Body mass index and shortened telomere length in middle-aged female and male
RUNNING HEAD: Middle-aged and shortened telomere length

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Abstract:
Obesity is a risk factor associated with age-related disorders that accelerate aging, and it increases the risk of metabolic diseases. Therefore, this study was conducted to investigate the association of leukocyte telomere length (LTL) with the presence of higher body weight in middle-aged females and males. The study subjects comprised 160 (80 control and 80 higher body mass index BMI groups) with ranging ages of 30-50 years included and stratified for BMI. The physio-biochemical analysis was measured using enzymatic determination. Mean telomere length was determined by using the southern blotting technique. The association analysis revealed a significant variance (P < 0.01) in biochemical parameters between higher BMI groups and control including waist, lipid profile, and the level of estradiol, testosterone, follicle-stimulating hormone, and luteinizing hormone. Mean telomere length was shorter in middle-aged males compared to the females of higher BMI groups and control groups for both age groups. LTL was shorter in the overweight and obese patients compared with the control group, and these differences in LTL obese group were shorter compared to the overweight group. In conclusion, shorter telomere length was observed in middle-aged males associated with higher body weight and lipid abnormalities. Lipid/lipoprotein abnormalities can be used as a predictor for the shortened telomere length and the reduction in adiposity indices can improve the telomere length in both overweight and obese subjects.

Keywords: Gender, obesity, physio-biochemical analyses, telomere.

Introduction:
Telomeres, the repetitive sequence TTAGGG at the terminal of linear chromosomes that protect deoxynucleotide acid DNA-protein structures by constituting a loop to prevent end-to-end fusions of the chromosomes 1, 2, 3. Telomeres also prevent loss of genomic DNA at the terminal of mammalian chromosomes, thereby protecting against cell death 4, 5. Telomere length extends to 10–15 kb in humans and decreases with age 4. In leukocytes, with each cell cycle telomeres undergo attrition leading to progressive telomere shortening with age 6. The attrition in telomere length increases with age 7. When telomeres attrition reaches a critical length, the cell cycle arrest or apoptosis is triggered 6.

Telomere length averages are affected by genetic factors, and non-genetic factors including oxidative stress, inflammation, body composition, and gender 8, 9. Telomere attrition rate increased by inflammation and oxidative stress that is increased with age and obesity 4, 5. In general, obesity and overweight promote inflammation and oxidative stress predictive of shorter telomeres in adults 10, 11, 12. Moreover, obesity represents one of the most important age-related disorders 13 that accelerate aging, and it increases the risk of metabolic diseases that increase the risk of age-related disorders 14. Besides obesity, leukocyte telomere length (LTL) is influenced by gender being shorter in adult males compared to adult females 15. This discrepancy in the rate of telomere attrition between genders is probably caused by the effects of estrogen 16. The estrogen hormone that has antioxidant properties to reduce oxidative stress and inflammation 8, also stimulates telomerase activity by stimulating nitric oxide production 17. While in males, testosterone increases susceptibility to oxidative stress that could participate in the acceleration of telomere shortening 18.

The prevalence of obesity all over the world threatens public global health that has currently
reached epidemic proportions. The current research aims to evaluate the correlation between higher body weight and shortened telomere length in middle-aged females and males. According to our knowledge, there is no study regarding the relationship between higher body mass indexes and telomere length with some physio-biochemical parameters in middle-aged females and males.

Materials and Methods:

Study population

This study was conducted between May 2019-March 2020 and it was carried out at the Department of Nutrition/ Marjan Teaching Hospital/ Babylon province/Iraq. The current study was approved by Al-Qasim Green University (Approval No. 12.10.15) and informed written consent was obtained from all subjects before the initiation of the study. The study subjects comprised 160 (80 control and 80 higher BMI group) with ranging ages of 30-50 years included and stratified for BMI. Individuals with diabetes, hypertension, any chronic disease, smokers, breast cancer, hormonal disturbance, alcoholic patients, and pregnant female were excluded. The questionnaire of each subject was taken; it included age, smoking, lifestyle, diet, family history of the disease and use of hormone replacement therapy.

