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Full Length Research Paper

## Effect of some traditional edible oils on sex hormones of albino Wistar rats

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The effect of some traditionally extracted edible seed oils (sesame, peanut and melon oils) on some sex hormones - prolactin, progesterone, testosterone, estradiol, luteinizing hormone (LH) and follicle stimulating hormone (FSH) of albino Wistar rats was studied. Sixty (30 - 50 g) weanling rats (20 males and 40 females) were purchased and housed separately until they weighed 120 to 150 g. Afterwards, the rats were cohabited in a mating ratio of 1 male : 2 females respectively, to give rise to 10 groups (n = 6), and fed rat chow (control), rat chow supplemented with 5, 10 and 20% sesame oil, 5, 10 and 20% peanut oil or 5, 10 and 20% melon oils, respectively. The animals were observed and pregnant females were separated into individual cages, allowed to litter and after weaning; the parent male and female rats were sacrificed and blood samples collected for hormonal assays. The results indicate that 5 and 10% supplemented seed oils caused significant increase ( $p < 0.05$ ) in prolactin level (with a corresponding decrease in progesterone), LH, estradiol and testosterone relative to the controls. This favourable impact on endocrine environment suggests that the consumption of these seed oils, especially sesame oil, may enhance fertility.

**Key words:** Sesame oil, peanut oil, melon oil, sex hormones.

### INTRODUCTION

The term seed oils refer generally to vegetable oils obtained from the seeds (endosperm) of food plants, rather than the fruit (pericarp). The oil-seed-bearing plants are usually grown for the economic and nutritional importance of the oils extracted therefrom; although a few serve for food or textile fibers production, in which case oil is obtained only as a by-product. Sesame, groundnut and melon oils were studied in the present investigation.

Sesame oil is an edible vegetable oil extracted from

sesame seeds (also known as beniseed in Nigeria), produced by the sesame plant (*Sesamum indicum*). *Sesamum indicum* (family *Pedaliaceae*) originated from tropical Africa (RMRDC, 2004) and is cultivated in the middle belt and some northern states of Nigeria (Olanyanju et al., 2006); hence the seeds constitute a staple food among many ethnic groups in Nigeria. The seeds are either eaten fresh, dried, fried or as a blend with sugar; as well as a paste in some traditional soups.

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Several studies have demonstrated the nutritional and health benefits of sesame seeds. The seeds are known to contain reasonable amounts of dietary proteins and fibre and minerals such as iron, magnesium, manganese, copper, phosphorous, zinc and calcium (Aremu et al., 2006). The chemical constituents of the Nigerian variety are akin to those grown in other parts of the world (Bamigboye et al., 2010). Lignans obtained from sesame seeds have been shown to possess antihypertensive, hypolipidemic, anti-inflammatory, anti-cancer and antioxidant properties (Ide et al., 2003; Kang et al., 1998; Matumura et al., 1995; Yamashita et al., 1992 and Hirose et al., 1992).

The seed oil is said to combat health conditions like cold, chronic cough and in turn prevents bronchial lung disease (Kanu, 2011). Besides its broad use in cooking, the oil is also used in the manufacture of margarine and some pharmaceuticals (Sangha et al., 2004). Sesamin, the most abundant lignan in sesame oil (Fukuda et al., 1986), is known to contain unique amounts of phytoestrogens (Jacklin et al., 2003), and hence may exert some profound effects on sex physiology. It is thought also that sesame oil can boost sex drive and improve sperm counts in males because of its reported high zinc content (Bamigboye et al., 2010). Zinc-rich diet and supplementary zinc are shown to be effective in raising testosterone level, hence improved body composition, sex drive and increase semen volume in men (Shafiei et al., 2011). This claim has however not been established firmly by scientific research, hence form part of the major objectives of the present investigation.

Peanuts oil is a vegetable oil obtained from the grains/seeds of the *Arachis hypogaea* plant (family *Fabaceae*). The oil is available in refined, unrefined, cold pressed and roasted varieties; the latter with a strong peanut flavor and aroma, analogous to toasted sesame seeds (Liu et al., 2011). It is used as general cooking oil, with a high smoke point relative to many other cooking oils, which makes it suitable for frying of foods. The major fatty acid components are oleic acid (46.8%), linoleic acid (33.4%) and palmitic acid (10.0%) (USDA, 2011). The oil also contains some stearic, arachidic, arachidonic, behenic and lignoceric acids and other fatty acids. The oil is a significant source of resveratrol, a compound associated with reduction in risk of cardiovascular disease and cancer (Sanders et al., 2000), lowering blood cholesterol level (Kritchevsky, 1988), weight loss (by decreasing appetite), and constipation relieve when topically applied to the rectum. Topical application to the skin is also known to provide relieve from arthritic and joint pains, scalp crusting and scaling, dry skin and other skin problems (Stampfer et al., 1998). However, to our knowledge, there is no available information in respect to its effect on reproductive function.

The *Citrullus lanatus* plant seeds commonly called melon (or "egusi" in Nigeria), are a source of another traditional cooking oil - melon oil. The seeds have notable

nutritional and cosmetic importance. The seeds are famous for their high oil (50%) and protein (35%); vitamins C and B<sub>2</sub>, minerals, fat and carbohydrate compositions (Rûgheimer, 1997). Its industrial importance includes use in production of moisturizers and skin regenerating/restructuring products (Jacks et al., 1976, Lazos, 1986). The oil predominantly contains unsaturated fatty acids hence exhibits high antioxidant activity (Lazos, 1986).

In spite of the extensive literature reports on the nutritional and medicinal properties of these oils, as well as the several claims to their role on reproduction, no detailed scientific study has been carried out to validate or invalidate this claim on the reproductive functions, particularly the oils extracted from the seeds grown in Nigeria. The present study was therefore undertaken to evaluate the possible impact of these vital and commonly consumed oils on reproductive functions in male and female rats using the sex hormones as the biochemical markers of reproduction. The study ensures that the oils were extracted using procedures akin to the traditional methods.

## MATERIALS AND METHODS

### Collection of seeds and extraction of the oils

Sesame, peanut and melon seeds were purchased from Watt market, Calabar in Cross River State, Nigeria. The seeds were cleaned, washed and sun-dried and afterwards ground thoroughly into a paste using a manual grinder (Corona). The extraction method employed was the traditional approach used in Nigeria, described by Tunde-Akintunde et al. (2012). In the method, water at 50 - 60°C was added to the blended seed paste in small quantities, periodically and massaged with the hands. The hot water permeates and percolates flour particles and causes the less dense oil to float as the uppermost layer, from where it is carefully removed by skimming. The extract was heated on a cooking mantle to evaporate the water (moisture); allowed to cool, then stored in air-tight coloured bottles with screw cap to prevent peroxidation. From 1 kg each of the sesame, peanut and melon seeds, 263.9 ml (26.4%), 333.3 ml (33.3%) and 319.4 ml (31.9%) of oil was extracted, respectively.

### Preparation of experimental diets

Rat chow purchased from Vital Feed Depot in Calabar, Cross River State, Nigeria, was used to compound the experimental diets thus: oil : feed (w/w) - 1 : 19 (5% oil); 1 : 9 (10% oil) and 1 : 4 (20% oil). These ratios were similarly used for the three different oils - sesame, peanuts and melon oils.

### Experimental animals

Sixty (60) weanling albino Wistar rats (20 males and 40 females) obtained from the animal house, Faculty of Science, University of Calabar, were used for this study. The animals were housed and fed in the animal house of the Department of Biochemistry until they weighed between 120 to 150 g. During this period, the males were separated from the females to avoid mating. The animal housing facility was maintained under standard conditions. Thereafter, the rats were cohabited in a mating ratio of 1 male: 2 females on the basis of their within and between groups average weight to give 10

**Table 1.** Experimental design/feeding schedule.

Group	Number of rats	Diet fed
1	6	Rat chow only (control)
2	6	5%sesame oil
3	6	10% sesame oil
4	6	20% sesame oil
5	6	5% peanut oil
6	6	10% peanut oil
7	6	20% peanut oil
8	6	5% melon oil
9	6	10% melon oil
10	6	20% melon oil

mating groups of six animals each (two males and four females). From commencement of cohabiting, the animals were fed the oils-supplemented diets and water *ad libitum* according to the schedule shown in Table 1.

During the mating period, the rats were closely observed for signs of pregnancy and the pregnant females were separated into individual cages while maintaining the initial groupings until they produce litters. The litter size was recorded for each animal and allowed to cohabit with mother until the litters were weaned; the parent animals were removed and sacrificed. This procedure lasted for about seven months - January to July 2013. The animal procedures were carried out in line with the University of Calabar; College of Medical Sciences approved protocol.

