Parental Obesity and Serum Leptin Levels among North Indian Obese Adults

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Abstracts: **Background And Objectives:** Obesity is increasing worldwide as an epidemic. Recent advances in biology of adipose tissue have revealed that adipose tissue in addition to its role as an energy reservoir, modulates energy metabolism via secretion of circulating adipocytokines. Leptin is one such adipocytokine which is essential for body weight homeostasis. There exists complex interaction of genetic and environmental factors in obesity. Various studies have shown genetic influence of parental fatness in childhood obesity but the effect of parental obesity in adult obesity as well as leptin level is not clear, therefore present study was aimed to determine whether parental obesity might contribute to adult obesity as well as serum leptin levels in obese adults. **Material and Methods:** Study consisted of forty five obese adults with Body Mass Index≥27 and control group included forty five lean adults with Body Mass Index≤22. Information regarding parental obesity was obtained from each participant in a prestructured questionnaire. Blood samples were collected from both groups and serum leptin levels were measured by Radioimmunoassay. **Results:** About 62 percent of case group was found to have parental history of obesity. In contrast 29 percent subjects in control group had obese parents. Moreover, the mean of serum leptin levels in obese adults with history of obese parents was significantly higher than obese adults without the history of obese parents (males:19.26±2.69 vs 15.75±2.19,p=0.04 and females: 37.0±7.55 vs26.86±3.72,p =0.02). **Conclusion:** Parental obesity plays an important role in obesity and serum leptin level during adulthood. [Khatri A NJIRM 2014; 5(5):1-4]

Key Words: Body Mass Index, Leptin, Parental obesity

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Introduction: Obesity is considered as one of the most pressing problem in industrialized world. Prevalence of obesity is increasing at an epidemic rate globally with more than 1 billion adults overweight and at least 300 million of them clinically obese [1].This is expected to rise further in next twenty to thirty years. In India obesity has reached epidemic proportion too. According to National Family Health Survey (NFHS) report 5% of Indian population has been affected by obesity[2]. Obesity though not a lethal disease by itself, but increases the morbidity and mortality by increasing the propensity of various diseases like diabetes mellitus, hypertension, and dyslipidemia disorders. Hence, there is an urgent need to address the trouble and efforts should be made to prevent the epidemic of obesity and its allied health disorders.

Adipose tissue was once considered to be an inert depot for storing fuel as lipids, now adipose tissue is known to operate as an endocrinologically active tissue that releases adipocytokines[3]. Leptin is one such adipocytokine which is essential for body weight homeostasis[4]. Leptin, the obese (ob) gene product, has been shown to be expressed in adipocytes in humans and animals [5]. It has been proposed to act as a satiety factor between adipose tissue and the central nervous system[6,7]. Both human and animal studies have indicated a strong positive relationship between the amount of adipose tissue and serum leptin levels [8].The increased serum leptin concentration in obesity was proposed to be secondary to leptin resistance[9].

Obesity is a complex multifactorial disease which is influenced by a wide-range of genetic and non genetic factors, with interactions between many of these. Previous studies have shown that heavy newborns tend to become fat children [10,11] and that fat children tend to become fat adults [12]. However, in all these studies the influence of parental body composition on the development of adult obesity was not evaluated. Hence the present study sought to determine whether parental obesity might contribute to adult obesity as well as serum leptin levels in obese adults.

Material and Methods: Study was conducted in physiology and medicine department of King George's Medical College, Lucknow. Ninety age and gender matched lean and obese subjects were

recruited for the study. Forty five (23 Males and 22 Females) lean subjects with body mass index (BMI) ≤22 were included in controls and forty five (23 Males and 22 Females) obese subjects with Body Mass Index ≥27 were included in case group. Participants in the study were selected from healthy volunteers and subjects who have recently enrolled themselves in various fitness centres have recently enrolled themselves in various fitness centres, but not yet have started the exercise regime. Informed consent was taken from all participants and study protocol was approved by institutional ethics committee.

After detailed medical history thorough Physical examination was carried out and routine haematological test, chest X Ray and ECG were done in order to rule out any disease. Subjects having diabetes, hypertension, any other chronic illness, smokers, alcoholics, pregnant, lactating and postmenopausal females and subjects who were engaged in heavy physical activity likewise sports persons and manual labourers were excluded from study. BMI of participants was calculated as: BMI=weight (kg)/height (m^2).

Assays: Blood samples were collected from all the subjects in the morning after overnight fasting. Sera that were separated, immediately after centrifugation were stored at -20^{0}C until the assays for leptin were performed. Plasma leptin concentrations were determined with a commercially available double-antibody radioimmunoassay (RIA; Human Leptin Specific MO Kit, LINCO Research, St Charles, USA) which detects leptin levels down to 0.5 ng/ml and which does not cross react with human insulin, proinsulin, glucagon, pancreatic polypeptide or somatostatin. The intra-assay coefficient of variation was 3.9% and the interassay coefficient of variation was 4.7% for a mean level of 10.4ng/ml.

