Effect of alcohol consumption on testosterone, luteinizing hormone and follicle stimulating hormone levels in males residing in Nnewi Metropolis, Anambra state, Nigeria

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Abstract
This study investigated the effect of alcohol consumption on some male reproductive hormone (Luteinizing Hormone (LH), Follicle Stimulating Hormone (FSH), and Testosterone (TT) levels in Nnewi Metropolis. Seventy two subjects comprising of 41 male (21 acute and 20 chronic) alcohol consumers and 31 controls aged between 18 and 50 years old were recruited for this study. Anthropometric data (height, weight and body mass index (BMI) as well as socio-demographic data of the subjects were obtained. Thereafter, 5mls of blood sample was collected from each subject and dispensed into plain container and used for laboratory analysis. Serum LH, FSH, and TT levels were estimated using Enzyme Linked Immunosorbent Assay (ELISA) method. The result revealed a significantly higher mean weight, BMI as well as serum LH levels respectively in alcohol consumers than in control ($p<0.05$), but the mean serum concentration of FSH and TT remained the same in both groups ($p>0.05$). More so, the mean weight, BMI, as well as serum LH and FSH levels were similar in both acute and chronic alcohol consumers respectively ($p>0.05$), While there was a significant decrease in the mean serum TT level in chronic alcohol consumers than in acute alcohol consumers ($p<0.05$). In conclusion, this study shows that moderate alcohol consumption did not adversely affect serum fertility hormone levels in males, while chronic consumption may induce weight gain and also affect fertility hormones, leading to hypogonadism.

Keywords: Alcohol consumption, Luteinizing Hormone (LH), Follicle Stimulating Hormone (FSH), Testosterone, Weight, Body mass index, Hypogonadism.

Introduction
Alcohol is as old as human history and its consumption in different socio-cultural milieus extends beyond the last ten thousand years (Smart, 2007). The term alcohol originally refers to the primary alcohol ethanol (ethyl alcohol), which is used as a drug and is the main alcohol present in alcoholic beverages (IUPAC, 2006), and due to its low lipid solubility, ethanol is primarily distributed to the water soluble parts of the body (Marshall *et al*., 2003). The oxidation of alcohol is primarily carried out by alcohol dehydrogenase which oxidizes ethanol to acetaldehyde, a product which is far more toxic than ethanol itself (Salaspuro *et al*., 2003). In recent decades, the pattern, quantity and reason for consumption of alcohol are changing rapidly, especially among youths (Chikere and Mayowa, 2011), with parents and siblings having enormous influence on the drinking behaviour of young people (Mares *et al*., 2011; Mares *et al*., 2012). A number of reasons have been attributed for the current use of alcohol among the young population ranging from the need to enhance sexual pleasure, to feeling high as well as feeling more sociable (Chikere and Mayowa, 2011). Alcohol intake has been implicated in male infertility in a number of studies (Frias *et al*., 2000; Muthusami and Chinnaswamy, 2005; Maneesh *et al*., 2006). According World Health Organization (WHO), 2.3 billion people are current drinkers globally and in 2016, the harmful use of alcohol resulted in 3 million deaths (5.3% of all deaths) worldwide and 132.6 million disability-adjusted life years (DALY), accounting for 5.1% of all DALYs in 2016 (WHO, 2018). Also, mortality resulting from alcohol consumption is higher than that caused by diseases such as tuberculosis, HIV/AIDS and diabetes. Among men in 2016, an estimated 2.3 million deaths and 106.5 million DALYs were attributable to the consumption of alcohol (WHO, 2018). Several authorities have also reported either no changes or decreases in the serum levels of LH, FSH, and Testosterone in alcoholics (Frias *et al*., 2000; Oremosu and Akang, 2014; Dosumu *et al*., 2010; Dosumu *et al*., 2014; Karitha *et al*., 2014; Rao *et al*., 2015). Therefore, this study investigated the effect of alcohol consumption on male testosterone, luteinizing hormone and follicle stimulating hormone levels in Nnewi Metropolis, Nigeria.

