Scientific Research Publishing

Food and Nutrition Sciences, 2025, 16(9), 1249-1264
https://www.scirp.org/journal/fns
ISSN Online: 2157-9458
ISSN Print: 2157-944X

# Metabolic Obesity Phenotypes and Risk of Chronic Kidney Disease: A Cross-Sectional Study from Communities in the Center Region of Cameroun

**Céline Sylvie Mimboe Bilongo[1,2*], Henriette Thérèse Dimodi[1], Anne Christine Abomo[1], Boris Ronald Tonou Tchuente[1], Marie-Modestine Kana Sop[2], Gabriel Medoua Nama[1]**

[1]Laboratory of Epidemiology and Nutritional Status, Centre for Food, Food Security and Nutrition Research, Institute of Medical Research and Medicinal Plant Studies, Yaoundé, Cameroon
[2]Department of Biochemistry, Faculty of Sciences, University of Douala, Douala, Cameroon
Email: *celinebilongo@yahoo.com

**How to cite this paper:** Mimboe Bilongo, C.S., Dimodi, H.T., Abomo, A.C., Tchuente, B.R.T., Kana Sop, M.-M. and Medoua Nama, G. (2025) Metabolic Obesity Phenotypes and Risk of Chronic Kidney Disease: A Cross-Sectional Study from Communities in the Center Region of Cameroun. *Food and Nutrition Sciences*, **16**, 1249-1264.
https://doi.org/10.4236/fns.2025.169071

**Received:** July 15, 2025
**Accepted:** September 21, 2025
**Published:** September 24, 2025



## Abstract

**Background and Aims:** Obesity is a risk factor for chronic kidney disease (CKD). Prior studies have identified two subtypes of obesity: metabolically healthy obesity (MHO) and metabolically unhealthy obesity (MUO), the latter being associated with an increased risk of CKD, while the condition of the former is contradictory and largely uninvestigated in Africa. **Methodology:** A cross-sectional study was conducted involving 317 obese participants (metabolically healthy subjects and metabolically unhealthy subjects) in two areas of the center region of Cameroon. Obesity was defined as a body mass index (BMI) of 27.0 kg/m$^2$ or greater. Chronic kidney disease (CKD) was evaluated using the estimated glomerular filtration rate (GFR) of less than 60 ml/min per 1.73 m$^2$, as per the CKD-EPI definition. **Results:** The prevalence of CKD was significantly higher in urban areas (28%) compared to peri-urban/rural areas (14.6%). Individuals with MUO phenotype had higher rates of CKD compared to participants with MHO phenotype; however, there was a difference in the prevalence of CKD between the metabolically healthy subjects in urban areas (24.6%) and in peri-urban/rural areas (14.3%). After adjustment for confounding factors, the risk factors for CKD in urban areas were: age over 50 years (OR: 7.54; 95% CI = 3.09 - 7.45), a high Total Cholesterol/HDL cholesterol ratio (TC/HDL ratio) (OR: 2.06; 95% CI = 0.97 - 4.41), and a high atherogenic index of plasma (AIP) (OR: 2.94; 95% CI = 1.16 - 7.45). MUO phenotype also had a higher risk of CKD, but the P-value was of borderline significance. The risk factors in peri-urban and rural areas were a high TC/HDL ratio (OR: 4.73; 95%

CI: 1.33 - 16.86) and high visceral fat (OR: 4.94; 95% CI: 1.33 - 16.86). **Conclusion:** While the MUO phenotype increased risk of chronic kidney disease (CKD), MHO phenotype is not a harmless condition. A substantial prevalence of CKD has also been observed among individuals with this phenotype.

## Keywords

Obesity Phenotypes, Chronic Kidney Disease, Urban and Peri-Urban/Rural Areas

## 1. Introduction

Chronic kidney disease (CKD), also known as chronic renal failure (CRF), is characterized by a significant decrease in kidney function, irrespective of the cause. This results in the kidneys' diminished ability to filter the body's blood effectively, a condition that is permanent and irreversible. CKD is a major public health issue, affecting over 10% of the global population, which equates to more than 800 million people. Furthermore, it has emerged as one of the leading causes of death in the twenty-first century [1]. In Africa, the prevalence of CKD is often underestimated due to its silent nature and the fact that most prevalence studies conducted in Africa are suboptimal [2]. A meta-analysis published in 2018 revealed that the overall prevalence of CKD was higher in Sub-Saharan Africa compared to North Africa [3]. In Cameroon, a study by Kaze *et al.* (2015) found a higher prevalence of chronic kidney disease in rural areas compared to urban areas, with rates of 14.1% and 10.9%, respectively [4]. Furthermore, Aseneh *et al.* (2020) demonstrated that the prevalence in urban areas varied between 10.0% and 14.2%.

Obesity is a significant risk factor for CKD and end-stage renal disease due to its associated conditions. In fact, the global prevalence of chronic kidney disease is on the rise in correlation with obesity and metabolic disorders [5]. A particular study discovered that being overweight or obese heightens the risk of developing CKD, regardless of the presence of diabetes, hypertension, or cardiovascular disease [6].

Although the indirect effects of obesity on the kidneys are well understood, primarily through the development of other risk factors for chronic kidney disease, such as diabetes and arterial hypertension, some obese individuals exhibit few or no metabolic complications [7]. These subjects are referred to as "metabolically healthy obese" (MHO). However, the association between the MHO phenotype and chronic kidney disease remains largely unknown, and the studies reviewed present contradictory findings. One study has demonstrated that this phenotype is associated with a lower risk of developing kidney failure [8], while others have indicated that this phenotype, like its counterpart with metabolic abnormalities, is associated with a high risk of developing kidney failure [5] [9]. In Africa, few studies have evaluated the relationship between the obesity phenotype and chronic kidney disease [10]-[14], and in Cameroon [3] [15] [16], most tend to focus on discussing risk factors and the prevalence of chronic renal failure. The aim of this study was therefore to investigate the association between the obesity phenotype and the risk of chronic kidney disease in an adult population in both urban and peri-urban/rural areas of Cameroon.

## 2. Methodology

### 2.1. Study Design

This cross-sectional study was carried out in three localities in the Centre Region of Cameroon, from March to July 2022 in urban localities (Yaoundé) and from September to October 2022 in peri-urban/rural localities (Obala and Efok). The study involved participants of both sexes, aged between 20 and 75 years. We used a consecutive sampling technique, and all volunteers who met the inclusion criteria were recruited. Physically disabled individuals, those with mental disorders (delirium, mental confusion), seriously ill individuals (extreme thinness, visible breathing difficulties, terminal cancer), breastfeeding and pregnant women, and people suffering from known kidney disease were excluded. A questionnaire was developed for this study and was utilized to collect the following information from each participant: sex, age, marital status, level of education, and place of residence. The sample size was calculated using Magnani's formula, considering a 95% confidence level, a 5% margin of error, and the prevalence of obesity in Cameroon, which was 9.6% in 2016 [17]. In the end, the study included 317 participants, with 183 from urban areas and 134 from semi-urban/rural areas. The study included men and women aged between 20 and 70 years who had been residing in the study areas for at least 6 months, had provided their informed consent, and had a BMI of ≥27 kg/m$^2$.