#### Collection of samples and hormonal assays

At the end of study period, each of the adult rats was anaesthetized in chloroform vapour and immediately dissected. Blood samples were collected by cardiac puncture into plain sample tubes and allowed to stand for about an hour at room temperature; after which they were centrifuged at 3000 rpm for 10 min using a bench top centrifuge (MSE, England). Sera obtained from the respective sample tubes were stored frozen until ready for hormonal assays. Heart, kidney and liver of each animal were surgically removed and weighed, and the relative organ weight was calculated thus:

$$\frac{\text{Weight of the organ (g)} \times 100}{\text{Weight of the animal (g)}}$$

The sex hormones including prolactin, progesterone, estradiol, luteinizing and follicle stimulating hormones, were evaluated by enzyme linked immunosorbent assay (ELISA) using ELISA assay kits (Accu-Bind Elisa microwell, monobind, USA, 100 North Pointe Drive, Lake Forest California, 92630, USA). The procedure followed was as contained in the kit inserts.

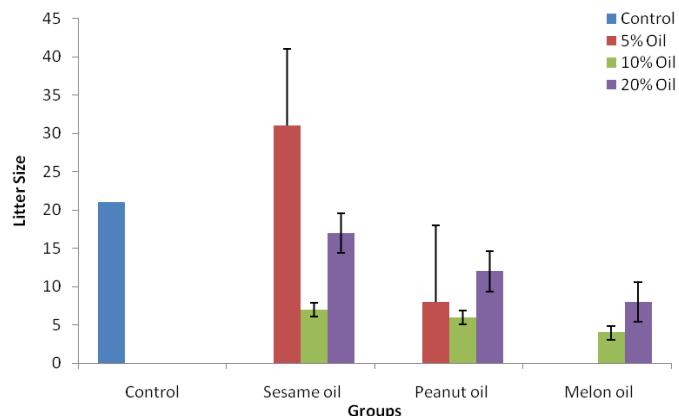
#### Statistical analysis

All values were expressed as the mean  $\pm$  SEM. The data obtained were analyzed by one way ANOVA using the SPSS statistical package, version 17 and the post hoc comparison with LSD. Differences test were considered significant at  $p < 0.05$ .

## RESULTS

### Effect of seed oils-supplemented diets on litter size

The result of the litter sizes of the various groups of rats



**Figure 1.** Number of litters produced by animal groups fed diets supplemented with 5, 10 and 20% of traditionally extracted sesame, peanut and melon oils.

treated with graded percentages of the three traditionally extracted oils is shown in Figure 1. The total number of litters produced was highest in sesame oil supplemented group (55), followed by peanut oil group (26) and melon oil fed group produced the lowest number of litters (12). Considering the individual treatment groups shows clearly that the oils exerted a non-significant effect on number of litters produced, except in the 5% sesame oil - fed group 31 l were produced as against 21 l in the control group ( $p > 0.05$ ). Moreover, there was no dose dependent effect of the oils on litter size.

### Effect of seed oils-supplemented diets on body weight

The weight gain of the experimental female and male rats fed the seed oils-supplemented diets is shown in Table 2. The 5% peanut oil supplemented group showed the greatest gain in weight ( $41.2 \pm 0.001$  g) followed by 10% sesame oil fed group ( $39.75 \pm 11.85$  g) and lastly the 5% melon oil-supplemented group ( $39.20 \pm 6.70$  g). Although not statistically significant, the weight gain decreased with increasing percentage of peanut oil supplementation was evident in both female and male rats. A similar trend was also observed in the melon oil supplemented male rats. The relative organ weights (hearts, kidney and liver) of the test groups were not significantly different from that of the control group (data not shown).

### Effect of seed oils-supplemented diets on sex hormones of female rats

The effect of seed oils-supplemented diets on sex hormones of female rats is shown in Table 3. As indicated in the result, the serum prolactin concentration was significantly increased in animal groups fed the 5 and 10% seed oils-supplemented diets ( $p < 0.05$ ). However the 20% seed oils-supplemented diet exerted a null effect



**Table 2.** Weight changes (gain) of experimental rats fed traditional oils-supplemented diets (g).

Sex	Control	Sesame oil	Peanut oil	Melon oil
<b>Female</b>	24.45 ± 4.93			
5% oil		29.03 ± 20.26	35.55 ± 14.62	30.07 ± 8.53
10% oil		24.95 ± 6.10	23.71 ± 5.73	20.77 ± 8.35
20% oil		31.35 ± 4.61	19.92 ± 4.39	22.60 ± 2.02
<b>Male</b>	32.75 ± 1.75			
5% oil		34.75 ± 10.25	41.20 ± 0.01	39.20 ± 6.7
10% oil		39.75 ± 11.85	34.70 ± 12.50	33.57 ± 3.33
20% oil		37.90 ± 3.40	26.20 ± 0.001	31.25 ± 6.25

Values are the mean ± SEM

**Table 3.** The concentration of sex hormones measured in female rats fed the seed oils-supplemented diets (ng/ml).

Sex hormone	Control	Sesame oil	Peanut oil	Melon oil
<b>Prolactin</b>	6.50 ± 0.23			
5% oil		9.0 ± 1.71*	7.6 ± 0.27*	8.00 ± 0.94*
10% oil		7.3 ± 0.36*	7.0 ± 0.001*	8.00 ± 0.41*
20% oil		6.6 ± 0.27	6.6 ± 0.27	6.75 ± 0.16
<b>Progesterone</b>	4.82 ± 1.76			
5% oil		1.11 ± 0.16*	2.42 ± 0.55*	1.50 ± 0.57*
10% oil		1.22 ± 0.38*	1.73 ± 0.23*	1.77 ± 0.56*
20% oil		5.62 ± 0.27	4.75 ± 1.44	5.12 ± 0.23
<b>Estradiol</b>	10.92 ± 1.92			
5% oil		42.50 ± 20.62*	27.50 ± 18.33*	43.75 ± 20.86*
10% oil		8.75 ± 4.33	81.25 ± 45.84*	37.50 ± 18.02*
20% oil		11.00 ± 3.40	31.25 ± 16.89*	6.25 ± 4.30
<b>LH</b>	8.52 ± 1.34			
5% oil		113.75 ± 57.70*	16.25 ± 4.33*	15.25 ± 4.75*
10% oil		27.50 ± 9.88*	18.13 ± 4.90*	15.37 ± 3.82*
20% oil		6.25 ± 0.28	6.50 ± 0.33	7.50 ± 1.10
<b>FSH</b>	12.00 ± 0.01			
5% oil		12.5 ± 0.57	12.75 ± 0.86	19.00 ± 1.97*
10% oil		12.5 ± 0.57	14.50 ± 2.81*	12.50 ± 0.57
20% oil		12.5 ± 0.57	12.50 ± 0.00	15.00 ± 1.09*

Values are mean ± SEM; \* $P < 0.05$  vs. control.

on prolactin level. Conversely, the progesterone concentration was decreased significantly in those groups with increased concentrations of prolactin ( $p < 0.05$ ). Also, 20% seed oils-supplemented showed no observable effect on serum progesterone concentration. The estradiol concentration in the entire peanut oil supplemented group showed a significantly higher level than that of the control ( $p < 0.05$ ). A similar effect was observed in the animals fed the low % supplemented sesame and melon oils, but not at 20% supplementation. Serum LH concentration

was increased in the three test groups at 5 and 10% seed oils replacements only ( $p < 0.05$ ); whereas the effect of the seed oils on FSH concentration in the female rats was rather irregular.

#### Effect of seed oils-supplemented diets on sex hormones of the male rats

The result of male sex hormones assayed in rats fed traditional oils-supplemented diets is shown in Table 4.

**Table 4.** The concentration of sex hormones measured in male rats fed with seed oils supplemented diets (ng/ml).

Sex hormone	Control	Sesame oil	Peanut oil	Melon oil
<b>LH</b>	7.38 ± 0.17			
5% oils		25.50 ± 7.07*	11.00 ± 0.07*	11.0 ± 0.35*
10% oil		37.50 ± 10.60*	11.63 ± 4.77*	10.0 ± 0.35*
20% oil		6.50 ± 0.71	6.00 ± 0.07	6.0 ± 0.07
<b>FSH</b>	13.00 ± 1.41			
5% oil		15.00 ± 1.41	14.00 ± 2.82	11.5 ± 0.71
10% oil		12.50 ± 0.70	15.95 ± 0.07	13.0 ± 1.41
20% oil		11.95 ± 0.07	11.95 ± 0.07	15.0 ± 1.41
<b>Testosterone</b>	0.71 ± 0.07			
5% oil		0.55 ± 0.071*	2.00 ± 0.00*	2.6 ± 1.70*
10% oil		0.20 ± 0.071*	1.75 ± 1.06	7.6 ± 6.22*
20% oil		0.75 ± 1.77	0.65 ± 0.071	0.53 ± 0.25

Values are Mean ± SEM; \**P* < 0.05 vs. control.

