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FULL PAPER

Effectiveness of feline body mass index (fBMI) as new diagnostic tool for obesity

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Abstract

Feline body mass index (fBMI), BW/PCL, length from top of patella to end of calcaneus, was developed as a new diagnostic tool for obesity in cats. To evaluate the effectiveness of fBMI for obese cats in short term, 6 cats were induced weight gain by over-feeding with high fat diet and then they were induced weight reduction by restrict-feeding with low fat diet to measure changes in fBMI and plasma metabolite concentrations and enzyme activities. BCS 3 is correlated with fBMI 24.6–32.0, BCS 4 is correlated with fBMI 33.1–37.1 and BCS 5 is correlated with fBMI 29.9–40.3, respectively. On the correlation coefficient analysis by Pearson's method ($P < 0.05$), positive correlations ($r = 0.403$) were seen between the fBMI and plasma triglyceride (TG) levels. From these findings, fBMI seems to be more sensitive and useful indicator for obesity diagnosis in cats.

Key Words: Cat, Feline body mass index, Obesity

Introduction

Obesity is a pathological condition in which excess body fat has accumulated. Prevalence of overweight and obesity is reported as 25%–50% in cats[1,6,16]. In Japan, metabolic syndrome (MS), which is based on obesity, has increased in cats in recent years[14]. To diagnose MS at early stage, simple diagnostic tool for obesity is necessary for veterinary medicine. 5-point body condition score (BCS) are usually used for obesity judgement for cats in Japan. 5-point BCS method is convenient but is lacking in objectivity. For early diagnosis of obesity for cats the objective method should be developed[1,6]. Several studies of feline body mass index (BMI) or body fat mass calculation have been reported[2,5,8,9,11,15,17]. However, these reported methods have not been used among veterinary clinics because of their inaccuracy and complexity. We developed a new BMI for cats (feline BMI, fBMI)[13]. It was calculated as follows: fBMI = BW / length from top of patella to end of calcaneus (PCL) (kg/m). fBMI were precisely reflected body weight change and were positively correlated

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with plasma non-esterified fatty acid (NEFA) and triglyceride (TG) concentrations at early stage of feline obesity. fBMI $\geq$ 28.0 was regarded as overweight or obesity. This study was designed to clarify the detailed relationship between fBMI, BCS, and plasma metabolites concentrations in cats with fBMI $\geq$ 28.0. To evaluate the effectiveness of fBMI, cats were induced weight gain by over-feeding with high fat diet and then they were induced weight reduction by restrict-feeding with low fat diet and were measured changes in fBMI, BCS, and plasma metabolite concentrations and enzyme activities.

Material and Methods

Animals: Six clinically healthy mongrel male cats (41.0 $\pm$ 1.0 months, 4.2 $\pm$ 0.3 kg, fBMI 28.1 $\pm$ 1.6, BCS3.0 $\pm$ 0.3) were used in this study. Two cats were regarded as overweight by fBMI at starting period of this study. One was BW 4.64 kg, fBMI 32.0, BCS 3 and the other was BW 5.24 kg, fBMI 33.8, BCS 4. Firstly, all cats were fed on a high-fat dry diet (Nippon Pet Food Co., Ltd., Tokyo, Japan; water, 6.1%; crude protein, 30.6%; ester extract, 27.3%; crude fiber, 0.9%; crude ash, 4.7%; 489.7 kcalME/100 g) with 2.0 $\times$daily energy requirement (DER; $1.4 \times 70 \times BW^{0.75}$) for 4 weeks to induce weight gain. Secondly, these cats were fed on a low-fat dry diet (Nippon Pet Food Co., Ltd., Tokyo, Japan; water, 7.3%; crude protein, 40.8%; ester extract, 11.9%; crude fiber, 6.8%; crude ash, 5.2%; 382.3 kcalME/100 g) with 1.0$\times$ DER for 2 weeks to maintain their body weight. Finally, cats were fed on low-fat diet with 0.8$\times$ the amount of rest energy requirement (RER; $70 \times BW^{0.75}$) for 2 weeks to induce weight reduction according to Burkholder's weight reduction program[4]. During experiment, all cats were fed twice a day (8 : 00 AM and 4 : 00 PM). Daily amount of food consumption was calculated by subtracting the amount of left-over food from the amount of supplied food. All cats were maintained in individual cages and provided with water *ad libitum* at Narita Animal Science Laboratory Co., Ltd. (Narita, Japan). The animal room was maintained at 24.0 $\pm$ 2.0°C and 55.0 $\pm$ 10.0% relative humidity on a 12 : 12 h light; dark cycle (light on 8 : 00 AM to 8 : 00 PM). Approval for this work was given by the Narita Animal Science Laboratory Co., Ltd. Research Animal Ethical Committee (15-F043).

