Abstract. – OBJECTIVE: Obesity is a serious public health problem associated with excessive food intake. Regulation of food intake in highly organized organisms is under the control of a large number of orexigenic and anorexigenic molecules. Therefore, the main purpose of this study has been to determine the relationship between obesity and some of the circulating orexigenic and anorexigenic peptides that have a role in appetite control and to determine whether the concentrations of these molecules differ according to blood groups.

PATIENTS AND METHODS: The study included 400 individuals of whom 100 were obese women, 100 obese men, 100 healthy men and 100 healthy women. Obese women and men were divided into 4 groups, according to their blood groups. In the control group, healthy women and men were similarly divided into 4 blood groups. Each blood group within the groups, therefore, had 25 participants.

RESULTS: When leptin, nesfatin-1, obestatin and neuropeptide-Y, ghrelin and galanin levels of the control group and obese participants were compared, regardless of blood groups, leptin, nesfatin-1, obestatin and neuropeptide-Y were significantly higher, whereas only the ghrelin levels were significantly lower in obese patients. When the amounts of these hormones were measured according to gender, the situation was similar. When leptin, nesfatin-1, obestatin and neuropeptide-Y values of the control and obese participants’ blood groups were compared with each other; these hormones were high in all blood groups; however, leptin levels in A blood group, nesfatin-1 levels in AB and O blood group, obestatin levels in AB blood group, neuropeptide-Y levels in A, B, AB blood groups were significantly higher. When the ghrelin levels of the blood groups in the control group and obese participants were compared, it was only significantly lower in the AB blood group. The ghrelin levels in the other blood groups of the obese individuals were again low, but not significantly so. When the distribution of hormones according to gender was evaluated, a situation parallel to the above results was recorded.
CONCLUSIONS: Leptin, nesfatin-1, obestatin and neuropeptide-Y and galanin levels of obese individuals were significantly higher than the control values, whereas the ghrelin values were significantly lower regardless of blood groups. Also, these hormones in blood partly varied with ABO blood groups. These different concentrations of hormones in ABO blood groups might be related with stimulation or suppression of appetite in human. However, further studies in other ethnic groups are needed to confirm these results.

Key Words: Anorexigenic peptides, Blood group, Obesity, Orexigenic peptides.

Introduction

Obesity is due to an increase in the amount of adipose tissue in the body following intake of food that contains more energy than the body needs. This disease is one of the most important health problems of our era, with an increasing incidence in all developed and developing countries. The etiopathology of obesity has not been fully elucidated, but to date, neuroendocrine mechanisms, environmental and genetic factors that regulate energy balance have been implicated, and thus it has been considered a multifactorial disease. Energy intake and body weight in humans are partly regulated by peripheral orexigenic and anorexigenic peptide concentrations.

