



Body composition and its effects on Glucose metabolism in Obese and non-obese adult males

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Abstract

The prevalence of obesity is rising and is regarded as a global issue. Excessive fat deposition in the body is a complex medical problem that has a negative impact on health and increases the risk of developing several illnesses. The current study is aimed to observe the effects of total body fat on the HbA1c levels in normal, non-diabetic, and apparently healthy male adults. A case/control study was conducted in which total body fat was obtained by the BIA method, anthropometric indices were recorded, and HbA1c was done. The mean total body fat in the normal group aged between 30-39 years is 21.95 ± 2.8 and in the same group mean total body fat in obese is observed as 33.3 ± 3.8 , similarly, in the age group 40-49 years the mean TBF observed in the non-obese is 19.3 ± 3.7 whereas, mean TBF in the obese group is 32.8 ± 5.1 . In the case of HbA1c 10 (14.2%) participants in an obese group of the age group 30-39 show HbA1c in the pre-diabetic range however, 1 (1.4%) participant in normal body composition shows pre-diabetic HbA1c. Whereas among participants of age Group 40-49 13 (27%) participants in the obese group and 02 (4.1%) in the non-obese showed HbA1c in the pre-diabetic range. Profound differences were observed in the glucose parameter of healthy obese and non-obese participants. The people with more body fat showed deranged HbA1c levels.

Keywords: Obesity, HbA1c, diabetes, body composition, total body fat.

Introduction

Obesity is a multifactorial medical condition in which excess fat deposition occurs in the body that causes detrimental effects to the health (Upadhyay, Farr, Perakakis, Ghaly, & Mantzoros, 2018). Obesity has now become a potential health issue which shows exponential and rapid prevalence throughout the world, Research shows that prevalence of obesity and overweight has increased two fold since 1980, nearly a one third of world population is classified as obese (Mehrabani & Khazraei, 2018). It has been reported that obesity was a primary cause for 3.4 million deaths in 2010 worldwide (Elffers et al., 2017), prevalence of obesity becomes doubled in adults and kids under 11 years of age and increased three folds in adolescents in comparison to last three decades (Upadhyay et al., 2018) these figures indicate obesity in developed countries as well as under developed /developing world, data suggest that obesity is more prevalent in developed countries in all age groups. According to researchers in United States of America obesity rates are 12.4% for adolescent males 31.7% for adult males, 13.4% for adolescent girls and 33.9% for adult women. Prevalence rates goes up between 1992 and 2002 (Ng et al., 2014).

It is often denounced and considered a false interpretation that obesity is solely associated with a physically inactive lifestyle and unhealthy eating habits. Nonetheless, there is strong evidence-based literature, supporting the other potential factors which interplay in the etiology of obesity, which can be environmental, genetic, and behavioral (Campbell, 2016; Kostovski et al., 2017).

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To face the challenge of increased obesity, awareness of body composition, fat distribution in different parts of the body and its clinical association is critical to get measures at the right time (Shuster, Patlas, Pinthus, & Mourtzakis, 2012). Hippocrates the Greek physician was the first to observe the relationship between obesity and the disease, he observed that “sudden death is more prevalent in those who are obese/fat than in the lean (Xia & Grant, 2013). The health consequences associated with obesity have generally been widely recognized for many years and continuous increase is observed in the progression of diabetes Mellitus (T2DM), cardiovascular disease(CVD) (Kusminski, Bickel, & Scherer, 2016) hypertension,(Seravalle & Grassi, 2017) hyperlipidemia (Sugai et al., 2016) stroke (Mitchell et al., 2015) hormonal imbalances certain cancers (Neuhouser et al., 2015) sleep disorders, fatty liver disease, and other liver problems(Ogrodnik & Jurk, 2017), GIT disease (Phatak, Phadke, & Pashankar, 2016) osteoarthritis (Kulkarni, Karssiens, Kumar, & Pandit, 2016) and infertility especially in females (Broughton & Moley, 2017). All of these put a negative effect on healthcare costs, quality of life, and functional impairment (Warsi, Mahar, Ansari, & Shah, 2020).

Impairment in glucose uptake by cells and its utilization is one of the vital ailments caused due to excess body fat. A number of studies were conducted in order to examine the association between the incidence of diabetes and obesity, the study conducted in Japan reveals obesity as the sole factor for the progression of glucose impairment and diabetes independent of other factors for instance genetics/family history (Tchernof & Després, 2013). Furthermore, central obesity is considered a potential risk factor for insulin resistance than body mass index alone (Doyle, Donohoe, Lysaght, & Reynolds, 2012).

The Glycated hemoglobin or (HbA1c) analysis provides a record of the average value of glucose levels in the blood during the last two to three months in an individual, which is equal to the average life span of red blood cells (RBCs) (McPherson & Pincus, 2011). HbA1c is currently suggested as a standard of care (SOC) for diagnosis and screening of diabetes, more especially Diabetes type II (Sherwani, Khan, Ekhzaimy, Masood, & Sakharkar, 2016).

