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Research Article

# Investigations of insulin resistance in obese dogs

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**ABSTRACT**

The aim of the study was to investigate insulin resistance in overweight and obese dogs. Obesity is excessive fat accumulation in the body and is defined as being 30% above the ideal body weight. In the study, a total of 30 dogs were divided into 3 equal groups: ideal weight, overweight and obese. Fasting serum samples were collected and used to measure insulin (INS) and asprosin (ASP) levels using dog-specific ELISA kits. Glucose (GLU) and fructosamine (FRU) were also determined using biochemistry analyzer and Idexx test kits, respectively. HOMA-IR (homeostasis model assessment of insulin resistance), HOMA-β% (homeostasis model assessment of β cell function) and insulin-glucose ratio (IGR) were calculated using glucose and insulin values. In the study, ASP ($p<0.05$), INS ($p<0.05$), HOMA-β ($p<0.05$) and IGR ($p<0.05$) values of both overweight and obese dogs were higher than the ideal weight group. Very strong correlations were detected between INS and HOMA-IR ($p<0.01$), HOMA-β ($p<0.01$), IGR ($p<0.01$) in both groups. It was determined that insulin resistance developed in 60% of overweight dogs and 90% of obese dogs. It is thought that HOMA-IR, HOMA-β, IGR, INS, FRU and ASP can be used in the evaluation of insulin resistance in obese dogs.

## INTRODUCTION

Obesity is defined as accumulation of excess fat in the body. It is the most common nutrition-related health disorders in dog and cats. It generally occurs as a result of energy imbalance and develops when the energy consumed is more than the energy used (Preet et al., 2021; Ronja and Kölle, 2021). Various predisposing factors contribute to the development of obesity in dogs. These predisposing factors include inactivity, high-energy diet, race, gender, neutering, and the presence of some metabolic disorders including hyperadrenocorticism, hypothyroidism, and insulinoma (Buishand and Kirpensteijn, 2023; Preet et al., 2021; Ronja and Kölle, 2021). Obesity is a growing chronic health problem in dogs that 22-66 percent of dogs are claimed to be obese (Munoz-Prieto et al., 2018; Preet et al., 2021; Ronja and Kölle, 2021). Studies have revealed that cancer, diabetes mellitus (DM), heart diseases, hypertension, joint diseases and skin diseases are more common in obese dogs (Preet et al., 2021; Ramos and Castillo, 2020; Ronja and Kölle, 2021). Therefore, obesity negatively affects the health and welfare of dogs.

Insulin resistance is the phenomenon of tissues such as liver, muscle and fat not responding to insulin. In this case, the pancreas produces more insulin, and on the other hand, blood sugar rises because glucose cannot be used in energy production. Therefore, insulin resistance develops and this ends with the development of type 2 DM. Over time, these animals develop complications such as obesity, high blood pressure, high cholesterol and triglycerides, and low HDLP (Marchi et al., 2022; Preet et al., 2021; Ronja and Kölle, 2021).

Insulin resistance has been shown to be more common in both obese people (Li et al., 2021; Xu et al., 2021) and obese pets (Ramos and Castillo, 2020), and a relationship between type 2 DM and obesity is noted. In studies, increases in parameters such as HOMA-IR (homeostasis model assessment of insulin resistance), HOMA-β% (homeostasis model assessment of β cell function) insulin, glucose, triglyceride, total cholesterol and high density lipoprotein (HDLP) were determined in obese dogs (Imamura et al., 2013; Preet et al., 2021; Ronja and Kölle, 2021; Sung et al., 2010; Villar and Bravo, 2022). Additionally, significant increases in blood asprosin levels have been detected in obese people, and a relationship between asprosin and obesity and insulin resistance has been determined (Li et al., 2021; Naiemian et al., 2020; Silistre and Hatipoglu, 2020; Xu et al., 2021). There are many studies on obesity and insulin resistance in humans, but there are very limited studies on the development of insulin resistance in obese dogs. Asprosin is also suggested as a biomarker to identify insulin resistance in obese adults and children (Naiemian et al., 2020; Romere et al., 2016; Silistre and Hatipoğlu, 2020). However, there is no study on asprosin and insulin resistance in obese cats and dogs

Therefore, the aim of this study was to investigate insulin resistance in overweight and obese dogs. Additionally, the diagnostic value of asprosin in determining insulin resistance in these dogs was also investigated.



