International Journal of Sciences: Basic and Applied Research (IJSBAR)

ISSN 2307-4531
(Print & Online)

http://gssrr.org/index.php?journal=JournalOfBasicAndApplied

Serum Lipid Profile of the Adult Habitual Consumers of Two Traditional Alcoholic Drinks Made in Benin

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Abstract

Alcohol consumption modifies many biological parameters. This study aimed to describe the profile of the serum lipids of adult habitual consumers of *Tchoukoutou and Sodabi*, two traditional alcoholic drinks made in Benin. We carried out a descriptive, cross-sectional and analytic study from May 1st, 2013 to August 31, 2013. The target of the study was a population consisting of 60 habitual consumers of *Tchoukoutou* (mean age: 35.85 ± 10.47 years; 39 men), 60 habitual consumers of *Sodabi* (mean age: 39.65 ± 11.85 years; 57 men) and 60 non-consumers of alcoholic beverages (mean age: 29.71 ± 11.34 years; 43 men) selected in the town of Parakou (Republic of Benin). The serum lipid parameters were determined in each subject. The analysis of variance (ANOVA) refined by Student's t test enabled to compare averages between different groups of the study. The different correlations were determined through linear regression. The results shows that *Tchoukoutou and Sodabi* consumers had HDL cholesterolemia, triglyceridemia and VLDL cholesterolemia means significantly higher than the ones of non-consumers ($p<0.01$). On the other hand, LDL cholesterolemia mean was significantly higher in non-consumers than in *Tchoukoutou* consumers ($p=0.03$). The mean atherogenic index of *Tchoukoutou and Sodabi* consumers was significantly lower than in non consumers ($p<0.001$). The mean values of the lipid parameters of *Tchoukoutou and Sodabi* consumers did not significantly vary.

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Based on consumption duration, correlations were average as regards triglyceridemia and VLDL cholesterolemia (r = +0.293 and +0.294 respectively) in S*odabi* consumers and regarding LDL cholesterolemia (r = +0.201) in *Tchoukoutou* consumers. With daily average alcohol intake, as far LDL cholesterolemia is concerned, *Tchoukoutou* consumers showed triglyceridemia and VLDL cholesterolemia average correlations (r = + 0.207; + 0.206 and +0.201 respectively). As a conclusion; we can say that *Tchoukoutou and Sodabi* consumption modifies serum lipids' values to the effect that it reduces the development of atheromatosis**.**

Keywords: Benin; lipid profile; serum; *Tchoukoutou*; *Sodabi.*

1. Introduction

Alcohol consumption is one of the main factors that contribute to early mortality and the cause of avoidable morbidity in the world. It is an actual public health issue. In fact, in 2011, according to the World Health Organization, alcohol consumption causes 2.5 million deaths each year [1]. Moreover, 320, 000 youths aged from 15 to 29 years die every year due to causes related to alcohol; this represents 9% of the whole mortality in this age group [2].

Home-made production of traditional drinks represents an important part of alcoholic beverages production in many African countries. The advantage of these drinks is that they are sold at a low cost and they play an important social and cultural role [3, 4]. Consumption of home-made beer is far from being insignificant in Africa. Actually, in Kenya, according to a study conducted by Shaffer et *al.* [5], 54% of the rural population excessively consumes locally made drinks. In Benin, according to the data disclosed by the Office National de la Sécurité Alimentaire (The National Food security Office) in 2001, sorghum consumption per inhabitant in the North was 115 kg per year and drink making is one of the forms of consumption [6].

Tchoukoutou is a variety of home-made beer obtained by fermentation of red sorghum or millet most cultivated in Sub-Saharan Africa, and its ethanol content is 3.03% [7]. It is the local beer most drunk in North-Benin. *Sodabi*, an alcoholic drink extracted from palm wine after a natural fermentation, with 37.6% of ethanol [4], is one of the most consumed drinks, particularly in the South-Benin.

