Relationship between Soluble Leptin Receptor, Leptin, Lipid Profiles and Anthropometric Parameters in Overweight and Obese Thai Subjects

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Median, range and 95% confidence interval (CI) for median of age, anthropometric variables, soluble leptin receptor, serum leptin and lipid profile levels of 48 overweight (Body mass index (BMI) = 25.00-29.99 kg/m^2) and obese (BMI $\geq$ 30.00 kg/m^2) Thai males and 166 overweight and obese Thai females, compared with 26 males and 81 females in a control group (BMI = 18.50-24.99 kg/m^2), were determined. The study subjects were persons who turned up regularly for physical check-ups at the Out-patient Department, General Practice Section, Ratchawithi Hospital, Bangkok, aged between 18-60 years. Serum leptin, triglyceride and low density lipoprotein cholesterol/high density lipoprotein cholesterol ratios (LDL-C/ HDL-C ratio) were significantly higher in the overweight and obese males and females. Soluble leptin receptor and HDL-C were significantly lower in the overweight and obese males and females. Cholesterol and LDL-C were significantly higher in the overweight and obese females, but there was no significant difference in the overweight and obese males when compared with the control males. Low soluble leptin receptor levels were found in 38.1% (8/21) of the overweight and obese males, while 31.5% (29/92) were found in the overweight and obese females. Elevated leptin levels were found in 66.7% (32/48) and 89.8% (149/166) of the overweight and obese males and females, respectively. Both low soluble leptin receptor levels and elevated leptin levels were found in 9.5% (2/21) and 29.4% (27/92) of the overweight and obese males and females, respectively. A significant positive correlation was found between soluble leptin receptor and cholesterol, and between weight, BMI, waist, hip and HDL-C, with leptin. Serum soluble leptin receptor levels were significantly negatively correlated with leptin and BMI. The results can elucidate the causes and consequences of obesity, and are expected to aid the provision of care for overweight and obese Thai people.

Keywords: *Serum leptin receptor, leptin, BMI, lipid profiles, obesity*

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Obesity has increased rapidly and is a health problem among the Thai people. The prevalence of obesity is 11% among the Thai elderly[1] and 23.6% among female Thai construction-site workers[2]. Obesity is determined by an interaction between environmental, psychosocial, and genetic factors[3].

Leptin is a protein hormone that is encoded by the *ob* gene and is secreted by adipose tissue into

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the circulation. Leptin acts mainly in the hypothalamus by binding to specific leptin receptor and regulates food intake and energy balance[4]. Serum leptin concentrations have shown a positive correlation with BMI, percentage of body fat, and fat mass[5].

Leptin receptor was identified as a member of the cytokine family of receptors. The leptin receptor gene was found to encode at least five alternatively spliced forms, ob-Ra, ob-Rb, ob-Rc, ob-Rd, and ob-Re[6]. A soluble form of leptin receptor (sob-R) is an extracellular region plasma protein that binds leptin in the circulation. Leptin receptor has been found in most tissue; particularly in the central nervous system, pancreas, kidney, liver, skeletal muscles, adrenal marrow and cortex, endothelia, reproductive organs, and hematopoetic structure[7,8].

In Thailand, leptin receptor data have not been studied. Therefore, the aim of the present study was to investigate changes in serum soluble leptin receptor in overweight and obese subjects compared with control subjects, and to evaluate the relationship between serum soluble leptin receptor, serum leptin, lipid profiles, and anthropometric parameters.

Material and Method

Subjects

The study subjects were 48 male and 166 female overweight and obese Thais (BMI ≥ 25.00 kg/m^2), and the control subjects were 26 male and 81 female Thais (BMI = 18.50-24.99 kg/m^2). Thai subjects who turned up regularly for a physical check-up at the Obesity Clinic, Out-patient Department, General Practice Section, Ratchawithi Hospital, Bangkok, aged between 18-60 years, were investigated in the present study. The age, marital status, place of origin, drinking and smoking habits were assessed through standardized questionnaires. Exclusion criteria were diabetes mellitus, hypertension, cardiovascular disease, and unwillingness. All subjects were apparently healthy. Physical examinations were conducted by the same medical doctor throughout the study. The study protocol was approved by the Ethics Committee of the Faculty of Tropical Medicine, Mahidol University, Bangkok, and informed consent was obtained from each participant. More detailed information concerning the subjects is shown in Table 1.