### 2.2. Ethical Considerations

This work received approval from the Centre Regional Ethics Committee for Human Health Research under the reference number CE N˚0885/GRERSHC/2022. The administrative authorities overseeing the selected study settings also granted their approval. Participants who agreed to partake in the study signed a free and informed consent form. The study adhered to the Declaration of Helsinki on human medical ethics.

### 2.3. Physical and Clinical Examination

Anthropometric measurements were carried out by trained personnel using standard procedures. Wearing minimal clothing, subjects were weighed to the nearest 0.01 kg decimal place using an electronic scale (OMRON Body Composition Monitor BF 508). Height was measured to the nearest 0.1 cm decimal place using a portable gauge. The body mass index (BMI) was calculated as weight (kg)/height (m)$^2$. Waist circumference was measured to the nearest 0.1 cm using a non-elastic tape measure at the midpoint between the subcostal and supracostal planes, taken at the end of a normal exhalation. Hip circumference was also measured to the nearest 0.1 cm using a tape measure after asking the participant to empty their pockets.

Body composition was determined using an OMRON Body Composition Monitor (BF 508). Blood pressure was measured at the elbow crease, in participants who had been at rest for at least 10 minutes, using an OMRON Electronic Radial Sphygmomanometer.

After fasting for 10 to 12 hours, approximately 6 ml of venous blood was collected from the participants into dry tubes. The blood was then centrifuged, and the resulting serum was used to perform the following assays: total cholesterol, HDL cholesterol, triglycerides, urea, and creatinine. These assays were conducted spectrophotometrically using kits from the Cypress brand. Low-density lipoprotein cholesterol (LDLc) was estimated using the Friedewald equation [18]. Fasting blood glucose was measured using the glucose oxidase peroxidase method (GOP-POD) with a glucometer and test strips (One-Touch Plus), applied directly to the participant's fingertip.

## 2.4. Diagnosis of Cardiometabolic Risk Factors and Determination of Obesity Phenotypes

The metabolic status was determined using the definition of NCEP-ATP III (National Cholesterol Education Program Adult Treatment Panel III). According to this definition, a person was considered metabolically healthy if they had fewer than three of the following risk factors: increased waist circumference (>102 cm for men, >88 cm for women), elevated triglycerides (≥1.50 g/l), low HDL cholesterol (<0.4 g/l in men, <0.5 g/l in women), hypertension (≥130/≥85 mmHg), and impaired fasting glucose (>1.0 g/l) [19].

A BMI threshold of >27 kg/m$^2$ has been used to diagnose obesity. This is because a BMI of ≈27 kg/m$^2$ for both men and women corresponds to a weight 20% higher than desirable. It is associated with an increased risk of hypertension, hypercholesterolemia, and diabetes mellitus, as well as premature death [20]. According to Suwala's study in 2024, a novel threshold for BMI (27.6 kg/m$^2$) was suggested, which increases the risk of cardiovascular disease by 3.3 - 5.3 times, depending on gender [21]. Colman's study in 2012 guidelines recommended that only people with a BMI > 27 kg/m$^2$ should be eligible for drug treatment for obesity [20]. Furthermore, a study by Nguedjo *et al.* in 2022 found that in Cameroon, people with a BMI ≥ 27 kg/m$^2$ were considered obese [22]. Obesity phenotypes were classified into two groups:

- Metabolically healthy obese individual (MHO), having fewer than three cardiometabolic risk criteria.
- Metabolically unhealthy obesity (MUO), presenting three or more cardiometabolic risk criteria.

We calculated the atherogenicity indices from the lipid balance parameters: the atherogenic index of plasma (AIP) and the TC/HDL ratio (total cholesterol/high-density lipoprotein). The AIP was calculated as the logarithm of the ratio of triglycerides to HDL (high-density lipoprotein) (log [TG/HDL]). An AIP greater than 0.15 was considered high [23]. This was used to determine cardiovascular risk.

## 2.5. Diagnosis of Chronic Kidney Disease

The glomerular filtration rate (GFR) was estimated using the CKD epidemiology collaboration (CKD-EPI) formula.

- $GFR = 141 \times \min(Scr/\kappa, 1)^{\alpha} \times \max(Scr/\kappa, 1)^{-1.209} \times 0.993^{Age} \times 1.018$ [if female] $\times$ 1.159 [if black].
- Scr is serum creatinine (mg/dl).
- $\kappa$ is 0.7 for females and 0.9 for males.
- $\alpha$ is −0.329 for females and −0.411 for males.
- min indicates the minimum of $Scr/\kappa$ or 1, and max indicates the maximum of $Scr/\kappa$ or 1 [24].

We used online software to automatically calculate it. A normal GFR ranges between 90 - 120 ml/min/1.73m$^2$. According to the KDIGO guidelines, a GFR less than 60 ml/min/1.73m$^2$ is considered pathological.

## 2.6. Statistical Analysis

We analyzed the data using SPSS (Statistical Package for Social Sciences) Version 25. We checked the data for extremes, outliers, and missing values, making corrections where necessary. Categorical variables were presented as frequency (N) and percentage (%), and quantitative variables as mean ± standard deviation (M ± SD). We used the chi-square test to compare the characteristics of participants in urban and peri-urban/rural areas and the Student's t-test to compare the means of participants in the two areas and between the two phenotypes. We performed binary logistic regression analyses to assess the risk factors predictive of CKD in our population. We ran two models: Model 1 (unadjusted) and Model 2 (adjusted for the covariates sex, waist circumference, triglycerides, hypertension, high-density lipoprotein (HDL), blood glucose, and BMI). The significance level was $P < 0.05$ for all tests performed.

# 3. Results

Table 1 presents the characteristics of the population in different areas. Of the 317 participants enrolled, the MHO phenotype was predominant among our obese individuals, accounting for 62.8% in urban areas and 64.7% in peri-urban/rural areas. Females were more represented in both areas; the prevalence of MHO increased with age in both areas.

