

Predicting Plasma Leptin With Anthropometric & Bioelectrical Impedance Analysis Measures Of Adiposity In A Multiethnic Young Adult Population In Malaysia

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Abstract:

Background: Obesity bears multivariate causes and manifestations. Simple anthropometric and non-invasive physical markers are being proposed for easy and early detection of obesity-induced disrupted energy homeostasis and metabolic disorders. Adipose-tissue-derived hormone, leptin is largely known for its role in energy homeostasis and paradoxically, higher serum leptin corresponds to higher degree of obesity. Thus, this study aims to investigate the association of plasma leptin with common physical measures of adiposity in a multiethnic, young adult Malaysian population. **Methodology:** Based on BMI, 185 volunteering participants were grouped as underweight, normal weight, overweight and obese. Standardized anthropometric and bio-electrical impedance assessment (BIA) measure of adiposity was done using Karada Scanner while plasma leptin was measured using a sandwich ELISA assay technique. **Results:** A total of 61, 45, 56 and 23 Malaysian Malays, Chinese, Indian and other minor groups, respectively were included in this study. Out of this, 28 were underweight, 100 were normal weight, 29 were overweight and 27 were obese (n=27). One-way ANOVA revealed a significant difference among all variables except height and waist-hip ratio. The multiple regression analysis predicted plasma leptin value based on age, weight, height, body age, resting metabolic rate, visceral fat, total body fat, and body mass index in both male ($R^2=0.54$) and females ($R^2=0.23$). The results show that weight, resting metabolic rate, visceral fat and body were reliable predictors for plasma leptin between all the groups. **Conclusion:** Anthropometric indices can be used as predictors of serum leptin in adults irrespective of their body weight. This observation thus emphasizes the clinical significance of simple non-invasive physical markers for detection of obesity-induced metabolic disorders.

1. INTRODUCTION

Obesity, a modifiable and preventable disorder has witnessed a rise in prevalence especially in the last decade with an alarming upsurge explicitly seen among children and adolescents (James et al., 2001, Wang and Lim, 2012, Wright and Harwood, 2012, Jaiprakash

et al., 2019). Although some countries have seen this rise plateau, it is still a growing concern, especially among many developing nations (Ogden et al., 2012). Some of the factors associated with the current epidemic of obesity are genetics (Mutch and Clément, 2006, Hastuti et al., 2016), epigenetics (Van Dijk et al., 2015, Herrera et al., 2011), environmental factors, endogenous factors such as stress-induced altered endocrine milieu, increase in cortisol levels etc (Chandola et al., 2006), sleep deprivation (Reiter et al., 2012), "screen time" (Sgoifo et al., 2003) and dietary factors (Jquier and Maffeis et al., 2015). Obesity is a phenotype of numerous pathologies involving neural mechanisms, hormonal mechanisms and dysregulations of increased energy intake, decreased energy expenditure or increased energy storage in the adipose tissue.

Adipose tissue is a metabolic and endocrine organ that is responsible for the storage of fat and secretion of biologically active molecules called adipokines such as leptin, cytokines, chemokines, growth factors and others (Coelho et al., 2013). The primary function of leptin is to regulate appetite, energy balance, fat deposition, glucose homeostasis, lipid metabolism, immunity, insulin resistance, angiogenesis, inflammation process and blood pressure (Jung and Choi, 2014). Factors such as effects of metabolic hormones, gender, developmental age and current body energy requirement and others can influence the plasma leptin concentration. Recent studies have also noted variation in plasma leptin can be innate to specific ethnic groups (Allison et al., 2013, Mente et al., 2010). Identified, as the first obesity-related protein that is ubiquitous and easily measured in vivo, leptin has become the focus of numerous studies (Considine and Caro, 1997, Dodd et al., 2014, Haffner et al., 1996). The finding that the relationship between relative obesity and plasma leptin is consistent across populations and does not seem to be influenced by variations in the environment adds to our growing body of knowledge about this protein (Luke et al., 1998).

