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Assessment of association of obesity with diabetes mellitus among general population of Gulberg, Lahore

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List of Abbreviations

Abbreviation	Explanation
• WHO	World Health Organization
• SPSS	Statistical Product for Service Solutions
• DHS	Demographic and Health Survey
• NGO	Non-Governmental Organization
• WMA	World Medical Association

Abstract

Introduction:

Obesity & Diabetes is one of the leading problems in the world. Many factors influence this. Among them are diet , life style, sleep patterns and inactivity. Insulin sensitivity decreases rapidly and markedly without adequate compensation in beta cell function, resulting in an elevated risk of diabetes. Obesity is a strong risk factor for Type 2 diabetes. Type 1 diabetes is juvenile and is also due to beta cell dysfunction.

Objectives:

1) To assess the association of obesity with diabetes mellitus among general population of Lahore
2) To suggest and recommend health authorities and community to share the responsibility and to participate actively in health activities for the betterment of society and nation at large.

Method:

A descriptive cross sectional survey will be conducted & data was calculated through a self-administered questionnaire from general population of Lahore. Questionnaire was consist of information about association of obesity with diabetes mellitus .

Results:

The results was calculated after compilation of data by SPSS software version 22

Conclusions:

Conclusion was drawn after the compilation of data.

Key Words:

Obesity, Diabetes mellitus, glucose tolerance, energy.

Introduction

Diabetes mellitus once regarded as a single disease entity but now is seen as a heterogeneous group of disease characterized by a state of chromic hyperglycemia resulting from a diversity of etiologies, environmental and

genetic acting jointly **(K.Park,2015).**Obesity may be defined as an abnormal growth of the adipose tissues due to an enlargement of fat cell size (hypertrophic obesity) or an increase in fat cell number (hyperplastic obesity) or a combination of both. Obesity is also expressed in terms of BMI, more than 25.0 are obese **(K.Park,2015).**

On studying the largest survey in United States ,shows a continuing increase of obesity and diabetes in both sexes, all ages, all races, all educational levels and all smoking levels. Because of the strong association between overweight and obesity and several well established risk factors for morbidity and mortality, reversing the obesity epidemic is an urgent priority **(AH Molded, Es' Ford, BA Bowman, WH Dietz..... jam 2003)**.The most common underlying cause of diabetes is the defective production or action of insulin a hormone that controls glucose, fat and amino acid metabolism. Chronic hyperglycemia from whatever cause leads to a number of complications i-e cardiovascular, renal, neurological, ocular and other incurrent infections **(K.Park,2015).**

Obesity is an important risk factor for cardio metabolic disease including diabetes, hypertension and coronary heart disease. Several leading national and international institutions including WHO and the national institution of health have provided guidelines for classifying weight status. **(S Klein, DB Allison, DE Kalley... 2007)** .Both obesity and diabetes are preventable. Previous studies have demonstrated that changes in lifestyle are effective in preventing both diabetes and obesity in high risk adults with impaired glucose tolerance. Increasing physical activity, improving diet, then sustaining these lifestyle changes can reduce both body weight and risk of diabetes **(AH Mokdad, ES Ford, BA Bowman, WH Dietz .... jama 2003)**.

We previously reported that less than 20% of US adults who were trying to lose or maintain weight were following recommendation to eat fewer calories and increase physical activity to at least 150 per week. Health professionals must continue to stress the importance of balanced diet and physical activity for healthy weight loss **(K.PARK 22nd edition)**

We will do this research because of our keen interest for the health of our people. As our study finds that educated people are more aware about the disease but as regards risk assessment scale being people living in rural communities and even educated were prone towards diabetes possibly due to dietary habits and lifestyle. So we want to save our people by educating them. The proportion of individuals found at high risk and low risk needs further screening for diabetes and

health education regarding diabetes mellitus and obesity is required in these areas. This work is aimed to give suggestions to Pakistan government (Ministries) ,World health organization(WHO) & other nongovernmental organizations (NGOs) to take measures to create general awareness regarding association of obesity with diabetes mellitus in people & fill the gaps & lags present in this field.

Literature Review

Diabetes kills 90,000 patients every year in Pakistan By Muhammad Qasim

Pakistan has seen dramatic increase in rates of diabetes prevalence and incidence over recent years and the key risk factor for type-2 diabetes, obesity, in young people is rising concomitantly rapidly in Pakistan, especially in the cities and towns. Approximately, diabetes kills 90,000 people annually in Pakistan of which majority are women. Studies reveal that Pakistan ranks 7th among the top 10 countries with the highest number of people living with diabetes. The diabetes prevalence rate in Pakistan is 12 per cent, which is expected to rise up to 30 per cent by 2025 and then Pakistan will rank 5th in the world. Also the age of onset of diabetes in Pakistan is one of the lowest in the world and it is becoming a disease of young generation.

Note all changes in people's levels of physical activity and food consumption leads to changes in population weight status. For example, if more people met the guideline of being active for 30 mints on most days, this by itself might not be sufficient to prevent weight gain, because energy intakes also important. Conversely, reducing energy intake may not prevent weight gain, especially in those who are already obese, unless there are simultaneously physical activities **(Brown et al., 2011).**

A research looked at the relationships between changes in lifestyle, diet and long-term weight changes. The study was based on three separate prospective cohort studies that included 120,877 men and women in the US. Their aim was to investigate the relationship between multiple lifestyle changes and long-term weight gain in studies that had followed people up over time (prospectively). Aggregate dietary changes were associated with substantial differences in weight change (3.93 lb across quintiles of dietary change). Other lifestyle factors were also independently associated with weight change ($P<0.001$), including physical activity (−1.76 lb across quintiles); alcohol use (0.41 lb per drink per day), smoking (new quitters, 5.17 lb; former smokers, 0.14 lb), sleep (more weight gain with <6 or >8 hours of sleep), and television watching (0.31 lb per hour per day) **(Dariush Mozaffarian, Tao Hao, Eric, et al, 2011)**

Another research was conducted to examine the long-term relationship between

changes in water and beverage intake and weight change. It was carried out on 3 cohorts of 50 013 women aged 40–64 years, 52 987 women aged 27–44 years and 21 988 men aged 40–64 years without obesity and chronic diseases at baseline. They assessed the association of weight change within each 4-year interval, with changes in beverage intakes and other lifestyle behaviors during the same period. They found out that replacement of 1 serving per day of SSBs(Sugar sweetened beverages) by 1 cup per day of water was associated with 0.49 kg (95% CI: 0.32–0.65) less weight gain over each 4-year period, and the replacement estimate of fruit juices by water was 0.35 kg (95% CI: 0.23–0.46). Substitution of SSBs or fruit juices by other beverages (coffee, tea, diet beverages, low-fat and whole milk) were all significantly and inversely associated with weight gain and concluded that increasing water intake in place of SSBs or fruit juices is associated with lower long-term weight gain.**(A Pan, V S Malik, T Hao, W C Willett, D Mozaffarian and F B Hu, 2011)**

