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RESEARCH ARTICLE

ASSESSMENT OF SYSTEMIC URIC ACID LEVELS AMONG CONSUMERS OF ALCOHOLIC BEVERAGES IN PORT HARCOURT NIGERIA

***,[1]Igwe, F. U., [1]Ndon, O. M. and [2]Brown, H.**

[1]Department of Biochemistry, Faculty of Science, Rivers State University of Science and Technology, P.M.B 5080, Port Harcourt, Rivers State, Nigeria

[2]Department of Medical Laboratory Sciences, Faculty of Science, Rivers State University of Science and Technology, P.M.B 5080, Port Harcourt, Rivers State, Nigeria

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ABSTRACT

The alteration in systemic uric acid homeostasis following consumption of certain beverages is generating concern among clinical scientists. There is renewed speculation that high levels of uric acid in body fluids can cause accumulation of urate crystals in joints which may result in gouty conditions. This study assessed the relationship between consumption of various types of alcoholic beverages and corresponding uric acid levels in the blood and urine of regular consumers in Port Harcourt, Nigeria. Randomly selected adult male and female volunteers, who accepted to undergo alcohol, caffeine and aspirin fast for 48 hours prior to consumption of specified volume of the various alcoholic beverages, were studied. The volunteers were divided into groups according to sex and preferred choice of alcoholic beverage. Blood and urine samples were collected, before and one hour after consumption of the beverage. Serum and urine uric acid levels were determined using uricase methods. There was significant ($P<0.05$) increase in the serum uric acid levels of male volunteers that consumed stout and larger beer. The urine uric acid levels of the male volunteers that consumed stout, larger beer and brandy were significantly ($P<0.05$) lower than the corresponding levels before consumption. For the female volunteers, there was significant ($P<0.05$) increase in the serum uric acid levels among those that consumed stout, brandy and table wine. Conversely, there was significant ($P<0.05$) decrease in the urine uric acid levels among those that consumed larger beer. The elevation of serum uric acid and its decreased elimination through urine may lead to uric acid accumulation in blood and predispose regular consumers of high doses of these alcoholic beverages to gout arising from deposition of uric acid crystals. Appropriate choice of the less susceptible brands of alcoholic beverage and moderation in consumption frequency is advocated.

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INTRODUCTION

The use of alcoholic beverages for social, medical, cultural and religious purposes dates back to antiquity. There is renewed debate about the association of alcohol consumption, uric acid levels in blood and gout. Several reports have suggested that the worldwide incidence and prevalence of gout is increasing (Harris *et al*, 1995; Klemp *et al*., 1997; Aromdee *et al*., 2002; Mikuls *et al*., 2005). Gout may occur when monosodium urate precipitates from supersaturated body fluid. The condition is thought to be associated with urate crystals in joint fluid and with deposits of the crystals (tophi) in tissue surrounding the joints. As serum uric acid levels rises and the physiological saturation threshold for uric acid is exceeded in the body fluids, the formation and deposition of monosodium urate crystals occurs in and around joints. This is responsible for the clinical signs and symptoms of severe pains and inflammation affecting peripheral joints of patients (Roddy *et al.,* 2007). Gout is the most prevalent form of inflammatory arthritis and is associated with impaired quality of life (Roddy *et al*., 2007). It is sometimes classified as primary or secondary. Primary gout is associated with essential hyperuricemia with polygenic basis like the metabolism of purines, decreased renal excretion and increased dietary intakes. While secondary gout is as a result of hyperuricemia attributable to several other identifiable causes (Wallace *et al*., 1977). An association between gout and dietary factors has been recognized for centuries. Dietary consumption of meat and seafood was associated with an increased risk of gout, consumption of purine-rich vegetables was not associated with the development of gout and

Corresponding author: **Igwe,**
Department of Biochemistry, Faculty of Science, Rivers State University of Science and Technology, P.M.B 5080, Port Harcourt, Rivers State, Nigeria.

