A STUDY ON PROFILE OF DIETARY STATUS, BMI AND PHYSICAL ACTIVITY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS

BY

Dr. SUMIT GARG, MBBS

DISSERTATION SUBMITTED TO

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH,

TAMAKA, KOLAR, KARNATAKA

IN PARTIAL FULFILLMENT

OF THE REQUIREMENTS FOR THE DEGREE OF

DOCTOR OF MEDICINE
IN
PHYSIOLOGY

Under the guidance of

DR. VINUTHA SHANKAR.M.S. MD

DEPARTMENT OF PHYSIOLOGY
SRI DEVARAJ URS MEDICAL COLLEGE, KOLAR

2012

SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION & RESEARCH,

TAMAKA, KOLAR, KARNATAKA.

DECLARATION BY THE CANDIDATE

I HEREBY DECLARE THAT THIS DISSERTATION/THESIS ENTITLED

"A STUDY ON PROFILE OF DIETARY STATUS, BMI AND PHYSICAL ACTIVITY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"

IS A BONAFIDE AND GENUINE RESEARCH WORK CARRIED OUT BY ME UNDER THE DIRECT GUIDANCE OF

DR.VINUTHA SHANKAR.M.S, MD

PROFESSOR, DEPARTMENT OF PHYSIOLOGY,

SRI DEVARAJ URS MEDICAL COLLEGE, KOLAR

DATE:

PLACE: KOLAR

SIGNATURE OF THE CANDIDATE

DR. SUMIT GARG

CERTIFICATE BY THE GUIDE

THIS IS TO CERTIFY THAT THE DISSERTATION ENTITLED

"A STUDY ON PROFILE OF DIETARY STATUS, BMI AND PHYSICAL ACTIVITY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"

IS A BONAFIDE RESEARCH WORK DONE

BY

DR. SUMIT GARG

IN PARTIAL FULFILLMENT OF THE REQUIREMENT FOR THE DEGREE OF

M.D IN PHYSIOLOGY

DATE:

PLACE:

SIGNATURE OF THE GUIDE

DR.VINUTHA SHANKAR.M.S.MD
PROFESSOR
DEPARTMENT OF PHYSIOLOGY

ENDORSEMENT BY THE HOD, PRINCIPAL/HEAD OF THE INSTITUTION

THIS IS TO CERTIFY THAT THE DISSERTATION ENTITLED

"A STUDY ON PROFILE OF DIETARY STATUS, BMI AND PHYSICAL ACTIVITY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"

IS A BONAFIDE RESEARCH WORK DONE

BY

Dr. SUMIT GARG

UNDER THE GUIDANCE OF

Dr. VINUTHA SHANKAR.M.S,

PROFESSOR,

DEPARTMENT OF PHYSIOLOGY.

SEAL & SIGNATURE OF THE HOD

Dr.KARTHIYANEE KUTTY

DATE:
PLACE: KOLAR

SEAL & SIGNATURE OF THE PRINCIPAL

DR. M.B.SANIKOP

DATE:
PLACE:KOLAR

COPYRIGHT

DECLARATION BY THE CANDIDATE

I HEREBY DECLARE THAT

SRI DEVARAJ URS UNIVERSITY, TAMAKA, KOLAR, KARNATAKA

SHALL HAVE THE RIGHTS TO PRESERVE,

USE AND DISSEMINATE THIS DISSERTATION / THESIS

IN PRINT OR ELECTRONIC FORMAT FOR ACADEMIC / RESEARCH PURPOSE.

DATE:

PLACE: KOLAR

SIGNATURE OF THE CANDIDATE

DR. SUMIT GARG

SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR.

ETHICAL COMMITTEE

CERTIFICATE

THIS IS TO CERTIFY THAT, THE ETHICAL COMMITTEE OF SRI DEVARAJ URS MEDICAL COLLEGE, TAMAKA, KOLAR HAS UNANIMOUSLY APPROVED

DR. SUMIT GARG,

POST GRADUATE STUDENT IN THE DEPARTMENT OF PHYSIOLOGY OF SRI DEVARAJ URS MEDICAL COLLEGE TO TAKE UP THE DISSERTATION WORK ENTITLED

"A STUDY ON PROFILE OF DIETARY STATUS, BMI AND PHYSICAL ACTIVITY IN PATIENTS WITH TYPE 2 DIABETES MELLITUS"

TO BE SUBMITTED TO THE
SRI DEVARAJ URS ACADEMY OF HIGHER EDUCATION AND RESEARCH,

TAMAKA, KOLAR.

MEMBER SECRETARY **PRINCIPAL**

ACKNOWLEDGEMENT

It is a pleasure to thank the people who made this thesis possible. I thank each and everyone for the knowledge they imparted to me & for the time they graciously gave me.

I wish to express my deepest gratitude to Dr. Vinutha Shankar, Professor, Department of Physiology, SDUMC, Kolar at the beginning of this study, who kindly suggested the subject of this study to me,who has the substance of a genius and who has been my teacher and supervisor in scientific work. I thank her for her excellent and tireless guidance, encouragement and patience throughout the years of the study.

No words can express my gratitude to Dr. Karthiyanee Kutty, Professor & HOD, Department of Physiology, who continually and convincely conveyed a spirit of adventure in regard to research. She showed and maintained a constant and most active interest during the entire period of this work. It was a pleasure to be associated with her contagious enthusiasm, persuasive and stimulating personality.

I also thank Dr. Sunanda Nayak., Professor, Department of Physiology, for having been a source of inspiration. I am very fortunate to obtain benefit of her vast experience, valuable guidance, sincere suggestion, keen interest and constant encouragement at each step throughout my post-graduate career.

My special thanks to Dr. Jagadamba, Assistant Professor, Department of Physiology for helping me in collecting samples. I am grateful to Mrs. Usha Shenoy and Dr. Raja Reddy, Assistant Professor, Department of Physiology for their invaluable guidance, kind support, co-operation and timely advice.

I render my hearty thanks to Dr. Deepthi Kiran, Assistant Professor, Department of Community Medicine, SDUMC & Dr. Ravi Shankar, Statistician, SDUMC for helping me in statistical analysis.

I wish to express my deepest gratitude to Dr. Kavana G Venkatappa, Dr. Shankarappa, Dr. Padmini, Dr. Ashwini, Dr. Soumya , my seniors for their excellent guidance.

I remain very grateful to the support and help rendered by my dear friends and colleagues Dr. Ramya and Dr. Bhanu, Dr. Ashwini Priyanka, Dr. Sindhu and Dr. Vineetha for their constant support and encouragement throughout.

I express my sincere heart felt gratitude to all the subjects for giving their willingness to participate in this study.

I thank all non teaching staff of the department for their kind co-operation.

Above all I am proud of my parents Dr. Mukesh Kumar and Dr. Nirmal, my grandmother Smt Sarala Devi, my brother and sister in law, Dr. Rohit Garg and Dr. Priyanka, my niece and nephew Parnika Garg and Satwik Garg for their constant encouragement, blessings, love & attention which have made me accomplish this work. They have formed the basis of this work without which it would not have seen the light of the day.

In the end, I thank The Almighty for the blessing for the fulfillment of this work.

Date:

Place: Kolar *Dr. Sumit Garg*

LIST OF ABBREVIATIONS

DM	-	Diabetes Mellitus
T2DM	-	Type 2 Diabetes Mellitus
BMI	-	Body mass index
IGT	-	Impaired glucose tolerance
WHO	-	World Health Organisation
MET	-	Metabolic Equivalent
PAL	-	Physical activity level
GLUT	-	Glucose transporter
BMR	-	Basal metabolic rate
GI	-	Glycemic index
GL	-	Glycemic load
WC	-	Waist circumference

ABSTRACT

Background and objectives:

Diabetes, a major non-communicable disorder, is a life long and life altering illness with no proven cures and demands only management. India leads the world in diabetes. This increased prevalence of Type 2 diabetes mellitus can be attributed to increased prevalence of overweight and obesity and lifestyle changes. It is already known that high proportion of body fat is associated with insulin resistance.

In addition to conventional treatment, life style modification including diet and physical exercise has also been accepted as cornerstone of treatment for diabetic patients as it helps to maintain euglycemic levels and hence reduce the associated complications and expenditure.

Rural population is generally unaware about the importance of diet and physical activity in control of DM. There is a need to evaluate the dietary pattern of these diabetic patients in order to provide appropriate information to plan a proper diet which includes locally available food items in right proportions. This study has been designed to assess the dietary status and physical activity profile in diabetic patients, so as to implement the necessary dietary and exercise interventions to control DM.

The aim of the study was to calculate body mass index (BMI), assess dietary status and determine the physical activity profile in patients with type 2 Diabetes Mellitus.

Materials & Methods:

Three hundred patients with diagnosed T2DM without any obvious complications were selected considering the inclusion and exclusion criteria. Their BMI (kg/m^2) was

calculated. Dietary intake was determined by dietary recall. Total calorie and protein intake were estimated for calculation of calorie and protein deficiency or excess after noting the recommended daily allowance for that age. Physical Activity profile was studied using standardized questionnaire and average duration of the activity per day was calculated. The resulting data was statistically analysed.

Results:

65% of the subjects had high BMI. 68% had sedentary lifestyle. 35% of the subjects were consuming excess calories and 65% were having deficient calories intake and 83% were deficient in daily protein intake. Excess calories intake was present in 61% of subjects in high BMI group. Sedentary lifestyle was observed in 73.3% of the subjects having high BMI and 89.6% of the subjects taking excess calories.

Conclusion:

Sedentary lifestyle and high BMI were more commonly observed in the diabetic patients studied. Awareness regarding lifestyle modification with respect to diet, physical activity and weight control should be created among diabetic patients by counseling and educative programs. Dietary prescription should be made keeping in mind the presence of pre existing calorie and protein deficiency.

Key words: Type 2 diabetes mellitus, BMI, Diet, Physical activity.

CONTENTS

LIST OF TABLES

LIST OF GRAPHS

LIST OF FIGURES

INTRODUCTION

INTRODUCTION

Diabetes, a major non-communicable disorder, is a life long and life altering illness with no proven cures and demands only management. India leads the world in diabetes prevalence with 61.3 million diabetics estimated to rise to 101.2 million by 2030 only behind China and hence the allied complications are expected to find at their peaks in the country with a serious trend of westernized life style aggravating the ethnic susceptibility. [1]

This increased prevalence of Type 2 diabetes mellitus (T2DM) can be attributed to increased prevalence of overweight and obesity and lifestyle changes. It is already known that high proportion of body fat is associated with insulin resistance. People in South Asia tend to develop diabetes with a lesser degree of obesity at younger ages and suffer longer with complications of diabetes due to ethnic susceptibility. Moreover rapid economic developments have improved the availability of nutrients, specifically an energy dense diet in these countries predisposing people to both obesity and type 2 diabetes. Furthermore, with rapid shifts in income, changes in occupation linked with reduced physical activity have added to rise in obesity. [2]

The increased prevalence of impaired glucose tolerance (IGT) and diabetes in urban part of India has been documented. Rural sector is fastly catching up with the urban trends due to rapid industrialization and urbanization. [3,4]

In addition to conventional treatment, life style modification including diet and physical exercise has also been accepted as cornerstone of treatment for diabetic patients as it helps to maintain euglycemic levels and hence reduce the associated complications and expenditure.

Rural population is generally unaware about the importance of diet and physical activity in control of DM. There is a need to evaluate the dietary pattern of these diabetic patients in order to provide appropriate information to plan a proper diet which includes locally available food items in right proportions. This study has been designed to assess the dietary status and physical activity profile in diabetic patients, so as to implement the necessary dietary and exercise interventions to control DM.

AIMS AND OBJECTIVES

AIMS AND OBJECTIVES OF THE STUDY

1. To calculate body mass index (BMI) to determine obesity in patients with type 2 Diabetes Mellitus.
2. To assess their dietary status using dietary recall.
3. To determine their physical activity profile using standard questionnaire.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

A. History of DM [5,6]

1552 B.C.	• Earliest known record of diabetes mentioned on 3rd Dynasty Egyptian papyrus by physician Hesy-Ra; mentions polyuria (frequent urination) as a symptom.
Up to 11th Century	• Diabetes commonly diagnosed by 'water tasters,' who drank the urine of those suspected of having diabetes; the urine of people with diabetes was thought to be sweet-tasting. The Latin word for honey (referring to its sweetness), 'mellitus', is added to the term diabetes as a result.
1870s	• French physician, Bouchardat, notices the disappearance of glycosuria in his diabetes patients during the rationing of food in Paris while under siege by Germany during the Franco-Prussian War; formulates idea of individualized diets for his diabetes patients
19th Century	• French researcher, Claude Bernard, studies the workings of the pancreas and the glycogen metabolism of the liver.
1900-	'Fad' diabetes diets include: the 'oat-cure' (in which the majority

1915	of diet was made up of oatmeal), the milk diet, the rice cure.
1910-1920	• Frederick Madison Allen and Elliot P. Joslin emerge as the two leading diabetes specialists in the United States. Joslin believes diabetes to be 'the best of the chronic diseases' because it was 'clean, seldom unsightly, not contagious, often painless and susceptible to treatment.'
1919	• Frederick Allen publishes Total Dietary Regulation in the Treatment of Diabetes.
October 31, 1920	• Dr. Banting conceives of the idea of insulin after reading Moses Barron's 'The Relation of the Islets of Langerhans to Diabetes with Special Reference to Cases of Pancreatic Lithiasis' in the November issue of Surgery, Gynecology and Obstetrics. For the next year, with the assistance of Best, Collip and Macleod, Dr. Banting continues his research using a variety of different extracts on de-pancreatized dogs.
1921	• Insulin is 'discovered'. A de-pancreatized dog is successfully treated with insulin.
1955	• Oral drugs are introduced to help lower blood glucose levels.

1959

- Two major types of diabetes are recognized: type 1 (insulin-dependent) and type 2 (non-insulin-dependent) diabetes.

1983

- First biosynthetic human insulin is introduced.

1993

- Diabetes Control and Complications Trial (DCCT) report is published. The DCCT results clearly demonstrate that intensive therapy (more frequent doses and self-adjustment according to individual activity and eating patterns) delays the onset and progression of long-term complications in individuals with type 1 diabetes.

1998 The United Kingdom Prospective Diabetes Study (UKPDS) is published. UKPDS results clearly identify the importance of good glucose control and good blood pressure control in the delay and/or prevention of complications in type 2 diabetes.Relation of diet and exercise to the rate of development of type 2 diabetes in high risk population was established in Diabetes Prevention program, USA.

2006 The United Nations recognizes diabetes as a global threat and designates World Diabetes Day, November 14.

India leads the world in diabetes prevalence and hence, the allied complications are expected to find at their peaks in the country with a serious trend of westernized life style aggravating the ethnic susceptibility.

B. ETIOLOGIC CLASSIFICATION OF DM [7]

I. Type 1 diabetes (beta cell destruction, usually leading to absolute insulin deficiency) A. Immune-mediated B. Idiopathic
II. Type 2 diabetes (may range from predominantly insulin resistance with relative insulin deficiency to a predominantly insulin secretory defect with insulin resistance)
III. Other specific types of diabetes A. Genetic defects of beta cell function characterized by mutations in: 1. Hepatocyte nuclear transcription factor (HNF) 4α (MODY 1) 2. Glucokinase (MODY 2) 3. HNF-1α (MODY 3) 4. Insulin promoter factor-1 (IPF-1; MODY 4) B. Genetic defects in insulin action 1. Type A insulin resistance 2. Leprechaunism C. Diseases of the exocrine pancreas—pancreatitis, pancreatectomy, neoplasia D. Endocrinopathies—acromegaly, Cushing's syndrome, glucagonoma, hyperthyroidism, E. Drug- or chemical-induced—glucocorticoids, β-adrenergic agonists F. Infections—congenital rubella, cytomegalovirus, coxsackievirus G. Uncommon forms of immune-mediated diabetes— "stiff-person" syndrome, anti-insulin receptor antibodies H. Other genetic syndromes sometimes associated with diabetes
IV. Gestational diabetes mellitus (GDM)

Among these T2DM is the most common type and accounts for 90–95% of those with diabetes, previously referred to as non–insulin dependent diabetes or adult-onset diabetes,

encompasses individuals who have insulin resistance and usually have relative (rather than absolute) insulin deficiency. Most patients with this form of diabetes are obese, and obesity itself causes some degree of insulin resistance.[8]

C. DIAGNOSTIC CRITERIA FOR DM

In 1979 and 1980, two groups, the National Diabetes Data Group in the United States and the World Health Organization (WHO), published reports addressing diabetes diagnostic criteria.[9]

Subsequently, in 1997 and 1999, two new reports on diabetes diagnostic criteria were published. The first was sponsored by the American Diabetes Association (ADA) and was the report of its Expert Committee on the Diagnosis and Classification of Diabetes Mellitus. The second was from WHO.[10] In 2003, the Expert Committee published a follow-up report in which it carefully considered new data since its 1997 report.[8]

	YEAR OF REPORTS		
CRITERIA	1979 & 1980	1997 & 1999	2003
Fasting plasma glucose			
Diabetes	$\geq$ 140 mg/dl	$\geq$ 126 mg/dl	$\geq$ 126 mg/dl
Impaired fasting glucose	Not considered	110 - 125 mg/dl	100 – 125 mg/dl
2 hour plasma glucose			
Diabetes	$\geq$ 200 mg/dl	$\geq$ 200 mg/dl	$\geq$ 200 mg/dl
Impaired fasting glucose	140 - 199 mg/dl	140 - 199 mg/dl	140 - 199 mg/dl

D. PATHOPHYSIOLOGY OF DIABETES [7]

Type 2 DM is characterized by impaired insulin secretion, insulin resistance, excessive hepatic glucose production, and abnormal fat metabolism. Obesity is very common in type 2 DM. In the early stages of the disorder, glucose tolerance remains near-normal, despite insulin resistance, because the pancreatic beta cells compensate by increasing insulin output. As insulin resistance and compensatory hyperinsulinemia progress, the pancreatic islets in certain individuals are unable to sustain the hyperinsulinemic state. Impaired glucose tolerance (IGT), characterized by elevations in postprandial glucose, then develops. A further decline in insulin secretion and an increase in hepatic glucose production lead to overt diabetes with fasting hyperglycemia. Ultimately, beta cell failure ensues.

Abnormal Muscle and Fat Metabolism

Insulin resistance, the decreased ability of insulin to act effectively on target tissues (especially muscle, liver, and fat), results from a combination of genetic susceptibility and obesity. Insulin resistance is relative, however, since supranormal levels of circulating insulin will normalize the plasma glucose. Insulin dose-response curves exhibit a rightward shift, indicating reduced sensitivity, and a reduced maximal response, indicating an overall decrease in maximum glucose utilization (30–60% lower than in normal individuals). Insulin resistance impairs glucose utilization by insulin-sensitive tissues and increases hepatic glucose output; both effects contribute to the hyperglycemia. Increased hepatic glucose output predominantly accounts for increased fasting plasma glucose levels, whereas decreased peripheral glucose usage results in postprandial hyperglycemia. In skeletal muscle, there is a greater impairment in

non oxidative glucose usage (glycogen formation) than in oxidative glucose metabolism through glycolysis. Glucose metabolism in insulin-independent tissues is not altered in type 2 DM.