From the results, the LH and testosterone concentrations were significantly increased in animal groups fed 5 and 10% supplemented with the sesame, peanut and melon oils (*p* < 0.05). The 20% oils-supplemented diets-fed groups showed no significant effects on the levels of these hormones. The FSH was not significantly impacted by any of the three traditional oils fed as supplemented diets.

## DISCUSSION

The present investigation was carried out to assess the impact of traditionally extracted sesame, peanut and melon oils on sex hormones and hence fertility in Wistar rats. The result obtained, showed statistically significant improvement in biochemical markers of fertility in both males and females along with a comparative increase in the litter number (though not reaching statistical significance) in the animals fed with sesame oil-supplemented diet compared to peanut and melon oils, indicating an overall positive effect of the oil on fertility. It is likely that the phytoestrogens contained in the oil as earlier reported by Jacklin et al. (2003) may be responsible for this enhanced fertility action of the sesame oils in the experimental rats in a manner akin to animal estrogens. Yildiz (2005) had shown that sesamin, a major component of sesame oil contains phytoestrogen capable of producing estrogenic effect by binding to the estrogen receptors. Moreover, the assayed estradiol level was highly increased, about two to seven folds in the test diets-fed groups compared to the control, probably resulting from the effect of one or more of the phyto-components of the oils. Estrogens in general are known to promote mitotic activity in the uterine muscle and endometrium, cause rapid gene transcription in uterine

tissues and influence the secretion of the gonadotrophic hormones in the anterior pituitary (Deb, 2011). The combined effects of the increased estradiol and the phytoestrogen may have caused the increased litter size, hence enhanced fertility.

The plasma concentration of LH a typical gonadotrophic hormone of the anterior pituitary was also selectively and significantly raised in tandem with the litter size and the raised estradiol level in the oil-treated female rats. In females, an acute rise in LH and FSH triggers ovulation and development of the corpus luteum which may lead to multiple pregnancies while in the male, they stimulate leydig cell production of testosterone (Louvet et al., 1975). The high plasma LH in groups fed 5 and 10% oils supplemented diets (both male and female rats) may justify the observed multiple births. The oils might have triggered multiple ovulations and development of the corpus luteum in the female rats and production of testosterone from the leydig cells; combined mechanisms that work in synergy to enhance multiple pregnancies/births.

At the percentages supplemented, the oils exerted a null effect on the relative organ (heart, kidney and liver) weights implying that on the basis of weight alone, the oils have no detrimental effect on these organs and thus may be safe for consumption at the levels used in this study.

Prolactin is known to stimulate lactogenesis or milk production after birth (Bowen, 2002) and it decreases the level of sex hormones, estrogen in females and testosterone in males (Kumar and Clark, 2005). In this study, measured prolactin level was significantly high in test groups fed the 5 and 10% supplemented diets compared to the control implying that the seed oils may influence prolactin release. Prolactin being a peptide hormone, it is possible that the seed oils have a

modulatory effect on the prolactin release mechanism. Prolactin released, exerts a direct effect on the mammary gland via prolactin receptors which are located on the plasma membrane of the secretory cells thereby stimulating breast milk production. This seed oil-induced plasma prolactin increase may explain the mechanism by which milk production is increased and as such scientifically validates the claim that some seed oils particularly sesame oil; enhance the production of breast milk ([www.whyfood.com](http://www.whyfood.com)). It is also quite interesting and striking too, to note that in these treatment groups (groups fed 5 and 10% oils-supplemented diets) progesterone production was concomitantly decreased. Progesterone is secreted majorly by the corpus luteum of the ovary; it is responsible for preparing the inner lining of the uterus (endometrium) for pregnancy and prevents ovulation during this period (Chard, 1988); its concentration decreases with increased prolactin production (Lelte et al., 1992, Deb, 2011). This observed reciprocal action of the seed oils clearly corroborates our earlier submission on the impact of prolactin on breast milk production, as the oils simultaneously increased prolactin and decreased progesterone in favour of increased milk production. This effect on enhanced milk production mechanism was however not indicated at high supplementation levels (20% oils supplementation), suggesting that maximum efficacy in milk production is obtained only when the oils are used as minor component of the diet and not as a major component in which case cross interferences with other mechanisms may compromise the milk production enhancement function. The study oils by this mechanism have proven to be good agents for maximum lactation or increased milk production in lactating mothers. However, this requires further research to be firmly established.

In this study, testosterone, a hormone primarily secreted in Leydig cells of testes, responsible for the development of the male sex organs and secondary sexual characteristics such as physique, strength, sex drive and performance man (Waterman and Keeney, 1992) was significantly increased in experimental animals fed oils-supplemented diets. The observed increase in testosterone concentration suggests consumption of sesame oil and lower melon and groundnut oils at 5 and 10% may boost sex drive and enhance fertility and potency in male via increase in testosterone production. It is plausible to think that this testosterone increase may be an effect of the zinc component of the seed oils. Sesame oil is reportedly rich in zinc (Kanu, 2011), and previous studies have also shown that zinc rich-diets and diet supplementation with zinc are extremely effective at raising testosterone levels and helping men to improve their body composition, sex drive and increase semen volume (Shafiei et al., 2011). The oils-incorporated diets have by this testosterone and LH productions also shown possible capacity to positively influence male fertility. Further and comprehensive research is however needed to fully establish this.

Overall, data from this study suggest that the consumption of these seed oils at 5 and 10%, especially sesame oil,

may contribute effectively to increased incidence of multiple births, enhanced fertility in males and females as well as improves lactation.

## Conflict of interests

The authors did not declare any conflict of interest.

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Full Length Research Paper

## Plasma lipid profile including the high density lipoprotein (HDL) subclasses in hypertensive patients in Ouagadougou, Burkina Faso

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The study was undertaken to evaluate the interest of cholesterol subclasses in the management of hypertensive patients recruited at University Hospital Yalgado Ouedraogo of Ouagadougou (Burkina Faso, West Africa). The distribution of hypertensive was reported as 45 (35.4%) without complications, 42 (33%) with cardiovascular complications and 40 (31.4%) with diabetes. Any difference in lipids profile was observed when balanced hypertensive was compared to non-balanced hypertensive. The total cholesterol (TC), high density lipoprotein cholesterol (HDLC) and HDL3 cholesterol (HDL3C) were significantly higher in hypertensive compared to the control group ( $p < 0.001$ ). Significant decrease of TC and HDLC levels was observed in women within hypertensive group ( $p < 0.05$ ). The increase in triglyceride (TG) and low density lipoprotein cholesterol (LDLC) was significant in obese compared to non-obese. The HDLC level was higher ( $p < 0.01$ ) in treated hypertensive compared to untreated, particularly females. The HDLC increased significantly in treated hypertensive without complications ( $p < 0.01$ ). The TC and LDLC levels were higher in treated hypertensive with diabetes ( $p < 0.05$ ). The HDL2 cholesterol (HDL2C) was significantly lower in treated hypertensive with diabetes ( $p < 0.05$ ), and particularly in obese compared to non-obese. A significant decrease of HDL2C was observed in female stage 3 hypertensive ( $p < 0.05$ ). The HDL2C might be a better predictor of cardiovascular risks in hypertensive if the relationship between its decrease with severity of hypertension is confirmed by further studies.

**Key words:** Lipids profile, HDL subclasses, hypertensive.

### INTRODUCTION

Hypertension (HT) is one of the most important risk factors of cardiovascular diseases (Durakoğlugil et al., 2014). High blood pressure levels have been associated

with elevated atherogenic blood lipid (Saidu et al., 2014). Hence, screening for lipid abnormalities should be an essential part of management of hypertensive patients. It

is recommended to perform an annual screening for blood fasting lipids, including total cholesterol (TC), triglyceride (TG), high density lipoprotein cholesterol (HDL) and low density lipoprotein cholesterol (LDL) in hypertensive patients (McPherson et al., 2006). The HDL has long been considered the main biological marker of protection against the cardiovascular disease. However, many studies have reported cases of patients with low HDL without additional risk of cardiovascular events, and other increased risks, despite a high value of HDL (Joy et al., 2008). This atheroprotective role has been attributed to the HDL<sub>2</sub> fraction of HDL cholesterol, differentiated on the basis of its density ( $d = 1,063-1,125$  g / ml), whereas the HDL<sub>3</sub> fraction ( $d = 1,125-1,210$  g / ml) does not seem involved (Bakogianni et al., 2001; Moriyama et al., 2014). Therefore, the HDL<sub>2</sub> subfraction seems to be more atherosclerotic protective than the HDL<sub>3</sub> subfraction (McPherson et al., 2006). An inverse correlation has been established between the HDL<sub>2</sub> subfraction and atherosclerosis where an increase in the HDL<sub>2</sub> subfraction results in decrease in atherosclerosis (Maeda et al., 2012). Several methods of measurement of HDL subclasses have been proposed and many authors have demonstrated the value of knowledge of the HDL subclasses in the monitoring of metabolic diseases (Superko, 2009; Shuhei et al., 2010). This has shown that there could be an association between HDL<sub>2</sub> and atherosclerosis in hypertensive patients. This study was undertaken to further evaluate this association using the patients in Burkina Faso in order to build further the body of knowledge in this area. This study also aimed to validate a cost effective method for determining an effective threshold concentration of HDL<sub>2</sub> used in assessing cardiovascular risk. The authors propose that this will help Burkina Faso, a resource limited country, in preventing coronary heart diseases.