consumption of dairy products appeared to be protective (Choi *et al*., 2004). Choi *et al*. (2007) also reported that consumption of six or more cups of coffee per day appeared to be protective against the development of gout compared with no consumption. A significant protective effect was also seen in those drinking smaller amounts of decaffeinated coffee. Tea consumption was not associated with development of gout as was the intakes of sugar sweetened soft drinks and fructose (Choi and Gurhan, 2008). Hyperuricemia is considered one of the most important risk factor for the development of gout. Lin *et al*. (2000) reported that the prevalence of gout was high among men with hyperuricemia. Further reports had indicated that the risk of developing gout increases with increasing serum uric acid level in both men and women (Aromdee *et al*., 2002; Bhole *et al*., 2010). Other reported risk factors for development of gout include; metabolic Syndrome (Grindy *et al*., 2004; Choi *et al.,* 2007), waist-hip ratio and obesity (Bhole *et al*., 2010), renal disease (Cohen *et al*., 2008) and use of diuretics (Choi *et al*., 2005; Jassens *et al.,* 2006; Bhole *et al*., 2010). Recent epidemiological data as reported by Mikuls *et al*. (2005) and Bhole *et al.* (2010) support the relationship between alcohol consumption and risk of developing gout. In a study of 321 acute attacks in 197 subjects, a dose-dependent relationship was found between the number of alcohol drinks consumed in the previous 48 hours and acute attacks of gout (Zang *et al*., 2006). However, the variability according to the type of alcoholic beverages consumed has not been ascertained. Given the association of gout with some co morbid diseases and reduction in quality of life, it is important that the effects of alcoholic beverage consumption and uric acid levels in body fluids be evaluated properly to generate useful information that will help individuals make responsible decisions about their choices, consumption habits and social life. Also, the association of alcoholic beverage consumption and risk of gout among Nigerians has not been fully investigated. Consequently, this study is aimed at assessing the systemic uric acid levels of some Nigerian adults following the consumption different types of alcoholic beverages.

MATERIALS AND METHODS

Experimental Design

Forty (40) adult male and forty (40) adult female volunteers aged between 19 and 30 years and weighing between 60 – 75kg were randomly selected from Rivers State University of Science and Technology, Port Harcourt, Nigeria. They observed alcohol, caffeine and aspirin fast for 48 hours prior to the consumption of the test alcoholic beverage. The alcoholic beverages (Larger Beer, Stout, Table Wine and Brandy) were obtained from a certified retail shop at the Port Harcourt Mall in Port Harcourt, Nigeria. The volunteers were separated into groups of 10 persons according to sex and usual preference for a particular alcoholic beverage. Each volunteer in the "Larger Beer" and "Stout" group consumed 600ml of the drink while each volunteer in the "Table Wine" and "Brandy" group consumed 300ml of the drink.

Sample Collection

The blood and urine samples of each volunteer were collected before consumption and one hour after consumption of the beverages. Venous blood samples were collected using sterilized hypodermic needle and syringe and transferred into plain sterile bottles. The blood was centrifuged and serum collected and stored at freezing temperatures. Urine samples were collected in plain sterile sample bottles and urinalysis conducted same day using cumbosreen urinalysis stripe.

Determination of Uric Acid Levels

The serum uric acid and urine uric acid levels were measured by the uricase method (Drum, *et al*., 1981). The principle is based on uricase acting on uric acid to produced allontoin, hydrogen peroxide and carbon dioxide, which under the influence of peroxidase, oxides 3,4,-dichloro-2-hydroxybenzensulfonic acid and 4-aminophenazone to form a red-violet compound that is measured at 520nm.

Statistical Analysis

All data with normal distribution were expressed as mean ± standard error of mean. Student's t-test was used to compare the variables within groups. A level of $P<0.05$ was considered statistically significant. Schematic presentations were expressed as aligned dot plots. All the data were analyzed using Prism Graph Pad 5.1.

RESULTS

Results of the serum and urine uric acid levels of female volunteers that consumed various alcoholic beverages are presented in Table 1.1 and Figures 1 - 8.

