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Pitout JDD, Church DL, Gregson DB, Chow BL, McCracken M, Mulvey M, Laupland KB (2007). Molecular epidemiology of CTXM-producing Escherichia coli in the Calgary Health Region: emergence of CTX-M-15-producing isolates. Antimicrob. Agents Chemother. 51: 1281-1286.

Pelczar JR, Harley JP, Klein DA (1993). Microbiology: Concepts and Applications. McGraw-Hill Inc., New York, pp. 591-603.

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Full Length Research Paper

# Evaluation of vegetable consumption in South Eastern Nigeria

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Vegetable consumption in the South East of Nigeria was evaluated with the objective of identifying the effects of cost and availability of vegetables in the areas and adequacy of consumption. A market survey was done in 9 markets spread across the zones including Anambra, Enugu and Ebonyi States. Questionnaires were used to assess 24 h vegetable consumption. The quantity of vegetable consumed was estimated indirectly from the market cost. The results show that the highest vegetable diet consumed in area is mixed vegetable soup (170.6 g/meal), followed by mixed okra/"ugu" soup (154 g/meal). The results showed that cost and availability do not affect vegetable consumption, and that the quantity of vegetable consumed in the area is lower than 400 g/day as recommended by World Health Organization (WHO). It was recommended that nurses and health educators who work with homemakers (mothers) should sensitize them on the need for increase in vegetable consumption.

Key words: Vegetable, consumption, cost, availability.

#### INTRODUCTION

Vegetables are edible parts of plants that are consumed whole or in parts, raw or cooked as part of main dish. The tropical and sub-tropical countries of the world are blessed with varieties of vegetables some of which are domesticated, while others grow wild and their prices are relatively affordable when compared with other food items in the areas (Uzueqbu and Eke, 2000; Okaka et al., 2010; Schippers, 2000). It is estimated that up to 2.7 million lives could potentially be saved each year if fruits and vegetables consumption are sufficiently increased (WHO, 2002). The health benefits of diet rich in vegetables have also been recognized and there is evidence that nutrient content of fruits and vegetables such as dietary fibre, folate, antioxidants, vitamins and phytochemicals are associated with low risk of cardiovascular diseases (Strandhagen, 2000; WHO, 2002).

Vegetables have a low glycaemic index and foods of

low glycaemic index are associated with low risk of type II diabetes and coronary heart disease, prolonged satiety responses which leads to weight control (Joffe and Robertson, 2001; WHO, 2003). In the last four decades, there are indications of partial shifts from staple food towards vegetable, oil and sugar in low and lower middle income countries like Nigeria while there is a shift toward vegetable, oil and meat in higher income countries (FAO/WHO, 2004). WHO recommended that the consumption standard quality for vegetable is 80 g/meal and a total of 400 g/day. The actual guantity consumed tends to be less, and it was recommended that vegetable intake should be flexible and adequate in local circumstances, because of wide variability of dietary pattern, vegetable availability, food preferences and cultural consideration of food. Countries were advised to set up vegetable promotion initiative with the aim of increasing intakes to

140 g serving vegetable per day for children and up to 400 g serving per day for adults (WHO, 2005).

The effort of vegetable promotion initiatives is to meet vegetable consumers' needs and preferences, increasing consumer awareness of the benefits of diets rich in vegetables, diversification of vegetable production, understanding consumer expectation in taste, texture, form, price convenience, quality and safety attributes (Ndie, 2010). Promoting the consumption of vegetable for health reasons also implies a need to improve vegetable supply and distribution systems to ensure their safety and qualities.

Research has shown that vegetable consumption is low is such Saharan Africa (27 to 114 kg/capita/year) which is below the WHO/FAO recommendation of 146 kg/capita/year (WHO, 2005). This study is aimed at identifying the consumption pattern of vegetable as it relate to availability and cost in the South Eastern Nigeria. South Eastern Nigeria is located at the equatorial rain forest of West Africa and is predominantly occupied by the Igbos.

#### Objectives of the study

The objectives of this study are as follows:

1) To identify the commonly consumed vegetables in South Eastern Nigeria.

2) To evaluate the effect of cost and availability of vegetables on the consumption pattern.

3) To assess the adequacy of vegetable consumption in the states.

#### **Research questions**

The research questions of the study are as follows:

1) What are the commonly consumed vegetables in South Eastern Nigeria?

2) What are the effect of cost and availability of vegetables on the level of consumption?

3) Is vegetable consumption of the people of South Eastern Nigeria adequate?

#### METHODOLOGY

#### **Research design**

A survey research design was used to elicit information on the cost and consumption pattern of vegetable in South Eastern Nigeria.

#### Population and sampling method

Simple random sampling method was used to select Anambra, Enugu and Ebonyi from five states of South Eastern States of

Nigeria. A convenient sampling method was used to select 10 households from each of the three senatorial zones of the three states. A total of 90 households representative samples were used for each state.

#### Estimate of vegetable consumption

The quantity of vegetables consumed by the individuals in the family was estimated indirectly using the following method.

#### Market survey

The weight of the edible part of different vegetables costing one hundred Naira (N100) were gotten from main markets of the nine senatorial zones of the three states: Anambra, Enugu and Ebonyi. The market survey was done from January to December, 2009.

The homemakers' (mostly mothers) in the 90 selected families in each state were asked the cost of various vegetables they use in preparing different foods for their families, from these costs the weight of the vegetable consumed were estimated by dividing the weight by the number of plates served from the food prepared.

#### Vegetable and recall

Vegetable food (24 h) recall was conducted using questionnaires to collect information on vegetable contained in the food served to the family within 24 h from the homemakers. The questionnaires were based on United Kingdom National Diet and Nutrition Survey (UK NDNS, 2002) modified to suit the environment and was first used in a pilot study. The amended copy was used for the study.

#### Statistical analysis

Data were collected and analyzed statistically using Statistical Packages for Social Sciences (SPSS) computer package. Calculation of mean vegetable consumption per day was also obtained.

#### RESULTS

The commonly consumed vegetables and their 24 h recall frequency in South Eastern Nigeria is shown in Table 1. The result show that *Telfairia occidentalis* (ugu leave), Vernonia amygdalina (bitter leave), Pterocarpus soyauxii (ora), Amaranthus species (Green Amalant) and Abelmoschus esculentus (okra) are with consumption rate of 68 to 86% daily in South Eastern Nigeria. The other vegetables that are consumed in the area at moderate rate are "Uzuza" (25 to 35%, Solanum melongena (anara leave), curry and "nchuanwu". "Okazi" and water leaves are consumed sparingly (11 to 18%). The means fluctuation in cost and consumption of vegetable in South Eastern Nigeria in 2009 is shown on Table 2. The results show that the cost of vegetables is low between April and September which corresponds to the rainy season and high between October and March which corresponds to the dry season. The only exception to this pattern of cost is *P. soyauxii* (ora) which is cheap between October and March. The results also show that

| Veretekle                              | Ana | mbra | En | ugu  | Ebo | onyi |
|--|-----|------|----|------|-----|------|
| vegetable                              | n   | %    | n  | %    | n   | %    |
| Telfairia occidentalis (ugu)           | 78  | 86.6 | 75 | 83.3 | 72  | 80   |
| Vernonia amygdalina (bitter leave)     | 78  | 86.6 | 72 | 80   | 72  | 80   |
| <i>Pterocarpus soyauxii</i> (ora)      | 75  | 83.3 | 72 | 80   | 72  | 80   |
| Amaranthus spp. (green amaranth)       | 72  | 80   | 70 | 78   | 70  | 78   |
| Abelmoschus esculentus (okra)          | 61  | 68   | 66 | 73   | 63  | 70   |
| Uzuza                                  | 35  | 39   | 32 | 35   | 29  | 32   |
| <i>Solanum melongena</i> (Anara leave) | 27  | 30   | 25 | 28   | 27  | 30   |
| Water leave                            | 15  | 17   | 11 | 13   | 14  | 15   |
| Curry leave                            | 27  | 30   | 29 | 32   | 27  | 30   |
| Nchuanwu                               | 29  | 32   | 27 | 30   | 27  | 30   |
| Okazi                                  | 14  | 15   | 18 | 20   | 11  | 13   |

Table 1. Commonly consumed vegetable and their consumption frequency (%) in South East Nigeria per day.

Table 2. Mean fluctuations in cost and consumption by weight/meal of vegetable in South East Nigeria.

|  | JanMarch         |               | April-June       |               | July-S           | ept.          | OctDec.          |               |
|--|------------------|---------------|------------------|---------------|------------------|---------------|------------------|---------------|
| Vegetable                              | Cost<br>(g/N100) | Gram/<br>meal | Cost<br>(g/N100) | Gram/<br>meal | Cost<br>(g/N100) | Gram/<br>meal | Cost<br>(g/N100) | Gram/<br>meal |
| <i>Telfairia occidentalis</i> (ugu)    | 450              | 73.3          | 500              | 80            | 800              | 80            | 850              | 76            |
| Vernonia amygdalina (bitter leave)     | 150              | 78.3          | 450              | 80            | 500              | 80            | 250              | 78.3          |
| <i>Pterocarpus soyauxii</i> (ora)      | 400              | 71.7          | 100              | 70            | 80               | 70            | 300              | 72            |
| Amaranthus spp. (green amaranth)       | 500              | 84.3          | 800              | 80            | 1000             | 89            | 600              | 84            |
| Abelmoschus esculentus (okra)          | 150              | 60            | 160              | 61            | 500              | 65            | 500              | 61            |
| Uzuza                                  | 20               | 5.8           | 20               | 5.8           | 50               | 6.1           | 30               | 5.5           |
| <i>Solanum melongena</i> (Anara leave) | 75               | 46.3          | 74               | 45.8          | 74               | 45.8          | 76               | 46            |
| Water leave                            | 100              | 62            | 1800             | 63            | 2000             | 64            | 1500             | 62            |
| Curry leave                            | 25               | 5.5           | 30               | 5.5           | 50               | 5.5           | 30               | 5.5           |
| Nchuanwu                               | 25               | 10            | 25               | 10            | 50               | 9.4           | 40               | 9.8           |
| Okazi                                  | 40               | 30            | 50               | 35            | 50               | 35            | 40               | 35            |

**Table 3.** Main vegetable diet of South Eastern Nigeria.

| Type of diet        | Vegetable                   | Gram/meal |
|---------------------|-----------------------------|-----------|
| Bitter leave soup   | Bitter leave                | 84.9      |
| Ugu soup            | Ugu/Uzuza                   | 70.1      |
| Ora soup            | Ora                         | 84.00     |
| Mix vegetable soup  | Ugu/Okazi/Water leave/Uzuza | 170.6     |
| Mix okra/ugu soup   | Okra/Ugu/Uzuza              | 154.1     |
| Boil yam/green      | Green amaranth              | 80.1      |
| Rice/green amaranth | Green amaranth              | 81.1      |
| Tapioca/Anara leave | Anara leave                 | 48        |

the cost of vegetable varies.