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## MATERIALS and METHODS

*Dogs*

A total of 30 owned dogs were used in the research. Dogs were divided into 3 groups: 10 overweight (n=10), obese (n=10), and ideal weight (n=10). The dogs were of different ages, breeds and genders.

This study was approved by the Animal Ethics Committee (AEC), Burdur Mehmet Akif University, Türkiye (No:929/2022).

*Evaluation of Obesity*

Body condition score (BCS) was used to assess obesity in dogs as described elsewhere (Chun et al., 2019; Williams and Buzhardt, 2022). Thus, BCS in dogs was scored in the range of 1-9. BCS 4-5/9 was considered ideal weight, while BCS 6/9 and 7-9/9 were considered overweight and obese, respectively. Dogs with any systemic disease and those that had surgery within 6 weeks were not included in the study.

*Blood Samples*

Fasting (8-12 hours) blood samples were taken from each animal into plane tubes and centrifuged at 4000 rpm for 20 minutes to prepare serum samples. These serum samples were then stored at -80°C until used.

*Biochemical analysis*

In the study, serum concentrations of insulin (SunRed, catalog number: 201-15-0201, Shangai-CHINA) and asprosin (My-Biosource catalog number: MBS2612398, San Diego-USA) were detected by using dog-specific ELISA kits. ELISA tests were applied according to the manufacturer's recommendations. Serum fructosamine values were determined by using dog-specific Idexx test kits (Idexx Catalyst One Chemistry Analyzer, USA). Serum glucose levels were measured by using a biochemistry device (Roche cobas integra 400 Plus, USA). Insulin and glucose values were then used to calculate HOMA-IR, HOMA-β and insulin-glucose ratio (IGR) for each dog. The formulas used in HOMA-IR, HOMA-β and IGR calculations are as shown below.

HOMA-IR: fasting glucose (mg/dl) x fasting insulin (mU/L)/405

HOMA-β: 20 x fasting insulin (µIU/ml)/fasting glucose (mmol/L)-3.5.

IGR: fasting insulin (mU/ml)/fasting glucose (mmol/L).

In the study, 2-fold dilutions were made for standard insulin (80mU/L-6.25mU/L) and asprosin (10ng/ml-0.156ng/ml) and the optical density (OD) of each well for insulin and asprosin was determined with a micro-ELISA plate reader (96A, Minray, CHINA) at a test wave-length of 450 nm. Regression analysis was performed with the OD values of the obtained standard dilutions in the Excel program, and the formulas derived for insulin and asprosin were used to calculate their levels in the test samples as follows.

Insulin in the test samples was calculated using the formulas y=66.459x-32967 ($R^2$=0.998) and asprosin y=4.9091x-1.4838 ($R^2$=0.9992).

*Statistical analysis*

The normality of the distribution of the data obtained in the study was determined by the Kolmogorov-Smirnov test. One-Way ANOVA (posthoc Duncan) test was used to analyze

**Table 1.** Parameters of Ideal weight, Overweight and Obese dogs (mean±standard deviation. SD)

| Parameters | Ideal weight (n=10) | Overweight (n=10) | Obese (n=10) | Median (min-max) |
|---|---|---|---|---|
| **ASP (ng/mL)** | 1.38±0.26 [b] | 2.15±0.61 [a] | 1.96±0.53 [a] | 1.69 (1.14-3.2) |
| **GLU (mg/dL)** | 74.87±12.00 [a] | 74.7±10.49 [a] | 86.5±19.39[a] | 76.5 (56-126) |
| **INS (mU/L)** | 13.49±5.19 [b] | 34.42±22.6 [a] | 43.13±27.75[a] | 20.32 (7.3-82.4) |
| **FRU (µmol/L)** | 273±34.91 [b] | 308±42.13[ab] | 344.16±42.13 [a] | 305 (194-427) |
| **HOMA-IR** | 2.44±0.88 [b] | 6.17±3.86 [ab] | 9.44±6.64[a] | 3.7 (1.36-19.73) |
| **HOMA-β (%)** | 64.06±33.62 [b] | 171.74±132.6 [a] | 178.61±115.88 [a] | 108.36 (3-438) |
| **IGR (µU/mL)** | 0.18±0.09 [b] | 0.48±0.36[a] | 0.5±0.32 [a] | 0.31 (0.09-1.22) |

