REVIEW OF LITERATURE

➢ BMI

World Health Organization,(2000) reported that BMI >25.0 and >30.0 kg/m2 was taken as cut-offs for overweight and obesity, respectively. WC cut-offs were taken as >94 cm for males and >80 cm for females to define overweight[15].

Webb GP ,( 2002)The cut-off used for WHR were >0.9 kg/m2 for males and >0.8 kg/m2 for females[31].

Coutinho T et al ,(2011) said that people with central obesity have higher mortality even when the BMI is normal and so waist circumference should be recorded and pursued in every person in addition to BMI for good risk stratification and therapeutic considerations[32].

Smita t patil et al , (2012) have taken 111 males and 93 females. Out of 204 patients, 111 (54%) were males and 93 (46%) were females. BMI (18-22.9 kg/m ) was found in 39 patients (19%), 114 patients (58%) were overweight (23-24.9 kg/m ) and 51 patients (23%) were obese BMI >25 kg/m. Women had a higher incidence of abnormal waist circumference compared to the men. 53% males BMI = 25 Kg/m2 were defined as obese. (n=60) had waist circumference above 90cm and 72% females (n=66) had waist circumference above 80cm[33].

Olumide A Abiodun et al , (2014) have taken total 776 individuals, 200 (25.8%) males and 576 (74.2%) females were examined. Among them, 232 (29.9%) were overweight and 136 (17.5%) were obese while, 28 (3.6%) were underweight[34].

➢ Hypertension

Huang Z et al ,(1998)The Nurses’ Health Study supported the findings that put on the weight in middle age is a risk factor for high blood pressure [35].

King DS et al, (2000) told that one-third of all high blood pressure cases are pertained to obesity, while being obese increase the risk for growing high blood pressure triplex [36]. Information from the cohort of 5,209 women and men in the Framingham Heart Study denote that for every 10-pound of put on weight, systolic blood pressure raises about an average of 4.5 mm Hg [36]. And a decrease of 2.2 pounds in body weight outcome in a decrease of 0.3 to 1 mm Hg.

Mertens IL et al , (2000) got that Even a moderate weight destruction (about 5%-10% of body weight) can decrease blood pressure, and this seems to be independent of sodium deduction[37].
Webb GP et al, (2002) said that Diastolic and Systolic blood pressures rose significantly with increasing BMI status and were significantly richly with abnormally raised waist hip ratio than in participants with normal waist hip ratio[31].

Anjum Humayun et al, (2010) said that It is proved that out of 541, males, 340 (63%) were hypertensive and 315 out of 465 (68%) females were hypertensive. The correlation of normal BMI with high blood pressure is 34% and overweight is 58% while that of obese is 77% showing a powerful relationship of hypertension with BMI[38].

➢ Diabetes

Chan JM et al, (1994) have gotten the data from the Professionals Health Study established the same correlations between body weight and type 2 diabetes mellitus among men. The dangerous factors of diabetes mellitus among men with a BMI of 35+ was 42 times that of men with a BMI of < 23 [39].

Colditz GA et al, (1995) got that many obese people, those have BMIs of 40+, are over with 53 times dangerous for type 2 diabetes. Even lowest overweight shows a risk; the Nurses’ Health Study narrated that women have BMIs in the range of 24 - 24.9 had a 5 times more risk of diabetes when assimilated with women with BMIs of < 22 [40].

Caterson ID et al, (1997) said that obesity is one of an assemblage of manufacturer for type 2 diabetes together called as Syndrome X, insulin resistance syndrome, or metabolic syndrome. Visceral (abdominal) fat is trusted by a lot of researchers to be more strongly associated with Syndrome X than subcutaneous fat [41].

Abdominal fat is an active metabolic tissue and discharges fatty acids, which accelerate in the liver and peripheral tissues, decreasing the effect of insulin on muscle cells and liver. The free fatty acids are utilized by the muscles at the expense of glucose, resulting raised levels of glucose in the blood that in turn result in raised insulin outcome by the pancreas[41].

Kushner RF et al, (1999) have demonstrated to the Academy of Managed Care Pharmacy in 1999, that obesity is liable for 61% of type 2 diabetes in the United States. Wolf and Colditz found that obese persons have a 27.6-folds extra risk of developing type 2 diabetes than do normal weight persons [42].

Dietz WH et al, (2001) said that pediatric type 2 diabetes has been founded to chance very frequently among obese females aged 12 to 14 years [43].