The results show that water leave is the cheapest (1000 to 2000 g/N100) in the states followed by *Amaranthus* spp. (green amaranth, 500 to 1000 g/N1000). Most consumed vegetable by weight per meal is *Amaranthus* spp. (80 to 89 g/meal). This is followed by *T. occidentalis* (78.25 to 80 g/meal), veronica amygdaline

(78 to 80 g/meal) and the least consumed are curry and "Uzuza" (5 to 10 g/meal). The results show that the consumption rate increases as the cost is lowered but this increase is not statistically significant ( $P \ge 0.05$ ).

The common vegetable diet of South Eastern Nigeria is shown in Table 3. The results show that the highest quantity of vegetable is in mix vegetable soup (170.6/meal) followed by mix okra/ugu soup (154 g/meal). The least is "tapioca/anara" leave (48 g/meal).

#### DISCUSSION

It may be inferred from this study that the quantity of these vegetables consumed are constant irrespective of the change in the cost. This may be explained by the fact that there is a maximum quantity needed for a particular size of the diet being prepared, and when this quantity is not reached or exceeded, the taste of the food is affected negatively (Ndie, 2010). This study indicates that the low consumption of vegetable is not due to low production or high cost as stated by Hodder (2004) and Bondoin (2004), because even when the vegetable is cheap and available the consumption rate did not increase significantly. This phenomenon probably informed WHO's fear that some countries that presently consume less than the recommended 400 g/day may never increase their consumption rate to meet this recommendation level even if availability of vegetable is increased and the cost reduced.

The main vegetable diet of South Eastern Nigeria is shown in Table 3. The results show that South East diet is low in vegetable intake per day. For example, an individual who consumed a mixed vegetable soup for lunch and boiled rice or yam with vegetable at dinner has consumed 250 g per day. This is still lower than 400 g/day recommended by WHO (2005).

It may be concluded from this study that varieties of vegetable are available all the year round in the South Eastern States of Nigeria and at affordable price too. The availability and cost do not influence significantly the quantity of these vegetables consumed per meal over the year. This may be due to the fact that the quantity of vegetables in a diet as a recipe is pre-determined by the type of diet. The results show that vegetable is added mostly in soups in South Eastern diet. To encourage vegetable consumption in the area, the people should be encouraged to increase their soup consumption rather than the accompanying pounded yam or garri or cassava fufu. Consumption of mix vegetable soup should also be encouraged.

#### Conclusion

Availability and cost do not significantly affect the consumption of vegetable since the quantity that needs to be added into a particular diet is predetermined by the quantity of the dish being prepared and taste attached to the diet by cultural food habit.

#### RECOMMENDATION

It is recommended that the homemakers in the area should be advised to increase the amount of soup ration served to the family to help improve the quantity consumed.

#### IMPLICATION TO NURSING

Nurses who understand the need of vegetable in diet and who from time to time are called to give health education to homemakers (mothers in Antenatal clinics and other maternity services as well as infant welfare clinics) should use these findings to educate mothers on the need for increase in vegetable consumption since the vegetables are available and also affordable in South East, Nigeria.

#### LIMITATION OF THE STUDY

A major limitation of the study was the sample size which limited the generalization of the result when compared with the populations of the households in the area.

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Full Length Research Paper

# Protective effect of vitamin C supplementation on oxonate-induced hyperuricemia and renal injury in rats

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Recent studies have suggested a potential direct role of mild hyperuricemia in development of chronic kidney disease independent of urate crystal formation. The present study was designed to investigate the possible anti-hyperuricemic and renoprotective effect of vitamin C, as a natural antioxidant with uricosuric property on a rat model with chronic mild hyperuricemia-induced nephropathy. A model of mild hyperuricemia was induced in male Wistar rats with an uricase inhibitor, oxonic acid (OA) (750 mg/kg per day for 4 weeks by gastric gavage). Rats were divided into four groups: (1) control; (2) OA only: (3) OA + vitamin C (200 mg/kg for 4 weeks by gastric gavage); and (4) vitamin C only. At the end of the study, rats were sacrificed under diethyl ether general anesthesia and serum levels of uric acid, creatinine and blood nitrogen urea (BUN) as well as glutathione (GSH) levels and activities of superoxide dismutase (SOD), glutathione peroxidase (GPx) and glutathione S-transferase (GST) were measured as indices of oxidative stress and anti-oxidative status in kidney tissues. Also, histopathological examination of isolated kidney tissues was performed. The administration of OA resulted in 2.4 fold increase in serum uric acid levels, and was associated with development of kidney damage characterized by a significant increase in serum levels of creatinine and BUN, and significant decreases in renal GSH levels and activities of SOD, GPx and GST. By contrast, simultaneous administration of vitamin C significantly ameliorated all these biochemical changes induced by OA. The histopathological findings supported these biochemical observations, whereby vitamin C supplementation remarkably reduced OA-induced tubulointerstitial damage and cellular infiltration in rat kidneys. These results indicate that vitamin C therapy significantly attenuated the biochemical indices, histopathological findings and oxidative stress parameters of OA-induced hyperuricemia and nephrotoxicity in rats. This may provide insight into the possible potential renoprotective effect of vitamin C supplementation against hyperuricemia nephropathy.

Key words: Vitamin C, oxonic acid, hyperuricemia, nephrotoxicity, oxidative stress.

#### INTRODUCTION

Vitamin C (Ascorbic acid) is the most important vitamin in fruits and vegetables, and has been regarded as the most potent natural antioxidant. Although most of the higher animals can synthesize vitamin C in their liver or kidneys, in humans; the terminal enzyme in its synthetic pathway is absent and thus, vitamin C has become an essential dietary component for human survival (Vissers et al., 2011). Vitamin C is required for the prevention of scurvy, and in addition to its powerful antioxidant activity, it plays an important role as a cofactor in enzymes activation and immune function (Schlueter and Johnston, 2011). Vitamin C has anti-inflammatory effects, prevents endothelial dysfunction and apoptosis, and reduces the risk of arteriosclerosis, cardiovascular disease and some

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forms of cancer (Ozkanlar and Akcay, 2012). In concordance with the aforementioned research findings, beneficial effects of vitamin C in kidneys have been reported by several other researchers. For example vitamin C treatments improved kidney function in renal allograft recipients (Williams et al., 2001), decreased renal inflammation and improved impaired renal function in salt-sensitive hypertensive rats (Tian et al., 2007), and inhibited oxidative damage and renal injury in experimental modalities of chemical-induced nephrotoxicity (Atasayar et al., 2009).

Hyperuricemia is a common metabolic disorder and a well-established causative factor for development of gouty arthritis, tophi formation, uric acid kidney stones and acute kidney failure (Edwards, 2008). However, recent epidemiologic studies have suggested the potential direct role of chronic mild hyperuricemia in development of interstitial nephritis and progressive renal failure (Mok et al., 2012). Hyperuricemia as an independent risk factor, has also been associated with metabolic syndrome. cardiovascular disease, hypertension, obesity, obstructive sleep apnea, stroke, vascular dementia, and preeclampsia (Feig et al., 2008). More importantly, it should be emphasized that the currently used anti-hyperuricemic medications, such as allopurinol or probenecid, carry significant side effect profiles and are not indicated in patients with kidney or heart disease. The use of these agents is supported only in the treatment of gout, the treatment of uric acid kidney stones, and the prevention of tumor lysis syndrome (Juraschek et al., 2011). Recommendations to use dietary approaches to lower uric acid have been suggested (Gelber, 2008). In this aspect, supplementation with vitamin C has recently attracted a great deal of attention as an alternative dietary anti-hyperuricemic approach (Gao et al., 2008; Lagowska-Lenard et al., 2010). Despite these promising trials, future studies are needed to precisely determine whether vitamin C treatment can effectively prevent hyperuricemia and its associated co-morbidities (Juraschek et al., 2011). An animal model of chronic mild hyperuricemia induced by an uricase inhibitor, oxonic acid (OA), has recently shed new light on a potential mechanism of elevated uric acid in inducing renal tissue injury and microvascular changes (Sanchez-Lozada et al., 2008). Therefore, this study was aimed to investigate whether vitamin C supplementation could attenuate the development and outcome of chronic hyperuricemia nephropathy by using this experimental model of OA-induced mild hyperuricemia and renal injury in rats.

#### MATERIALS AND METHODS

#### Animals and experimental approach

A total of 50 adult male Wistar albino rats weighing 200 to 250 g obtained from Animal Center Laboratory, Umm Al-Qura University, Saudi Arabia, were housed in metabolic cages with a 12:12-h light:dark cycle at a constant temperature of 23 to 25°C, and had free access to standard commercial rat food (pellet form) and tap

water. To induce the experimental rat model with mild hyperuricemia, an uricase inhibitor, OA was given orally by gastric gavage at a dose of 750 mg/kg/day for 4 consecutive weeks as described previously (Sanchez-Lozada et al., 2008). The animals were randomized into four groups as follow: Group 1 (n = 10) served as normal controls; Group 2 (n = 15) received only OA and served as disease untreated group; Group 3 (n = 15) received OA (as in group 2) and simultaneously treated with vitamin C (200 mg/kg/day by gastric gavage for 4 consecutive weeks), and Group 4 (n = 10) received only vitamin C (as in group 3). The selected dose of vitamin C was based on our preliminary study and on those tested previously. All experiments were performed in accordance with the Standards Regulations and Guidelines Relating to the Care and Management of Animal Experimentation, and all standard reagents were purchased from Sigma (St. Louis, Mo., USA), unless indicated otherwise.

#### Sacrificing and sampling

At the end of experimental period of 4 weeks, the animals were fasted overnight but still allowed free access to water, weighed, and then sacrificed under ether anesthesia. Blood samples were drawn from the inferior vena cava and allowed to clot and retract at room temperature for 1-h before centrifugation at 3,000 ×g for 10 min. Supernatants (that is, serum samples) were separated and stored at -20 °C until used. Both kidneys of each rat were immediately excised, weighed, and rinsed with a phosphate buffered saline (PBS) solution, pH 7.4, to remove any blood cells and clots. The right kidney was used for histological examination, while the left one was cut into small pieces, homogenized in 5 volume of ice-cold Tris HCl buffer (50 mM, pH 7.4), and centrifuged at 10,000 ×g for 10 min. The volume of the resultant supernatant was measured and stored at -20 °C until used.