Leptin, an anorexigenic peptide derived from adipose tissue, sends information on adipose tissues to the brain, reducing nutrient intake and preventing excessive accumulation of fat. As leptin levels decrease, leptin resistance develops or age increases, weight gain increases, which means that one (or more) of the pathways leading to obesity become(s) important. Another satiety molecule, nesfatin-1 was discovered in 2006 by Oh-I et al. It has 82 amino acids with a molecular weight of 9.7 kDa. Intracerebroventricular (i.c.v) injection of nesfatin-1 reduces nutrient uptake in a dose-dependent manner. Appetite stimulation is seen when nesfatin-1 is neutralized by antibody injection. There is also the idea that nesfatin-1, encoded by the NUCB2 gene, is a satiety sensation in all animal experiments to date, and is capable of possessing the effects of a new leptin molecule. Another appetite-suppressing molecule is obestatin, encoded by the same gene as the ghrelin hormone, which suppresses weight gain. Obestatin is synthesized in many tissues, including the stomach, small intestine, hypothalamus and pituitary. It is released during eating and gives the feeling of satiety. Ghrelin triggers eating, whereas obestatin suppresses appetite. On the other hand, ghrelin (comprised of 28 amino acids) is also known as a fasting hormone. In biological fluids, there are 4 forms of ghrelin, including the one which is bound at the third serine amino acid at the N-terminus by octanyl, decanoic or desenoyl fatty acids, or there is one not bound to fatty acids (des-acyl ghrelin). The most important form that acts physiologically and biochemically is octenyl fatty acid bonded to Ghrelin. Therefore, octanoylated ghrelin measurement has been preferred in our investigations. However, unless otherwise stated, only the term “ghrelin” will be used instead of octanoylated ghrelin. Although the main site of synthesis of this peptide is the stomach, it might be synthesized in the hypothalamus, pituitary, salivary gland, thyroid gland, small intestine, kidneys, heart, pancreas alpha cells and gonads. The first physiological effect of ghrelin is on the so-called growth hormone-releasing receptor (GHS-R). In babies that are small for their gestational age, ghrelin affects pre-meal appetite more than post-meal appetite. Neuropeptide Y (NPY) is expressed abundantly in the mammalian central nervous system, particularly in the hypothalamus, which is also important in controlling nutrition. NPY is one of the most important appetite-stimulating agents known in mammals and is a widely dispersed peptide of 36 amino acids found in the central and peripheral nervous system. Acute administration of NPY has effects on fasting. Another molecule that is involved in the control of appetite is galanin, a peptide of 30 amino acids synthesized in the central nervous system and intestine, which regulates nutrition. Furthermore, the galanin receptors (GAL1R, GAL2R and GAL3R) are commonly found in central and peripheral tissues. When galanin is given, a feeling of hunger arises and increases energy intake, its chronic stimulation leading to weight gain.

Diet can be related to blood groups in order to provide regulate energy production. Orexigenic and anorexigenic peptides are involved in energy regulation. The metabolism of individuals varies according to blood groups; if this is the case, obese and healthy individuals with O, A, B and AB blood groups are likely to show differences in their concentrations of orexigenic and anorexigenic peptides. Despite our extensive literature
search, we found no evidence as to whether orexigenic and anorexigenic peptides levels differ with the blood groups. Therefore, our main purpose was to determine the amount of certain circulating orexigenic and anorexigenic peptides important in terms of appetite in obese and healthy individuals. By classifying these obese and healthy individuals according to their blood groups, we explored whether there is any connection between blood groups and obesity as indicated by possible differences in the amounts of orexigenic and anorexigenic peptides they express.

**Patients and Methods**

This study was approved by the Local Research and Ethics Committee (Firat University Ethics Committee, Approval Date: 06.02.2011; Numbered: 2011-151). The study was conducted in accordance with the principles of the Declaration of Helsinki. Informed consent has been obtained from all participants. Body mass index (BMI) was used to determine the degree of obesity of the participants. If the BMI value was 18.5-24.9 kg/m², this was accepted as normal weight (control); these healthy individuals did not have any known medical condition. The obese group was composed of first-degree obese patients with BMI values between 30-34.9 (kg/m²). The study included in all 400 participants, 100 obese women (25 individuals of each blood group), 100 obese men (25 individuals of each blood group) and 100 healthy men and women (25 individuals of each blood group). None of participants had irritable bowel syndrome, gastritis, Helicobacter infection.

Patients with high blood pressure, morbid obesity, diabetes, breastfeeding, alcohol use, gastrointestinal disorders, exercising regularly and of Rh (-) blood group were excluded. The obese patients included did not have any medical problems other than obesity. From these volunteer participants, 5 mL fasting blood at 09.00 am was taken into EDTA biochemical tubes containing aprotinin (protease inhibitor, after overnight fasting), as previously described. The plasma obtained by centrifugation at 4.000 rpm was stored at -80°C until examined. For blood group determination, monoclonal ABO-Rh blood group determination anti-sera were used.