Weight index

The weighted index recommended by the world health organization (WHO) including body mass index (BMI) and waist circumference was included in this study. This measure was based on the heights and weights measured without shoes and with minimal clothing as the following equation BMI= weight (kg)/ height\(^2\) (m\(^2\)). BMI was classified as underweight, normal, overweight, obesity, and morbid obesity (BMI<18), (BMI 18 - 24.9), (BMI 25 - 29.9), (BMI 30-39.9), and (BMI > 40) respectively. The waist circumference was measured at the narrowest point of the torso wisdom usually just above the belly button, while the subject stood up, which was in male ≤ 102 cm and female ≤ 88 cm.  

Biochemical assay

From each subject in the study, 5 ml of fasting blood samples were collected. Two milliliters were collected into EDTA containing tubes to measure the telomere length. While the sera were collected by centrifuging at 3000 rpm for 15 min. The sera were used for the measurement of lipid profile and hormonal assay. The serum total cholesterol (TC), triglyceride (TG), and high-density lipoprotein-cholesterol (HDL-C) were analyzed by an enzymatic determination (CHOD-PAP). The low-density lipoprotein-cholesterol (LDL-C) was calculated by the following equation: LDL-C = TC – HDL-C – (TG × 0.2). Bioassay Technology Laboratory company ELISA kit was used to measure the hormones (estradiol with catalog number E1034Hu, testosterone with catalog number E1036Hu, luteinizing hormone (LH) with catalog number E1037Hu, and follicle-stimulating hormone (FSH) with catalog number E1001Hu).

DNA extraction and telomere length

Genomic DNA was extracted from whole blood by using a Genaid kit and was quantified by Nanodrop. The southern-blotting technique was used to measure terminal restriction fragment (TRF) lengths. Briefly, 2 μg of DNA was digested with restriction enzymes including HinfI (20 U) and RsaI (20 U) (Roche) for 2 h at 37°C to produce TRFs that contain both subtelomeric and telomeric repetitive sequence. Genomic DNA was digested into small fragments while the repetitive telomere sequences (TTAGGG)n remain intact according to the restriction enzyme sites. The TRFs were loaded on 0.8% agarose gel then denatured with 0.5 M NaOH/1.5 M NaCl and neutralized for 30 min in 0.5 M Tris and 1.5 M NaCl. The capillary transfer was used overnight to transfer DNA to a nylon membrane positively charged. The nylon membranes were then hybridized for 3 h in the hybridization solution with telomeric probe digoxigenin. The blotting membrane was washed 3 times with 2x SSC (3 M NaCl, 0.3 M sodium citrate, pH 7.0). The telomeric probe was revealed by the digoxigenin luminescent procedure after being exposed to X-ray film. As the telomeres shorten with age, all samples were taken at the same age group (30-40, 41-50) to determine the telomere length.

Statistical analysis

SPSS v23.0 (IBM, NY, USA) was used to calculate association analysis. Means of BMI group and equal variances of the normalized data were tested with the Kolmogorov-Smirnov test. The significant effect of the BMI group, sex and age group on the various parameters studied were performed with the following model:

\[
Y_{ijkl} = \mu + G_i + S_j + A_k + (GSA)_{ijk} + e_{ijkl}
\]

where \(Y_{ijkl}\) = phenotypic traits, \(\mu\) = overall mean, \(G_i\) = fixed effect of \(i^{th}\) BMI group (i = control, overweight, obese), \(S_j\) = fixed effect of \(j^{th}\) sex (j = male, female), \(A_k\) = fixed effect of \(k^{th}\) age group (30-40,41-50), (GSA)\(_{ijk}\) = the interaction effect and \(e_{ijkl}\) = random residual error associated...
Results:

Results of the association analysis showed a significant difference (P < 0.01) in biochemical parameters between higher BMI and control groups (Table 1 and Table 2). There was significant elevation (P<0.01) in the waist and lipid profile in both overweight and obese patients compared to the control group, in male compared to the female and age group (30-40 years) compared to the (41-50 years). Moreover, the results showed a significant reduction (P<0.01) in the level of estradiol, testosterone, FSH, LH, and telomere length in the higher BMI group compared to the control group, also this reduction in male compared to the female and age group (30-40 years) compared to the (41-50 years) (Table 2).