Many studies demonstrated that alcohol consumption raises serum ratios of HDL cholesterol [8-13], triglycerides and VLDL cholesterol [14]. On the other hand, alcohol consumption does not seem to influence LDL cholesterol ratio [8, 12]. Some authors had even noted a decline of LDL cholesterol in alcohol chronic excessive drinkers [11]. This rise of HDL cholesterol does not depend on the type of alcoholic drink [13] and does not vary in both sexes [8].

In Benin, except works on dyslipidemias and atherogenic risk in diabetic [15], the apoprotein reference values [16], serum lipid profile in the Fulani [17] and lipids in habitual tobacco consumers [18], to the best of our knowledge, there are no data available on the lipid biochemical parameters in alcoholic drink consumers. The aim of this study was to describe the serum lipid profile of habitual adult consumers of *Tchoukoutou and Sodabi*, two local alcoholic drinks made in Benin.

2. Materials and methods

2.1. Ethics

This study was approved by the institutional ethic committee.

2.2. Study setting

This study was conducted in the town of Parakou as far as subjects selection is concerned, and at the laboratory of the Regional Hospital of Borgou as for samples handling. Both places are located in the Republic of Benin.

2.3. Materials

The materials used were one Microlab 300 spectrophotometer (Vital Scientific, Dieren-The Netherlands). The reagents used were: cholesterol measurement kits, triglyceride measurement kits, precipitation reagent of low density lipoproteins and one test serum (LINEAR CHEMICALS SL, Barcelona, Spain).

2.4. Methods

2.4.1. Nature and period of study

This study was a cross-sectional, descriptive and analytic one carried out from May 1st, 2013 to August 31, 2013.

2.4.2. Target population

The target population of this study consisted of subjects of both sexes, selected after an informed consent read and approved among the population of Parakou who uses to consume one of the two local alcoholic drinks, i.e. *Tchoukoutou* and *Sodabi*. The subjects who are alcoholic drink non-consumers were selected in the same general population of Parakou.

Were included in this study adult subjects (with age higher than or equal to 18 years), habitual consumers of one of the local alcoholic drinks (*Tchoukoutou* or S*odabi*).

Any subject consuming one of these beverages at a minimal frequency of three (03) times a week was considered as a habitual consumer of one of the local alcoholic drinks. Any adult who does not consume any kind of alcoholic drink (local and industrial) was considered as a subject who does not consume alcohol.

Were not included in the study tobacco consumers, subjects consuming other local alcoholic drinks or industrial alcoholic drinks, obese subjects or subjects with previous diabetes mellitus**,** liver disease, arterial hypertension, gout, dyslipidemia revealed or being under a medication therapy of any kind, pregnant women and subjects who did not give their consent.

2.4.3. Samplings

The sampling was a random one. A census of the habitual consumers of locally made alcoholic drinks was done in the markets and most renowned points of sale of Parakou during the period of survey.

Regarding selection of *Tchoukoutou* consumer subjects, we put ourselves in the centre of the market and then, by turning a bottle on the ground, we chose a direction at random (the one indicated by the bottle's neck). We counted the number of female sellers in the direction chosen and we numbered them. Later on, we selected one female seller among those numbered. This selected seller is the first we visited. The next female sellers were gradually visited. All the sellers with whom there is one subject aged 18 years at least were selected. All the subjects aged 18 years and above who were with a same seller were selected. When the number of subjects was not reached after the visit to all the sellers identified, the process was resumed as from the centre of the market till the number of subjects desired is reached.

As regards the selection of S*odabi* consumer subjects, we localized some points of sale, particularly in sellers commonly known as « *dadjè* ». Then, we selected one seller among those counted. This seller selected is the first we visited. All the subjects aged 18 years and above who were with a same seller were selected. The next sellers were gradually visited till the number of subjects desired is reached.

The non-consumer subjects who gave their free consent were selected in the town of Parakou.

A total of 180 subjects were selected including 60 S*odabi* habitual consumers (mean age 39.65 ± 11.85 years ; 57 men ; 03 women), 60 *Tchoukoutou* habitual consumers (mean age 35.85 ± 10.47 years ; 39 men ; 21 women), and 60 do not consume alcoholic drinks (mean age 29.71 ± 11.34 years ; 43 men ; 17 women).