ASP: asprosin. GLU: glucose. INS: insulin. FRU: fructosamine. HOMA-IR: homeostasis model assessment of insulin resistance. HOMA-β: homeostasis model assessment of beta-cell function. IGR: Insulin/glucose ratio. The significance of the deviations between the values of the groups is indicated with superscript letters. and the presence of different letters on the same line indicates the significance between the groups (p <0.05).

the significance of data differences between groups. Additionally, the correlation between parameters was analyzed with the Pearson's correlation coefficient (r) test. In the correlation test, negative (-) or positive (+) correlation values (r) were accepted as very weak (-,+ 0-0.19), weak (-,+ 0.2-0.39), moderate (-,+ 0, 4 -0.59), strong (-,+ 0.6-0.79) and very strong (-,+ 0.8-1.00) as defined by Meghanathan (2016). In the study, the cut-off value of each parameter in overweight or obese dogs was determined by ROC analysis and individual increases or decreases for each value were determined according to the cut-off values. As a result of ROC analysis, the parameters were given as AUC (Area), %sensitivity (%sns), %specificity (%sps) and cut-off value. Significance levels were accepted as $p<0.05$. Parameters were expressed as mean±standard deviation (mean±SD), median, minimum-maximum (min-max). SPSS 27.0 for Windows® package program (version 27.0 for Windows, SPSS Inc, Chicago) was used to perform the statistical analysis.

## RESULTS

In the study, serum concentrations of ASP ($p<0.05$), INS ($p<0.05$), HOMA-β ($p<0.05$) and IGR ($p<0.05$) were significantly higher in both overweight and obese dogs than ideal weight dogs. Additionally, FRU ($p<0.01$) and HOMA-IR ($p<0.01$) values of obese dogs were found to be higher than those of ideal weight group, however, these parameters of obese dogs were not statistically different those of overweight dogs (Table 1).

In the study, cut-off values were calculated for each parameter in the ROC analysis (Table 2), and the number and percentages of dogs showing an increase for a parameter were calculated (Table 3). In the obese group, increases in ASP, GLU, INS, FRU, HOMA-IR, HOMA-β and IGR values were detected in 8(80%), 9(90%), 9(90%), 10(100%), 9(90%), 8(80%) and 7(70%) dogs, respectively (Table 3). However, an increase in these parameters was detected in fewer dogs in the overweight group than in obese dogs (Table 3).

**Table 2.** Cut-off values of the parameters.

| | AUC (Area) | Cut-off (95 confidence intervals) (lower-upper bound%) | p value | sensitivity%-specificity% |
|---|---|---|---|---|
| **ASP (ng/mL)** | 0.125 | 1.62 (0-0.273) | 0.002 | 31.8-75 |
| **GLU (mg/dL)** | 0.338 | 76.5 (0.124-0.553) | 0.181 | 40.9-75 |
| **INS (mU/L)** | 0.227 | 15.89 (0.063-0.392) | 0.024 | 36.4-62.5 |
| **FRU (µmol/L)** | 0.151 | 295.5 (0.013-0.288) | 0.004 | 27.3-75 |
| **HOMA-IR** | 0.216 | 2.4 (0.056-0.376) | 0.019 | 27.3-75 |
| **HOMA-β (%)** | 0.239 | 60.23 (0.07-0.407) | 0.031 | 31.8-62.5 |
| **IGR (µU/mL)** | 0.253 | 0.175 (0.082-0.423) | 0.041 | 31.8-62.5 |

ASP: asprosin. GLU: glucose. INS: insulin. FRU: fructosamine. HOMA-IR: homeostasis model assessment of insulin resistance. HOMA-β: homeostasis model assessment of beta-cell function. IGR: Insulin/glucose ratio.

**Table 3.** Number and percentages of dogs showing increases in parameters.

| Parameters | Cut-off | Overweight (n=10) | Obese (n=10) |
|---|---|---|---|
| **ASP (ng/mL)** | 1.62 | 7(70%) | 8(80%) |
| **GLU (mg/dL)** | 76.5 | 6(60%) | 9(90%) |
| **INS (mU/L)** | 15.89 | 7(70%) | 9(90%) |
| **FRU (µmol/L)** | 295.5 | 4(40%) | 10(100%) |
| **HOMA-IR** | 2.4 | 6(60%) | 9(90%) |
| **HOMA-β (%)** | 60.23 | 6(60%) | 8(80%) |
| **IGR (µU/mL)** | 0.175 | 6(60%) | 7(70%) |

AASP: Asprosin. GLU: glucose. INS: insulin. FRU: fructosamine. HOMA-IR: homeostasis model assessment of insulin resistance. HOMA-β: homeostasis model assessment of beta-cell function. IGR: Insulin/glucose ratio.

