Compares Hemodynamic Variables, Food Habits, And Obesity Grades Between Obese and Non-Obese Males

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ABSTRACT
Most nations' obesity rates have been on the rise since the 1980s, confirming the existence of a worldwide pandemic. Hypertension, dyslipidemia, type 2 diabetes, and sleep problems are incident cardiovascular risk factors that obesity directly contributes to. Even after controlling for other risk factors, obesity increases the likelihood of cardiovascular disease and its death. Waist circumference, rather than body mass index, is a more modern indicator of abdominal obesity as a risk factor for cardiovascular disease. Imaging techniques that assess visceral adiposity and other aspects of body composition have also come a long way. Research looking at the quantification of fat depots (including ectopic fat) lends credence to the idea that increased visceral adiposity is a standalone predictor of worse cardiovascular outcomes. Metabolic syndrome, along with its associated systemic inflammation and endothelial dysfunction, may be ameliorated via changes in lifestyle and, subsequently, weight reduction. On the other hand, medicinal weight loss has not been shown in clinical studies to lower risks of coronary artery disease.

KEYWORDS: Healthcare students, obesity, overweight, prevalence

INTRODUCTION

Obese individuals who more commonly show features of the MS exhibit a clustering of phenotypes associated with increased cardiovascular risk. Insulin resistance is suggested to be central of this syndrome's pathogenesis and explains the association between obesity and vascular dysfunction. However, recent additions to this clustering, such as an elevation of levels of plasminogen-activator inhibitor, microalbuminuria and endothelial dysfunction cannot be clearly explained by insulin-mediated mechanisms. Moreover, obesity is associated with features of acute-phase activation and low-grade inflammation is recognized as a component of atherosclerosis. C-reactive protein (CRP), IL-6, fibrinogen and TNF-α are associated with adiposity in children. Markers of systemic low-grade inflammation worsen with increasing adiposity. The molecular mechanism of CRP related to insulin resistance has been examined with the purpose that CRP induces the phosphorylation of jun N-terminal kinase (JNK) and Insulin receptor substrate-1 (IRS-1) Ser307 site through a spleen tyrosine kinase (Syk) and RhoA-activation signaling pathway. An increased level of is prostates, as a measure of oxidative stress, was seen in association with greater adiposity in adolescents. All these factors could affect vascular function by their local and distant actions.

Many interactions between metabolism and vascular tissues contribute to major relationships between insulin resistance and endothelial function. Adipose tissue has become increasingly important in understanding the role of obesity in vascular disease. It produces several biologically-active cytokine-like molecules that could mediate the increased risk of CVD associated with obesity. The two most promising of such molecules for an effect on vascular function are leptin and adiponectin. The action of leptin could be mediated *via* receptors widely distributed on endothelial cells, to stimulate smooth muscle cell proliferation and migration, and hence impair arterial elasticity. This suggests that a high leptin concentration is a key link between obesity and vascular disease.

Adiponectin, which acts as a regulator of MS, might be cardio-protective by inhibiting TNF- α-mediated monocyte adhesion, formation of foam cells and smooth muscle cell proliferation and also by promoting blood vessel growth and endothelial NO production. Endothelial function is impaired in adiponectin knock-out mice, whereas forced adiponectin expression reduces atherosclerotic lesions in a mouse model of atherosclerosis. Hypo adiponectin has been associated with impaired endothelial function in patients with mild hypertension and type 2 diabetes and in healthy adult controls.

There are relatively few data for human beings, especially for young individuals, that support an independent anti-atherogenic action of adiponectin. Adiponectin contributes to the maintenance of insulin sensitivity in young, nonobese individuals but does not affect the development of early endothelial dysfunction. Leptin, adiponectin, free fatty acids (FFA), and ghrelin are emerging biomarkers of insulin resistance; the three latest have also been implicated as biomarkers of coronary artery disease. Many prospective studies have shown that individuals with MS have a greater risk of developing type 2 diabetes and cardiovascular disease. Assessment of CVD risk and MS in children and adolescents has involved the analysis of serum or plasma biomarkers including total cholesterol (TC), triglycerides (TG), HDL cholesterol (HDL-C), insulin and C-peptide. Lipids and lipoproteins play an important role in the development as well as in consequences of MS. The dyslipidemia results in increased production of TG and secretion of

very low-density lipoprotein along with associated abnormalities like reduction of in HDL-C and increased density of LDL.