**Analysis of Biological Samples**

Glucose, total cholesterol (TC), HDL-cholesterol (HDL-C), LDL-cholesterol (LDL-C), VLDL-cholesterol (VLDL-C), triglyceride (TG) values were measured by an autoanalyzer (Cobas 6000, Roche Hitachi, Tokyo, Japan). Orexigenic and anorexigenic peptides were determined by ELISA as indicated by the manufacturer. Leptin (Cat No: E1559Hu), NUCB2/nesfatin-1 (E3820Hu), obestatin (Cat No: E0790Hu), and galanin (Cat No: E1332Hu) were obtained from ELISA kit manufacturers (Bioassay Technology Laboratory, Shanghai, China). Ghrerlin (Cat No: A0506 was obtained from the ELISA kit manufacturer (SPI-BIO, Bertin Pharma, Bretonex, France). Neuropeptide-Y (Cat No: RSCYK080R) was obtained from the ELISA kit manufacturer (BioVendor Group, Brno, Czech Republic). The intra-assay (coefficient of variation; CV) and inter-assay (CV) values of the ghrelin ELISA kit were <10.3%, and <10.9%, respectively, and the minimum measured value was 1.5 pg/mL. The intra-assay value of the leptin ELISA kit used was <8%, the inter-assay value was <10% and the minimum measured value was 0.021 ng/mL. The intra-assay (CV) value of the NUCB2/nesfatin-1 ELISA kit was <8%, the inter-assay value was <10% and the minimum measured value was 0.021 ng/mL. The intra-assay (CV) value of the Neuropeptide-Y ELISA kit was 6.08-8.52%, the inter-assay (CV) value of the obestatin ELISA kit were <8%, and <10%, respectively, and the minimum measured value was 0.26 ng/L. The intra-assay (CV) value of the Galanin ELISA kit was <8%, the inter-assay value was <10% and the minimum measured value was 0.26 ng/L. The intra-assay (CV) value of the Neuropeptide-Y ELISA kit was 6.08-8.52%, the inter-assay value was 5.45-10.26%, and the minimum measured value was 0.082 ng/mL. At the end of the study, the kits with dif-
ferent measurement units were converted to ng/mL to help in their interpretation.

**Statistical Analysis**
Statistical analysis used the SPSS 22.00 (SPSS Inc., IBM, Armonk, NY, USA) package program. The spearman correlation correlation test was used to compare the data between the groups. 2 way ANOVA was used to analyze the data with control/obese and blood type as variables. Study parameters were given as mean ± standard deviation. $p < 0.05$ was considered statistically significant.

**Results**

Glucose, TC, LDL-C, VLDL-C and TG level of obese groups were higher, and HDL-C was lower compared with the control group. The differences were statistically significant (Table I). When similar data was obtained according to blood groups, there was little difference. Since these data showed no effect of blood group, it is appropriate to present the data here. Leptin, nesfatin-1, obestatin, galanin and NPY hormone values of the obese group were significantly increased and ghrelin values were significantly decreased when they were compared with the control group values (Figure 1-part I). Based on gender; obese women had higher concentrations of leptin, nesfatin-1, obestatin, galanin and NPY hormone levels, whereas ghrelin levels were lower than in obese men (Figure 1 (Tables II and III)). On the blood group basis without separating according to gender, leptin and NPY in the A blood group, NPY in the B blood group, galanin, nesfatin-1, obestatin and NPY in the AB blood group and nesfatin-1 in the O blood group were significantly higher in obese patients than in the control groups. On the other hand, although ghrelin was significantly lower in the AB blood group in obese patients, unlike the other blood groups (Figure 2).

On examining blood groups according to gender, obese women leptin and NPY in the A blood group, NPY in the B blood group, galanin, nesfatin-1, obestatin and NPY in the AB blood group and nesfatin-1 in the O blood group among obese women were significantly higher than those of control women in same blood groups. Although ghrelin was significantly lower in the AB blood group in obese women than in control women, the difference in other blood groups was not statistically significant (Figure 3). Hormone levels and changes in male and female groups were observed to be parallel to each other (Figure 4).