BW and BCS measurement: BW measurement and BCS assessment by 5 point scale (1. Thin, 2. Lean, 3. Ideal, 4. Overweight, 5. Obese), commonly used in Japan, were performed before blood collection.

Zoometry measurement: fBMI was calculated as BW/PCL (kg/m) as previously described[13]. PCL, length from top of patella to end of calcaneus, was measured using a commercial tape measure contacted to the body (Fig. 1). The measurements were performed by only one researcher to minimize the measurement error.

Blood Sampling: Three ml of blood were withdrawn from jugular vein into heparinized plastic tubes at start (0wk), 4wks and 8wks of experiment. Plasma samples were separated by centrifugation at 1,200 g for 10 min, 4°C. These samples were stored at −80°C until use.

Plasma parameter analysis: GLU, TG, total cholesterol (T-Cho) concentrations and aspartate aminotransferase (AST), alanine aminotransferase (ALT), alkaline phosphatase (ALP), lactate dehydrogenase (LDH) activities were measured using an auto-analyzer (JCA-BM2250, JOEL Ltd., Tokyo, Japan) with the manufacture's reagents at Monolis Inc. (Tokyo, Japan). NEFA was measured with commercial kits (NEFA-C, Wako Pure Chemical Industries Ltd., Osaka, Japan) at Nippon Veterinary and Life Science University. Plasma insulin (INS) and adiponectin (ADN) concentrations were measured with commercial ELISA kits, Lbis cat insulin kit (SHIBAYAGI Co., Gunma, Japan), mouse/rat adiponectin kit

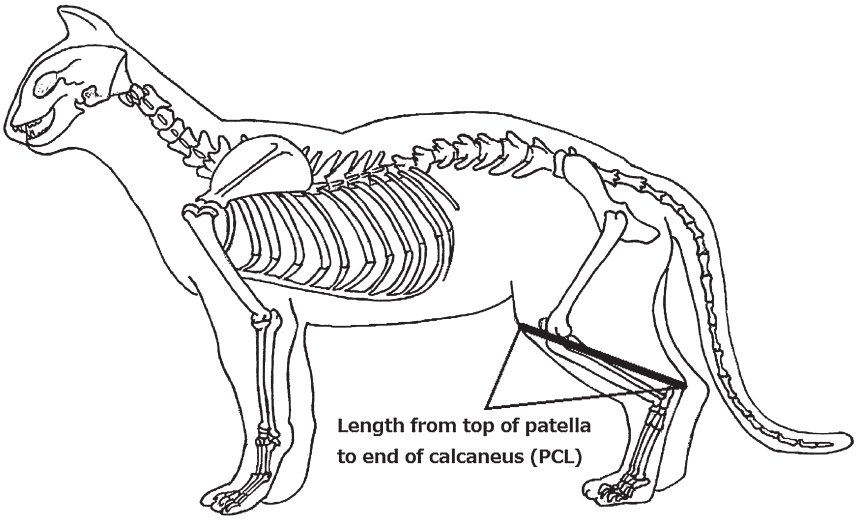

Fig. 1. Anatomic sites of length from top of patella to end of calcaneus in cats. PCL: Length from top of patella to end of calcaneus

(Otsuka Pharmaceutical Co., Ltd, Tokyo, Japan), respectively.

Statistical analysis: Results were presented as means ± standard error (SE). Statistical significances were determined by paired *t* -test. The significance levels were set at $P < 0.05$. Correlation coefficient between the fBMI and each parameter value was calculated by Pearson's method. The significance level was set at $P < 0.05$.