Hypertriglyceridemia is associated with predominance of small dense LDL particles which is due to relative depletion of unesterified cholesterol, esterified cholesterol and phospholipids with either no change or an increase in LDL triglyceride. Small dense LDL is more toxic to endothelium, can transit through endothelial basement membrane easily, adheres well to glycosaminoglycans, is more susceptible to oxidation Andis more selectively bound to scavenger receptors on monocytes derived macrophages. Recently, emerging biomarkers such as apolipoprotein (apo)-AI and apo-B have been proposed as precise predictors of atherogenicity and CVD risk.

Obesity might also promote preclinical atherosclerotic changes *via* a direct effect on vascular physiology. Greater adiposity in childhood or adolescence has been associated with greater cardiovascular and all-cause mortality in adult life. Several reports now suggest that obesity impairs vascular function. Obese individuals show an impaired endothelial-mediated vasodilator response to increase blood flow and to insulin.

Similarly, obesity is associated with greater arterial stiffness and visceral adiposity is particularly detrimental. The precise mechanism by which obesity in childhood increases CVR above that of fatness in adult life remains unexplained. One possibility is that of a prolonged exposure of arteries to the metabolic milieu associated with obesity (such as a high insulin concentration). Endothelial dysfunction and arterial stiffness in young individuals who are obese is now strong evidence from an early age. The underlying mechanism for this effect is contentious. Childhood obesity exerts its effects on coronary heart disease by way of its persistence into adulthood. The tools to obtain functional and morphologic characteristics of arteries in adults have been successfully used to analyze arterial function in the pediatric population. Ultrasound evaluation of morphology and mechanical properties of great elastic arteries and endothelial function of brachial artery have been used in the clinical setting to assess coronary risk in childhood obesity as well as in other high cardiovascular risk populations like heterozygous familial hypercholesterolemia, type 1 diabetes or homozygous homocystinuria.

LITERATURE REVIEW

Wright (2012) With approximately 68% of US people being overweight and 35% being obese, the prevalence of obesity has been continuously growing over the previous few decades and is presently at historic proportions (Flegal et al., JAMA 303:235-241, 2010). The data shows that there has been a rise in every age group, regardless of sex, ethnicity, or smoking status. Interestingly, there has been a disproportionately larger increase in the highest weight category (defined as BMI > 40 kg/m (2)) compared to the lower weight category (defined as BMI < 35 kg/m (2)). Many other nations have also seen a sharp increase in obesity, and researchers still don't know what's causing it (Hill and Melanson, 1999).

Lam (2023) One of the most detrimental diseases to human health is obesity. Despite this, doctors only recommend weight loss to about half of their obese patients, and some of those patients may not even realize that their weight is an issue. By outlining the negative effects of obesity, this paper aims to stress the need of controlling excess body fat. To summaries, there is significant evidence from Mendelian randomization experiments to demonstrate the causal relationship between obesity and over 50 medical problems.

Moorthy (2022) Diabetes, high blood pressure, heart attacks, strokes, and hormonal imbalances are all much more common in those who are overweight or obese. Reducing the prevalence of obesity in India would need a shift in public perception, education, and action. Examining the prevalence of obesity among Indian adults is the primary goal of this research. Methods: Both the city itself and its surrounding rural and urban regions were surveyed using a community-based cross-sectional survey. All participants have to be adults (18+) and eager to take part. With the use of a pre-validated questionnaire, data was obtained from 300 participants. We measured things like height, weight, and KAP scores. To determine whether demographics were associated with positive KAP towards obesity, chi-square tests were used.