Materials and Methods
Subjects and Study Design
This is a case-control study designed to assess the effect of alcohol consumption on male testosterone, luteinizing hormone and follicle stimulating hormone levels in Nnewi Metropolis, Anambra State, Nigeria. A total of seventy two (72) apparently healthy male subjects comprising of 41 male alcohol consumers and 31 non-alcohol consumers used as test and control subjects respectively were randomly recruited for the study. All subjects were interviewed using

a questionnaire with regard to alcohol intake and drinking habits. Personal interviews were conducted with all alcoholic and control subjects to obtain relevant clinical data: age, sex, marital status, diet, history of alcohol consumption, the amount and kind of alcohol consumed, infertility status, past medical illness and treatment, and history of smoking. Also, the respective height and weight of all the subjects were obtained using a meter rule and weighing scale respectively and used for the calculation of their respective body mass index (BMI). All subjects signed an informed consent to participate in this study. All were in the age groups between 18-50 years.

Thereafter, 5mls of venous blood sample was collected from each subject and dispensed into plain container. The blood samples were centrifuged to obtain the clear supernatant (serum) and the supernatant were stored at -18°C until it was used for laboratory analysis. Serum LH, FSH, and TT levels were estimated using Enzyme Linked Immunosorbent Assay (ELISA) method.

Inclusion Criteria

The study included two subject groups, controls and alcoholics aged between 18 and 50 years. Subjects in the control group were volunteers who were free from any disease and who had never consumed alcoholic drinks and who had never smoked. Subjects in the alcoholic group were nonsmokers who had consumed a minimum of 180 mL of alcohol (beer, brandy and whisky, 5%–50% alcohol content) per day for a minimum of 3-5 days per week in the past year and had been drinking for a period of 2-3 years previously.

Exclusion Criteria

Known smokers, subjects with known disorders such as; liver diseases, diabetes mellitus, hypertension, kidney diseases, subjects with history of other drug abuse, as well as any other physical illness or causes of infertility, were excluded from the study.

Ethical Consideration

The ethical approval for this study was sought and obtained from the Ethics Committee of College of Health Sciences and Technology, Nnamdi Azikiwe University, Nnewi, Anambra State, Nigeria.

Estimation of Serum Testosterone (TT) Level

Serum TT level was estimated using enzyme- linked immunosorbent assay method as described by Singer, (1992).

Estimation of Serum Follicle-Stimulating Hormone (FSH) Level

Serum FSH level was estimated using enzyme- linked immunosorbent assay method as described by Seth *et al.*, (1989).

3.5 Estimation of serum luteinizing hormone (LH)

Serum LH level was estimated using enzyme- linked immunosorbent assay method according to Kosasa, (1981).

Statistical Analysis

The data obtained was statistically analyzed using Statistical Package for Social Sciences (SPSS) version 20.0. Students't-test and pearson correlation were used to compare means. The results were expressed as mean ±SD and confidence limits was chosen at 95% ($P < 0.05$). $P < 0.05$ was considered statistically significant while $P > 0.05$ was insignificant.

Result

The mean age of alcohol consumers was significantly higher compared to control subjects (28.17± 6.75 Vs 23.77 ± 4.12; p=0.02). There was no significant difference between the mean height of alcohol consumers and the control subjects (p>0.05). However, both mean weight and BMI were significantly higher in alcohol consumers compared to the control group (p=0.046; 0.031) respectively, See table 1.

Table 1: mean ±SD Anthropometric parameters in the subjects studied

Variables	Age	Height	Weight	BMI
Test (n=41)	28.17 ±6.75	1.71 ±0.08	73.07 ± 13.25	25.46 ±4.94
Control (n=31)	23.77 ±4.12	1.72 ± 0.07	67.42±9.22	23.20 ±3.92
t-value	8.625	0.001	7.921	7.406
p-value	*0.002	0.707	*0.046	*0.031

*Statistically significant at p<0.05

The mean serum LH level was significantly raised in alcohol consumers compared with control subjects (20.46± 14.67 Vs 11.93±3.58; p=0.014). However, the mean serum levels of FSH and Testosterone did not differ significantly between the alcohol consumers and control subjects respectively (p>0.05). See Table 2.

