arterial disorder and aortic aneurysm. According to Messner et al. [53], smoking is one of the most powerful risk factors for CVD worldwide. Doyle et al. [54] had previously demonstrated that smokers had higher risk of MI. A Japanese study demonstrated a higher risk of CHD among smokers [55]. Many other studies agreed that smoking has a negative effect on cardiovascular endpoints.

Consistent with findings of the present study, a recent study conducted by Lasebikan et al. [3] in an urban city in Nigeria reported higher prevalence of smoking among night clubbers than non-clubbers [3]. Induction of inflammation and sub-clinical atherosclerosis are the postulated patho-physiological mechanisms of smoking-induced CVD. This is evident by elevated serum markers of inflammation and atherosclerosis including elevated serum levels of highly sensitive C-reactive protein, fibrinogen, and coronary artery calcium as well as reduced serum level of HDL-C [56] in smokers. Some of these lifestyle behaviors either alone or in combination with other risk factors are known to initiate or worsen extant CVD. For instance, alteration of circadian rhythms due to altered sleeping pattern can cause derangements in several psychophysical and metabolic functioning of the body [57] and CVD risk factors including aging, diet-induced obesity, T2DM, hypertension and dyslipidemia and therefore augments the development of CVD. Lopez et al. & Merikanto et al. [58,59] reported association between sleep disorders and raised BP, smoking and re-infarction.

Night clubbers had significant higher mean SUA level than non-clubbers. This could probably be due to poor lifestyle habits known to be etiologically implicated in hyperuricemia, including poor dietary habits, binge drinking of alcoholic beverages, exposure to first and second hand tobacco smoking, exposure to indoor air pollutants, consumption of junk foods/meats dense with high cholesterol but low in fruits and vegetables and poor sleeping habits which were more common among night-clubbers than non-clubbers [60]. The observed higher SUA level and other poor lifestyle habits among night clubbers could partly provide explanation for the observed clinical and biochemical aberrations found among night clubbers as strong correlations have been established between SUA and these disorders including dyslipidemia [61], hypertension [62-64], IR, [65,66] abnormal adiposity indices [67] and atherogenesis [68]. Besides the indirect relationship, other lifestyle habits have been shown to directly impact several metabolic pathways leading to the observed clinical and biochemical aberrations. For instance, binge drinking is associated with IR, dyslipidemia, abnormal adiposity and abnormal BP indices. Likewise, short sleep duration, poor dietary habits, physical inactivity and poor hydration status have been found to directly influence metabolic processes and leading to abnormal clinical and biochemical endpoints as observed among night clubbers in the present study.

### Conclusion

CVD risk factors are more prevalent among night clubbers than non-clubbers. Night club, discotheque, tavern, drinking point and bar patrons should be included among target populations in the development of interventions necessary to reduce the high prevalence of CVD risk factors in the general populations because current risk factors become future disease and public health burden and knowledge of risk factors can be used to shift their population distribution.

### Competing Interest

None declared.

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