## METHODOLOGY

### Ethics statement

The study protocol and consent procedure were approved by the Burkina Faso National Ethics Committee for Research Ouagadougou, Burkina Faso approval number #2012-06-52 on the 7<sup>th</sup> June 2012. As required by the Helsinki declaration a written informed consent was obtained from all participants prior to conducting any study procedures. After consenting, personal and epidemiological data were collected and recorded. All the data used in this study were anonymous.

### Type and period of the study

This was a case-control study conducted between September to

December 2012 in Ouagadougou, the capital city of Burkina Faso in West Africa. The hypertensive subjects were recruited at University Hospital Yalgado Ouedraogo of Ouagadougou and the control group at the Regional Center of Blood Transfusion of Ouagadougou. All laboratory tests were performed at the University Hospital laboratory Yalgado Ouedraogo of Ouagadougou.

## Recruitment of study population

### Inclusion criteria

The study population comprised of consecutive hypertensive patients attending the cardiology department of University Hospital Yalgado Ouedraogo of Ouagadougou during the period of the study. The hypertensive patients were divided into 4 groups: Group 1: untreated hypertensive; patients; Group 2: treated hypertensive patients without complications; Group 3: treated hypertensive patients with complications (coronary artery diseases; peripheral vascular and cerebrovascular disease; left ventricular systolic dysfunction, nephropathy, retinopathy); Group 4: Treated hypertensive patients with diabetes; Group 5 comprised apparently healthy normotensive controls were recruited at the Regional Center of Blood Transfusion of Ouagadougou.

### Exclusion criteria

The hypertensive patients were excluded if taking drugs known to increase blood pressure like steroids and contraceptive pills or to modify the lipids such as statins, nicotinic acid, fibrates and resins. Pregnant women and subjects who are enlisted in any other concomitant study were also excluded.

## Biochemical analysis

After an overnight fast, venous blood was collected in a dry tube for biochemical analysis. Serum was separated by centrifugation at 3000 g for 10 min at 4°C, stored at -80°C and analyzed within a week. Serum total cholesterol (TC) and triglycerides (TG) were determined using an automated Spintech 240 Biolis 24j analyzer (Spintech, Barcelona, Spain) and the fully enzymatic methods (Spinreact kits Cholesterol-LQ reference TK41021 and Triglycerides-LQ reference TK41031). The dual-step precipitation of HDL subfraction was performed according to the procedure described by Hirano et al. (2008). To isolate total HDL by precipitation, a combined precipitant consisting of 100  $\mu$ l (0.02 mmol/L) of dextran sulfate (Mr 500000, SIGMA, France) and 25  $\mu$ l (200 mmol/L) of  $MnCl_2$  ( $MgCl_2 \cdot 6H_2O$ , MERCK, France) was added to 1 ml of serum. After 15 min of standing at room temperature, the mixture was centrifuged at 3,400 g for 20 min at 4°C. Aliquots of the resulting supernatant (S1) were taken for the assay of the HDL and precipitation of the HDL<sub>2</sub>. The HDL<sub>2</sub> was precipitated by a combined precipitant consisting of 100  $\mu$ l (0.02 mmol/L) of dextran sulfate (Mr 500000, SIGMA, France) and 50  $\mu$ l (200 mmol/L) of  $MnCl_2$  ( $MgCl_2 \cdot 6H_2O$ , MERCK, France) added to 500  $\mu$ l of supernatant (S1). After 2 hat room temperature, the mixture was centrifuged at 3,400 g for 20 min at 4°C. Aliquots of the resulting supernatant (S2) were taken for the assay of the HDL<sub>3</sub>. The measured value for total HDL was multiplied by 1.125 and that for HDL<sub>3</sub> was multiplied by 2.92 to correct for dilution by the

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**Table 1.** The accuracy and precision of the measurements and day to day coefficient of variations.

Parameter	Accuracy (n = 20)		Precision		
	Reference mean	Mean obtained	Student test calculated t	Intra-serial CV (n = 20)	Inter-serial CV (n=20)
CT	2.87	2.81	0.83	0.56	0.98
HDL-c	0.77	0.65	0.37	1.05	0.94
LDL-c	1.54	1.39	1.67	0.98	1.56
TG	1.23	1.16	0.80	1.39	0.67

TC= total cholesterol; HDL-c= HDL-cholesterol; LDL-c= LDL-cholesterol; TG=triglycerides; CV=coefficient of variations.

reagents. HDL3C was measured by the direct HDLC homogenous assay instead of the original TC assay. The sub-fraction HDL2C was calculated by the following formula: cholesterol HDL2C = HDLC - HDL3C. We calculated the LDLC (in mmol/L) by using the Friedewald formula: LDLC = TC – HDLC – TG/2.2 (Srisawasdi P et al., 2011).

### Quality control

Laboratory analysis was conducted by trained and competent personnel following standard laboratory procedures as stated above. The laboratory was enrolled in a National External Quality Assurance (EQA) program for cholesterol and HDL fractions determination with a frequency of 2 surveys per year. During the period of the experiment, the laboratory performance was within the stated acceptable limits of National EQA program. To ensure the accuracy and precision of the test results, internal quality controls were performed daily before analysis and Standard Deviation (SD) and coefficient of variations (CV) calculated. In Table 1 the accuracy and precision of the measurements during the study were within the acceptable criteria stated in literature (Thalameh et al.,1986; Vassault et al., 1999).

### Statistical analysis

Quantitative variables were expressed as means  $\pm$  standard deviation and qualitative variables in percentages. The Analysis of Variance (ANOVA) was used to determine quantitative variables with normal distribution, followed by the Bonferonni multiple comparisons test to compare the means between groups. The statistical analysis was performed using the statistical software PASW, version 18 for Windows (SPSS CPSC., Chicago, USA). Probability levels of 0.05 or less were considered significant.

## RESULTS

A total of 201 study subjects of which 158 (78.6%) were hypertensive with an average age of  $55 \pm 11$  years were studied (Table 2). The other 43 (21.4%), making the control group, were normotensive with an average age of  $51 \pm 8$  years. The 158 hypertensive patients, 53 (33.5%) and 105 (66.5%) were males and females respectively. Of the 43 in the control group, 24 (55.8%) were males and 19 (44.8%) females. There were significantly more females hypertensive than males hypertensive in the study group ( $p < 0.05$ ). The hypertensive patients were

significantly older than the controls (Control group mean =  $51 \pm 8$ ; hypertensive group mean =  $55 \pm 11$ ;  $p < 0.05$ ). The male hypertensive patients were older (mean =  $58 \pm 12$  years) than female (mean =  $53 \pm 10$  years). The body mass index (BMI) for hypertensive patients (Mean =  $27 \pm 6$  kg/m<sup>2</sup>) was significantly higher ( $p < 0.001$ ) compared to normotensive controls (Mean =  $21.7 \pm 3$  kg/m<sup>2</sup>). The prevalence of obesity in hypertensive patients was 57.6% (87.5% in females versus 12.5% in males). Among the 158 hypertensive patients, 127 (80%) were on antihypertensive therapy. The 127 treated hypertensive patients comprised 45 (35.4 %) without complications, 42 (33%) with cardiovascular complications and 40 (31.4 %) with diabetes. Both systolic and diastolic blood pressures (BP) for untreated hypertensive patients were significantly higher than those on treatment. According to the World Health Organization (WHO) classification of hypertensive stages of severity, the patients were divided into 3 groups: 42 (26.6%) in stage I; 33 (20.9%) in stage II; 16 (10.1%) in stage III and 67 (42.4%) were balanced.

The TC, HDLC and HDL3C (Table 3) were significantly higher in hypertensive patients compared to control group ( $p < 0.001$ ). Particularly, the increase in TC and HDLC was significant in women within hypertensive group ( $p < 0.05$ ).

Significant increase of the triglyceride and LDLC was observed in obese compared to non-obese and the decrease of HDLC and HDL3C was significant in obese ( $p < 0.05$ ), while an decrease in HDL2C was observed in obese males compared to non-obese males (Table 4).