Results

Changes in physical measurements

At 4 weeks of weight gain period, BW, fBMI, BCS, were significantly increased. Average of fBMI and BW increased by 21.2% and 23.4%, respectively. At 8 weeks-weight reduction period, cat BW, fBMI, and BCS were decreased significantly compared to those at 4wks. Average of fBMI and BW at 8wks were decreased by 7.9%, 8.3%, respectively. There were no significant changes in PCL during whole experimental periods (Table 1). Cats with BCS 3 indicated in the range of fBMI 24.6–32.0 while cats with BCS 4 indicated in the range of fBMI 33.1–37.1, cats with BCS 5 indicated in the range of fBMI 29.9–40.3 (Table 3).

Changes in plasma parameters

At 4 weeks of weight gain period, plasma parameter levels of GLU, TG, TC, AST, ALT and ALP changed in the range of reference values. Plasma biomarker levels of LDH indicated above reference values. Plasma ADN concentrations significantly increased compared to those at start (0wk). At 8 weeks of weight reduction period, plasma NEFA concentrations were significantly higher than those at 4wks-weight gain period. Plasma INS concentrations at 8 weeks increased compared to those at 4 weeks while ADN concentrations decreased (Table 2).

Correlation between the fBMI and plasma parameter values

On the correlation coefficient analysis by Pearson's method ($P < 0.05$), positive correlations ($r = 0.403$) were seen between the fBMI and plasma TG level (Fig. 2). Correlation coefficient between the fBMI and GLU, NEFA, TC, ADN and INS levels were −0.207, −0.150, −0.308, −0.121 and −0.127, respectively.

Table 1. Changes in physical measurements in 6 cats

	Start 0wk	Weight gain period 4wks	Weight reduction period 8wks
BW (kg)	4.2 ± 0.3	5.2 ± 0.4[a)]	4.8 ± 0.3[a,b)]
fBMI (kg/m)	28.1 ± 1.6	34.1 ± 1.8[a)]	31.4 ± 1.8[a,b)]
BCS (/5)	3.0 ± 0.3	4.3 ± 0.3[a)]	3.3 ± 0.2[b)]
PCL (cm)	14.9 ± 0.2	15.2 ± 0.3	15.1 ± 0.3

Values are presented as means ± standard error.
a) Significantly different from the 0wk (Paired *t*-test, $P < 0.05$)
b) Significantly different from the 4wks (Paired *t*-test, $P < 0.05$)
BW: Body weight, fBMI: feline body mass index, BCS: Body condition score, PCL: Length from top of patellato end of calcaneus

Table 2. Changes in plasma metabolites at start (0wk), weight gain (4wk), and weight reduction period (8wk) in 6 cats

	Start 0wk	Weight gain 4wks	Weight reduction 8wks
GLU (mg/dl)	126.2 ± 13.0	97.8 ± 4.8	89.0 ± 1.8
TG (mg/dl)	18.7 ± 0.6	34.5 ± 4.8[a)]	23.7 ± 2.3
NEFA (mEq/l)	0.23 ± 0.04	0.20 ± 0.03	0.35 ± 0.07[b)]
T-Cho (mg/dl)	133.5 ± 4.2	113.7 ± 7.0	103.5 ± 4.9[a)]
AST (IU/l)	15.8 ± 1.4	20.5 ± 4.5	18.2 ± 2.8
ALT (IU/l)	42.7 ± 3.0	48.0 ± 3.3	49.5 ± 3.6
ALP (IU/l)	70.5 ± 10.8	152.0 ± 15.6	124.8 ± 20.8[a)]
LDH (IU/l)	119.8 ± 14.4	217.0 ± 76.9	132.2 ± 20.5
INS (ng/ml)	2.6 ± 0.1	2.9 ± 0.1	3.9 ± 0.9
ADN (μg/ml)	6.6 ± 1.8	11.7 ± 1.5	5.1 ± 1.1

Values are presented as means ± standard error.
a) Significantly different from the 0wk (Paired *t*-test, $P < 0.05$)
b) Significantly different from the 4wks (Paired *t*-test, $P < 0.05$)
GLU: Glucose, TG: Triglyceride, NEFA: Non-esterified fatty acid, T-Cho: Total cholesterol, AST: Aspartate aminotransferase, ALT: Alanine aminotransferase, ALP: Alkaline phosphatase, LDH: Lactate dehydrogenase, INS: Insulin, ADN: Adiponectin