Pearson correlation test was applied to determine correlations between parameters obtained from overweight and obese dogs. As a result of the analyses, very strong positive correlations were detected between INS and HOMA-IR, HOMA-β and IGR, between HOMA-IR and HOMA-β and IGR, and also between HOMA-β and IGR ($p<0.01$).

Asprosin is produced from adipose tissue in cases of hunger and anorexia and stimulates both glucose production from the liver and appetite by crossing the brain barrier (Duerrschmid et al., 2017; Li et al., 2021). Blood asprosin levels are found to be high in type 2 DM cases with insulin resistance (Li et al., 2021; Naiemian et al., 2020; Wang et al., 2018). However,

**Table 4.** Correlation coefficient (r) values between parameters of overweight and obese dogs.

| Groups | | ASP | INS | GLU | FRU | HOMA-IR | HOMA-β | IGR |
|---|---|---|---|---|---|---|---|---|
| **Overweight** | **ASP** | 1 | 0.279 | -0.059 | 0.578 | 0.294 | 0.249 | 0.257 |
| | **INS** | | 1 | -0.330 | -0.150 | 0.947** | 0.962** | 0.961** |
| | **GLU** | | | 1 | 0.069 | -0.029 | -0.558 | -0.560 |
| | **FRU** | | | | 1 | -0.106 | -0.189 | -0.182 |
| | **HOMA-IR** | | | | | 1 | 0.823** | 0.823** |
| | **HOMA-β** | | | | | | 1 | 1.00** |
| | **IGR** | | | | | | | 1 |
| **Obese** | **ASP** | 1 | -0.269 | 0.552 | 0.411 | -0.038 | -0.463 | -0.462 |
| | **İNS** | | 1 | 0.196 | -0.254 | 0.953** | 0.939** | 0.940** |
| | **GLU** | | | 1 | 0.472 | 0.453 | -0.109 | -0.108 |
| | **FRU** | | | | 1 | -0.131 | -0.373 | -0.377 |
| | **HOMA-IR** | | | | | 1 | 0.794** | 0.796** |
| | **HOMA-β** | | | | | | 1 | 1.00** |
| | **IGR** | | | | | | | 1 |

ASP: Asprosin. GLU: glucose. INS: insulin. FRU: fructosamine. HOMA-IR: homeostasis model assessment of insulin resistance. HOMA-β: homeostasis model assessment of beta-cell function. IGR: Insulin/glucose ratio. Pearson's correlation test: *: $p<0.05$. **:$p<0.01$.

## DISCUSSION

Body weight gain is defined as overweight or obesity and characterized by excess fat tissue increase. It is a increasing common nutritional disease in cats and dogs. An increase of 10-20% in ideal body weight is considered overweight, and increases above 20% are considered obesity (Preet et al., 2021). The causes of obesity are diverse and often result from an imbalance between energy intake and use. In other words, the dog takes in more calories than it consumes, and the excess energy consumed is stored as fat in the body. Nowadays, obesity emerges as a chronic problem, especially in animals that are housed in a home environment, neutered, and fed with a high-energy diet (Preet et al., 2021; Ronja and Kölle, 2021). It is a growing problem and 40-60% of dogs are reported to be overweight or obese (Munoz-Pierto et al., 2018; Preet et al., 2021). Studies have revealed that there is a relationship between obesity and the incidence of diseases such as osteoarthritis, diabetes mellitus, hypothyroidism, hypertension, cardiovascular diseases, respiratory distress, hyperthermia and cancer (Munoz-Pierto et al., 2018, Preet et al., 2021; Ronja and Kölle, 2021). Diabetes mellitus, hypothyroidism and hyperadrenocorticism have been reported to occur in 9%, 6% and 13% of dogs, respectively. It has also been shown that 40% of dogs with at least one of these diseases are obese (Oh, 2011; Preet et al., 2021).