The blood lipids profile in Table 5 shows that the HDLC level increased significantly ( $p < 0.01$ ) in treated hypertensive compared to untreated hypertensive, especially in females. The highest HDLC level was recorded in treated hypertensive without complications ( $p < 0.01$ ). The TC and LDLC levels were significantly higher in treated hypertensive with diabetes ( $p < 0.05$ ). In males, the decrease of HDL2C was significant in treated hypertensive with diabetes.

The study of lipid levels according to the stage of hypertension recorded in Table 6 shows only a significant decrease of HDL2C in stage 3 females hypertensive ( $p < 0.05$ ). No difference in lipids profile was observed between balanced hypertensive and non-balanced hypertensive.

**Table 2.** Demographic and clinical characteristics of the population.

Parameter		Non hypertensive n= 43 M 24 F 19	Hypertensive					
			Total n=158 M 53 F 105	Untreated hypertensive n=31 M=13 F=18	Treated hypertensive n=127 M=40 F=87	Treated hypertensive without complications n=45 M=9 F=36	Treated hypertensive with complications n=42 M=21 F 21	Treated hypertensive with diabetes n=40 M=10 F=30
Age(years)	Participants	51 ± 8	55±11	49±13	56±11	53±11	60±11	56±10
	Male	54 ± 9	58±12	53±14	60±12	54±14	62±13	60±7
	Female	48 ± 7	53±10	46±12	54±10	52±10	58±9	54±11
Sex	Male	24(55.8%)	53(34.6)	13(24,5%)	40(31,5%)	9(17%)	21(39%)	10(18,9%)
	Female	19(44.2)	105(66.4)	18(17,1%)	87(68,5%)	36(34,3%)	21(20%)	30(28,6%)
	Sex ratio	1.3	0.5	0.7	0.4	0.2	1	3
BMI (Kg/m <sup>2</sup> )		21.7±3	27±6	27±5	28±6	28±6	25±5	30±7
Waist circumference (cm)	Male	85±9	91±11	87±11	93±13	90±9	93±14	96±12
	Female	87±10	94±11	89±12	95±11	94±10	92±11	99±11
Blood pressure (mmHg)	Systolic	120±10	144±21	155±21	141±22	143±23	141±23	140±18
	Diastolic	85±10	84±10	93±13	83±10	85±10	82±10	80±10
Hypertension Stage	Stage 1	-	42(26.6%)	10(24%)	32(25%)	15(36%)	7(17%)	10(24%)
	Stage 2	-	33(20.9%)	8(24%)	25(20%)	9(27%)	8(24%)	8(24%)
	Stage 3	-	16(10.1)	8(50%)	8(6%)	3(19%)	4(25%)	1(6%)
	Balanced		67(42.4)	5(8%)	62(92%)	18(27%)	23(34%)	21(31%)

## DISCUSSION

The present study assessed plasma lipid profile among hypertensive subjects. The total cholesterol (TC) and HDLC increased significantly in hypertensive compared to control group. The increase of TC is common in hypertensive but generally a low HDLC is reported in hypertensive (Kanaya et al., 2003; Cai et al., 2012; Sun et al., 2014). The findings of higher mean HDLC among hypertensive patients when compared with normal controls was also documented in Nigeria (Okeahialam et al.,

2003; Karaye et al., 2008; Adamu et al., 2013; Saidu et al., 2014). Particularly, significant elevation of TC and HDLC levels was noted in women within hypertensive group. This can be explained by the number of postmenopausal women in the group (53 ± 10 years). This observation is consistent with other studies (Freedman et al., 2004; Ai et al., 2010; Skoczyńska et al., 2013) as well as with National Cholesterol Education Program report, ATPIII: prior to the age of menopause, females have lower total cholesterol levels than males of the same age. After menopause, however,

cholesterol levels tend to rise in women (Stone et al., 2005).

In this study, the relationship between dyslipidemia and the traditional cardiovascular risk factors including body mass index (BMI), waist circumference (WC) and blood pressure (BP) was studied. The triglyceride and LDLC increased in obese compared to non-obese and the decrease of HDLC was significant in obese. These findings are in agreement with those of previous studies (Kanaya et al., 2003; Cai et al., 2012; Wang et al., 2012; Sun et al., 2014).



**Table 3.** Lipids values of hypertensive group compared to non-hypertensive.

Lipids (mmol/L)		Hypertensive n=158 (M 53 F 105)	Non hypertensive n=43 (M 24, F 19)	p
Total Cholesterol	Population	5.39±1.20	4.69 ± 0.84	0.0004
	Male	5.11±1.18	4.71 ± 0.94	0.147
	Female	5.52±1.17	4.67 ± 0.72	0.04
	p	0.039	0.883	
Triglycerides	Population	1.24±0.65	1.10 ± 0.43	0.18
	Male	1.24±0.70	1.09 ± 0.40	0.27
	Female	1.25±0.58	1.10 ± 0.47	0.28
	p	0.90	0.965	
LDL cholesterol	Population	3.42±1.04	3.23 ± 0.8	0.26
	Male	3.26±1.03	3.26 ± 0.86	0.24
	Female	3.49±1.04	3.20 ± 0.74	
	p	0.189	0.821	
HDL cholesterol	Population	1.53±0.39	1.23 ± 0.3	0.00005
	Male	1.42±0.30	1.19 ± 0.27	0.002
	Female	1.57±0.40	1.28 ± 0.34	0.003
	p	0.017	0.331	
HDL <sub>3</sub> cholesterol	Population	1.02±0.23	0.76 ± 0.18	0.000001
	Male	0.99±0.22	0.76 ± 0.20	0.000003
	Female	1.03±0.22	0.76 ± 0.15	0.000001
	p	0.23	0.941	
HDL <sub>2</sub> cholesterol	Population	0.51±0.36	0.50 ± 0.28	0.86
	Male	0.44±0.30	0.45 ± 0.29	0.89
	Female	0.54±0.37	0.56 ± 0.28	0.81
	p	0.08	0.216	

**Table 4.** Comparison of Lipid levels between obese and non-obese.

Lipids (mmol/L)	Non obese (BMI <25 Kg/m <sup>2</sup> )			Obese (BMI ≥25 Kg/m <sup>2</sup> )		
	Total	Male	Female	Total	Male	Female
Triglyceride	1.10±0.5	1.06±0.5	1.14±0.5	1.50±0.92 <sup>a</sup>	1.72±1.2	1.29±0.64
Total Cholesterol	5.23±1.12	4.95±1.11	5.52±1.13 <sup>c</sup>	5.48±1.33	5.39±1.44	5.57±1.23
LDL cholesterol	3.24±1.15	3.1±1.00	3.39±1.03	3.60±1.09 <sup>a</sup>	3.66±1.09 <sup>b</sup>	3.55±1.09
HDL cholesterol	1.55±0.30	1.49±0.33	1.62±0.32	1.42±0.38 <sup>a</sup>	1.28±0.31 <sup>b</sup>	1.57±0.46
HDL <sub>2</sub> cholesterol	0.50±0.30	0.46±0.34	0.55±0.28	0.47±0.34	0.39±0.27 <sup>b</sup>	0.55±0.42
HDL <sub>3</sub> cholesterol	1.06±0.19	1.05±0.22	1.08±0.21	0.95±0.20 <sup>a</sup>	0.88±0.22 <sup>b</sup>	1.02±0.24

Significant difference (P<0.05) between <sup>a</sup>Non obese hypertensive and obese hypertensive; <sup>b</sup>male Non obese hypertensive and Male obese hypertensive; <sup>c</sup>male non-obese hypertensive and female non-obese hypertensive.

The blood lipids profile showed a significant elevation of the TC and HDLC in treated hypertensive compared to untreated and particularly in females. Moreover, the TC and LDLC levels were higher in treated hypertensive with diabetes. The dyslipidaemia seen in the Type 2 diabetes mellitus (T2DM) patients with hypertension could be due

to the effects of antihypertensive treatment. Indeed, in other studies, treatment of hypertension with b-blockers, as well as high doses of thiazide diuretics have been shown to exacerbate the dyslipidaemia in patients with hypertension and diabetes mellitus (Andrew and Clifford, 1994; Iaccarino et al., 2005).