Table 1.1 Uric acid levels of female volunteers following consumption of alcoholic beverages

Alcoholic Beverage	Serum Uric Acid Level (mg/dl)			Urine Level (mg/dl)		
	Before	After	% Change	Before	After	% Change
Larger Beer	7.67	9.17	19.50 P=0.0707	1140.825	687.50	-39.34 P= 0.0012
Stout	9.83	13.00	32.23 P=0.0304	1081.68	605.00	-44.07 P= 0.0777
Table Wine	7.17	9.84	37.24 P=0.0160	1044.94	1319.96	26.32 P=0.5324
Brandy	6.50	8.17	25.64 P= 0.0218	1214.60	509.175	-58.08 P=0.0564

Values are mean of ten determinations. (-) indicate percent decrease. P-values from the student t-test is indicated

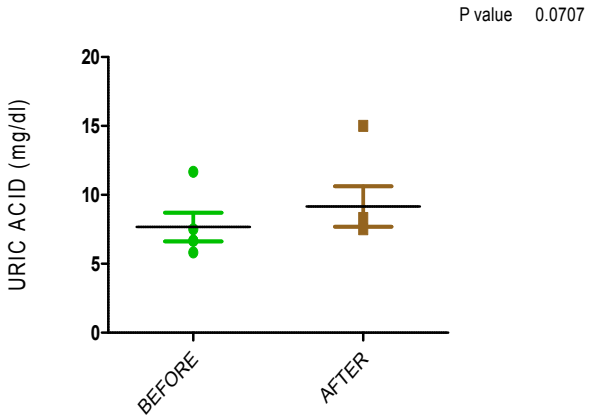


Figure 1 SERUM URIC ACID LEVELS OF FEMALES ON STAR BEER.

Serum uric acid levels of the females that consumed Lager Beer is shown and illustrated in Figure 1. There was no significant difference ($P>0.05$) between uric acid level before consumption and one hour after consumption. There was

significant increase (P<0.05) in the mean uric acid levels of females after consumption of Stout (Figure 2). A significant increase (P<0.05) in serum uric acid level was also observed for the females that consumed Brandy (Figure 3) and Table Wine (Figure 4).

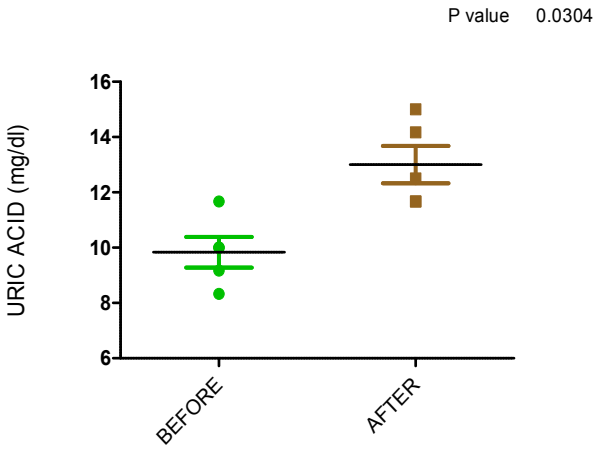


Figure 2 SERUM URIC ACID LEVELS OF FEMALES ON STOUT BEER.

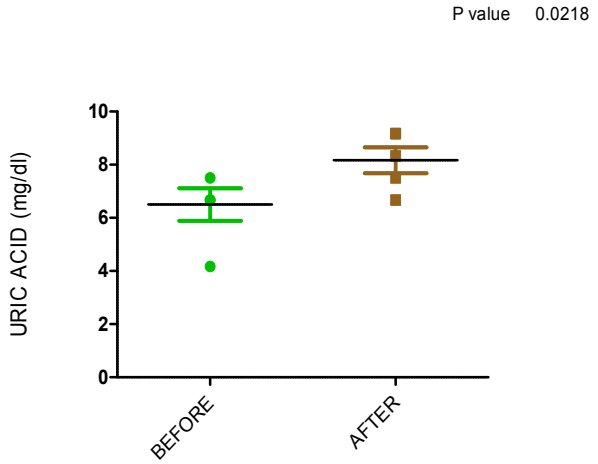
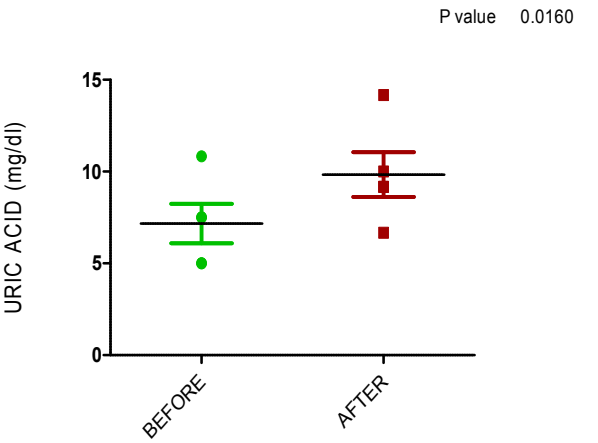


Figure 3 SERUM URIC ACID LEVELS OF FEMALES ON BRANDY.



