THE RELATIONSHIP OF PON1 ACTIVITY WITH LIPID METABOLISM IN OBESE WOMEN

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**OBEZ KADINLARDA PARAOKSANAZ 1 AKTİVİTESİNİN LİPİD METABOLİZMASI İLE İLİŞKİSİ**

**Giriş:** Obesite, Tip 2 Diabetes Mellitus, kardiyovasküler hastalık ve kanser riskini artıran, dünya genelinde önemi giderek artan ciddi bir hastalıktır.

**Amaç:** Bu çaşılmanın amacı serum lipidleri, Apolipoprotein A-I ( Apo A-I), Apolipioprotein B ( Apo B), Paraoksanaz 1 (PON 1) aktivitesinin kadınlarda obesiteye etkilerini saptamaktır. **Yöntem ve Gereçler:** Çalışmaya toplam 90 kadından oluşan, yaşları 18-48 yaş arası değişen, 30 abdominal obez ( Grup I), 30 jinoid obez ( Grup II), 30 sağlıklı kontrol ( Grup III) olmak üzere üç grup dahil edildi.

**Bulgular:** HDL-K, Apo B, D-LDL-K, hsCRP, HOMA-IR testlerinde, Grup I ve II arasında istatistiksel anlamlı fark saptandı (p=0.001, p=0.000, p=0.016, p=0.001, p=0.000, p=0.000, sırasıyla). HDL-K, D-LDL-K, hsCRP and HOMA-IR testlerinde Grup I ve III arasında anlamlı fark saptandı (p=0.023, p=0.023, p=0.000, p=0.001, respectively). TG, Apo B, HDL-K, D-LDL-K parametrelerinde abdominal obesitede belirgin olmak üzere fark saptanırken, PON 1 düzeylerinde gruplar arası fark olmadığı ortaya kondu.

**Sonuçlar;** Sonuçlarımız obesite de lipid metabolizmasının değiştiğini gösterirken, PON1 aktivitesinde değişiklik olmadığını ortaya koymuştur.

Anahtar Kelimeler : *Lipid Metabolizması; Obesite; Paraoksonaz 1*

**IN OBESE WOMEN THE RELATIONSHIP OF PARAOXONASE 1 ACTIVITY WITH LIPID METABOLISM**

**Summary**

**Bacground:** Obesity is a serious disease with increasing importance all around the world; it increases the risk for Type 2 Diabetes Mellitus, cardiovascular diseases and cancer.

Purpose: The aim of this study was to identify the effects of serum lipids, Apolipoprotein A-I (Apo A-I), Apolipoprotein B (Apo B) and Paraoxanase/Arylesterase (PON1) activity on obesity in women.

**Material and Methods**: The study was conducted on 90 subjects categorized into three groups, there were 30 women in each group with an age range of 18-48 years. Group I had 30 abdominal obese patients, Group II had 30 gynoid obeses and Group III included 30 healthy control subjects.

**Results:** There was a statistically significant difference between the triglyceride, HDL-C, Apo B, D-LDL-C, hsCRP, HOMA-IR levels of Group I and II (p=0.001, p=0.000, p=0.016, p=0.001, p=0.000, p=0.000, respectively). For HDL-C, D-LDL-C, hsCRP and HOMA-IR levels, there were statistically significant differences between Group I and III (p=0.023, p=0.023, p=0.000, p=0.001, respectively). TG, Apo B, HDL-C, D-LDL-C parameters differed significantly in abdominal obesity, whereas there was no difference among the groups for PON1 activity.

**Conclusion:** Our results indicate that lipid metabolism is altered in obesity, but PON1 activity appears not to be affected.

Key words: *Lipid Metabolism; Obesity; Paraoxonase 1*

**Introduction**

 Increasing prevalence of obesity all around the world causes great concerns both for individual health and for the national health prevention systems (1). Obesity has been shown to predispose individuals to several chronic diseases like atherosclerosis, arterial hypertension (HT), glomerulopathies that go together with proteinuria, diabetes mellitus (DM) and osteoarthritis (2).