Weight-related disorder characterizes 82.5%, 76.4%, and 73.6% of people with Type 2 diabetes, hypertension, and dyslipidemia, respectively. Increased lipid accumulation in the cardiovascular, hepatic and musculoskeletal system along with activation of protein kinase C pathway may contribute to insulin resistance (Yung and Giacca, 2020). Physical compression in the kidney due to accumulating fat through renin-angiotensin-aldosterone-system is known to contribute towards obesity-induced hypertension (Park et al., 2004, Ramachandran et al., 1997, Shintani et al., 2002). The early identification of these metabolic disorders can allow us to intervene at an early stage and prevent these preventable consequences.

In this context, anthropometric indices, which are non-invasive, low cost, standardized technique-dependent and easy in measurement, offers the most convenient and reliable alternative in screening a large population (Gluszek et al., 2015). Most well-studied indices include waist circumference, hip circumference, waist-hip ratio (Johnstone et al., 2006). However, reliability has slowly shifted towards the use of body mass index. Studies have shown the use of skinfold with satisfying results but since the introduction of bioelectrical impedance (BIA) technique has opened the Pandora's box in providing information on the total, regional and visceral fat distribution, body age, total and regional muscle mass etc (Malavolti et al., 2003). Despite all these measures, there still exist challenges in identifying which among the array of tools are best for early identification of this non-communicable disorder.

Thus, in our study, we addressed the question whether plasma leptin concentration is related to adiposity as measured by BIA in a multiethnic Malaysian young adult population and if these BIA measures can be used to predict the changes in plasma leptin so that appropriate clinical intervention can be introduced early as a treatment. To our knowledge, no previous study has examined the association of plasma leptin with adiposity assessed by a measure other than skinfold thickness among different ethnic subgroups in Malaysian young adult population. Our study aimed to measure plasma leptin levels in healthy young

Malaysian adults and investigate its association with age, height, weight, hip & waist circumference, waist-hip ratio, visceral & total body fat and BMI.

2. METHODOLOGY

The university ethical and research management committee reviewed the protocol and approved this study (Ethical Approval No: RMC/E87/2019 dated 20th August 2019 from MAHSA University and JKEtika 4/20(12) dated 15th September, 2020 from UMS). All procedures were followed in accordance with the university directives on human experimentation and the Helsinki Declaration of 1975 and subsequent revisions. Informed written consent was obtained from all the participants of this study after they were briefed on the aim, objectives, outcome and benefits of this study.

This cross-sectional study was conducted between August to November, 2019 at MAHSA University, Malaysia. Using stratified-random sampling method 185 volunteering participants were recruited in this study at a specific point of time. Malaysian young adults between the age group of 18-25 years (Kee et al., 2008) with no pre-existing medical illness or on any treatment with no habits or addiction were included in this study. All the invited participants voluntarily participated in this study.

Anthropometric Measurements

The height and weight of each subject were measured using a research-grade stadiometer and health scale. We used Omron Karada Scanner (Omron Healthcare, Japan) was used to measure body weight, BMI, total body fat (%), resting metabolic rate (in kcal), visceral fat (%). The Full Body Sensor Body Composition Monitor and Scale works on bioelectrical impedance method to send feeble electric current of 5 kHz and less than 5 μ A through the body to determine the amount of water in each tissue (Healthcare, 2008). The subject will not notice or feel the electrical current. The bioelectrical impedance technique used to assess body composition with the Karada scan has been shown to match well with other techniques such as dual-energy X-ray absorptiometry and also computed tomography. Body age, a biological age calculation, was estimated on the basis of body mass, body fat, and resting metabolism, based on health and fitness level.