#### Uric acid measurement and biochemical assays

Serum levels of uric acid (UA), creatinine (Cr), blood urea nitrogen (BUN), total cholesterol (TC) and triglycerides (TG) were measured using commercial assay kits (BioAssay Systems, Hayward, CA) and following the manufacturers' instructions.

#### Histological examination

Isolated kidney specimens were fixed in 10% neutral formalin, paraffinized and were processed for histological examination. Paraffin sections, at 4  $\mu$ m thickness, were prepared from each kidney, stained with hematoxylin and eosin (H&E) and then, microscopically examined for the existence of renal tubular and glomerular damage, tubulointerstitial nephritis as well as cellular infiltration. All histological analyses were performed blinded by a pathologist unaware of the type of treatment so as to avoid biased results, and the scoring was done as none (-), mild (+), moderate (++) and severe (+++).

#### Assessment of renal antioxidant defense elements

Levels of glutathione (GSH) and enzymatic activity of superoxide dismutase (SOD), glutathione peroxidase (GPx), and glutathione Stransferase (GST); as strong indices of tissue antioxidant mechanisms, were spectrophotometrically assayed in the prepared supernatants of kidney tissue homogenates using commercial enzyme-linked immunosorbent assay (ELISA) kits (Sigma-Aldrich, St. Louis, Mo., USA), and according to the manufacturers' instructions.

#### Statistical analysis

Statistical analysis was performed using the Statistical Package for Social Science (SPSS) version 16 (SPSS Inc., Chicago, IL, USA.). All data are presented as means  $\pm$  SD. Differences in the various parameters in more than two groups were evaluated by a one-way analysis of variance (ANOVA). Continuous variables between two groups were analyzed using Student's *t*-test. Differences between groups were considered significant at p < 0.05.

#### RESULTS

## Vitamin C improved OA-induced hyperuricemia and renal injury

This study was designed on the hypothesis that persistent mild hyperuricemia at concentrations that do not lead to intrarenal urate crystal deposition can induce chronic renal injury and dysfunction, and vitamin C supplementation could produce anti-hyperuricemic effect and improve hyperuricemia associated nephropathy. To test this hypothesis, an experimental model of mild hyperuricemia in rats with OA were induced, which is a urate oxidase inhibitor, and the animals were simultaneously treated or not treated with vitamin C. As shown in Table 1, after four weeks of OA administration, there was a significant increase in serum uric acid level compared to normal control rats  $(3.1 \pm 0.5 \text{ versus } 1.3 \pm 0.1 \text{ mg/dl}, \text{ p})$ < 0.01); while co-administration of vitamin C significantly (p < 0.05) reduced this elevation (Table 1). In comparison to normal controls, OA also significantly increased kidney weight as well as serum levels of creatinine and BUN, as biomarkers of renal dysfunction, and significantly decreased serum albumin concentration. However, simultaneous treatment with vitamin C significantly ameliorated these alterations on kidney weight and serum levels of creatinine, BUN and albumin that were induced by OA (Table 1). At the end of the study, there were no significant differences (P = NS) in the values of body weights and serum concentrations of sodium and potassium ions among all experimental animal groups, suggesting that OA and/or vitamin C administration did not affect food consumption during the experimental period (Table 1).

Histopathological observations strongly supported the aforementioned biochemical findings, whereby OA administration (Figure 1B) caused prominent damage in the kidney interstitial tissues compared with the control rats (Figure 1A). The kidneys of rats administered with OA alone showed marked deleterious tubulointerstitial inflammation, sloughing of the tubular epithelial cells and predominant interstitial cellular infiltration (Figure 1B). In contrast, simultaneous treatment of these OA-administered rats with vitamin C almost preserved normal histo-architecture of the kidney (Figure 1C). The renal histopathological changes among all animal groups were

graded and summarized in Table 1.

## Vitamin C restored OA-induced depletion of renal antioxidant defence elements

The research hypothesis suggested that hyperuricemia could induce renal injury due to induction of oxidative stress. To confirm this hypothesis, the levels of total GSH and the activities of antioxidant enzymes, SOD, GPx and GST were measured in the renal tissue homogenates of all experimental groups at the end of the study. As shown in Table 2, OA administration significantly decreased (p < p0.05) GSH levels as well as activities of SOD, GST and GPx, and this is suggestive of induction of oxidative stress phenomenon in OA administered rats; however, simultaneous therapy with vitamin C significantly (p < 0.05) elevated the decreased GSH, and also significantly increased (p < 0.05) the reduced antioxidant enzyme activities towards almost their normal values (Table 2). The kidneys of normal rats treated with vitamin C alone also showed significant increases in the levels of GSH and in the activities of SOD, GPx and GST antioxidant enzymes in comparison of normal control groups, indicating the natural antioxidant property of vitamin C (Table 2).

#### DISCUSSION

The present study demonstrates the attenuating effects of vitamin C supplementation on experimental rat model induced with chronic mild hyperuricemia resulting to remarkable intrarenal oxidative damage and renal injury. These data support the notion that an elevation in circulating uric acid, rather than being a surrogate biomarker of kidney dysfunction, is actually an active player in the pathogenesis of renal disease (Inaba et al., 2013). It also support the hypothesis that vitamin C might be an effective dietary approach in the prevention and management of hyperuricemia and its related diseases (Choi et al., 2009; Juraschek et al., 2011), particularly in patients with nephropathy.

In the present study, the hypothesis that mild elevation of serum uric acid may have a direct causal role in the development of renal disease was tested by development of a rat model of mild hyperuricemia induced by OA, which is an uricase inhibitor and, thus, prevents the breakdown of endogenous uric acid (Mazzali et al., 2001). After four weeks of OA administration, there was a significant (2.4 fold) increase in serum uric acid level, as well as in kidney weights and serum levels of creatinine and BUN (as indicators of impaired kidney function) (Table 1). The histopathological observations were consistent with these biochemical changes, whereby a marked tubulointerstitial inflammation and a striped pattern of interstitial cellular infiltration were observed in

| uric | acid,   | creatinine,    | blood  | urea     | nitrogen     | (BUN), | albumin, | sodium | and | potassium | ions; | and |
|------|---------|----------------|--------|----------|--------------|--------|----------|--------|-----|-----------|-------|-----|
| scor | es of I | renal injury a | and ce | llular i | nfiltration. |        |          |        |     |           |       |     |
|      |         |                |        |          |              |        |          |        |     |           |       |     |

Table 1. Effects of oxonic acid (OA) and vitamin C on body and kidney weights; serum levels of

|                              |                 | (                        | Group                      |                          |
|------------------------------|-----------------|--------------------------|----------------------------|--------------------------|
| Parameter                    | Control         | OA                       | OA + Vitamin C             | Vitamin C                |
|                              | (n = 10)        | (n = 15)                 | (n = 15)                   | (n = 10)                 |
| Body weights (g)             | 274 ± 5.9       | 272 ± 7.3 <sup>c</sup>   | $273 \pm 4.5^{\circ}$      | $276 \pm 6.5^{\circ}$    |
| Kidney weights (g)           | 0.98 ± 0.13     | 1.32 ± 0.27 <sup>a</sup> | 1.05 ± 0.11 <sup>d,c</sup> | 0.98 ± 0.19 <sup>c</sup> |
| Uric acid (mg/dl)            | 1.3 ± 0.1       | 3.1 ± 0.5 <sup>b</sup>   | $1.6 \pm 0.2^{d,c}$        | 1.1 ± 0.1 <sup>c</sup>   |
| Creatinine (mg/dl)           | $0.63 \pm 0.03$ | 1.31 ± 0.09 <sup>a</sup> | $0.89 \pm 0.08^{d,c}$      | $0.64 \pm 0.04^{\circ}$  |
| BUN (mg/dl)                  | 32.5 ± 1.6      | 79.4 ± 5.5 <sup>a</sup>  | $39.0 \pm 1.7^{d,c}$       | 31.7 ± 1.5 <sup>c</sup>  |
| Albumin (g/dl)               | 1.2 ± 0.2       | $0.67 \pm 0.06^{a}$      | $0.90 \pm 0.06^{c,e}$      | $1.2 \pm 0.4^{c}$        |
| Na <sup>+</sup> (mmol/L)     | 142 ± 9.7       | 139 ± 13.5 <sup>c</sup>  | 143 ± 5.6 <sup>c,e</sup>   | 141 ±8.3 <sup>c</sup>    |
| K⁺ (mmol/L)                  | $5.80 \pm 0.30$ | $5.56 \pm 0.5^{\circ}$   | $5.67 \pm 0.33^{c,e}$      | $5.9 \pm 0.5^{\circ}$    |
| Tubulointerstitial nephritis | _               | +++                      | +                          | -                        |
| Renal cellular infiltration  | -               | +++                      | +                          | -                        |

Values are expressed as the mean  $\pm$  SD. <sup>a</sup>P < 0.05, <sup>b</sup>P < 0.01 and <sup>c</sup>P = NS versus normal control group; <sup>d</sup>P < 0.05 and <sup>e</sup>P = NS versus oxonic acid (OA) alone received group. Severity of renal histological changes and levels of intrarenal cellular infiltration were scored on a scale of none (–), mild (+), moderate (++) and severe (+++) degree as described in part of the methodology.

Table 2. Changes in the antioxidant defense elements in kidney homogenates of experimental animal groups.

|  |                 | (                        | Group                     |                          |
|--|-----------------|--------------------------|---------------------------|--------------------------|
| Parameter  | Control         | OA                       | OA + Vitamin C            | Vitamin C                |
|  | (n = 10)        | (n = 15)                 | (n = 15)                  | (n = 10)                 |
| Glutathione content (µmol/ml)                    | 5.37 ± 1.0      | $2.3 \pm 0.3^{a}$        | $5.40 \pm 0.9^{c}$        | 7.3 ± 1.1 <sup>a</sup>   |
| Superoxide Dismutase activity (Units/ml)         | $0.20 \pm 0.01$ | 0.09 ± 0.01 <sup>b</sup> | $0.19 \pm 0.03^{d,c}$     | $0.24 \pm 0.02^{a}$      |
| Glutathione peroxidase activity (Units/ml)       | 33.3 ± 5.27     | 17.34 ± 2.5 <sup>a</sup> | 31.4 ± 6.2 <sup>d,c</sup> | 40.0 ± 5.1 <sup>a</sup>  |
| Glutathione S-transferase activity (µmol/ml/min) | 2.63 ± 0. 3     | $0.85 \pm 0.09^{b}$      | $2.29 \pm 0.08^{d,c}$     | 3.14 ± 0.84 <sup>a</sup> |

Values are expressed as the mean  $\pm$  SD. <sup>a</sup>P < 0.05, <sup>b</sup>P < 0.01 and <sup>c</sup>P = NS versus normal control group; <sup>d</sup>P < 0.05 and <sup>e</sup>P = NS versus oxonic acid (OA) alone received group.