The regression analysis of telomere length with study parameters persisted the biochemical changes investigated in this study (Table 3). There was a linear increase in leukocyte telomere length with sex (β=0.38, CI (95%) =0.33 to 0.96), HDL (β=0.69, CI (95%) =0.42 to 2.84) and estradiol (β=0.01, CI (95%) =0.01 to 0.03) respectively. Shorter leukocyte telomere length was associated with age (β=-0.53, CI (95%) = -1.08 to 0.30), BMI (β=-0.73, CI (95%) = -0.93 to 0.07), waist (β=- 0.76, CI (95%) = -1.04 to 0.92), cholesterol (β=-2.03, CI (95%) = -3.92 to -1.26), TG (β= -0.68, CI (95%) = -1.55 to 0.47), LDL (β= -1.50, CI (95%) = -2.34 to 0.51), and LH (β= -0.53, CI (95%) = -1.05 to 0.94) respectively. Moreover, Pearson’s correlation analysis was used to assess the correlation between the telomere length and variable parameters (Table 4). Telomere length was found to correlate positively and significantly with sex (r = 0.38, P < 0.05), and estradiol (r = 0.61, P < 0.05), while significant negative correlation was found between telomere length and other variable parameters including age (r = 0.53, P < 0.05), BMI (r = 0.73, P < 0.05), waist (r = 0.62, P < 0.05), cholesterol (r = -0.72, P < 0.05), LDL (r = -0.73, P < 0.05), VLDL (r = -0.49, P < 0.05), and testosterone (r = -0.39, P < 0.05).

The detection of terminal restriction fragments (TRFs) of genomic DNA of control and higher BMI subjects measured in kilobase pairs (Kb) is shown in Figure 1 and Table 2. Telomere length analysis in control and higher BMI subjects according to the gender showed that telomere length (TL) was variant between male and female (Table 2). Telomere length mean was shorter in males compared to females of control subjects for both age groups (12.56 bp) vs. (14.16 bp) and (11.53 bp) vs. (12.86 bp) and these differences decreased as aging increased. In higher BMI subjects, this gender difference in TL was revealed among the overweight and obese patients as shown in Table 2. TL was shorter in the overweight and obese patients compared to the control subjects were, the obese male (10.43) of age (30-40) were longer compared to the obese male (9.13) of age (41-50). While obese females (11.13) of age (30-40) were longer compared to the obese female (10.23) of age (41-50). Both obese males and females for both age groups were shorter compared to the overweight male and female age groups.
Table 1. Comparison of some biochemical parameters among BMI groups according to gender and age groups

| Gender | Age groups (years) | BMI groups | Indices (LSM ± SE) |
|--------|-------------------|------------|--------------------|
|        |                   |            | WC (cm) | Cholesterol (mmol/l) | TG (mmol/l) | HDL (mmol/l) | LDL (mmol/l) | VLDL (mmol/l) | P-value |
| Men    | 30-40 (40)        | Control    | 90.00±10.11 | 2.86±0.03  | 0.81±0.02  | 1.60±0.02  | 1.34±0.02  | 0.16±0.01  | 0.001  |
|        |                   | Overweight | 95.33±8.21  | 4.36±0.04  | 1.13±0.03  | 1.33±0.03  | 1.69±0.02  | 0.31±0.01  | 0.001  |
|        |                   | Obese      | 106.00±7.12 | 4.66±0.31  | 1.56±0.43  | 0.93±0.03  | 3.22±0.01  | 0.37±0.02  | 0.001  |
|        | 41-50 (40)        | Control    | 95.00±10.21 | 3.76±0.32  | 1.30±0.44  | 1.40±0.01  | 2.60±0.04  | 0.46±0.01  | 0.001  |
|        |                   | Overweight | 100.66±9.22 | 5.13±0.42  | 1.70±0.22  | 1.10±0.03  | 3.49±0.04  | 0.54±0.03  | 0.001  |
|        |                   | Obese      | 114.33±8.21 | 5.63±0.51  | 2.20±0.05  | 0.70±0.02  | 3.90±0.05  | 0.60±0.04  | 0.001  |
| Women  | 30-40 (40)        | Control    | 81.00±9.11  | 2.11±0.02  | 0.73±0.02  | 1.90±0.01  | 1.33±0.02  | 0.14±0.03  | 0.001  |
|        |                   | Overweight | 90.66±10.23 | 3.13±0.34  | 1.00±0.05  | 1.53±0.01  | 1.58±0.05  | 0.28±0.01  | 0.001  |
|        |                   | Obese      | 100.33±8.52 | 3.26±0.32  | 1.30±0.24  | 1.13±0.03  | 3.00±0.03  | 0.33±0.03  | 0.001  |
|        | 41-50 (40)        | Control    | 90.66±9.21  | 2.66±0.41  | 0.86±0.34  | 1.53±0.03  | 2.28±0.04  | 0.38±0.02  | 0.001  |
|        |                   | Overweight | 95.33±9.31  | 4.23±0.22  | 1.36±0.33  | 1.13±0.02  | 2.96±0.06  | 0.40±0.04  | 0.001  |
|        |                   | Obese      | 110.33±7.22 | 5.43±0.34  | 1.76±0.21  | 0.71±0.01  | 3.61±0.07  | 0.58±0.04  | 0.001  |