**Table 5.** Blood Lipids profile of untreated hypertensive and treated hypertensive.

Parameters (mmol/L)		Untreated hypertensive n=31 M=13 F=18	Treated hypertensive n=127 M=40 F=37	Treated hypertensive without complications n=45 M=9 F=36	Treated hypertensive with complications N=42 M=21 F 21	Treated hypertensive with diabetes N=40 M=10 F=30
Triglyceride	Population	1.22±0.91	1.25±0.6	1.16±0.52	1.17±0.48	1.44±0.76
	Male	1.40±1.29	1.18±0.56	1.10±0.48	1.08±0.49	1.46±0.73
	Female	1.09±0.52	1.29±0.62	1.18±0.53	1.25±0.46	1.44±0.79
Total cholesterol	Population	5.19±1.20	5.44±1.22	5.47±1.31	5.04±1.14	5.83±1.11 <sup>h</sup>
	Male	5.32±1.00	4.98±1.27	4.83±1.35	4.71±1.22	5.7±1.14
	Female	5.10±1.35	5.65±1.16 <sup>f</sup>	5.62±1.28	5.38±0.97	5.88±1.11
LDL Cholesterol	Population	3.24±0.98	3.46±1.08	3.31±1.00	3.26±1.07	3.85±1.11 <sup>h</sup>
	Male	3.34±0.97	3.21±1.08	2.9±1.15	3.08±1.06	3.78±0.91
	Female	3.16±1.00	3.58±1.08	3.41±0.95	3.43±1.08	3.88±1.18
HDL Cholesterol	Population	1.38±0.37	1.57±0.40 <sup>d</sup>	1.61±0.47 <sup>h</sup>	1.54±0.39	1.56±0.34
	Male	1.43±0.40	1.44±0.32	1.47±0.26	1.48±0.38	1.32±0.15 <sup>g</sup>
	Female	1.34±0.35	1.63±0.42 <sup>e,f</sup>	1.64±0.50	1.6±0.39	1.65±0.35
HDL3 cholesterol	Population	0.98±0.23	1.04±0.23	1.01±0.22	1.04±0.23	1.07±0.25 <sup>h</sup>
	Male	1.05±0.29	0.99±0.21	0.93±0.14	1.03±0.23	0.95±0.23 <sup>g</sup>
	Female	0.92±0.17	1.06±0.24	1.02±0.23	1.05±0.22	1.1±0.25
HDL2 Cholesterol	Population	0.41±0.33	0.54±0.38	0.6±0.45	0.51±0.34	0.5±0.31
	Male	0.39±0.37	0.46±0.31	0.54±0.3	0.48±0.35	0.36±0.17 <sup>h</sup>
	Female	0.43±0.31	0.57±0.40	0.61±0.48	0.55±0.33	0.55±0.34

Significant difference (P<0.05) between <sup>D</sup>untreated hypertensive and Treated hypertensive, <sup>E</sup>female untreated hypertensive and female treated hypertensive, <sup>F</sup>male treated hypertensive and female treated hypertensive, <sup>G</sup>male treated hypertensive with diabetes and female treated hypertensive with diabetes, <sup>H</sup> treated hypertensive without complications and treated hypertensive with diabetes

**Table 6.** Lipids levels according to the grade of hypertension.

Parameters (mmol/L)		Balanced hypertensive	Non balanced hypertensive	Hypertensive		
				Stage 1	Stage 2	Stage 3
Triglyceride	Population	1.23±0.7	1.26±0.66	1.13±0.71	1.28±0.66	1.28±0.53
Total Cholesterol	Population	5.39±1.2	5.39±1.25	5.29±1.2	5.33±1.19	5.77±1.48
LDL Cholesterol	Population	3.46±0.98	3.39±1.13	3.28±1.09	3.38±1.13	3.69±1.24
	population	1.57±0.37	1.51±0.43	1.60±0.51	1.42±0.33	1.46±0.35
HDLCholesterol	Male	1.46±0.41	1.42±0.28	1.43±0.34	1.41±0.25	1.43±0.25
	Female	1.61±0.34	1.56±0.48	1.68±0.56	1.43±0.38	1.48±0.39
HDL2cholesterol	population	0.52±0.33	0.51±0.40	0.58±0.47	0.45±0.30	0.46±0.38
	Male	0.44±0.34	0.45±0.31	0.44±0.35	0.44±0.29	0.48±0.34
HDL3cholesterol	Female	0.55±0.32	0.54±0.44	0.64±0.51	0.46±0.32	0.45±0.41 <sup>i</sup>
	Population	1.05±0.21	1.01±0.24	1.02±0.25	0.98±0.24	1.05±0.25
	Male	1.01±0.2	0.99±0.24	0.99±0.21	0.97±0.23	1.12±0.41
	Female	1.06±0.22	1.01±0.24	1.04±0.27	0.98±0.25	1.03±0.15

Significant difference (P<0.05) between <sup>i</sup>Stage 1; Stage 2 and stage 3 hypertensive.

The measurement of cholesterol sub-fractions reported a significant increase of HDL3C in hypertensive compared to control group and HDL3C level was in accordance to

TC and HDLC values. This observation is consistent to over studies reporting that HDLC of hypertensive patients had a markedly increased relative content of HDL3C

while their HDL2C fraction was reduced by other 50% (Dobiasova et al., 1992). The HDL3C level was higher in treated hypertensive with diabetes and particular in males. This HDL3C elevation can be due to the hypertension effect on lipids profile and also to the treatment reported to increase HDL3C in patients on antihypertensive drugs as beta-blokers (Szollár et al., 1990).

The high density lipoprotein 2 (HDL2C) decreased in obese compared to non-obese. In males, the decrease in HDL2C was significant in treated hypertensive with diabetes. This decrease in HDL2C can be explained by the fact that the apoA1, major protein component of HDL2C, is reduced in type 2 diabetes (Van Linthout et al., 2010). According to the stage of hypertension, a significant decrease of HDL2C was observed only in stage 3 female hypertensive while the other lipids levels were not related to the severity of hypertension.

## Conclusion

The HDL2C might be a better predictor of hypertensive complications if the relationship between its decreases with hypertensive stage of severity is confirmed by further studies.

## Conflict of interests

The authors did not declare any conflict of interest.

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Full Length Research Paper

## Diabetes and pre-diabetes in adult Nigerians: Prevalence, and correlations of blood glucose concentrations with measures of obesity

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The quantification of the number of people with diabetes is an important factor that allows for effective planning and distribution of scarce resources for the management of diabetes. This study therefore investigated the prevalence of diabetes and impaired fasting glucose in a convenient population of adults in Umudike, a semi-urban town in South-East Nigeria. The relationship between measures of obesity and blood glucose concentration was also evaluated. Standard protocols were followed for all measurements, determinations and definitions. The results show that the prevalence of diabetes in the studied population is 3.0% (3.6% for females and 2.3% for males). Impaired fasting glucose was found in 1.1% of the population (females 1.6%; males 0.6%), such that 4.1% of the population had dysglycemia. Diabetic females had significantly ( $P < 0.02$ ) higher body mass index (BMI), fat mass, waist-to-height ratio (WHtR) and waist-to-hip ratio (WHpR) compared to non-diabetic female subjects. For the males, mean BMI and fat mass were statistically similar between the groups whereas WHtR and WHpR were significantly ( $P < 0.01$ ) higher in diabetics. The assessed measures of obesity (except for BMI in males) were weakly but significantly ( $r < 0.5$ ,  $P < 0.05$ ) correlated with blood glucose concentration in all subjects. The results are discussed and their public health significance highlighted.

**Key words:** Diabetes, dysglycemia, impaired fasting glucose, obesity, pre-diabetes.

### INTRODUCTION

Diabetes mellitus (DM) is an aetiologically multifactorial metabolic disorder, characterised by chronic hyperglycaemia. It results in aberrations in carbohydrate, fat and protein metabolism, which arise due to defects in insulin

secretion, and/or action. DM is currently a very prevalent disease, especially in Africa (Bos and Agyemang, 2013). Globally, at the end of 2013, as much as 382 million people had the disease; while the number is expected

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to reach 592 million by 2035. Interestingly, the burden of diabetes is highest in low and middle income countries (LMICs) (it is reported that in 2011, for instance, 14 million Africans had diabetes; and 80% of diabetics currently live in LMICs) (IDF, 2013).

LMICs will experience significant increases in the prevalence of the disease over the next 22 years (Guariguata et al., 2014). The increasing burden of DM in LMICs is thought to be related to the changes in physical activity (due to the availability of energy-sparing devices), dietary patterns (which are becoming westernised), and an improvement in life expectancy (Van Dieren et al., 2010). It is however worrisome that cases of undiagnosed diabetes are quite rampant in LMICs, reaching 50% (Chow et al., 2006) to 75% (IDF, 2009) of all cases.

Owing to the fact that one important factor (in diabetes management) that allows for effective planning and distribution of scarce resources is the quantification of the number of people with diabetes (Soriguer et al., 2012), this study investigated the prevalence of diabetes and impaired fasting glucose concentration in a convenient sample of a sub-urban adult population in Nigeria, and assessed the relationship between fasting blood glucose concentrations and measures of obesity. The findings are expected to be useful in guiding future research efforts and public health policy formulation and action.