Clear and full instructions were given to each subject on how to stand on the Karada scan sensor platform in an upright position, barefooted, and holding the fat analyzer with extended arms. A measuring tape was used while standing to measure each subject's waist circumference (WC) and hip circumference (HC) to the nearest 0.1 cm. At the midpoint between the lower costal boundary (tenth rib) and the iliac crest, WC was assessed at the end of normal expiration. In comparison, at the full circumference around the buttocks, HC was measured. Then, the waist-to-hip ratio (WHR) was determined. The participants were grouped into four categories based on the BMI classification of the World Health Organization (WHO): lean or underweight group, BMI <18 kg/m²; normal weight group, BMI 18-24.9 kg/m²; overweight group, BMI 25-29.9 kg/m²; and obese group, BMI \geq 30 kg/m². The measurements were carried out by two laboratory technicians who were trained to use these devices. The overall process was supervised by the authors.

Sample Collection

The participants were briefed about the process of blood collection and at 8:00 am observing strict aseptic precaution 3ml of blood was collected in EDTA vacutainers by certified phlebotomists. Within 30mins of collection, the samples were centrifuged at 3000rpm, and plasma was collected in aliquots to be stored in -80^oC.

Estimation of Serum Leptin

All the samples were estimated in under two weeks after sample collection. The samples were thawed overtime to bring plasma to room temperature before analysis. Leptin was

analyzed using the sandwich ELISA method using research-grade kits from R&D Systems, USA (Catalogue No: DY1707). Each sample was measured in duplicates against the standard curve along with positive and negative controls.

Statistical Analysis

The data was compiled and analyzed using IBM SPSS version 26.0. We used a one-way ANOVA test using Tukey as post-hoc test to measure the difference in the mean between groups for all parameters. For each group, multiple linear regression analysis was used to investigate the impact of age, height, weight, total fat, total subcutaneous fat, visceral fat and body age as independents variables. Multiple regression analysis was performed to assess the predictive value of anthropometric measures, including WC, HC, WHR on plasma leptin concentration within each group. During this test, plasma leptin was used as the dependent variable. An alpha of $p < 0.05$ was considered significant.

3. RESULTS

This cross-sectional study recruited 185 healthy Malaysian young adult participants, out of which 55 were male, and 130 were female. Out the 185 participants, 61 were Malaysian Malays, 45 were Malaysian Chinese, 56 were Malaysian Indian, and 23 were Malaysians from other minor ethnic groups. These participants were group based on their BMI as underweight (n= 29), normal weight (n=100), Overweight (n=29), Grade 1 Obese (n=19), Grade 2 Obesity (n=3), Grade 3 Obesity (n=5). All of the measured parameters were found to be significantly different among groups, except age, height & waist-hip ratio (*Table 1*).

Table 1 a: Male

Parameters	Underweight (n=10)	Normal (n=28)	Overweight (n=4)	Obese (n=13)	p-value
Age	19.8 ± 1.7	20.6 ± 1.8	20.5 ± 1.2	20.6 ± 1.2	0.615
Height (cms)	167.2 ± 7.6	170.0 ± 7.5	175.7 ± 2.8	169.8 ± 8.8	0.328
Wt (Kg)	49.1 ± 5.5	64.1 ± 8.0	79.2 ± 5.4	101.1 ± 28.5	0.000
Hip Circumference	87.4 ± 13.9	91.5 ± 11.5	102.7 ± 12.8	111.2 ± 15.1	0.000
Waist Circumference	69.1 ± 10.6	78.9 ± 7.3	86.1 ± 6.9	92.5 ± 15.8	0.000
Waist-Hip Ration	0.81 ± 0.04	0.84 ± 0.08	0.83 ± 0.05	0.85 ± 0.07	0.567
Body Age	16.9 ± 0.0	27.3 ± 6.4	37.4 ± 2.1	41.0 ± 11.2	0.000
Resting Metabolic Rate	1258.1 ± 227.8	1519.4 ± 149.2	1759.2 ± 83.1	1792.9 ± 179.4	0.000
Visceral Fat	-.22 ± 2.6	6.6 ± 2.5	10.4 ± .94	13.5 ± 2.9	0.000
Total Body Fat	18.2 ± 6.7	20.9 ± 5.9	22.6 ± 2.0	33.6 ± 6.3	0.000
Body Mass Index	15.1 ± 1.8	23.1 ± 2.7	27.9 ± .8	33.3 ± 2.7	0.000
Plasma Leptin	2345.6 ± 548.0	2482.7 ± 446.9	2486.0 ± 594.5	3274.1 ± 798.8	0.001