Analytical method

The nutritional status of all subjects was assessed by means of anthropometric measurements. The body weight of each individual dressed in light clothing was measured using a carefully calibrated beam balance (Detecto®). The height of each individual was measured using a vertical-measuring rod; waist and hip circumferences were also measured to calculate waist/hip ratio. BMI was calculated as weight

Table 1. Descriptive data for the overweight / obese and control subjects

	Obese/overweight		Control	
	Male n (%)	Female n (%)	Male n (%)	Female n (%)
Age distribution: (years)				
18-30	9/48 (18.8%)	46/166 (27.7%)	9/26 (34.6%)	23/81 (28.4%)
31-40	15/48 (31.2%)	51/166 (30.7%)	7/26 (26.9%)	28/81 (34.6%)
41-50	18/48 (37.5%)	63/166 (38.0%)	8/26 (30.8%)	27/81 (33.3%)
> 50	6/48 (12.5%)	6/166 (3.6%)	2/26 (7.7%)	3/81 (3.7%)
Income: (Baht)				
Low (≤ 5,000)	8/45 (17.8%)	63/164 (38.4%)	5/26 (19.2%)	14/79 (17.7%)
Middle (5,001-10,000)	17/45 (37.8%)	65/164 (39.6%)	7/26 (26.9%)	28/79 (35.5%)
High (≥ 10,001)	20/45 (44.4%)	36/164 (22.0%)	14/26 (53.9%)	37/79 (46.8%)
Alcohol drinking:				
No	11/45 (24.4%)	109/164 (66.4%)	3/26 (11.5%)	53/80 (66.3%)
Yes	26/45 (57.8%)	47/164 (28.7%)	22/26 (84.6%)	26/80 (32.5%)
Quit	8/45 (17.8%)	8/164 (4.9%)	1/26 (3.9%)	1/80 (1.2%)
Smoking:				
No	28/45 (62.2%)	149/163 (91.4%)	16/26 (61.5%)	81/81 (100%)
Yes	11/45 (24.4%)	10/163 (6.1%)	7/26 (26.9%)	0/81 (0%)
Quit	6/45 (13.4%)	4/163 (2.5%)	3/26 (11.6%)	0/81 (0%)
Obese parent:				
No	18/42 (42.9%)	60/156 (38.5%)	16/25 (64%)	46/79 (58.2%)
Yes	24/42 (57.1%)	96/156 (61.5%)	9/25 (36%)	33/79 (41.8%)

(in kg) divided by squared height (in m^2). Subjects were grouped according to BMI as follows: underweight, BMI < 18.50; normal weight, 18.50 ≤ BMI < 25.00; overweight, 25.00 ≤ BMI < 30; and obese, 30.00 ≤ BMI < 35.00 (class I), 35.00 ≤ BMI < 40.00 (class II), and BMI ≥ 40.00 (class III)[9]. Waist and hip circumferences were also measured to calculate waist/hip ratios (normal value for females < 0.77, males < 0.90)[10,11].

Blood samples were taken early in the morning, 12 hours postprandially. About 10 ml of venous blood was drawn from the subjects. The serum samples were stored at 2-5°C for not more than 24 hours prior to lipid profile determination. A serum aliquot was stored frozen at -70°C for serum leptin and soluble leptin receptor.

Laboratory techniques

Cholesterol, high density lipoprotein cholesterol (HDL-C), low density lipoprotein cholesterol (LDL-C) and triglycerides (TG) were determined using a commercially available Boehringer Mannheim (Germany) test kit. Values of ≥ 200.0 mg/dl or ≥ 250.0 mg/dl of cholesterol, ≥ 150.0 mg/dl of LDL-C, ≤ 35.0 mg/dl of HDL-C, and ≥ 200.0 mg/dl of TG, were set as cut-off points[11].