**Figure 1.** Histopathological findings. (A) Kidneys of normal rats showing normal glomerular and tubular structure. **(B)** Kidneys of rats that received oxonic acid (OA) alone; demonstrating clear hallmarks of tubulointerstitial nephritis with excessive number of interstitial cellular infiltration. **(C)** Kidneys of rats that received a combination of OA plus vitamin C; revealing significant improvements in all renal histomorphological structures.

the kidneys of hyperuricemic rats (Figure 1). These biochemical and histopathological findings have also

been previously reported by Sanchez-Lozada et al. (2008), and support the direct involvement of hyperuricemia

in induction of renal disease. In agreement, Nakagawa et al. (2003) showed that hyperuricemia has direct proinflammatory effects and causes glomerular hyper-trophy in rat kidneys independent of intrarenal crystal formation. Moreover, soluble uric acid has been found to stimulate monocyte chemotaxis and the release of proinflammatory mediators from vascular cells (Kanellis et al., 2003). Virtually, chemotaxis and recruitment of inflammatory cells into kidney tissues play pivotal roles in development of renal injury and progressive renal diseases through the spillover of proinflammatory mediators, modulate extracellular matrix synthesis, and promote oxidative stress (Sean and Cockwell, 2005).

In this study, co-administration of vitamin C with OA interestingly resulted in significant anti-hyperuricemic and renoprotective effects that were reflected by reducing the elevations in serum levels of uric acid, creatinine and BUN, and almost preserved the normal histoarchitecture of the kidney (Table 1 and Figure 1). Previously, it has been reported that vitamin C can lower serum uric acid via a direct uricosuric effect, which is likely due to a competition for renal reabsorption of uric acid via an anion-exchange transport system at the proximal tubule (Choi et al., 2009), or by increasing glomerular filtration rate, and thus providing another potential mechanism for the uricosuric effect of vitamin C intake (Huang et al., 2005). Moreover, effective antioxidant vitamin C decreases free radical-induced damage to body cells, thereby reducing production and ultimately serum concentration of uric acid (Gao et al., 2008). Human observational studies have confirmed the uricosuric effect of vitamin C and an inverse association between vitamin C intake and serum uric acid concentrations has been reported (Gao et al., 2008). Vitamin C supplementation has been shown to lower serum uric acid in haemodialysis patients and during exhaustive exercise (Tauler et al., 2003). Moreover, a recent randomized trial of daily intravenous infusion of 500 mg of vitamin C for 10 days in patients with acute ischemic stroke resulted in a significant reduction in serum uric acid compared to placebo infusion (Lagowska-Lenard et al., 2010). Moreover, the specific renoprotective effects of vitamin C have also been reported in other modalities of kidney diseases at both the clinical and experimental levels (Tian et al., 2007; Atasayar et al., 2009).

It remains speculative whether the antioxidant action of vitamin C may have a protective effect against hyperuricemiainduced nephropathy. There is a strong body of evidence that increased oxidative damage due to an excess of free radicals, or generation of reactive oxygen species (ROS), is one of the most important pathogenic mechanisms in the development of many diseases and pathophysiological problems, including renal disease (Wilcox and Gutterman, 2005; Sanchez- Lozada et al., 2008). Generation of ROS can lead to various forms of cellular injury, such as inflammation, cell death, necrosis, and DNA damage or fragmentation (Passos and Von Zglinicki, 2006). The kidney tissues are rich sources of nicotinamide adenine

dinucleotide phosphate-oxidase (NADPH) oxidasederived ROS, which under pathological conditions can contribute to renal dysfunction and damage (Gill and Wilcox, 2006). On the other hand, ROS can be detoxified by a battery of antioxidant cellular defences, including scavenging action of glutathione (GSH) as well as antioxidant activities of SOD, GST, GPx and other antioxidant enzymatic systems. Modulations of these enzymes and concentrations of GSH appear to be sensitive indicators of the overall health of a cell and its ability to resist toxic challenges (Matés, 2000). The findings of the present study are in harmony with these collective facts, whereby OA-induced hyperuricemia and renal injury was significantly associated with a significant decrease in GSH levels and activities of SOD, GST and GPx (Table 2), suggesting that OA-induced hyperuricemia in rats was associated with oxidative damage of their renal tissues. By contrast, simultaneous therapy with vitamin C significantly restored the renal content of GSH as well as activities of SOD, GST and GPx towards almost their normal values (Table 2). Similar observations that OA induced hyperuricemia and renal injury triggered intrarenal oxidative stress have also been reported by Sanchez-Lozada et al. (2008). Moreover, in an in vitro study, uric acid was able to stimulate oxidant generation and reduction in antioxidant levels in cultured cells (Sautin et al., 2007).

#### Conclusion

These findings show that vitamin C supplementation exhibits anti-hyperuricemic, anti-oxidation and nephroprotective activity in rat model of hyperuricemia-induced oxidative damage and renal injury. This study may provide an evidence to support clinical therapeutic value of vitamin C in treatment of hyperuricemia with renal dysfunction.

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Full Length Research Paper

## Physiological and anthropometric correlates of metabolic risk factors among selected non obese adults in Zaria, Northern Nigeria

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Recent publications on clinical definitions have transformed the metabolic syndrome from a physiological curiosity to a major focus of research, clinical and public health interests on non obese adults. The purpose of this study therefore, was to determine the relationship between anthropometrics and cardiometabolic variables that reflect the existence of metabolic syndrome among non obese adults. One hundred and seventy four (174) adults, with mean age of 47.13 ± 8.10 (male) and 44.96 ± 9.58 (female) were recruited for this cross sectional study. Descriptive statistics, partial correlation and multiple regression analysis were used to determine the relationship between anthropometric measurements and cardiometabolic variables after controlling for age. Anthropometric indices, lipid profile, fasting glucose and blood pressure were among the variables assessed using standard procedures. The best correlation among the anthropometrics ( $p \le 0.05$ ) was presented between waist circumference and waist-height ratio (male r = 0.925, female r = 0.916) and body fat (%) and fat mass (male r = 0.956, female r = 0.944). Fat mass, waist-height ratio and waist circumference in male and waist-height ratio in female were found to have the largest correlation relative to at least 3 risk factors. Combination of three components of cardiometabolic risk factors were significantly more in male than female. Regression analysis also showed that waist-height ratio appears optimal for predicting components of cardiometabolic risk factors among non obese adults. Among all obesity measures studied, waist-height ratio, waist circumference and fat mass explained comparatively larger amount of variance of cardiometabolic risk factors among non-obese adults. Non obese male were significantly more likely to have two or more risk factors than female participants. However, the greater risk of developing metabolic syndrome was associated with increasing waist-height ratio, which could be used as simple and non-invasive method for detecting dyslipidemia among non obese adults, and use of this method was suggested in clinical and epidemiological fields.

Key words: Metabolic syndrome, non obese adults, anthropometric indices.

#### INTRODUCTION

Obesity and its associated metabolic abnormalities have

been the focus of many researchers lately, especially

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physiologists, epidemiologists and geneticists. More so, there are individuals who by standard weight/body mass index (BMI) tables are not obese or even overweight, but have metabolic abnormalities that who are characteristically associated with adult onset obesity. The constellation of these abnormalities also known as metabolic syndrome (MetS) have been widely investigated (Song et al., 2006; Opie, 2007; Zhao et al., 2010).

International Diabetes Federation (IDF) proposed a definition for MetS for use in epidemiology studies and clinical practice, which would allow for comparison between different population groups and the assessment of its relationship with various health outcomes (Motala et al., 2009). The IDF, (2005) defined a participant as having MetS if he or she had central obesity (Ethnicity specific waist circumference values) plus at least 2 of the following criteria: (1) triglyceride level  $\geq$  1.7 mmol/L; (2) reduced high density lipoprotein-cholesterol (HDL-C) levels of less than 1.03 mmol/L in men, less than 1.29 mmol/l in women; (3) raised systolic or diastolic blood pressure of 130/85 mmHg or higher, or previously diagnosed hypertension; and (4) raised fasting plasma glucose level of 5.6 mmol/L or higher, or previously diagnosed type 2 diabetes mellitus (T2DM) (Cornier et al., 2008).

A prerequisite in the IDF definition of the MetS is central obesity measured by ethnicity-specific waist circumference and it is recommended that European cut points be used for populations for which such data are not available, for example in Sub-Saharan Africans (Motala et al., 2009). Because risk factors cluster, and because the cardiovascular risk factors for the MetS are linear in their damaging effects, different definitions of the metabolic syndrome make little difference in the prognostic implications (Opie, 2007). Although data in the developing countries and particularly in the sub-Saharan African remain scarce (Okosun et al., 2000; Addo et al., 2007; Motala et al., 2009; Kimani-Murage et al., 2010), high prevalence of MetS has often been documented in developed countries and increasingly so in developing countries (Ford et al., 2008; Kieldsen et al., 2008; Rufus et al., 2008).

Measurement of waist circumference is recommended by the US National Cholesterol Education Program (NCEP) for the assessment of central obesity, whereas the World Health Organization (WHO) recommends waist-hip ratio (WHpR) for the same purpose. All of the mentioned anthropometric indices have been found to be associated with all-cause mortality, diabetes mellitus, cardiovascular morbidity and mortality in prospective studies (WHO, 1997; Ahmet et al., 2008). Some authors have proposed waist-height ratio (WHtR) as the best anthropometric index to predict cardiovascular disease (CVD) risk, and MetS and hip circumference (HC) has been found to be inversely associated with diabetes, CVD morbidity and mortality in a prospective study (Lissner et al., 2001; Hsieh and Muto, 2006).