Data are expressed as mean \pm SD, * $p < 0.05$ significant difference among the groups using a One-Way ANOVA

Parameters	Underweight (n=20)	Normal (n=73)	Overweight (n=23)	Obesity (n=14)	p-value
Age	20.5 \pm .94	20.3 \pm 1.2	21.0 \pm 2.5	21.5 \pm 2.8	0.074
Height (cms)	156.9 \pm 7.5	159.2 \pm 7.2	164.7 \pm 22.8	160.7 \pm 5.4	0.134
Wt (Kg)	42.3 \pm 5.8	54.7 \pm 6.9	71.2 \pm 9.6	88.0 \pm 12.6	0.000
Hip Circumference	85.4 \pm 11.8	91.3 \pm 10.4	99.8 \pm 12.5	113.0 \pm 10.9	0.000
Waist Circumference	59.9 \pm 10.9	72.4 \pm 9.9	81.8 \pm 8.7	92.4 \pm 8.3	0.000
Waist-Hip Ration	0.76 \pm 0.05	0.80 \pm 0.08	0.81 \pm 0.07	0.80 \pm 0.07	0.150
Body Age	17.6 \pm 2.7	27.9 \pm 6.1	39.5 \pm 4.1	47.3 \pm 4.3	0.000
Resting Metabolic Rate	957.2 \pm 184.5	1236.9 \pm 152.1	1469.8 \pm 138.6	1628.7 \pm 129.3	0.000
Visceral Fat	-1.4 \pm 1.4	3.7 \pm 2.7	9.4 \pm 1.9	13.8 \pm 2.1	0.000
Total Body Fat	25.2 \pm 5.3	27.9 \pm 4.7	35.8 \pm 5.3	40.6 \pm 3.4	0.000
Body Mass Index	14.2 \pm 2.7	22.4 \pm 2.6	28.8 \pm .88	33.4 \pm 2.1	0.000
Plasma Leptin	2890.2 \pm 382.7	3135.4 \pm 735.3	3277.5 \pm 650.1	3533.6 \pm 635.5	0.042

Data are expressed as mean \pm SD, * $p < 0.05$ significant difference among the groups using a One-Way ANOVA

We compared the mean values of anthropometric measure and plasma leptin with underweight, normal weight, overweight and obese groups based on ethnicity and gender (Table 2). The results indicate that weight, HC, WC, Body Age, resting metabolic rate, visceral fat, total body fat, & BMI were significantly different between the subgroups. At the same time, plasma leptin showed a significant difference only in Malay female, Indian male and Female of other minor ethnic population.

Parameters	Malay Female (48)	Malay Male (n=13)	Chinese Female (26)	Chinese Male (19)	Indian Female (33)	Indian Male (23)	Other Female (n=23)
Age	0.043	0.121	0.683	0.126	0.383	0.919	0.598
Height (cms)	0.889	0.406	0.000	0.461	0.276	0.392	0.638
Weight (Kg)	0.000	0.007	0.000	0.000	0.000	0.002	0.000

Hip Circumference	0.000	0.045	0.115	0.137	0.000	0.026	0.298
Waist Circumference	0.000	0.001	0.049	0.047	0.000	0.134	0.013
Waist-Hip Ratio	0.017	0.682	0.708	0.877	0.260	0.974	0.288
Body Age	0.000	0.007	0.000	0.000	0.000	0.005	0.000
Resting Metabolic Rate	0.000	0.07	0.000	0.003	0.000	0.000	0.003
Visceral Fat	0.000	0.037	0.000	0.000	0.000	0.000	0.000
Total Body Fat	0.000	0.153	0.018	0.006	0.001	0.002	0.002
Body Mass Index	0.000	0.002	0.000	0.000	0.000	0.000	0.000
Plasma Leptin	0.046	0.163	0.441	0.400	0.222	0.012	0.044
<i>*Significant association p<0.05</i>							