2.4.4. Study variables

The variables studied were: total cholesterol serum ratio, HDL cholesterol serum ratio, LDL cholesterol serum ratio, triglyceride serum ratio, atherogenic index, type of alcoholic drink consumed. We also studied the daily quantity of alcoholic drink consumed and the length of time of alcoholic drink consumption.

2.4.5. Data collection

It was done by means of a questionnaire administered to each selected subject. Each subject of the study was submitted to venous blood collection in order to measure lipid parameters.

2.4.6. Getting blood samples

Blood samples (4 mL) were collected through superficial venous puncture at the bend of elbow on dry tubes in each study subject on an empty stomach since 12 hours. The samples obtained in this way were centrifuged at 4000 turns/minute during 5 minutes, and then the serums were decanted to make measurements on the same day.

2.4.7. Measurement of lipid parameters

Total cholesterol was measured through enzymatic method in end point with cholesterol oxydase [19], HDL cholesterol by precipitation method with phosphotungstic acid in presence of magnesium ions [20], triglycerides by enzymatic method in end point with glycerol phosphate oxydase [19]. LDL cholesterol was determined by calculation using the formula of Friedewald et *al.* [21], VLDL cholesterol by formula: *VLDL = triglycerides (g/L)/5* and atherogenic index by dividing *total cholesterol / HDL cholesterol* [19].

2.5. Data analysis

Data were analyzed with Epi Info 3.5.1 and EXCEL softwares. Quantification of alcohol consumption was done in number of standard cups [22]. Results were expressed in ratios and means with their standard deviation. The analysis of variance (ANOVA) refined by Student's t-test allowed to compare means between the different groups of the target population. The linear regression used to determine correlations between lipid parameters means and duration of local alcoholic drink consumption on the one hand and daily quantity of alcohol intake on the other hand. The difference was significant for $p < 0.05$.

3. Results

There was no significant difference between the serum lipid parameters of S*odabi* and *Tchoukoutou* consumers (Table 1).

Table 1: Comparison of mean values ± standard deviation of lipid parameters (in g/L) between *Sodabi* and *Tchoukoutou* consumers.

	***Sodabi* consumers** (i)	***Tchoukoutou* consumers** (j)	**Difference of mean (i-j)**	**p**
TC	1.63±0.38	1.54±0.35	0.09	0.17
HDL- C	0.60±0.10	0.57±0.09	0.03	0.06
TG	1.22±1.07	1.16±0.87	0.06	0.73
LDL- C	0.80*±0.35	0.74*±0.33	0.06	0.32
VLDL-C	0.25±0.22	0.23±0.17	0.02	0.71
AI	2.69±0.60	2.74±0.61	- 0.05	0.64

p = significance ; n = number ; TC = total cholesterolemia ; HDL- C = HDL cholesterolemia ; TG = triglyceridemia ; LDL- C = LDL cholesterolemia ; VLDL -C = VLDL cholesterolemia; AI = atherogenic index; * = calculated based on n = 59 ; i = mean of S*odabi* consumers; j = mean of *Tchoukoutou*.consumers.

The mean values of HDL cholesterolemia, triglyceridemia and VLDL cholesterolemia were significantly higher in *Sodabi* consumers than in non-consumers ($p<0.05$). On the other hand, the athérogenic index was significantly higher than in non-consumers ($p = 0.001$) (Table 2).

Table 2 : Comparison of mean values ± standard deviation of lipid parameters (in g/L) between non-consumers and *Sodabi* consumers.

	Non consumers (k) **(n = 60)**	***Sodabi* Consumers** **(i)** **(n = 60)**	**Difference of mean** **(k-i)**	**p**
TC	1.54±0.57	1.63±0.38	- 0.09	0.31
HDL-C	0.48±0.14	0.60±0.10	- 0.12	0.00
TG	0.63±0.37	1.22±1.07	- 0.59	0.000
LDL- C	0.91±0.53	0.80*±0.35	0.11	0.19
VLDL-C	0.13±0.07	0.25±0.22	-0.12	0.000
AI	3.46±1.74	2.69±0.60	0.77	0.001

p = significance; n = number; TC = total cholesterolemia; HDL-C = HDL cholesterolemia ; TG = triglyceridemia ; LDL- C = LDL cholesterolemia ; VLDL-C = VLDL cholesterolemia; IA = atherogenic index; * = calculated based on n = 59 ; i = mean of S*odabi* consumers; k = mean of non-consumers.