Statistical Analysis: Data are expressed as mean±S.D. Statistical analysis was performed using student t-test. Correlation of leptin with Body Mass Index was evaluated via Pearson's correlation analyses. The Chi-square test was also performed and for all statistical assessments a value of $p<0.05$ was accepted to be statistically significant.

Results: Table 1 shows that, age did not differ significantly between the 2 study groups. However, BMI and leptin were significantly higher in the case group than the control group. Significant correlation of leptin with BMI was found in our study(r=0.81,p=.0001)**.**

Table 1: Description of Study Population

	Cases N=45	Controls N=45	P value
Age(years)	30.84±5.29	30.38±5.29	0.69
BMI(Kg/m^2)	30.69±2.62	20.08±1.79	0.001*
Leptin(ng/ml)	25.90±10.45	6.43±2.58	0.001*

*Statistically significant

Results of Table2 and Table 3 show that the mean of serum leptin hormone concentrations of obese adults with parental obesity was more significant than obese adults without parental obesity in males (0.04) and females (P = 0.02).

Table 2: Relationship between Parental Obesity and Serum Leptin Levels among Obese Male Adults of the Case Group

	Males		
Parental obesity	Present	Absent	Pvalue
Leptin(ng/ml)	19.26±2.69	15.75±2.19	0.04*

*Statistically significant

Table 3: Relationship between Parental Obesity and Serum Leptin Levels among Obese Female Adults of the Case Group

	Females		
Parental obesity	Present	Absent	Pvalue
Leptin(ng/ml)	37.0±7.55	26.86±3.72	0.02*

*Statistically significant

Table 4 shows that about 62% of the case individuals had parental obesity and about 38% had ideal body weight parents. In contrast, for the control individuals, about 29% had parental obesity and about 71% had normal ideal weight parents**.** This frequency distribution was statistically significant (p=0.01)

Table 4: Frequency Distributions of the Study Individuals by Parental History of Obesity

Parental body weight	*Cases		*Controls	
	Frequency	Valid %	Frequency	Valid %
Obese	28	62.22	13	28.88
Ideal	17	37.77	32	71.11
Total	45	100	45	100

*The frequency distribution among the case and control group is statistically significant (p=.01)

Discussion: Obesity is increasing worldwide as an epidemic. Various studies have shown the effect of parental composition of fat on childhood obesity but there are paucity of studies especially in india, showing the influence of parental fat in adult obesity and serum leptin level. Hence present study was planned to evaluate whether parental obesity might contribute to adult obesity and serum leptin levels among obese adults.

As previous studies have reported, the present study indicates that leptin levels were higher in obese subjects than in normal-weight individuals [13,14]. Hyperleptinemia in obesity suggests a state of leptin resistance which may be due to a diminished response in the leptin receptor signalling pathway [9,15] or poor penetration of the blood-brain barrier by leptin[16].The influence of different factors on the level of circulating leptin has been investigated in many studies during the past years. Almost all studies report a high correlation between BMI and leptin levels [8], [17]. Our study confirmed the results of other investigators. The BMI showed a high correlation with leptin levels.

Significant finding of our study was about influence of the parental history of obesity on serum leptin concentrations. It was found that adults with obese parents were more likely to have obesity and high leptin level than adults of non-obese parents. This means that fat cells generated in one or both parents due to genetic factors and family feeding styles contribute to transmission of obesity risk and high leptin level during adulthoods[18].In a study by Tarquini et al[19] it was reported that cord blood leptin concentration is elevated in the presence of the family history of obesity on paternal side.

The finding that parental obesity affects the development of obesity is also in agreement with other studies in the literature. Longitudinal studies in infants[20,21] have shown that parental body composition is an important predisposing factor in the development of obesity in children. In a retrospective study, by Whitaker et al the probability of obesity in young adulthood was examined in relation to the presence or absence of obesity at various times throughout childhood and the presence or absence of obesity in the child's parents [22]. That study showed that obese children without obese parents were at low risk of obesity in adulthood. The risk of adult obesity was significantly greater if either parent was obese. Finally Frisancho AR [23] concluded that the tracking of high birth weight to adolescence depends on the body composition of the parents such that heavy newborns become fat adolescents only if either the mother or father is also fat.

Conclusion: Results of our study indicated that in the presence of parental obesity, risk of adult obesity is increased and these obese adults had higher serum leptin levels when compared in obese adults without family history of obesity. The present study gives us an insight into the relationship between adult and parental obesity at the molecular level. The examination of leptin genes of obese individuals and their parents at molecular level should be conducted so as to understand the nature of cause of obesity at the molecular level which in future will provide us fruitful information regarding primordial and primary prevention of obesity.

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Conflict of interest: None
Funding: None