The multiple regression analysis (Table 3) was done to predict plasma leptin value based on age, weight, height, Body Age, resting metabolic rate, visceral fat, total body fat, & body mass index in both male and females. In both male and female groups, there was an overall linear association between the standardized residual and standardized predicted value, since the points were uniformly distributed in the scattered for both the groups indicating homoscedasticity and the value Darbin-Watson for male and Female was 1.05 (normal 1.50 to 2.50) indicating that the errors in the model are independent and there were no outliers. With the above tests, we ensure that all the assumption for performing multiple linear regression were fulfilled.

Table 3: Model summary of multiple linear regression analysis using plasma leptin as dependent variable and age, weight, height, Hip & Waist Circumference, waist-hip ratio, Body Age, resting metabolic rate, visceral fat, total body fat, & body mass index as independent variables

Group	R	R squared	Adjusted R square	F	p-value
Male	0.738	0.544	0.405	3.909	0.001*
Females	0.487	0.237	0.154	2.849	0.003*

**Significant association p<0.05, Dependent Variable: Plasma Leptin. Predictors: (Constant), Body Mass Index, Waist-Hip Ration, Height, Age, Waist Circumference, Total Body Fat, Body Age, Resting Metabolic Rate, Visceral Fat, Weight, Hip Circumference*

A significant equation was found (F(11,36), 3.909, $p < 0.005$), with an R^2 of 0.544 for males and (F(11,101), 2.849, $p < 0.005$), with an R^2 of 0.237 for females.

The adjusted R square in males & females, indicating 40.5% & 15.4% consecutively, accounted for the variance observed in the plasma leptin values by the predictors, it also indicating goodness of fit. Since the p-value for both the groups is less than 0.05, it suggests that the independent variables provide a satisfactory explanation for the changes in plasma leptin levels.

The coefficient table (Table 4a & 4b) shows that the t-test for the variable for hip circumference, body age was statistically significant indicating the ability to generalize the result to the whole of this population subset.

Table 4a: Multiple linear regression analysis of plasma leptin concentration as a dependent variable with age, weight, height, Hip & Waist Circumference, waist-hip ratio, Body Age, resting metabolic rate, visceral fat, total body fat, & body mass index as independent variables among Malaysian young adult male population.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
(Constant)	4737.931	2539.121		1.866	0.070
Age	56.784	53.375	0.129	1.064	0.294
Height (cm)	-10.427	17.604	-0.141	-0.592	0.557
Weight (Kg)	74.282	17.306	1.970	4.292	0.000*
Body Age	-4.778	19.132	-0.082	-.250	0.804
Resting Metabolic Rate	-2.760	1.159	-1.110	-2.381	0.022*
Visceral Fat	96.098	58.263	0.781	1.649	0.107
Total Body Fat	-21.094	17.185	-0.275	-1.227	0.227
Body Mass Index	-99.005	43.719	-0.997	-2.265	0.029*

*Significant association $p < 0.05$, Dependent Variable: Plasma Leptin

From Table 2 it can be predicted that participant's plasma leptin is equal to $4737.9 + 74.28(\text{Weight}) - 4.78(\text{RMR}) - 99.0(\text{BMI})$ in males and $2083.09 + 48.16(\text{Body Age}) + 67.53(\text{BMI})$ in females, where weight was measured as Kg and resting metabolic rate (RMR) was measured as kCal.

From Table 3a it is estimated that male participant's plasma leptin increased 74.28 pg/mL for each Kg increase in weight and decreased 4.78 pg/mL for every kCal increase in RMR and decreased 99 pg/mL for every unit increase in BMI. From table 3b, it is estimated that female participant's plasma leptin increased by 48.16 pg/mL for every unit increase in body age and increased 67.53 pg/mL for every unit increased in BMI.

Table 4b: Multiple linear regression analysis of plasma leptin concentration as the dependent variable with age, weight, height, Hip & Waist Circumference, waist-hip ratio, Body Age, resting metabolic rate, visceral fat, total body fat, & body mass index as independent variables among Malaysian young adult female population.