 In obese patiens, changes have been demonstrated in lipid levels and lipoprotein metabolism (3). Changes in the levels and components of lipoproteins are thought to be in relation with the generation of cardiovascular disease (CVD) risk that accompanies obesity. Obese patients experience increased oxidative stress. When obese patients were compared to healthy individuals, low density lipoprotein cholesterol (LDL-C) was shown to have undergone lipid peroxidation. Increases in oxidative damage could be related to the decreases in the antioxidant defense. There is strong evidence in support of the fact that oxidative stress, resulting from metabolic changes secondary to insulin resistance, contributes to the complications of obesity. The decrease in the activity of PON1, which is an antioxidant enzyme related to high density lipoprotein cholesterol (HDL-C), is found to be in relation with dyslipidemia, diabetes mellitus, advanced age and increased oxidative stress (4).

 Paraoxonase /Arylesterase (PON1) enzymes have a glycoprotein structure synthesized by the liver, it hydrolyzes aromatic esters of carboxylic acid and it is an esterase firmly bound to HDL-C. PON1 is an enzyme with important roles in the prevention of atherosclerosis and the detoxification of organophosphates (5). In 1970s, among the scientific circles, toxicologists were the first to focus on the protective effects of PON1 in organophosphate poisoning. PON1 is an exyme that accompanies HDL, while it metabolizes lipid hydroperoxides. The studies performed in this field have demonstrated that PON1, an antioxidant enzyme found in the structure of HDL, hydrolyzes lipid peroxides and protects it against the oxidation of LDL-C (dragonov). PON1 stimulates the flow of cholesterol from macrophages through inhibition of LDL oxidation. PON1 is in association with Apolipoprotein A-I (Apo A-I) and Apolipoprotein J (Apo J) proteins of HDL-C (6). Several studies in recent years concentrated on the role of PON1 in instances of CVD, diabetes, neurological diseases, HIV and some other bacterial infections, chronic liver diseases and the use of alcohol (7).

Oxidation of LDL on the arterial wall is an important step in the generation of atherosclerosis, PON1 breaks down the active lipid found in oxidized LDL (OxLDL) and prevents the inflammatory response on the arterial cell wall (8). In this study, the measurement of PON1 levels, lipid profile and apoprotein levels and the identification of the relationships between them in obese women, aims at contributing to the literature by delineating the mechanism of effect for atherosclerotic vascular disease that develops in obesity.

**Materyal and methods**

This study was conducted in Abant Izzet Baysal University (AIBU), School of Medicine, Bolu Research and Implementation Center. Among the patients or relatives who come to the Blood Sampling Unit for routine control purposes, 60 obese women without any systemic diseases were selected (android, n=30 and gynoid, n=30) and 30 individuals were chosen as the control group to yield a population of 90 individuals. Obesity group was determined based on the National Institute of Health (NIH) Criteria (9) Individuals with BMI of > 30 kg/m2 qualified as obese, among these, those with a waist-to-hip (WTH) ratio of larger than 0.85 were classified as android obeses and those with a WTH ratio of smaller than 0.85 as gynoid obeses. Among the individuals participating in the study, the following were determined as exclusion criteria: a history of cerebrovascular disease within the last three months, significant weight loss, giving birth within the last six months, HT, DM, previous MI, history of anorexia/bulimia, medication use (Aspirin, anti-inflamatory drugs, oral contraseptive medications, hypolipemic drugs etc.), vitamins, antioxidants, smoking, pregnancy, menopausal-postmenopausal state, hepatic, renal, thyroid or atherosclerotic diseases, pituitary disease or dysfunction. An approval was obtained from AIBU Clinical and Laboratory Research Ethics Committee stating that the study was in abidance with ethical principles.