The aim and objective of the study were to observe the effects of total body fat on the hba1c levels in normal, non-diabetic, and apparently healthy male adults.

Materials and Methods

A case/control study was conducted from July to September 2020, at the department of physiology university of Sindh. Data was collected from the

Hyderabad district. The total number of participants was (n) 118. Nondiabetic, adult and apparently healthy obese and non-obese males were recruited for the study aged between 30 to 50 years. Written consent was obtained before the study was conducted. A self-structured questionnaire was given which contained the demographic characters and medical history of the participants. Anthropometry was done, including weight, height, hip circumference, waist circumference, waist-to-hip ratio, and waist-to-height ratio, and BMI was taken by standard methods (Faheem, Warsi, & Shah, 2019). Total body fat was calculated by using Omron fat monitor (Model: BF508, Japan). In Bio-impedance analysis (BIA) body composition was measured by applying slight alternate current to the body, and by computing reactance and measuring electrical resistance. Tetra polar BIA machines have four electrode sites (2 per foot or & 2 per hand), which yields body fat estimation of the complete body. According to Total body fat %, the participants were divided into two age groups 30-39 years and 40-49 years. Blood was drawn as per the standard protocol of phlebotomy (Warsi, Faheem, Laghari, & Memon, 2019) for the performance of serum glycated hemoglobin HbA1c test and it was performed by the immune-turbid-metric method through the commercially available kit of Cobas.

Statistical analysis

The chi-square test and student t-test (with Yates correction, at 0.05 significant levels) were applied to see the significance level, the odds ratio was also calculated.

Results

Out of 118 individuals, 70 (59.3%) belong to age group 30-39 years and 48 (40.7%) belong to the age group 40-49 years. Among participants of 30-39 years 21(30%) showed normal total body fat and 49(70%) showed increased total body fat. Among participants of 40-49 years 17(35.41%) showed normal total body fat and 31 (64.5%) showed increased total body fat (**Figure 1**).

As shown in **Table 1**, mean total body fat in the non-obese group aged between 30-39 years is 14.7 ± 3.1 and mean total body fat in obese is observed as 31.1 ± 5.2 , similarly in age group 40-49 years the mean TBF observed in non-obese is 15.3 ± 2.6 whereas mean TBF in obese group is 30.6 ± 5.7 .

Table 2 shows comparative anthropometric indices of obese participants of both age groups with their controls, weight, BMI, WHR, WHtR Waist, & hip circumference of both age groups shows significant differences statistically with p -value < 0.05 .

Table 3 shows comparison of the mean HbA1c of obese and non- obese participants of both age groups.

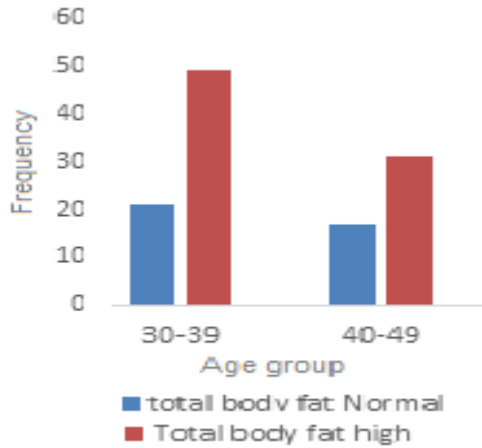


Figure 1. Frequency of obese and non- obese participants according to age

Age (years)	Mean Total body fat		Odd ratio	χ^2	p-value
	Non-obese	Obese			
30-39	14.7 $\pm$ 3.1	31.1 $\pm$ 5.2	0.9	0.05	0.82
40-49	15.3 $\pm$ 2.6	30.6 $\pm$ 5.7			

In non-obese participants of age group 30-39 mean HbA1c is reported as 4.9 ± 0.4 conversely obese participants in the same age group showed mean HbA1c 5.65 ± 0.7 with mean difference of -0.7 . In another age group 40-49 the mean HbA1c in non-obese participants is 4.6 ± 0.3 conversely in their counter parts it is 5.77 ± 0.4 with mean difference of -1.1 . In each age group the mean of obese group is significant statistically with p value <0.05 .

Table 4 indicates the HbA1c range in study participants, 10 (14.2%) participants in obese group of age group 30-39 shows HbA1c in pre diabetic range however 1 (1.4%) participant in normal body composition shows pre-diabetic HbA1c, whereas among participants of Age Group 40-49 13 (27%) participants in obese group and 02 (4.1%) among non-obese showed HbA1c in pre-diabetic range. Odd ratio of 5.1 and 5.4 shows that the odds of getting diabetes in obese participants estimated to be 5.1 times the odds of diabetes in non-obese in age group 30-39 however in age group 40-49 the odds of getting diabetes in obese are 5.4 times higher than non-obese. Since odd ratio reveals that obese people are more vulnerable to become diabetic in future in both age groups.

Table 2. Comparative anthropometric indices (mean $\pm$ SD) of obese and Non-obese individuals.