**Discussion**

It was assessed for the first time whether orexigenic and anorexigenic hormones differed in obese and control groups according to their blood groups. In this study, leptin, nesfatin-1, obestatin, NPY, ghrelin and galanin values of obese participants were compared with control groups regardless of blood groups; leptin, nesfatin-1, galanin, obestatin and NPY values of obese patients were usually significantly higher, whereas ghrelin values were significantly lower. In previous studies, leptin and NPY were high in obese individuals worldwide\(^4\)\(^{,27,28}\). On the other hand, when the concentrations of nesfatin-1, galanin and obestatin in obese individuals are compared with the control values, no consensus has yet been formed. In some populations, obese individuals had high values of nesfatin-1\(^29\), galanin\(^30\) and obestatin\(^31\). On the other hand, citizens from some countries (such as China and Turkey) have low val-

<table>
<thead>
<tr>
<th>Study Parameters</th>
<th>Control (n: 200)</th>
<th>Obese (n: 200)</th>
<th>$p$-value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glucose (mg/dL)</td>
<td>97 ± 11.82</td>
<td>117.82 ± 23.21</td>
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</tr>
<tr>
<td>TC (mg/dL)</td>
<td>164.73 ± 25.91</td>
<td>195.44 ± 37.54</td>
<td>0.0001</td>
</tr>
<tr>
<td>TG (mg/dL)</td>
<td>94.22 ± 38.02</td>
<td>146.42 ± 59.43</td>
<td>0.0002</td>
</tr>
<tr>
<td>HDL-C (mg/dL)</td>
<td>48.61 ± 7.43</td>
<td>43.22 ± 5.92</td>
<td>0.0231</td>
</tr>
<tr>
<td>LDL-C (mg/dL)</td>
<td>92.1 ± 21.41</td>
<td>123.41 ± 28.91</td>
<td>0.0001</td>
</tr>
<tr>
<td>VLDL-C (mg/dL)</td>
<td>18.94 ± 6.24</td>
<td>28.12 ± 16.92</td>
<td>0.0002</td>
</tr>
</tbody>
</table>

Values were given as mean ± standard deviation. HDL-C = high-density lipoprotein cholesterol; LDL-C = low-density lipoprotein cholesterol; TC = total cholesterol; TG = triglyceride; VLDL-C = very low-density lipoprotein cholesterol.
Figure 1. Comparison of the orexigenic and anorexigenic hormones of obese and healthy participants without consideration of blood groups. I = females + males; II = only females; III = only males; a = control vs. obese ($p < 0.05$); b = control vs. obese ($p < 0.01$).
Table II. Pearson’s correlation of BMI with glucose and lipids without considering blood groups and genders.

<table>
<thead>
<tr>
<th>Pair</th>
<th>Control (r-value)</th>
<th>Obese (r-value)</th>
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<tr>
<td>BMI vs. Glucose</td>
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<td>.49</td>
</tr>
<tr>
<td>BMI vs. TC</td>
<td>.26</td>
<td>.54</td>
</tr>
<tr>
<td>BMI vs. TG</td>
<td>.19</td>
<td>.37</td>
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<tr>
<td>BMI vs. HDL-C</td>
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<td>-0.042</td>
</tr>
<tr>
<td>BMI vs. LDL-C</td>
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<td>.42</td>
</tr>
<tr>
<td>BMI vs. VLDL-C</td>
<td>.22</td>
<td>.38</td>
</tr>
</tbody>
</table>

BMI = Body mass index; HDL-C = High-density lipoprotein cholesterol; LDL-C = Low-density lipoprotein cholesterol; TC = Total cholesterol; TG = triglyceride; VLDL-C = Very low-density lipoprotein cholesterol.

Table III. Pearson’s correlation of BMI with orexigenic and anorexigenic peptides.