The individuals participating in the study provided venous blood samples at 9:00am in the morning following a 12-hour fast, the samples were obtained while they were sitting. Tubes used for serum separation were either dry tubes with gel separators containing clot activators (Vacuette, Greiner Bio-one GmbH, Kremsmünster, Austria) or those with K3EDTA (Vacuette, Greiner Bio-one GmbH, Kremsmünster, Austria); 8 ml. of sample was placed in the first, 2 ml in the second. For the clotting to take place, the obtained blood samples were kept at room temperature for 30 minutes. They were then centrifuged at +4 °C at 1250 g for 15 minutes to separate the plasma and the serum. Serum samples were stored at -80 °C until the day of the analyses. Frozen samples were sequentially thawed immediately before the analysis. We refrained from repetitive thawing and freezing.

Measurement of biochemical parameters

Serum paraoxonase activity was measured by utilizing Rel Assay Diagnostics commercial kit (Catalog no: 5D43, Gaziantep, Turkey). CV% values of the test were reported as 4.1, 1.7 ve 1.5 for low, medium and high serum pools by the manufacturer. The absorbance produced in a minute by p-nitrophenol generated as a result of the reaction was measured by spectrophotometry at 412 nm. Molar absorbtion coefficient for p-nitrophenol was Є412= 18290 M-1 cm-1. One unit of paraoxonase activity was defined as the amount of enzyme hydrolysing one µmole of paraoxone under these conditions and enzyme activity was expressed as U/L.

Serum arylesterase activity was measured by using Rel Assay Diagnostics arylesterase activity measurement kit (Catalog no: 4D23. Gaziantep, Turkey). CV% values of the test were reported as 4.0, 3.3 and 3.1 for high, medium and low serum pools by the manufacturer. The method is based on the colorimetric measurement of the phenol produced as a result of the enzymatic hydrolysis of the phenylasetate used as a substrate. The absorbance produced in a minute by the phenol generated as a result of the reaction was measured by a spectrophotometer at 548 nm. Molar absorbtion coefficient for phenol was Є548= 4000 M-1 cm-1. Under these conditions one unit of arylesterase activity was defined as the amount of enzyme hydrolysing one micromole of phenylacetate in a minute and was expressed as U/L.

High sensitive-CRP (hs-CRP) levels were measured by BN Prospec (Siemens Healthcare Diagnostics, Deerfield, IL, ABD) nephelometer by using a commercial kit and employing a nephelometric method.

Glucose, triglyceride, HDL-cholesterol (HDL-C), Total cholesterol, Apolipoprotein A-I (Apo A-I), Apolipoprotein B (Apo B), creatinine, direct-LDL-cholesterol (D-LDL-C) and serum insulin levels were measured with chemiluminescence microparticle enzyme immunological method by using Abbott commercial kits (Chicago, IL, ABD) and Abbott Arcitect c8200i analyzer. Of the calculated biochemical parameters, HOMA-IR (Homeostasis model insulin resistance) index was calculated with the following formula: fasting plasma glucose (mg/dL) x fasting plasma insulin (μIU/dL)/ 405 (10). MDRD (Modification of Diet in Renal Disease) renal function was assessed with the help of the estimated glomerular filtration rate (eGFR) calculated with MDRD equation (11).

Whether the numerical changes showed a normal distribution or not was assessed with Kolmogorov Smirnov test, and its homogenicity was analysed with Levene test. All the data were presented as mean± standard deviation. For parameters with normal distribution, the groups were evaluated with One Way ANOVA. For parameters that were significant, post hoc Tukey test was used to see the differences between the groups. For parameters that did not have a normal distribution following Kruskal Wallis variance analysis, the difference between the groups was analysed with Mann Whitney U. Arithmetical mean and standard deviation were used for parametric data, median and standard error were used for nonparametric data. Pearson correlation and BMI were calculated with multiple linear regression analysis. Results were evaluated within 95% confidence interval and p value of <0.05 was accepted as statistically significant.

**Results**

The study group consisted of healthy non-obese control group (Group I, n=30), abdominal obesity group (Group II, n=30) and gynoid obesity group (Group III, n=30) adding up to 90 female subjects.