The results of prospective and cross-sectional studies that have attempted to find the best anthropometric index are not uniform, studies from the Eastern Mediterranean area do not agree on the best anthropometric index to predict cardiometabolic risk (Yalcin et al., 2005; Ahmet et al., 2008). Work in Turkey has suggested that WHpR might better indicate CVD risk than BMI and WC, the same study also showed that both WC and WHpR were strongly associated with BMI, age, diastolic blood pressure and plasma triglyceride (TG); also, WHpR was significantly associated with prevalent coronary heart disease (CHD) only in Turkish women. Fasting glucose, insulin, HDL cholesterol (HDL-C) and Low density lipoprotein cholesterol (LDL-C) levels were not ascertained, and the association of anthropometric indices with MetS was not reported in that study (Onat et al., 1999). On the other hand, Kayode et al. (2009) showed in their study that WHtR and abdominal height (measured as the distance from the exam table to the top of the belly when the patient is lying supine) predicted CVD than any other anthropometric measurement including BMI, WC, WHpR and skin-fold thickness.

The Japanese Society of Internal Medicine also announced a definition of the MetS for the Japanese which is now used in Japan. Most of these definitions employ WC as an indicator of central or abdominal obesity. However, several reports have argued that other indices, for example the WHtR and WHpR are superior to WC for identifying subjects with cardiovascular risk factors. In particular, reports from Japan have proposed using the WHtR (Ishikawa-Takata et al., 2002; Masayuki et al., 2008). Little is known about the relationship between the anthropometric indices and various metabolic disorders in Nigerian adult population (Okosun et al., 2000; Olatunbosun et al., 2000) and there has been paucity of recent literatures relating to local incidence, particularly in Kaduna, Nigeria (Bello-Sani et al., 2007).

The aim of this study is to investigate the interrelationship between anthropometrics and MetS among non-obese adults of Zaria, Northern Nigeria. This study will attempt to investigate the correlations between anthropometric indices and risk factor accumulation (RFA) defined as the existence of any two (or three) of the following disorders: hypertension, dyslipidaemia (defined by high TG and/or low levels of HDL) and fasting hyperglycaemia, each of which is a component of MetS (Masayuki et al., 2008), and their cut-off points in this study were based on the IDF (2005) criteria of metabolic syndrome because they consider the sub-Sahara Africans in their values.

#### Statements of hypotheses

On the basis of previous research evidences, it was

hypothesized that there are no significant relationships among anthropometric indices and RFA, and that central adiposity does not predict metabolic risk factors better than overall obesity.

#### MATERIALS AND METHODS

#### **Research design**

This study is a correlational research that cross-sectionally examines the covariation among some physiological and anthropometric indices, and assess their predictability as regards to metabolic risk indicators.

#### Sampling technique

The subjects for this study were form Zaria, northern Nigeria; based on interest and willingness to participate in the study. The stratified random sampling technique was used to stratify the subjects by wards, and subjects were subsequently constituted by using purposive sampling technique. These were adopted because only subjects who satisfied the predetermined exclusion and inclusion criteria of being healthy, non-obese and aged 35 to 70 years were selected for the research. The criteria for BMI (Lincoln et al., 2002) were adopted to assess obese and non-obese subjects.

#### Study population

The participants for this study were 174 male and female adults in Zaria; northern Nigeria; aged between 35 and 70 years. The subjects were not obese, not on any special diet and not hypertensive or diabetic (if so, medically controlled). Exclusion criteria also include diagnosed major medical conditions such as CHD and kidney disease. Also, subjects on any medication known to influence energy regulation were not included. The subjects were certified fit by a physician at the Ahmadu Bello University Teaching Hospital (ABUTH), Family Medicine Out-Patient Department, and were adequately informed about the rationale behind this study. In addition, participating adult female subjects were ensured not to have been pregnant or had a laparotomy procedure in the past 2 years.

#### Study site

This study was carried out between May and October, 2011 in Zaria, Kaduna state, Northern Nigeria. Zaria is a cosmopolitan city, inhabited by about 408,198 people as stated by the 2006 census report and is the second largest city in Kaduna state. It is located at latitude 11°3' N and longitude 7° 42' N. The city lies on the high plains of Northern Nigeria, in the Sub-Saharan Africa. It is about 643.7 km from the coast of Nigeria. Different tribes and ethnic groups could be found in Zaria, the three major ethnic groups: Hausa, Yoruba and Igbo and minorities grouped together as others. Zaria has many tertiary institutions of learning and research, including Ahmadu Bello University, Zaria. The climate is savannah with annual rainfall ranging from 0.0 to 816.0 mm/month and minimum and maximum temperature of 15.3 and 36.25°C, respectively (Avidime et al., 2011).

#### Instruments

The equipments for this study were stadiometer, weighing scale,

sphygmomanometer, tape rule measure, skinfold caliper, stopwatch and spectrophotometer. The IDF (2005) guideline of the MetS with ethnic specific WC for Sub-Sahara Africans was used as the main outcome measures. Normal-weight BMI was defined as a range of 18.5 to 24.9 kg/m<sup>2</sup> and overweight BMI  $\leq$  27 kg/m<sup>2</sup> was considered because BMI  $\geq$  28 kg/m<sup>2</sup> has been shown to be a significant prognostic factor for all-caused and cardiovascular mortality among adults (Asefeh et al., 2001; Ofei, 2005).

#### Ethical committee approval

This research was submitted to the Joint Institutional Scientific and Ethical Committee of the Ahmadu Bello University Teaching Hospital, Shika, Zaria, for review, screening and approval. The informed consent of the subjects was sought before they were included in the study and confidentiality was maintained in accordance with standard medical practice.

#### Format of testing

All the readings and measurements were carried out in the Department of Human Physiology Laboratory, Faculty of Medicine, Ahmadu Bello University, Zaria. Each subject in their gymnasium kits made a total of two visits to this same venue for the purpose of this study. The first visit was basically familiarization and screening of subjects to ensure their fitness for the study. All the exclusion and inclusion criteria were strictly observed. A routine BMI check for obese subjects was also carried out and consent forms signed. The actual readings and measurements were taken on the second visit. The subjects reported at 09.00 h in their gymnasium kits to the same venue in the morning of the appointed day (expected to have fasted for at least 12 h). They were allowed to rest for about 30 min after which the readings and measurements were taken as follows:

#### Anthropometric measurements

These were used for body composition assessments. The methodology is based on the assumption that body fat is distributed at various sites on the body specifically chest, abdomen and thigh in men; triceps, suprailiac and thigh in women (Gallagher et al., 1996). With the use of a skinfold caliper, the skinfold thickness in (mm) at these sites was taken twice, and the average at each site recorded. WC, weight, height and HC were also measured, and WHpR and WHtR were calculated.

#### Physiological parameters

These included the blood pressure, heart rate and blood sample analysis of TC, triglyceride (TG), HDL and fasting plasma glucose. LDL was calculated using the Friedewald's formula:

LDL = TC - HDL - (TG / 2.17) mmol/l (provided TG values are less than 4.52 mmol/l).

#### Data analysis

Data was analyzed using statistical package for social sciences (SPSS Inc, version 16. 0; Chicago). Descriptive statistics of mean and standard deviation was computed for the purpose of data interpretation. Partial correlation analysis was used to identify the significance and trend of relationships among the variables.

| Magguramanta                         | Male (n=91)  | Female (n=83) |
|--------------------------------------|--------------|---------------|
| Measurements                         | Mean±SD      | Mean±SD       |
| Age (years)                          | 47.13±8.10   | 44.96±9.58    |
| Weight (kg)*                         | 69.55±9.60   | 62.90±7.96    |
| Height (cm)*                         | 173.32±6.42  | 162.58±6.09   |
| Waist circumference (cm)             | 87.90±8.17   | 86.87±7.70    |
| Hip circumference (cm)*              | 93.37±6.12   | 99.27 ±6.48   |
| Body mass index (kg/m <sup>2</sup> ) | 23.13±2.73   | 23.78±2.42    |
| Waist-hip ratio*                     | 0.941±0.050  | 0.876±0.06    |
| Waist-height ratio*                  | 0.508±0.048  | 0.535±0.05    |
| Percentage body fat (%)*             | 20.20±5.89   | 34.682±7.65   |
| Fat mass (kg)*                       | 14.40±5.47   | 22.14±6.55    |
| Fat free mass (kg)*                  | 55.154±6.034 | 40.763±4.68   |

Table 1. Anthropometric characteristics of the study participants.

\*Statistical significant difference of equality of means at (95% CI, P  $\leq$  0.05; critical value: 1.960; Df: 172).

Correlations were considered significant at  $P \le 0.05$  with critical values located at 0.2050 (male), 0.2172 (female). To find the variables that best prevent the prediction errors, the accuracy of simple and derived anthropometrics were assessed further by stepwise multiple regression analysis in which the data included the predictive variables that correlated with the RFA as dependent variables. Differences were considered significant at  $P \le 0.05$ .

#### RESULTS

## Anthropometric characteristics of the study participants

Participants were within the BMI class of normal weight (male: 23.13 ± 2.73, female: 23.78 ± 2.42). A total of 56 subjects were excluded on the basis of the study criteria. the remaining 174 subjects were included in statistical analyses. The physical and anthropometric characteristics of study participants are summarized in Table 1. The mean age of study participants was 47.13 years for male and 44.96 years for female. Using statistical t-test of equality of means at confidence interval: 95%,  $P \le 0.05$ ; critical value: 1.960; Df: 172, male were significantly taller (173.32  $\pm$  6.42), with narrower hips (93.37  $\pm$  6.12), smaller fat mass (14.40  $\pm$ 5.47) and higher resting energy expenditure (1571  $\pm$ 166.85) as compared to female (162.58  $\pm$  6.09, 99.27  $\pm$ 6.48, 22.14 ± 6.55 and 1.347.17 ± 97.38), respectively. The waist-height ratio (0.535  $\pm$  0.05) and fat% (34.682  $\pm$ 7.65) were higher in female than in male  $(0.508 \pm 0.048)$ , 20.20 ± 5.89), respectively. No significant difference was observed between males and females with respect to waist circumference (male: 87.90 ± 8.17, female: 86.87 ± 7.70) and BMI (male: 23.13 ± 2.73, female: 23.78 ± 2.42).