Study conducted by WHO provides an important basis for action. It synthesizes available evidence for action and aims to facilitate the development and implementation of national action plans to improve diets and increase physical activities, with specific reference to healthy weight.**(WHO Global Strategy on Diet , Physical activity and Health 2010).**The evidence on population intervention to address weight gain and overweight and obesity in adults has been the subject of number of recent reviews **(The report published by the Heart Foundation of Australia 2013.)**

The case for screening for undiagnosed diabetes is probably somewhat stronger than it was at the last review, because of the greater options for reduction of cardiovascular disease, principally through the use of statins, and because of the rising prevalence of overweight and hence type 2 diabetes. However, there is also a good case for screening for IGT, with the aim of preventing some future diabetes and reducing cardiovascular disease.**(McNamee P, Gillett M, Brennan A, Goyder E, et al2007)**

Objectives

- To assess the association of obesity with diabetes mellitus among general population of Lahore.
- To suggest and recommend health authorities and community to share the responsibility and to participate actively in health activities for the betterment of society and nation at large

MATERIAL & METHODS

Study Variables:

a) Dependent variable : Obesity related to diabetes.

b) Independent variable: Sedentary life style and unawareness of disease.

Study Design:

Descriptive Cross Sectional.

Study Setting:

gulberg.

Study universe:

Lahore.

Study population:

General population of Lahore.

Study duration:

a)Commencement Time: Three Times.

b)Completion date: September 18th 2016

Study subjects selection criteria:

Inclusion criteria: General population of all age groups. All males and females were included in the research. All those who gave consent were included in the research.

Exclusion criteria: All non-obese men and women. All ill due to some disease wee not included in the research. All those not giving consent were not included in the research.

Social and Ethical Considerations:

Observing cultural ethics.

Consent will be obtained for interview.

The information about the names addresses etc. will not be disclosed to anyone and will not be used for unethical purpose.

Sample size calculations:

It will be calculated through SPSS software version 22.

Sampling Technique:

Random sampling will be done.

Date Collection plan:

The data will be collected by a team of 5 members.

Data compilation and analysis:

The data will be compiled and analyzed through SPSS software version 22.

Pre-tested:

Before carrying out the actual exercise of data collection, questionnaire will be tested on some subjects on experimental basis to observe any deficiency in questionnaire.

Work Plan

Activity	1St Week	2nd Week	3rd Week	4th Week	5th Week	6th Week
Finalization and approval of Synopsis	August 7th-14th					
Data Collection		August 15th-22th				
Data Compilation			August 23th-29th			
Data Analysis				August-September 30th-5th		
Report Writing					September 6th-12th	
Report Presentation						September 13th-19th

Budget

The various aspects of this exercise which demand financial support are as follows:

Photostats	**Rs. 500**
Prints	**Rs. 500**
Stationary	**Rs. 380**
Computer	**Rs. 700**
Final prints	**Rs. 850**
Total	**Rs. 2930**

Results

Table1:

Gender percentage of representing population

Respondent's Sex					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Female	20	40.0	40.0	40.0
	Male	30	60.0	60.0	100.0
	Total	50	100.0	100.0	

Results:

It states that:

- 40 % people were female.
- 60% people were male.

Table2:

Age of population selected for the research

Respondent's Age					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Age group b/w 20-30	28	56.0	56.0	56.0
	Age group b/w 30-40	20	40.0	40.0	96.0
	Age group b/w 40-50	2	4.0	4.0	100.0
	Total	50	100.0	100.0	

Results:

It states that:

- 56% people belongs to 20-30 years age group
- 40% people belongs to 30-40 years age group
- 4% people belongs to 40-50 years age group

Table3:

Awareness of association of obesity with diabetes in society

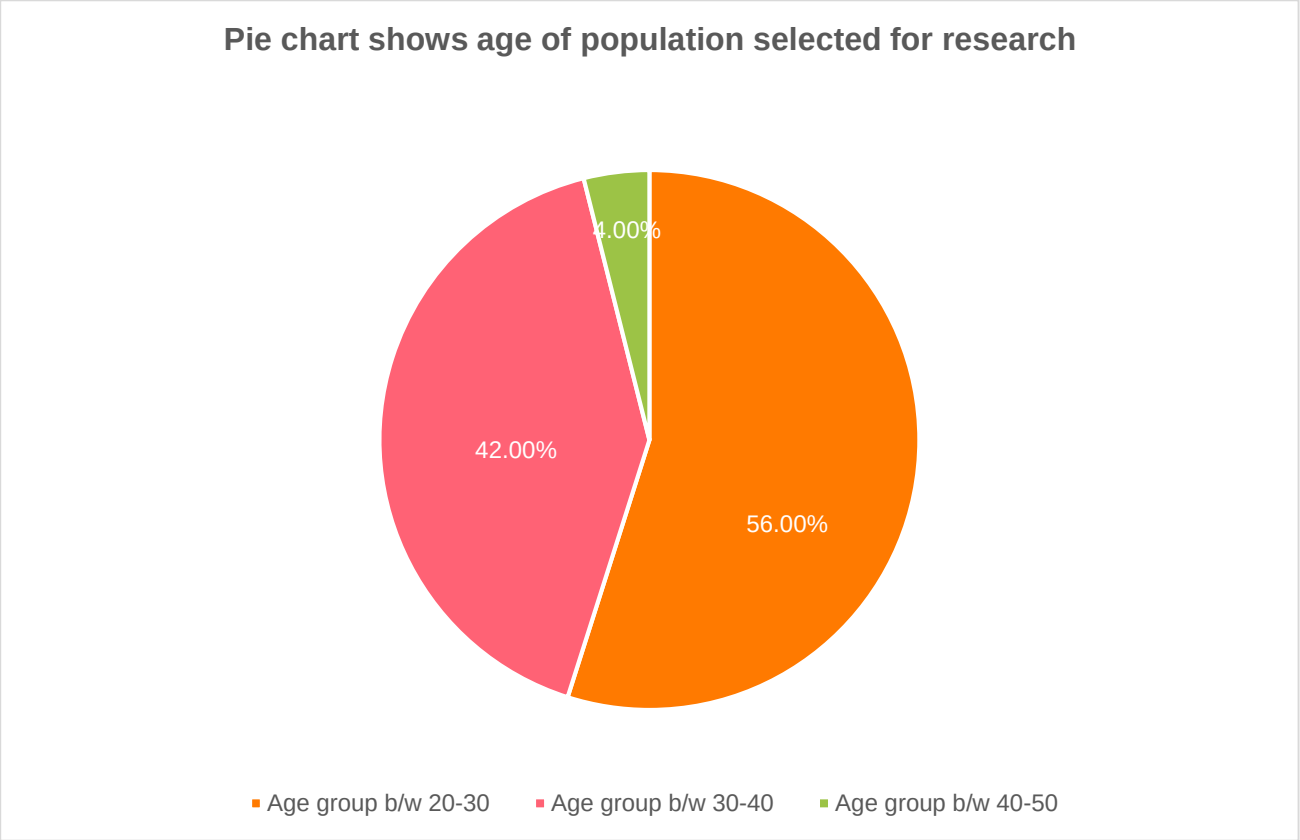
Awareness of association of obesity with diabetes in society					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	No	21	42.0	42.0	42.0
	Yes	29	60.0	58.0	100.0
	Total	50	100.0	100.0	