Mean age, SD and range of the groups were found as; Group I 31.5 ± 7.5 (21-48) years, Group II 35.6 ± 8.6 (18-47) years and Group III 34.0 ± 8.2 (18-47) years, demonstrating no statistical difference (p=0.163). The duration of obesity was 5.0 ( 2.0 – 10.0) years for Group II and 4.0 (1.0 – 12.0) years for Group III. There was a statistically significant difference between the groups (p=0.000) A statistically significant difference was seen between Group I and II (p=0.000) and Group I and III (p=0.000), but not with Group II and III (p=0.525).

The measured and calculated values for the study groups were classified as parametric and nonparametric with Kolmogorov Smirnov test based on their distributions. Nonparametric tests were identified as duration of obesity, BMI, systolic blood pressure (SBP), diastolic blood pressure (DBP), Insulin, HOMA-IR and ARE acitivity and the results were presented as median and standard error. Parametric tests were identified as age, WTH ratio, glucose, creatinine, MDRD, Total cholesterol, triglyceride, HDL-C, Apo A-I, Apo B, PON1 activity, D-LDL-C and hsCRP and the results were presented as arithmetical mean and standard deviation. The results are presented on Tables I and II.

Pearson correlation was used between the tests that were measured. Significant positive correlations were seen between Total cholesterol and TG (r=0.614, p=0.000), Apo A-I (r=0.358, p=0.001), Apo B (r=0.840, p=0.000) and D-LDL-C (r=0.781, p=0.000). Triglyceride was in negative correlation with HDL-C (r=-0.281, p=0.007) and in positive correlation with Apo B (r=0.677, p=0,000), HOMA-IR (r=0.353, p=0.001), D-LDL-C (r=0.512, p=0.000) and BMI (r=0.357, p=0.001).

 HDL-C was in positive correlation with Apo A-I (r=0.817, p=0.000) and in negative correlation with hsCRP (r=-0.237, p=0.025) and BMI (r=-0.384, p=0.000). Apo B was in positive correlation with D-LDL-C (r=0.811, p=0.000) and BMI (r=0.284, p=0.007). A positive correlation was identified between HOMA-IR and BMI (r=0.381, p=0.000). There were positive correlations between D-LDL-C and hsCRP (r=0.262, p=0.013) and D-LDL-C and BMI (r=0.399, p=0.000). A positive correlation was identified between BMI and hsCRP (r=0.570, p=0.000). The results are presented on Table III.

When multiple linear regression analysis was performed for BMI and all other measured parameters, statistically significant differences were demonstrated (r2=0.524, p=0.000, BMI=23.24-0.003XTCHOL+0.010XTG-0.218XHDL-C+0.046XApoA-0.061XApoB- 0,014XPON1-0.000XARE+0.932 HOMA-IR+0,066XD-LDL-C+1,015XhsCRP).

When the other parameters were analysed separately, the most statistically significant ones were BMI and hsCRP (p=0,000) and BMI and HOMA-IR index (P=0,006). The results are shown on Table IV.

**Discussion**

At the heart of the disease causing mechanisms related to obesity, lies the low degree inflammatory process triggered by the metabolic events (12). Although, the changes in lipid and lipoprotein metabolisms have been demonstrated in obese cases, the level of PON1 activity and its relationship to lipid metabolism is not clear (13-14). In this study, the changes in PON1 activity in premenopausal abdominal and gynoid obesity groups were similar, changes have been shown in the D-LDL-C, HDL-C, TG and APOB components of the lipid panel together with changes in the levels of hsCRP, which is an indicator for endothelial inflammation.

In a study analysing the effects of different genetic and environmental factors on PON1 metabolism, in addition to different substrates, Rainwater et al. compared serum PON1 activity of 922 subjects by three different substrates as paraoxone, phenyl acetate and lactone dihydrocoumarine (15). Depending on the substrate, the activity differed up to 88-97%. Based on the allele they were located for PON1 activity, the order was as follows; QQ< QR< RR and MM< LM< LL (16). Browne et al. conducted a study in which they analyzed PON1 method and its biological variations. Human serum PON1 activity was measured in 70 individuals with Q192R polymorphisms grouped as 41 (QQ), 31 (QR) and 7 (RR) alleles; five different kinetic measurements were performed as paraoxonase activity, salt-stimulated paraoxonase activity, ARE activity with phenyl acetate, ARE activity with p-nitrophenyl acetate and finally ARE activity with phenyl acetate and p-nitrophenyl acetate in an attempt to identify the activity differences between the methods. Based on the alleles, PON1 acitvity was as follows; QQ< QR< RR. In the biological variation study conducted on 17 individuals, they demonstrated the differences between the alleles (17).