#### Clinical characteristics of the study participants

The summary of the clinical parameters of the study

participants is shown in Table 2. Participants were normotensive (mean arterial blood pressure:  $95.02 \pm$ 12.76 in male and  $99.398 \pm 17.94$  in female). Statistical mean values of the systolic blood pressure (127.03 ± 20.14), diastolic blood pressure (79.01 ± 10.76), mean arterial blood pressure (95.02 ± 12.76), and high density lipoprotein (0.944 ± 0.325) in male were lower than in female (132.77 ± 26.05, 82.71 ± 26.05, 99.398 ± 17.94, 1.150 ± 0.82, 0.955 ± 0.37), respectively. Triglyceride (1.37 ± 1.11) and total cholesterol (3.017 ± 0.83) were found to be slightly higher in male than female group (1.150 ± 0.82 and 3.017 ± 0.83), respectively. Statistical ttest of equality of means at (Confidence interval 95%, P ≤ 0.05; critical value: 1.960; Df: 172) did not show any significant difference except for pulse rate.

#### Relationships among measures of adiposity

Tables 3 and 4 show the relationships among all anthropometric variables that were examined in this study at critical value: 0.2050 (male), 0.2172 (female); P ≤ 0.05. In male, waist circumference exhibited the strongest correlation among the anthropometric variables studied (weight,  $r = 0.797^{**}$ ; BMI,  $r = 0.851^{**}$ ; waist-height, ratio r = 0.925<sup>\*\*</sup>; waist-hip ratio,  $r = 0.727^{**}$ ; fat mass, r =0.956<sup>\*\*</sup>), followed by waist-height ratio (weight, r =0.580\*\*; BMI, r = 0.840\*\*; waist circumference, r =0.925<sup>\*\*</sup>; waist-hip ratio,  $r = 0.807^{**}$ ; fat mass,  $r = 0.776^{**}$ ) as shown in Table 3. Similarly, in female, waist circumference exhibited the strongest correlation among the anthropometric variables studied (weight,  $r = 0.721^{**}$ ; BMI,  $r = 0.774^{**}$ ; waist-height ratio,  $r = 0.916^{**}$ ; waist-hip ratio,  $r = 0.692^{**}$ ; fat mass,  $r = 0.689^{**}$ ), followed by waistheight ratio (weight,  $r = 0.474^{**}$ ; BMI,  $r = 0.775^{**}$ ; waist circumference,  $r = 0.916^{**}$ ; waist-hip ratio,  $r = 0.666^{**}$ ; fat mass,  $r = 0.553^{**}$ ) as shown in Table 4. Comparatively,

| Maaauramanta                        | Male (n=91)  | Female (n=83) |
|-------------------------------------|--------------|---------------|
| Measurements                        | Mean±SD      | Mean±SD       |
| Systolic blood pressure (mmHg)      | 127.03±20.14 | 132.77±26.05  |
| Diastolic blood pressure (mmHg)     | 79.01±10.76  | 82.71±14.74   |
| Pulse rate (beats/min)*             | 68.73±8.92   | 75.81±11.79   |
| Mean arterial blood pressure (mmHg) | 95.02±12.76  | 99.398±17.94  |
| Total cholesterol (mmol/dl)         | 3.13±1.12    | 3.017±0.83    |
| Triglyceride (mmol/dl)              | 1.37±1.11    | 1.150±0.82    |
| High density lipoprotein (mmol/dl)  | 0.944±0.325  | 0.955±0.37    |
| Low density lipoprotein (mmol/dl)   | 1.63±0.904   | 1.581±0.71    |
| Fasting plasma glucose (mmol/dl)    | 3.997±1.40   | 4.174±1.32    |

Table 2. Clinical characteristics of the study participants.

\*Statistical significant difference of equality of means at (95% Cl, P  $\leq$  0.05; critical value: 1.960; Df: 172).

Table 3. Correlation matrix for measures of adiposity in male participants.

| Parameter | WT      | HT      | WC      | HC      | BMI     | WHpR    | WHtR    | FAT (%) | FM      | FFM   |
|-----------|---------|---------|---------|---------|---------|---------|---------|---------|---------|-------|
| WT        | 1.000   |         |         |         |         |         |         |         |         |       |
| HT        | 0.507** | 1.000   |         |         |         |         |         |         |         |       |
| WC        | 0.797** | 0.124   | 1.000   |         |         |         |         |         |         |       |
| HC        | 0.878** | 0.391*  | 0.842** | 1.000   |         |         |         |         |         |       |
| BMI       | 0.842** | -0.034  | 0.851** | 0.775** | 1.000   |         |         |         |         |       |
| WHpR      | 0.313** | -0.260* | 0.727** | 0.211*  | 0.530** | 1.000   |         |         |         |       |
| WHtR      | 0.580** | -0.261* | 0.925** | 0.651** | 0.840** | 0.807** | 1.000   |         |         |       |
| FAT (%)   | 0.624** | 0.053   | 0.825** | 0.734** | 0.690** | 0.537** | 0.782** | 1.000   |         |       |
| FM        | 0.816** | 0.232*  | 0.890** | 0.849** | 0.800** | 0.508** | 0.776** | 0.956** | 1.000   |       |
| FFM       | 0.852** | 0.597** | 0.462** | 0.628** | 0.615** | 0.037   | 0.220*  | 0.127   | 0.393** | 1.000 |

\*\*Correlation is significant at the 0.01 level (2-tailed) \*Correlation is significant at the 0.05 level (2-tailed) Critical value: 0.2050 P: ≤0.05

| Table 4. Correlation | matrix for m | easures of | adiposity in | female participants |
|----------------------|--------------|------------|--------------|---------------------|
|                      |              |            |              |                     |

| Parameter | WT      | HT      | WC      | HC      | BMI     | WHpR    | WHtR    | FAT (%)  | FM     | FFM   |
|-----------|---------|---------|---------|---------|---------|---------|---------|----------|--------|-------|
| WT        | 1.000   |         |         |         |         |         |         |          |        |       |
| HT        | 0.545** | 1.000   |         |         |         |         |         |          |        |       |
| WC        | 0.721** | 0.128   | 1.000   |         |         |         |         |          |        |       |
| HC        | 0.735** | 0.161   | 0.648** | 1.000   |         |         |         |          |        |       |
| BMI       | 0.796** | -0.07   | 0.774** | 0.761** | 1.000   |         |         |          |        |       |
| WHpR      | 0.251*  | 0.017   | 0.692** | -1.000  | 0.296*  | 1.000   |         |          |        |       |
| WHtR      | 0.474** | -0.280* | 0.916** | 0.559** | 0.775** | 0.666** | 1.000   |          |        |       |
| FAT (%)   | 0.563** | 0.086   | 0.589** | 0.547** | 0.618** | 0.261*  | 0.534** | 1.000    |        |       |
| FM        | 0.793** | 0.272*  | 0.689** | 0.681** | 0.751** | 0.261*  | 0.553** | 0.944**  | 1.000  |       |
| FFM       | 0.521** | 0.508** | 0.213   | 0.245*  | 0.248*  | 0.044   | -0.001  | -0.402** | -0.106 | 1.000 |

\*\*Correlation is significant at the 0.01 level (2-tailed). \*Correlation is significant at the 0.05 level (2-tailed), critical value: 0.2172,  $P \le 0.05$ .

male group had higher values of correlation coefficient and the strength of correlation was more than that of the female group. The pattern of correlation was also similar for waist circumference, waist-height ratio and BMI; with waist circumference showing the strongest attitude in both groups.

| Parameter | SBP     | DBP     | PULSE   | MABP    | TG      | HDL    | LDL     | FPG   |
|-----------|---------|---------|---------|---------|---------|--------|---------|-------|
| WT        | 0.124   | 0.284*  | 0.182   | 0.225*  | 0.102   | 0.092  | 0.327** | 0.16  |
| HT        | -0.172  | 0.108   | -0.093  | 0.03    | -0.131  | -0.003 | 0.286** | 0.066 |
| WC        | 0.258*  | 0.271** | 0.270** | 0.288** | 0.272** | 0.143  | 0.251*  | 0.131 |
| HC        | 0.13    | 0.271** | 0.2     | 0.221*  | 0.1     | 0.173  | 0.394** | 0.165 |
| BMI       | 0.241*  | 0.248*  | 0.264*  | 0.266*  | 0.203   | 0.106  | 0.202   | 0.152 |
| WHpR      | 0.282** | 0.131   | 0.226*  | 0.222*  | 0.352** | 0.043  | -0.043  | 0.027 |
| WHtR      | 0.312** | 0.216*  | 0.297** | 0.286** | 0.318** | 0.139  | 0.137   | 0.104 |
| FAT (%)   | -0.259* | 0.212*  | 0.269** | 0.255*  | 0.181   | 0.069  | 0.168   | 0.042 |
| FM        | 0.246*  | 0.271** | 0.264** | 0.281** | 0.159   | 0.078  | 0.246*  | 0.089 |
| FFM       | 0.026   | 0.206*  | 0.05    | 0.102   | 0.018   | 0.076  | 0.298** | 0.174 |

**Table 5.** Correlation between measures of adiposity and cardiometabolic risk factors in male participants.

\*\*Correlation is significant at the 0.01 level (2-tailed) \*Correlation is significant at the 0.05 level (2-tailed). Critical value: 0.2050.  $P \le 0.05$ .

 Table 6. Correlation between measures of adiposity and cardiometabolic risk factors in female participants.

| Parameter | SBP     | DBP     | PULSE   | MABP    | TG      | HDL    | LDL     | FPG   |
|-----------|---------|---------|---------|---------|---------|--------|---------|-------|
| WT        | 0.124   | 0.284*  | 0.182   | 0.225*  | 0.102   | 0.092  | 0.327** | 0.16  |
| HT        | -0.172  | 0.108   | -0.093  | 0.03    | -0.131  | -0.003 | 0.286** | 0.066 |
| WC        | 0.258*  | 0.271** | 0.270** | 0.288** | 0.272** | 0.143  | 0.251*  | 0.131 |
| HC        | 0.13    | 0.271** | 0.2     | 0.221*  | 0.1     | 0.173  | 0.394** | 0.165 |
| BMI       | 0.241*  | 0.248*  | 0.264*  | 0.266*  | 0.203   | 0.106  | 0.202   | 0.152 |
| WHpR      | 0.282** | 0.131   | 0.226*  | 0.222*  | 0.352** | 0.043  | -0.043  | 0.027 |
| WHtR      | 0.312** | 0.216*  | 0.297** | 0.286** | 0.318** | 0.139  | 0.137   | 0.104 |
| FAT (%)   | -0.259* | 0.212*  | 0.269** | 0.255*  | 0.181   | 0.069  | 0.168   | 0.042 |
| FM        | 0.246*  | 0.271** | 0.264** | 0.281** | 0.159   | 0.078  | 0.246*  | 0.089 |
| FFM       | 0.026   | 0.206*  | 0.05    | 0.102   | 0.018   | 0.076  | 0.298** | 0.174 |

<sup>\*\*</sup>Correlation is significant at the 0.01 level (2-tailed). \*Correlation is significant at the 0.05 level (2-tailed) critical value: 0.2172. P  $\leq 0.05$ .