it has also been shown that ASP contributes to the development of insulin resistance by impairing β cells secretion (Jung et al., 2019). Studies conducted in obese children and adults indicate that the level of asprosin in the circulation increases depending on the degree of obesity, and that there is a relationship between asprosin and obesity and insulin resistance (Li et al., 2021; Naiemian et al., 2020; Silistra and Hatipoglu, 202). In present study, ASP values of both overweight and obese dogs were found to be significantly higher than the ideal weight group ($p<0.05$). However, no statistically significant difference was found between these parameters in obese and overweight dogs. On the other hand, ASP values were found to be high in 7(70%) overweight and 8(80%) obese dogs compared to the ASP cut-off value. The reason for the high blood asprosin value determined in this study is probably due to stimulation of asprosin production from adipose tissue to balance the diet-induced energy consumption, which has been shown to decreases in obesity (Watanabe et al., 2006; Velasquez-Mieyer et al., 2003). It should not be forgotten that high blood asprosin level determined in obese dogs in this study may also play a role in the development of insulin resistance by affecting pancreatic functions as described by Jung et al (2019).

Insulin is a hormone produced by pancreatic β cells, and its production is stimulated when blood glucose rises above 110 mg/dl. It ensures that circulating glucose is taken up by cells and used for energy production. In cases where

blood glucose increases excessively, insulin induce that excess glucose is stored as glycogen in the liver and muscles (Li et al., 2021; Mehran and Johnson, 2012; Wondmkun, 2020). Maintenance of blood sugar homeostasis depends on the insulin sensitivity of tissues such as muscle, liver, and fat (Fazakerley et al., 2019; Petersen and Shulman, 2018). Insulin resistance is the phenomenon of liver, muscle and fat tissue cells not responding adequately to insulin. If this situation continues, insulin resistance develops and this situation ends with type 2 DM. In this case, pancreas begins to produce more insulin to keep blood sugar balanced. Depending on the degree of insulin resistance, blood insulin level increases while glucose level is normal/high. Over time, animals with insulin resistance develop complications such as obesity, high blood pressure, high cholesterol and triglycerides, and low HDLP (Munoz- Prieto et al., 2018; Ramos and Castillo, 2020; Wondmkun, 2020). Type-1 DM may develop in obese dogs when the pancreas cannot synthesize sufficient amounts of insulin, and insulin resistance may develop as a result of the pancreas producing excessive insulin (Petersen and Shulman, 2018). In Type 2 DM cases, glucose is constantly released from the liver and the pancreas has to constantly secrete insulin to reduce glucose (Sung et al. 2010; Villar and Bravo 2022). As a result, hyperglycemia, insulin resistance and pancreatic β cell dysfunction develop (Li et al., 2021; Titchenell et al., 2017). A link between insulin resistance and obesity has been indicated in studies. Diet-related energy expenditure decreases in obesity, and this decrease has been shown to be related to the degree of insulin resistance (Mehran and Johnson, 2012; Petersen and Shulman, 2018; Watanabe et al., 2006). Insulin resistance accompanied by hyperinsulinemia reduces diet-induced energy consumption despite increased appetite and decreased fat oxidation (Kasuga, 2006; Watanabe et al., 2006; Velasquez-Mieyer et al., 2003). In the current study, hyperinsulinaemia was observed in both obese and overweight dogs. However, no statistical difference was detected between glucose values of all the groups. According to the cut-off values, INS and GLU levels increased in 9 (90%) of the obese dogs, while INS and GLU levels increased in 7 (70%) and 6 (60%) of the overweight dogs, respectively. It has been reported that plasma insulin levels increase and glucose levels are normal or high due to the development of insulin resistance. (Sung et al., 2010; Villar and Bravo, 2022). Therefore, in this study, more insulin is probably produced by the pancreas to keep plasma glucose values at normal levels. Despite insulin resistance, glucose was kept at normal levels in some dogs, while in others this was not achieved (Sung et al., 2010; Villar and Bravo, 2022).

Fructosamine and HbA1C are used to monitor changes in blood glucose levels over the previous days. (Oikonomidis et al., 2023; Zeugswetter, 2021). According to the updated values of fructosamine levels in dogs (Idexx), the reference range for healthy dogs is given as 177-314µmol/L. In the present study, a similar cut-off value was calculated for dogs as 295.5µmol/L. In this study, FRU levels were found to be significantly higher in obese dogs than in both overweight and ideal weight dogs ($p<0.05$). According to cut-off values, FRU values were found to be high in 4 (40%) overweight and 10 (100%) obese dogs. It is thought that the number of dogs with increased FRU values observed in the study is related to the degree of obesity and insulin resistance.