ABC News, (2002) has demonstrated that a fresh study by researchers at Yale University of 167 adolescents and obese children found that 21% of the adolescents and 25% of the children had not paired glucose tolerance, an proved risk factor of type 2 diabetes [44].
Webb GP et al, (2002) got that RBS was also significantly higher in those with elevated waist hip ratio than in those with normal waist hip ratio. RBS tended to increase significantly with increase in BMI status but overweight individuals tended to have a higher RBS than the obese[31].

Arshad Parvez et al, (2010) have taken diabetic patients included 120 obese having BMI > 30, and 40 non-obese with BMI < 30. In the obese diabetics, there were 55% having <110 serum glucose, 8.3% having 110-126 serum glucose, 36.6% Having > 126 serum glucose and in the non-obese diabetics, there were 32.5% having <110 serum glucose, 5% having 110-126 serum glucose, 62.5% having >126 serum glucose[45].

The connection between raised BMI and an raised risk for type 2 diabetes has been showed in various populations, including those with both high rates of diabetes and traditionally low. Type 2 diabetes was formerly known adult-onset diabetes, an appellation no longer applicable like it is now being diagnosed with alarming frequency among young adults and adolescents as well as younger children[45].

Lipid

Khaodhiar L et al, (1999) founded that raised cholesterol levels have been long recognized as having an association with obesity. Obesity serves to result in an elevation in total cholesterol level and triglycerides and a deduction in high-density cholesterol (HDL). Abdominal obesity can result a raised production of low-density cholesterol (LDL) substance that are denser and smaller than normal, putting a person at greater risk of atherosclerosis, and also raised very-low-density lipoprotein (VLDL) and reduced HDL [46].

Myers MD et al, (2002) have been assessed that, on average, each 10 pounds of extra fat produces more 10 mg. of cholesterol daily, which is equivalent of eating one excess egg yolk every day[47].

Hardev Singh Sandhu et al, (2008) have done the study in which Serum cholesterol, the maximum mean value (222.97 mg/dl) was noted in age group 41 – 50 years and the minimum (189.42 mg/dl) in age group 31 – 40 years. For HDL – C, the maximum mean value (43.58 mg/dl) was found in age group 41 – 50 years and the minimum (40.06 mg/dl) in age group 61+ years. For LDL – C, almost equal distribution was recorded in all the four age groups. For triglyceride, the maximum mean value (264.19 mg/dl) was noted in age group 41 – 50 years and the minimum (235.08 mg/dl) in age group 31 – 40 years. In females, the maximum mean value (30.07 Kg/m2) was recorded in age group 31 – 40 years and the minimum (26.31 Kg/m2) in age group 51 – 60 years for BMI. For serum cholesterol, the maximum mean value (230.43 mg/dl) was noted in age group 31 – 50 years and the minimum (199.43 mg/dl) in age group 61+ years. For HDL – C, the maximum mean value (43.52 mg/dl) was recorded in age group 41 – 50 years and the minimum (42.30 mg/dl) in age group 61+ years. For LDL – C, the maximum mean value (115.08 mg/dl) was found in age group 51 – 60 years and the minimum (105.14 mg/dl) in age group 31 – 40 years. For triglyceride, the maximum mean value (258.44 mg/dl) was noted in age group 51 – 60 years and the minimum (227.48 mg/dl) in age group 61+ years [48]
Hubert HB et al, (1983) told that Extra fat tissue needs an raise in blood flow, the supply of which needs greater cardiac output and raised cardiac workload. As with high blood pressure, weight gain after years the young adult shows more risk independent of initial weight or additional risk factors associated with the gain[49].

Khaodhiar L et al, (1999) described that raised risk of coronary heart disease, obesity has been associated with congestive heart failure(CHF), cardiomyopathy, and myocardial hypertrophy [50].

Romero-Corr al A et al, (2006) and Lavie CJ et al, (2009) founded that On the non obese patients have a better survival when they develop a heart attack or undergo coronary procedures such as angioplasty, stent or bypass surgery. This thing has been commonly pertained to as the ‘obesity paradox’ [51,52].


Coutinho T et al, (2011) have cleared that an raised BMI was indeed associated with a 36% lower mortality in coronary heart disease CHD patients. However, abdominal obesity was associated with 70% higher mortality in individuals with and without high BMI. Both high waist circumference (WC) and high waist hip ratio (WHR) were used as an index of abdominal obesity. So, it is suggested that waist circumference and waist hip ratio are more reliable than BMI in stratifying mortality risk in coronary heart disease patients, and waist circumference and/or waist hip ratio should be documented in individuals with coronary heart disease and normal BMI for better risk stratification and therapeutic considerations [32].

Margot Shields et al, (2012) have gotten that when the odds of with at least two cardiovascular disease risk factors were examined within BMI classification, significant associations were observed for waist hip ratio and waist to height ratio among men in the normal-weight (2.98 for waist hip ratio and 2.38 for waist to height ratio based on standardized variables) and overweight (1.74 for waist hip ratio and 1.96 for waist to height ratio based on standardized variables) classifications [57].