The precise molecular mechanism leading to insulin resistance in type 2 DM has not been elucidated. Insulin receptor levels and tyrosine kinase activity in skeletal muscle are reduced, but these alterations are most likely secondary to hyperinsulinemia and are not a primary defect. Therefore, "postreceptor" defects in insulin-regulated phosphorylation/dephosphorylation appear to play the predominant role in insulin resistance. For example, a PI-3-kinase signaling defect might reduce translocation of GLUT4 to the plasma membrane. Other abnormalities include the accumulation of lipid within skeletal myocytes, which may impair mitochondrial oxidative phosphorylation and reduce insulin-stimulated mitochondrial ATP production. Impaired fatty acid oxidation and lipid accumulation within skeletal myocytes also may generate reactive oxygen species such as lipid peroxides.

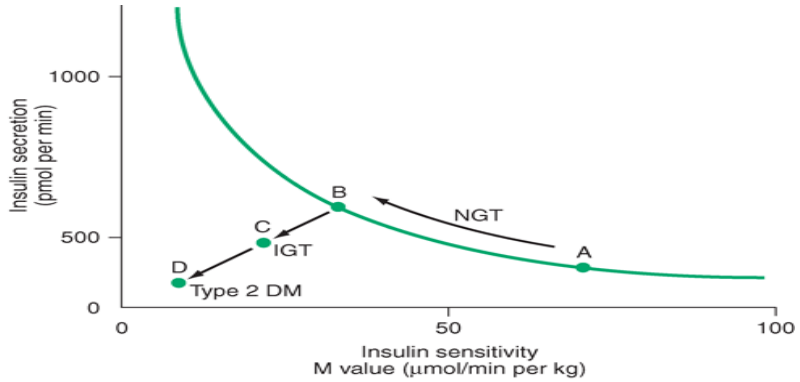
Impaired Insulin Secretion

Insulin secretion and sensitivity are interrelated (Figure 1). In type 2 DM, insulin secretion initially increases in response to insulin resistance to maintain normal glucose tolerance. Initially, the insulin secretary defect is mild and selectively involves glucose-stimulated insulin secretion. Abnormalities in proinsulin processing are reflected by increased secretion of proinsulin in type 2 DM. Eventually, the insulin secretary defect progresses to a state of inadequate insulin secretion.

The reason for the decline in insulin secretary capacity in type 2 DM is unclear. The assumption is that a second genetic defect—superimposed upon insulin resistance—leads to beta cell failure. Beta cell mass is decreased in individuals with long-standing type 2 diabetes. Islet

amyloid polypeptide or amylin is co-secreted by the beta cell and forms the amyloid fibrillar deposit found in the islets of individuals with long-standing type 2 DM. The metabolic environment of diabetes may also negatively impact islet function. For example, chronic hyperglycemia paradoxically impairs islet function ("glucose toxicity") and leads to a worsening of hyperglycemia. Improvement in glycemic control is often associated with improved islet function. In addition, elevation of free fatty acid levels ("lipotoxicity") and dietary fat may also worsen islet function.

Figure 1: Relation between Insulin secretion and sensitivity.



Increased Hepatic Glucose and Lipid Production

In type 2 DM, insulin resistance in the liver reflects the failure of hyperinsulinemia to suppress gluconeogenesis, which results in fasting hyperglycemia and decreased glycogen storage by the liver in the postprandial state. Increased hepatic glucose production occurs early in the course of diabetes, though likely after the onset of insulin secretary abnormalities and insulin resistance in skeletal muscle. As a result of insulin resistance in adipose tissue, lipolysis and free fatty acid flux from adipocytes are increased, leading to increased lipid [very low

density lipoprotein (VLDL) and triglyceride] synthesis in hepatocytes. This lipid storage or steatosis in the liver may lead to nonalcoholic fatty liver disease and abnormal liver function tests. This is also responsible for the dyslipidemia found in type 2 DM [elevated triglycerides, reduced high-density lipoprotein (HDL), and increased small dense low-density lipoprotein (LDL) particles].

E. OBESITY AND INSULIN RESISTANCE

Obesity is a major determinant of type 2 diabetes, and is associated with many metabolic aberrations that impair insulin sensitivity. These abnormalities include excess lipolysis causing increased concentrations of non-esterified fatty acids and triglycerides in blood and skeletal muscle. Glucose uptake by muscle is suppressed.[11]

Obesity is the main risk factor for type 2 diabetes apart from other environmental and genetic factors. However all obese people are not diabetic. An adipocyte-hormone called Resistin is known to provide link between obesity and diabetes", Resistin is a peptide hormone produced by adipocytes and regulates insulin sensitivity. It is more highly expressed in omental and abdominal subcutaneous white fat than in adipose tissue from the thigh or breast. It was initially isolated as an mRNA whose expression is suppressed in response to rosiglitazone. A significant correlation has been observed between resistin level and insulin resistance even after correcting for gender and BMI as measured by the HOMA-R technique (used for assessing Insulin levels and insulin resistance). [12]

The hormone leptin is secreted from white adipocytes, and serum levels of leptin correlate with adipose tissue mass. Leptin was first described to act on the satiety center in the hypothalamus through specific receptors (leptin receptor [ObR]) to restrict food intake and enhance energy expenditure. Important peripheral actions of leptin involve inhibition of insulin

biosynthesis and secretion in pancreatic cells. In turn, insulin stimulates leptin secretion from adipose tissue, establishing a hormonal regulatory feedback loop—the so called "adipo-insular axis." Multiple signal transduction pathways are involved in leptin signaling in pancreatic β cells. The proinsulin gene and protein phosphatase 1 gene has been identified as leptin repressed genes and the gene for the suppressor of cytokine signaling 3 proteins as a leptin-induced gene in pancreatic β cells. The molecular effects of leptin culminate to restrict insulin secretion and biosynthesis to adapt glucose homeostasis to the amount of body fat. In most overweight individuals, however, physiological regulation of body weight by leptin seems to be disturbed, representing "leptin resistance." This leptin resistance at the level of the pancreatic β cell may contribute to dysregulation of the adipo-insular axis and promote the development of hyperinsulinemia and manifest T2DM in overweight patients. [13]

BMI increases the risk of developing T2DM increases in a "dose-dependent" manner. The prevalence of type 2 diabetes is 3–7 times higher in obese than in normal-weight adults, and those with a BMI > 35 kg/m^2 are 20 times more likely to develop diabetes than those with a BMI between 18.5 and 24.9 kg/m^2. In addition, weight gain during adulthood is also directly correlated with an increased risk of type 2 diabetes. Obesity also complicates the management of type 2 diabetes by increasing insulin resistance and blood glucose concentrations. It is an independent risk factor for dyslipidemia, hypertension, and cardiovascular disease and thus, increases the risk of cardiovascular complications and cardiovascular mortality in patients with type 2 diabetes. [14]

There was an association between increased BMI or waist circumference (WC) and risk of glucose intolerance among older people by adjusting for the effect of physical activity and an

independent and inverse association between physical activity and the presence of glucose intolerance. [15]

Obesity in the Asian Indian population

Asian populations are multiracial and have multifactorial causes of type 2 diabetes. The mechanisms underlying development of the disease are complex and varied, even within these populations. The major etiological components of type 2 diabetes are impaired insulin secretion and impaired insulin action, which are aggravated by the presence and degree of glucotoxicity. Both components might also be genetically predetermined.[11]

Lipotoxicity plays an important part in causing insulin resistance and β-cell damage. In the natural history of type 2 diabetes, β-cell function undergoes a series of changes. With the development of obesity and other adverse effects on insulin sensitivity, β cells respond with compensatory hyperinsulinemia. Such changes are seen even in non diabetic people with strong familial history of diabetes. With increasing duration, β-cell function declines and insulin to glucose ratio diminishes, before an ultimate decompensation occurs with expression of clinical diabetes. Asian populations are more insulin resistant than are people of many other races. Insulin resistance and compensatory hyperinsulinemia are reported even in children and adolescents of Asian Indian origin. These factors probably play a major part in the escalating prevalence of type 2 diabetes in young populations in Asia.[11]

Obesity also impairs insulin action by changing secretion of cytokines, specifically of leptin and adiponectin, and leads to proinflammatory conditions. Features of insulin resistance, including hypertriglyceridemia and increased abdominal or visceral fat, are seen even in non-obese Asian populations. [11]

A study done on 900,000 adults from seven Asian countries confirmed the strong relationship between increased body mass index and prevalence of diabetes. The association between BMI and diabetes prevalence was stronger among younger than among older individuals. There are several possible explanations for this pattern: First, genetic factors could play a more important role in those with earlier than later onset of diabetes, and the stronger association in younger adults could be due, in part, to joint effects of high BMI and such genetic factors. Second, the stronger association in younger adults may be due to a shorter latency of the effect of substantial weight gain, which is more likely to have occurred in those of younger age.[16] Asian people generally have a lower body-mass index than do people of many other races, but the association between body-mass index and glucose intolerance is as strong as in any other population. Asian populations seem to differ from European populations in associations between body-mass index and percentage of body fat and health risks. On the basis of the evidence, a WHO expert consultation concluded that a substantially increased risk of type 2 diabetes and cardiovascular diseases occurs at body-mass index lower than 25 kg/m^2.[11]

The definition of the cut off value for "normal" BMI in a population would depend on identifying the risk association with a disorder strongly associated with BMI. In view of the high prevalence of diabetes and also its strong independent association with BMI in Asian Indians, it may be appropriate to use this association to derive the normal cut off value for BMI. Asian Indians have higher upper-body adiposity and higher visceral fat for a given BMI when compared with the Western population. In Asian Indian population, an interaction between upper-body adiposity and general adiposity increased the risk at lower tertiles of BMI in both men and women. The healthy BMI for an Indian is <23 kg/m^2. The risk of diabetes was significant at BMI >23 kg/m^2 for Indians of both sexes.[17,18]

Prevalence of obesity was highest for urban population (male = 5.5%, female = 12.6%) followed by urban slum (male = 1.9%, female = 7.2%) and rural populations (male = 1.6%, female = 3.8%). Urbanization increases the prevalence of non-communicable disease risk factors, with women showing a greater increase as compared with men. [19]

For a given BMI or WC, South Asian men had approximately 6% higher total body fat than Caucasian men. The threshold for obesity in South Asians is approximately 2 kg/m^2 lower than in Caucasians. A WC of 10 cm lower in South Asians confers a similar estimate of % body fat. These discrepancies may be indicative of body fat content, and adjustments required for BMI or waist circumference to define obesity do not entirely account for possible differences in inherent insulin resistance in the South Asian population. Truncal skinfolds thicknesses were higher in South Asians. This implies that South Asians show a preferential accumulation in truncal fat over gluteofemoral fat. This finding is consistent with reports, which have also shown a strong relationship between truncal subcutaneous fat and insulin resistance. South Asians had larger abdominal subcutaneous adipocytes than Caucasians. [20] Enlarged adipopcytes express and secrete increased amount of leptin, TNF α and IL-6, all of which are implicated in pathogenesis of insulin resistance and type 2 DM. [21]

F. ROLE OF DIET IN TYPE 2 DIABETES

Although type 2 diabetes is determined primarily by lifestyle and genes, diet also plays an important role in both its development and complications. In the pre-insulin era diet played a dominant role in the management of diabetes. Even after the discovery of insulin and several oral hypoglycemic agents, diet forms the sheet anchor of treatment. The dietary approaches have varied from time to time. The pendulum has swung from the starvation diets of Allen, to

restriction of carbohydrates with liberal fat, to the modern high carbohydrate and high fiber diets.[22]

WHO Expert Committee opined that under-nutrition protects populations against Diabetes and as under-nutrition is common in India, incidence and prevalence of Diabetes should be less in Indians, but on the contrary, it is more. Indian lifestyle habits, especially diet, might be invoked to explain the increased susceptibility to glucose intolerance based on Neel's thrifty genotype hypothesis. Diet may contribute to the development of Diabetes in two ways: quantitatively, by supplying calories and if activity is low by resultant obesity and qualitatively by the effects of specific foods. [23,24] South Indian population is rice-eating and eats preparations made from rice for all the meals during the day. Lentils too are consumed extensively, as accompaniment to the rice preparations. Coconut is also being used a lot in kitchens. Rice, and particularly white rice, tend to have high starch content and therefore may cause rapid increases in blood sugar. Higher white rice consumption was associated with a significantly elevated risk of type 2 diabetes. This association seems to be stronger for Asians than for Western populations. A dose-response analysis showed that each serving per day of white rice consumption was associated with an 11% increase in risk of diabetes in the overall population.[25] Intensive dietary advice has the potential to appreciably improve glycemic control and anthropometric measures in patients with type 2 diabetes and unsatisfactory HbA1c despite optimized hypoglycemic drug treatment. This effect occurred despite the fact that some of the patients considered to be on maximum drug treatment were able to reduce their dose of tablets or insulin. [26]

In non diabetics, glucose tolerance deteriorates during fasting, but calorie restriction improves glucose intolerance among obese NIDDM subjects, and there was even greater

improvement after weight loss. The main effect of calorie restriction and weight loss was to reduce hepatic glucose production.[27]

Obese type 2 diabetic women treated with diet control and different types of exercise modes demonstrated reduction in weight, BMI, fasting blood sugar level, post prandial blood sugar level, low density lipoprotein level, total cholesterol level, triglyceride level and body fat and an increase in high density lipoprotein level.[28]

Dietary fat is of particular interest because fatty acids influence glucose metabolism by altering cell membrane function, enzyme activity, insulin signaling and gene expression. Replacing saturated fats and trans fatty acids with unsaturated (polyunsaturated and/or monounsaturated) fats has beneficial effects on insulin sensitivity and is likely to reduce risk of type 2 diabetes. [29]

In a large prospective study of women, no association was found between total fat intake and risk of type 2 diabetes after controlling for known risk factors. However, polyunsaturated fatty acid intake was associated with a substantial reduction in risk, and trans fatty acids and dietary cholesterol were associated with increased risk. [30]

Very low caloric diet therapy results in rapid and marked improvement in fasting plasma glucose levels, improved insulin secretary responses and peripheral insulin sensitivity also. Changes in caloric intake, rather than subsequent changes in body weight, are a major factor underlying the glucose-lowering effect of hypo caloric diets. [31]

G. ROLE OF PHYSICAL ACTIVITY IN DIABETES

Physical activity is defined as any bodily movement produced by skeletal muscles that results in energy expenditure beyond resting expenditure. Exercise is a subset of physical activity

that is planned, structured, repetitive, and purposeful in the sense that improvement or maintenance of physical fitness is the objective.[32]

Aerobic exercise: This consists of rhythmic, repeated, and continuous movements of the same large muscle groups for at least 10 min at a time. Examples include walking, bicycling, jogging, continuous swimming, water aerobics, and many sports. When performed at sufficient intensity and frequency, this type of exercise increases cardiorespiratory fitness. [33]

Resistance exercise: Activities that use muscular strength to move a weight or work against a resistive load. Examples include weight lifting and exercises using weight machines. When performed with regularity and moderate to high intensity, resistance exercise increases muscular fitness. [33]

MET (metabolic equivalent): A MET is a unit of intensity equal to energy expenditure at rest. Physical activity at 3 METs uses three times as much energy as stationary sitting. [33]

The current human genome was moulded and refined through generations of time. The basic framework for physiologic gene regulation was selected during an era of obligatory physical activity, as the survival of our Late Palaeolithic (50000–10000 BC) ancestors depended on hunting and gathering. A sedentary lifestyle in such an environment probably meant elimination of that individual organism. The phenotype of the present day *Homo sapiens* genome is much different from that of our ancient ancestors, primarily as a consequence of expressing evolutionarily programmed Late Palaeolithic genes in an environment that is predominantly sedentary. So, human genome is maladapted, resulting in abnormal gene expression, which in turn frequently manifests itself as clinically overt disease. Some of these genes still play a role in survival by causing premature death from chronic diseases produced by physical inactivity. These disruptions in cellular homeostasis are diminished in magnitude in physically active

individuals compared with sedentary individuals due to the natural selection of gene expression that supports the physically active lifestyle displayed by our ancestors. Genes evolved with the expectation of requiring a certain threshold of physical activity for normal physiologic gene expression, and thus habitual exercise in sedentary cultures restores perturbed homeostatic mechanisms towards the normal physiological range of the Palaeolithic *Homo sapiens*. [34]

The genetic basis of diabetes was proposed by geneticist James V. Neel in 1962 in **thrifty gene hypothesis**. Neel suggested that genes which predispose to diabetes (called 'thrifty genes') were historically advantageous, but they became detrimental in the modern world. In his words they were "rendered detrimental by progress". Thrifty genes are genes which enable individuals to efficiently collect and process food to deposit fat during periods of food abundance. According to the hypothesis, the 'thrifty' genotype would have been advantageous for hunter-gatherer populations because it would allow them to fatten more quickly during times of abundance. Fatter individuals carrying the thrifty genes would thus better survive times of food scarcity. However, in modern societies with a constant abundance of food, this genotype efficiently prepares individuals for a famine that never comes. The result is widespread chronic obesity and related health problems like diabetes. [23]

Thrifty gene hypothesis is based on an analysis of the pattern and level of mortality during famines. Despite much anecdotal evidence used to suggest that famines cause substantial mortality, real data available from famines involve rather low levels of mortality and there is no evidence that fat people survive famines better than lean people. Mortality actually falls mostly on groups such as the very young and very old where differential mortality in relation to body composition is highly unlikely. Another alternative to the thrifty gene hypothesis is the **drifty gene hypothesis** proposed by the British biologist John Speakman. The main feature of this idea

was that the current pattern of obesity did not suggest that obesity under strong positive selection for a protracted period of time. Instead that the obesity came about because of genetic drift in the genes controlling the upper limit on our body fatness. Such drift might have started because around 2 million years ago ancestral humans effectively removed the risk from predators, which was probably a key factor selecting against fatness. [35]

H. MECHANISMS REGULATING GLUCOSE UPTAKE INTO SKELETAL MUSCLE[7,36,37]

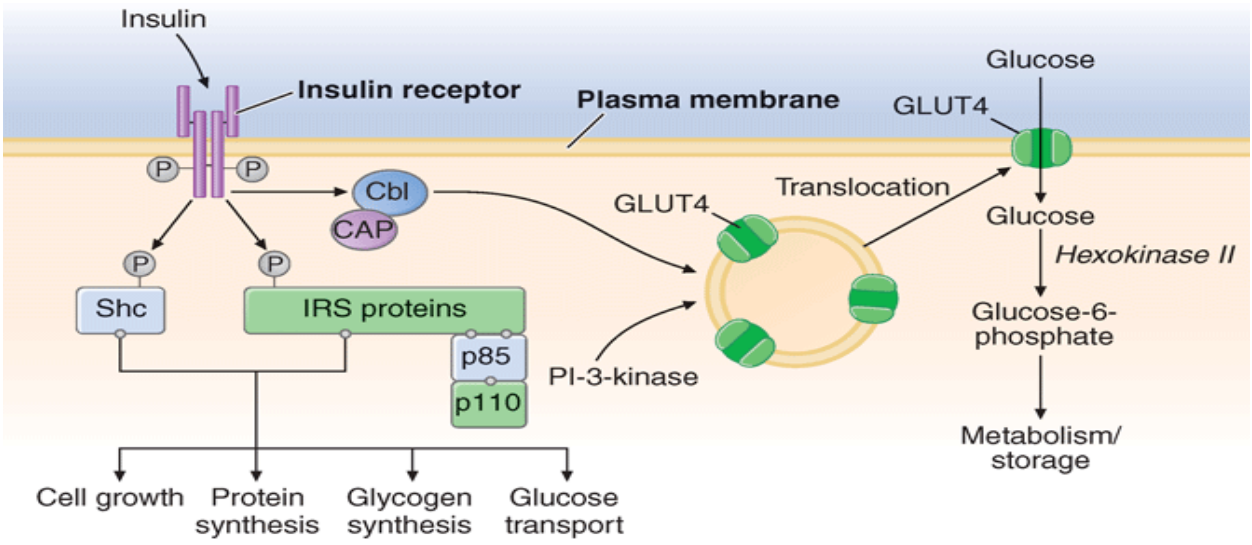
Skeletal muscle glucose uptake is mediated by glucose transporter 4, the major isoform that is responsive to hormones such as insulin, and by energy-demanding conditions such as exercise and hypoxia. Like insulin, muscle contraction (in vitro or in situ) and exercise increase glucose uptake into skeletal muscle. However, the contraction/exercise pathway of glucose uptake in skeletal muscle is an independent pathway to that of insulin. Indeed, skeletal muscle glucose uptake is normal during exercise in those who suffer from insulin resistance and diabetes. Thus, the pathway of contraction-mediated glucose uptake into skeletal muscle provides an attractive potential target for pharmaceutical treatment and prevention of such conditions, especially as skeletal muscle is the major site of impaired glucose disposal in insulin resistance.