Figuer 4 SERUM URIC ACID LEVELS OF FEMALES ON TABLE WINE.

Results of the urine uric acid levels of the females on Larger Beer, Stout, Brandy and Table Wine are presented in Figures 5 - 8 respectively. There was significant decrease (P<0.05) in urine uric acid levels of the females that consumed Larger Beer while the difference obtained for females that consumed Stout, Brandy and Table Wine were not statistically significant (P>0.05).

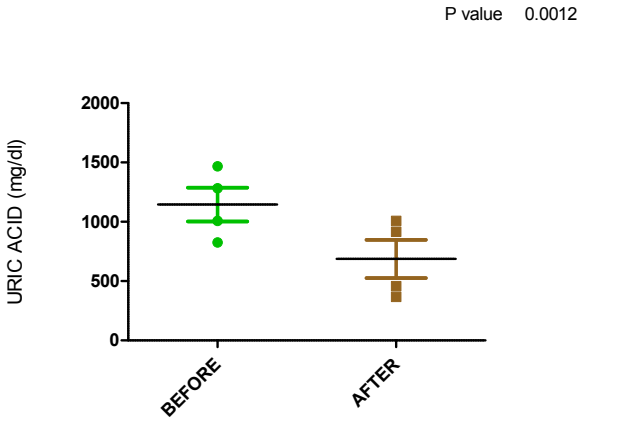


Figure 5 URINE URIC ACID LEVELS OFFE MALES ON STAR BEER

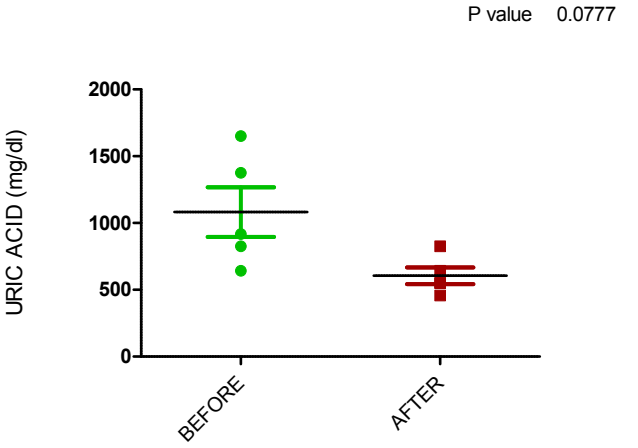


Figure 6 URINE URIC ACID LEVELS OF FEMALES ON STOUT BEER

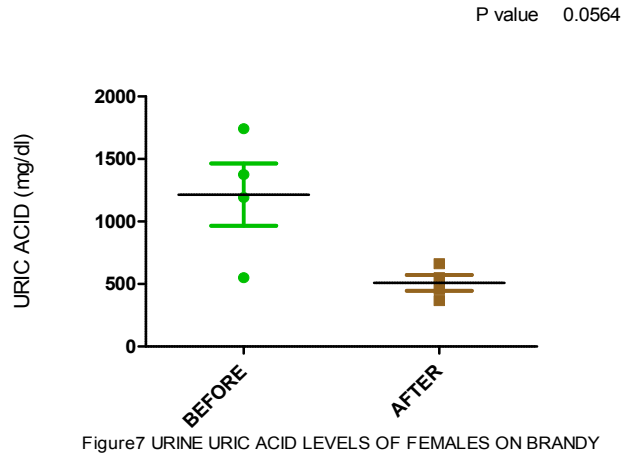


Figure7 URINE URIC ACID LEVELS OF FEMALES ON BRANDY

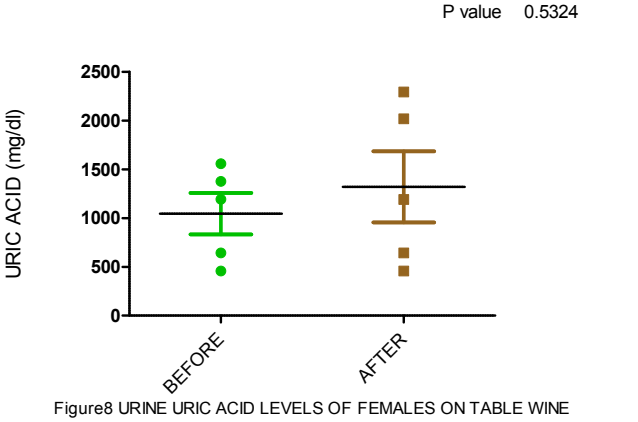


Figure8 URINE URIC ACID LEVELS OF FEMALES ON TABLE WINE

Results of the serum and urine uric acid levels of the male volunteers that consumed various alcoholic beverages are presented in Table 1.2 and Figures 9 - 16.

Table 1.2. Uric acid levels of male volunteers following consumption of alcoholic beverages