Regarding anthropometric parameters, in urban areas, MUO individuals had significantly higher mean waist circumference, weight, visceral fat, and waist/hip circumference ratio than MHO individuals. In peri-urban and rural areas, only

**Table 1.** Characteristics of participants (urban and peri-urban/rural areas) according to metabolic status.

| Parameter | | Urban area | | | Peri-urban/rural area | | |
|---|---|---|---|---|---|---|---|
| | | MHO N = 115 (62.8) | MUO N = 68 (37.2) | P-value | MHO N = 86 (64.7) | MUO N = 47 (35.3) | P-value |
| **Gender** | Female | 99 (66.9) | 49 (33.1) | 0.294 | 71 (62.8) | 42 (37.2) | 0.020* |
| | Male | 16 (45.7) | 19 (54.3) | | 15 (75) | 5 (25) | |
| **Age (years)** | 20 - 39 | 34 (75.6) | 11 (24.4) | 0.084 | 20 (76.9) | 6 (23.1) | 0.205 |
| | 40 - 59 | 57 (61.3) | 36 (38.7) | | 44 (64.7) | 24 (35.3) | |
| | >60 | 24 (53.3) | 21 (46.7) | | 21(55.3) | 17 (44.7) | |
| **Marital status** | Married | 71 (61.7) | 44 (38.3) | 0.027* | 39 (60.9) | 25 (39.1) | 0.267 |
| | Unmarried | 52 (77.6) | 15 (22.4) | | 47(70.1) | 20 (29.9) | |
| **Level study** | Not at school | 2 (40) | 3 (60) | 0.533 | 15 (60) | 10 (40) | 0.124 |
| | Primary | 20 (60.6) | 13 (39.4) | | 26 (53.1) | 23 (46.9) | |
| | Secondary | 40 (58.8) | 28 (41.2) | | 37 (77.1) | 11 (22.9) | |
| | University | 52 (69.3) | 23 (30.7) | | 7 (77.8) | 2 (22.2) | |
| **Waist circumference (cm)** | | 99 ± 12.46 | 107.27 ± 10.49 | 0.000* | 96.57 ± 11.34 | 100.93 ± 8.7 | 0.024* |
| **Weigh (kg)** | | 87.34 ± 13.1 | 95.58 ± 15.28 | 0.000* | 83.42 ± 14.02 | 86.79 ± 11.80 | 0.046 |
| **BMI (kg/m$^2$)** | | 33.57 ± 5.31 | 35.55 ± 5.85 | 0.020* | 31.25 ± 3.66 | 32.52 ± 4.42 | 0.080 |
| **Body fat (%)** | | 43.62 ± 8.53 | 43.80 ± 8.94 | 0.896 | 41.57 ± 6.9 | 43.86 ± 6.66 | 0.075 |
| **Visceral fat** | | 11.06 ± 3.3 | 13.44 ± 3.78 | 0.000* | 10.49 ± 3.65 | 11.37 ± 2.76 | 0.160 |
| **WHR** | | 0.85 ± 0.10 | 0.91 ± 0.10 | 0.000* | 0.86 ± 0.91 | 0.87 ± 0.64 | 0.508 |
| **SBP (mmHg)** | | 126.08 ± 18.83 | 140.04 ± 21.49 | 0.000* | 130.50 ± 23.47 | 141.91 ± 21.65 | 0.007* |
| **DBP (mmHg)** | | 79.65 ± 12.44 | 89.38 ± 13.22 | 0.000* | 81.65 ± 12.84 | 87.89 ± 11.67 | 0.007* |
| **Glycémia (mg/dl)** | | 90.27 ± 10.1 | 100.08 ± 34.4 | 0.005* | 87.65 ± 20.24 | 100.97 ± 45.02 | 0.021* |
| **TC (g/l)** | | 2.44 ± 1.11 | 2.69 ± 1.09 | 0.135 | 1.87 ± 0.80 | 1.87 ± 0.51 | 0.981 |
| **HDL (g/l)** | | 0.55 ± 0.19 | 0.5 ± 0.21 | 0.150 | 0.54 ± 0.17 | 0.45 ± 0.15 | 0.006* |
| **LDL (g/l)** | | 1.68 ± 1.07 | 1.82 ± 1.09 | 0.396 | 1.11 ± 0.76 | 1.15 ± 0.47 | 0.762 |
| **TG (g/l)** | | 0.96 ± 0.45 | 1.76 ± 1.12 | 0.000* | 1.24 ± 0.65 | 1.60 ± 0.59 | 0.002* |
| **TC/HDL** | | 4.76 ± 2.1 | 5.91 ± 2.97 | 0.003* | 3.71 ± 1.67 | 4.51 ± 1.98 | 0.015* |
| **AIP** | | 0.21 ± 0.27 | 0.49 ± 0.28 | 0.000* | 0.32 ± 0.25 | 0.53 ± 0.20 | 0.000* |
| **Creat (mg/l)** | | 10.84 ± 2.17 | 11.54 ± 2.03 | 0.033* | 10.13 ± 2.32 | 9.98 ± 2.51 | 0.725 |
| **Urea (g/l)** | | 0.24 ± 0.10 | 0.27 ± 0.13 | 0.139 | 0.29 ± 0.14 | 0.32 ± 0.13 | 0.282 |
| **GFR (ml/min/1.72m$^2$)** | | 77.89 ± 22.05 | 72.87 ± 20.71 | 0.131 | 90.28 ± 30.01 | 89.57 ± 25.95 | 0.882 |

Data were expressed in terms of numbers (n) and frequencies (%), and in terms of mean ± standard deviation. MHO: Metabolically Healthy Obese; MUO: Metabolically Unhealthy Obese; BMI: Body Mass Index; WHR: Waist-to-Hip Ratio; SBP: Systolic Blood Pressure; DBP: Diastolic Blood Pressure; TC: Total Cholesterol; HDL: High-Density Lipoprotein; LDL: Low-Density Lipoprotein; TG: Triglycerides; Creat: Creatinemia; AIP: Atherogenic Index of Plasma; GFR: Glomerular Filtration Rate. *P < 0.05 is significant.

the means of waist circumference and weight were significantly higher in MUO individuals compared to MHO. However, the means of SBP, DBP, glycemia, and triglycerides were significantly higher in MUO participants in the two areas. Conversely, the means of HDL-C were significantly higher in MHO individuals compared to MUO individuals. We found no significant difference in the glomerular filtration rate between the two obese subgroups in the different areas. Cardiovascular risk, as determined by the atherogenicity indices (TC/HDL and the logarithm of the TG/HDL ratio), was significantly higher in the MUO group in both urban and peri-urban/rural areas.