Serum leptin levels were measured using a radioimmunoassay from Linco Research Inc., which utilized ^{125}I-labeled human leptin and human leptin antiserum. The detection limit of the assay was 0.5 ng/ml and the upper limit of linearity was 100 ng/ml. Values for males of < 5.6 ng/ml and females of < 10.8 ng/ml of leptin were used as cut-off points[11].

Soluble leptin receptor levels were measured using a commercially available sandwich enzyme-linked immunosorbent assay (ELISA; Biovendor Laboratory Medicine, Czech Republic) composed of two monoclonal antibodies raised against the extracellular domain of the leptin receptor. This test is 100% specific for soluble leptin receptor and the sensitivity is 0.4 u/ml. A value of < 10.0 u/ml of soluble leptin receptor was set as the cut-off point. (1 unit (U) equivalent of soluble native human leptin receptor equals 2 ng of the recombinant standards.)

Statistical analysis

The statistical computer software package MINITAB[12] was used to analyze the data. The median, range, and 95% confidence interval (C.I.) for median, were calculated. The individual parameters detected in the control group and in the group of overweight and obese subjects were compared using the Mann-Whitney U Test (two tailed). The relationships between the individual parameters were evaluated using Spearman's correlation. P-value < 0.05 was considered statistically significant.

Results

Median, range and 95% confidence interval (CI) for age, anthropometric variables, soluble leptin receptor, serum leptin and lipid profile levels, are shown in Table 2. The median ages of the overweight

Table 2. Medians, ranges and 95%CI of age, anthropometric variables, leptin, leptin receptor and lipid profiles in overweight/obese and control subjects

	Overweight/obese			Control			p-value
	Median (range)		95% CI	Median (range)		95% CI	
Age (yrs)	38.0	(18.0-58.0)	38.8-45.2	37.0	(18.0-55.0)	30.0-42.0	0.194
Weight (kg)	76.8	(54.0-129.2)	81.0-91.5	54.4	(50.7-78.0)	58.9-64.7	0.000
Height (m)	1.56	(1.45-1.84)	1.67-1.72	1.59	(1.57-1.85)	1.65-1.71	0.105
BMI (kg/m^2)	31.02	(25.19-53.28)	29.18-31.93	21.91	(18.68-24.61)	20.61-23.23	0.000
Waist (cm)	91.0	(66.5)	93.0-103.0	72.5	(64.0-89.0)	74.7-83.7	0.000
Hip (cm)	108.0	(89.5-129.0)	103.4-110.0	92.0	(87.0-102.0)	91.0-95.4	0.000
Waist/hip ratio	0.84	(0.67-1.01)	0.91-0.94	0.78	(0.72-0.93)	0.82-0.88	0.000
Leptin (ng/ml)	19.55	(2.0-30.0)	6.1-9.8	9.0	(1.0-9.0)	2.5-5.0	0.000
Leptin receptor (unit/ml)	11.2	(4.4-36.3)	9.33-12.66	15.3	(10.4-25.0)	15.15-23.21	0.000
Cholesterol (mg/dl)	215.0	(136-284.0)	206.4-233.4	203.0	(155.0-280.0)	197.5-220.4	0.005
HDL-C (mg/dl)	49.0	(26.0-73.0)	40.0-46.0	59.0	(43.0-84.0)	48.0-58.4	0.000
LDL-C (mg/dl)	138.0	(55.0-206.0)	127.8-153.2	129.0	(81.0-193.0)	112.9-148.4	0.028
LDL-C/HDL-C ratio	2.78	(1.59-4.59)	2.87-3.66	2.19	(1.27-3.78)	2.02-2.98	0.000
Triglycerides (mg/dl)	123.5	(56.0-740.0)	131.8-169.4	69.0	(45.0-426.0)	67.3-105.1	0.000