Table 3: Comparison of mean values ± standard deviation of lipid parameters (in g/L) between *Tchoukoutou* non-consumers and consumers.

	Non-consumers (k) **(n = 60)**	***Tchoukoutou* Consumers** **(j)** **(n = 60)**	**Difference of mean** **(k-j)** **(n = 60)**	**p**
TC	1.54±0.57	1.54±0.35	0.00	0.98
HDL- C	0.48±0.14	0.57±0.09	- 0.09	0.000
TG	0.63±0.37	1.16±0.87	- 0.53	0.000
LDL-C	0.91±0.53	0.74*±0.33	0.17	0.03
VLDL-C	0.13±0.07	0.23±0.17	- 0.10	0.000
IA	3.46±1.74	2.74±0.61	0.72	0.003

p = significance ; n = number ; CT = Total cholesterolemia; HDL-C = HDL cholesterolemia ; TG = triglyceridemia ; LDL-C- = LDL cholesterolemia ; VLDL-C- = VLDL cholesterolemia; AI = Atherogenic Index; * = calculated based on n = 59 ; j = mean of *Tchoukoutou* consumers ; k = mean of non-consumers.

The mean values of HDL cholesterolemia, triglyceridemia and VLDL cholesterolemia were significantly higher in *Tchoukoutou* consumers than in non-consumers ($p<0.05$). On the other hand, the mean values of LDL cholesterolemia and atherogenic index were significantly higher in non-consumers ($p<0.05$) (Table 3).

Correlations were mean ($r > 0.20$) in *Sodabi* consumers as regards VLDL cholesterolemia and triglyceridemia and in *Tchoukoutou* consumers as for LDL cholesterolemia with consumption duration. This correlation was mean only in *Tchoukoutou* consumers as regards LDL cholesterolemia, VLDL cholesterolemia and triglyceridemia with daily mean quantity of alcohol intake (Table 4).

Table 4: Correlations between lipid parameters and daily mean quantity of alcohol intake and alcohol consumption duration.

		Correlation coefficient (r)	
		Daily mean quantity of alcohol intake	**Alcohol consumption duration**
TC	*Sodabi*	0.109	0.030
	Tchoukoutou	0.083	0.095
HDL-C	*Sodabi*	0.135	0.065
	Tchoukoutou	0.014	0.068
LDL- C	*Sodabi*	0.147	0.023
	Tchoukoutou	0.207	0.201
VLDL-C	*Sodabi*	0.010	0.294
	Tchoukoutou	0.201	0.154
TG	*Sodabi*	0.164	0.293
	Tchoukoutou	0.206	0.159
AI	*Sodabi*	0.164	0.094
	Tchoukoutou	0.068	0.157

T C= Total Cholesterolemia; HDL-C = HDL cholesterolemia; TG = triglyceridemia; LDL-C- = LDL cholesterolemia; VLDL-C- = VLDL cholesterolemia; AI = Atherogenic Index.

4. Discussion

In our study, the difference of mean total cholesterolemia between alcohol consumers and non-consumers was not statistically significant. The works carried out by Wannamethe and Shaper [23] got the same result.