Figure 1 displays the prevalence of CKD according to study areas. It reveals that CKD was significantly more prevalent in urban areas than in semi-urban/rural areas, with rates of 28% and 14.6%, respectively ($P < 0.05$). Figure 2 illustrates the prevalence of CKD as a function of obesity phenotype in the different populations. The prevalence of CKD was higher in MUO individuals in both urban and semi-urban/rural areas, at 33.8% and 15.2%, respectively. Conversely, the prevalence of CKD was higher in MHO individuals in urban areas (24.6%) compared to those in semi-urban/rural areas, but the difference was not statistically significant.

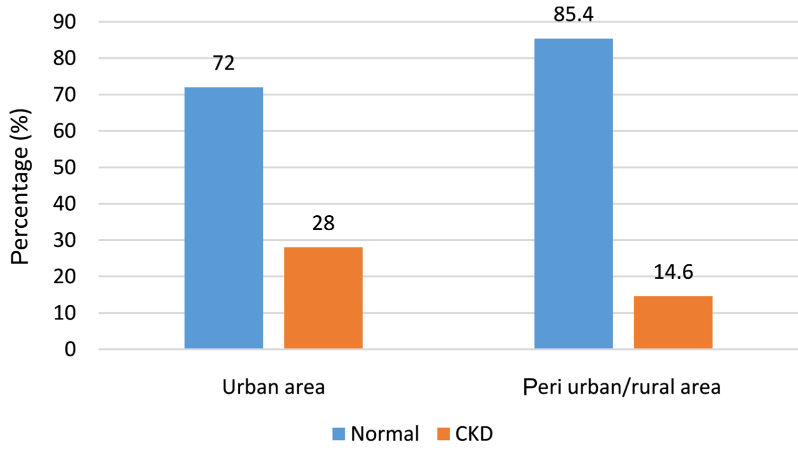


Figure 1. Prevalence of chronic kidney disease in different areas.

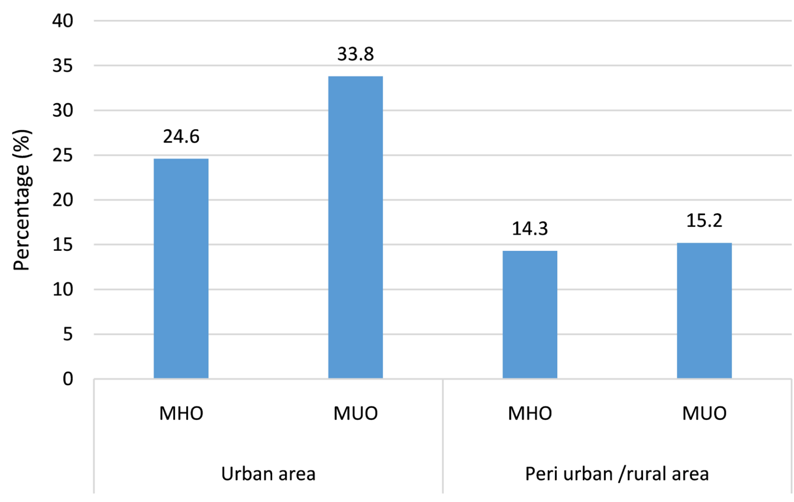


Figure 2. Prevalence of chronic kidney disease as a function of metabolic status in the different zones.

The risk factors for CKD in our study population were: urban area [2.274 (1.268 - 4.080)]; age groups of 40 - 59 years [5.711 (1.68 - 19.33)] and over 60 years [16.87 (4.90 - 58.11)]; high visceral fat [2.33 (1.207 - 4.507)]; and high TC/HDL ratio [2.134 (1.24 - 3.66)]; the differences were significant (P-value < 0.05) (Table 2).

**Table 2.** Risk factors associated with the onset of renal failure in the general population.

| Parameters | | OR (95% CI) | P-value |
| --- | --- | --- | --- |
| **Zone** | peri-urban | 1 | 0.006* |
| | Urban | 2.274 (1.268 - 4.080) | |
| **Sex** | Male | 1 | 0.063 |
| | Female | 2.227 (0.959 - 5.17) | |
| **Age (years)** | 20 - 39 | 1 | |
| | 40 - 59 | 5.711 (1.687 - 19.33) | 0.005* |
| | >60 | 16.87 (4.90 - 58.11) | 0.000* |
| **Metabolic status** | MHO | 1 | 0.214 |
| | MUO | 1.41 (0.82 - 2.42) | |
| **Visceral fat** | Normal | 1 | 0.012* |
| | High | 2.33 (1.207 - 4.507) | |
| **Waist circumference (cm)** | Normal | 1 | 0.498 |
| | High | 1.272 (0.635 - 2.549) | |
| **BMI (kg/m²)** | <30 | 1 | 0.835 |
| | >30 | 0.941 (0.529 - 1.672) | |
| **Blood glucose (g/l)** | Normal | 1 | 0.054 |
| | High | 1.935 (0.990 - 3.78) | |
| **Triglycerides (g/l)** | Normal | 1 | 0.280 |
| | High | 0.722 (0.400 - 3.66) | |
| **Blood pressure (mmHg)** | Normal | 1 | 0.280 |
| | High | 1.345 (0.785 - 2.305) | |
| **HDL** | Normal | 1 | 0.679 |
| | Low | 0.888 (0.505 - 1.560) | |
| **Ratio TC/HDL** | Normal | 1 | 0.006* |
| | High | 2.134 (1.245 - 3.660) | |
| **AIP** | Normal | 1 | 0.551 |
| | High | 1.215 (0.640 - 2.308) | |

MHO: Metabolically Healthy Obese; MUO: Metabolically Unhealthy Obese; BMI: Body Mass Index; AIP: Atherogenic Index of Plasma; TC/HDL: Ratio Total Cholesterol/High Density Lipoprotein; *P < 0.05.

After adjusting for covariates, the risk factors for CKD in urban areas were age greater than 50 years (OR: 7.54; 95% CI = 3.09 - 7.45), high TC/HDL ratio (OR:

2.06; 95% CI = 0.97 - 4.41), and high AIP (OR: 2.94; 95% CI = 1.16 - 7.45). MUO phenotype (OR: 2.12; 95% CI = 0.92 - 7.72) and visceral fat (OR: 2.70; 95% CI = 0.95 - 7.66) also had a higher risk of CKD, but the P-value was not significant. The risk factors in semi-urban/rural areas were high TC/HDL ratio (OR: 4.73; 95% CI = 1.33 - 16.86) and high visceral fat (OR: 4.94; 95% CI = 1.33 - 16.86) (Table 3).