Results:

It states that

- 42 % people were not aware of the association of obesity with diabetes.
- 58% people were aware of the association of obesity with diabetes.

Age of population selected for research



Results:

- 56% people belongs to 20-30 years of age group.
- 42% people belongs to 30-40 years of age group.
- 4% people belongs to 40-50 years of age group.

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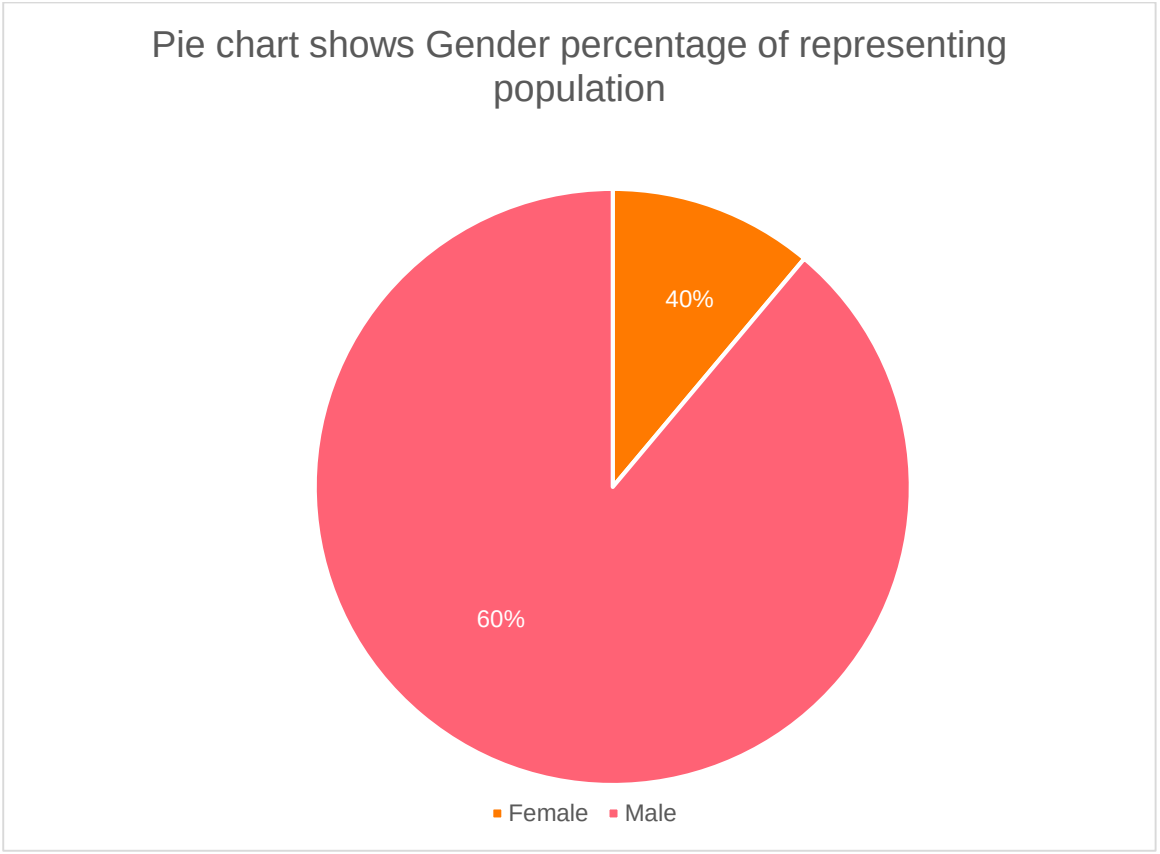
Pie chart shows Gender percentage of representing population



Results:

- 40% people were female.
- 60% people were male.

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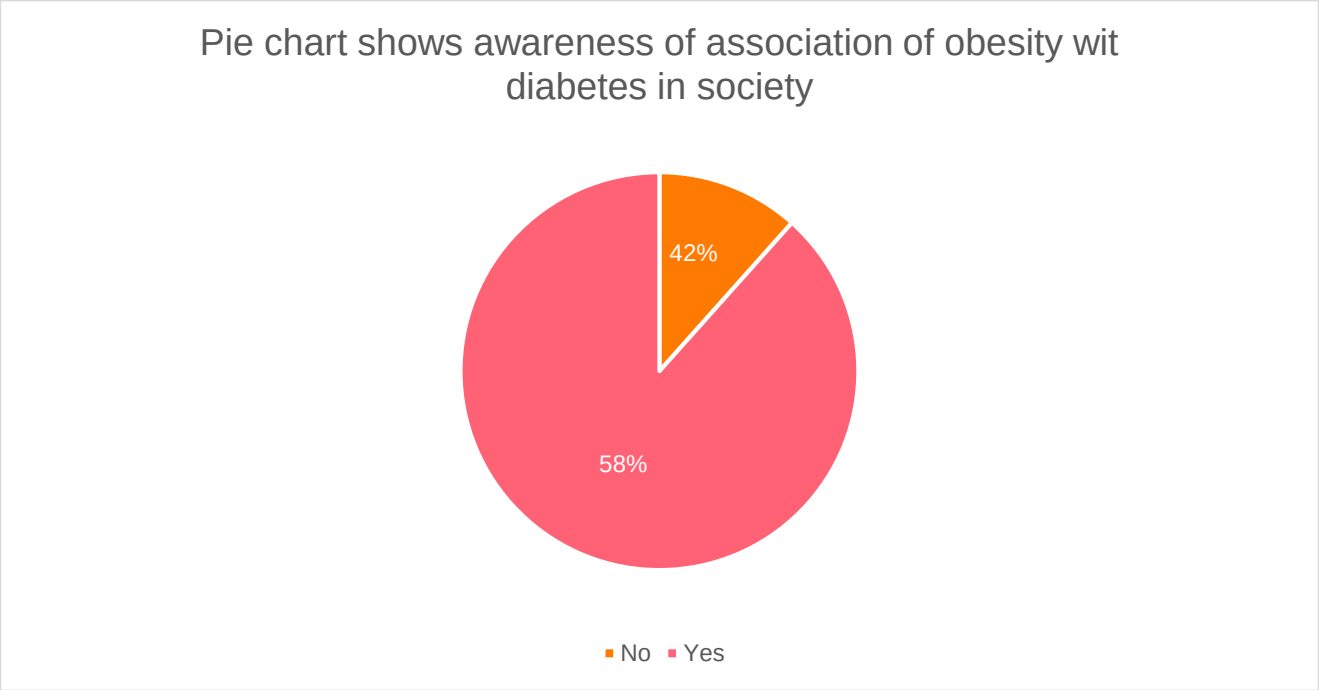
Pie chart shows awareness of association of obesity wit diabetes in society