Energy metabolism has been reported to be affected in obese dogs, and in addition to increases in insulin and fructosamine levels, HOMA-IR level has also been reported to increase (Ramos and Castillo, 2020; Sung et al., 2020; Villar and Bravo,2022; Zeugswetter, 2021). HOMA-IR, HOMA-β, and IGR are suggested to be useful biomarkers in diagnosing insulin resistance and type 2 DM in humans (Imamura et al., 2013; Sung et al., 2020; Villar and Bravo, 2022). However, there is no standard cut-off value for HOMA-IR and HOMA-β in both humans and animals, and different values have been obtained in studies. It is stated that the cut-off value for HOMA-IR varies between 2.54 and 2.80, and the cut-off value for HOMA-β (%) ranged between 72% and 87%. Therefore, values above 2.5 for HOMA-IR were considered insulin resistance in humans (Chissini et al., 2020; Harbuwono et al., 2023). Furthermore, HOMA-β, below 72-87% is considered a loss of pancreatic β-cell function, and above this value is considered an increase in pancreatic β-cell function (Endukuru et al., 2020; Ghasemi et al., 2015). In the present study, the cut-off values for HOMA-IR and HOMA-β (%) were found to be 2.4 and 60.23%, respectively. HOMA-β and IGR values of overweight and obese dogs were determined to be significantly higher than the ideal weight group ($p<0.05$). Additionally, HOMA-IR values of obese dogs were higher than ideal weight dogs ($p<0.01$). However, these parameters of obese dogs were not significantly different from those of overweight dogs. According to the cut-off values, increases in HOMA-IR, HOMA-B and IGR values were observed in 9 (90%), 8 (80%) and 7 (70%) dogs in the obese group, respectively. On the other hand, increases in these parameters were detected in a smaller number of overweight dogs. Increases in HOMA-IR, HOMA-β and IGR levels indicate that more insulin is produced from pancreatic β cells in obese dogs than in the ideal weight group, but the sensitivity of the cells to insulin decreases. Most likely, the severity and duration of insulin resistance affects blood insulin and glucose levels in obese and overweight dogs.

In the present study, very strong correlations were observed between INS and HOMA-IR, between HOMA-β and IGR, between HOMA-IR and HOMA-β and IGR, and between HOMA-β and IGR in both groups. Similar correlations between obesity and asprosin, insulin resistance, HOMA-β and HOMA-IR have also been reported in obese individuals (Li et al., 2021; Silistre and Hatipoğlu, 2020; Xu et al., 2021). The existence of strong positive correlations between HOMA-IR, HOMA-β and IGR reveals that insulin resistance develops in obese dogs and these parameters can be used in the diagnosis of insulin resistance in obese dogs.

Increases in INS, ASP, FRU, HOMA-β, HOMA-IR and IGR values reveal that energy metabolism is also affected in obese dogs and insulin resistance has developed in some of obese and overweight dogs. Results of the study indicated that insulin resistance in obese dogs is more severe than in overweight dogs. The possible reason for this is that the severity and period of obesity affect the development of insulin resistance, as reported in previous studies (Mehran and Johnson, 2012; Petersen and Shulman, 2018; Watanabe et al., 2006).

## CONCLUSION

In the present study, ASP, INS, FRU, HOMA-IR, HOMA-β and IGR values were high in both obese and overweight dogs, and insulin resistance developed in 60% of overweight and 90% of obese dogs. The existence of strong correlations between insulin resistance indices such as HOMA-IR, HOMA-β and IGR, and an increase in INS, FRU and ASP indicated that these parameters can be used to evaluate insulin resistance in obese dogs.

## DECLARATIONS

### Ethics Approval

This study was approved by the Animal Ethics Committee (AEC), Burdur Mehmet Akif University, Türkiye (No:929/2022).

### Conflict of Interest

Authors do not have any conflict of interest fort his study.

### Consent for Publication

Consent on publication was confirmed with approval from the Republic of Türkiye Ministry of Agriculture and Forestry, Directorate of Burdur Provincial (No: E-69877819-325.04.02-5917267).

### Competing Interest

The authors declare that they have no competing interests

### Author contribution

Idea, concept and design: HİG, EES

Data collection and analysis: HİG, EES

Drafting of the manuscript: HİG, EES

Critical review: HİG, EES

### Data Availability

Not applicable.

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