## MATERIALS AND METHODS

Adult subjects in Umudike, a University town in Abia State, South-East Nigeria, aged 18 years and older, were approached and the goals of the study explained to them. Those who gave an informed consent were then recruited. Exclusion criteria included overt or reported ill-health and pregnancy or recent delivery (in women). A total of 365 subjects (52.9% females) participated in this cross-sectional study.

Self-reported age and diabetic status were recorded per subject while weight, height, waist and hip circumferences were measured using standard protocols as described previously (Ejike and Ijeh, 2012). From the above variables, body mass index (BMI), waist-to-hip ratio (WHpR), and waist-to-height ratio (WHtR) were calculated using standard equations. Fat mass (percent) was measured using a bio-electrical impedance analysis (BIA) machine (Omron BF-400, Omron Healthcare Europe BV, Hoofddorp, The Netherlands). Fasting capillary blood glucose concentrations of the subjects who had fasted for at least 12 h (overnight) were determined using a glucometer (Accu-check Advantage, Roche Diagnostics, Mannheim, Germany).

Diabetes was diagnosed as a fasting blood glucose concentration of  $\geq 126$  mg/dL ( $\geq 7$  mmol/L), or self-reported use of glucose lowering medication. Impaired fasting glucose (IFG) was defined as a fasting blood glucose concentration between 110 and 125 mg/dL (6.1-6.9 mmol/L) (WHO, 2006). Subjects with either diabetes or IFG were regarded as having dysglycemia. Where necessary, subjects were stratified into four dissimilar age ranges thus: 18 to 30 years, 31 to 45 years, 46 to 60 years, and more than 60 years, for convenience purposes.

This study was carried out between the months of August and September 2014. The design for this study, which is in accordance with the Helsinki declaration, was approved by the Board of the

Department of Biochemistry, Michael Okpara University of Agriculture, Umudike since the University is yet to constitute a Human Experiments Ethics Review Board.

## Statistical analysis

Descriptive statistics and frequency counts were done on the data generated and the results reported as means  $\pm$  standard deviations and percentages, respectively. Pearson's correlation coefficients were calculated to assess the correlation between blood glucose concentration and measures of obesity. Differences between group means were separated using one-way analysis of variance (ANOVA), with the significant threshold fixed at  $P < 0.05$ . Data analysis was carried out using the statistical software IBM-SPSS version 20.0 (IBM Corp., Atlanta, GA) while graphs were plotted using Microsoft Excel (Microsoft Corp., Redmond, WA).

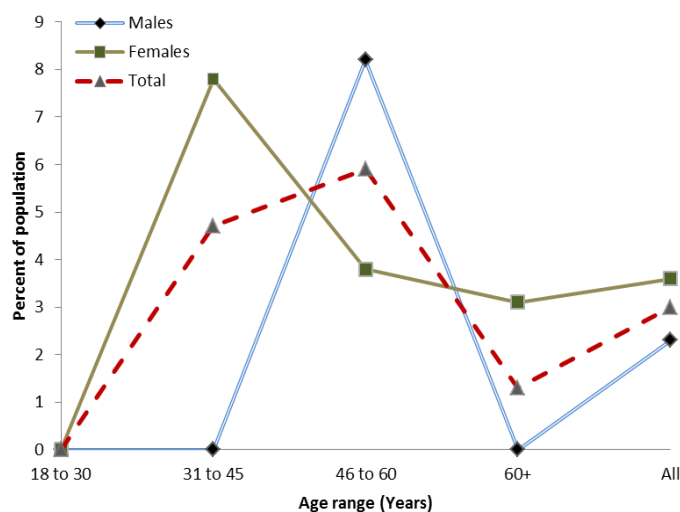
## RESULTS

The prevalence of diabetes in the studied population was 3.0% (3.6% for females and 2.3% for males). Females had an earlier onset of the disease (7.8% in the 31 to 45 years group) compared to males (0.0% in the same age group). Figure 1 shows the distribution of the prevalence of diabetes stratified by age and sex of respondents. IFG was found in 1.1% of the population (females 1.6% aged 20 to 41 years; males 0.6% aged 26 years). From Figure 2, it is seen that 4.1% of the population (5.2% for females and 2.9% for males) had dysglycemia. The distribution of the state by sex and age group is also shown in the figure.

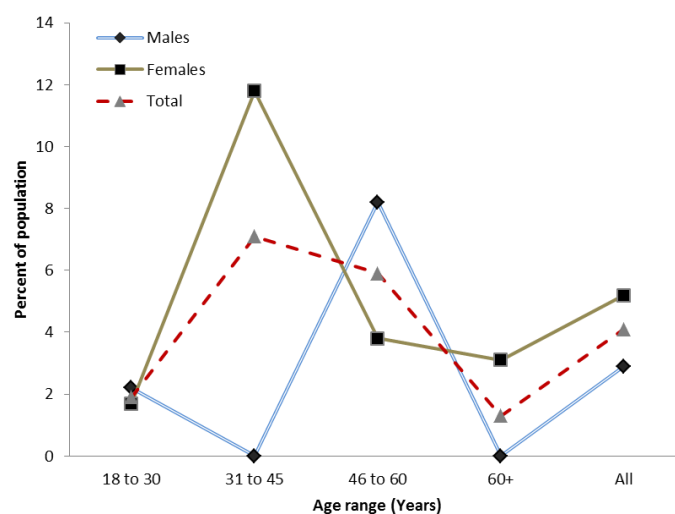
All the diabetic males had prior knowledge of their status while blood glucose concentrations were lower in females of known diabetic status compared to those who had no prior knowledge of their status. No female diabetic, younger than 46 years knew of her status. IFG was found only in those aged 18 to 45 years (Table 1). Diabetic females had significantly ( $P < 0.02$ ) higher BMI, fat mass, WHtR and WHpR compared to non-diabetic female subjects. For the males, mean BMI and fat mass were similar between the groups; whereas, WHtR and WHpR were significantly ( $P < 0.01$ ) higher in diabetics. Irrespective of sex, all the measures of obesity were significantly ( $P < 0.01$ ) higher in diabetics relative to non-diabetics (Table 2). The assessed measures of obesity (except for BMI in males only) were weakly but significantly ( $r < 0.5$ ,  $P < 0.05$ ) correlated with blood glucose concentration in all subjects. BMI in males was not significantly ( $P > 0.05$ ) correlated with BGC (Table 3). No significant correlation was found between lean mass and BGC.

## DISCUSSION

The prevalence of diabetes and dysglycemia reported in this study [3.0% (3.6% for females and 2.3% for males) and 4.1% (5.2% for females and 2.9% for males),



**Figure 1.** Prevalence of diabetes mellitus stratified by sex and age range.



**Figure 2.** Prevalence of dysglycemia (diabetes mellitus + impaired fasting glucose) stratified by sex and age range.

respectively] are within the reported ranges in Nigeria. Chinenye et al. (2008) noted that the prevalence of diabetes in Nigeria ranged from 0.65% in rural Mangu village to 11.0% in Lagos, an urban centre. Recently, Enang et al. (2014) reported a prevalence of 6.5% for diabetes in Calabar, another urban centre. It is understandable that, given the contributions of lifestyle modifications in the aetiology of chronic diseases, the prevalence of diabetes is higher in urban areas compared to rural areas. This may also explain the figure reported in this study as the setting is a semi-urban area. Arguments about the variations being due to fewer studies in rural areas and lower access to healthcare facilities in those areas are nonetheless plausible.

The higher prevalence of diabetes (3.0%) compared to impaired fasting blood glucose (1.1%) may be indicative of a rapid progression of the disease. A slower progression would have ensured that those with impaired blood glucose homeostasis would be more than those with the full blown disease. The design of the present study however forecloses any emphatic deduction in the direction of disease progression. Moreover, the use of capillary blood for this study may have had an impact on the data. The female preponderance of diabetes found in this study is not an isolated finding. Prior to this report, Ohwovoriole et al. (1998), Chinenye et al. (2008) and Enang et al. (2014) had reported a female preponderance of diabetes in Nigeria. Such preponderance was not however found in some other studies in Nigeria (Okoro et al., 2002; Rotimi et al., 2004). Though male *homo sapiens* are larger than female *homo sapiens*, females often have more obesity-related challenges arising likely from their proportionally larger visceral adipose tissue. This coupled with variations in steroid

hormones that may affect metabolism, may be responsible for this female preponderance of diabetes reported here and elsewhere.