Alcoholic Beverage	Uric Acid Level (mg/dl) Serum				Urine	
	Before	After	% Change	Before	After	% Change
Larger Beer	9.334	10.832	16.05 P=0.0087	879.920	733.340	- 16.66 P=0.0350
Stout	7.166	9.836	37.30 P=0.0160	769.940	696.680	-9.51 P=0.0336
Table Wine	9.153	10.418	13.82 P=0.1713	1420.850	1307.925	-7.95 P=0.0788
Brandy	8.332	9.664	15.99 P=0.1204	1265.000	1063.320	-15.94 P=0.0295

Values are mean of ten determinations. (-) indicates percent decrease. P-values from the student t-test is indicated

The mean serum uric acid levels of the male volunteers that consumed Stout are shown in Figure 9. There was significant increase ($P<0.05$) in uric acid levels after consumption. The mean serum uric acid level of the male volunteers that consumed Larger Beer was also significantly ($P<0.05$) higher after consumption of the beverage (Figure 10). There were no significant differences ($P>0.05$) in the male volunteers that consumed Brandy and Table Wine (Figures 11 and 12 respectively).

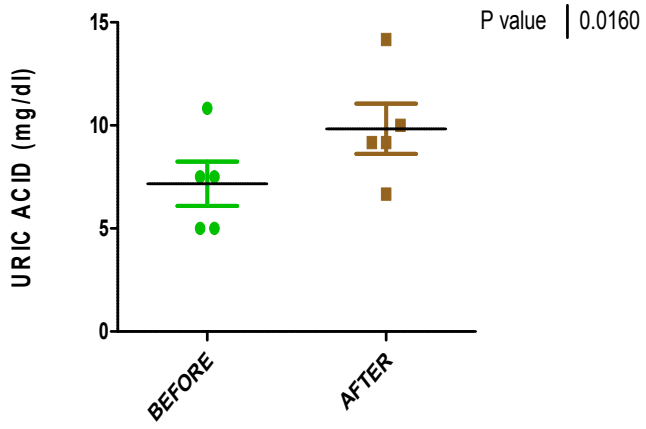


Figure 9 SERUM URIC ACID LEVELS OF MALES ON STOUT BEER.