Table 2. Comparison of reproductive hormones and telomere length among BMI groups according to gender and age groups

| Gender | Age group (years) | BMI group | Indices (LSM ± SE) |
|--------|-------------------|-----------|--------------------|
|        |                   |            | E (pg/ml) | T (ng/ml) | FSH (mIU/ml) | LH (mIU/ml) | Telomere length (Kb) | P-value |
| Men    | 30-40 (40)        | Control    | 3.08±0.23 | 11.83±0.44 | 12.08±0.32 | 6.13±0.23 | 12.56±0.93 | 0.001  |
|        |                   | Overweight | 6.23±0.92 | 9.50±0.92 | 8.13±0.72 | 5.50±0.42 | 11.56±1.21 | 0.001  |
|        |                   | Obese      | 8.32±0.83 | 5.18±1.32 | 7.13±1.21 | 3.66±0.32 | 10.43±1.11 | 0.001  |
|        | 41-50 (40)        | Control    | 4.33±0.42 | 11.31±1.22 | 11.17±1.32 | 5.50±0.53 | 11.53±0.82 | 0.001  |
|        |                   | Overweight | 6.24±0.92 | 8.34±0.83 | 8.04±0.93 | 3.66±0.21 | 10.33±1.21 | 0.001  |
|        |                   | Obese      | 9.43±0.84 | 4.18±1.11 | 6.70±0.72 | 2.50±0.42 | 9.13±1.23 | 0.001  |
| Women  | 30-40 (40)        | Control    | 38.08±1.36 | 0.28±0.02 | 34.34±1.23 | 24.13±0.31 | 14.16±1.23 | 0.001  |
|        |                   | Overweight | 23.23±1.20 | 0.39±0.01 | 22.58±1.40 | 16.76±0.42 | 12.33±0.92 | 0.001  |
|        |                   | Obese      | 20.01±2.21 | 0.42±0.01 | 17.56±2.32 | 10.27±2.22 | 11.13±1.23 | 0.001  |
|        | 41-50 (40)        | Control    | 32.41±2.20 | 0.33±0.03 | 20.54±1.31 | 20.90±1.12 | 12.86±0.98 | 0.001  |
|        |                   | Overweight | 20.66±3.11 | 0.50±0.02 | 14.06±2.31 | 16.03±2.31 | 11.13±0.99 | 0.001  |
|        |                   | Obese      | 19.75±2.62 | 0.66±0.03 | 11.44±1.11 | 6.90±1.12 | 10.23±1.12 | 0.001  |

LSM ± SE, Least square means ± Standard error; BMI, body mass index; WC, waist circumference; TG, triglyceride; HDL, high-density lipoprotein; LDL, low-density lipoprotein; VLDL, very low-density lipoprotein. The P-value with statistical significance is indicated in bold numbers.
Table 3. Association analysis of telomere length with anthropometric and biochemical parameters

| Variables                                      | Regression coefficient (β) | Confidence interval (95%) | P-value |
|------------------------------------------------|----------------------------|---------------------------|---------|
| Gender                                         | 0.38                       | 0.33 to 0.96              | 0.001   |
| Age                                            | -0.53                      | -1.08 to 0.30             | 0.001   |
| Body mass index (BMI)                          | -0.73                      | -0.93 to 0.07             | 0.001   |
| Waist circumference (cm)                       | -0.76                      | -1.04 to 0.92             | 0.001   |
| Cholesterol (mmol/l)                           | -2.03                      | -3.92 to -1.26            | 0.001   |
| Triglyceride (TG) (mmol/l)                     | -0.68                      | -1.55 to 0.47             | 0.001   |
| High density lipoprotein (HDL) (mmol/l)        | 0.69                       | 0.42 to 2.84              | 0.012   |
| Low density lipoprotein (LDL) (mmol/l)         | -1.50                      | -2.34 to 0.51             | 0.019   |
| Very low density lipoprotein (VLDL) (mmol/l)   | -0.35                      | -1.27 to 0.08             | 0.194   |
| Estradiol (pg/ml)                              | 0.01                       | 0.001 to 0.03             | 0.009   |
| Testosterone (ng/ml)                            | -0.39                      | -1.01 to 0.50             | 0.065   |
| Follicle stimulating hormone (FSH) (mIU/ml)     | -0.22                      | -1.002 to 0.42            | 0.094   |
| Luteinizing hormone (LH) (mIU/ml)              | -0.59                      | -1.05 to 0.94             | 0.001   |