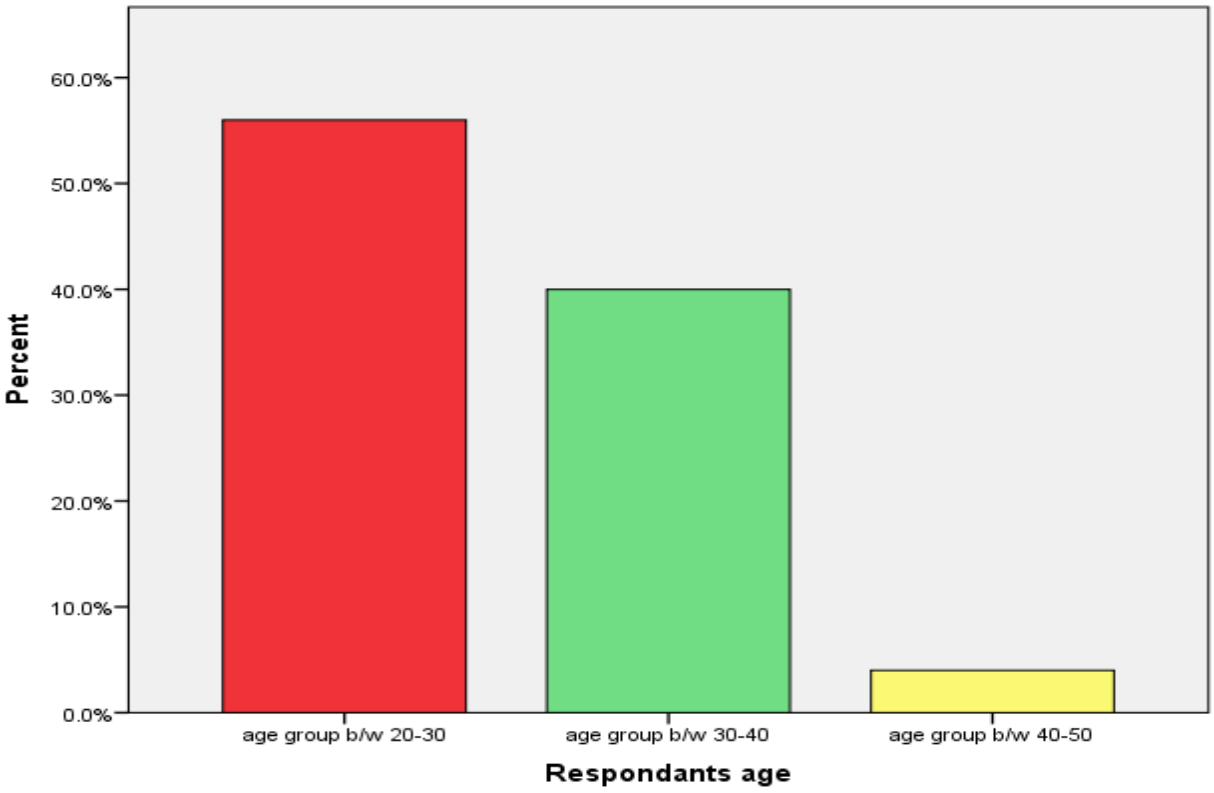


Results:

- 42% people were not aware of the association of obesity with diabetes.
- 58% people were aware of the association of obesity with diabetes.