Papamargaritis (2022) Obesity is a long-term health problem that may lead to major health problems and even death. Modest long-term weight loss occurs after making changes to one's lifestyle, which may be attributable to compensatory biological adaptations that cause one to eat more and burn less energy, both of which contribute to an increase in body mass index. Only bariatric surgery, up until recently, could reliably cause weight reduction and maintenance of at least 15%. The development of new drugs for the treatment of obesity, with the primary objective being the lowering of appetite, has been driven by our improved knowledge of the endocrine control of hunger.

RESEARCH METHODOLOGY

DATA COLLECTION

We used a semi-structured proforma to get everyone's name, age, gender, and food preferences. Additionally, anthropometric data such as weight, height, and body mass index were recorded.

Additionally, information was documented on the subject's food habits, amount of exercise, heart rate, blood pressure, and BP. Also recorded were the results of lipid profile and fasting blood sugar assessments,

MEASUREMENT OF ANTHROPOMETRIC VARIABLES

Statistically speaking, a person's weight in relation to their height is called their body mass index (BMI). The most used diagnostic technique for determining whether a population is underweight, overweight, or obese is the body mass index (BMI), even though it does not assess body fat percentage. This is because BMI is easy to measure and calculate.

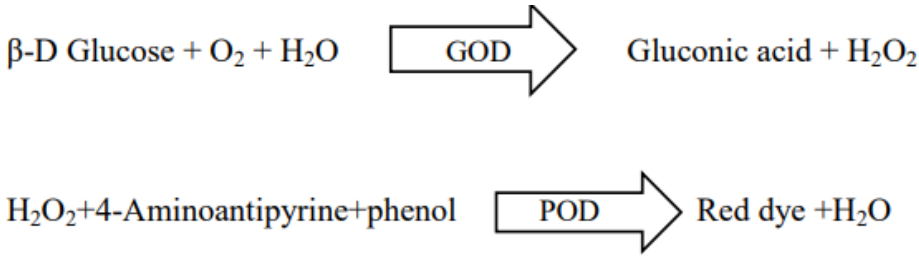
The formula for calculating a person's body mass index is their weight in relation to the square of their height.

The calculations that are utilised everywhere in medicine come up with a measurement of kg/m2. A body mass index (BMI) chart is another way to find someone's BMI. This chart shows the relationship between weight and height, with various contour lines representing different BMI values or colours representing different BMI categories.

MEASUREMENT OF GLYCEMIC LEVELS

In order to estimate fasting blood sugar, patients' blood was drawn after they had fasted for at least 8 hours the night before. Two hours after a typical meal, blood was drawn again to estimate post-prandial blood sugar.

Principle: In the process of glucose oxidation, gluconic acid is produced. Red quinone imine colour is produced when the hydrogen peroxide that is created in this process oxidatively interacts with 4 amino antipyrine and phenol in the presence of peroxidase (POD). A wavelength of 505 nm (500-550 nm) is the upper limit of this dye's absorbance. There is a clear correlation between the specimen's glucose concentration and the colour complex intensity.



ANALYSIS

Under the inclusion criteria (no history of diabetes, hypertension, respiratory illness, alcohol misuse, or steroid/drug dependency), young healthy volunteers (ranging in age from 5 to 18 years old) were recruited and enrolled in the research once their signed agreement was obtained.

Each participant had their anthropometric, hemodynamic, and demographic data recorded on an individual case record form, and all of them were tested for glycemic and lipid profiles in the lab.

Group I consisted of 150 patients with an obesity-related BMI more than 25 kg/m2, whereas Group II included 150 subjects without an obesity-related BMI less than 25 kg/m2. Table 1 shows the distribution of the study population by group.

Table 1: Group-wise Distribution of the Study Population

Group	**Obesity**	**No. of Subjects**	**Percentage**
Group I	Cases (Obese)	150	50%
Group II	Controls (non-obese)	150	50%

Table 2: Gender-wise distribution of the study population

Gender	**Group I (n=150)**	**Group II (n=150)**	**Total (N=300)**
Male	75	75	150
Female	75	75	150

The research included two groups of subjects: Group-I consisted of 50 obese males and 50 obese females with a body mass index (BMI) more than 25 kg/m2, and Group-II consisted of 50 non-obese males and 50 non-obese females with a BMI less than 25 kg/m2.