Despite considering non obese subjects in this study, FM showed some interesting trend and strength with other variables in male (weight,  $r = 0.816^{**}$ ; waist circumference,  $r = 0.890^{**}$  and BMI,  $r = 0.800^{**}$ ) and (weight,  $r = 0.793^{**}$ ; waist circumference,  $r = 0.689^{**}$  and BMI,  $r = 0.751^{**}$ ) in female group. However, both groups presented best correlation between fat% and fat mass: r = 0.956 in male, r = 0.944 in female followed by waist circumference and waist-height ratio: r = 0.925 in male, r = 0.916 in female.

## Correlation between measures of adiposity and cardiometabolic risk factors

Tables 5 and 6 show correlation between anthropometrics and cardiometabolic risk factors at

critical value: 0.2050 (male), 0.2172 (female);  $P \le 0.05$ . Among male subjects, higher values of waist-height ratio and waist-hip ratio were associated with increasing cardiometabolic risk factors (mean arterial blood pressure,  $r = 0.286^{**}$  and  $0.222^{*}$ ; triglyceride, r =0.0.318<sup>\*\*</sup> and 0.352<sup>\*\*</sup>; waist circumference,  $r = 0.925^{**}$ and 0.727\*\*), respectively, and overall association did not vary substantially among the indices. Associations were somewhat weaker in the female group with only waistheight ratio showing association with mean arterial blood pressure ( $r = 0.257^*$ ) and waist circumference (r =0.916\*\*). Gender differences in the correlation of obesity pattern with metabolic risk factors were further confirmed. Among the male group, waist-height ratio demonstrated the strongest gradient in association with risk factors, followed by waist-hip ratio. Though, the correlation of risk factors with waist-height ratio among female was weaker,

| Parameter | Wt      | HC      | WHtR    | WHpR    | BMI     | Fat (%) | FM      |
|-----------|---------|---------|---------|---------|---------|---------|---------|
| WC        | 0.797** | 0.842** | 0.925** | 0.727** | 0.851** | 0.825** | 0.890** |
| SBP       | -       | -       | 0.312** | 0.282*  | 0.241*  | -0.259* | 0.246*  |
| DBP       | 0.284*  | 0.271*  | 0.216*  | -       | 0.248*  | 0.212*  | 0.271*  |
| TG        | -       | -       | 0.318** | 0.352** | -       | -       | -       |
| HDL       | -       | -       | -       | -       | -       | -       | -       |
| FPG       | -       | -       | -       | -       | -       | -       | -       |

**Table 7.** Multivariate correlations of measures of adiposity and risk factor accumulations in male participants.

\*\*Correlation is significant at the 0.01 level (2-tailed). \*Correlation is significant at the 0.05 level (2-tailed). Critical value: 0.2050,  $P \le 0.05$ .

| Parameter | Wt      | HC      | WHtR    | WHpR    | BMI     | Fat (%) | FM      |
|-----------|---------|---------|---------|---------|---------|---------|---------|
| WC        | 0.721** | 0.648** | 0.916** | 0.692** | 0.774** | 0.589** | 0.689** |
| SBP       | -       | -       | 0.267*  | -       | -       | -       | -       |
| DBP       | -       | -       | 0.234*  | -       | -       | -       | -       |
| TG        | -       | -       | -       | -       | -       | -       | -       |
| HDL       | -       | -       | -       | -       | -       | -       | -       |
| FPG       | -       | -       | -       | -       | -       | -       | -       |

**Table 8.** Multivariate correlations of measures of adiposity and risk factor accumulations in female participants.

\*\*Correlation is significant at the 0.01 level (2-tailed). \*Correlation is significant at the 0.05 level (2-tailed) Critical value: 0.2172 . P: ≤0.05.

the existence of waist-height ratio in both groups was phenomenal and no correlation existed with fasting plasma glucose in both groups.

## Multivariate correlation of risk factors accumulation with measures of adiposity

Multivariate correlation of risk factors accumulation with various measures of adiposity at critical value: 0.2050 (male), 0.2172 (female);  $P \le 0.05$  is presented in Tables 7 and 8. The tables show the relationships of various measures of adiposity with risk factors accumulation. Waist-height ratio showed the strongest relationship with risk factors accumulation in male (waist circumference, r =  $0.925^{**}$ ; systolic blood pressure,  $r = 0.312^{**}$ ; diastolic blood pressure,  $r = 0.216^*$  and triglyceride,  $r = 0.318^{**}$ ) (Table 7). Waist-hip ratio also showed relationship with risk factors accumulation, but not as strong as waistheight ratio in the male group, and no relationship was found in the female group (Table 8). The common combination of risk factors accumulation was the existence of large waist circumference, high blood pressure and hypertriglyceridemia. Risk factors accumulation was present in male group not in female group.

## Logistic regression analysis of measures of adiposity and cardiometabolic risk factors

Logistic regression analyses were used to investigate the independent relation between the obesity indices and cardiometabolic risk factors after adjusting for age (\*significant at 95% confidence interval and  $P \le 0.05$ ). In the male group (Table 9), only waist-height ratio predicted the existence of large waist circumference (0.000\*) and two other components of metabolic syndrome, that is, elevated triglycerides (0.002\*) and systolic blood pressure (0.003\*). None of the obesity measures predicted elevated fasting blood glucose. No prediction of these combinations of cardiometabolic risk factors existed in the female group. Similarly, using the odd ratio values  $(R^2)$ , Figure 1 further showed the strength of waist-height ratio in predicting existence of risk factors accumulation in the male group (waist circumference,  $R^2 = 0.855$ ; systolic blood pressure,  $R^2 =$ 0.098 and triglyceride  $R^2 = 0.101$ ), while waist-hip ratio predicted systolic blood pressure ( $R^2 = 0.069$ ) and triglyceride ( $R^2 = 0.124$ ). Other measures of adiposity did not exhibit predictions with risk factors accumulation. No prediction was found for risk factors accumulation in female participants (Figure 2). Measures of adiposity did not show relationships with fasting plasma glucose, and

| Dependent variables predictors              | β     | R <sup>2</sup> | Significance |
|---|-------|----------------|--------------|
| Systolic blood pressure, waist-height ratio | 0.312 | 0.098          | 0.003*       |
| Waist-hip ratio                             | 0.263 | 0.069          | 0.007*       |
| Waist circumference, waist-height ratio     | 1.027 | 0.855          | 0.000*       |
| Height                                      | 0.393 | 0.144          | 0.000*       |
| Triglyceride waist-Hip ratio                | 0.352 | 0.124          | 0.001*       |
| Waist-height ratio                          | 0.318 | 0.101          | 0.002*       |

 Table 9. Regression analysis of obesity measures and cardiometabolic risk factors in male participants

\*Significant (95% CI, P ≤ 0.05).



**Figure 1.** Predictability of measures of adiposity for risk factors accumulation in male participants. WC – waist circumference, SBP – systolic blood pressure, DBP – diastolic blood pressure, HDL – high density lipoprotein, TG – triglyceride, FBG – fasting blood glucose.

no prediction was exhibited in both male and female groups.

#### Summary of result

In summary, statistical correlation analysis showed that four variables (waist-height ratio, waist circumference, fat% and fat mass) were sufficient to explain relationships among the factors being investigated in non-obese adults in this study.

1. Weight, waist-height ratio and waist circumference reflected strength of correlation among all anthropometric indices in both male and female groups.

2. Male group had higher base-line levels of total and abdominal obesity, triglyceride, low density lipoprotein and lower high density lipoprotein, fasting plasma glucose, systolic blood pressure, diastolic blood pressure and fat mass. 2. Fat mass, waist-height ratio and waist circumference in male group and waist-height ratio in female group were found to have the strongest correlation coefficients relative to at least three risk factors. Risk factors accumulation is present in male not in female.

3. Regression analysis showed that waist-height ratio was optimal for predicting components of cardiometabolic risk factors among non-obese male adults, there were no predictions found in the female group.

4. The variability of low density lipoprotein with other anthropometric indices was higher than that of high density lipoprotein, and no correlation or prediction was found for fasting plasma glucose in both groups.

#### Test of hypothesis

Based on the findings of this study, the test of hypothesis showed that among non-obese adult subjects investigated in this study:



**Figure 2.** Predictability of measures of adiposity for risk factors accumulation in female participants. WC – waist circumference, SBP – systolic blood pressure, DBP – diastolic blood pressure, HDL – high density lipoprotein, TG – triglyceride, FBG – fasting blood glucose.

 There were strong and positive relationships between anthropometric indices and risk factors accumulation.
 Central adiposity predicts cardiometabolic risk factors

better than overall obesity.

3. The initial null hypotheses were therefore rejected.

#### DISCUSSION

Metabolically obese normal weight (MONW) individuals, who despite having a normal weight BMI, present with metabolic disturbances typical of obese individuals. And the associated metabolic disorders among non obese are often ignored relatively to obesity (Ruderman et al., 1998; Succuro et al., 2008). The main findings of this study suggested that waist-height ratio provides a marginally superior tool for discriminating high cardiometabolic risks among non-obese adults compared with waist circumference, BMI and waist-hip ratio. Slightly higher odd ratios were observed in males compared to females, suggesting that discrimination is more precise on average, in male. Linear correlation analyses also showed that measures of adiposity were positively and significantly associated with cardiometabolic risk factors in both sexes, with the exception of fasting plasma glucose.

## Relationship between anthropometrics and cardiometabolic risk factors

The results from this study showed that WC, WHtR, fat

(%) and FM were the strength of correlation between anthropometrics and cardiometabolic risk factors. In addition, WHtR, HC and WC had a greater influence on cardiometabolic risk factors and the influence was more conspicuous among the male group of non-obese adults investigated. The correlation among the anthropometric indices observed in this study however shows that they carry the same information of adiposity and the associated health risk factors among non obese adults. The results are in agreement with the findings of other investigators who earlier showed that the concept of the metabolically obese normal weight individual is based on the observation that these same characteristics may be found in normal weight individuals with disorders often associated with obesity (St-Onge et al., 2004; Tsai, 2009).