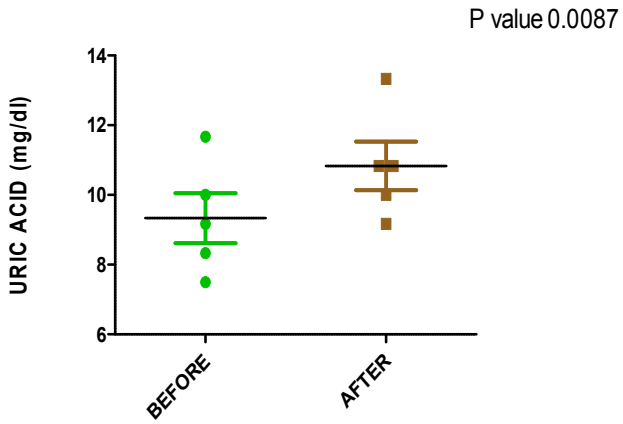


Figure10 URIC ACID LEVELS OF MALES ON STAR LARGER BEER

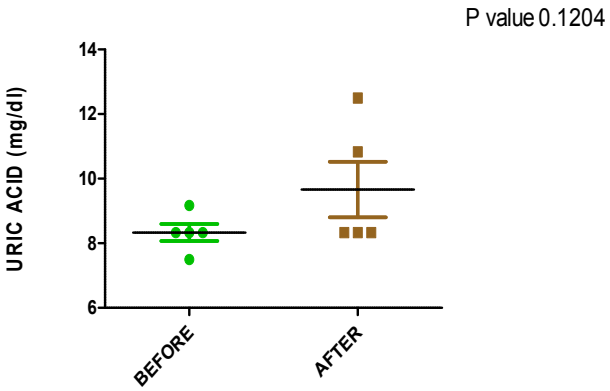


Figure11 URIC ACID LEVELS OF MALES ON BRANDY

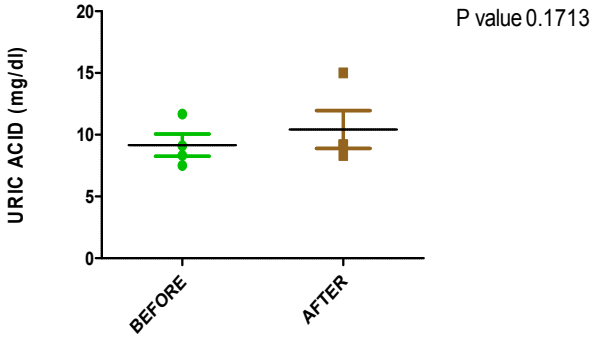


Figure 12 URIC ACID LEVELS OF MALES ON TABLE WINE

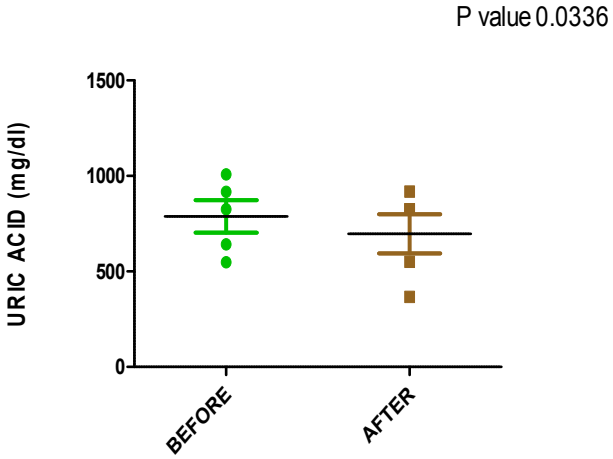


Figure 13 URINE URIC ACID LEVELS OF MALES ON STOUT BEER

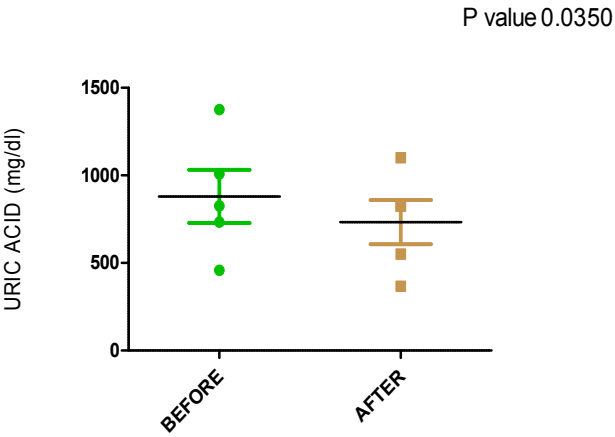


Figure 14 URINE URIC ACID LEVELS OF MALES ON STAR BEER

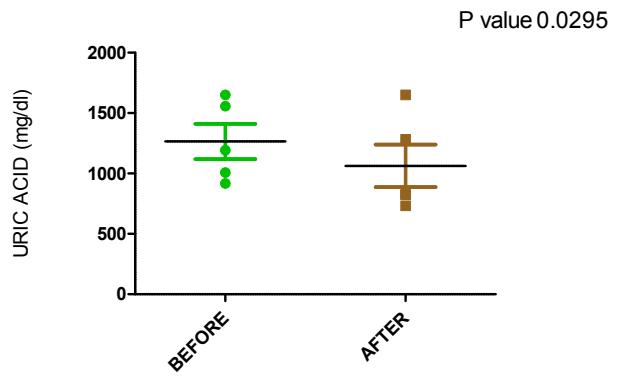


Figure 15 URINE URIC ACID LEVELS OF MALES ON BRANDY

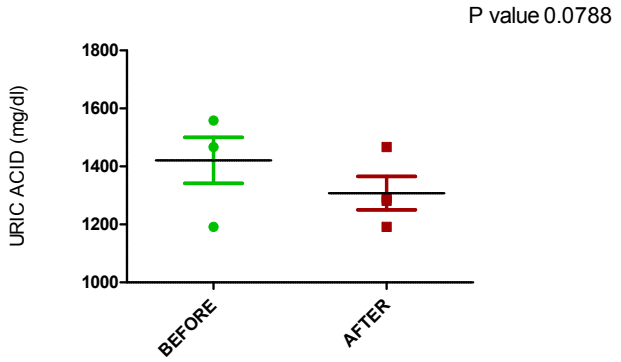


Figure 16 URINE URIC ACID LEVELS OF MALES ON TABLE WINE

DISCUSSION

The mean serum uric acid levels among the female volunteers were generally higher than the normal range of 2.4 to 5.7 mg/dl for females (Table 1.1) and 3.4 to 7.0 mg/dl for males (Table 1.2). Consumption of alcoholic beverages led to increases in the serum uric acid levels. Whereas Stout produced the highest increase (37.30%) among males, Table Wine produced the highest increase (37.24%) among females. Both increases were statistically significant ($P<0.05$). Consumption of Stout produced significant ($P<0.05$) elevation of serum uric acid levels in both sexes (Table 1.1 and 1.2) compared to the other alcoholic beverages. This suggest that in addition to the alcohol present in Stout other compounds containing purine nucleotides, which can be metabolized to uric acid, may be present in Stout. Faller and Fox (1982) and Fam (2002) reported that hyperuricemia can be attributed to ethanol-induced accelerated degradation of purine nucleotides among other factors. The percent increase in serum uric acid levels for those that consumed Larger Beer, Table Wine and Brandy were higher in females than in males. This