Table 2: Mean ±SD serum levels of LH, FSH and Testosterone in male alcohol consumers

Variables	LH	FSH	Testosterone
Alcohol consumers (n=41)	20.46±14.6	8.30±5.66	4.25±0.84
Control (n=31)	11.93 ±3.58	8.13 ±7.58	4.54 ±0.82
t-value	0.601	1.607	0.112
p-value	*0.014	0.914	0.144

*Statistically significant at p<0.05

The mean age of the subjects was significantly lower in acute alcohol consumers (24.81± 3.19) than in chronic alcohol consumers (31.70 ± 7.73), (p=0.001). The mean weight of subjects was significantly decreased in acute alcohol consumers (68.76 ± 10.5) than in chronic alcohol

consumers (76.60 ± 16.74), (p=0.077). However, there were no significant differences observed in the mean height, body mass index, and serum levels of LH and FSH between acute alcohol consumers and chronic alcohol consumers respectively (p>0.05). However, mean serum level of Testosterone was significantly decreased in the chronic alcohol consumers than in the acute alcohol consumers (4.01 ± 0.98 Vs 4.63 ± 0.44; p=0.012). See table 3.

Table 3: Mean ±SD levels Age, Height, weight, BMI, LH, FSH and Testosterone between acute and chronic alcohol consumers

Variables	Age	Height	Weight	BMI	LH	FSH	TT
AAC (n=21)	24.81 ±3.19	1.70 ±0.07	68.76 ±10.25	24.55 ± 5.35	17.04 ±12.61	7.69 ± 4.42	4.63 ±0.44
CAC (n=20)	31.70 ±7.73	1.72 ±0.09	76.60 ±16.74	26.42± 4.41	24.05 ±16.10	8.94 ± 6.80	4.04 ± 0.98
t-value	16.120	0.007	4.560	0.060	2.576	3.241	11.248
p-value	*0.001	0.659	*0.077	0.231	0.128	0.487	*0.012

*Statistically significant at p<0.05;
AAC=Acute alcohol consumers, CAC=Chronic alcohol consumers, TT=Testosterone.

In the study, there was a positive correlation between BMI_2 versus HT_1 (r=0.040), $Test_2$ versus BMI_1 (r=0.021). But there was a negative correlation between LH_2 versus Age (r=-0.032), LH_2 versus $Test_1$(r=-0.043), FSH_2 versus Age (r=-0.017), FSH_2 versus FSH_1 (r=-0.040), $Test_2$ versus WT (r=-0.007), $Test_2$ versus LH_1 (r=-0.008). See Table 4.

Table 4: levels of association between parameters studied in test(1) and control(2)

Parameters	Correlation pearson r	f-value
BMI_2 v HT_1	0.040	0.831
LH_2 v Age_1	-0.032	0.862
LH_2 v LH_1	-0.043	0.819
FSH_2 v Age	0.017	0.927
FSH_2 v FSH	-0.040	0.831
$Test_2$ v WT	-0.007	0.971
$Test_2$ v BMI_1	0.021	0.911
$Test_2$ v LH_1	-0.008	0.967

*Statistically significant at p<0.05

Discussion

In this study, both the mean age, weight as well as body mass index were significantly higher in alcohol consumers than in control group. Following WHO classification of BMI levels (WHO, 1995): underweight ≤ 18.5kg/m^2, normal weight= 18.5-24.9kg/m^2, obesity class I =30.0-34.9kg/m^2, obesity class II= 35.0-39.9kg/m^2, and obesity class III=>40.0kg/m^2; Our present study revealed that alcohol consumers were overweight in contrast with the control group who had normal body mass index. A number of factors have been implicated in the development of adult obesity, one of which is the consumption of alcohol (WHO, 2015). Alcohol is one of the second most energy dense macronutrient and has an appetite enhancing effect, which may lead to an increase in energy intake, inducing an increase in body mass index (Colditz *et al*., 1991; Westerterp-Plantenga and Verwegen, 1999). More so, it is a known fact that alcohol suppresses the oxidation of fat, hence favouring fat storage (Prentice, 1995). Our finding is in consonance with some other previous studies which had earlier reported weight gain or positive relationship between alcohol and BMI (Williamson *et al*., 1987; Thomson *et al*., 1988). BMI appears to be one of the most powerful predictors of biochemical hypogonadism (Ventimiglia *et al*., 2016a).