Figure: 1

Bar chart shows age of population selected for research



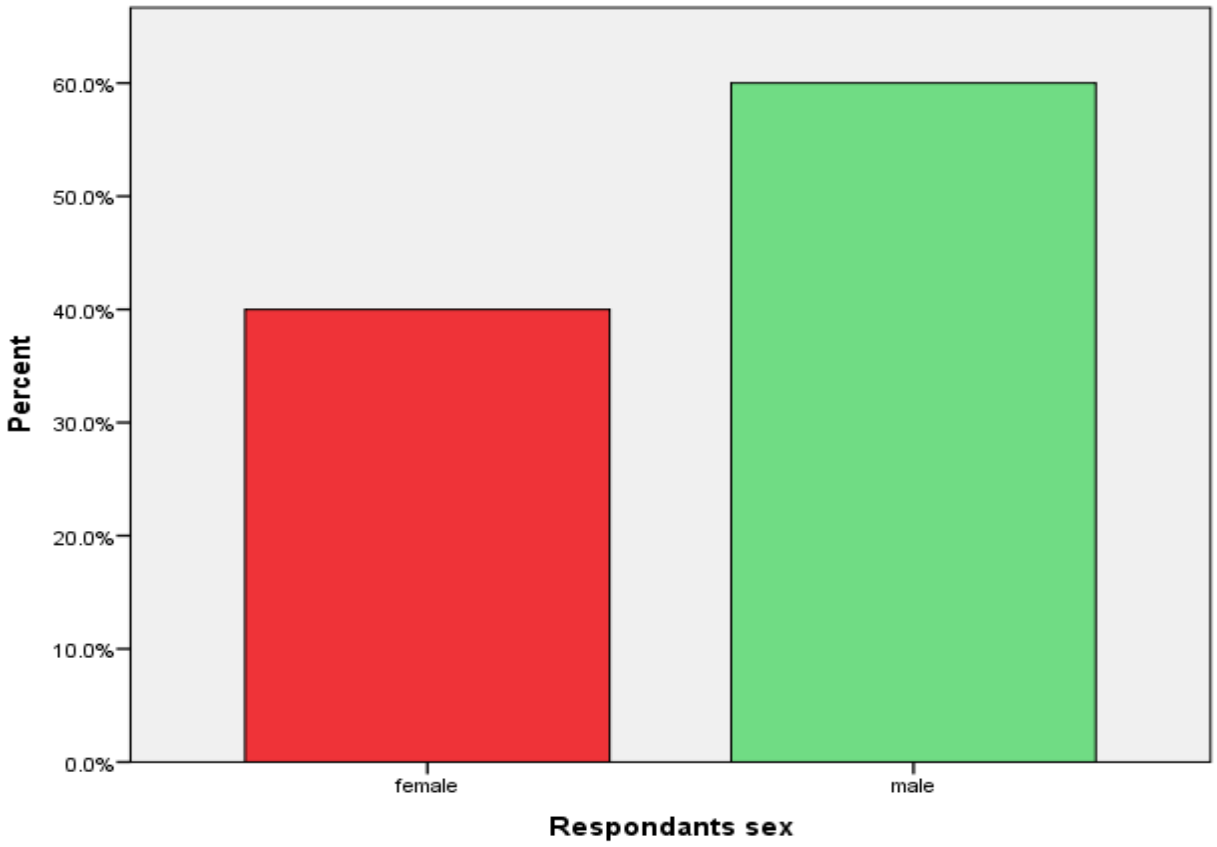
Results:

It states that:

- 56% people belongs to 20-30 years age group
- 40% people belongs to 30-40 years age group
- 4% people belongs to 40 -50 years age group

Figure 2:

Bar chart shows Gender percentage of representing population



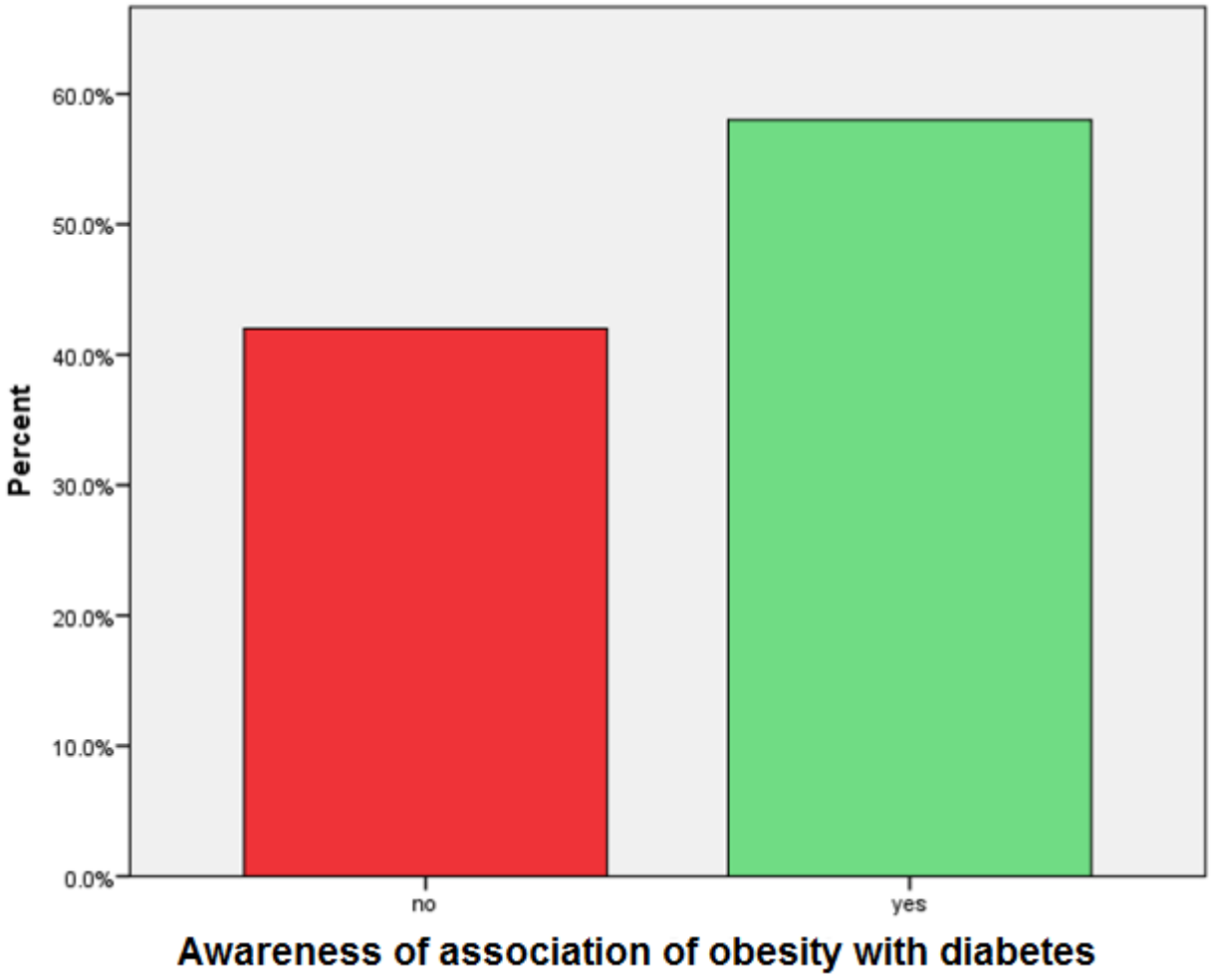
Results:

It states that:

- 40% people were female
- 60% people were male

Figure 3:

Bar chart shows awareness in the community regarding association of obesity with diabetes



Results:

It states that:

- 42% people were not aware of association of obesity with diabetes.
- 58% people were aware of association of obesity with diabetes.

DISCUSSION

- Our sedentary lifestyle is killing us. The human body has evolved into a fat-hoarding machine. Food that our bodies don't immediately use for energy gets quickly stored as fat, and we convert this fat back into fuel for our brains and muscles when food is scarce. This was a great thing for our ancestors who didn't know when their next meal would be. But for us, with our drive-through restaurants and jam-packed junk food aisles, this survival mechanism is wreaking havoc on our health.
- About 72 million Americans — more than one-third of all adults — are obese. The condition substantially increases the risk for a long list of ailments, including heart disease,

stroke, high blood pressure, type 2 diabetes, infertility, Alzheimer's disease and endometrial, breast, prostate and colon cancer.