P < 0.05, Significant; P > 0.05, Non-significant. The P-value with statistical significance is indicated in bold numbers.

Table 4. Correlation among telomere length with anthropometric and biochemical parameters

| Variables                                      | r     | P-value |
|------------------------------------------------|-------|---------|
| Gender                                         | 0.38  | 0.021   |
| Age                                            | -0.53 | 0.001   |
| Body mass index (BMI)                          | -0.73 | 0.001   |
| Waist circumference (cm)                       | -0.62 | 0.001   |
| Cholesterol (mmol/l)                           | -0.72 | 0.001   |
| Triglyceride (TG) (mmol/l)                     | -0.02 | 0.886   |
| High density lipoprotein (HDL) (mmol/l)        | 0.03  | 0.830   |
| Low density lipoprotein (LDL) (mmol/l)         | -0.73 | 0.001   |
| Very low density lipoprotein (VLDL) (mmol/l)   | -0.49 | 0.002   |
| Estradiol (pg/ml)                              | 0.61  | 0.001   |
| Testosterone (ng/ml)                            | -0.39 | 0.018   |
| Follicle stimulating hormone (FSH) (mIU/ml)     | -0.09 | 0.588   |
| Luteinizing hormone (LH) (mIU/ml)              | 0.02  | 0.866   |

P < 0.05, Significant; P > 0.05, Non-significant. The P-value with statistical significance is indicated in bold numbers.
abnormalities and atherogenic indices have been revealed in obese and overweight subjects, including elevated cholesterol, triglycerides, and decreased HDL cholesterol levels. These lipid abnormalities and the prevalence of obesity are higher in males compared to females. The possible explanation for the gender difference might be due to biological gender differences. Estrogen has atheroprotective effects on serum lipid concentrations that can reduce the development of early lesions of atherosclerosis. Estradiol through atheroprotective effects can reduce lipid deposits in the endothelium, with a decrease in LDL-C and an increase in HDL-C in the plasma. Besides, the estrogen effect in females is of greater activity in an improvement of the blood lipid profile by removing LDL and VLDL from the plasma compared to males.

Overweight and obesity are one of the most dangerous public health that increases with age. Generally, the risk of obesity increases with age especially middle age. Middle age is the main risk factor of obesity in both males and females. This may belong to the visceral fat and subcutaneous abdominal fat, which are the riskiest factors for insulin resistance and metabolic diseases that increase with aging and reach peak values at middle age. While at older ages, the relative effects of obesity likely decrease because individuals are more likely to develop metabolic diseases independent of obesity.

Studies have shown that overweight and obese females and males exhibit a significant reduction in levels of estradiol, testosterone, FSH, and LH hormones compared to the control group. Obesity is an increasingly common health risk that causes many female reproduction disorders. Higher body mass is reported to inhibit estradiol production by the ovary and causes the functional impairment of the granulosa cells. Additionally, the low levels of gender hormone-binding globulin (SHBG) in overweight and obese females result in decreased estrogen inactivation and consequently lower levels of estradiol. Besides, obesity decrease FSH and LH level by decreasing sensitivity and feedback inhibition of the hypothalamic-pituitary-gonadal (HPG) axis that results in blunted LH responsiveness to gonadotropin stimulation. FSH is also lower in females with obesity and overweight. HPG axis sensitivity may be relatively reduced in obese females that led to a decrease in FSH. This functional impairment happened by increased free fatty acids incorporation into the hypothalamic-pituitary-ovarian axis at the level of the pituitary leading to pausing gonadotropin gene translation affecting LH and FSH synthesis and/or translation affecting LH and FSH synthesis and/or

Discussion:

The present study investigated some anthropometric–biochemical parameters and the length of telomere in middle-aged females and males. The study revealed significant elevation (P<0.01) in the waist circumference and lipid profile in overweight and obese patients compared to the control group, also in males compared to the females and age group (41-50 years) compared to the (30-40 years). Body mass index and waist circumference are widely used as a marker of adiposity and atherogenic indices. Lipid
secretion. Moreover, in overweight and obese males, low testosterone levels with elevated estradiol (E2) level results from an up-regulated aromatase activity suppress the HPG axis. Elevated estradiol levels may also affect gonadotropin-releasing hormone (GnRH), LH, and FSH release, resulting in hypogonadotropic hypogonadism and decreased fertility in overweight and obese male subjects. Decreased FSH and levels of LH are reported in obese adult males.