Our study found that *Sodabi* and *Tchoukoutou* significantly increase HDL cholesterolemia. These results are similar to the ones of some former studies [23, 24, 25]. The mechanisms through which alcohol consumption raises HDL cholesterol are still unknown and several assumptions have been put forward. This rise in HDL cholesterol ratio by alcohol would be due to an increase of synthesis and secretion of apoprotein A-I by hepatic

cells [26]. Actually, HDL cholesterol is synthesized as from liver in immature form, consisting of A-I apolipoproteins surrounded by phospholipids. A-I apoprotein is a protein cofactor of lecithin cholesterol acyltransferase, responsible for the esterification of free cholesterol and its assimilation within HDL [27]. Another mechanism would imply the increase of lipoprotein lipase ratio; as lipolysis increases, free cholesterol flow and VLDL apoprotein to HDL would also increase [28]. *In vivo* activity of the esterified cholesterol transfer protein (CETP) provokes decline of HDL cholesterol [29]. In heavy alcoholic drinkers, CETP activity declines [30]. This decline of CETP activity could explain the rise of HDL cholesterol in *Tchoukoutou* and *Sodabi* consumers.

In accordance with the results of former works [23, 31], *Tchoukoutou* and *Sodabi* consumers had their triglyceridemia means significantly higher than those of non-consumers. This triglyceridemia modification in alcohol consumers could be explained by the fact that alcohol consumption leads to reduction of mitochondrion NAD+, indispensable for the oxidization of fatty acids. As a result, there is an inhibition of acetoacetate metabolism and fatty acid oxidization in the liver. Thus, the rise of NADH/NAD+ ratio fosters the synthesis of fatty acids implying an accumulation of triglycerides [32]. According to Siler et *al.* [33], alcohol consumption causes a rise in triglycerides and VLDL production; this explains the elevation of triglyceridemia in *Tchoukoutou* and *Sodabi* consumers compared to non-consumers.

Tchoukoutou consumers had LDL cholesterolemia significantly lower than the one of non-consumers as former research works reported [11, 34]. This decline of LDL cholesterolemia in alcohol consumers is due to the fact that alcohol consumption generates an increasing activity of paraoxonase that is one of the key enzymes of HDL antioxidizing action opposite LDL lipoproteins [35]. Paraoxonase confers to HDL antioxidizing properties by making possible the hydrolysis of LDL oxidized phospholipids [35].

The VLDL cholesterolemia means of *Tchoukoutou* and *Sodabi* consumers were significantly higher than the ones of non-consumers in our study. Crouse and Grundy [36] found similar results. These results are logic as the same observation had been made with triglyceridemia ; VLDL cholesterolemia was obtained by dividing triglyceridemia rate by five (5).

The atherogenic index was significantly lower in *Tchoukoutou* and *Sodabi* consumers compared to the one of non-consumers in our study. Wakabayashi [37] found identical results. The analysis of our results and the ones of former works [31, 37] allows to note that the higher daily alcohol intake quantity is, the lower the atherogenic index is. Therefore, non-consumers have a higher risk of exposure to atheromatosis than alcohol consumers. *Tchoukoutou* and *Sodabi* moderate consumption would have a beneficial effect on health.

There is no significant difference between the means of *Tchoukoutou* and *Sodabi* consumumers lipid parameters. The increase of the values of *Tchoukoutou* and *Sodabi* consumers lipid parameters compared to non-consumers would be related to alcohol effect and not to the other components of these alcoholic beverages.

The correlations of alcohol consumption duration with triglyceridemia and VLDL cholesterolemia noted in our study could be due to the fact that it is a dose-dependent relationship. Actually, the higher alcohol consumption is, the higher triglyceridemia is [38]. Moreover, excessive consumption is most often observed in chronic consumers; thus this correlation with alcohol consumption duration.

With the daily mean quantity of alcohol intake, correlations with lipid parameters found in our research work could be due to the difference in the composition of both drinks. In fact, the nutritional value of beers made with sorghum is generally higher than the one of *Sodabi*, because of presence of yeast, bacteria, lactic acid and other suspended particles [39]. In addition, the baseline process of traditional beer making in West Africa is characterized by malting where starch is transformed into simple sugar and then in acetic acid [3]; this process of acidification of the product would foster a substantial modification of lipid parameters [40, 41].