further confirms the assertion that the likely negative effect of alcohol consumption will be prevalent among females than males as females have smaller muscle mass and only about 55% of the female body mass is available for the distribution of alcohol while about 68% of the male body mass can accommodate alcohol. The consistent elevation and maintenance of high serum uric acid levels can lead to its physiological saturation in body fluids and subsequent formation of urates crystals especially around the joints. This can predispose regular consumers of alcoholic beverages to gout. Results from this study indicate that females are therefore more susceptible to this condition than males. Gout occurs when monosodium urate precipitates from supersaturated body fluid. The formation and deposition of monosodium urate crystals in and around joints are responsible for the clinical signs and symptoms of severe pains and inflammation affecting peripheral joints of patients (Roddy *et al.,* 2007). In both male and female volunteers, there was significant decrease in the urine uric acid level after the consumption of alcoholic beverages. This probably explains the increase in serum uric acid levels. The overall percent decrease is greater in females compared to males. Consumption of brandy resulted in 58.08% decrease in the urine uric level in females (Table 1.1) while a

Table 1.3. Urine pH and SG of male volunteers following consumption of alcoholic beverages

Alcoholic Beverage	pH			SG		
	Before	After	Change (%)	Before	After	Change (%)
Larger Beer	6.60	5.80	-12.12	1.018	1.058	3.929
Stout	8.20	6.80	-17.07	1.017	1.008	-0.885
Table Wine	8.00	5.00	-37.50	1.013	1.025	1.185
Brandy	6.80	5.40	-20.59	1.024	1.022	-0.195

Values are mean of ten determinations. (-) indicates percent decrease.