Model	Unstandardized Coefficients		Standardized Coefficients	t	Sig.
	B	Std. Error	Beta		
(Constant)	2083.086	1564.629		1.331	0.186
Age	21.174	46.917	0.042	0.451	0.653
Height (cms)	-1.892	6.333	-0.033	-0.299	0.766
Wt (Kg)	-3.155	15.690	-0.067	-0.201	0.841
Body Age	48.165	23.177	0.688	2.078	0.040*
Resting Metabolic Rate	-.668	1.005	-0.233	-0.665	0.507
Visceral Fat	-82.887	48.081	-0.578	-1.724	0.088
Total Body Fat	-22.235	21.521	-0.224	-1.033	0.304
Body Mass Index	67.534	35.074	0.564	1.925	0.047*

*Significant association $p < 0.05$, Dependent Variable: Plasma Leptin

Discussion

The present study demonstrated that plasma leptin concentration was significantly associated with all the anthropometric measures that were included in this study except height and waist-hip ratio in the Malaysian young adult population. We observed that most of the anthropometric measurements were significantly different among underweight, normal weight, overweight & obese population among the three ethnic groups. We further report that among the three ethnic groups, plasma leptin was significantly different in lean, overweight & obese individuals in only Malaysian Malay women, Malaysian Indian men and women of other minor ethnic population. We also report that these anthropometric measures could statistically be accounted for the changes observed in plasma leptin in both men and women in all the ethnic subgroups.

Since it is a fact that plasma leptin is significantly higher in women than in men, we presented the data according to gender. Studies have shown women to have a higher concentration of subcutaneous fat than men, while men had a higher proportion of visceral fat than women. Thus, when associated with the fact that the subcutaneous fat predominantly produces leptin, it explains the variation of plasma leptin observed between men and women (Rosenbaum et al., 1996, Hellström et al., 2000, Flanagan et al., 2007). In agreement with these studies, we too observed that plasma leptin was significantly higher among women when compared to men. The role of ethnicity in association with plasma leptin is well documented. A study conducted in by Mente *et al.* (2010) showed that South Asians had an unfavorable adipokines profile, which is characterized by lower adiponectin and higher leptin for the same degree of adiposity observed in Chinese, Aboriginal, and European Canadians ethnic subpopulation (Mente et al., 2010). Earlier studies conducted in Malaysian community indicates that genetically leptin was more strongly associated among Indian men when compared to Malay population (Fan and Say, 2014, Ng et al., 2014). In another study conducted on school children in Brunei, they observed no significant increase in leptin among the overweight population (Idris et al., 2016). In our study majority of Malaysian Malays were Bumiputra, Malaysian Indians comprised of Tamils, Punjabis and Telugus with ancestries tracing back to the Indian subcontinent while Malaysian Chinese subjects in this study include mostly of Han Chinese descent. We observed that only Malay women, Indian men and women of other minor ethnic population showed significant changes in plasma leptin when compared between lean, overweight & obese subject. These could be due to genetic factors observed by the polymorphic modifications of leptin and leptin receptor gene (Gopalakrishnan et al., 2012, Fan and Say, 2014, Ng et al., 2014) which in turn could be affected by external factors such as physical, psychological & emotional stress, lifestyle choices, sleep etc. (Lovibond and Lovibond, 1995, Lin et al., 2016, Hishan et al., 2018, Mohanraj, 2014).