However, our study has some limits. Actually, the difficulty of matching age, sex and weigh did not enable us to conduct a case-control study. Nevertheless, most of the results achieved were significant indeed. The dosage of apoproteins, lipoprotein (a), HDL2-cholesterol and HDL3-cholesterol would make this research work more exhaustive. The measurement of alcoholic drink consumption was self-reported; in these circumstances, some subjects may have underrated their consumption level. But according to Frone [42], in general alcohol intake assessments are considered reliable and valid.

5. CONCLUSION

Our study enabled to find out that T*choukoutou* and S*odabi* consumption has variable influences on serum lipid parameters. *Tchoukoutou* and S*odabi* increase HDL cholesterolemia, triglyceridemia and VLDL cholesterolemia. On the other hand, these two local alcoholic drinks inhibit atherogenic index. Only T*choukoutou* decreases LDL cholesterolemia. There is no significant difference between the lipid parameters values of *Tchoukoutou* and S*odabi* consumers. The modification of lipid parameter values is not enough correlated with consumption duration on the one hand, and daily mean quantity of alcohol intake on the other hand.

Therefore, this study shows that the consumption of *Tchoukoutou* and S*odabi*, two traditional drinks made in Benin, modify serum lipid values to the effect of reducing exposure to atheromatosis.

REFERENCES

[1] World Health Organization. World Status Report on Alcohol and Health, 2011. Website. http://www.WHO.int. Consulted on November 14, 2013.

[2] World Health Organization. Alcohol consumption. Notes N° 349 ; 2011. Website. http://www.WHO.int. Consulted on November 14, 2013.

[3] A.P.P. Kayodé, J.D. Hounhouigan, M.J.R. Nout, A. Niehof. 'Household production of sorghum beer in Benin: technological and socio-economic aspects.' *International Journal of Consumer Studies*, vol. 31, pp. 258-264, 2006.

[4] R.G. Noll. 'The Wines of West Africa: History, Technology and tasting notes.' *Journal of Wine Economics*, vol. 3, pp. 85- 94, 2008.

[5] D.N. Shaffer, R. Njeri, A.C. Justice, W.W Odero, W.M. Tierney. 'Alcohol abuse among patients with and without HIV infection attending public clinics in western Kenya.' *East African Medical Journal*, vol. 81, pp. 594-598, 2004.

[6] B.N. Seïdou, A.P.P. Kayodé, G. Dalodé-Vieira, L. Baba-Moussa, S.O. Kotchoni, A.J.D. Hounhouigan. 'Improvement of the traditional technology for the production of « *gowé* », a sour and sweet beverage from Benin.' *Afr. J. Food Sci.*, vol. 5, pp. 806-813, 2011.

[7] A.P.P. Kayodé, G. Vieira-Dalodé, A.R. Linnemann, O.S. Kotchoni, A.J.D. Hounhouigan, M.A.J.S. van Boekel, M.J.R. Nout. 'Diversity of yeasts involved in the fermentation of *tchoukoutou*, an opaque sorghum beer from Benin.' *Afr. J. Microbiol. Res.*, vol. 5, pp. 2737- 2742, 2011.

[8] W.P. Castelli , J.T. Doyle, T. Gordon, C.G. Hames, M.C. Hjortland, S.B. Hulley, A. Kagan, W.J. Zukel. 'Alcohol and blood lipids : The cooperative lipoprotein phenotyping study.' *Lancet*, vol. 2, pp. 153-155, 1977.

[9] S.R. Choudhury , H. Ueshima ,Y. Kita , K.M. Kobayashi, A. Okayama, M. Yamakawa, Y. Hirao, M. Ishikawa, Y. Miyoshi. 'Alcohol intake and serum lipids in a Japanese population.' *Int J Epidemiol*, vol. 23, pp. 940-947, 1994.

[10] A. Branchi, A. Rovellini, C. Tomella, L. Sciariada, A. Torri, M. Molgora, D. Sommariva. 'Association of alcohol consumption with HDL subpopulations defined by apolipoprotein A-I and apolipoprotein A-II content.' *Eur J Clin Nutr*, vol. 51, pp. 362-365, 1997.