<table>
<thead>
<tr>
<th>Pair</th>
<th>Control (r-value)</th>
<th>Obese (r-value)</th>
</tr>
</thead>
<tbody>
<tr>
<td>BMI vs. Ghrelin</td>
<td>-0.31</td>
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<td>BMI vs. Galanin</td>
<td>.28</td>
<td>.56</td>
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<tr>
<td>BMI vs. Obestatin</td>
<td>.22</td>
<td>.41</td>
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<tr>
<td>BMI vs. Neuropeptide Y</td>
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<td>0.49</td>
</tr>
<tr>
<td>BMI vs. Leptin</td>
<td>.33</td>
<td>.55</td>
</tr>
<tr>
<td>BMI vs. Nesfatin-1</td>
<td>.28</td>
<td>.47</td>
</tr>
</tbody>
</table>

BMI = Body mass index.

Figure 2. Comparison of orexigenic and anorexigenic hormones according to blood groups of obese and healthy all participants. a=control vs. obese (p < 0.05).

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ues,32-36 and in some societies, obese individuals indicated no significant difference of obestatin and galanin level30,37. Our data support the results of researchers who found that blood leptin, nesfatin-1, obestatin, NPY and galanin levels are higher in obese individuals4,27,28,38,39. In addition, this study found that blood ghrelin levels of obese individuals were as low as in previously reported studies40,41. Although the levels of blood ghrelin are low in obese individuals, the mechanisms underlying the high levels of leptin, nesfatin-1, obestatin and NPY and galanin can be explained as follows. Leptin hormone gives the message that “you are full up, stop eating” and ghrelin gives the message of “you are hungry, start eating” making. Leptin hormone terminates eating and ghrelin initiates it. Therefore, the likely cause of the high levels of leptin hormone circulating in obese individuals is that high levels of leptin are intended to prevent further weight gain by providing satiety, as many researchers have previously suggested4,27,28,38,42-44. Again, it is known that the hormone ghrelin gives the signal to become hungry; a decrease in the weight of obese individuals is intended to prevent weight gain by reducing appetite45. Therefore, when we

**Figure 3.** Comparison of orexigenic and anorexigenic hormones according to blood groups of obese and healthy only female participants. a=control vs. obese (p < 0.05).
Appetite hormones in the blood groups

combine the existing data on ghrelin and leptin with the findings of previous researchers, it is clear that losing weight reduces the amount of leptin in the blood and increases the amount of ghrelin\(^4,27,28,38,42-44\). In addition, when anorexic people gain weight, the amount of ghrelin in their blood decreases\(^46\). In other words, as the weight decreases, the amount of leptin hormone decreases and the decrease in leptin encourages eating\(^47,48\). The amounts of ghrelin and leptin in blood are an important factor governing body weight and have a major influence on energy balance\(^46,48\).

Neuropeptide Y blood concentrations were higher in obese\(^27\). NPY is found in central and peripheral neurons. This peptide is one of the most potent orexigenic peptides for eating motivation. In other words, it is a peptide that

Figure 4. Comparison of orexigenic and anorexigenic hormones according to blood groups of obese and healthy only male participants. a= control vs. obese (\(p < 0.05\)).
regulates nutrition and body weight and triggers carbohydrate consumption rather than fat consumption\textsuperscript{46}. Chronic administration of NPY to the brain causes chronic overnutrition and consequently obesity\textsuperscript{50}. Therefore, it is thought that obesity develops because of the high levels of NPY in obese individuals that encouraging their eating habits. Leptin inhibits NPY and food intake, but stimulates energy expenditure and thus reduces body weight\textsuperscript{51}. If this is the case, we cannot explain why NPY concentrations are not suppressed in obese subjects despite increased leptin levels. Because the physiological role of NPY in nutrition has not been established yet, and the natural effects of NPY in the hypothalamus cannot be fully explained either.