**Table 3.** Factors associated with the onset of renal failure in the different zones.

| **Parameters** | | **Urban area** | | **Peri-urban/rural areas** | |
|---|---|---|---|---|---|
| | | OR (95% CI) | | OR (95% CI) | |
| | | Model I | Model II | Model I | Model II |
| **Metabolic status** | MHO | 1 | 1 | 1 | 1 |
| | MUO | 1.57 (0.81 - 3.03) | 2.12 (0.92 - 7.72) | 1.07 (0.39 - 2.95) | 0.42 (0.067 - 2.65) |
| **Visceral fat** | Normal | 1 | 1 | 1 | 1 |
| | High | 1.83 (0.81 - 4.14) | 2.70 (0.95 - 7.66) | 2.78 (0.86 - 8.93) | 4.94 (1.14 - 21.34)* |
| **Ratio TC/HDL** | Normal | 1 | 1 | 1 | 1 |
| | High | 1.63 (0.85 - 3.12) | 2.06 (0.97 - 4.41)* | 2.76 (1.01 - 7.51) | 4.73 (1.33 - 16.86)* |
| **AIP** | Normal | 1 | 1 | 1 | 1 |
| | High | 1.42 (0.67 - 3.013) | 2.94 (1.16 - 7.45)* | 1.185 (0.31 - 4.45) | 2.09 (0.40 - 10.71) |
| **Age (years)** | <50 | 1 | 1 | 1 | 1 |
| | ≥50 | 5.66 (2.61 - 12.27) | 7.54 (3.09 - 18.4)* | NA | NA |
| **WHR** | Normal | 1 | 1 | 1 | 1 |
| | High | 0.92 (0.48 - 1.76) | 1.12 (0.54 - 2.34) | 2.20 (0.74 - 6.54) | 2.66 (0.72 - 9.78) |

Model I: Unadjusted; Model II: Adjusted for sex, waist circumference, triglycerides, hypertension, High-Density Lipoprotein (HDL), blood glucose, and body mass index. *$P < 0.05$. Ratio CT/HDL: Ratio Total Cholesterol/High-Density Lipoprotein; AIP: Atherogenic Index of Plasma; WHR: Waist-to-Hip Ratio.

## 4. Discussion

The aim of this study was to investigate the association between obesity phenotypes and the risk of chronic kidney disease in an adult population in urban and peri-urban/rural areas in the Center region of Cameroon. The main finding of our study is that the MUO phenotype was associated with a higher prevalence of CKD; this prevalence was higher in urban areas. Our study showed a high prevalence of the metabolically healthy obesity (MHO) phenotype, with a prevalence of 62.8% in urban areas and 64.7% in peri-urban/rural areas. The prevalence of metabolically healthy obese individuals is consistent with previous studies, which found a prevalence ranging from 6% to 75% [25]. A study conducted by Mbanya *et al.* in 2015 showed that approximately 85.8% of overweight or obese Cameroonians had a healthy profile, with a significantly higher proportion in rural areas in terms of standard cardio-metabolic risk factors [26].

We found a high prevalence of CKD in urban areas (28%) compared with peri-urban/rural areas, where the prevalence was 14.6%. Our results contradict those obtained by Kaze *et al.* in 2015, who studied urban and rural populations in West Cameroon and found a higher prevalence of chronic kidney disease in rural areas than in urban areas (14.1% and 10.9%, respectively) [4]. The high prevalence we observed in urban areas may be due to our population consisting entirely of overweight individuals, as obesity is associated with the development and progression of chronic kidney disease. Furthermore, the prevalence of chronic kidney disease seems to correlate with the increase in adiposity in Sub-Saharan Africa [12].

In the present study, 24.6% of MHO subjects were suffering from CKD in urban areas, compared with 14.3% in peri-urban/rural areas. This difference in prevalence may be due to urbanization. Various studies conducted in low- and middle-income countries have shown that rapid urbanization is at the root of an increased prevalence of metabolic diseases, which coincide with an increase in the prevalence of CKD [27]. This adds to a growing body of evidence indicating that the MHO phenotype is not a benign state [5] [9]. Several studies have suggested that overweight and obese patients are not protected from the risk of CKD by healthy metabolic profiles [5] [28] [29]. Obesity itself could be harmful to renal function, and there could be other mechanisms directly linking obesity to renal damage, independently of metabolic risk factors [30].

The results of our study showed that living in an urban area was a predictive risk factor for the development of CKD, which is consistent with studies conducted in Ghana [31] and Congo [32]. The age groups of 40 - 59 years [5.711 (1.68 - 19.33)] and more than 60 years [16.87 (4.90 - 58.11)], high visceral fat [2.33 (1.207 - 4.507)], and a high arterogenicity index [2.134 (1.24 - 3.66)] were also risk factors for the occurrence of CKD in our population. Indeed, the glomerular filtration rate (GFR) decreases with age, and the prevalence of CKD is higher in the elderly [33].

After adjusting for waist circumference, triglycerides, hypertension, HDL, glycaemia, sex, and BMI, age greater than 50 years and a high atherogenic index (AIP and TC/HDL ratio) were identified as risk factors for the onset of CKD in urban areas. The glomerular hyperfiltration characteristic of a high BMI generally begins at a young age. Therefore, it is possible that the progressive loss of renal function becomes more evident in individuals who have been exposed to the effects of obesity for a longer period, which could explain the increase in renal failure with age [34]. It should also be noted that ageing itself leads to an increase in glomerular permeability, a decrease in individual glomerular volume, glomerular sclerosis, and a decrease in the number of nephrons [35].

Most obese individuals are prone to lipid disorders such as dyslipidemia. Atherogenic indices, which are assessed by various lipid profile ratios including the atherogenic index of plasma (AIP) and the TC/HDL ratio, serve as indicators of abnormal lipid metabolism and are identified as significant contributors to atherosclerosis and cardiovascular disease (CVD) [36]. Our findings suggest that a high TC/HDL ratio or a high AIP escalates the risk of chronic kidney disease (CKD) in obese individuals residing in urban areas. Similar results have been found by other researchers, notably Huang *et al.* and Li *et al.*, who conducted their studies on a population of Chinese adults [36] [37]. The pathogenic mechanism underlying the increased risk of kidney problems associated with AIP is unclear. However, there are several possible explanations. First, AIP can serve as an indirect indicator of the presence of smaller LDL-C particles, which are associated with an increased risk of atherogenicity [38]. Secondly, a decrease in HDL-C levels leads to a reduction in reverse cholesterol transport and an accumulation of lipids in the glomeruli. Consequently, the accumulation of foam cells results in glomerulosclerosis and the progression of renal dysfunction [39]. Thirdly, elevated AIP is associated with insulin resistance, and the resulting hyperinsulinemia appears to play a role in renal function by inducing glomerular hyperfiltration and increased vascular permeability [40]. Hyperfiltration can be a sign of early kidney damage and can lead to further progression of the disease, particularly in conditions like diabetes and obesity.