Veiga and colleagues performed a study on 81 obese and 75 healthy Portuguese women. They measured the PON1 activity by using paraoxone substrate, similar to findings of our study, they could not show any difference between the groups. When all the samples were classified on the basis of Q192R and L55M polymorphisms, PON1 activity changed depending on the alleles as has been shown in other studies. Similar to our findings, in this study, obese group had increased levels of SBP, DBP, triglyceride and hsCRP (a p<0.001, p<0.001, p<0.001and p<0.001 respectively) and HDL-L level was decreased (p<0.001) (18). A study by Tabur and colleagues including 30 obese and 40 non-diabetic metabolic syndrome patients together with 30 control subjects could not find a difference in PON1 activity between the groups and this is similar to our findings (4).

In a study by Seres et al. conducted on patients with adult and childhood obesity, adiponectin, resistin and PON1 activity were shown to have decreased. In this research study, the PON1 form with decreased activity was in Q192R polymorphism group; of the A and B isoenzymes, the activity was related to the B isoenzyme (13). In a study by Ferretti et al., 30 obese and 62 control children were found to have decreased PON1 activity (p<0.05), increased ARE activity (p<0.01), increased leptin levels (p<0.0001) and decreased adiponectin levels (p<0.001) (19). The findings of this study do not correlate with ours. As a specific polymorphism or allele was not chosen, the samples used in our study represented a heterogeneous group. In order not to be influenced by other hormonal or metabolic problems, the obese group consisted of women at reproductive age; furthermore these individuals did not have any diabetic, renal, hepatic or endocrine problems. Adding to all these limitations, the duration of obesity was considerably short and the number of patients was inadequate in our study.

In a study by Ayer et al., 19 obese and 19 non-obese young individuals were recruited; the study included arterial dilatation with exogenous nitrate stimulation in a subgroup and these individuals had significantly high levels of hsCRP and insulin together with low levels of HDL-C (20). In studies performed with the aim of identifying the correlation between calculated and direct LDL-C, for providing guidance in antilipemic therapy, direct measurements proved to be more valuable (21-22). In our study, we also preferred direct LDL-C.

In a study by Revenga-Faruca et al. on obese children (126) and adolescents (60 boys ve 66 girls), when belly fat and metabolic risks were analyzed, similar to our study, differences were shown in the levels of insulin CRP, TG and HDL-C levels (23).

The studies performed in the field show that abdominal obesity has higher cardiometabolic risks than gynoid obesity (24-25). In our study, when we compared the abdominal obesity group with controls; we found that they had developed insulin resistance and that they had increased levels of TG, Apo B, D-LDL-C and hsCRP together with low levels of HDL-C. When gynoid obesity group was compared to controls, there was no insulin resistance, their D-LDL-C and hsCRP levels were high and HDL-C levels were low. The insulin resistance seen in abdominal obesity is responsible for NIDDM, HT and CVD (26). When we compared the two obese groups with each other in our study, there was a statistically significant difference for HOMA-IR. Only abdominal obesity group was shown to have developed insulin resistance (> 2.7). In our study, the frequency of those with HOMA-IR of > 2.7, was 53% (n=16) in the abdominal obesity group and 46% (n=14) in gynoid obese group.

When we analyzed the relationship of PON1 to lipid metabolism in obese women, we arrived at the following conclusions:

 PON1 activity was similar between the groups. PON1 acitivity might be measured by different methods and can be influenced by polymorphisms even alleles. Moreover, in the biological variation studies of these tests, as individual index was < 0.6; instead of choosing a reference range, the suggestion was to monitor individual differences. When PON1 activity was being identified, for it to be affected minimally by demographical variables, women of similar age who did not smoke or use OCS were included in the study. PON1 activity tests can only be monitored for an individual for certain genotypes and by employing certain substrates.