Activation of the insulin receptor substrates (IRS)/phosphatidylinositol 3-kinase (PI3K) axis is indispensable to insulin-stimulated GLUT4 translocation and glucose uptake. Similarly, 2 serine/threonine kinases participate in mediating insulin's metabolic actions in skeletal muscle and fat. In adipose cells, c-Cbl-associated protein (CAP), adaptor protein associated with

pleckstrin homology and Src homology2 (SH2) domain (APS) and the small GTPase TC10 may also be involved in mediating insulin regulation of GLUT4 traffic and glucose uptake.

The mechanisms regulating skeletal muscle glucose uptake during contraction have not been fully elucidated. Potential regulators include Ca21 (via CaMK's and/or CaMKK), AMPK, ROS, and NO signaling, with some redundancy likely to be evident within the system.

Figure 2: Insulin signal transduction pathway in skeletal muscle.



Source: Longo DL, Fauci AS, Kasper DL, Hauser SL, Jameson JL, Loscalzo J: *Harrison's Principles of Internal Medicine, 18th Edition*: www.accessmedicine.com

Muscle Glycogen Concentration:

Skeletal muscle glucose uptake in exercising humans has been reported to be either enhanced or unaffected by low muscle glycogen levels. In vitro studies have shown that glucose transport is elevated in response to low muscle glycogen content in fast-twitch (glycolytic), but not slow-twitch (oxidative) muscle fibers. Because GLUT4 is hypothesized to be structurally bound to glycogen particles, it has been speculated that depleting glycogen releases GLUT4 to

facilitate glucose transport. If muscle glycogen has some influence on contraction mediated glucose uptake, it is likely to be via one or more of the signaling pathways.

Alternative pathways leading to glucose uptake: [36,37]

As with exercise, hypoxia also enhances glucose transport into skeletal muscle and muscle cells in culture. Hypoxia is imposed on isolated cells or isolated skeletal muscle by oxygen deprivation and its effects are mimicked by pharmacological interference with the mitochondrial oxidative chain. DNP is a mitochondrial uncoupler that transiently reduces cellular adenosine triphosphate (ATP) levels. DNP stimulates glucose uptake into both L6 myotubes and isolated fast-twitch muscle independently of PI3K activity. Hence, exercise, hypoxia and DNP are said to engage "alternative" signalling pathways.

Role of 5'-AMP-activated protein kinase (AMPK) in stimulation of muscle glucose uptake:

The heterotrimeric enzyme AMPK has been proposed as an "energy sensor" that is rapidly phosphorylated and activated during exercise/contraction in skeletal muscle. As muscle glucose transport correlated well with AMPK activity, it was proposed that AMPK might be a signal in exercise/contraction-stimulated glucose transport into skeletal muscle. Hypoxia and DNP also increased AMPK activity in L6 myotubes and in isolated skeletal muscle, providing further support for a link between AMPK activation and glucose uptake.

Role of cytosolic Ca^{2+} in the stimulation of muscle glucose uptake:

A hallmark of muscle contraction is Ca^{2+} release from the sarcoplasmic reticulum (SR) that is induced through depolarization of the adjacent transverse tubules. Increase cytosolic Ca^{2+} stimulate glucose uptake independently of contraction. The plasma membrane depolarization caused by high extracellular K+ elevates glucose uptake into L6 muscle cells and this effect is completely inhibited by dantrolene, an inhibitor of Ca^{2+} release from the SR. it can be concluded

hypoxia may induce Ca^{2+} release from the mitochondria. Indeed, glucose uptake into hypoxic muscle was also completely inhibited by dantrolene. Similarly, a role for Ca^{2+} in DNP-stimulated glucose uptake was suggested from the partial inhibition of uptake in L6 myotubes upon cytosolic Ca^{2+} chelation.

AS160 (Akt substrate of 160 kDa): in the regulation GLUT4 traffic:

Activation of the PI3K target Akt by muscle contraction/exercise remains equivocal and appears to be rapid, transient, muscle fibre-type specific and to exhibit lower activation in muscle that is contracted in vitro compared with in vivo. Interestingly, both in vitro muscle contraction and AICAR increased the phosphorylation of AS160, a recently identified Akt substrate that functionally participates in insulin regulation of GLUT4 traffic in 3T3-L1 adipocytes and L6 skeletal muscle cells.

Regulation of glucose uptake by adiponectin

Adiponectin (Ad), a recently identified adipokine, is proposed to be an endogenous insulin sensitizer. Liver appears to be the major target tissue of adiponectin, where it enhances insulin suppression of endogenous hepatic glucose production. Ad also increased glucose uptake and GLUT4 translocation in L6 muscle cells. Ad-induced AMPK phosphorylation in skeletal muscle and in muscle cells in culture, and transfection of myocytes with a dominant-negative AMPK mutant attenuated the stimulatory effects of gAd and Ad on glucose uptake. These results suggest that adiponectin engages ≥1 "alternative pathways" to regulate glucose uptake.

Nitric Oxide

The mu neural isoform of NOS (nNOSl) is the primary isoform expressed in skeletal muscle. nNOSl is constitutively active and its activity, and therefore NO production, increases by 1.5–2-fold with contraction. The NO donor sodium nitroprusside (SNP) increases NO production

in isolated skeletal muscle and enhances glucose transport independently of insulin. The NO donor, SNP appears to increase glucose uptake in muscle through a cGMP-PKG (cGMP-dependent protein kinase) dependent pathway. NO is known to also exert its effects via cGMP-independent signaling such as through the formation of peroxynitrite (ONOO2) from superoxide (O2). In adipocytes, NO increases GLUT4 translocation and glucose uptake independent of the cGMP/PKG pathway and AMPK activation, with the s-nitrosylation of proteins appearing to be responsible. Thus, there is building evidence that NO plays a critical role in signaling glucose uptake during contraction.

Reactive Oxygen Species (ROS)

Physiological levels of ROS may be important signaling molecules regulating various cellular processes, metabolism and gene expression seemingly contrasting dynamics of ROS is likely to be a product of duration and extent of exposure, with chronic low level oxidative stress or acute large increases in ROS generation having deleterious effects on muscle cells and acute modest changes initiating signaling cascades. Exogenous hydrogen peroxide can increase basal glucose uptake in isolated rat epitrochlearis muscle implicating ROS in the regulation of glucose uptake during contraction. In support of H_2O_2 being the primary ROS associated with the regulation of glucose uptake, the anti-oxidant catalase (which reduces H_2O_2 to H_2O) but not SOD, inhibits the increase in rat EDL muscle glucose uptake in response to the superoxide generating system hypoxanthine1xanthine oxidase.

Figure 3: Mechanisms of glucose uptake into skeletal muscle. (A) insulin-stimulated glucose uptake, (B) Potential mechanisms involved in contraction-stimulated glucose uptake. 1) Glucose

delivery to the muscle cell, 2) glucose transport through the membrane 3) glucose phosphorylation and therefore flux through metabolism.

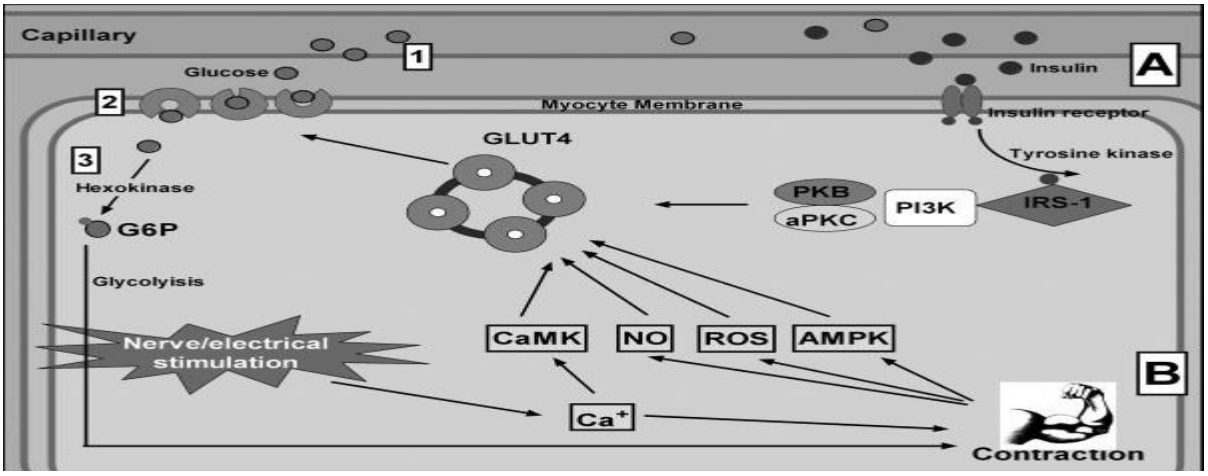
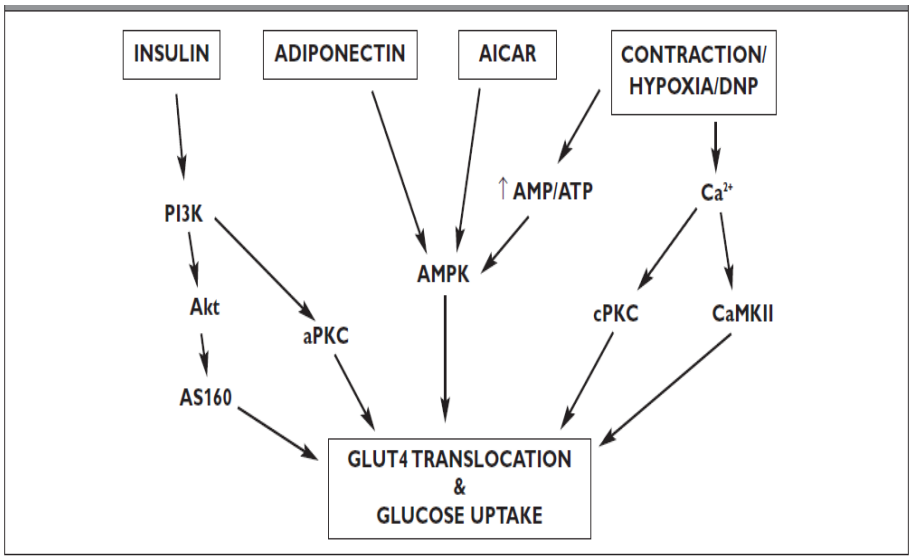


Figure 4: Alternative pathway for glucose transport in skeletal muscle



*AICAR = 5-amino imidazole-4-carboxamide ribonucleoside

*AMPK = 5'-AMP-activated protein kinase

*aPKC = atypical PKC

*CaMKII = Ca^{2+}/calmodulin-dependent protein kinase

*cPKC = conventional protein kinase C

Many studies done earlier have shown the importance of physical activity in controlling obesity and hence occurrence of diabetes mellitus. Even in diabetic patients physical activity reduces the blood sugar levels effectively through pathways independent of insulin as explained above.

There was a strong positive association between obesity (BMI) and increased risk for diabetes in both genders. The prevalence of diabetes was significantly lower for subjects who reported at least 30 min per day of sport activity. This substantially reduced the risk of diabetes in physically active subjects and remained significant after adjustment for age, BMI, WHR, cardiovascular disease and educational level.[38]

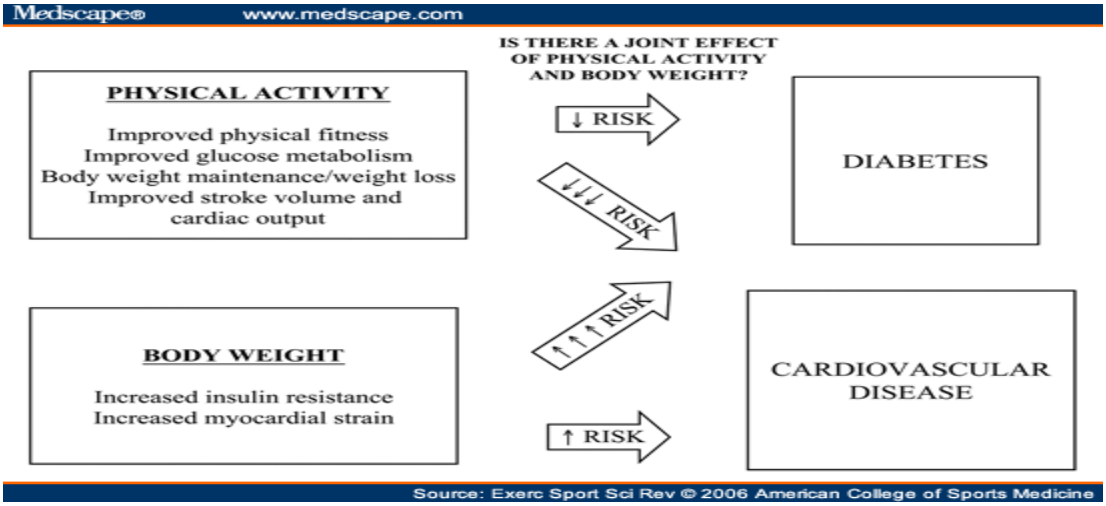
Researchers have showed that recommended physical activities have great role in decreasing important variables such as: fasting blood sugar, postprandial blood sugar and hemoglobin A1c (Hb A1c). The intake of medicine and insulin didn't have significant difference (i.e., medication didn't differ) but doing physical activity according to the recommended pattern caused the above variables to decrease significantly. So, physical activity and exercise are critical components of diabetes management. The recommended physical activity can affect most in controlling the diabetes mellitus. If any individual in regard to his physical condition cannot do physical activity, respecting a proper food pattern is in the second place of importance. [39]

Structured exercise training that consists of aerobic exercise, resistance training, or both combined is associated with HbA_{1c} reduction in patients with type 2 diabetes. Structured exercise training of more than 150 minutes per week is associated with greater HbA_{1c} declines than that of 150 minutes or less per week. Physical activity advice is associated with lower HbA_{1c}, but only when combined with dietary advice. [40,41] Higher physical activity is associated with reduced risk of dysglycemia over 5 years even after accounting for BMI. [42]

The total sitting time was more closely related to BMI than total physical activity time and subjects with a higher weight status were more sedentary. Specific group of diabetic or pre-diabetic patients with overweight or obesity had the total sitting time been more closely related to BMI than total PA time. Subjects with high sitting time had a higher BMI, even after correction for total PA time. Even those diabetics or pre-diabetics who meet public health guidelines for leisure time PA may be at risk for co-morbidities if they spend a large proportion of their residual leisure time in sedentary activities. [43]

Beyond the effect of activity on body mass and composition, physical activity may reduce the risk for type 2 diabetes directly through improvements in insulin sensitivity. However, a large portion of the effect of physical activity in decreasing insulin resistance is short lived and may last only a few days. Thus, the consistency of an individual's activity throughout the years plays a key role in the mechanisms underlying the relation between physical activity and diabetes prevention.[44]

Figure 5: Role of physical activity & BMI in DM and cardiovascular disease



BMI plays a much larger role than physical activity in the development of diabetes. In contrast, physical activity is more important than BMI in reducing the risk of cardiovascular disease.[45]

A higher step activity at the end of a five year follow-up period was associated with a lower body mass index, lower waist to hip ratio, and higher HOMA insulin sensitivity at the five year follow-up. The observed association of step activity with enhanced HOMA insulin sensitivity was largely accounted for by body mass index. This may indicate a mediation effect through change in body mass index. [46]

I. COMPLICATIONS OF DIABETES MELLITUS

The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease. The main "macrovascular" diseases (related to atherosclerosis of larger arteries) are ischemic heart disease (angina and myocardial infarction), stroke and peripheral vascular disease.

Diabetes also causes "microvascular" complications—damage to the small blood vessels. Diabetic retinopathy, which affects blood vessel formation in the retina of the eye, can lead to blindness. Diabetic nephropathy can lead to scarring changes in the kidney tissue, loss of protein in the urine, and eventually chronic kidney disease requiring dialysis. Diabetic neuropathy, most commonly causes numbness, tingling and pain in the feet and also increasing the risk of skin damage due to altered sensation. Together with vascular disease in the legs, neuropathy contributes to the risk of diabetes-related foot problems (such as diabetic foot ulcers) that can be difficult to treat and occasionally require amputation.[47]

Molecular basis of diabetic complications [48]

Hyperglycaemia causes overproduction of superoxide by the mitochondrial electron-transport chain. The metabolism of glucose through glycolytic pathway and the tricarboxylic acid cycle produces reducing equivalents that are used to drive ATP synthesis via oxidative phosphorylation in the mitochondria. Byproducts of mitochondrial oxidative phosphorylation include free radicals such as superoxide anion and their generation is increased by high glucose lcvcls. Glucose auto oxidation also creates free radicals that can damage cellular proteins as well as mitochondrial DNA. Increased oxidant stress reduces nitric oxide levels, damages cellular proteins and promotes leukocyte adhesion to the endothelium while inhibiting its barrier function causing various vascular, renal and neurological complications.

METHODOLOGY

MATERIALS AND METHODS:

Source of data:

The study group comprised of 300 diabetic subjects. The subjects were recruited from Diabetology OPD of RL Jalappa Hospital and Research, Tamaka, Kolar. Ethical clearance for the study was obtained from the Institutional Ethical Committee.

Basis for sample size:

Considering the number of cases attending Diabetology OPD expecting around 1000 cases in a year (population), the sample size was estimated to be 278 rounded off to 300 with 5% error at 95% confidence interval.

Criteria for selection of study group:

a. Inclusion criteria

1.The subjects with diagnosed Type 2 Diabetes Mellitus.

2.The subjects > 30 years of age.

b. Exclusion criteria:

1. Subjects with any obvious complications of DM.
2. Subjects with H/O diagnosed tuberculosis, congestive cardiac failure and carcinoma.