The present study revealed a significantly higher mean serum concentration of LH (Miu/ml) in alcohol consumer compared with control subjects, whereas the mean serum FSH concentration remained similar in both groups. Also the mean serum Testosterone (ng/ml) level did not differ significantly between the values observed in alcohol consumers and control subjects. This increase in the mean serum concentration of LH in the alcoholics may be as a result of the inhibitory effect of alcohol on the biosynthesis of testosterone which results in a compensatory increase in the LH secretion, thereby leading to the temporary maintenance of normal serum concentration of testosterone. This is called compensated hypogonadism and it may be a biomarker for testosterone decline within the reference range, indicating a readjustment of the HPT feedback set point in alcoholics to compensate for deficiencies in testicular function and/or defective TT feedback at the hypothalamic-pituitary level. This condition may therefore, be a forerunner of overt primary hypogonadism, being characterized by elevated LH. Men with increased comorbidity and/or other as yet undefined factors may eventually progress from compensated to overt primary hypogonadism (Tajar *et al*., 2010). This increase in the mean serum LH concentration in the present study is in consonance with the findings of some other previous similar studies (Muthusam and Chinnaswamy, 2005; Schilep *et al*., 2015). However, some authors had also reported either no changes in the serum level of LH or decreased level of serum LH in alcoholics in contrast to our findings (Frias *et al*., 2000; Oremosu and Akang, 2014; Dosumu *et al*., 2010; Dosumu *et al*., 2014; Karitha *et al*., 2014; Rao *et al*., 2015).

Furthermore, the mean serum concentration of FSH did not differ significantly in alcohol consumers compared to control subjects. However, it is known that FSH acts on sertoli cells to stimulate gametogenesis and the synthesis

and release of inhibin (Burtis *et al*., 2008). Our finding is in concert with the report of some similar research (Frias *et al*., 2000; Frias *et al*., 2002; Maneesh *et al*., 2006; Dosumu *et al*., 2010; Dosumu *et al*., 2014), whereas, some other researchers earlier reported a differing opinion (Maneesh *et al*., 2006; Kavitha *et al*., 2014; Muthusami and Chinnaswany, 2015).

More so, the mean serum concentration of testosterone was found to be similar in both alcohol consumers and control subjects. This is in contrast to the reports of other previous studies which reported either increased or decreased levels of testosterone in alcoholics (Kavitha *et al*., 2014; Dosumu *et al*., 2014; Grover *et al*., 2014; Oremosu and Akang, 2014; Condorelli *et al*., 2015). This normal serum concentration of testosterone despite a prevailing increased secretion of LH and unaffected FSH levels may suggest that the hypothalamus which is responsible for the luteinizing hormone releasing hormone (LHRH) do not function to the feedback when testosterone level remained normal; instead it was hypersecretory. Although, TT levels in this group remained normal, it may be insufficient to maintain previous levels of physical functions (Zitzmann *et al*., 2006). Also, the inability of the pituitary gland to respond appropriately to a normal testosterone level may suggest that the alcohol has a central effect on the interaction between the nervous system and the endocrine system. Furthermore, the findings could also be attributable to genetic factors as well as differing environmental factors which affect the studied population.

Interestingly, the mean weight, BMI as well as serum concentration of LH and FSH were similar in acute alcohol compared with chronic alcohol consumers whereas, the mean serum TT level was significantly higher in acute alcohol consumers than in chronic alcohol consumers (4.63 ± 0.44 Vs 4.01±0.98; p=0.012). This implies that with increasing quantity of alcohol consumed, there will be a corresponding increase in its deleterious effect on the TT levels.

Conclusion

The present study revealed a significantly higher mean weight and body mass index in alcohol consumers than in control subjects. Also, there was a significant increase in the mean serum concentration of LH with no significant changes in FSH and testosterone levels in alcohol consumer compared with control subjects. Further, the mean weight was significantly increased in chronic alcohol consumers than in acute consumers, the BMI as well as serum concentration of LH and FSH were similar, while mean serum testosterone level was significantly higher in acute alcohol consumers than in chronic alcohol consumers. This may suggest that alcohol consumption induces weight gain and affects sex hormones, leading to hypogonadism.

Conflict of Interest: None.

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