In this study blood galanin levels of obese individuals high. To date, the results of blood galanin levels in obese individuals are conflicting, some reports finding that their galanin levels are significantly higher compared with healthy controls\textsuperscript{30,52}, whereas others reported that they could not detect significant differences\textsuperscript{34,53}. Administration of galanin to the hypothalamus, particularly to the paraventricular nucleus, increases food intake in rats and fat consumption in mammals\textsuperscript{54}. In addition to mammals, galanin has an orexigenic effect on chicks and aquarium fish\textsuperscript{55,56}. Therefore, based on the high galanin levels reported in obese people, we suggest that high galanin causes weight gain, leading to obesity, by encouraging eating.

In this study, obestatin levels were also significantly higher in obese subjects compared to control subjects. However, there was no consensus regarding the control values and obestatin concentrations in obese individuals. In one study, obestatin was significantly lower in obese individuals compared with thin controls and might have played a role in long-term body weight regulation\textsuperscript{37}. The high levels of obestatin found in obese participants suggest a compensatory increase of obestatin for food intake and the control of body weight. In early studies with mice; obestatin had an anorexigenic effect after peripheral or intracerebroventricular injection\textsuperscript{58}. Our findings confirm these preliminary results, but they have recently been contradicted by studies reporting no inhibitory effect on food intake and body weight gain\textsuperscript{59,61}. It has also been reported that obestatin has no role in modifying serum levels of leptin\textsuperscript{10}. Guo et al\textsuperscript{62} indicated that obestatin increased with obesity and that preprandial ghrelin also increased, with the concentration being positively correlated with body mass index\textsuperscript{52}.

In this study, NUCB-2/nesfatin-1 levels were higher in obese individuals when compared with control subjects. Although there have been some exceptional results\textsuperscript{35,58}, the NUCB-2/nesfatin-1 amounts of obese individuals were significantly higher compared to the controls\textsuperscript{29}. Expression of adipose tissue NUCB-2/nesfatin-1 increases with obesity and changes in nutrition and malnutrition. Intraperitoneal (i.p.), nesfatin-1 administration reduces food intake in both thin and db/db leptin-resistant obese mice. Furthermore, peripheral nesfatin-1 induces anorexia independently of leptin. Thus, high NUCB-2/nesfatin-1, which we detected in obese individuals, is an anorexigenic hormone that is involved in weight control by mediating restriction of food intake\textsuperscript{63}.

In this study, when leptin, nesfatin-1, obestatin and NPY values of the control and obese participants’ blood groups were compared with each other; leptin levels in A blood group, nesfatin-1 levels in AB and O blood group, obestatin levels in AB blood group, neuropeptide-Y levels in A, B, and AB blood groups were higher. When the ghrelin levels of the blood groups in the control group and obese participants were compared, it was significantly lower only in the AB blood group. The ghrelin levels in the other blood groups of the obese individuals were also low but were not significantly different. Thus, the relationship between the fate of orexigenic and anorexigenic hormones and ABO blood groups is a novel feature of our investigation. Regardless of whether the amounts of orexogenic and anorexigenic hormones are related to ABO blood groups, some studies have found a link between obesity and ABO blood groups, while others have reported no link between obesity and ABO blood groups\textsuperscript{64,65}. The association of ABO blood groups with DM has been also observed in many epidemiological and genetic studies\textsuperscript{64,66,73-75}.

Results of the previous studies and this present study show that there is a direct link between some hormones that increase/decrease the appetite and obesity. The amount of orexigenic and anorexigenic hormones can vary with ABO blood groups. Therefore, these data on the relationship between ABO blood types and molecules that increase and decrease the appetite indicate that obesity may have genetic and immunological aspects. Since the present study is novel in investigating how the amounts of orexigenic and anorexigenic hormones change according to ABO blood groups, it might be useful to compare with the findings with future studies.
In one study, women with blood type B positive (B +) had a 35% chance of developing type 2 diabetes, but it was also reported that women with AB+, A and A+ blood types had a 26, 22 and 17% higher risk for developing type 2 diabetes, respectively. Many researchers have shown that the amounts of leptin, nesfatin-1, obestatin and NPY, ghrelin and galanin measured in patients with diabetes may be associated with this disease (although there are some conflicting reports). The obtaining different results with these hormones in studies with diabetic patients may be due to different distribution of the blood groups. So that blood leptin, nesfatin-1, obestatin ve NPY, ghrelin ve galanin were altered with blood types in obese subjects when compared with those in control subjects. So it appears that this observation supports the above-proposed argument.