BMI = body mass index, HDL-C= high density lipoprotein cholesterol, LDL-C= low density lipoprotein cholesterol
*Mann-Whitney U Test (two- tailed). Significant difference between overweight/obese and control subjects p < 0.05

and obese males and females were 40.5 years (range 18.0 to 55.0) and 37.0 years (range 18.0 to 58.0), respectively. The median ages of the control males and females were 35.5 years (range 19.0 to 54.0) and 37.0 years (range 18.0 to 55.0), respectively. The median age did not differ significantly between the two groups. All of the anthropometric variables, except height, were significantly higher in the overweight and obese males and females. Serum leptin, triglyceride and LDL-C/HDL-C ratio were significantly higher in the overweight and obese males and females. Conversely, soluble leptin receptor and HDL-C were significantly lower in the same group. Cholesterol and LDL-C were significantly higher in the overweight and obese females but not in the males. The median serum leptin levels of overweight and obese females were significantly higher than those of the males in the same group, whereas the median soluble leptin receptor of the overweight and obese females did not show any significant difference between the sexes.

Elevated leptin levels were found in 66.7% (32/48) and 89.8% (149/166) of the overweight and obese males and females, respectively. Low soluble leptin receptor levels were found in 38.1% (8/21) and 31.5% (29/92) of the overweight and obese males and females, respectively. Both low soluble leptin receptor levels and elevated leptin levels were found in 9.5% (2/21) and 29.4% (27/92) of the overweight and obese males and females, respectively. Cholesterol concentrations of ≥ 250.0 mg/dl were found in 18.8 and 21.1% of the overweight and obese males and females, respectively. However, the prevalence of low HDL-C (HDL-C ≤ 35.0 mg/dl) was found to be 20.8 and 4.2% in the overweight and obese males and females, respectively (Table 3).

The relationships between the studied parameters in the overweight and obese group are shown in Table 4. A significant positive correlation was found between soluble leptin receptor and cholesterol. In addition, significant positive correlations were found between weight, BMI, waist, hip and HDL-C, with leptin. Significant negative correlations were detected between leptin and age, height, waist/hip ratio and LDL-C/HDL-C ratio, in both overweight and obese subjects.

There was a strong positive correlation between serum leptin level and BMI, as shown in Fig. 1. On the contrary, serum soluble leptin receptor levels were significantly negatively correlated with BMI, as shown in Fig. 2. Serum soluble leptin receptor levels were also significantly negatively correlated with leptin, as shown in Fig. 3.

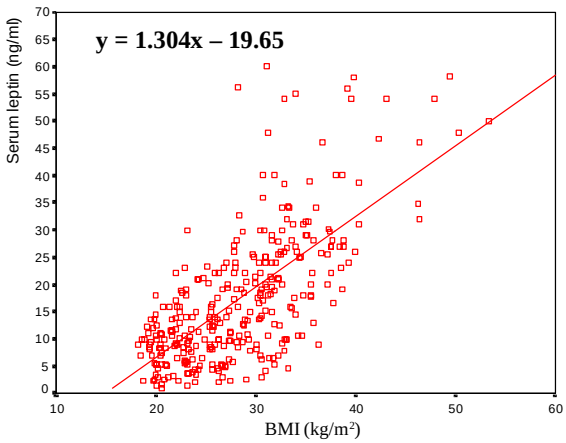

Fig. 1 Relationships between serum leptin levels and body mass index (BMI) in all subjects, as determined by linear regression analysis

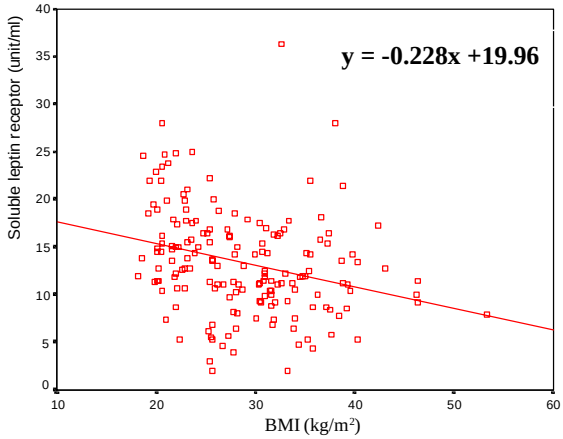

Fig. 2 Relationships between soluble leptin receptor levels and body mass index (BMI) in all subjects, as determined by linear regression analysis