Method of collection of data:

Data was collected from the subjects volunteering for study after taking informed consent. Study included 300 patients with diagnosed T2DM attending Diabetology OPD of RL Jalappa Hospital, Kolar, without any obvious complications. Systematic random sampling was done for collection of samples by recruiting every alternate patient attending the OPD between 9 am to 11 am from Monday to Saturday. General information about each subject was noted. [Annexure 1]

Body weight was measured in kg by a mechanical scale to the nearest kg. Height was measured to the nearest one cm. BMI was calculated by the Quitelet's formula: weight/ height^2 (kg/m^2) and subjects were classified under low, normal and high BMI groups according to South Asian Standards. [Annexure 2].[18]

Dietary intake was determined by dietary recall. Total calorie and protein intake were estimated for calculation of calorie and protein deficiency or excess after noting the recommended daily allowance for that age [Annexure 3].[49] The Nutritive Value of different food items was calculated using Dietary Guidelines Manual, National Institute of Nutrition, Hyderabad.[50]

Physical Activity profile was studied using standardized questionnaire and average duration of the activity per day was calculated. [Annexure 4] [51]

Physical Activity Level is a composite index of physical activity and is given by the formula:

24 hour energy expenditure / basal metabolic rate.

24 hours energy expenditure is calculated as the sum of energy expenditures of all reported activities for a single day. The activities which are reported for one week are recomputed for 24 hours as

the sum of energy expenditure related to sleep, occupational energy expenditure, discretionary leisure time energy expenditure and residual energy expenditure. Energy expenditure for each of these components BMR/min is calculated first and MET (Metabolic equivalent) of that activity is applied as a multiple of BMR. Therefore

24 hour Energy expenditure = $\sum$ MET ***** Average duration of activity per day (min/day) *****Basal metabolic rate.

Residual energy expenditure relates to those periods of a day which are unaccountable for by recall and for which intensities of activities have to be assumed. Since reports from different literatures suggest that individuals tend to underreport sedentary activities, a uniform MET of 1.4 was employed for all residual time.[51]

BMR was calculated from age and gender specific regression equations recommended by WHO, that include height and weight as predictor variables.[52] [Annexure 5]

Physical activity Level (PAL) values:

- < 1.4 - Sedentary
- 1.4 – 1.75 - moderate physical activity
- >1.75 - heavy physical activity

Statistical Treatment of the data:

The data was suitably arranged into tables under different headings [Annexure 6]. Descriptive statistical analysis was carried out and data was presented in terms of proportions, means & standard deviation. Results on continuous measurements were analysed using independent t test and correlation between quantitative variables was done using Pearsons correlation. Significance was assessed at 5% level of significance. Conclusions are drawn based on the outcome of this statistical treatment.

RESULTS AND ANALYSIS

RESULTS AND ANALYSIS

In the present study, 300 subjects with type 2 diabetes mellitus were selected considering the inclusion and exclusion criteria and their BMI, dietary intake and physical activity levels were assessed.

Presentation of data

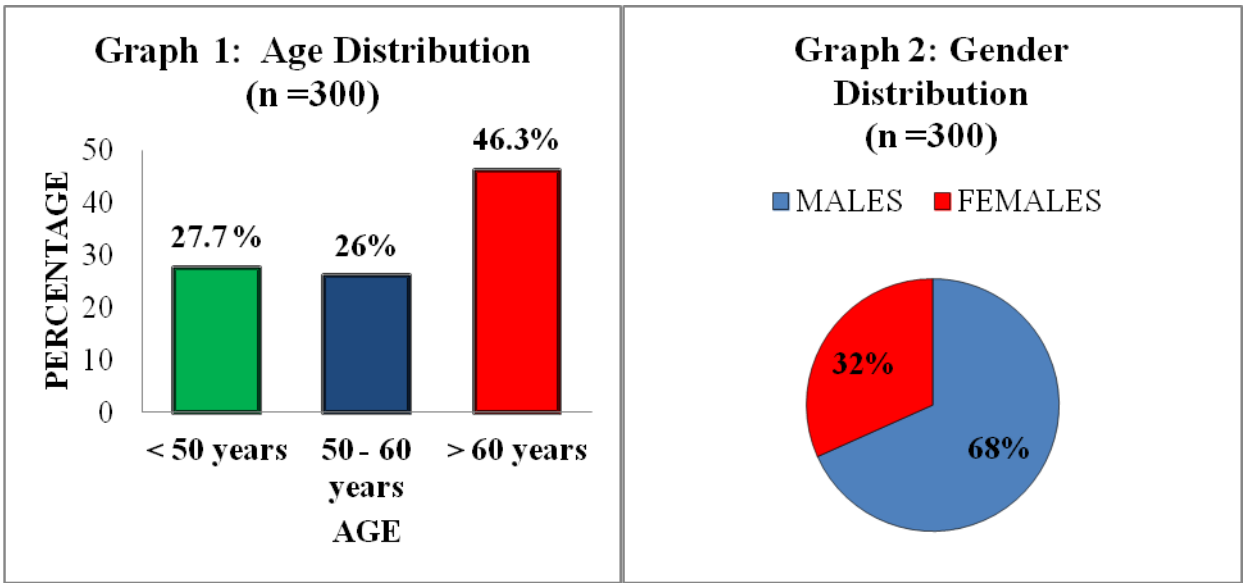
Master chart showing daily intake of calories & proteins, weight, height and physical activity levels with age and sex of the subjects and duration of diabetes. [Annexure 6]

Table 1: Characteristics of the study population

PARAMETERS				
Age		< 50 years (27.7%)	50– 60 years (26%)	>60 years (46.3%)
Gender		Males (68%)	Females (32%)	
BMI (kg/m^2)		Low (6%)	Normal (29%)	High (65%)
Physical activity		Sedentary (68%)	Moderate (16.3%)	Heavy (15.7%)
Diet				
	Calories	Excess (35%)	Deficiency (65%)	
	Protein	Excess (17%)	Deficiency (83%)	
Duration of diabetes		<10 years (71.7%)	10 –20 years (20.3%)	>20 years (8%)

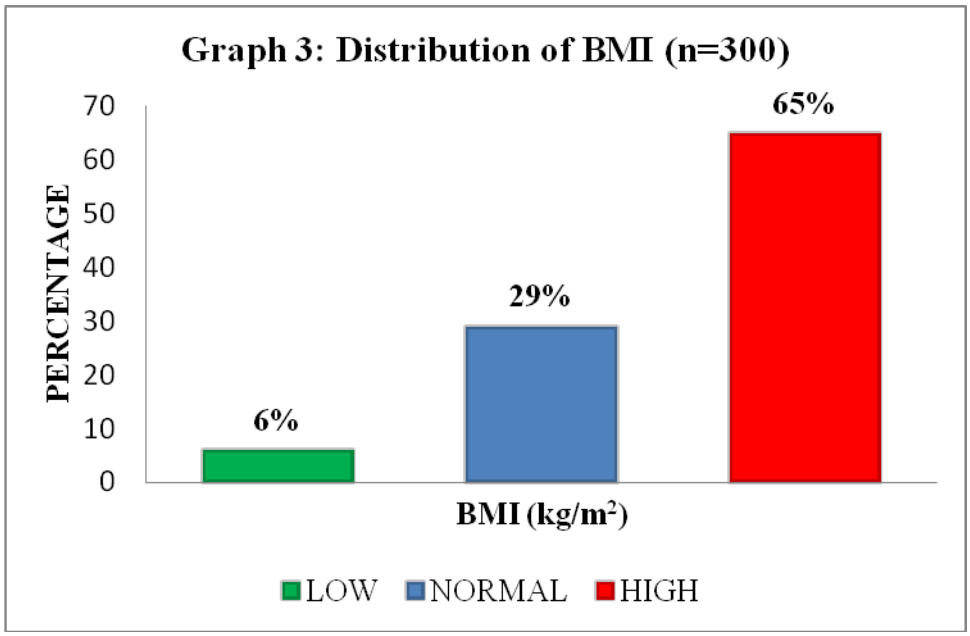
AGE AND GENDER DISTRIBUTION:

The mean age of the study group was 57.3 ± 10.81 years varying from 34 years to 82 years. As shown in Graph 1, 27.7% of the subjects were < 50 years of age, 26% were between 50-60 years and rest 46.3% > 60 years. Out of the 300 subjects, 68% were males and 32% were females (Graph 2).



DISTRIBUTION OF BMI

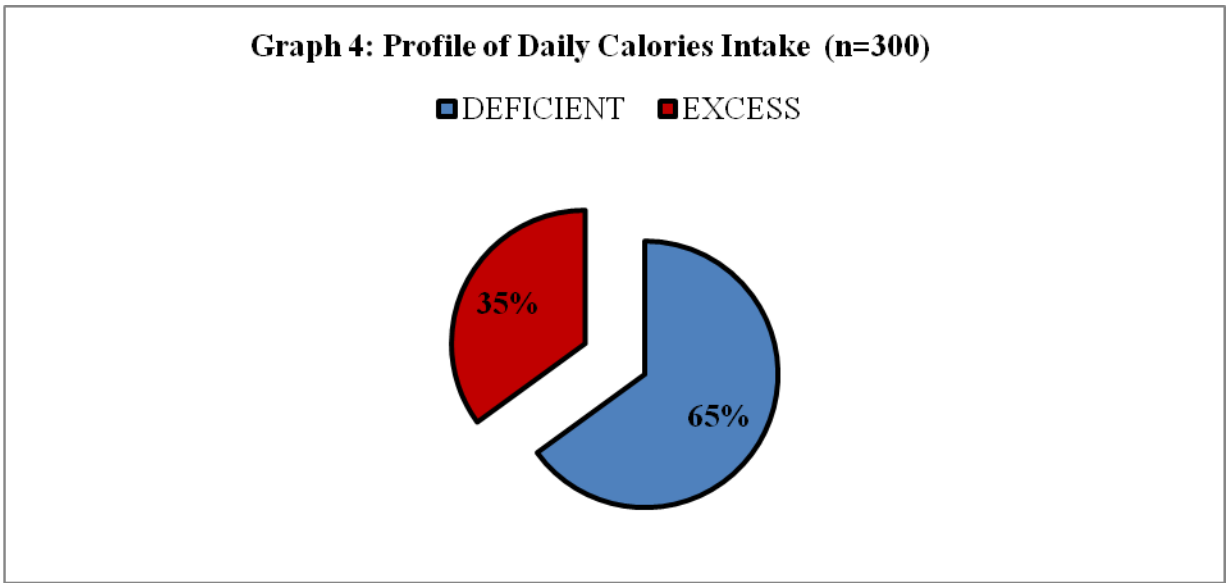
Taking the normal BMI range (18.9 – 22.9 kg/m^2) according to Asian standards, 6% of the subjects were having low BMI, 29% normal BMI and 65% were having high BMI (Graph 3).



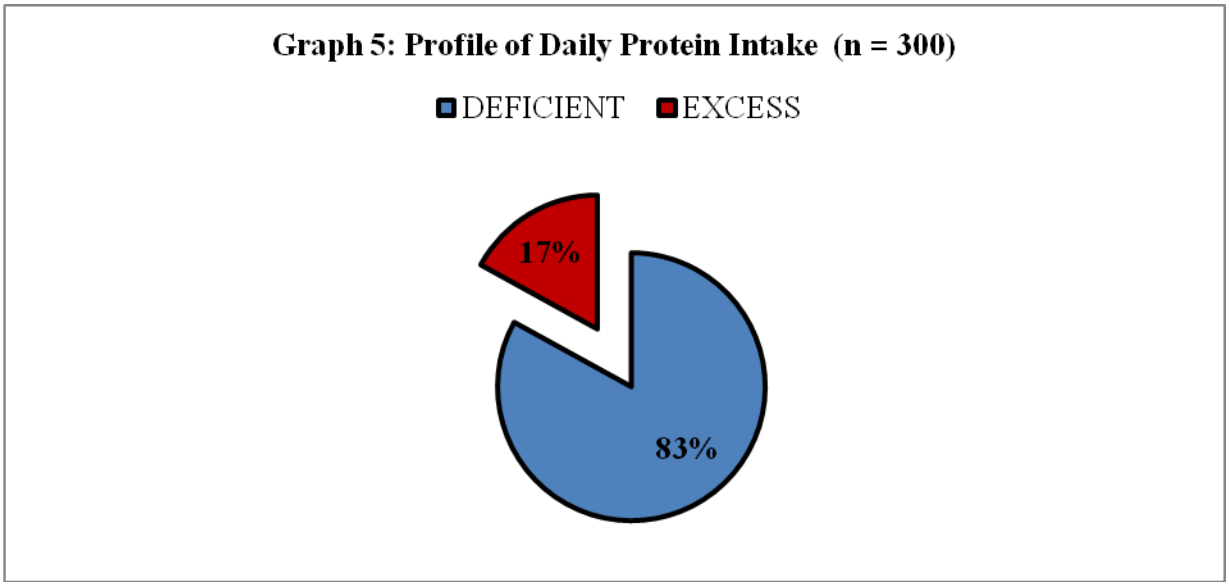
PROFILE OF DIETARY STATUS:

With regard to the dietary status, 35% of the subjects were consuming excess calories and 65% were having deficient calories intake whereas regarding protein, 83% were deficient in daily protein intake and just 17% were consuming excess protein. (Graph 4,5)

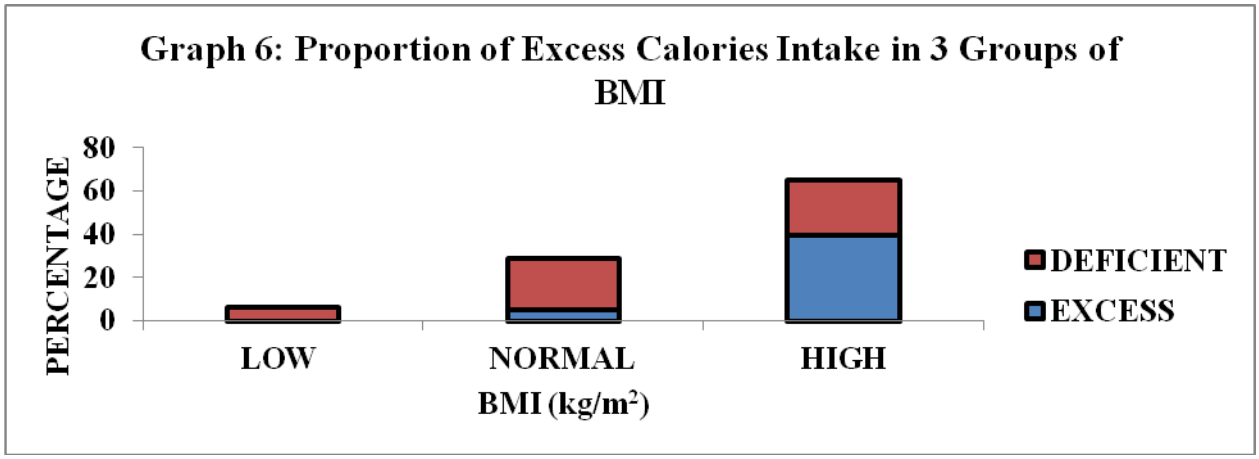
Graph 4: Profile of Daily Calories Intake (n=300)



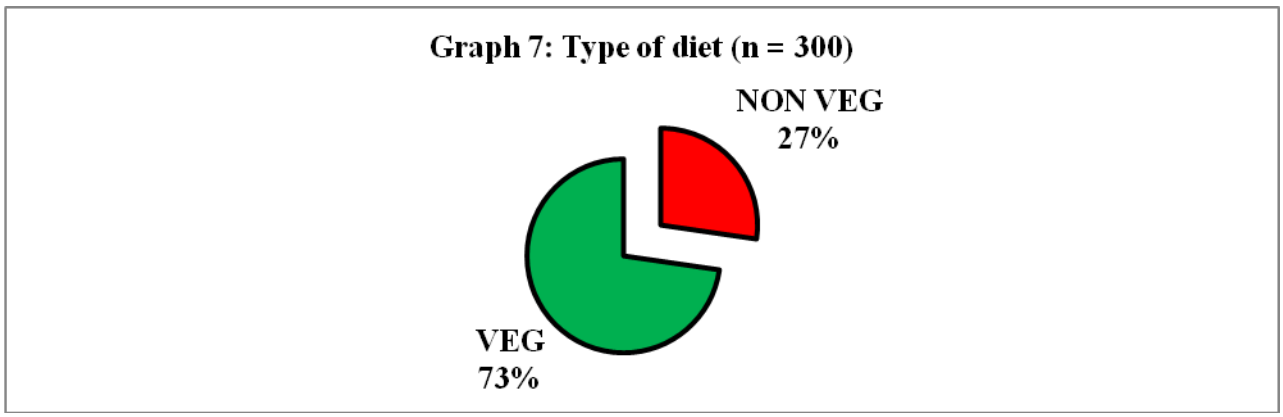
Graph 5: Profile of Daily Protein Intake (n = 300)

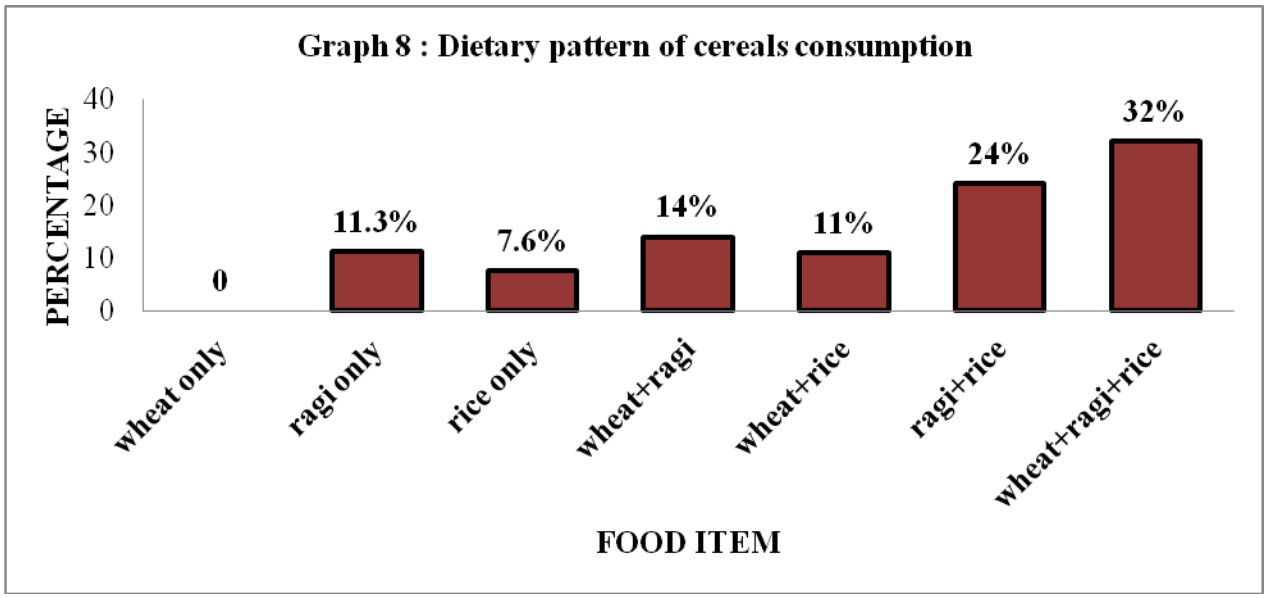


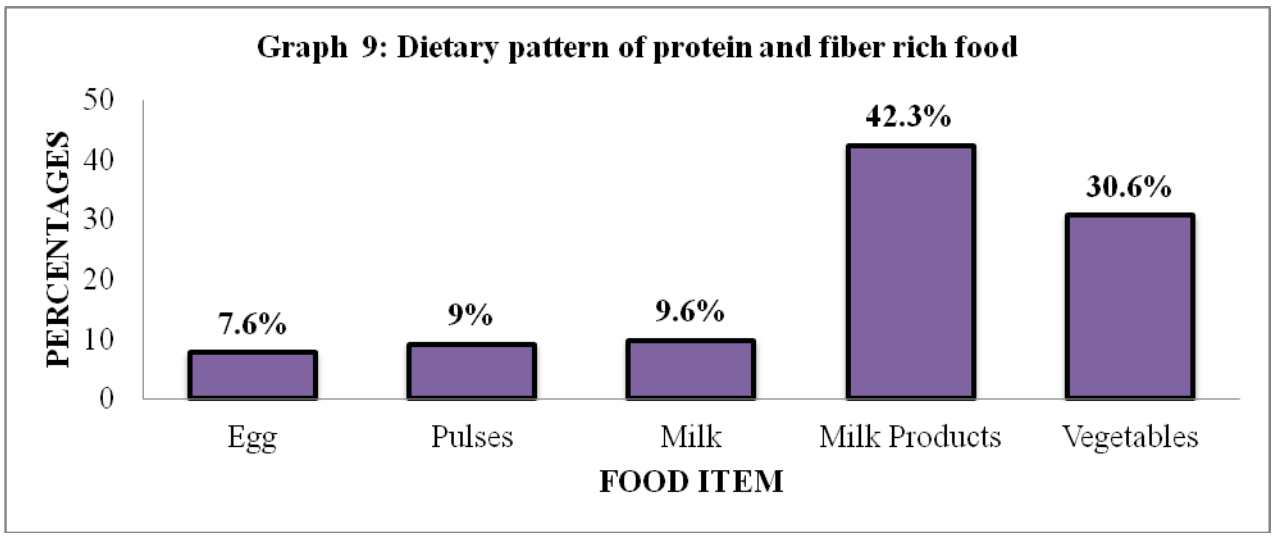
Excess calories intake was present in 61% of subjects in high BMI group while it was 18.3% in normal BMI group and none in low BMI group. (Graph 6)



73% of the subjects were consuming vegetarian diet and rest 27% were non vegetarians (Graph 7). Food items with highest daily consumption were white rice, ragi and wheat. Most of the subjects (32%) consume a mixture of all three cereals. While 24% consumed rice and ragi, 11.3% only ragi, 11% ragi and wheat, 7.6% only white rice and none was having only wheat (Graph 8). Out of them, 9% were consuming pulses. 42.3% were consuming milk products and out of them milk was taken by only 9.6% of subjects. Egg was consumed by 7.6% of subjects. Vegetables, a good source of dietary fibers were consumed by 30.6% of the subjects (Graph 9).