The risk factors for the development of CKD in peri-urban/rural areas include a high TC/HDL ratio (OR: 4.73; 95% CI = 1.33 - 16.86) and high levels of visceral fat (OR: 4.94; 95% CI = 1.33 - 16.86). In this area also, we found that, although the P-value was not significant, the odds of CKD in individuals with a high waist-to-hip ratio (WHR) increased from 2.20 in the unadjusted model to 2.66 in the adjusted model; prior studies have shown that a higher WHR, indicating a greater proportion of visceral fat, is associated with an increased risk of developing CKD [41]. It is important to note that, in obese individuals, visceral adipose tissue plays a significant role in the development of metabolic complications. This is because it is much more metabolically and hormonally active than its subcutaneous counterpart. Additionally, it has pro-inflammatory properties and is subject to lipolysis [42]. An increase in visceral adipose tissue leads to hyperfiltration and hyperperfusion of the renal glomeruli, which can result in glomerular hypertrophy, proteinuria, and the development of CKD [43]. Our findings align with those obtained by Lee and colleagues, who demonstrated that visceral adipose tissue is a risk factor for the progression of CKD in Korean adults [44].

Although not significant, the present study also indicated that in urban areas, after adjusting for potential confounders, the odds of CKD in the MUO phenotype increased (2.12 times higher) compared to the MHO phenotypes, while these odds decreased in peri-urban/rural areas. The same factor can both increase and decrease the odds in two areas; in this case, this may be explained by the fact that rapid urbanization is often associated with the adoption of Western diets, lifestyle changes, and the consumption of highly processed foods that contribute to decreased physical activity and an increased risk of metabolic and cardiovascular diseases [45]. In addition, the risk of CKD increases as the number of metabolic syndrome components a person has. A study by Chen *et al.* shows that individuals with metabolic syndrome (MetS) had a 2.6-fold increased risk of developing incident chronic kidney disease (CKD), defined as an estimated glomerular filtration rate (eGFR) of less than 60 ml/min, compared to those without MetS. The risk of

CKD increased with the number of MetS components, rising from 1.89 in individuals with one component to 5.85 in adults with all five. Adults with MetS had twice the chance of developing microalbuminuria compared to adults without. The risk of microalbuminuria increased gradually with the number of MetS components [46].

There are also several limitations of this analysis, including the use of single measurements of creatinine; however, as subjects were not acutely ill at the time of study evaluation, these values are likely consistent with chronic kidney function. A second limitation is the lack of albuminuria data, an important risk factor for the development of kidney disease that may also be associated with obesity. Third, the cross-sectional design, which prevents causal inference, the use of a single GFR measurement to diagnose CKD, and the non-random sampling method represent additional limitations.

## 5. Conclusion

In conclusion, our study examined the association between obesity phenotypes and the risk of chronic kidney disease (CKD) in an adult population living in urban, peri-urban/rural areas of Cameroon. Our results suggest that although the MUO phenotype is associated with a higher risk of CKD, the MHO phenotype is not a harmless condition, a substantial prevalence of CKD was also observed among individuals with this phenotype. Therefore, relying solely on standard metabolic markers may underestimate the true risk. Additional factors such as visceral adiposity and atherogenic indices should be integrated into the assessment of metabolic health to better capture the complexity of risk in obese populations.

## Acknowledgements

The authors appreciate the cooperation of the patients in this study.

## Ethics Approval and Consent to Participate

This work received approval from the Centre Regional Ethics Committee for Human Health Research under the reference number CE N˚0885/GRERSHC/2022. The administrative authorities overseeing the selected study settings also granted their approval. Participants who agreed to partake in the study signed a free and informed consent form. The study adhered to the Declaration of Helsinki on human medical ethics.

## Availability of Data and Materials

All data generated or analyzed during this study are included in this published article.

## Funding

This research received specific funding from the Public Investment Budget of IMPM/MINRESI.

## Authors' Contributions

CSMB, GMN, MMKS and HTD designed the study plan; GMN, MMKS, and HTD planned the work; HTD, BRTT, ACA, and CSMB collected the data; CSMB and BRTT analyzed the data; CSMB wrote the manuscript; HTD, BRTT, ACA, MMKS, and GMN read and revised the article. All authors read and approved of the final manuscript.

## Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

## References


[1] Kovesdy, C.P. (2022) Epidemiology of Chronic Kidney Disease: An Update 2022. *Kidney International Supplements*, **12**, 7-11. https://doi.org/10.1016/j.kisu.2021.11.003

[2] Hariparshad, S., Bhimma, R., Nandlal, L., Jembere, E., Naicker, S. and Assounga, A. (2023) The Prevalence of Chronic Kidney Disease in South Africa—Limitations of Studies Comparing Prevalence with Sub-Saharan Africa, Africa, and Globally. *BMC Nephrology*, **24**, Article No. 62. https://doi.org/10.1186/s12882-023-03109-1

[3] Kaze, A.D., Ilori, T., Jaar, B.G. and Echouffo-Tcheugui, J.B. (2018) Burden of Chronic Kidney Disease on the African Continent: A Systematic Review and Meta-Analysis. *BMC Nephrology*, **19**, Article No. 125. https://doi.org/10.1186/s12882-018-0930-5

[4] Kaze, F.F., Meto, D.T., Halle, M., Ngogang, J. and Kengne, A. (2015) Prevalence and Determinants of Chronic Kidney Disease in Rural and Urban Cameroonians: A Cross-Sectional Study. *BMC Nephrology*, **16**, Article No. 117. https://doi.org/10.1186/s12882-015-0111-8

[5] Chang, Y., Ryu, S., Choi, Y., Zhang, Y., Cho, J., Kwon, M., *et al.* (2016) Metabolically Healthy Obesity and Development of Chronic Kidney Disease. *Annals of Internal Medicine*, **164**, 305-312. https://doi.org/10.7326/m15-1323

[6] Herrington, W.G., Smith, M., Bankhead, C., Matsushita, K., Stevens, S., Holt, T., *et al.* (2017) Body-Mass Index and Risk of Advanced Chronic Kidney Disease: Prospective Analyses from a Primary Care Cohort of 1.4 Million Adults in England. *PLOS ONE*, **12**, e0173515. https://doi.org/10.1371/journal.pone.0173515

[7] Lin, L., Peng, K., Du, R., Huang, X., Lu, J., Xu, Y., *et al.* (2017) Metabolically Healthy Obesity and Incident Chronic Kidney Disease: The Role of Systemic Inflammation in a Prospective Study. *Obesity*, **25**, 634-641. https://doi.org/10.1002/oby.21768