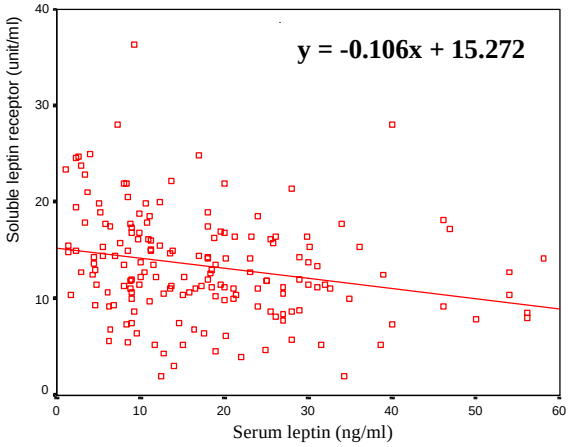

Fig. 3 Relationships between soluble leptin receptor levels and serum leptin levels in all subjects, as determined by linear regression analysis

Table 3. Number and percentage of individuals with abnormal leptin, abnormal leptin receptor, prevalence of dyslipidemia, in overweight and obese subjects

Parameter	Male		Female		Total	
	n/Total	%	n/Total	%	n/Total	%
Grading of overweight and obese by BMI (kg/m^2)						
Overweight or preobese (BMI = 25.00-29.99)	21/48	43.8	61/166	36.7	82/214	38.3
Obese grade I (BMI=30.00-34.99)	20/48	41.6	66/166	39.8	86/214	40.2
Obese grade II (BMI=35.00-39.99)	7/48	14.6	28/166	16.9	35/214	16.4
Obese grade III (BMI $\geq$ 40.00)	0/48	0.0	11/166	6.6	11/214	5.1
Waist/Hip ratio					181/214	84.6
Male $\geq$ 0.90	36/48	75.0				
Female $\geq$ 0.77			145/166	87.3		
Leptin					181/214	84.6
Male >5.6 ng/ml	32/48	66.7				
Female >10.8 ng/ml			149/166	89.8		
Leptin receptor: < 10.0 unit/ml	8/21	38.1	29/92	31.5	37/113	32.7
Leptin: Male >5.6 ng/ml, Leptin receptor: < 10.0 unit/ml	2/21	9.5				
Leptin: Female >10.8 ng/ml, Leptin receptor: < 10.0 unit/ml			27/92	29.4		
Dyslipidemia						
Cholesterol $\geq$ 200.0 mg/dl	32/48	66.7	112/166	67.5	144/214	67.3
Cholesterol $\geq$ 250.0 mg/dl	9/48	18.8	35/166	21.1	44/214	20.6
HDL-C $\leq$ 35.0 mg/dl	10/48	20.8	7/166	4.2	17/214	7.9
LDL-C $\geq$ 150.0 mg/dl	20/48	41.7	61/166	36.7	81/214	37.9
LDL-C/HDL-C > 5.0	0/48	0.0	3/166	1.8	3/214	1.4
Triglycerides $\geq$ 200.0 mg/dl	13/48	27.1	22/166	13.3	35/214	16.4

Table 4. Correlation coefficients of age, anthropometric variables, leptin, leptin receptor and lipid profiles in overweight/obese males and females (BMI $\geq$ 25.00 kg/m^2)

Parameter	Leptin	Cholesterol	HDL-C	LDL-C	LDL-C/HDL-C	Triglycerides
Age	-0.291**	0.287**	0.222**	0.231**	0.017	-0.002
Weight	0.355**	-0.109	-0.155*	-0.124	0.006	0.096
Height	-0.322**	-0.102	-0.207*	-0.077	0.081	0.098
BMI	0.618**	-0.051	-0.044	-0.086	-0.039	0.052
Waist	0.275**	-0.045	-0.127	-0.070	0.036	0.081
Hip	0.576**	-0.090	0.014	-0.131	-0.103	0.036
Waist/Hip ratio	-0.170*	0.029	-0.201**	0.033	0.162*	0.085
Leptin	1.000	-0.034	0.175*	-0.086	-0.176*	-0.069
Leptin receptor	-0.003	0.197*	0.168	0.082	-0.042	0.076
Cholesterol	-0.034	1.000	0.194**	0.885**	0.553**	0.333**
HDL-C	0.175*	0.0194**	1.000	0.053	-0.598**	-0.348**
LDL-C	-0.086	0.885**	0.053	1.000	0.726**	-0.149*
LDL-C/HDL-C	-0.176*	0.553**	-0.598**	0.726**	1.000	0.119
Triglycerides	-0.069	0.333**	-0.348**	-0.149*	0.119	1.000