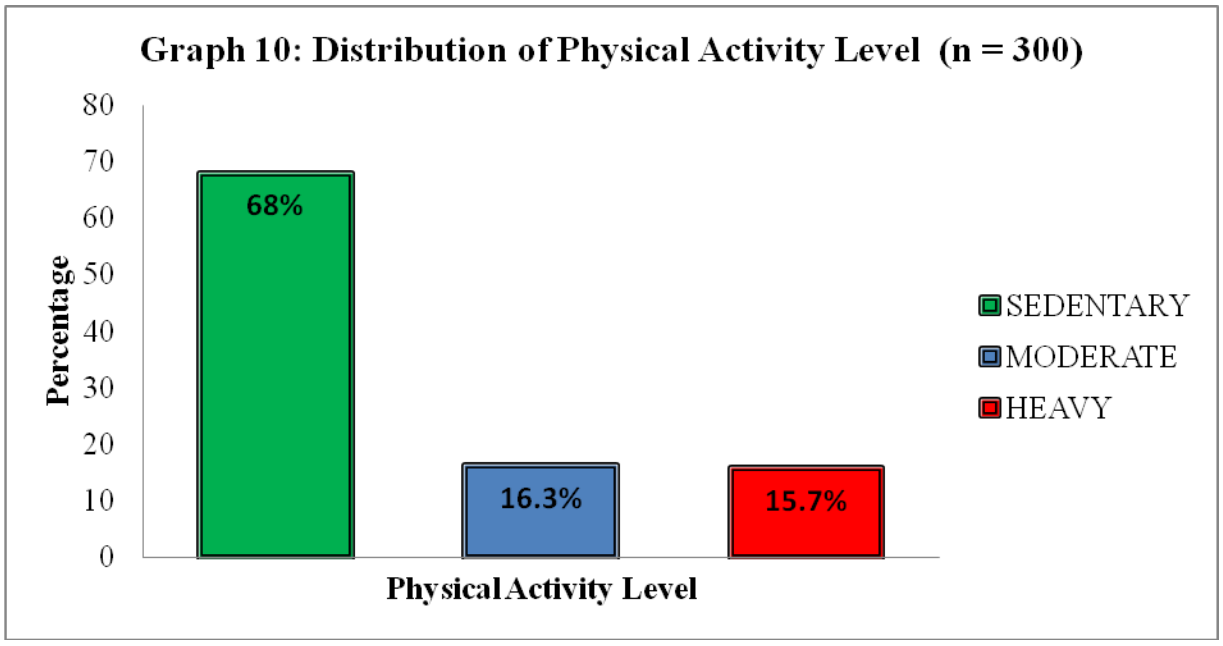

Graph 7: Type of diet (n = 300)
NON VEG
27%
VEG
73%

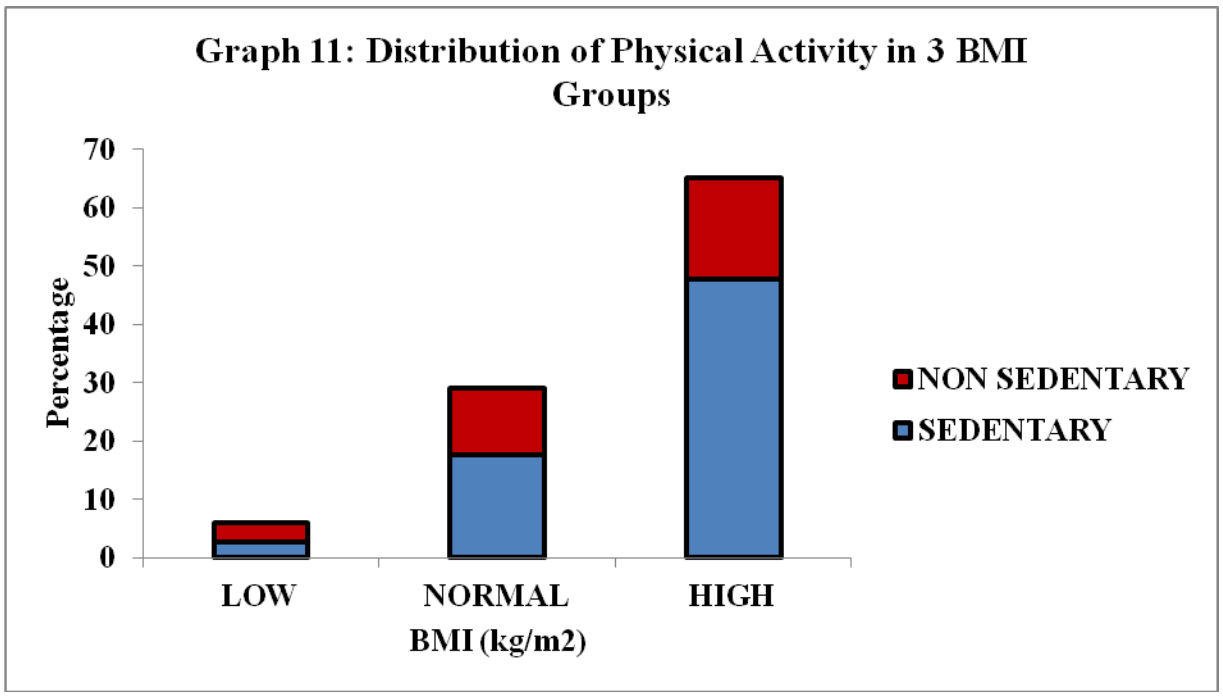

Graph 8 : Dietary pattern of cereals consumption
PERCENTAGE
40
30
20
10
0
0
11.3%
7.6%
14%
11%
24%
32%
wheat only
ragi only
rice only
wheat+ragi
wheat+rice
ragi+rice
wheat+ragi+rice
FOOD ITEM


Graph 9: Dietary pattern of protein and fiber rich food
PERCENTAGES
50
40
30
20
10
0
7.6%
9%
9.6%
42.3%
30.6%
Egg
Pulses
Milk
Milk Products
Vegetables
FOOD ITEM

DISTRIBUTION OF PHYSICAL ACTIVITY LEVELS

As far as physical activity level was concerned, 68% of the subjects were having sedentary lifestyle, 16.3% were moderately active and 15.7% were doing heavy physical activity (Graph 10). Sedentary lifestyle was observed in 73.3% of the subjects having high BMI, 60.9% of the normal BMI and 44.4% of low BMI group (Graph 11). 89.6% of the subjects taking excess calories were having sedentary lifestyle whereas 50.3% of the subjects with deficient calories intake had sedentary lifestyle (Graph 12).




Graph 11: Distribution of Physical Activity in 3 BMI Groups
70
60
50
40
30
20
10
0
Percentage
LOW
NORMAL
HIGH
BMI (kg/m2)
NON SEDENTARY
SEDENTARY

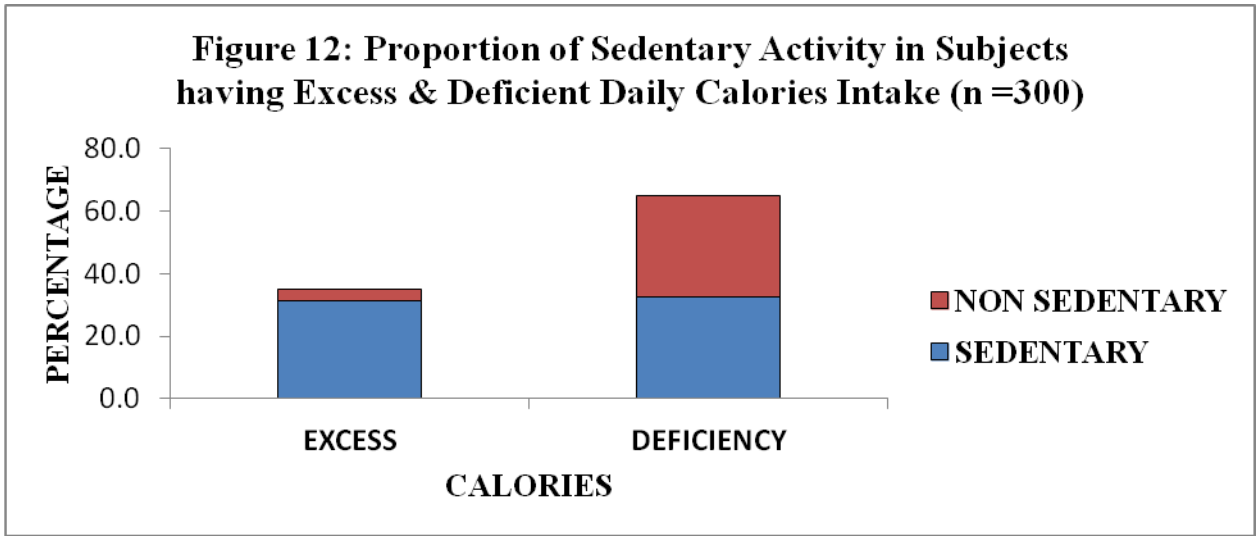
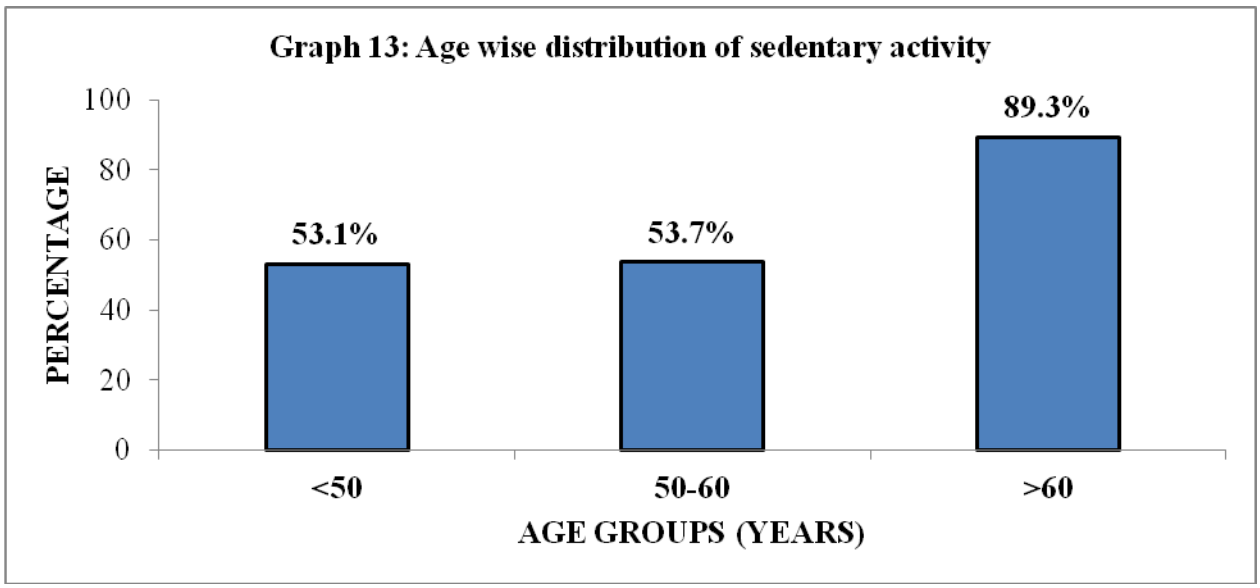


Figure 12: Proportion of Sedentary Activity in Subjects having Excess & Deficient Daily Calories Intake (n =300)
80.0
60.0
40.0
20.0
0.0
PERCENTAGE
EXCESS
DEFICIENCY
CALORIES
NON SEDENTARY
SEDENTARY

As far as age wise distribution of physical activity is concerned, 89.3% of the subjects > 60 years of age, were sedentary. In the same way 53.7% and 53.1% of subjects had sedentary lifestyle in 50 – 60 year and <40 year age group respectively (Graph 13).



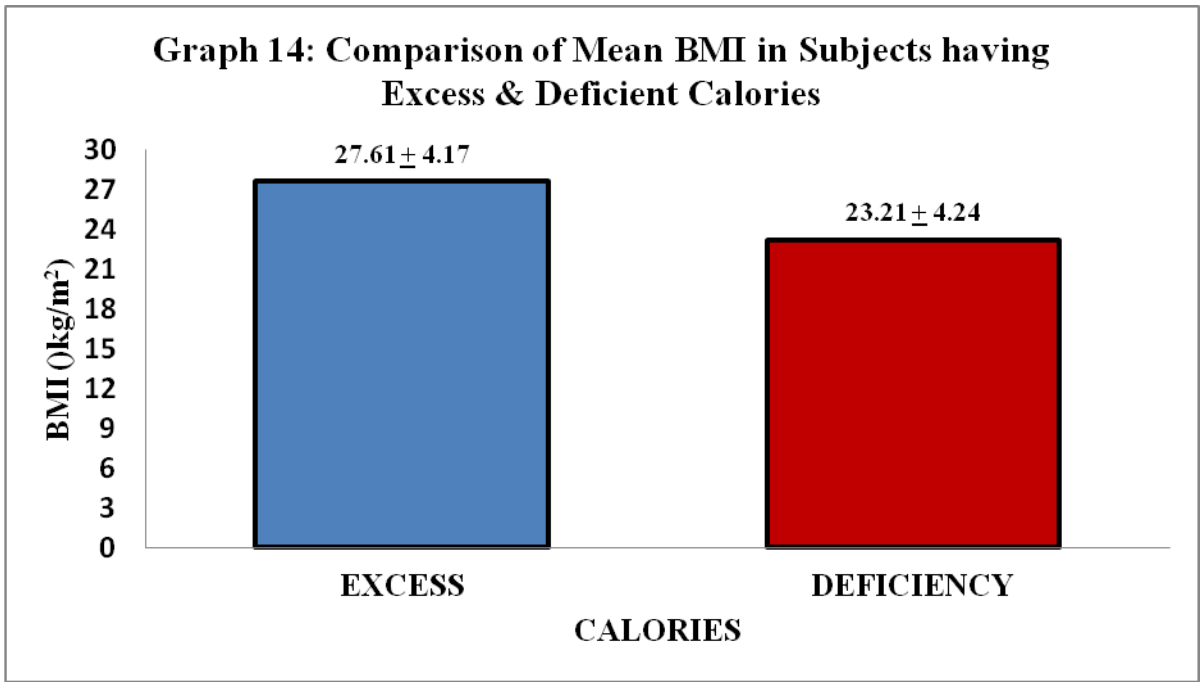
The mean BMI of the subjects taking excess calories intake was significantly higher (p < .0001) than those who were deficient in calories intake (Table 2, Graph 14). Similarly the mean BMI of the subjects consuming significantly higher protein intake (p< .0001) was also higher than those who were deficient in protein (Table 3, Graph 15).