Abdominal obesity group was shown to have developed insulin resistance. When compared with the controls, abdominal obesity group had higher levels of TG, ApoB, D-LDL-C and hsCRP and low levels of HDL-C; gynoid obesity group had high levels of D-LDL-C and hsCRP and low levels of HDL-C. These results point out to the fact that metabolic dysfunction has started both in gynoid and abdominal obesity groups. This process has obviously affected abdominal obesity group more significantly. D-LDL-C is a marker that is currently used in clinical biochemistry labs, its levels were found to be high for both groups of obesity and was more significantly so in the abdominal obesity group; this is a new information that contributes to the literature and can be regarded as important for that very reason.

**Funding**

This research has been supported by the Scientific Research Unit of Abant Izzat Baysal University (Project Number: 2008.08.01.296).

**Declaration of interest:** The authors report no conﬂicts of interest. The authors alone are responsible for the content and writing of the paper.

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Table I. Results of the nonparametric tests of the study groups presented as median and standard error.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|  | GROUP In=30 | GROUP IIn=30 | GROUP IIIn=30 | I-IIP | I-IIIp | II-IIIP |
| BMI (kg/m2) | 21.0 ± 0.42 | 33.5 ± 0.63 | 32.0 ± 0.87 | 0.000 | 0.000 | 0.534 |
| SBP(mm/Hg) | 100 ± 1.71 | 120 ± 2.57 | 120 ± 1.83 | 0.000 | 0.000 | 0.671 |
|  |  |  |  |  |  |  |
| DBP (mm/Hg) | 65 ± 1.41 | 80 ± 1.30 | 80 ± 1.67 | 0.000 | 0.000 | 0.185 |
| Insulin (µU/mL) | 8.6 ± 0.60 | 11.3 ± 1.23 | 10.4 ± 1.50 | 0.000 | 0.003 | 0.336 |
| HOMA-IR | 1.90 ± 1.87 | 2.75 ± 0.35 | 2.53 ± 0.35 | 0.000 | 0.001 | 0.496 |
| ARE (U/L) | 953 ± 89.7 | 1110 ± 79.6 | 1042 ± 85.8 | \* | \* | \* |
| ALT | 12.0 ± 0.81 | 16.0 ± 1.06 | 12.5 ± 0.75 | \* | \* | \* |

\*Kruskal Wallis did not identify any statistical significance.

SBP: sistolic blood pressure; DBP : diastolic blood pressure; HOMA-IR: Homeostasis Model of Assessment - Insulin Resistance; ARE: arylesterase.

Table II. The results of the parametric tests of the study groups presented as mean and SD.

|  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- |
|   | GROUP In=30 | GROUP IIn=30 | GROUP IIIn=30 | I-IIP | I-IIIP | II-IIIp |
| Glucose (mg/dL) | 92.0 ± 12.2 | 96.3 ± 9.7 | 98.7 ± 16.8 | \* | \* | \* |
| Creatinine (mg/dL) | 0.72 ± 0.07 | 0.71 ± 0.07 | 0.71 ± 0.08 | \* | \* | \* |
| MDRD | 104.3 ± 11.4 | 101.5 ± 12.8 | 101.7 ± 13.7 | \* | \* | \* |
| T.Chol. (mg/dL) | 162.7 ± 29.3 | 174.4 ± 35.5 | 175.2 ± 29.0 | \* | \* | \* |
| TG (mg/dL) | 94.2 ± 42.2 | 151.1 ± 70.1 | 128.9 ± 55.1 | 0.001 | 0.055 | 0.294 |
| HDL-C (mg/dL)  | 52.9 ± 9.6 | 43.9 ± 6.8 | 47.2 ± 7.8 | 0.000 | 0.023 | 0.250 |
| APO A-I (mg/dL) | 133.7 ± 20.1 | 126.5 ± 14.9 | 125.5 ± 16.8 | \* | \* | \* |
| APO B (mg/dL) | 68.7 ± 21.4 | 83.3 ± 21.6 | 75.9 ± 16.6 | 0.016 | 0.355 | 0.322 |
| PON (U/L) | 31.5 ± 19.3 | 29.2 ± 16.4 | 30.6 ± 18.7 | \* | \* | \* |
| D-LDL-C (mg/dL) | 97.9 ± 23.9 | 122.6 ± 30.1 | 116.2 ± 28.1 | 0.001 | 0.023 | 0.627 |
| hsCRP  | 0.77 ± 0.70 | 5.05 ± 3.35 | 3.76 ± 2.06 | 0.000 | 0.000 | 0.080 |