## Correlation and prediction of cardiometabolic risk factors exhibited by WHtR

Palacios et al. (2011) showed in their study that general and abdominal adiposity were both associated with cardiometabolic risks, and that WC, WHpR and WHtR appear to be slightly better predictors than BMI among the population studied. Although several anthropometric indices serve as simple clinical tools for the measurement of central adiposity, it is not particularly clear which surrogate marker is the most reliable predictor of metabolic risk factor accumulation. BMI measurements in childhood are associated with abnormal metabolic clustering in adulthood, but the usefulness of BMI is limited for the following reasons: (1) its inability to distinguish fat from muscle mass; (2) its tendency to under-represent body fat distribution; and (3) its inability to measure central adiposity in a direct fashion, and for WC, there are currently no agreements about a health-related classification; varying percentile has been considered as a cut-off point for high WC. Both of these measures are also age and sex dependent (Vasan et al., 2011).

WHtR takes into account the distribution of body fat in the abdominal region which has been shown to be more associated with cardiovascular risks than body weight. WHtR adds significantly to cardiometabolic risk prediction over BMI and waist circumference in men, and it is an important index of central obesity, which is free from any bias due to hip width changes along with waist circumference of short and tall subjects (Dhall et al., 2011). The significant correlation and strength of prediction exhibited by WHtR in this study could then mean that it carries the burden of cardiometabolic risks among non obese individuals investigated in this study; so much so that the recommended optimal cut-off point of 0.5 for men and women (Browning et al., 2010) is higher in this study (0.508 in men and 0.535 in female). This is in agreement with the findings of Park et al. (2009) and Nambiar et al. (2010) who showed WHtR to be a better predictor of cardiovascular diseases than other anthropometric measurements, including BMI, WC, WHpR and skinfold thickness.

This observation was also reflected in recent studies on non obese adults by Gomes-ambrosi et al. (2011). Knowels et al. (2011) and Vasan et al. (2011) who proved that WHtR was the best predictor of both hypertension and dyslipidaemia for both male and female, and that BMI was the least accurate predictor of hypertension. Similar observation was also noted in this study, but more pronounced in male than female. The heterogeneity in findings across studies that have assessed cardiometabolic risk factors in relation to indices of adiposity may be attributable to difference in race, ethnicity, age and gender distribution of participants across study population (Duncan et al., 1995; Deurenberg et al., 1998).

WHtR is a recently introduced index to assess central fat distribution; an increased circumference is most likely associated with elevated risk factors because of its relation with visceral fat accumulation, and the mechanism may involve excess exposure of the liver to fatty acid. The combination of WC and Ht that is, WHtR could manifest better, the morphology of an enlarged abdomen with inappropriate short stature (Lin et al., 2002; Kayode et al., 2009). As was also observed in Kayode et al. (2009) and Wint et al. (2011), HC assessment in non-obese is more relevant than in obese individuals. This is particularly so because BMI does not reflect body fat distribution. Indeed, other anthropomeric

indices such as WC, HC, WHtR and WHpR have been used as alternatives.

This study also showed that both HC and WHpR were significantly correlated with various cardiometabolic risk factors, but in a smaller degree than WC and WHtR. This means that the use of Ht rather than HC adjusted for WC better indicates the clustering of cardiometabolic risk factors. Generally, incorporating either HC or Ht may provide more information on cardiometabolic risk than WC alone; the universal use of WC therefore may cause overestimation of risk factors in tall persons and underestimation in short persons (Ahmet et al., 2008).

Hypertension is now recognised globally as a major public health problem in terms of well-known risk factors for cardiometabolic diseases (Nobukazu et al., 2005; Ghosh and Bandyopadhyay, 2007). In this study, WHtR showed strong and consistent correlation with systolic blood pressure (SBP) > diastolic blood pressure (DBP) > mean arterial blood pressure (MABP) in both male and female groups, followed by WC, FM and abdominal skinfold. This fact could then mean that WHtR, WC and abdominal skinfold carry same information of visceral obesity. Therefore, the significant correlations with SBP, DBP and MABP observed also in this study could suggest that a decrease in intra-abdominal fat could reduce blood pressure and shows consistence with previous studies (Ilse and Luc, 2000; Wang and Hoy, 2004; Roopakala et al., 2009).

#### Metabolic syndrome among non-obese adults

Noteworthy, obesity is defined as an excess accumulation of body fat with the amount of this excess fat being responsible for most obesity associated health risks (Vasan et al., 2011). In this study, BMI cut off values, even at the highest quintile (27.0 in male and 26.60 in female) did not show strong associated risk of MetS compared to WC, WHtR and fat (%). The findings signify that although BMI may help in categorising obesity, it is not a reliable predictor for MetS in this study and may not reflect cardiometabolic risks clustering among non obese adults.

Since the correlation between BMI and central obesity can vary considerably from one individual to another, it has been suggested that what causes normal weight individuals to have MetS is having a greater body fat or abdominal obesity at a normal BMI range (Hadaegh et al., 2007). The association of uric acid with CVD is controversial (Tsai, 2009) but it is a risk factor in definition of MONW individuals (Ruderman et al., 1998).

Insulin resistance, not obesity (Baba et al., 2010), non alcoholic fatty liver disease (Kim et al., 2004) and independent role of leptin concentration (Esteghamati et al., 2011) have also been found to be significantly associated with MetS according to IDF definition and is independent of overall and central obesity among obese and non-obese subjects. This could also, in combination with familial and genetic background, explain the non correlation or prediction for FPG found in male and female of this study. Although mean fasting blood glucose was higher in female compared to male, none of the study population had fasting blood sugar greater than 5.6 mmol/L; more so, since fasting blood glucose did not show any correlation with anthropometrics and no prediction were shown, therefore it cannot be encountered as a metabolic risk factor in this study.

Similar finding was detected in a population of non obese Egyptian children (Saleh et al., 2010) and Iranian (Esmaillzadeh al., 2006) adolescents et with hypertriglyceridemic and waist phenotype, where they had higher prevalence of all metabolic risk factors except elevated fasting glucose. Dietary intake might be an important factor in determining blood glucose (Parillo and Riccardi, 2004). Moebus et al. (2011) also observed that the carbohydrate content and metabolic demands of last meal before the time of fasting blood glucose measurement, coupled with observed fasting state by subjects, might be another contributing factor. Infections (Ugwu et al., 2008) and stress (Wing et al., 1985) have also been mentioned.

Normal weight, low fitness individuals have also been identified. In a study of 855 coronary artery disease patients by Kashish et al. (2011), It was found that overweight, high-fitness subjects (determined by cardiopulmonary exercise testing) had a much lower risk of dying compared with normal-weight, low-fitness subjects. It is believed that a number of protective factors may come into play, including the site of excess weight (abdominal weight is more dangerous than weight carried around the hips), good physical activity levels, and following a healthy weight-reduction diet, along with normal metabolic biomarkers, such as blood pressure, blood sugar, and blood lipid levels.

#### Gender differences and prevalence of MetS

Studies on gender differences and prevalence by sex of MetS among non-obese supported results from this study which showed high prevalence of 20.4% in men and 15.3% in women (Hwang et al., 2007) and 15.5% in men and 10.5% in women (Lin et al., 2006). The most common component was hypertension and abdominal obesity. More so, the gender difference in the development of MetS among non-obese has been attributed to low serum sex-hormone-binding-globulin, low total testosterone and symptomatic androgen deficiency, and may provide early warning signs for cardiovascular risk and consequently an opportunity for early intervention in non-obese men (Varant et al., 2006). Generally, the hormonal environment plays a key role in determining body fat distribution. This is because sex hormones are known to affect regional fat deposition; the changing hormonal environment during puberty may contribute to the development of sex differences and large individual changes in fat distribution (Goran and Gower, 1999).

Gender specificity has also been attributed to the heterogeneity of two different anthropometric indices in prediction of cardiometabolic risk factors among nonobese males and females in this study. The females were more acclimatized to household work and not involved in vigorous physical activities voluntarily or habitually, thus disposing them to be more overweight or obese. On the other hand, males being in business sector also had sedentary behaviour along with omnipresent anxiety which comes with business competition, this not only predisposed them to prehypertension, it also increased the fat amount which might have exaggerated the already existing androidal fat pattern typical of males, thus the relation of WHtR among males (Dhall et al., 2011). Sexrelated differences in MetS prevalence are not universal; the differences within specific countries may also be due. for example, to differing socio-economic status, workrelated activities, and cultural views on body fat (Cameron et al., 2007).

#### CONCLUSION

Based on the findings of this study and in view of its limitations, the following conclusions were drawn:

1. Same anthropometric indices which were correlated in obese individuals, were also correlated among non-obese adults in this study.

2. This study clearly revealed the gender specificity and relative effectiveness of anthropometric indices in the relationship and prediction of cardiometabolic risks among non-obese adults.

3. Waist circumference and waist-height ratio mediate the correlation between cardiometabolic risk factors and anthropometrics among non-obese adults, with men being at greater risk than women.

4. Of all the anthropometric indices investigated, waistheight ratio appears to be better predictor of cardiometabolic risk factors among non-obese adults.

#### RECOMMENDATIONS

Based on the findings of this study, the following recommendations are proposed:

1. Optimal intervention strategies should be directed towards prevention of fat accumulation and improving fitness by a regular, coordinated and supervised exercise

programme and dietary modifications.

2. Waist-height ratio, as shown in this study, should be used by physicians as screening tool for metabolic abnormalities in persons with a BMI at the upper end of the normal weight and lower end of the overweight spectrum, since the early detection of metabolic obese normal weight individuals may be beneficial in the prevention of diabetes and cardiovascular disease.

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#### ABBREVIATIONS

BMI, Body mass index (kg/m<sup>2</sup>); CHD, coronary heart disease; CVD, cardiovascular disease; DBP, diastolic blood pressure (mmHg); FFM, fat free mass (kg); FM, fat mass (kg); HC, hip circumference (cm); HDL, high density lipoprotein (mmol/l); LDL, low density lipoprotein (mmol/l); MetS, metabolic syndrome; MABP; mean arterial blood pressure; SBP, systolic blood pressure (mmHg); T2DM, type 2 diabetes mellitus; TC, total cholesterol (mmol/l); TG, triglyceride (mmol/l); WC, waist circumference (cm); WHpR, waist-hip ratio; WHtR, waistheight ratio; Wt, body weight (kg).

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