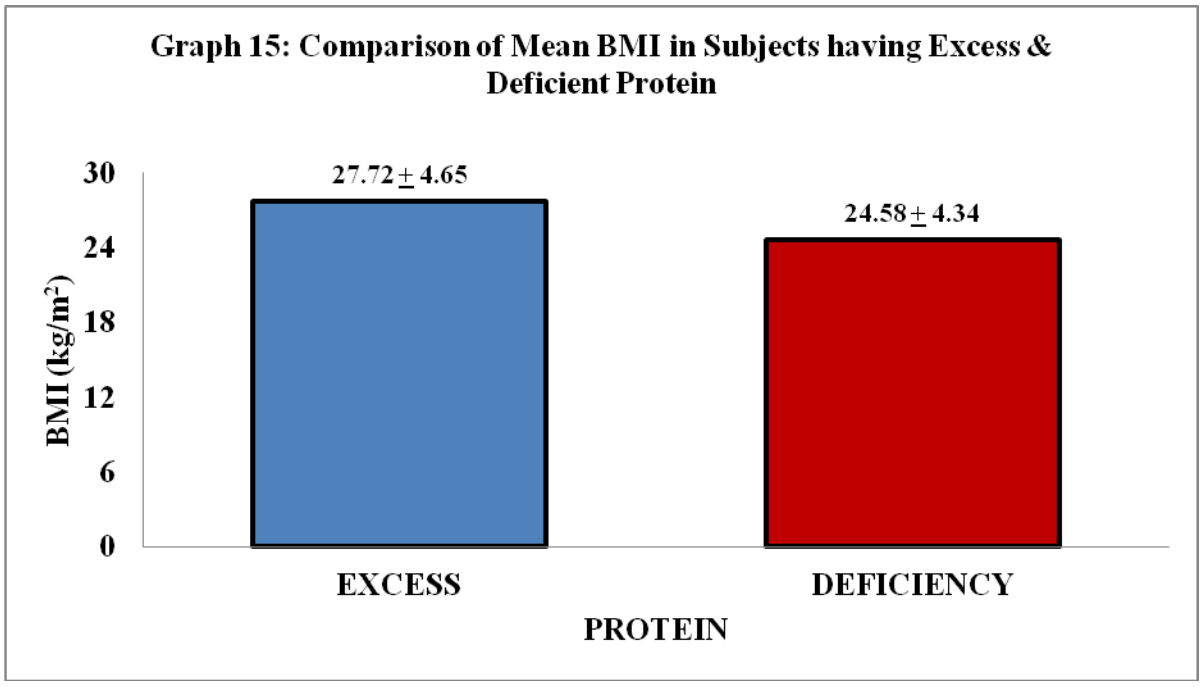
Table 2: Independent t test comparing BMI in excess and deficient calories intake groups

	CALORIES	N	Mean ± Std. Deviation	t value	p value
BMI	EXCESS	135	**27.61 ± 4.17**	9.005	**< .0001**
	DEFICIENT	165	**23.21 ± 4.24**		

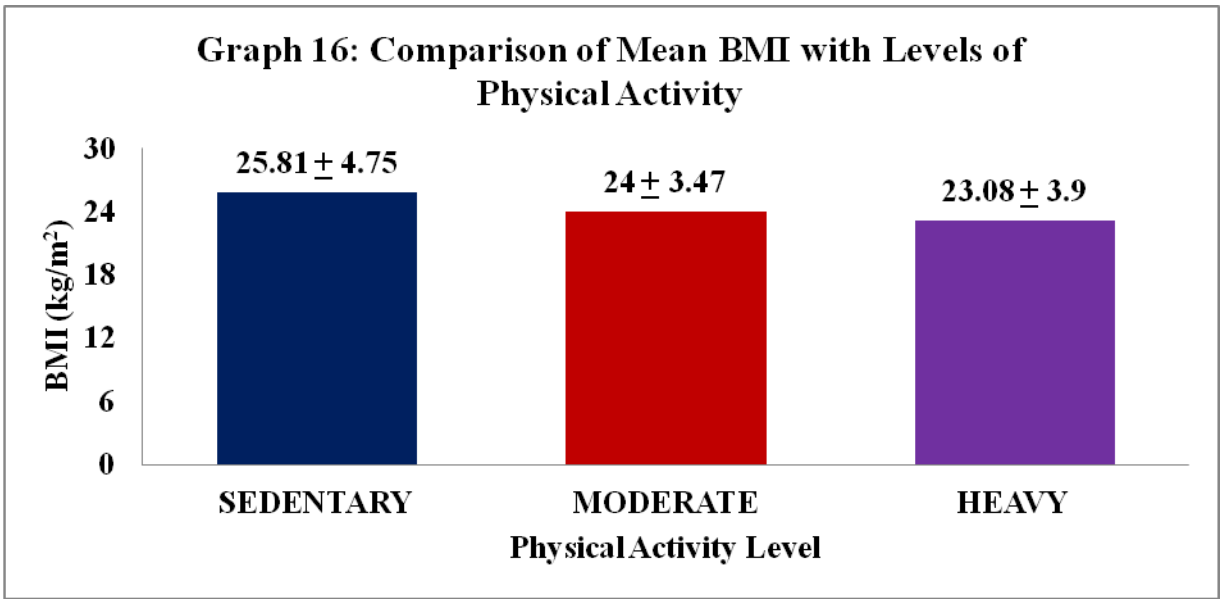
Table 3: Independent t test comparing BMI in excess and deficient protein intake groups

	PROTEIN	N	Mean Std. Deviation	t value	p value
BMI	EXCESS	51	**27.72 ±** 4.65	4.638	**< .0001**
	DEFICIENCY	247	**24.58 ±** 4.34		


Graph 14: Comparison of Mean BMI in Subjects having Excess & Deficient Calories
30
27
24
21
18
15
12
9
6
3
0
BMI ()kg/m²
27.61 ± 4.17
23.21 ± 4.24
EXCESS
DEFICIENCY
CALORIES


Graph 15: Comparison of Mean BMI in Subjects having Excess & Deficient Protein
30
24
18
12
6
0
BMI (kg/m²)
27.72 ± 4.65
24.58 ± 4.34
EXCESS
DEFICIENCY
PROTEIN

The mean BMI of subjects in different physical activity groups is shown in Graph 16 and table 4a).



The mean BMI was significantly more in subjects with sedentary lifestyle in contrast to subjects having moderate ($p = 0.033$) and heavy activity ($p = 0.001$) . The mean BMI of moderate activity group was not statistically significant when compared with heavy activity group. (Table 4 a,b , Graph 16)

TABLE 4 a): ANOVA results comparing BMI in physical activity groups

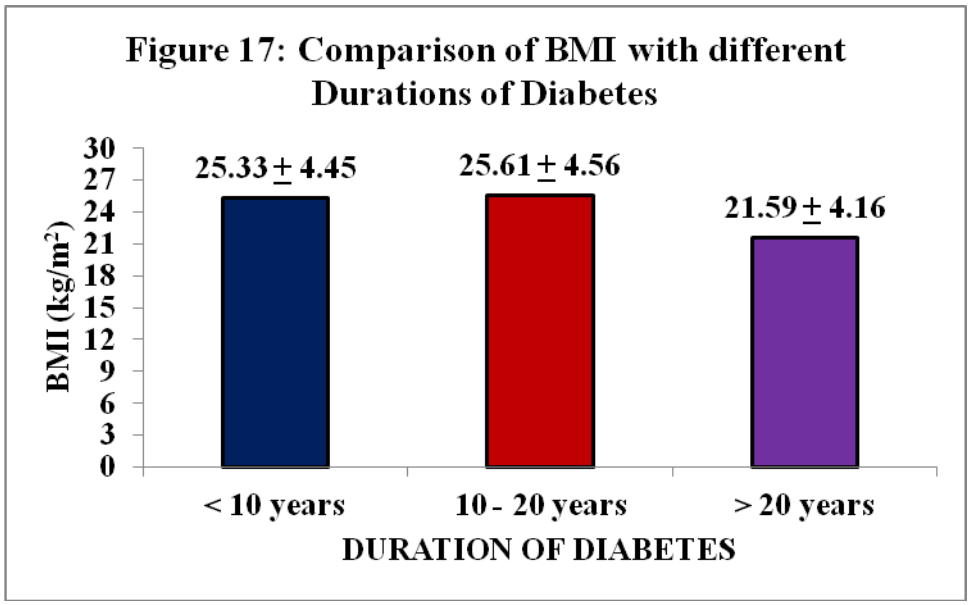
	SEDENTARY ACTIVITY	MODERATE ACTIVITY	HEAVY ACTIVITY	ANOVA (F VALUE)
BMI (mean $\pm$ SD)	25.81 $\pm$ 4.75	24 $\pm$ 3.47	23.08 $\pm$ 3.9	8.925 $p < 0.0001$

TABLE 4 b) POST HOC (BONFERRONI TEST) comparing means of BMI

	Sedentary vs Moderate	Sedentary vs Heavy	Moderate vs Heavy
BMI	$p = 0.033$	$p = 0.001$	$p = 0.939^{ns}$

ns: not significant

The subjects were divided according to the duration of diabetes (<10 years, 10 – 20 years and > 20 years). The mean BMI of subjects in different duration groups is shown in Graph 13 and table 5a).



The mean BMI was significantly lower in subjects with duration of diabetes > 20 years compared to < 10 years group ($p < 0.0001$) and 10 – 20 years group ($p = .001$). But there was no statistical difference in BMI of subjects with duration < 10 years and 10 – 20 years. [Table 5 a), b)]

TABLE 5 a) ANOVA results comparing BMI in different duration of diabetes

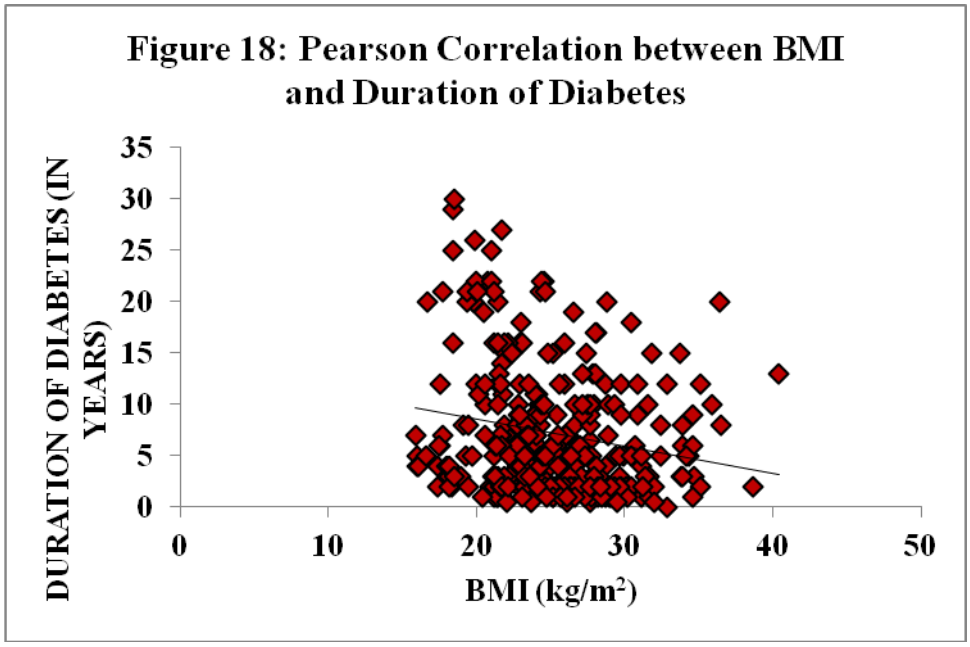
	< 10 YEARS	10 – 20 YEARS	>20 YEARS	ANOVA (F VALUE)
BMI (mean ± SD)	25.33 ± 4.45	25.61 ± 4.56	21.59 ± 4.16	8.127 $p < 0.0001$

TABLE 5 b) POST HOC (BONFERRONI TEST) to compare means of BMI

	< 10 YEARS VS 10 – 20 YEARS	< 10 YEARS VS >20 YEARS	10 – 20 YEARS VS >20 YEARS
BMI	$p = 1.00^{ns}$	$p < 0.0001$	p =.001

ns: not significant

Pearson's correlation showed a significant negative correlation between BMI and duration of diabetes ($r = - 0.194$, $p < 0.001$). (Graph 18).



DISCUSSION

DISCUSSION

Diabetes mellitus is a major public-health problem worldwide. Its incidence is increasing rapidly, and by 2030, this number is estimated to almost double.[1] The increase in incidence in developing countries follows the trend of urbanization and lifestyle changes. Individuals with T2DM are considered on high priority as they are potential candidates for rapid evaluation to prevent and halt the progression of complications.

This study presented descriptive data from a large number of subjects with diabetes type 2 attending the Diabetology OPD of our hospital.

Obesity was prevalent in 65% of the representative sample of type 2 diabetes patients. These results are comparable to other studies done in India and abroad. Higher prevalence of overweight and obesity in diabetics with 58.1% of subjects having high BMI was observed in Chennai. [53] Similarly in Gujarat, overweight and obesity in diabetics was even more common observed in 83% of the diabetics. [54] Another study from South Africa revealed 78% of obesity in diabetics. [55] Whereas obesity prevailed among 86% of diabetics attending a hospital diabetic clinic in UK. [56]

The higher prevalence of obesity in these subjects may be due to various factors which of diet and sedentary lifestyle being most important. As far as overall dietary status is concerned, 35% of the subjects were consuming excess calories. However in relation to obesity, 61% of the high BMI group was having high calorie diet making dietary calories an important cause of obesity in diabetes. Inspite of only 35% of diabetics consuming excess calories, 65% were having high BMI. This discrepancy may be due to under reporting because of low education levels (32% of subjects were illiterate and 20% had only primary education). The problem of

under reporting in nutritional research has been reported earlier and it is said to be particularly common among the obese. [57]

Regarding diet, it is not only the quantity of food intake but also the quality of food item which is important. High intake of foods with a high glycemic index (GI) and glycemic load (GL) may increase the risk of type 2 diabetes mellitus. Foods with higher GI and GL can cause rapid post-prandial increase in blood glucose resulting in hyperglycemic state. [58]

South Indian population (Karnataka, Andhra Pradesh, Tamil Nadu and Kerala) is rice-eating and eats preparations made from rice for all the meals during the day. But in Karnataka and in our study, diet does not solely contain rice; it also constitutes ragi and wheat.

75% of our subjects had rice in their diet. Rice has been a staple food in Asian populations for thousands of years, the transition in nutrition to more refine white rice may render Asian populations more susceptible to the adverse effects of high intakes of white rice. Higher white rice intake is associated with a significantly elevated risk of type 2 diabetes, especially among Asian populations. [25] This is due to high glycemic index and low dietary fibre in white rice. [58] In addition, the dose-response relations indicate that even for Western populations with typically low intake levels, relatively high white rice consumption may still modestly increase risk of diabetes.The deleterious effects of refined grains (predominantly white rice) and its strong association with type 2 diabetes and also with metabolic syndrome were evident from Chennai Urban Rural Epidemiology Study (CURES). [59]

As far as Ragi is concerned, around 81% of subjects were consuming ragi daily. Though it has high dietary fibre content and dietary fibre is known to exert an inhibitory effect on the starch digestibility. [60,61] But it elicites a glycaemic response equivalent to that of the glucose load. [62] So, the usefulness of ragi as a part of diabetic diet is questionable. Different studies have

shown different results. According to American Journal of Clinical Nutrition, ragi has high glycemic index and load which is even higher than rice. [63] But according to Association of Food Scientists & Technologists (India) ragi contains phenolics that inhibit Malt amylase, α glucosidase, pancreatic amylase reducing postprandial hyperglycemia by partially inhibiting the enzymatic hydrolysis of complex carbohydrates. It also inhibits Aldose reductase, prevents the accumulation of sorbitol and reduces the risk of diabetes induced cataract diseases. Methanolic extract in ragi prevents glycation and crosslinking of collagen and reduce complication of diabetes by improving antioxidant status. Regular consumption of ragi as a food or even as snacks helps in managing diabetes and its complications by regulation of glucose homeostasis and prevention of dyslipideamia.[64] But to date there are no long term intervention trials helpful to bring to light the health benefits of ragi consumption. [65]

Only 57% of subjects consumed wheat in their diet and none of them were purely consuming wheat. Wheat has a low glycemic index compared to rice and ragi and also contains high dietary fiber. In addition to fiber wheat also contains resistant starch which is defined as starch that is not absorbed in the small intestine of humans. This resistant starch has glycemic index lowering capacity and hence helps to manage type II diabetes. [66]

In the National Urban Diabetes Study (NUDS) study, the prevalence rates of diabetes was higher in three southern cities (Hyderabad, Chennai and Bangaluru) where rice is the staple diet, compared to three northern cities (Delhi, Kolkata and Mumbai) where it is wheat. [67]

Regarding protein more than 80% of the subjects were protein deficient. Majority ie 73% of the subjects were vegetarian, among them only 9% were consuming pulses. Although around 40% were consuming milk products but it was in the form of either butter milk or curd in small quantities (1 or 2 small cups) and milk was taken by only 9.6% of subjects. Egg which is rich

protein was consumed by around 8% of subjects. Only 30% of the subjects consumed vegetables which are good source of dietary fibers and micronutrients.

An increase in protein content in the diet, particularly if associated with a decrease in carbohydrate content would result in a decrease in the integrated glucose concentration. Such diet is useful for controlling blood glucose in persons with type 2 diabetes, provided it does not result in any adverse effects. [68]

The other important factor responsible for obesity is physical activity and majority of our subjects (68%) were having sedentary lifestyle and only 32% had either moderate or heavy activity. Similar results were revealed from other studies. Sedentary lifestyle was observed in 84% of subjects in Gujarat. [54] In a study done in Chandigarh, it was present in 47.5% of the subjects. [69] Prevalence of diabetes was significantly higher among subjects with light grade activity (17.0%) compared to moderate grade (9.7%) and heavy grade activity (5.6%) was shown in Chennai study.[57] 33.3% of the subjects fell into the low physical activity in Malaysia. [70] In our study, among the high BMI group, sedentary activity was observed in around 73% of the subjects making it an important contributing factor for obesity. In excess calories group, around 90% (94 subjects) were sedentary. Overall, around 30% of the subjects had high BMI, excess calories and sedentary lifestyle. Highest percentage of sedentary subjects was in > 60 year age group, so age may be a barrier to exercise. In other age groups also more than 50% of subjects had sedentary lifestyle, this may be due to ignorance about importance of physical activity or lack of motivation.

When compared with duration of diabetes, mean BMI was lowest in diabetics with duration > 20 years confirming the fact that BMI reduces as duration of diabetes increases.[71] Also BMI was negatively correlated to duration of diabetes. Although weight loss is more

common in type 1 diabetes but it also occurs in type 2 though after several years of onset of the disease. In the later stages of type 2 diabetes, the pancreatic beta cells become "exhausted" and are unable to produce enough insulin. Insulin is required for protein synthesis especially in muscles and an insulin deficiency can both decreases muscle synthesis and increases its breakdown of protein. Moreover failure to use glucose for energy leads to increased utilization and decreased storage of proteins as well as fat. Therefore, a person with severe untreated diabetes mellitus suffers weight loss.[72]

As a consequence of industrialization & urbanization, there has been an increase in the standard of living leading to a nutritional transition with consumption of diet which is energy dense and high in fat and sugar component. Moreover, with changes in occupation from predominantly agriculture based manual labour jobs to sedentary office type jobs; there is an immense decrease in physical activity. This is the basis for the rapid weight gain and obesity seen in several parts of the sub-continent.

Type & quantity of diet and exercise form a solid foundation for the prevention and treatment of diabetes mellitus. Regular physical activity increases insulin sensitivity, improves pharmacotherapy, lowers blood sugar concentrations, reduces body fat content, builds muscle and improves cardiovascular fitness and function.[73]

CONCLUSION AND SUMMARY

CONCLUSION

Obesity was prevalent among diabetics establishing it a major risk factor for type 2 diabetes. Majority (68%) of them had sedentary lifestyle. Excess calorie intake was observed in those with high BMI and sedentary lifestyle. Most of them were consuming a protein deficient diet.

Majority of the subjects in our study were consuming a mixture of cereals, white rice and ragi being most important among them. White rice has high glycemic index and low fiber whereas ragi's benefit in diabetes is to be proved. Emphasis should be placed on improving the intake of whole grain cereals like wheat, legumes and vegetables in the right quantities which are affordable source of dietary fibre with a low glycemic index rather than refined grains.

Awareness regarding lifestyle modification with respect to diet, physical activity and weight control should be created among diabetic patients by counselling and educative programs. Dietary prescription should be made keeping in mind the presence of pre existing calorie and protein deficiency.

SUMMARY

This cross sectional study was conducted to calculate BMI, assess dietary status and determine physical activity levels in patients with type 2 diabetes. 300 diabetic subjects were enrolled in the study. BMI was calculated using Quitelet's index and classified as low, normal and high BMI group. Dietary history was obtained by dietary recall and calorie & protein excess or deficiency was calculated after noting the recommended daily allowance for that age. Physical activity profile was assessed by standard questionnaire and physical activity level determined and classified as sedentary, moderate and heavy activity. Statistical analysis revealed high BMI and sedentary lifestyle in majority of diabetics were having high BMI and were sedentary. Most of subjects in high BMI group were consuming disproportionate diet with high calories and low protein. Therefore, awareness regarding importance of lifestyle modification by exercise and diet control should be spread in addition to treating them conventionally.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. International Diabetes Federation. IDF Diabetes Atlas [homepage on the Internet]. Brussels: International Diabetes Federation;[updated 2011; cited 2012 Sept 12]. The Global Burden. [About 6 screens]. Available from: http://www.idf.org/diabetesatlas/5e/the-global-burden.