- The easiest way to determine if you're obese is by calculating your body mass index (BMI), the ratio of your weight to your height. Adults with a BMI of 25 or higher are considered overweight; with a BMI of 30 or higher, they're considered obese. (Get your BMI with our online BMI calculator.)
- Granted, genes play a strong role in determining obesity — some of us store fat more easily and shed it less readily than others, even if we follow the same eating and exercise programs. Environmental factors influence obesity too. Drastic changes in our society over the past 30 years have thrown us into an environment that our bodies were not built for. We spend more time sitting, driving and eating big portions of high-calorie, high-fat food than ever before. And we spend less time being physically active.
- But regardless of the reasons for the rise in obesity, maintaining healthy weight ultimately comes down to balancing the number of calories you eat and drink with the number of calories you burn through exercise and everyday activities. And we all have the power to achieve this balance. Every day we can make choices to eat differently, move our bodies more and enjoy a healthier life.

- Mechanisms of obesity-associated insulin resistance
- The influence of obesity on type 2 diabetes risk is determined not only by the degree of obesity but also by where fat accumulates. Increased upper body fat including visceral adiposity, as reflected in increased abdominal girth or waist-to-hip ratio, is associated with the metabolic syndrome, type 2 diabetes, and cardiovascular disease (3), although underlying mechanisms remain uncertain. Whether subcutaneous fat lacks the pathological effects of visceral fat or is simply a more neutral storage location, for example, requires further study. Beyond differences in body fat distribution, emerging evidence suggests that different subtypes of adipose tissue may be functionally distinct and affect glucose homeostasis differentially. Adult humans have limited and variable numbers of brown fat cells (4), which play a role in thermogenesis and potentially influence energy expenditure and obesity susceptibility (5). Improved understanding of the function of different fat cell types and depots and their roles in metabolic homeostasis is a priority for investigation into the pathogenesis and complications of obesity. Likewise, adipose tissue is composed of heterogeneous cell types. Immune cells within adipose tissue also likely contribute to systemic metabolic processes. As the study of adipose biology progresses, it will be important to consider whether additional subtypes of adipocytes or other cell types can be identified to refine our understanding of obesity complications and generate novel approaches to prevention.

- At least three distinct mechanisms have been proposed to link obesity to insulin resistance and predispose to type 2 diabetes: 1) increased production of adipokines/cytokines, including tumor necrosis factor-α, resistin, and retinol-binding protein 4, that contribute to insulin resistance as well as reduced levels of adiponectin (6); 2) ectopic fat deposition, particularly in the liver and perhaps also in skeletal muscle, and the dysmetabolic sequelae (7); and 3) mitochondrial

dysfunction, evident by decreased mitochondrial mass and/or function (8). Mitochondrial dysfunction could be one of many important underlying defects linking obesity to diabetes, both by decreasing insulin sensitivity and by compromising β-cell function.

- Mechanisms of progressive β-cell dysfunction in obese individuals
- The link between obesity and hyperinsulinemia, first identified ~50 years ago (9), reflects compensation by insulin-secreting β-cells to systemic insulin resistance. Although mechanisms underlying this coupling (e.g., mild hyperglycemia and raised levels of circulating free fatty acids) remain elusive, obese normoglycemic individuals have both increased β-cell mass and function (9–12). Obesity-induced glucose intolerance reflects failure to mount one or more of these compensatory responses (13).
- Factors predisposing to β-cell decompensation could also be primarily genetic or epigenetic. A clear, mechanistic basis for this decompensation has remained elusive. Genetic studies have helped identify the role of some key molecules in β-cell biology that may be important in this regard. For example, recent rodent studies have demonstrated diabetogenic effects of reduced pancreatic expression of the Pdx1 gene (14,15). While these animal studies have demonstrated that PDX1 deficiency relates mechanistically to diabetes through β-cell apoptosis, and PDX1 deficiency is linked to MODY4 (16), it is not clear yet that PDX1 deficiency has a role in common forms of type 2 diabetes in humans. This example illustrates how a growing understanding of genetics and cellular function of the β-cell can identify potential mediators predisposing obese individuals to type 2 diabetes and further may provide insights for the development of new therapeutic agents.
- Genetic factors linking obesity and diabetes
- Genome-wide association scans (GWAS) and candidate gene approaches now have identified ~40 genes associated with type 2 diabetes (17,18) and a similar number, albeit largely different, with obesity. Most type 2 diabetes genes appear to be related to β-cell dysfunction, with many fewer involved in pathways related to insulin resistance independent of obesity (19,20). Not surprisingly, many obesity gene variants appear to be involved in pathways affecting energy homeostasis. Although numerous diabetes- and obesity-associated genes have been identified, the known genes are estimated to predict only 15% of type 2 diabetes and 5% of obesity risk (21). Although additional genes with important roles will undoubtedly be discovered, this low predictive power may reflect the importance of environmental factors, less frequent genetic variants with stronger effects, or gene-environment, gene-gene, and epigenetic interactions that are not readily identified through methods based on population genetics. Methods for detecting gene-gene interactions exist, but the population size needed to detect them is substantially greater than is required for detection of single genes of relatively small effect. Alternatively, pathway analyses or a systems biology approach combining information from DNA variations with transcript, protein, and metabolite profiles may better capture the genetic influences on metabolism than studying single genes. One should also keep in mind that the missing heritability could be

an illusion of inferring additive genetic effects from epidemiological data (22).