Table 3. Comparison of fBMI ranges with BCS

BCS	fBMI		
	mean ± SE	minimum	maximum
2	23.7 (1)		
3	28.4 ± 0.8 (9)	24.6	32.0
4	34.6 ± 0.8 (5)	33.1	37.1
5	36.2 ± 3.2 (3)	29.9	40.3

Discussion

In the present study, we could induce early stage of obesity in adult cats. Small changes at PCL, length from top of patella to end of calcaneus, were measured using a commercial tape measure at 4 weeks of weight gain period. It is not clear they may come from fat deposition or muscle enlargement in legs. It is well known that obese cats increase fat deposition in organs, such as adipose tissue, muscle, and liver[12,18,20)]. We have to clarify PCL is constant or not in young cats since fBMI may be appropriate to access their obese degree. Changes in fBMI are well correlated with those of BW and BCS at start, weight-gain period and weight-reduction period. As compared to BCS, fBMI seems to be a more sensitive in overweight cats. From our previous data[13)], it was suggested that fBMI could evaluate

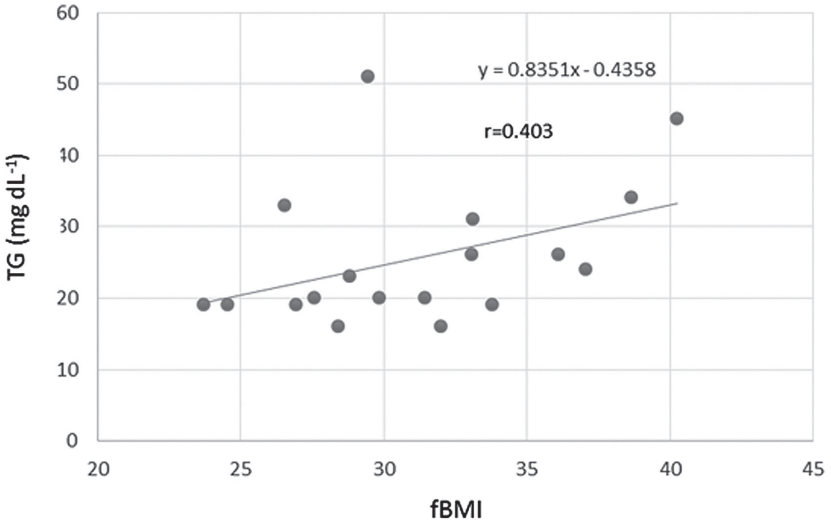

Fig. 2. Correlation analysis between the fBMI and plasma TG level by Pearson's method. Significance level was set at R $\geqq$ 0.600, $<$ $-$0.600, P $<$ 0.05.

early stage of obesity before onset of INS resistance and injury in liver function. Precise reference ranges of fBMI should be further studied since cats with BCS 5 indicated wide fBMI ranges in the present study.

At 4 weeks of weight gain period, ADN, a plasma lipid metabolic hormone, increased. ADN is a cytokine secreted from adipose tissue, and regulates glucose and lipid metabolism and increase insulin sensitivity and has anti-inflammatory effect[3]. In general, plasma ADN concentrations significantly decrease and metaflammation occurs in obese animals[7].

At 8 weeks of weight reduction period, plasma NEFA levels increased compared to those of 4wks-weight gain period. Hoenig *et al.* (2007) reported obese cats increased NEFA clearance in their plasma[10]. Wilding (2007) reported elevated plasma NEFA levels could lead to insulin resistance[19]. In the present study, plasma INS concentrations increased at 8wks- weight reduction period and were accompanied by decrease of ADN concentrations.

From the present data, cats at 4wks -weight gain period seem to be healthy and not pathological. We define this BW gaining stage is "healthy weight gain" in cats. We demonstrated the effectiveness of fBMI in this period. Further study should be focused on the effectiveness of fBMI for 1) young cats, 2) cats with pathological obesity and 3) species specificity.

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