2. Yoon KH, Lee JH, Kim JW, Cho JH, Choi YH, Ko SH et al. Epidemic obesity and type 2 diabetes in Asia. Lancet 2006; 368:1681–88.

3. Diabetes India [Homepage on the Internet]. The Indian Task on Diabetes Care in India. Mumbai; [cited 2010 Sept 29]. Available from: http://www.diabetesindia.com/diabetes/itfdci.htm.

4. Ebrahim S, Kinra S, Bowen L, Anderson E, Shlomo YB, Lyngdoh T et al. The Effect of Rural-to-Urban Migration on Obesity and Diabetes in India: A Cross-Sectional Study. PLoS Med [Serial on Internet].2010 Apr [cited 2010 Sept 10];7:[About 12 pages]. Available from: http://www.plosmedicine.org/article/info%3Adoi%2F10.1371%2Fjournal.pmed.100026.

5. Ventura CS. The History of Diabetes Mellitus A Maltese perspective. Malta: the author;2002.

6. Canadian Diabetes Association [homepage on the Internet]. Toronto: The Association[cited 2012 Sept 15].The History of Diabetes. [About 5 screens]. Available from: http://www.diabetes.ca/diabetes-and-you/what/history/.

7. Powers AC. Diabetes Mellitus. In:Longo DL, Kasper DL, Jameson JL, Fauci AS, Hauser SL, Loscalzo J, editors. Harrison's Principles of Internal Medicine. 18th ed. New York: Mc Grawhill;2012. p.2968-3002.

8. American Diabetes Association. Diagnosis and Classification of Diabetes Mellitus. Diabetes Care 2010;33:s62-69.

9. World Health Organisation. WHO Expert Committee for Diabetes. Geneva: WHO;1980. 80 p.

10. World Health Organization, Department of Noncommunicable Disease Surveillance. Definition, Diagnosis and Classification of Diabetes Mellitus and its Complications. Geneva: WHO;1999.59p.

11. Ramachandran A, Ma RCW, Snehalatha C. Diabetes in Asia. Lancet 2010; 375: 408–18.

12. Silha JV, Krsek M, Skrha JV, Sucharda P, Nyomba BLG, Murphy LJ. Plasma resistin, adiponectin and leptin levels in lean and obese subjects: correlations with insulin resistance. Eur J Endocrinol 2003;149: 331–35.

13. Seufert J. Leptin Effects on Pancreatic -Cell Gene Expression and Function. Diabetes 2004;53:s152-s158.

14. Klein S, Sheard NF, Sunyer XP, Daly A, Rosett JW, Kulkarni K. Weight Management Through Lifestyle Modification for the Prevention and Management of Type 2 Diabetes: Rationale and Strategies. Diabetes Care 2004; 27:2067-73.

15. Li CH, Chen SY, Lan C, Pan WH, Chou HC, Bai YB. The effects of physical activity, body mass index (BMI) and waist circumference (WC) on glucose intolerance in older people: A nationwide study from Taiwan. Arch Gerontol Geriatr 2011;52:54–59.

16. Boffetta P, McLerran D, Chen Y, Inoue M, Sinha R, He J et al . Body Mass Index and Diabetes in Asia: A Cross-Sectional Pooled Analysis of 900,000 Individuals in the Asia Cohort Consortium Paolo Boffetta. PLoS One. [serial on the Internet] 2011 Jun [cited 2012 mar 30];6:[about 10 p.]. Available from: http://www.plosone.org/article/info:doi/10.1371/journal.pone.0019930.
17. Snehalatha C, Viswanathan V, Ramachandran A. Cutoff Values for Normal Anthropometric Variables in Asian Indian Adults. Diabetes Care 2003;26:1380–84.
18. World Health Organisation. The Asia Pacific perspective: Redefining obesity and its treatment. Australia: Health Communications;2000.
19. Yadav K, Krishnan A. Changing patterns of diet, physical activity and obesity among urban, rural and slum populations in north India. Obes Rev 2008 Sep;9:400-8.
20. Chandalia M., Lin P, Seenivasan T, Livingston EH, Snell PG, Grundy SM et al. Insulin Resistance and Body Fat Distribution in South Asian Men Compared to Caucasian Men. PLoS One 2007;2: e812.
21. Weyer C, Foley JE, Tataranni PA, Pratley RE. Enlarged subcutaneous abdominal adipocyte size, but not obesity itself, predicts Type 2 diabetes independent of insulin resistance. Diabetologia 2000;43:1498-1506.
22. Sahay BK. dietary carbohydrate content in Indian diabetic patients. Med Update 2012;22:235-39.
23. Neel JV. Diabetes Mellitus: A “Thrifty” Genotype Rendered Detrimental by “Progress”? Am J Hum Genet 1962;14:353–62.
24. Mitra A , Basu B, Mukherjee S. Significance of Different Dietary Habits in Sections of Indian Diabetics. J Hum Ecol 2009; 26: 89-98.

25. Hu EA, Pan A, Malik V, Sun Q. White rice consumption and risk of type 2 diabetes: meta-analysis and systematic review. Br J Med [serial on the Internet]. 2012 Jan 19. [cited 2012 Sept 1]; 344: [about 3 Screens]. Available from: http://www.bmj.com/content/344/bmj.e1454.

26. Coppell KJ, Kataoka M, Williams SM, Chisholm AW, Vorgers SM, Mann J. Nutritional intervention in patients with type 2 diabetes who are hyperglycaemic despite optimised drug treatment—Lifestyle Over and Above Drugs in Diabetes (LOADD) study: randomised controlled trial. Br Med J 2010;341:3337-44.

27. Kelley DE, Wing R, Buonocore C, Sturis J, Polonsky K, Fitzsimmons M. Relative Effects of Calorie Restriction and Weight Loss in Noninsulin-Dependent Diabetes Mellitus. J Clin Endocrinol Metab 1993;77:1287-93.

28. Nazni P, Vijayakumar TP, Angamuthu K. Effect of Exercise on BMI and Biochemical Profile of Selected Obese Diabetic Women. J Exerc Sci Physiother 2006;2:66-70.

29. Riserus U, Willett WC, Hu FB. Dietary fats and prevention of type 2 diabetes. Prog Lipid Res2009;48: 44–51.

30. Salmerón J, Hu FB, Manson JAE, Stampfer MJ, Colditz GA, Rimm EB. Dietary fat intake and risk of type 2 diabetes in women. Am J Clin Nutr 2001;73:1019–26.

31. Henry RR, Scheaffer L, Olefsky JM. Glycemic Effects of Intensive Caloric Restriction and Isocaloric Refeeding in Noninsulin-Dependent Diabetes Mellitus. J Clin Endocrinol Metab 1985;61:917-25.

32. Thompson PD, Buchner D, PiN IL, Balady GJ, Williams MA, Marcus BH et al. Exercise and Physical Activity in the Prevention and Treatment of Atherosclerotic Cardiovascular Disease. Circulation 2003;107:3109-16.

33. Sigal RJ, Kenny GP, Wasserman DH, Sceppa CC. Physical Activity/Exercise And Type 2 Diabetes. Diabetes Care 2004;27:2518-39.

34. Booth FW, Chakravarthy MV, Spangenburg EE. Exercise and gene expression: physiological regulation of the human genome through physical activity. J Physiol 2002; 543: 399–411.

35. Speakman JR. Thrifty genes for obesity, an attractive but flawed idea, and an alternative perspective: the 'drifty gene' hypothesisThe 'drifty gene' hypothesis. Int J Obes 2008;32:1611-17.

36. Wijesekara N, Thong FSL, Antonescu CN, Klip A. Diverse Signals Regulate Glucose Uptake into Skeletal Muscle. Canadian J Diabetes 2006;30:80-88.

37. Merry TL, McConell GK. Skeletal Muscle Glucose Uptake During Exercise: A Focus on Reactive Oxygen Species and Nitric Oxide Signaling. IUBMB Life 2009; 61:479–84.

38. Defay R, Delcourt C, Ranvier M, Lacroux A, Papoz L. Relationships between physical activity, obesity and Diabetes Mellitus in a French elderly population: the POLA study. Int J Obes 2001;25:512-18.

39. Seyyednozadi M, Shakeri MT, Rajabian R, Vacfaee A. Role of physical activity and nutrition in controlling Type 2 Diabetes Mellitus – 2007. J Biol Sci 2007;8:794–98.

40. Umpierre D, Ribeiro PAB, Kramer CK, Leitão CB, Zucatti ATN, Azevedo MJ. Physical Activity Advice Only or Structured Exercise Training and Association With HbA_{1c}Levels in Type 2 Diabetes. J Am Med Assoc 2011;305:1790-1799.

41. American Diabetes Association. Standards of Medical Care in Diabetes—2011. Diabetes Care 2011;34(1):S11-S61.

42. Ponsonby AN, Sun C, Ukoumunne OC, Pezic A, Venn A, Shaw JE et al. Objectively Measured Physical Activity and the Subsequent Risk of Incident Dysglycemia. Diabetes Care 2011: 34;1497–1502.

43. Helmink JHM, Kremers SPJ, Brussel-Visser FNV, de Vries NK. Sitting Time and Body Mass Index in Diabetics and Pre-Diabetics Willing to Participate in a Lifestyle Intervention. Int J Environ Res Public Health 2011: 8;3747-58.

44. Kriska AM, Saremi A, Hanson RL, Bennett PH, Kobes S, Williams DE et al. Physical Activity, Obesity, and the Incidence of Type 2 Diabetes in a High-Risk Population. Am J Epidemiol 2003;158:669–75.

45. Weinstein AR, Sesso HD. Joint Effects of Physical Activity and Body Weight on Diabetes and Cardiovascular Disease. Exerc Sport Sci Rev 2005;34:10-15.

46. Dwyer T, Ponsonby AL, Ukoumunne OC, Pezic A, Venn A, Dunstan D et al. Association of change in daily step count over five years with insulin sensitivity and adiposity: population based cohort study. Br Med J 2011;341:c7249.

47. Boussageon R, Angoulvant TB, Elahi MS, Lafont S, Bergeonneau C, Kassaï B et al. Effect of intensive glucose lowering treatment on all cause mortality, cardiovascular death, and microvascular events in type 2 diabetes: meta-analysis of randomised controlled trials. Br J Med [serial on the Internet]. 2011 July 26. [cited 2012 Aug 4]; 343: [about 12 pages]. Available from: http://www.bmj.com/content/343/bmj.d4169.

48. Brownlee M. Biochemistry and molecular cell biology of diabetic complications. Nature 2001; 414:813-20.

49. FAO/WHO. Energy and protein requirements, Report of a Joint FAO/WHO Ad Hoc Expert Committee. Geneva:FAO/ WHO; 1973.p. 118. Report No.:FAO Nutr. Meet, Rep. Ser. No. 52; WHO Tech. Rep. Ser. No. 522.

50. National Institute of Nutrition. Dietary Guidelines for Indians -A Manual. 2nd ed. Hyderabad: National Institute of Nutrition;2010.

51. Vaz M. Evaluating Habitual Physical Activity Patterns. State Level Workshop on Human Experiments in Physiology – Setting up innovative experiments with minimal equipment and expenditure; 2000 Jan 28-29; Bangalore. p. 70-81.

52. FAO/WHO/UNU Expert Consultation. Energy and protein requirements. Geneva: FAO/WHO/UNU;1985. p.120. Technical Report Series 724.

53. Mohan V, Shanthirani CS, Deepa R. Glucose intolerance (DM and IGT) in a selected South Indian population with special reference to family history, obesity and lifestyle factors – the Chennai urban population study (CUPS 14). J Asso Physicians India 2003;51:756-57.

54. Patel M, Patel IM, Patel YM, Rathi SK. A Hospital-based Observational Study of Type 2 Diabetic Subjects from Gujarat, India. J Health Popul Nutr 2011;29:265-72.

55. Adebayo FO. Dietary habits and prevalence of obesity among type 2 Diabetes patients seen at Scott Hospital, Morija, Lesotho [dissertation]. Morija: University of Limpopo;2007.

56. Daousi C, Casson I F, Gill G V, MacFarlane I A, Wilding J P H, J H Pinkney JH. Prevalence of obesity in type 2 diabetes in secondary care: association with cardiovascular risk factors. Postgrad Med J 2006;82:280-84.

57. Klesges RC, Eck LH, Ray JW. Who underreports dietary intake in a dietary recall? Evidence from the Second National Health and Nutrition Examination Survey. J Consult Clin Psychol 1995 Jun;633:438-44.

58. Villegas R, Liu S, Gao YT, Yang G, Li H, Zheng W et al. Prospective study of dietary carbohydrates, glycemic index, glycemic load, and incidence of type 2 diabetes mellitus in middle-aged Chinese women. Arch Intern Med 2007;167:2310-6.

59. Radhika G, Dam RMV, Sudha V, Ganesan A, Mohan V. Refined grain consumption and the metabolic syndrome in urban Asian Indians (Chennai Urban Rural Epidemiology Study 57). Metabolism 2009;58:675-81.

60. Raghuram TC, Pasricha S, Upadhyaya AC, Krishnaswamy K. Glycaemic Index Of Indian Foods. Hyderabad: National Institute of Nutrition; 1987.6 p.

61. Torangatti G, Naik Rk. Glycemic And Lipemic Response of Composite Foods In Normal and NIDDM Subjects. Int J Diab Dev Countries 1999;19:167-71.

62. Mani UV, Prabhu BM, Damle SS, Mani I. Glycaemic index of some commonly consumed foods in western India. Asia Pacific J Clin Nutr 1993; 2:111-114.

63. Powell KF, Holt SHA, Miller JCB. International table of glycemic index and glycemic load values: 2002. Am J Clin Nutr 2002;76:5–56.

64. Devi PB, Vijayabharathi R, Sathyabama S, Malleshi NG, Priyadarisini VB. Health benefits of finger millet (Eleusine coracana L.) polyphenols and dietary fiber: a review. J Food Sci Technol [serial on the Internet]. 22 November 2011 [cited 2012 Sept 12]:[About 20 screens]. Available from: http://link.springer.com/article/10.1007/s13197-011-0584-9.

65. Shobana S. Health Benefits of Ragi.In: Pradeepa RG,editor. Diabetes Monitor. 32 ed. Chennai: Direct, A Charitable Trust for Diabetes;2011. p. 9-11.

66. Sramkova Z, Gregova E, Sturdik E. Chemical composition and nutritional quality of wheat grain. Acta Chimica Slovaca 2009;2:115–138.

67. Mohan V, Radhika G, Vijayalakshmi P, Sudha V. Can the diabetes/cardiovascular disease epidemic in India be explained, at least in part, by excess refined grain (rice) intake? Indian J Med Res 2010;131: 369-72.

68. Gannon MC, NuttallFQ , Saeed A, Jordan K, Hoover H. An increase in dietary protein improves the blood glucose response in persons with type 2 diabetes. Am J Clin Nutr 2003;78:734–41.

69. Puri S, Kalia M, Swami M, Singh A, Abhimanyu, Mangat C, Kaur A, Kaur S et al . Profile of diabetes mellitus in elderly of Chandigarh, India. Internet J Endocrinol [ISSN 1540 – 2606]. 2007 [cited 2010 Oct 14];4. Available from: http://www.ispub.com/journal/the_internet_journal_of_endocrinology/volume_4_number_1_16/ article/profile_of_diabetes_mellitus_in_elderly_of_chandigarh_india.html.

70. Shazwani MNN, Suzana S, Mastura YH, Lim CJ1, Teh SC, Fauzee MZM. Assessment of Physical Activity Level among Individuals with Type 2 Diabetes Mellitus at Cheras Health Clinic, Kuala Lumpur. Mal J Nutr 2010;16:101-12.

71. Looker HC, Knowler WC, Hanson RL. Changes in BMI and Weight Before andAfter the Development of Type 2 Diabetes. Diabetes Care 2001; 24:1917-22.

72. Insulin, Glucagon and Diabetes Mellitus. In: Guyton AC, Hall JE, editors. Textbook of Medical Physiology. Philadelphia: Elsevier Saunders;2011. p. 939-54.
73. Zisser H, Sueyoshi M, Krigstein K, Szigiato A,Riddell MC. Advances in exercise, physical activity and diabetes mellitus. Int J Clin Pract 2012;66: 62–71.

ANNEXURES

ANNEXURE 1

GENERAL INFORMATION:

1. NAME:

2. AGE:

3. SEX:

4. OCCUPATION:

5. DURATION OF DM:

6. WEIGHT:

7. HEIGHT:

ANNEXURE 2

BMI Cut off values (ASIAN STANDARDS) [2]

Category	**BMI range – kg/m^2**
Emaciation	less than 14.9
Underweight	from 15 to 18.4
Normal	from 18.5 to 22.9
Overweight	from 23 to 27.5
Obese	from 27.6 to 40
Morbid obesity	More than 40

ANNEXURE 3

Questionnaire for dietary recall:

S. No.	Time	Food Item	Quantity taken each time
			It was estimated by showing a known volume of utensil glass and cup).

Daily Recommended Calories intake :

MALES		
AGE (Years)	**TYPE OF WORK**	**kCAL/DAY**
Up to 40	Sedentary	2425
	Moderately active	2875
	Heavy worker	3800
40 – 50	Sedentary	2303
	Moderately active	2731
	Heavy worker	3610
50 – 60	Sedentary	2182
	Moderately active	2587
	Heavy worker	3420
> 60	Sedentary	1940
	Moderately active	2300
	Heavy worker	3040

FEMALES		
AGE (Years)	**TYPE OF WORK**	**kCAL/DAY**
Up to 40	Sedentary	1875
	Moderately active	2225
	Heavy worker	2925
40 – 50	Sedentary	1781
	Moderately active	2113
	Heavy worker	2779
50 – 60	Sedentary	1687
	Moderately active	2002
	Heavy worker	2633
> 60	Sedentary	1500
	Moderately active	1780
	Heavy worker	2340

- Daily Protein Requirement for both adult Males and Females is – 1 gm / kg Body Weight.