[8] Hashimoto, Y., Tanaka, M., Okada, H., Senmaru, T., Hamaguchi, M., Asano, M., *et al.* (2015) Metabolically Healthy Obesity and Risk of Incident CKD. *Clinical Journal of the American Society of Nephrology*, **10**, 578-583. https://doi.org/10.2215/cjn.08980914

[9] Lin, L., Peng, K., Du, R., Huang, X., Lu, J., Xu, Y., *et al.* (2017) Metabolically Healthy Obesity and Incident Chronic Kidney Disease: The Role of Systemic Inflammation in a Prospective Study: Metabolically Healthy Obesity and CKD. *Obesity*, **25**, 634-641. https://doi.org/10.1002/oby.21768

[10] Egbi, O.G., Okafor, U.H., Miebodei, K.E., Kasia, B.E., Kunle-Olowu, O.E. and Unuigbe, E.I. (2014) Prevalence and Correlates of Chronic Kidney Disease among Civil Servants in Bayelsa State, Nigeria. *Nigerian Journal of Clinical Practice*, **17**, 602-607. https://doi.org/10.4103/1119-3077.141426

[11] Lunyera, J., Stanifer, J.W., Ingabire, P., Etolu, W., Bagasha, P., Egger, J.R., *et al.* (2016) Prevalence and Correlates of Proteinuria in Kampala, Uganda: A Cross-Sectional Pilot

Study. *BMC Research Notes*, **9**, Article No. 97.
https://doi.org/10.1186/s13104-016-1897-6

[12] Oluyombo, R., Banjo Oguntade, H., Soje, M., Obajolowo, O. and Karim, M. (2022) Obesity and CKD in Sub-Saharan Africa: A Narrative Review. *Kidney Medicine*, **4**, Article ID: 100403. https://doi.org/10.1016/j.xkme.2021.11.001

[13] Seck, S.M., Doupa, D., Gueye, L. and Abdou Dia, C. (2014) Prevalence of Chronic Kidney Disease and Associated Factors in Senegalese Populations: A Community-Based Study in Saint-Louis. *Nephro-Urology Monthly*, **6**, e19085.
https://doi.org/10.5812/numonthly.19085

[14] Stanifer, J.W., Maro, V., Egger, J., Karia, F., Thielman, N., Turner, E.L., *et al.* (2015) The Epidemiology of Chronic Kidney Disease in Northern Tanzania: A Population-Based Survey. *PLOS ONE*, **10**, e0124506. https://doi.org/10.1371/journal.pone.0124506

[15] Aseneh, J.B., Kemah, B.A., Mabouna, S., Njang, M.E., Ekane, D.S.M. and Agbor, V.N. (2020) Chronic Kidney Disease in Cameroon: A Scoping Review. *BMC Nephrology*, **21**, Article No. 409. https://doi.org/10.1186/s12882-020-02072-5

[16] Teuwafeu, D.G., Joseph, N.B., Mahamat, M., Aristide, N., Francois, K.F. and Gloria, A. (2022) Epidemiological and Clinical Profiles of Chronic Kidney Disease Patients Presenting for Emergency Hemodialysis: A Five-Year Retrospective Study in Two Dialysis Centres in Cameroon. *Open Journal of Nephrology*, **12**, 75-86.
https://doi.org/10.4236/ojneph.2022.121007

[17] OMS (2020) Obésité et surpoids.
https://www.who.int/fr/news-room/fact-sheets/detail/obesity-and-overweight

[18] Friedewald, W.T., Levy, R.I. and Fredrickson, D.S. (1972) Estimation of the Concentration of Low-Density Lipoprotein Cholesterol in Plasma, without Use of the Preparative Ultracentrifuge. *Clinical Chemistry*, **18**, 499-502.
https://doi.org/10.1093/clinchem/18.6.499

[19] Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (2001) Executive Summary of the Third Report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). *JAMA*: *The Journal of the American Medical Association*, **285**, 2486-2497.
https://doi.org/10.1001/jama.285.19.2486

[20] Colman, E. (2012) Food and Drug Administration's Obesity Drug Guidance Document. *Circulation*, **125**, 2156-2164. https://doi.org/10.1161/circulationaha.111.028381

[21] Suwała, S. and Junik, R. (2024) Body Mass Index and Waist Circumference as Predictors of Above-Average Increased Cardiovascular Risk Assessed by the SCORE2 and SCORE2-OP Calculators and the Proposition of New Optimal Cut-Off Values: Cross-Sectional Single-Center Study. *Journal of Clinical Medicine*, **13**, Article 1931.
https://doi.org/10.3390/jcm13071931

[22] Nguedjo, M.W., Ngondi, J.L., Ntentie, F.R., Kingue Azantsa, B.G., Ntepe Mbah, J.L. and Oben, J.E. (2022) Clinical Characteristics and Classification of Cameroonians with Obesity and Metabolically Normal Phenotype in the West Region of Cameroon. *Heliyon*, **8**, e11652. https://doi.org/10.1016/j.heliyon.2022.e11652

[23] Hannech, E., Slouma, M., Dhahri, R., Mehmli, T., Gharsallah, I., Metoui, L., *et al.* (2020) Profil lipidique au cours de la polyarthrite rhumatoïde et risque d'athérogénicité. *La Revue de Médecine Interne*, **41**, A202-A203.
https://doi.org/10.1016/j.revmed.2020.10.346

[24] Levey, A.S., Stevens, L.A., Schmid, C.H., *et al.* (2009) A New Equation to Estimate Glomerular Filtration Rate. *Annals of Internal Medicine*, **150**, 604-612.

https://doi.org/10.7326/0003-4819-150-9-200905050-00006

[25] Matsha, T.E., Hartnick, M.D., Kisten, Y., Eramus, R.T. and Kengne, A.P. (2013) Obesity Phenotypes and Subclinical Cardiovascular Diseases in a Mixed-Ancestry South African Population: A Cross-Sectional Study. *Journal of Diabetes*, **6**, 267-270. https://doi.org/10.1111/1753-0407.12089

[26] Mbanya, V.N., Echouffo-Tcheugui, J.B., Akhtar, H., Mbanya, J. and Kengne, A.P. (2015) Obesity Phenotypes in Urban and Rural Cameroonians: A Cross-Sectional Study. *Diabetology & Metabolic Syndrome*, **7**, Article No. 21. https://doi.org/10.1186/s13098-015-0016-5

[27] Jagannathan, R. and Patzer, R.E. (2017) Urbanization and Kidney Function Decline in Low and Middle Income Countries. *BMC Nephrology*, **18**, Article No. 276. https://doi.org/10.1186/s12882-017-0685-4