- Does a shared pathogenesis underlie both obesity and type 2 diabetes?
- Although the link between obesity and type 2 diabetes is widely held to involve two discrete lesions—obesity-induced insulin resistance and β-cell failure—both disorders may share an underlying defect. This "unified field theory" raises questions about whether defects favoring progressive weight gain and metabolic impairment also contribute to β-cell decompensation.
- One potential link could be sustained cell exposure to nutrient concentrations exceeding energy requirements. Deleterious cellular effects of nutrient excess can include impaired inflammatory signaling, endoplasmic reticulum stress, excess production of reactive oxygen species, mitochondrial dysfunction, accumulation of triglycerides and/or fatty acyl intermediates, and activation of serine-threonine kinases (23). These responses are not mutually exclusive, and induction of one may trigger another, leading to a cascade of damage. Obesity-associated cellular injury can in turn recruit and activate macrophages and other immune cells that exacerbate tissue inflammation (23,24). Collectively, these responses contribute to the pathogenesis of insulin resistance in the liver, skeletal muscle, and adipose tissue, and some (e.g., acquired mitochondrial dysfunction and inflammation) may occur in β-cells as well via mechanisms discussed above. In susceptible individuals, therefore, obesity-induced metabolic impairment can favor insulin resistance on the one hand and progressive β-cell dysfunction on the other. Reduced insulin secretion can in turn worsen the nutrient excess problem by raising circulating concentrations of glucose, free fatty acids, and other nutrients. In this way, a vicious cycle arises whereby obesity-induced nutrient excess triggers inflammatory responses that cause insulin resistance, placing a greater demand on the β-cell, and as β-cell function declines the cellular toll taken by nutrient excess increases. Since not all obese individuals develop hyperglycemia, however, an underlying abnormality of the β-cell must coexist with nutrient excess to promote type 2 diabetes (13).
- Brain neurocircuits governing energy homeostasis also affect insulin sensitivity in the liver and perhaps other peripheral tissues (25), and inflammation similar to that induced by obesity in peripheral insulin-sensitive tissues also occurs in these areas of the brain (26). If obesity is associated with impairment of neurocircuits regulating both energy balance and insulin action, obesity-induced insulin resistance may arise not only as a direct consequence of excessive adipose mass but via neuronal mechanisms as well. Whether disturbed neurocircuits also contribute to deteriorating β-cell dysfunction as obesity and its sequelae progress is an active area of investigation (27).
- Managing body weight by behavioral change and medications
- The dramatic increase in incidence and prevalence of obesity over the past 50 years, associated in part with major worldwide changes in caloric intake and dietary composition, has focused attention on lifestyle intervention to reverse or ameliorate caloric imbalance. In general, programs including individual or group counseling to modify behavior result in 5–10% weight loss and are effective for 6–12 months, after which weight regain is the rule. Some longer-term

lifestyle intervention studies with sustained interventions demonstrate more durable weight loss (28,29), with extent of weight loss in the first 3–6 months generally predicting longer-term success. Successful lifestyle intervention programs typically involve self-monitoring of weight, dietary intake, and activity; behavioral modification; frequent contact; and caloric balance through diet, with or without exercise. For example, short-term intervention studies suggest that dietary changes, which emphasize less fat and refined carbohydrates, make it easier to reduce total caloric intake in obese adults and overweight children (30,31).

- Medications have been used to assist in weight loss for almost 80 years, but adverse effects frequently restrict utility. Medications have been developed based on physiological insights, more recently targeting central nervous system control of appetite and metabolism, or opportunistically when weight loss was noted as a side effect of approved medications. Table 1 lists medications that have been available and others under development. In general, weight loss achieved with these medications ranges from 2 to 8% greater than placebo, with some suggestion that combination therapy may either increase weight loss or ameliorate side effects and increase tolerability. However, most drug trials last only 6–12 months, and thus there are few long-term data that weight loss can be sustained. Moreover, high drop-out rates, which approach 50%, are characteristic of many weight-loss trials and result in survivor effects in efficacy analyses, thereby potentially amplifying drug benefits and limiting generalizability. Furthermore, concern regarding adverse effects, including cardiovascular disease risk and central effects (e.g., depression) in drugs crossing the blood-brain barrier, continue to limit approval and application.
- Managing body weight by bariatric surgery
- Health benefits of bariatric surgery, determined largely from nonrandomized studies, are being increasingly recognized. These benefits include substantial and sustained weight loss (32), resolution of comorbidities such as diabetes, hypertension, and dyslipidemia (33,34), and reduced myocardial infarction, cancers, and associated mortality (35). For extreme obesity, surgery is now the preferred and currently only effective treatment modality. Acute morbidity and mortality of surgical approaches have been dramatically reduced, enabling widespread use of these procedures. Furthermore, over the long term, bariatric surgery might reduce aggregate health care expenditures (36). There is also a growing movement toward using surgery to control diabetes, independent of severe excess weight, but there are currently few scientifically valid data to support this clinical path.
- Bariatric surgery falls into two general categories: purely restrictive procedures such as the laparoscopic adjustable gastric band devices, which appear to improve diabetes via weight loss, and procedures bypassing the proximal gut, such as the Roux-en-Y gastric bypass (RYGB) or newer gastric sleeve procedures. The latter approaches (“metabolic” surgery) appear to produce unique effects on enteroendocrine hormones and neuronal signaling pathways and produce more weight loss and diabetes remission than banding alone (34,37). Metabolic surgeries are associated with increases in anorexigenic and decreases in orexigenic hormones, changes largely absent in band or restrictive

procedures, and may explain the differential outcomes (38). Although mechanisms leading to weight loss and diabetes remission are only beginning to be understood, the above endocrine, peptide, and neural effects may mediate these benefits because of structural changes including isolation of the gastric cardia; exclusion of the distal stomach, duodenum, and proximal jejunum; exposure of the distal intestine to undigested nutrients; and partial vagotomy. Longer duration of diabetes and insulin use, both typically associated with decreased β-cell function and possibly surrogates for reduced β-cell mass, are associated with reduced postsurgical remission rates, thus suggesting that residual β-cell function may be a critical factor for metabolic benefits (39).

- Known differences in mechanism and efficacy, along with risks and patient priorities (e.g., weight loss vs. metabolic/diabetes goals) already inform the choice of surgical procedure. However, many questions remain, including the following: How much weight loss is required for health benefits? What is the effect of different interventional methods on long-term outcomes? What mechanisms underlie the heterogeneous responses? Further, regarding diabetes, Is the optimal timing for treatment the same or different from obesity? Are β-cells preserved or do they even grow? Why do not we see the same efficacy and durability of response for other obesity-related pathologies (e.g., hypertension) as for glycemic control? Ongoing randomized clinical trials (40) promise to answer many questions regarding patient selection, optimal procedure, when to intervene, and where initial and chronic care should be delivered.
- Barriers to effective management
- A vast array of barriers—ranging from deficits in basic research to socioeconomic and individual psychological factors beyond the scope of the conference—undermines current efforts to manage obesity, particularly in individuals with type 2 diabetes. Lessons learned from efforts such as those applied to tobacco cessation may be quite relevant (41).
- Lifestyle programs (especially long-term) are often plagued by inadequate reimbursement. Further, there is a lack of evidence-based individualized goals and strategies combining lifestyle and medications, or appreciation of sequential (stepped) therapy. As mechanisms leading to obesity and its maintenance are not fully understood, questions remain about which interventions, be they lifestyle or pharmacological, might be most effective during various stages of weight gain, loss, and regain. In addition, medications under development may carry indeterminate risk. Likewise, surgery is an imperfect remedy due in part to perceived risks and high cost. With laparoscopic banding now approved for BMI >30 kg/m2 with a comorbidity such as diabetes or hypertension, 27 million Americans would be eligible for surgery. However, the large-scale feasibility of such an approach is uncertain and compounded by issues related to reimbursement. Thus, the search must continue for how to implement optimal lifestyle interventions and to find effective drugs and/or minimally invasive devices.
- These barriers are further complicated in the context of type 2 diabetes. Obese patients with hyperglycemia are often poorly characterized not only in terms of their history of obesity but also in the duration of their glucose intolerance. Further, interventions are typically started late in the disease, with