Indices	Age (Years)	Non-obese	Obese	p-value
Age	30-39	32.3 $\pm$ 3.00	33 $\pm$ 2.9	0.05
	40-49	44.8 $\pm$ 3.35	43.2 $\pm$ 2.9	0.69
Weight	30-39	55.2 $\pm$ 2.7	79.1 $\pm$ 10.9	0.001
	40-49	56 $\pm$ 5.43	78.7 $\pm$ 10.3	0.001
Height	30-39	168 $\pm$ 6.73	165 $\pm$ 5.3	0.07
	40-49	164 $\pm$ 8.0	165 $\pm$ 4.4	0.62
Waist	30-39	76.4 $\pm$ 10.5	98.8 $\pm$ 8.1	0.001
	40-49	80.70 $\pm$ 4.5	102.2 $\pm$ 11.1	0.001
Hip	30-39	93 $\pm$ 6.2	105.8 $\pm$ 7.7	0.001
	40-49	91.58 $\pm$ 5.71	107 $\pm$ 8.6	0.001
BMI	30-39	20.4 $\pm$ 1.2	28.9 $\pm$ 4.1	0.001
	40-49	20.65 $\pm$ 1.0	28.8 $\pm$ 4	0.001
WHR	30-39	0.81 $\pm$ 0.1	0.93 $\pm$ 0.03	0.001
	40-49	0.88 $\pm$ 0.07	0.95 $\pm$ 0.04	0.001
WHtR	30-39	0.45 $\pm$ 0.06	0.59 $\pm$ 0.11	0.001
	40-49	0.49 $\pm$ 0.03	0.62 $\pm$ 0.07	0.001

Discussion

The worldwide incidence and prevalence of overweight and obesity have approximately increased two folds since 1980 to an extent is roughly estimated that more than one-third of the total world's population is classified as overweight or obese. Furthermore it estimated that up to 57.8% of the world population will be classified as overweight by the year 2030 if the same trends will be practiced (Kelly, Yang, Chen, Reynolds, & He, 2008).

There is a well-established epidemiological association between obesity prevalence and insulin resistance (Zatterale et al., 2020), nonetheless this relationship has been investigated less extensively in non-diabetic individuals with normal glucose metabolism than in individuals with DM or IGT.

Table 3: Mean $\pm$ SD HbA1c of obese and non-obese participants of both age groups.

Age (Years)	Mean HbA1c		p-value
	Normal (TBF)	high (TBF)	
30 – 39	4.9 $\pm$ 0.43	5.48 $\pm$ 0.69	0.001**
40 - 49	4.8 $\pm$ 0.47	5.48 $\pm$ 0.61	0.003***

However, most investigations involved subjects with metabolic syndrome and/or impaired glucose regulation. Most of related studies on visceral adiposity involved mainly female subjects who vary physically and metabolically from the male subjects, thus present study revealed about glycemic impairment in non-diabetic individuals of different age groups.

Table 4: Frequency of pre-diabetic and normal HbA1c in study participants.

Age	Hba1c range	TBF Normal	TBF High	Odd ratio	X ²	p-value
30-39	Pre-diabetic	01 (1.4%)	10 (14.2%)	0.27	2.91	0.08
	Normal	20 (28.5%)	39 (55.7%)			
40-49	Pre-diabetic	02 (4.1%)	13 (27%)	0.29	4.65	0.03
	Normal	15 (31.2%)	18 (37.5%)			

HbA1c is in obese individuals is observed high as compared to non-obese normal population, which coincide with another study by Bower et al. (Bower, Meadows, Foster, Foraker, & Shoben, 2017) showing that total fat of the body and trunk fat, is closely linked to increased HbA1c. Our results shows the same trends as reported earlier by (Tchernof & Després, 2013) in a prospective they reported that visceraally obese men is on markedly increased risk of developing diabetes as compared to non-obese men, as obesity is associated with derange glucose parameters, These results are in consistent with the hypothesis presented earlier that certain pro inflammatory cytokines secreted by adipose tissue could contribute to impaired glucose metabolism and insulin resistance (Cartier et al., 2008; Kern, Ranganathan, Li, Wood, & Ranganathan, 2001). Persistent inflammation is considered as a potential risk factor for progressing many diseases including metabolic diseases, cardiovascular problems, malignancies, and diabetes (Chughtai, Zai, Mughal, & Faheem, 2018). As a risk factor, obesity leads to a pro-inflammatory state by increasing inflammatory mediators. Another study was carried out to see the effects of adiposity on glycemic index and it was observed that obese men have more tendency towards getting type 2 diabetes by

showing increased glycemic and insulinemic response towards glucose challenge test as compared to lean controls.

Conclusion

Present study concludes that obesity is closely related with impairment in glucose/insulin metabolism in the body, individuals with more body fat shows derange HbA1c levels when compared with non-obese control.

Limitation

Due to small sample size, we could not generalize our results to large number of people, larger studies with big sample size are required for clearer picture.

Conflict of interest

The authors have no conflict of interest and nothing to disclose, no financial aid is received from any funding body.

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