Significant difference: *p < 0.05, **p < 0.01

Discussion

In the present study, higher leptin concentrations were found in Thai overweight and obese males and females (BMI $\geq$ 25.00 kg/m^2) than the normal controls. The medians of leptin in the overweight and obese females were significantly higher than those of the overweight and obese males. This can reflect gender differences in body composition and fat distribution, or the inducing effects of sex steroids such as estrogen, progesterone and androgens on leptin production[13,14]. However, some obese humans have low levels of leptin. Although a high leptin concentration was found to correlate positively with the degree of obesity in humans, a high level does not effect a decrease in weight. The reason for this may be the presence of an abnormal leptin and leptin receptor protein in humans.

The results also showed that soluble leptin receptor was significantly lower in the overweight and obese males and females than the normal control males and females. The medians for soluble leptin receptor of the overweight and obese females were not significantly different from the overweight and obese males. The reduction in soluble leptin receptors might reflect a downregulation of leptin receptor, because the number of leptin receptors may be reduced parallel to increased circulating leptin concentrations and might be a certain compensation for leptin resistance in obese subjects, by increasing the fraction of free unbound leptin. More recently, it was found that blood-brain barrier transportation had a threshold level for serum leptin (about 25-30 ng/ml), above which increases in serum levels were not translated into proportional increases in cerebrospinal or brain leptin levels, which means that it may result in apparent leptin resistance and obesity[15]. Some humans with leptin resistance due to a defect in their leptin receptor are severely obese and have high plasma leptin levels[16].

In the present study, only 1.4% of the overweight subjects had LDL-C/HDL-C ratios > 5, whereas 3.0% of a rural population group in northeast Thailand had a ratio > 5, which was risk factor for coronary heart disease.

In the subjects investigated in the present study, the relationship between leptin and BMI was linear, as can be seen in Fig. 1. The findings showed the same results as previously reported in Japanese obese women[17], where a linear relationship between serum leptin and BMI had been found. On the contrary, the relationship between leptin and BMI in Caucasian individuals is not linear in very obese persons because Caucasians have different metabolic states from Asians, and one reason for the differences might be the greater height of Caucasians, so that an increase in the volume of fat tissue to a given height does not correspond in the same way as in Asians. The increase in fat tissue in Asians, with a relatively shorter height, results in a more direct relationship between fat tissue and BMI.

The results showed that soluble leptin receptor concentrations were negatively correlated with BMI. Interestingly, when fat mass decreased, soluble leptin receptor levels increased (Fig. 2). Ogier et al, reported that soluble leptin receptor increase after weight loss positively correlated with loss of fat mass[18]. Consequently, soluble leptin receptor concentrations were significantly negatively correlated with serum leptin, as shown in Fig. 3. These results suggested that a leptin receptor might defect, causing leptin resistance, or high leptin levels can reflect a downregulation of leptin receptor.

An inverse correlation between LDL-C/HDL-C ratio and serum leptin was found in the male and female overweight and obese subjects. A positive correlation was found between HDL-C and serum leptin in the overweight and obese males and females (Table 3). It can be concluded that leptin production occurs mainly in adipocytes and is related to lipid profiles, especially HDL-C.

Further studies will be needed to examine leptin and the leptin receptor genes of obese Thais to determine the causes of obesity.