[11] A. Van Tol, M.S. Van Der Gaag, L.M. Scheek, T. Van Gent, H.F.J. Hendricks. 'Changes in postprandial lipoproteins of low and high density caused by moderate alcohol consumption with dinner.' *Atherosclerosis*, vol. 141, pp. 101-103, 1998.

[12] E. De Oliveira, D. Foste, M. McGee Harpe, C.E. Seidman, J.D. Smith, J.L. Breslow, E.A. Brinton. 'Alcohol consumption raises HDL cholesterol levels by increasing the transport rate of apolipoproteins A-I and A-II.' *Circulation*, vol. 102, pp. 2347-2352, 2000.

[13] D.R. Parker, J.B. McPhillips, C.A. Derb, K.M. Gans, T.M. Lasater, R.A. Carleton. 'High-density lipoprotein cholesterol and types of alcoholic beverages consumed among men and women.' *Am J Public Health*, vol. 86, pp. 1022-1027, 1996.

[14] K. Kervinen, M.J. Savolainen, Y.A. Kesaniem. 'A rapid increase in lipoprotein (a) levels after ethanol withdrawal in alcoholic men.' *Life Sci*, vol. 48, pp. 2183-2188, 1991.

[15] S.A. Akpona, S. Iwole, I. Kora, P. Lebreton, Josse R. 'Dyslipidemia and atherogenic among population at risk in Cotonou.' *Méd. Afr. Noire*, vol. 42, pp. 145-151, 1995.

[16] S.A. Akpona, M. Gomina Assoumanou, M.M. Soumanou, R. Adegbindin, I. Abdoulaye. 'Reference values of lipoparticle (a) and other apoproteins among Benin citizens through agarose gel electrophoresis.' *Rev. CAMES-Série A*, vol. 8, pp. 72-75, 2009.

[17] M. Gomina Assoumanou, K.D. Djaliri, A.S. Akpona. 'Serum lipid profile of nomad adult Fulani in the North-Benin.' *J. Appl. Biosci*, vol. 62, pp. 4620-4627, 2013.

[18] M. Gomina, G. Yarou Ngobi, S.A. Akpona. 'Serum lipid profile of adult habitual tobacco consumers in Benin.' *European Scientific Journal*, vol. 9, pp. 125-140, 2013.

[19] M-L. Solera. 'Lipid and lipoprotein metabolism', in *Biochimie clinique*, Paris : cedex, 2000, pp.168-186.

[20] G.R. Warnick, M.C. Cheung, J.J. Albers. 'Comparison of current methods for high-density lipoprotein cholesterol quantification.' *Clin Chem*, vol. 25, pp. 596-604, 1979.

[21] W.T. Friedewald, R.I. Levy, D.S. Fredrickson. 'Estimation of concentration of low density lipoprotein cholesterol in plasma without use of ultracentrifuge.' *Clin Chem*, vol. 18, pp. 499-502, 1972.

[22] F. Paille. 'Practical assessment of alcohol consumption. Classifications and definitions of alcoholzation behaviors.' *Gastroenterol Clin Biol*, vol. 26, pp. 141-148, 2002.

[23] S.G. Wannamethe, A.G. Shaper. 'Blood lipids: the relationship with alcohol intake, smoking and body weight.' *J Epid Com Healh*, vol. 46, pp. 197-202, 1992.

[24] A. Roy, D. Prabhakaran, P. Jeemon, K.R. Thankappan, V. Mohan, L. Ramakrishnan, P. Joshi, F. Ahmed, B.V. Mohan, R.K. Saran, N. Sinha, K.S. Reddy. 'Impact of alcohol on coronary heart disease in Indian men.' *Atherosclerosis*, vol. 210, pp. 531-535, 2010.

[25] C. Schäfer, A. Parlesak, J. Eckoldt, C. Bode, J.C. Bode, W. Marz, K. Winkler. 'Beyond HDL-cholesterol increase: phospholipid enrichment and shift from HDL3 to HDL2 in alcohol consumers.' *J Lipid Res*, vol. 48, pp. 1550-1558, 2007.

[26] M.S. Brow, P.T. Kovanen, J.L. Goldstein. 'Regulation of plasma cholesterol by lipoprotein receptors.' *Science*, vol. 212, pp. 628-635, 1981.