\*ANOVA did not identify any statistical difference.

MDRD: Modification of Diet in Renal Disease; PON: Paraoxonase; hsCRP: high sensitive C-Reactive Protein.

Table 3. Pearson correlation results of Total cholesterol, TG, HDL-C, Apo A-I, Apo B, hsCRP, PON, ARE, D-LDL-C and BMI in all groups.

|  |  |  |  |  |  |  |  |  |  |  |  |
| --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- | --- |
|  |  | TG | HDL-C | Apo A-I | Apo B | PON | ARE | HOMA-IR | D-LDL.C | BMI | hsCRP |
| T.Chol. | r | 0.614 | \* | 0.358 | 0.840 | \* | \* | \* | 0.781 | \* | \* |
|  | p | 0.000 | \* | 0,001 | 0,000 | \* | \* | \* | 0,000 | \* | \* |
| TG | r |  | -0.281 | \* | 0.677 | \* | \* | 0.353 | 0.512 | 0.357 | \* |
|  | p |  | 0.007 | \* | 0.000 | \* | \* | 0.001 | 0.000 | 0.001 | \* |
| HDL.C | r |  |  | 0.817 | \* | \* | \* | \* | \* | -0.384 | -0.237 |
|  | p |  |  | 0.000 | \* | \* | \* | \* | \* | 0.000 | 0.025 |
| Apo A-I | r |  |  |  | \* | \* | \* | \* | \* | \* | \* |
|  | p |  |  |  | \* | \* | \* | \* | \* | \* | \* |
| Apo B | r |  |  |  |  | \* | \* | \* | 0.811 | 0.234 | \* |
|  | p |  |  |  |  | \* | \* | \* | 0.000 | 0.007 | \* |
| PON | r |  |  |  |  |  | \* | \* | \* | \* | \* |
|  | p |  |  |  |  |  | \* | \* | \* | \* | \* |
| ARE | r |  |  |  |  |  |  | \* | \* | \* | \* |
|  | p |  |  |  |  |  |  | \* | \* | \* | \* |
| HOMA-IR | r |  |  |  |  |  |  |  | \* | 0.381 | \* |
|  | p |  |  |  |  |  |  |  | \* | 0.000 | \* |
| D-LDL.C | r |  |  |  |  |  |  |  |  | 0.399 | \* |
|  | p |  |  |  |  |  |  |  |  | 0.000 | \* |
| BMI | r |  |  |  |  |  |  |  |  |  | 0.570 |
|  | p |  |  |  |  |  |  |  |  |  | 0.000 |

\* Pearson correlation did not identify any statistical difference.

Table IV. Multiple linear regression analyses of BMI and other tests

|  |  |  |
| --- | --- | --- |
|  | β coefficeint | P  |
| T. Cholesterol | 0.012 | 0.957 |
| TG | 0.087 | 0.536 |
| HDL- Cholesterol | -0.286 | 0.153 |
| APO A-I | 0.119 | 0.498 |
| APOB | -0.187 | 0.329 |
| ARE | 0.010 | 0.900 |
| PON | 0.036 | 0.665 |
| D-LDL- Cholesterol | 0.275 | 0.103 |
| HOMA-IR | 0.238 | 0.006 |
| hsCRP | 0.436 | 0.000 |