Correlation
Comparison of the correlation between 3 anthropometric measurements (WHtR, WC, BMI) with glycaemic control

Webb GP et al, (2002) said, SBP, DBP, RBS and WHR had positive correlation with BMI. SBP, DBP, RBS also had a positive correlation with WHR. After controlling for effects of age and sex the correlation of RBS, SBP and DBP with WHR decreased to 0.078, 0.184) and 0.166 respectively. The correlation of SBP and DBP with BMI also decreased to 0.178 and 0.123 respectively whereas the correlation between RBS and BMI slightly increased to 0.084. RBS still had no correlation with SBP (-0.016) and DBP (0.009)[31].

Balsam Mahdi Nasir Al-Zurfi et al (2012) founded correlations between anthropometric parameters (BMI, WC and WHtR) and glycaemic control were not significant, weak and inversed [58].

Onyesom Innocent1 et al, (2013) got the age group, 16-20 years, the 35 female subjects showed an optimistic but weak correlation between body mass index and blood glucose level while the 35 male subjects showed an optimistic but very important correlation between body mass index(BMI) and blood glucose level (BGL). As a whole, there was a very important optimistic correlation between body mass index (BMI) and blood glucose level (BGL) among 70 subjects in this age. For the age group, 21-25 years, the 46 female and 84 male subjects showed a optimistic significant. As a whole, there was a optimistic and statistical meaningful correlation between body mass index and blood glucose level among 130 subjects in this group. For the age group, 26-30 years, there was a optimistic and low statistical correlation between body mass index and blood glucose level among the 21 female and 32 male subjects. As a whole, body mass index (BMI) and blood glucose level (BGL) showed a optimistic and weak correlation among 53 subjects in this age group. Over all there is a relationship between body mass index (BMI) and blood glucose level (BGL) irrespective of sex and age. BMI and BGL showed a optimistic but weak correlation among 151 male subjects. Among the 102 female subjects, there was a optimistic and very important correlation. As a whole, for the 253 subjects, body mass index (BMI) and blood glucose level (BGL) showed a optimistic but very low significant correlation [59]. The study included 234 subjects with a mean age from 39 years to 50.4 years and BMI of 25.2±3.8 kg/m2. The mean SBP were 137.9±9.6 mmHg and DBP were 94.4±8.8 mmHg. The mean BMI, HDL, and LDL were higher in males than the females, which was statistically meaningful . The mean waist circumference (WC) was higher in males, which was not statistically meaningful.

Kamrun Nahar Choudhury et al, (2014) founded, The mean age of hypertensive patients were from 43.4 to 51.8 years and normotensives were 38.4±3.7 years. Serum levels of total cholesterol (TC) were 238.3±3.4 mg/dl, Triglyceride (TG) were 178.3±6.3 mg/dl, and low density lipoprotein (LDL) were 151.3±7.8 mg/dl, in hypertensive patients while in normotensive patients, they were 187±6.2 mg/dl TC, 141.5±11.2 mg/dl TG, and 110.3±6.3 mg/dl LDL, which were significantly higher in hypertensive patients. The serum HDL was significantly lower in
hypertensive patients (41.2±3.2 mg/dL) than in normotensive subjects (44.3±5.6 mg/dL). The mean SBPs of hypertensives were 146.8±8.5 mmHg and normotensives were 119.2±9.3 mmHg, respectively, and mean DBPs of hypertensives were 98.9±7.3 mmHg and normotensives were 84.9±5.3 mmHg. The mean systolic blood pressure and diastolic blood pressure of hypertensives were higher than those of normotensives. WC, Age and BMI showed meaningful association with hypertensive patients but not with normotensive subjects[60].

ONI OA et al, (2014) Revealed the mean age of patients (59 years), mean arterial Pressure, systolic and diastolic blood pressure (SBP and DBP). Also, the mean BMI for women is slightly higher than the values for men. Rural (R) dwellers have a higher prevalence of hypertension than urban (UR) dwellers in this study. The associated hypertensive risk factors include Family history (90%), occupation (Farmers=69.2%), Environment: Rural(R) = 72.3%, alcohol consumption (56.9%), tobacco smoking (53.8%), Raised Lipid level (10.7%) and Age (Hypertension peaks at the 6th decade of life). More female had high blood Pressure from the third to the fifth decade of life while it equals at the sixth decade and subsequently higher in male than female [61].