Table 1.4 Urine pH and SG of female volunteers following consumption of alcoholic beverages

Alcoholic Beverage	pH			SG		
	Before	After	% Change	Before	After	% Change
Larger Beer	6.40	5.20	-18.75	1.027	1.006	-2.045
Stout	6.20	5.20	-16.13	1.017	1.006	-1.082
Table Wine	5.06	5.60	10.67	1.023	1.018	-0.489
Brandy	6.60	5.20	-21.21	1.054	1.005	-4.649

Values are mean of ten determinations. (-) indicates percent decrease.

The urine uric acid levels of male volunteers that consumed Stout, Larger Beer, Brandy and Table Wine are shown in Figure 13-16. There were significant differences ($P<0.05$) in the mean urine uric acid levels of males volunteers that consumed Stout, Larger Beer and Brandy (Figures 13, 14 and 15 respectively). However, there was no significant difference ($P>0.05$) in the urine uric acid levels for the male volunteers that consumed Table Wine (Figure 16) The urine pH and Specific Gravity (SG) of the male and female volunteers following consumption of the selected alcoholic beverages are presented in Tables 1.3 and 1.4. There was a general decrease in urine pH following consumption of the various alcoholic beverages. Table Wine showed the highest percent decrease in males (-37.50%) while Brandy gave the highest percent decrease in females (-21.21%). Changes in the SG of urine following consumption of the alcoholic beverages were less than 4% for both male and female volunteers

15.94% decrease was recorded for males (Table 1.2). This indicates that the renal elimination of serum uric acid through the urine is probably slower in the females than the males assayed. This decrease in urine uric acid levels may also be attributed to dilution arising from an increase in the renal elimination of water from the system following consumption of alcoholic beverages. Consumption of alcoholic beverages may have distorted the steady-state condition (homeostasis) of the uric acid pool accounting for a decrease in the urine and increase in blood. The presence of alcohol in the system induces accelerated metabolism and causes frequent urination as water is indeed one of the products of alcohol metabolism.

The urine pH profile showed a general decrease after consumption of alcoholic beverages. This could be due to increased presence of hydrogen ions produced as a result of increased metabolism initiated by the presence of alcohol in the system. The changes were minimal indicating that the buffering capacity of the urine was adequate to accommodate it. The SG which indicates the weight of solutes in urine, decreased after intake of alcoholic beverages. This implies that there was decrease in the production of solutes or increase in the production of water. The urine was probably diluted by increased glomerular filtration of water. Increased elimination of water through urination, sweating or breathing is common features among consumers of alcoholic beverages. This phenomenon which causes general dehydration of the system may further accelerate the formation of urate crystals in joint fluids as well as potentiate the manifestation of other deleterious effects of alcohol consumption. Several mechanisms by which alcohol predisposes to hyperuricemia have been proposed, including reduced renal urate excretion via lactic acids or lead poisoning, increased urate production via ethanol-induced accelerated degradation of purine nucleotides or the high purine content of some beverages enhancing substrate concentration and poor compliance with urate-lowering therapy (Fam, 2002).

Conclusion

Consumption of alcoholic beverages caused an increase in serum uric acid levels and decrease in the urine uric acid levels of the male and female volunteers studied. However, the percent change was higher in females compared to males. Consumption of Stout produced 37.30% increase in males and 32.23% increase in females. Lager Beer produced 16.05% increase in males and 19.50% increase in females. Table Wine produced 13.82% increase in males and 37.24% increase in females and Brandy produced 15.99% increase in males and 25.64% increase in females. The change in the concentration of uric acid in urine was such that consumption of Stout accounted for 9.51% decrease in males and 44.07% decrease in females. Consumption of Lager Beer resulted in 16.66% decrease in males and 39.34% decrease in females. Table Wine produced 7.95% decrease in males and 26.32% decrease in females while Brandy accounted for 15.94% decrease in males and 58.05% decrease in females. These results suggest that the elevated levels of serum uric acid and its decreased renal elimination following consumption of alcoholic beverages may predispose regular consumers of high doses of alcoholic beverages to possible accumulation of uric acid in blood with the concomitant formation of uric acid crystals in body fluids especially around the joints. Accumulation of urate crystals in joints causes gouty arthritis. Consequently, moderation in the rate of consumption of alcoholic beverages and appropriate choice of the type with least potential for uric acid accumulation is advocated.

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