ANNEXURE 4

Questionnaire for Physical Activity

1. a) On an average how many hours per day do you spend at work :

   b) Of the hours you spend at work how many hours do you spend:

2. On an average how many hours do you sleep in a day:

3. How do you normally travel to and from work:

4. Apart from work how do you spend time:

Type of activity	**Average duration in min**	**Daily**	**Weekly**
Sports /exercise			
Hobbies involving manual activity like gardening			
Household chores –cooking, sweeping			
Sedentary activity – reading, watching TV			
Eating			
Brushing and bathing			
Dressing			
Socializing (talking)			

ANNEXURE 5:

BMR for different age groups is given by:

	Age	**Equation(BMR in kJ)**
Males	10 – 18	69.4W+322.2H+2392
	18 – 30	64.4W-113.0H+3000
	30 – 60	47.2W+66.9H+3769
	>60	36.8W+4719.5H-4481
Females	10 – 18	30.9W+2016.6H+907
	18 – 30	55.6W+1397.4H+146
	30 – 60	36.4W-104.6H+3619
	>60	38.5W+2665.2H-1264
Where W – Weight in kgs H – Height in metres		

ANNEXURE 6 (MASTER CHART)

S.No.	Age (years)	Gender	Duration of DM (years)	Weight (kg)	Height (meters)	Calorie intake (KCal)	Protein intake (gm)	Physical activity level
1	76	M	22	65	1.77	1462	26.5	Sedentary
2	80	M	29	52	1.68	972	42.9	Sedentary
3	55	M	4	51	1.78	1963	41.6	Heavy
4	62	M	7	74	1.6	2087	68.3	Sedentary
5	60	M	2	60	1.68	1780	43.4	Sedentary
6	65	M	6	45	1.6	1006	41.8	Sedentary
7	42	M	3	76	1.65	3343	81.5	Heavy
8	71	M	9	78	1.68	2213	59	Sedentary
9	60	M	9	60	1.57	2121	40.8	Moderate
10	63	M	3	61	1.53	2028	46.1	Sedentary
11	65	M	4	55	1.78	1686	44.5	Heavy
12	65	M	12	46	1.62	1984	35.2	Heavy
13	50	M	3	60	1.78	2798	58.5	Heavy
14	50	M	6	105	1.76	2963	105.8	Sedentary
15	69	M	7	47	1.72	1384	42	Sedentary
16	45	M	2	56	1.61	2109	34	Heavy
17	57	M	6	50	1.69	1728	22.4	Moderate
18	78	M	20	51	1.75	1423	27.7	Sedentary
19	62	F	5	42	1.62	1384	42	Sedentary
20	60	F	8	54	1.52	1465	42.8	Sedentary
21	50	F	8	49	1.6	1173	32.8	Moderate

22	55	F	2	44	1.55	1060	29	Moderate
23	75	F	7	60	1.6	1490	43.5	Sedentary
24	50	F	2	51	1.48	1483	55.1	Moderate
25	68	F	4	39	1.56	611	19.5	Sedentary
26	61	F	3	63	1.55	2024	61.9	Sedentary
27	77	F	13	61	1.48	2095	75	Sedentary
28	45	M	1	62	1.71	1348	45.2	Sedentary
29	74	M	15	65	1.72	1376	42.8	Sedentary
30	60	M	16	62	1.7	1300	43.3	Moderate
31	40	F	3	46	1.45	1955	33.6	Heavy
32	67	F	9	84	1.68	2250	57.9	Sedentary
33	61	M	3	94	1.84	2516	77.6	Sedentary
34	55	F	7	75	1.7	1836	53.1	Sedentary
35	53	F	2	45	1.61	1219	22.5	Moderate
36	56	M	4	52	1.7	1534	34.9	Heavy
37	54	F	2	56	1.49	2438	43.2	Moderate
38	50	F	3	55	1.6	1457	38.4	Moderate
39	70	M	21	50	1.68	772	27.5	Sedentary
40	58	M	2	63	1.8	1844	40.9	Heavy
41	45	F	2	65	1.6	2311	72.9	Moderate
42	55	F	7	50	1.68	1390	36.9	Sedentary
43	65	F	3	47	1.61	1156	40.2	Sedentary
44	73	F	12	74	1.5	2574	71.8	Sedentary
45	65	F	10	62	1.49	2009	74.5	Sedentary
46	47	M	2	60	1.55	2902	35	Heavy

47	64	M	6	61	1.56	1787	46	Sedentary
48	76	F	25	46	1.58	1163	30.2	Sedentary
49	61	M	8	55	1.68	1755	53.3	Heavy
50	50	M	9	80	1.52	2432	87.5	Sedentary
51	63	M	20	70	1.56	1281	28.8	Sedentary
52	51	M	5	89	1.61	3199	89.6	Sedentary
53	41	M	2	56	1.48	1605	47.8	Moderate
54	69	F	20	92	1.59	2605	71.8	Sedentary
55	71	M	21	54	1.49	1362	44.7	Sedentary
56	55	M	9	62	1.61	2035	56	Sedentary
57	72	M	12	82	1.69	3121	73.1	Sedentary
58	39	F	2	78	1.42	2547	79.2	Sedentary
59	39	M	4	43	1.54	1566.5	40.4	Heavy
60	60	M	10	82	1.72	2586	64.8	Heavy
61	60	M	15	91	1.69	2757.5	108.1	Sedentary
62	41	F	2	58	1.43	1589	60.9	Moderate
63	45	M	4	89	1.78	2426.5	82.8	Moderate
64	70	M	16	60	1.52	1836	40.4	Sedentary
65	38	F	3	78	1.5	2256	75.6	Sedentary
66	39	M	1	80	1.52	1951	71	Sedentary
67	43	F	1	51	1.53	883	28.3	Moderate
68	72	M	17	80	1.69	1781	46.9	Sedentary
69	52	M	5	72	1.52	2310	41.8	Moderate
70	41	M	3	89	1.62	2260.5	88.8	Sedentary
71	55	M	5	48	1.7	996	26.8	Heavy

72	82	F	19	68	1.6	1530	16.2	Sedentary
73	51	F	6	80	1.52	2016	27.1	Sedentary
74	68	F	12	60	1.52	1886	32.9	Sedentary
75	44	M	2	78	1.72	1819	60.4	Sedentary
76	71	M	18	51	1.49	926.5	35.1	Sedentary
77	38	M	2	81	1.59	2050	71	Sedentary
78	49	F	2	49	1.53	1003	22.1	Moderate
79	40	F	1	83	1.62	2192	65	Moderate
80	70	M	11	70	1.71	1277	40.2	Sedentary
81	57	M	5	78	1.55	2118	58.2	Sedentary
82	67	F	0.5	72	1.48	1955	58.7	Sedentary
83	66	M	6	54	1.59	1455	42.4	Heavy
84	42	M	2	60	1.52	846	25.3	Sedentary
85	59	F	8	89	1.62	1844	66.4	Sedentary
86	45	F	2	42	1.52	717	25.6	Heavy
87	57	M	2	75	1.63	2095	61.8	Heavy
88	34	M	1	64	1.5	1521	33.2	Heavy
89	52	M	3	60	1.68	1642	47	Heavy
90	73	M	12	62	1.7	1520	36.6	Sedentary
91	57	F	8	71	1.48	2825	60.4	Sedentary
92	48	F	4	54	1.49	2552	76.2	Moderate
93	58	M	10	56	1.52	2320	57.8	Sedentary
94	50	M	10	74	1.64	1767	60	Sedentary
95	37	F	10	65	1.5	2368	81	Sedentary
96	40	F	3	57	1.57	1851	57.8	Sedentary

97	65	M	9	86	1.67	2357	90.8	Sedentary
98	68	F	15	80	1.54	2216	78.1	Sedentary
99	70	M	15	62	1.57	2079	84.1	Sedentary
100	50	M	4	69	1.61	2453	55.3	Sedentary
101	63	F	7	59	1.5	2147	66.6	Sedentary
102	76	M	5	57	1.6	1962	73.9	Sedentary
103	72	F	5	65	1.48	2596.5	73.3	Moderate
104	61	M	2	61	1.59	917.5	31.6	Heavy
105	67	F	2	75	1.8	2578	55	Sedentary
106	47	M	2	68	1.62	1848	62.2	Sedentary
107	66	F	3	75	1.49	2095	54.8	Sedentary
108	42	M	4	71	1.51	2421	53.1	Sedentary
109	60	M	10	58	1.68	2314	53	Sedentary
110	37	M	2	54	1.52	1973	65.5	Sedentary
111	56	F	5	60	1.51	1123	32.8	Moderate
112	62	M	4	72	1.72	2686	63	Sedentary
113	72	M	10	69	1.61	1879	46.6	Sedentary
114	49	M	5	69	1.59	2006	59.7	Heavy
115	60	M	5	64	1.57	2109	63.6	Sedentary
116	71	M	13	110	1.65	3732	138.6	Sedentary
117	71	M	15	65	1.54	2169	52.1	Sedentary
118	48	M	1	66	1.69	2718	60.3	Heavy
119	62	F	8	80	1.48	2853	75.3	Sedentary
120	51	M	6.5	64	1.52	2492	78.5	Sedentary
121	55	M	5	70	1.63	2444	66.3	Heavy

122	49	M	1	81	1.68	2859	62.4	Sedentary
123	67	M	9	66	1.56	2098	57	Moderate
124	40	M	2	74	1.58	923	28.6	Sedentary
125	49	F	2	81	1.6	1922	48.4	Sedentary
126	39	M	1	78	1.76	1364	37.1	Heavy
127	43	F	1	68	1.53	1133	32.7	Sedentary
128	55	M	5	81	1.63	3228	100.6	Moderate
129	68	F	12	78	1.49	2418	57.6	Sedentary
130	51	F	3	76	1.6	2578	79.4	Sedentary
131	48	F	2	54	1.58	1395.5	44	Sedentary
132	52	M	6	72	1.78	2462	67.4	Moderate
133	50	M	5	62	1.71	2223	47	Sedentary
134	49	M	3	54	1.71	1315.5	47.5	Heavy
135	47	M	4	51	1.49	989	33	Sedentary
136	45	M	2	84	1.7	3323	84.9	Heavy
137	71	M	22	62	1.59	1483	39.9	Sedentary
138	57	M	6	50	1.41	2437	76.8	Moderate
139	49	M	3	78	1.57	2506	85.4	Sedentary
140	48	M	5	68	1.71	1838	52.4	Sedentary
141	60	M	12	59	1.72	1400	47.8	Sedentary
142	79	M	18	81	1.63	2463	64.8	Sedentary
143	61	M	10	72	1.51	2410	58.2	Sedentary
144	60	F	10	52	1.51	1609.5	41.9	Sedentary
145	39	M	1	84	1.69	2641	79.9	Sedentary
146	41	M	3	69	1.71	1871.5	51.2	Heavy

147	61	F	5	61	1.58	1225	38.9	Sedentary
148	60	M	12	72	1.75	1364	36.1	Sedentary
149	70	M	20	50	1.59	1124	32.1	Sedentary
150	52	F	5	78	1.51	2947	84.1	Sedentary
151	68	M	10	66	1.55	2028	42.1	Sedentary
152	55	F	1	70	1.5	2617.5	88.3	Sedentary
153	40	M	0.5	62	1.45	2325	63.6	Sedentary
154	37	M	1	60	1.5	2674	75.5	Heavy
155	75	M	12	55	1.55	1232	38.7	Sedentary
156	48	M	2	59	1.65	1047	40.1	Heavy
157	72	M	6	60	1.68	1295	36.6	Sedentary
158	60	M	2	49	1.52	1094	34.4	Moderate
159	64	M	4	72	1.68	2557	49.1	Heavy
160	62	M	1	65	1.59	2329.5	61.2	Heavy
161	60	M	10	75	1.6	2393.5	70.5	Sedentary
162	60	M	5	57	1.6	1921	36.6	Heavy
163	45	M	10	73	1.73	1849	53.9	Sedentary
164	45	F	0.5	64	1.52	1931	71	Sedentary
165	70	M	10	93	1.61	2540	60.4	Sedentary
166	65	M	1	75	1.63	2425	63.4	Sedentary
167	60	M	0.5	75	1.53	2953.5	88.5	Heavy
168	48	M	3	65	1.55	2493	60	Sedentary
169	65	M	7	62	1.69	1846	45.2	Sedentary
170	69	M	13	79	1.68	2219.5	50.9	Sedentary
171	79	M	20	52	1.62	1596	38.3	Sedentary

172	45	M	3	68	1.75	1805	50.2	Moderate
173	59	M	8	78	1.71	2463	56.9	Sedentary
174	46	M	3	59	1.61	2408	62.9	Sedentary
175	72	M	27	59	1.65	1566	60.3	Sedentary
176	41	M	1	67	1.52	2700	69.1	Sedentary
177	65	M	19	62	1.74	2100	44.4	Sedentary
178	51	M	4	81	1.68	3137	73.3	Sedentary
179	68	M	16	67	1.71	2070	56.1	Sedentary
180	40	M	3	95	1.74	3314	91.4	Sedentary
181	81	M	30	54	1.71	1222	39.4	Sedentary
182	51	M	6	73	1.8	2801	69.8	Moderate
183	76	M	26	51	1.6	1368	38	Sedentary
184	66	M	16	55	1.61	1260	32.7	Sedentary
185	46	M	2	61	1.59	2265	57.1	Sedentary
186	55	M	9	69	1.7	3312	69	Moderate
187	75	M	20	57	1.63	1564	37.5	Sedentary
188	64	M	2	79	1.73	2440	41.1	Sedentary
189	39	M	2	78	1.63	3772	74.6	Sedentary
190	56	M	8	64	1.71	2005	54.5	Sedentary
191	45	M	2	69	1.58	2950	72.2	Heavy
192	61	M	8	78	1.79	2730	62.4	Sedentary
193	54	M	5	67	1.65	2373	57.3	Moderate
194	43	F	2	76	1.59	2364.5	77.4	Sedentary
195	77	M	25	60	1.69	2022	59.9	Sedentary
196	51	F	4	59	1.61	2182	52.3	Moderate

197	69	F	17	65	1.52	1887	56.6	Sedentary
198	57	F	3	70	1.69	2796	68.5	Sedentary
199	72	F	16	56	1.59	1340	44.4	Sedentary
200	64	F	9	65	1.6	1794	45.9	Sedentary
201	49	F	2	85	1.69	2540	64.1	Sedentary
202	71	M	14	59	1.65	1316	39.1	Sedentary
203	61	F	13	62	1.51	2265	55.9	Sedentary
204	52	M	6	78	1.75	2752	75.6	Moderate
205	46	M	1	69	1.67	2137	57.3	Heavy
206	45	F	2	70	1.62	2506	54.9	Moderate
207	51	F	1	61	1.69	1938	50.6	Sedentary
208	65	F	12	85	1.69	3148	83.5	Sedentary
209	71	F	16	56	1.6	1236	34.1	Sedentary
210	62	M	9	67	1.71	1961	45	Moderate
211	62	F	6	62	1.67	2120	49	Sedentary
212	69	M	15	70	1.68	2182	53.9	Sedentary
213	47	M	6	76	1.73	2751	69.4	Sedentary
214	56	M	2	69	1.56	2660	52.8	Heavy
215	56	M	9	70	1.75	1947	38.7	Moderate
216	45	M	1	59	1.7	2071	40.6	Heavy
217	57	M	5	59	1.73	2330	44.2	Heavy
218	59	F	5	85	1.7	2992	65.9	Sedentary
219	75	M	22	57	1.69	1575	48.6	Sedentary
220	67	F	6	56	1.6	1168	34.5	Sedentary
221	49	F	3	69	1.62	2277	63.7	Sedentary

222	65	M	6	69	1.7	2107	52.9	Sedentary
223	56	M	8	78	1.68	2836	65.9	Sedentary
224	56	M	2	51	1.56	1368	34	Sedentary
225	42	M	1	71	1.65	2679	62.9	Heavy
226	65	F	7	67	1.67	2048	45.5	Sedentary
227	53	M	1	69	1.59	2475	68	Moderate
228	61	M	6	65	1.73	1855	51	Moderate
229	43	M	0.5	60	1.65	2515.5	47.7	Heavy
230	62	M	6	64	1.63	2002	50.2	Sedentary
231	46	M	1	49	1.51	2109	58	Moderate
232	70	F	11	58	1.63	1308	36	Sedentary
233	49	M	2	64	170	2310.5	52.8	Heavy
234	61	M	7	70	1.78	2113	47.9	Sedentary
235	75	F	20	49	1.59	1322	41.7	Sedentary
236	56	M	5	63	1.61	2202.5	61.2	Sedentary
237	56	M	13	60	1.67	1429	34.3	Sedentary
238	48	M	3	72	1.76	2110.5	54.9	Moderate
239	69	F	11	68	1.68	1954	32.5	Sedentary
240	56	M	7	78	1.82	2781	74.7	Heavy
241	51	M	3	65	1.67	2585	58.8	Sedentary
242	74	F	11	51	1.59	1320	37.5	Sedentary
243	50	M	3	61	1.7	2115.5	55.9	Moderate
244	42	M	0.5	72	1.66	2757.5	62.2	Sedentary
245	61	M	4	65	1.63	2140	58.8	Sedentary
246	65	M	16	69	1.73	2343	57.7	Sedentary

247	66	M	12	64	1.72	1388	25.5	Sedentary
248	55	M	10	62	1.7	1749.5	53.1	Moderate
249	43	F	1	71	1.6	2702	70.9	Sedentary
250	52	M	1	69	1.7	2203	62.5	Sedentary
251	46	M	2	59	1.65	2517.5	58.8	Moderate
252	60	M	5	64	1.67	2565	59.3	Moderate
253	73	F	21	49	1.59	1468	44.4	Sedentary
254	63	M	6	62	1.69	2325.5	56.6	Sedentary
255	61	M	7	60	1.58	2020.5	47.2	Sedentary
256	72	M	12	54	1.62	1632	44.3	Sedentary
257	59	M	2	91	1.73	3252	78.6	Sedentary
258	72	M	22	60	1.69	1553	42.1	Sedentary
259	61	F	12	60	1.53	1368	38.4	Sedentary
260	51	F	5	62	1.56	2014.5	74.9	Moderate
261	49	F	5	50	1.61	1655	48	Moderate
262	70	M	21	62	1.71	1806	47.1	Sedentary
263	50	F	3	59	1.67	1835	57.1	Moderate
264	66	M	7	70	1.73	3164.5	78.8	Sedentary
265	42	F	6	69	1.6	1831.5	52	Sedentary
266	56	M	4	69	1.71	2217	50.6	Sedentary
267	49	F	7	62	1.56	2107	54.7	Moderate
268	42	M	0.5	63	1.63	2493	48.5	Moderate
269	44	F	1	71	1.68	2160	64.1	Sedentary
270	53	M	4	80	1.79	3425	101.6	Sedentary
271	63	F	7	56	1.61	1448	38.8	Sedentary

272	49	M	3	62	1.67	2627	67	Sedentary
273	54	F	6	71	1.52	3316	85.2	Sedentary
274	65	F	12	78	1.59	2749	76.3	Sedentary
275	50	M	5	65	1.67	3183	74.1	Heavy
276	65	M	7	78	1.73	2590	69.2	Sedentary
277	59	M	2	64	1.61	2792	60.3	Sedentary
278	45	M	2	69	1.74	3102	81	Sedentary
279	57	M	8	68	1.73	2228	56.4	Heavy
280	48	F	2	78	1.49	3215	83	Sedentary
281	51	M	2	65	1.52	1990.5	55	Sedentary
282	56	M	10	62	1.59	2830	79.5	Heavy
283	45	F	3	68	1.55	2612	63.8	Sedentary
284	56	M	5	98	1.81	2280	38.1	Sedentary
285	69	M	7	52	1.59	1614	36.9	Sedentary
286	61	F	2	60	1.51	1841	48	Sedentary
287	55	M	4	65	1.59	2128	66.1	Sedentary
288	49	M	2	82	1.62	2526	52	Moderate
289	60	M	15	61	1.65	1368	35	Sedentary
290	64	F	16	42	1.51	1424	36.2	Heavy
291	48	M	2	68	1.59	1994	59.6	Moderate
292	70	M	21	52	1.61	1475	39	Sedentary
293	76	M	22	68	1.67	1679	52.7	Sedentary
294	48	F	1	68	1.5	2453	57.1	Sedentary
295	55	F	2	61	1.52	1695.5	46.3	Sedentary
296	71	M	21	64	1.61	1372	34.7	Sedentary

297	51	F	2	72	1.6	2941.5	85.2	Sedentary
298	61	M	5	72	1.61	2219	51.6	Sedentary
299	64	M	10	73	1.64	1611	44.9	Heavy
300	52	M	8	58	1.6	2875	65.9	Sedentary