Our finding that diabetics were younger than 61 years is consistent with other reports from Nigeria (Chinenye et al., 2008; Enang et al., 2014); whereas in economically more developed countries, diabetics are usually older than 60 years. In the majority of countries in Europe for instance, the prevalence of diabetes is less than 10% in people younger than 60 and 10 to 20% in people aged 60 to 80 years (DECODE-Study-Group, 2003). Guariguata et al. (2014) summarised this scenario nicely when they reported that for the year 2013, “people with diabetes in high-income countries are predominantly over the age of 50 (74%) while those in low- and middle-income countries are mostly under the age of 50 (59%)”. This disparity may be explained by the poor state of healthcare in low- and middle-income countries, resulting in higher mortality rates and probably an earlier onset of the disease in such countries where the nutrition transition is already taking place. Whatever the explanation is, it does not bode well for these countries especially as economic development comes with improved life expectancy and an increased burden of non-communicable diseases. If the healthcare infrastructures in these countries are not strengthened now, the challenges of the future may be too much for them to cope with.

Many of the diabetics found in this study were not aware of the condition and were naive to treatment. Such high prevalence of undiagnosed diabetes (and other chronic diseases) is considerably common in low- and middle-income countries (Elbagir et al., 1996; Chow et al., 2006; Bo and Agyemang, 2013) where the cost of

**Table 1.** Concentrations of blood glucose in the subjects.

Subject		Blood glucose concentrations (mg/dL)				
		18 - 30 years	31 - 45 years	46 - 60 years	> 60 years	All
KD	Male	-	-	324 ± 53	-	324 ± 53
	Female	-	-	127 ± 21	-	127 ± 21
	Total	-	-	179 ± 83	-	179 ± 83
UKD	Male	-	-	-	-	-
	Female	-	151 ± 33	205 ± 49	-	183 ± 48
	Total	-	151 ± 33	205 ± 49	-	183 ± 48
IFG	Male	121 ± 0	-	-	-	121 ± 0
	Female	119 ± 0	113 ± 1	-	-	115 ± 4
	Total	120 ± 1	113 ± 1	-	-	117 ± 4
ND	Male	83 ± 11	88 ± 10	88 ± 10	86 ± 10	86 ± 10
	Female	84 ± 10	87 ± 10	89 ± 10	84 ± 11	86 ± 10
	Total	83 ± 11	87 ± 10	88 ± 10	85 ± 10	86 ± 10

KD, UKD, IFG and ND represent known diabetes, unknown diabetes, impaired fasting glucose and no diabetes, respectively. The distribution of the population by age range is: 18-30 years, 104 (58 females, 46 males); 31-45 years, 85 (51 females, 34 males); 46-60 years, 101 (52 females, 49 males); > 60 years, 75 (32 females, 43 males).

**Table 2.** Variations in measures of obesity between diabetic and non-diabetic subjects.

Subject	Diabetic				Non-diabetic			
	BMI (kg/m <sup>2</sup> )	FM (Kg)	WHtR	WHpR	BMI (kg/m <sup>2</sup> )	FM (Kg)	WHtR	WHpR
Female	30.1 ± 3.8	30.7 ± 6.6	0.61 ± 0.07	0.94 ± 0.07	25.2 ± 4.7	20.6 ± 8.6	0.52 ± 0.08	0.86 ± 0.08
P (F vs. M)					(0.019)	(0.001)	(0.001)	(0.001)
Male	26.6 ± 1.1	19.0 ± 4.9	0.59 ± 0.00	0.96 ± 0.02	23.5 ± 6.1	12.2 ± 5.9	0.46 ± 0.00	0.84 ± 0.06
P (F vs. M)					(0.270)	(0.118)	(<0.001)	(<0.001)
Total	28.8 ± 3.5	27.2 ± 9.8	0.60 ± 0.06	0.95 ± 0.06	24.4 ± 5.5	16.8 ± 8.4	0.49 ± 0.08	0.85 ± 0.07
P (F vs. M)					(0.009)	(<0.001)	(<0.001)	(<0.001)

F, M, BMI, FM, WC, HC, WHtR and WHpR represent female, male, body mass index, fat mass, waist circumference, hip circumference, waist-to-height ratio and waist-to-hip ratio, respectively.

**Table 3.** Correlations between blood glucose concentrations and anthropometric indices/measures of obesity.

Subject		Correlations [r (p)]							
		Height	Weight	BMI	FM	WC	HC	WHtR	WHpR
BGC	Females	-0.043 (0.553)	+0.258 (<0.001)	+0.282 (<0.001)	+0.264 (<0.001)	+0.333 (<0.001)	+0.235 (0.001)	+0.317 (<0.001)	+0.281 (<0.001)
	Males	-0.040 (0.600)	+0.018 (0.812)	+0.037 (0.634)	+0.116 (0.140)	+0.381 (<0.001)	+0.234 (0.002)	+0.376 (<0.001)	+0.327 (<0.001)
	All	-0.016 (0.759)	+0.090 (0.085)	+0.105 (0.044)	+0.124 (0.020)	+0.299 (<0.001)	+0.192 (<0.001)	+0.265 (<0.001)	+0.271 (<0.001)

BMI, FM, WC, HC, WHtR and WHpR represent body mass index, fat mass, waist circumference, hip circumference, waist-to-height ratio and waist-to-hip ratio, respectively.



regular medical check-ups may be out of reach of ordinary people and poor illness concepts are widespread (Ejike, 2014). The ratio of known to newly diagnosed diabetes is a good indicator of the level of diabetes awareness in a population. For this population, there is a need for aggressive public health education especially with respect to diabetes and related chronic diseases.

We found that measures of obesity were significantly higher in diabetics compared to non-diabetics (except for BMI and fat mass in males). The assessed measures of obesity (except for BMI in males) were weakly but significantly ( $r < 0.5$ ,  $P < 0.05$ ) correlated with blood glucose concentration in all subjects. It has been reported that the increase in the prevalence of diabetes is interwoven with the upsurge in obesity in affected regions. About 90% of type 2 diabetes is known to be attributable to excess adiposity (Hossain et al., 2007). The absence of correlation between BMI and blood glucose concentration in males may be as a result of higher muscle mass in the males. BMI as an instrument is known to be unable to differentiate sufficiently between bone, muscle and fat mass, such that an individual may be obese by BMI standards yet post normal metabolic profile as seen in the metabolically-healthy-obese phenotype reported in Nigeria (Ejike et al., 2009; Ijeh et al., 2010). Interestingly, no significant correlation was found between lean mass and fasting BGC in either males or females.

Furthermore, patients with a low WHpR rarely have diabetes, irrespective of BMI, indicating the role of visceral fat in diabetes. Obesity comes with increased visceral tissue mass. Increased visceral obesity results in increased free fatty acids which in turn induces oxidative stress, inflammation and insulin resistance. Since insulin resistance results in hyperinsulinemia, adipocyte hormone sensitive lipase under such conditions ensures sustained lipolysis and an increased free fatty acid concentration which in turn perpetuates the cycle. Due to the chronic nature of obesity, it causes a tonic low grade activation of inflammation; and the attendant sustained insulin resistance, through the afore-discussed and other pathways, ultimately results in diabetes mellitus (Bloomgarden, 2000; Dandona et al., 2005; Lumeng and Saltiel, 2011; Watson, 2014). This relationship explains the complicity of obesity in diabetes and the correlations reported in this study.

This study is limited by the small sample size which was necessitated by the limited funds at our disposal, cultural belief systems that make people scared of procedures that require their blood, and lack of honoraria paid to participant. The sample size is nonetheless sufficient to highlight the challenges of diabetes in resource-poor settings typified by the semi-urban location we studied. The sampling method used may have also introduced some bias, as it is possible that those who suspected they had problems with glucose homeostasis may have more readily volunteered to be part of the study.

The use of capillary blood glucose for this study is yet another limitation. Capillary blood glucose is known to have a wider coefficient of variation compared to venous plasma (Anjana et al., 2011). However, logistical constraints such as the limited funds and cultural encumbrances mentioned earlier, lack of phlebotomists, and the challenges of proper transportation and storage of samples, foreclosed the used of venous blood for this study. This limitation is however attenuated by the report of Priya et al. (2011) which shows that capillary blood glucose compares well with venous blood glucose in studies like ours. Again, diabetes and IFG would have been better diagnosed if the oral glucose tolerance test was administered to the subjects with elevated fasting blood glucose concentrations. This calls for caution in the interpretation of diabetes prevalence data and data for those with IFG (a fraction of who may be reclassified as having diabetes if the OGTT was performed).

In conclusion, this study assessed the prevalence of diabetes and pre-diabetes in a convenient sample of adult Nigerians and examined the correlation between blood glucose concentration and measures of obesity. The prevalence of diabetes was found to be 3.0% (3.6% for females and 2.3% for males). Except for BMI in males, the assessed measures of obesity were all weakly but significantly ( $r < 0.5$ ,  $P < 0.05$ ) correlated with blood glucose concentration in all subjects. Public health action is needed to educate the masses about diabetes, especially the need for early diagnosis and the development of healthy lifestyles that may prevent or delay the onset of the disease.

### Conflicts of interests

The authors declared that there is no conflict of interest.

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