As mentioned above, there are some reports that the amount of orexigenic and anorexigenic hormones varies in obese individuals from society to society. For example, nesfatin-1 is found to be low in obese Saudi individuals, whereas in obese Egyptian, nesfatin-1 was high. These different results of the researchers have been attributed to differences in ethnicity, race and applied research methods. However, available blood groups data suggest that these conflicting results may have been partly due to blood groups not being taken into account. Because the distribution of blood groups varies from society to society. A blood group is common in Turkish society whereas the zero (O) blood group is common in countries such as Chile and Ecuador. B blood group is common in countries such as Bangladesh and India, and the AB blood group is less common in all countries. In the studies carried out with molecules that open up and close down appetite, blood samples are taken randomly from the participants and are analyzed without regard for blood groups. The prevalence of blood groups in societies differs from each other; the differences between the populations and the lack of statistical significance may be since the blood groups of the participants were dissimilar. Since more common blood types in the society might be theoretically more blood samples (if you don’t check which blood types belong to your participants) when blood samples were randomly collected. Therefore, in these conditions, it is inevitable that the results will appear differently.

This study also had some limitations. First, we had low number of samples per group. Second, CART, CCK, GLP-1, PYY and so were be also considerable interest for these hormones in obesity research. Our study is the first to identify basal blood concentrations of some orexigenic and anorexigenic hormones in obese and nonobese individuals according to blood groups. These results warrant further studies to validate our first findings.

Conclusions

As a result, leptin, nesfatin-1, obestatin and NPY and galanin significantly higher in the circulation of obese individuals, whereas ghrelin was significantly lower when compared with regardless of blood groups. When the amounts of these hormones were related to gender, there was a similar situation. When leptin, nesfatin-1, obestatin and NPY values of the control group and obese participants’ blood groups were compared; these hormones were high in all blood groups, but leptin levels in the A blood group, nesfatin-1 levels in the AB and O blood group, obestatin levels in the AB blood group, neuropeptide-Y levels in the A, B, AB blood groups were significantly higher. When the ghrelin levels of the blood groups in the control group and obese participants were compared, it was significantly lower only in the AB blood group. The ghrelin levels in the other blood groups of the obese individuals were again low, but not significantly so. When the distribution of ABO blood group hormones according to gender was evaluated, a situation parallel to the above results was reported. Blood concentrations of the molecules that increase/decrease appetite differ in the ABO blood groups. Therefore, after the confirmation of our current results by independent laboratories in the future; if equal blood group distributions of the sample are achieved, then the inter-population differences in blood concentrations of the appetizing and unappetizing molecules can also be avoided. Present results might help to change the perspective of the struggle over obesity.

Conflict of Interest

The Authors declare that they have no conflict of interests.

Acknowledgements

Since some of the remaining materials used in this study were made with the contributions of Firat University, we would like to thank our university. Some preliminary results of this study were also presented orally at the International Congress of “Adnan Menderes University I. Internation-
al Health Sciences Congress” held between June 29 – 01 July in, Aydin 2017.

Authors’ Contribution
ZKK, SA (Suna Aydin), AA and SA (Suleyman Aydin) conceived the study, designed the experiments, and performed the experiments. ZKK, KU, YA, VC, MY, TB, MHY, MK, AA, AU, IS, TK, SA (Suleyman Aydin) and RFA analyzed data. ZKK and SA (Suleyman Aydin) drafted the manuscript. All authors revised the manuscript and approved the final manuscript.

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