The relation of leptin concentration with anthropometric measures of adiposity was examined earlier in many population studies. A study among the Saudi population showed that leptin significantly associated to the waist and hip circumference and they concluded that leptin concentrations are associated with overall adiposity; however, factors such as insulin level and activities of subclinical inflammation can alter this association (Al-Daghri et al., 2007, Cernea et al., 2020). In contrast, other researchers found an association of leptin concentration with waist circumference, independent of BMI or percentage body fat. They concluded that body fat distribution might also be an essential determinant of leptin concentration (De Silva et al., 1998, Monti et al., 2006, Shah and Braverman, 2012, Hijjawi et al., 2018). In another study, leptin concentrations were unrelated to waist circumferences after adjustment for fat mass but were associated with hip circumference in women (Bennett et al., 1997, Eldosouky et al., 2018). In our study anthropometric measures such as weight, waist circumference & hip-circumference and Bioelectrical Impedance Analysis measures

such as body age, resting metabolic rate, visceral fat, total body fat, and body mass index along with plasma leptin was significantly different among underweight, normal weight, overweight and obese individuals. Rather than indicating a role for specific fat depots in determining leptin concentration, these results may indicate the limitation of using BMI as a standalone measure of total fat mass.

Although anthropometric measures are less accurate than the measure of total fat mass and specific fat depots by methods such as computed tomography or dual-energy X-ray absorptiometry, we had no information from such an approach. However, our multiple linear regression model which used weight, resting metabolic rate (bioelectrical impedance method) and BMI, explained the degree of variance in leptin concentration in men and body age, resting metabolic rate (bioelectrical impedance method) & BMI in women. These were equivalent to those explained by models containing total fat mass and percentage body fat measured by computed tomography, dual-energy X-ray absorptiometry or bioelectrical impedance analysis (Ramachandran et al., 1997, Luke et al., 1998, Malavolti et al., 2003, Doña et al., 2018).

In a study conducted by Byrnes *et al.* reported an inverse relationship between plasma leptin and weight gain among Australian adolescence (Byrnes et al., 1999); likewise, Ahmed *et al.* reported that plasma leptin could predict the increase of percentage body fat more accurately in girls when compared to boys (Dietz, 1988). In our study, we observed that among young adult male participants plasma leptin increased 74.28 pg/mL for each Kg increase in weight and decreased 4.78 pg/mL for every kCal increase in resting metabolic rate and decreased 99 pg/mL for every unit increase in BMI. Among the young adult female participant's plasma leptin increased by 48.16 pg/mL for every unit increase in body age and increased 67.53 pg/mL for every unit increased in BMI. Various studies have examined the importance of gender for overweight and obesity in childhood (Sweeting, 2008, Skelton et al., 2009), some reported no gender-difference (Tremblay, 1996, Baskin et al., 2005), some reported higher overweight prevalence among boys (Watkins et al., 2005). In contrast, others reported higher overweight rates among girls (Salazar-Martinez et al., 2006, Bénéfice et al., 2001). We also observed that these anthropometric values were efficiently predicted plasma leptin among men ($R^2=0.54$) when compared to women ($R^2=0.23$). Some recent studies showed the presence gender differentials concerning the behavioral determinants of overweight including calorie intake (Al Sabbah et al., 2007) and physical and sedentary activities (Irving et al., 2003, Kirchengast and Marosi, 2008).

Limitations in this study may be the sample size, and disproportion in the state-wise representation of population, which could have a risk of sampling bias. With regard to the moderate size of the screened population, future studies should involve larger sample size of Malaysians from all the states with more balanced age groups, ethnicities, and genders to better reflect the entire Malaysian population.

Conclusion

We observed that Malay women, Malaysian Indian men and women from other minor ethnic groups showed a strong association between plasma leptin and measures of adiposity. We confirm that plasma leptin is significantly higher among women when compared to men. Our study showed that anthropometric measurements of obesity such as weight, resting metabolic rate and BMI were good predictors of changes in plasma leptin among both young adult men and women population. While factors such as height, waist-hip ratio, total body fat and visceral fat although significantly associated with plasma leptin were not good predictors. Finally, this study infers that BMI on its own does not reflect the changes in plasma leptin, however, in association with anthropometric measures such as weight, resting metabolic rate, visceral fat & body age it can better account for differences observed in plasma leptin. Early

and accurate detection of obesity can thus provide us an opportunity to effectively manage the progression of this preventable disorder.

Conflicts of Interest

The author declares that there is no conflict of interests regarding the publication of this paper.

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