FTO Gene

Yajnik CS et al, (2002) got that the results of FTO on BMI (per-allele change of 0.30 kg/m2) and other regional measurements have significant clinical implications since elevated cardiometabolic risk has been reported to relationship even with inferior cutoffs of adiposity in the group of Indians [62].

Frayling TM (2007), Scuteri A (2007), Saunders CL (2008), Thorleifsson G (2009), Willer CJ, (2009), Speliotes EK (2010), have got proof from genome-wide association study (GWAS) and meta-analyses there for have confirmed the relationship of FTO variants with obesity and related traits in Caucasians [63-39].

Li H(2009), Lindgren CM(2009) suggested that FTO variants elevate obesity risk by 1.20–1.32 times in Europeans [69] and by 1.25-times in Asians [70]. In Indians, they showed an OR of 1.15 elevate in obesity risk equal to per allele BMI elevate by 0.30 kg/m2 (790 g/allele for a individual 1.65 m tall). The confidence intervals (CI) for the assessments were overlapping between populations, and so they resulted that the effect of FTO on adiposity in Asian Indians is by large similar to what is reported in other populations. For more exact evaluate, larger study samples were required. Other than overall adiposity, their results furthermore conclude that rs9939609 also elevates point assessments of regional adiposity measurements (WC, HC, and WHR) to a magnitude same to that observed in the population of Eastern Asian (16), and slightly higher than previously reported in the group of Caucasians [68,71].

Vasan SK(2011), Heid IM(2010) previously showed that rs9939609 was relationship with elevate in regional skin-fold thicknesses that show subcutaneous fat depots [72]. They gave the powerful correlations between BMI, WC, and WHR, the relationship of FTO with regional adiposity traits is anticipated and is comparable to preceding reports in Caucasians [64,73].
Sihua Peng (2011) said that under per-allele comparison, the OR was not obtainable from the study by Li et al., leaving 29 studies for other thought. A total of 21 out of 29 studies reported a important, positive relationship between obesity and the rs9939609 genotype. This meta-analysis indicated that FTO might be represent a low-penetrance responsible gene for obesity risk. Separate studies with large numbers of sample sizes were required to further estimate the relationship between the polymorphisms and obesity risk in various populations of ethnic [74].

Meyre D (2012) told that the devaluation observed in the running and Li et al., meta-analysis warning us that small changes in BMI that help to estimate the FTO-T2DM relationship might be ineffective in Asians for the reason that BMI did not actually reflect the obesity-related diabetes risk in the same way like in the populations of Western and intrinsic ethnic differences in body composition were observed over different populations. So It is important that estimation of association between FTO-T2DM and adiposity should include adjustments to regional adiposity measurements. In this contexture, they acknowledged that non-availability of data from separate studies concerning diabetes risk adjusted for measurements such as WC, limits the running analysis to more investigation if the effect of FTO on T2DM risk was mediated even so central adiposity [75].

Wei-Wei Chey (2013) concluded that the minor A allele frequency (MAF) of the FTO rs9939609 SNP was low - as in other populations of Asian and there was no proof for the participation of the FTO rs9939609 SNP in obesity and obesity-associated traits amongst this multi-ethnic Malaysian study group. The distribution of the genotype and allele frequencies of this FTO gene variant was also important difference among ethnicities, with Malays having the significantly highest the minor A allele frequency. On the basis of the results available so far, the function of FTO (rs9939609 SNP) in obesity hang around inconclusive in the groups Malaysian subjects [76].

Association with Obesity and Related Traits

Senthil K (2014) did the study of meta-analysis included pooled data from 28,394 individuals (16,741 males and 11,653 females) for obesity and associated traits. Their results showed that the common variant of FTO elevates obesity risk in Indians with an OR of 1.15 (95% CI). Each additional risk allele of rs9939609 was related with the elevation in BMI by 0.30 kg/m2, WC by 0.74 cm, HC by 0.52 cm, and WHR by 0.002 units in the pooled population. The HC meta-analysis demonstrated moderate heterogeneity, and no heterogeneity was found for the other obesity-associated traits [77].

Association with type 2 Diabetes

Senthil K (2014) did the study meta-analysis of FTO-associated type 2 diabetes mellitus (T2DM) risk included six studies. Meta-analysis was performed for two individual models, the first one model was adjusted for age and gender and the second one for age, gender, and BMI. In the first model, an overall elevation in diabetes risk was observed with an OR of 1.11 (95% CI). This relation presented modest devaluation when more adjusted for BMI [77].
From the above account the relationship of FTO gene in Indian population is still not touched in details, further the results showed till date did not has clear demarcation in term of relation between FTO gene and fat obesity. To fulfill this gap we are proposing a research proposal in this thrust area. This research could be provide key notes in this area and may be helpful to improving health of mankind.