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ความสัมพันธ์ระหว่างเลปตินรีเซปเตอร์, เลปติน, ปริมาณไขมันในเลือดและสัดส่วนของร่างกายของคนไทยที่มีน้ำหนักเกิน และอ้วน

สุภลัคน์ โพธิ์พฤกษ์, รังสรรค์ ตั้งตรงจิตร, ปราณีต ผ่องแผ้ว, เบ็ญจลักษณ์ ผลรัตน์, อัญชลี ตั้งตรงจิตร, ศิริวรรณ ไตรบัญญัติกุล, ศุภร ปักษานนท์, นิยมศรี วุฒิวัย, Frank P Schelp

คณะผู้วิจัย ได้ทำการศึกษาระดับฮอร์โมนเลปติน, เลปตินรีเซปเตอร์, ปริมาณไขมันในเลือด และภาวะโภชนาการโดยการประเมินสัดส่วนของร่างกาย ของกลุ่มตัวอย่างซึ่งเป็นผู้ที่มีน้ำหนักตัวเกิน (ดัชนีความหนาของร่างกาย, body mass index = 25.00-29.99) และอ้วน (ดัชนีความหนาของร่างกาย ≥ 30.00) ประกอบด้วย อาสาสมัครเพศชาย 48 คนและเพศหญิง 166 คน เปรียบเทียบกับผู้ที่มีน้ำหนักอยู่ในเกณฑ์มาตรฐาน (ดัชนีความหนาของร่างกาย = 18.50-24.99) เพศชาย 26 คน และเพศหญิง 81 คน โดยอาสาสมัครทุกคนเป็นคนไทยที่มีสุขภาพดี อายุระหว่าง 18-60 ปี ซึ่งคัดเลือกจากผู้ที่มาตรวจสุขภาพที่คลินิกผู้ป่วยนอก โรงพยาบาลราชวิถี กรุงเทพฯ จากผลการศึกษาพบว่าระดับฮอร์โมนเลปติน, ไตรกลีเซอไรด์ และ สัดส่วนของโคเลสเตอรอล-ไลโปโปรตีนชนิดความหนาแน่นต่ำ (low density lipoprotein-cholesterol, LDL-C) กับโคเลสเตอรอล-ไลโปโปรตีนชนิดความหนาแน่นสูง (high density lipoprotein-cholesterol, HDL-C) เพิ่มสูงขึ้นอย่างมีนัยสำคัญทางสถิติ แต่ระดับเลปตินรีเซปเตอร์ และ HDL-C ลดลงอย่างมีนัยสำคัญทางสถิติ ซึ่งพบในกลุ่มผู้ที่มีน้ำหนักตัวเกิน และอ้วน นอกจากนี้พบว่าระดับโคเลสเตอรอลและ LDL-C ลดลงอย่างมีนัยสำคัญทางสถิติ ในเพศหญิงที่มีน้ำหนักตัวเกิน และอ้วน นอกจากนี้ พบว่า เพศชาย จำนวน 38.1% (8/21) และเพศหญิง จำนวน 31.5% (29/92) ที่มีน้ำหนักตัวเกิน และอ้วน มีระดับเลปตินรีเซปเตอร์ต่ำ นอกจากนั้น เพศชาย จำนวน 66.7% (32/48) และ เพศหญิง จำนวน 89.8% (149/166) ที่มีน้ำหนักตัวเกิน และอ้วน มีทั้งระดับเลปตินและเลปตินรีเซปเตอร์ต่ำ เมื่อทำการวิเคราะห์หาความสัมพันธ์ของ ระดับเลปตินรีเซปเตอร์กับโคเลสเตอรอล พบว่ามีความสัมพันธ์แบบแปรผันตรงเช่นเดียวกับความสัมพันธ์ของเลปติน กับ น้ำหนัก, เส้นรอบเอว, เส้นรอบสะโพก, ดัชนีความหนาของร่างกาย และ HDL-C แต่ในทางตรงกันข้าม ระดับเลปตินรีเซปเตอร์ กับ เลปติน และดัชนีความหนาของร่างกาย มีความสัมพันธ์แบบผกผัน และจากผลการศึกษานี้ทำให้มีความเข้าใจถึงสาเหตุและผลที่เกิดติดตามมาจากโรคอ้วนได้ชัดเจนขึ้น ซึ่งอาจจะใช้เป็นแนวทางในการดูแลสุขภาพ