minimal preventive efforts. In addition, as initial weight loss is the main determinant of longer-term weight loss, the typical initial goal of ~5–10% weight loss may be inadequate to produce glycemic control (42). Furthermore, although controlling body weight (either by reduction or by prevention of further rise) improves glycemic control by ameliorating both insulin resistance and β-cell dysfunction, the impact of pharmacologically induced improved glycemic control on body weight varies by individual drug. Glucose-lowering medications can be broadly categorized into those associated with weight gain and those essentially weight neutral or promoting weight loss (Table 2). Whether weight gain offsets any benefit of reduced glycemia on cardiovascular risk needs to be determined. Further, weight changes do not necessarily predict changes in glycemic control (43), and while specific therapies may work in certain diabetes subtypes, the response to glucose-lowering medications varies considerably. This latter topic was the focus of a similar workshop in 2009 on individualizing therapies in type 2 diabetes (44).

- Equally challenging is the problem of weight regain, which usually follows any degree of weight loss, however achieved (Fig. 1). Well studied and viewed as a normal response in lean individuals, this phenomenon is equally robust among the obese. It involves complex, highly integrated physiological responses that are similar to those invoked in weight-reduced, nonobese individuals. The biologic basis appears to be the tendency to defend attained weight, whether normal or excessive, which seems to be wired in multiple central nervous system defenses against weight loss. Current models of energy homeostasis predict genetic or acquired defects in key neurocircuits that undermine the normal response to adiposity-related humoral signals. Much of the basic science in this area has been performed in animal models of obesity (genetic or overfeeding); extrapolation to the pathophysiology of human obesity remains uncertain.

CONCLUSION

Increase in association between obesity and diabetes among adults of Pakistan continue in both sexes, all ages, all races, all educational levels and all smoking levels. Obesity is strongly associated with several major health risk factors.

Obesity and diabetes are major causes of morbidity and mortality in the Pakistan. Evidence from several studies indicates that obesity and weight gain are associated with an increased risk of diabetes and that intentional weight loss reduces the risk that overweight people will develop diabetes. Each year, an estimated 1,00,000 adults of Pakistan die because of causes related to obesity and diabetes is the sixth leading cause of death.

Correspondingly, both obesity and diabetes generate immense health care costs.

We recently reported that the prevalence of obesity and diabetes among adults of Pakistan has increased substantially from 2000 to 2016. Research indicates that people living in large cities in Pakistan are more exposed to the risks of obesity and diabetes as compared to those in the rural countryside. In addition, we examined the association between obesity and several other important health risk factors, as well as self-rated general health.

Recommendations

- Diet, physical activity, and behavioural therapy designed to achieve weight loss should be prescribed for overweight and obese patients with type 2 diabetes ready to achieve weight loss.
- Limit saturated fat to 7% of total calories, intake of fats should be minimized, dietary cholesterol should be limited to 200mg/day.
- Such interventions should be high intensity and focus on diet, physical activity, and behavioural strategies to achieve a 500–750 kcal/day energy deficit.
- Diets that provide the same caloric restriction but differ in protein, carbohydrate, and fat content are equally effective in achieving weight loss.
- For patients who achieve short-term weight loss goals, long-term comprehensive weight maintenance programs should be prescribed. Such programs should provide at least monthly contact and encourage on going monitoring of body weight (weekly or more frequently), continued consumption of a reduced calorie diet, and participation in high levels of physical activity.
- To achieve weight loss of >5%, short-term (3-month) high-intensity lifestyle interventions that use very low-calorie diets (≤800 kcal/day) and total meal replacements may be prescribed for carefully selected patients by trained practitioners in medical care settings with close medical monitoring. To maintain weight loss, such programs must incorporate long-term comprehensive weight maintenance counselling.
- Glucose-lowering medications for overweight or obese patients with type 2 diabetes
- Whenever possible, minimize the medications for comorbid conditions that are associated with weight gain.
- Weight loss medications may be effective as adjuncts to diet, physical activity, and behavioural counselling for selected patients with type 2 diabetes and BMI ≥27 kg/m^2. Potential benefits must be weighed against the potential risks of the medications.
- If a patient's response to weight loss medications is <5% after 3 months or if there are any safety or tolerability issues at any time, the medication should be discontinued and alternative medications or treatment approaches should be considered.

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Annexure

Consent Form

I……D/O S/O…… give consent to take part in research without any pressure. I am fully aware of the fact that this research is only for educational purposes which will be helpful for the betterment of my health and that of the community and the nation at large. It has also been made clear to me that my information and identity will be kept secret. I am ready to contribute and provide answers to questions.

Signature……

Questionnaire

Assessment of association of obesity with diabetes mellitus among general population of Lahore

Consent: _____

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Bio data:

Name: _____

Age: _____

Sex: _____

Occupation: _____

Education: _____

Residence: _____

Religion: Muslim/Non -Muslim

Knowledge:

1) Do you know about obesity? Yes/No

2) Do you know about Diabetes mellitus? Yes/No

3) Do you know about the association of obesity with Diabetes? Yes/No

4) Do you know, it runs in the family? Yes/No

5) Do you have any diabetic person in your family? Yes/No

6) Do you have any obese person in your family? Yes/No

7) Do you know about the complications of Obesity? Yes/No

8) Do you know about the complications of Diabetes? Yes/No

9) Do you engage in regular physical activity? Yes/No

10) Do you participate in any kind of sports? Yes/No

11) Are you in the habit of taking morning walk daily? Yes/No

12) Have you ever checked your sugar level? Yes/No

13) Have you ever calculated your BMI? Yes/No

14) Do you think that you are overweight? Yes/No

15) Is there any family history of diabetes in your family? Yes/No