Regarding the telomere length, the current results showed that subjects with overweight and obese were lower telomere length. Obesity disorder causes the accumulation of excess fat in adipose tissue; this continuous accumulation results in increased oxidative stress and inflammation and releases inflammatory cytokines. The adipokines and pro-inflammatory cytokines that are associated with obesity cause oxidative damage of DNA and shorter telomere length. Compared to genomic DNA, telomeric DNA is relatively less capable of DNA repair, so the acute oxidative damage of the G-rich telomeric sequence can accelerate telomere loss with each cell cycle leading to senescence. Besides, shorter telomeres lengths were recorded in males compared to their females counterparts. This may be due to the estrogen effect in females that is thought to activate telomerase, an enzyme that lengthens telomeres by adding TTAGGG repeats. Additionally, estrogen has antioxidant and protective properties that may protect telomeres against increased oxidative stress and damage telomeres. Estrogen directly activates the telomerase promoter through phosphoinositol-3-kinase/Akt and nitric oxide pathways and indirectly affects DNA repair through the P53 pathway. In contrast, testosterone in males lacks antioxidant properties that may increase the deleterious effects of oxidative stress on telomeres. Additionally, the larger body size in males causes more cell divisions leading to shorter telomeres.

Conclusion:
Shorter telomere length was observed in middle-aged males associated with higher body weight and lipid abnormalities. Lipid/lipoprotein abnormalities can be used as a predictor for the shortened telomere length and the reduction in adiposity indices can improve the telomere length in both overweight and obese subjects.

Author's declaration:
- Conflicts of Interest: None.
- I hereby confirm that all the Figures and Tables in the manuscript are mine. Besides, the Figures and images, which are not mine, have been given permission for re-publication attached with the manuscript.
- The author has signed an animal welfare statement.
- Ethical Clearance: The project was approved by the local ethical committee in the University of Al-Qasim Green.

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دلیل کتله الجسم وقصر طول التيلومير عند النساء والرجال في متوسط العمر

العنوان الدوار: متوسط العمر وقصر طول التيلومير

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الخلاصة

السمنة هي عامل الخطر المرتبط بالاضطرابات المرتبطة بالعمر والتي تسرع الشيخوخة وتزيد من خطر الإصابة بأمراض الايض. لذلك، أجريت هذه الدراسة للتحري عن ارتباط طول التيلومير في الكريات البيض (LTL) وزن الجسم في النساء والرجال في منتصف العمر (30-50 سنة) وتم مطابقتها لمؤشر كتلة الجسم في النساء والرجال في منتصف العمر. شملت مجامع الدراسة 160 مجموعة (80 مجموعة سيطرة و80 مجموعة مؤشر كتلة الجسم عالي) مع أعمار تراوحت بين 30-50 سنة. تم قياس التحاليل الفسيولوجية، الكيميائية، باستخدام التحديد الأنزيمي، وتم تحديد متوسط طول التيلومير (LTL) باستخدام تقنية مرسيد سومرن. أظهرت تحاليل الارتباط تباينًا معنويًا (P < 0.01) في المعايير الفسيولوجية والكيميائية بين مجامع مؤشر كتلة الجسم العالمي ومجاميع كتلة الجسم والصحة في منتصف العمر. كان تمتد طول التيلومير أقصر عند الذكور في منتصف العمر من الإناث في مجامع مؤشر كتلة الجسم العالية. تبين استخدام تحليل الحالة الأولى والثانية، لتحديد طول التيلومير كمؤشر على قصر طول التيلومير وكيف يمكن أن يؤدي تقليل مؤشرات السمنة إلى تحسين طول التيلومير في الأشخاص الذين يعانون من زيادة الوزن والسمنة.

الكلمات المفتاحية: الجنس، السمنة، التحاليل الفسيولوجية، الكيميائية، التيلومير.