Table 3: Distribution of cases according to grades of obesity

Obesity Grade (BMI)	**No. of subjects (150) & %**
Obese I (25-29.9 kg/m^2)	124
Obese II (≥30 kg/m^2)	25

Out of all the obese participants, 124, or 83%, were classified as Grade 1 obesity, while 25, or 17%, were classified as Grade II obese.

Table 4: Group-wise comparison of age

Age Group	Group I (n=150)		Group II (n=150)		Total. (n=300)	
	No.	%	No.	%	No.	%
5-11 years	91	61%	99	66%	190	63.5
12-18 years	58	39%	51	34%	109	36.5
	$\chi 2$=1.406, df=1, P=0.236					
Mean ±SD	23.98±3.011		23.67±3.327		23.82±3.169	

People in the research varied in age from five to eighteen years old. Group-I had a greater mean age (23.98±3.011 years) than Group-II (23.67±3.327 years).
While the remaining participants were under the age of 11, the percentage of subjects in Group-I was greater than that in Group-II among those aged 12–18 (39.00% vs. 34.00%). There was no statistically significant difference in the mean age of the two sets of participants.

Table 5: Group Wise Comparison of Food Habits

Food Habits	Group-I (n=150)		Group-II(n=150)		Total (N=300)	
	No.	%	No.	%	No.	%
Vegetarian	97	65.00	99	66.00	196	65.50
Non-Vegetarian	53	35.00	51	34.00	103	34.50
		$\chi 2$=0.022, df=1, p=0.882				

Overall, 65.50% of the population was vegetarian, while 34.50% were not. In group I, 64% were vegetarian and 35% were not; in group II, the numbers were even higher at 66% vegetarian and 34% nonvegetarian. A statistical analysis did not reveal a significant difference (p=0.882) in the eating habits of the participants in Group-I and Group-II.

Table 6: Group-wise comparison of Activity Level

Activity Level	Group-I (n=150)		Group-II (n=150)		Total (300)	
	No.	%	No.	%	No.	%
Heavy	3	2.00	14	9.00	17	5.50
Moderate	76	51.00	106	71.00	183	61.00
Sedentary	71	47.00	30	20.00	100	33.50
		$\chi 2$=18.61, df=2, p=0.009				

The percentage of individuals in Group I who were sedentary was 47% greater than in Group II, where it was 20%.

Table 7: Group wise comparison of Hemodynamic Variables

Parameter	Group-I (n=150)		Group-II (n=150)		Statistical significance	
	Mean	SD	Mean	SD	't'	'p'
Pulse rate	75.15	3.25	73.82	2.28	3.128	0.002
Systolic BP	123.24	4.29	120.68	3.51	4.799	<0.001
Diastolic BP	77.50	3.35	76.34	3.70	2.236	0.028

Compared to Group-II (73.82±2.28mm Hg), the pulse rate difference of Group-I participants was substantially larger (p=0.002) at 75.15±3.25mm Hg.
The systolic blood pressure of Group I was substantially higher (p=<0.001) than that of Group II (120.68±3.51mm Hg), with a value of 123.24±4.29mm Hg.
The patients in Group-I had a significantly higher diastolic blood pressure (77.5±3.35mm Hg) compared to those in Group-II (76.34±3.70mm Hg) (p=0.028).

Table 8: Comparison of Hemodynamic Variables between Obese Male and Non-obese Male

Parameter	Obese Male (n=50)		Non-obese Male(n=50)		Statistical values	
	Mean	SD	Mean	SD	't'	'p'
Pulse rate	74.66	2.861	73.80	2.356	1.584	0.120
Systolic BP	123.32	4.582	120.92	3.641	3.130	0.003
Diastolic BP	77.00	3.288	76.88	3.662	0.164	0.870

The pulse rate of men who were obese (74.66±2.861 beats/min) was greater than that of non-obese males (73.80±2.356 beats/min), however the difference was not statistically significant (p=0.120).

When comparing Obese Males (123.32±4.582mm Hg) to Non-obese Males (120.92±3.641mm Hg), the difference was found to be statistically significant (p=0.003).
There was no statistically significant difference in the diastolic blood pressure of obese males (77.00±3.288mm Hg) and non-obese males (76.88±3.662mm Hg).