[28] Jung, C.H., Lee, M.J., Kang, Y.M., Hwang, J.Y., Kim, E.H., Park, J., *et al.* (2015) The Risk of Chronic Kidney Disease in a Metabolically Healthy Obese Population. *Kidney International*, **88**, 843-850. https://doi.org/10.1038/ki.2015.183

[29] Moeinzadeh, F., Rouhani, M.H., Seirafian, S., Vahdat, S., Mortazavi, M., Clark, C.C.T., *et al.* (2023) Metabolic Health Status and Renal Disorders: A Cross-Sectional Study. *Scientific Reports*, **13**, Article No. 20794. https://doi.org/10.1038/s41598-023-48333-9

[30] Cho, Y.K., Lee, J., Kim, H.S., Park, J., Lee, W.J., Kim, Y., *et al.* (2020) Impact of Transition in Metabolic Health and Obesity on the Incident Chronic Kidney Disease: A Nationwide Cohort Study. *The Journal of Clinical Endocrinology & Metabolism*, **105**, e148-e157. https://doi.org/10.1210/clinem/dgaa033

[31] Adjei, D.N., Stronks, K., Adu, D., Beune, E., Meeks, K., Smeeth, L., *et al.* (2018) Chronic Kidney Disease Burden among African Migrants in Three European Countries and in Urban and Rural Ghana: The RODAM Cross-Sectional Study. *Nephrology Dialysis Transplantation*, **33**, 1812-1822. https://doi.org/10.1093/ndt/gfx347

[32] Masimango, M.I., Sumaili, E.K., Wallemacq, P., Malembaka, E.B., Hermans, M.P., Fillée, C., *et al.* (2020) Prevalence and Risk Factors of CKD in South Kivu, Democratic Republic of Congo: A Large-Scale Population Study. *Kidney International Reports*, **5**, 1251-1260. https://doi.org/10.1016/j.ekir.2020.05.028

[33] Ortiz, A., Mattace-Raso, F., Soler, M.J. and Fouque, D. (2022) Ageing Meets Kidney Disease. *Clinical Kidney Journal*, **15**, 1793-1796. https://doi.org/10.1093/ckj/sfac151

[34] Lu, J.L., Molnar, M.Z., Naseer, A., Mikkelsen, M.K., Kalantar-Zadeh, K. and Kovesdy, C.P. (2015) Age and Association of Body Mass Index with Loss of Kidney Function and Mortality. *The Lancet Diabetes & Endocrinology*, **3**, 704-714.

[35] McNamara, B.J., Diouf, B., Hughson, M.D., Hoy, W.E. and Bertram, J.F. (2009) Associations between Age, Body Size and Nephron Number with Individual Glomerular Volumes in Urban West African Males. *Nephrology Dialysis Transplantation*, **24**, 1500-1506. https://doi.org/10.1093/ndt/gfn636

[36] Huang, F., Wang, L., Zhang, Q., Wan, Z., Hu, L., Xu, R., *et al.* (2021) Elevated Atherogenic Index and Higher Triglyceride Increase Risk of Kidney Function Decline: A 7-Year Cohort Study in Chinese Adults. *Renal Failure*, **43**, 32-39. https://doi.org/10.1080/0886022x.2020.1853569

[37] Li, W., Du, Z., Wei, H. and Dong, J. (2022) Total Cholesterol to High-Density Lipoprotein Cholesterol Ratio Is Independently Associated with CKD Progression. *International Urology and Nephrology*, **54**, 2057-2063. https://doi.org/10.1007/s11255-021-03099-9

[38] Dobiášová, M. and Frohlich, J. (2001) The Plasma Parameter Log (TG/HDL-C) as an

Atherogenic Index: Correlation with Lipoprotein Particle Size and Esterification Rate Inapob-Lipoprotein-Depleted Plasma (FERHDL). *Clinical Biochemistry*, **34**, 583-588. https://doi.org/10.1016/s0009-9120(01)00263-6

[39] Oh, D., Lee, S., Yang, E., Choi, H.Y., Park, H.C. and Jhee, J.H. (2025) Atherogenic Indices and Risk of Chronic Kidney Disease in Metabolic Derangements: Gangnam Severance Medical Cohort. *Kidney Research and Clinical Practice*, **44**, 132-144.

[40] De Cosmo, S., Menzaghi, C., Prudente, S. and Trischitta, V. (2013) Role of Insulin Resistance in Kidney Dysfunction: Insights into the Mechanism and Epidemiological Evidence. *Nephrology Dialysis Transplantation*, **28**, 29-36. https://doi.org/10.1093/ndt/gfs290

[41] He, Y., Li, F., Wang, F., Ma, X., Zhao, X. and Zeng, Q. (2016) The Association of Chronic Kidney Disease and Waist Circumference and Waist-to-Height Ratio in Chinese Urban Adults. *Medicine*, **95**, e3769. https://doi.org/10.1097/md.0000000000003769

[42] Pluta, W., Dudzińska, W. and Lubkowska, A. (2022) Metabolic Obesity in People with Normal Body Weight (MONW)—Review of Diagnostic Criteria. International *Journal of Environmental Research and Public Health*, **19**, Article 624. https://doi.org/10.3390/ijerph19020624

[43] Miricescu, D., Balan, D., Tulin, A., Stiru, O., Vacaroiu, I., Mihai, D., *et al.* (2021) Impact of Adipose Tissue in Chronic Kidney Disease Development (Review). *Experimental and Therapeutic Medicine*, **21**, Article No. 539. https://doi.org/10.3892/etm.2021.9969

[44] Lee, J., Min, S., Oh, S.W., Oh, S., Lee, Y.H., Kwon, H., *et al.* (2023) Association of Intraabdominal Fat with the Risk of Incident Chronic Kidney Disease According to Body Mass Index among Korean Adults. *PLOS ONE*, **18**, e0280766. https://doi.org/10.1371/journal.pone.0280766

[45] Biadgilign, S., Mgutshini, T., Haile, D., Gebremichael, B., Moges, Y. and Tilahun, K. (2017) Epidemiology of Obesity and Overweight in Sub-Saharan Africa: A Protocol for a Systematic Review and Meta-Analysis. *BMJ Open*, **7**, e017666. https://doi.org/10.1136/bmjopen-2017-017666

[46] Chen, J., Muntner, P., Hamm, L.L., Jones, D.W., Batuman, V., Fonseca, V., *et al.* (2004) The Metabolic Syndrome and Chronic Kidney Disease in U.S. Adults. *Annals of Internal Medicine*, **140**, 167-174. https://doi.org/10.7326/0003-4819-140-3-200402030-00007