[27] L. Matthieu. 'Characterization of a new human apolipoprotein, Apo O.' Ph.D thesis. Paul SABATIER University -Toulouse III. France. 2006.

[28] G.H. Hartung, J.P. Foreyt, R.S. Reeves, L.P. Krock, W. Patsch, J.R. Patsch, A.M. Jr. Gotto. 'Effect of alcohol dose on plasma lipoprotein subfractions and lipolytic enzyme activity in active and inactive men.' *Metabolism*, vol. 39, pp. 81-86, 1990.

[29] P. Moulin. 'Cholesteryl ester transfer protein (CETP).' *Sang Thrombose Vaisseaux*, vol. 7, pp. 475-485, 1995.

[30] M.C. Guimon. 'Lipoprotein Lp(a) : its interest in the interpretation of lipid balance.' *Bio économie*, vol. 293, pp. 3-43, 1998.

[31] A.V. Kelly, M.B. Christie, D.F. Flavio, A.S. Richey, B. Eric. 'Relationship of Alcohol Consumption and Type of Alcoholic Beverages Consumed With Plasma Lipids Levels: Differences Between Whites and African Americans of the ARIC Study.' *Ann Epidemiol*, vol. 18, pp. 101-113, 2008.

[32] J.L. Schlienger. 'Alcohol nutritional and metabolic impact,' in *Les Entretiens de Bichat*, 2013, pp. 1-4.

[33] S.Q. Siler, R.A. Neese, E.J. Parks, M.K. Hellerstein. 'VLDL-triglyceride production after alcohol ingestion, studied using [2-$^{13}C_1$] glycerol.' *J Lipid Res*, vol. 39., pp. 2319-2328, 1998.

[34] L. Djoussé, I.M. Lee, E.B. Julie, G. Michael. 'Alcohol Consumption and Risk of Cardiovascular Disease and Death in Women: Potential Mediating Mechanisms.' *Circulation*, vol. 120, pp. 237-244, 2009.

[35] D. Bonnefont-Rousselot, M. Gardès-Albert, D. Jore, J. Delattre. 'HDL lipoproteins and atherosclerosis oxidation theory.' *Sang Thrombose Vaisseaux*, vol. 10, pp. 166-174, 1998.

[36] J.R. Crouse, S.C. Grundy. 'Effect of alcohol on plasma lipoproteins and cholesterol and triglycerides metabolism in man.' *J Lipid Res*, vol. 25, pp. 486-496, 1984.

[37] I. Wakabayashi. 'Relationships among alcohol drinking, blood pressure and serum cholesterol in healthy young women.' *Clin Chim Act*, vol. 388, pp. 192-195, 2008.

[38] A. Di Castelnuovo, S. Rotondo, L. Iacoviello, M.B. Donati, G. de Gaetano. 'Meta-analysis of wine and beer consumption in relation to vascular risk.' *Circulation*, vol. 105, pp. 2836-2844, 2002.

[39] G. Miljkovic, C.H. Bunker, R.E. Ferrell, C.M. Kammerer, R.W. Evans, V.W. Wheeler. 'Lipoprotein subclass and particle size differences in Afro-Caribbean, African Americans, and white Americans: associations with hepatic lipase gene variation.' *Metabolism*, vol. 55, pp. 96-102, 2006.

[40] B. Bergeret, J. Perisse, R. Masseyeff, S. Le Berre. 'Ingredients tables of some tropica foods.' *Annales de la nutrition et de l'alimentation*, vol. 11, pp. 45-89, 1957.

[41] R.O. Adeleke, O.A. Abiodun. 'Physico-chemical properties of Commercial Local Beverages in Osun-State, Nigeria.' *Pak J Nutr*, vol. 9, pp. 853-855, 2010.

[42] M.R. Fron. 'Prevalence and distribution of alcohol use and Impairment in the Workplace: A U.S. National Survey.' *Journal of Studies on Alcohol and Drugs*, vol. 67, pp. 147-156, 2006.