Table 9: Comparison of Hemodynamic Variables between obese female and non-obese female

Parameter	Obese Female (n=50)		Non-obese Female (n=50)		Statistical values	
	Mean	SD	Mean	SD	t	p
Pulse rate	75.64	3.572	73.84	2.243	-2.755	0.008
Systolic BP	123.16	4.022	120.44	3.411	-3.632	0.001
Diastolic BP	78.00	3.380	75.80	3.708	-3.080	0.003

When comparing the pulse rates of obese and non-obese females, the difference was considerably greater for the former (75.64±3.572 beats/min.) than for the latter (73.84±2.248 beats/min.).
Obese females had a substantially higher systolic blood pressure (123.16±4.022 mmHg) compared to non-obese females (120.44±3.411 mmHg) (p=0.870).
The difference between the diastolic blood pressure of obese women (78.00±3.380 mmHg) and non-obese women (75.80±3.708 mmHg) was shown to be statistically significant (p=0.870).

Table 10: Comparison of Hemodynamic Variables between the obese males and obese females

Parameter	Obese Male(n=50)		Obese Female(n=50)		Statistical values	
	Mean	SD	Mean	SD	t	p
Pulse rate	74.66	2.861	75.64	3.572	-1.549	0.128
Systolic BP	123.32	4.582	123.16	4.022	0.197	0.845
Diastolic BP	77.00	3.288	78.00	3.380	-1.488	0.143

When comparing obese men (74.66±2.861 beats/min.) to obese females (75.64±3.572 beats/min.), the difference in pulse rate was smaller for the former. as well as the fact that this variation did not reach statistical significance (p=0.128).
Although it was not statistically significant (p=0.845), the systolic blood pressure of obese men (123.32±4.582 mmHg) was higher than that of obese women (123.16±4.022 mmHg).
The diastolic blood pressure (BP) of obese men was shown to be lower (77.00±3.288 mmHg) than that of obese women (78.00±3.380 mmHg), however this disparity was not statistically significant (p=0.143).

Table 11: Group wise comparison of blood sugar level

Parameter	Group-I (n=150)		Group-II (n=150)		Statistical values	
	Mean	SD	Mean	SD	t	p
Blood sugar (Fasting)	85.82	5.60	84.74	5.52	1.38	0.171
Blood sugar (P.P.)	121.90	5.47	117.55	3.43	7.43	<0.001

This difference was not deemed statistically significant (p=0.171), but Group-I had higher fasting blood sugar levels (85.82±5.60 mg/dl) than Group-II (84.74±5.52 mg/dl).
There was a significant difference (p=<0.001) in the blood sugar (PP) levels between Group-I (121.9±5.47mg/dl) and Group-II (117.55±3.43mg/dl).

CONCLUSION

Obese men had far higher levels of the lipid variables (total cholesterol, triglycerides, very low-density lipoprotein, and low-density lipoprotein) than nonobese men. Neither group's HDL levels were significantly different from the other, but they were close. same pattern seen in female participants. Grade II obese participants had much greater mean calorie consumption and systolic blood pressure than grade I obese subjects. There was also no statistically significant change in the other metrics.
An association between body mass index and a number of health indicators was shown to be statistically significant. These indicators included systolic and diastolic blood pressure, heart rate, postprandial blood sugar, total cholesterol, triglyceride, very low-density lipoprotein, and low-density lipoprotein levels. Neither body mass index nor HDL levels were significantly correlated with one another. The results show that young people who are overweight or obese had higher blood pressure, sugar, and cholesterol levels than their leaner peers. Young people who were overweight had a higher risk of cardiovascular disease due to their sedentary lifestyle and energy imbalance. Even though no one in the research really had metabolic syndrome, the young people there were at high risk due to their susceptibility for high blood pressure, weight increase, and lipids. In order to curb obesity and its negative consequences on cardiovascular health, the research suggests raising awareness about the problem and encouraging young people to lead healthier lifestyles via more physical exercise and better food choices.

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