TO ASSESS THE PREVALENCE AND ATTRIBUTABLE RISK FACTORS OF PREDIABETES IN WESTERN INDIAN POPULATION

A THESIS SUBMITTED TO
THE SAURASHTRA UNIVERSITY, RAJKOT
FOR THE AWARD OF THE DEGREE OF

Doctor of Philosophy

IN
PHARMACY
(FACULTY OF MEDICINE)

RE-ACCREDITED GRADE 'B' BY NAAC (CGPA-2.93)

BY

CHIRAG HARSHADKUMAR SHAH

M. PHARM. (PHARMACOLOGY), PGDPM

GUIDE

PROF. (DR.) NAVIN R. SHETH

(M. PHARM., PH. D., LLB, DBM)

HEAD, DEPARTMENT OF PHARMACEUTICAL SCIENCES,

SAURASHTRA UNIVERSITY, RAJKOT (GUJARAT) INDIA

APRIL-2012

TO ASSESS THE PREVALENCE AND ATTRIBUTABLE RISK FACTORS OF PREDIABETES IN WESTERN INDIAN POPULATION

A THESIS SUBMITTED TO
THE SAURASHTRA UNIVERSITY, RAJKOT
FOR THE AWARD OF THE DEGREE OF

Doctor of Philosophy

IN
PHARMACY
(FACULTY OF MEDICINE)

RE-ACCREDITED GRADE 'B' BY NAAC (CGPA-2.93)

BY

CHIRAG HARSHADKUMAR SHAH
M. PHARM. (PHARMACOLOGY),PGDPM

GUIDE

PROF. (DR.) NAVIN R. SHETH
(M. PHARM., PH. D., LLB, DBM)
HEAD, DEPARTMENT OF PHARMACEUTICAL SCIENCES,
SAURASHTRA UNIVERSITY, RAJKOT (GUJARAT) INDIA

APRIL-2012

RE-ACCREDITED GRADE 'B' BY NAAC (CGPA-2.93)

CERTIFICATE

This is to certify that the thesis entitled **"To Assess the Prevalence and Attributable Risk Factors of Prediabetes in Western Indian Population"** represents bonafide and genuine research work of Mr. Chirag Harshadkumar Shah carried out under my guidance and supervision. The work presented in this dissertation was carried through various medical camps across Gujarat and is upto my satisfaction.

Date:
Place: Raikot

Prof. (Dr.) Navin R. Sheth
Head,
Department of Pharmaceutical Sciences, Saurashtra University, Rajkot, Gujarat, India.

RE-ACCREDITED GRADE 'B' BY NAAC (CGPA-2.93)

DECLARATION

I hereby declare that thesis entitled **"To Assess the Prevalence and Attributable Risk Factors of Prediabetes in Western Indian Population"** is a bonafide and genuine research work carried out by me, under the guidance of **Prof. (Dr.) Navin R. Sheth**, Head, Department of Pharmaceutical Sciences, Saurashtra University, Rajkot, Gujarat, India. The results presented in this dissertation are original and has not been submitted in part or full for any degree/diploma to any University.

Date:
Place: Rajkot

Mr.Chirag Harshadkumar Shah
Reg. No. 3485
Department of Pharmaceutical Sciences, Saurashtra University, Rajkot 360005.

RE-ACCREDITED GRADE 'B' BY NAAC (CGPA-2.93)

COPYRIGHT

DECLARATION BY THE CANDIDATE

I hereby declare that the Saurashtra University, Rajkot shall have the right to preserve, use and disseminate this dissertation/thesis in print or electronic format for academic/research purpose.

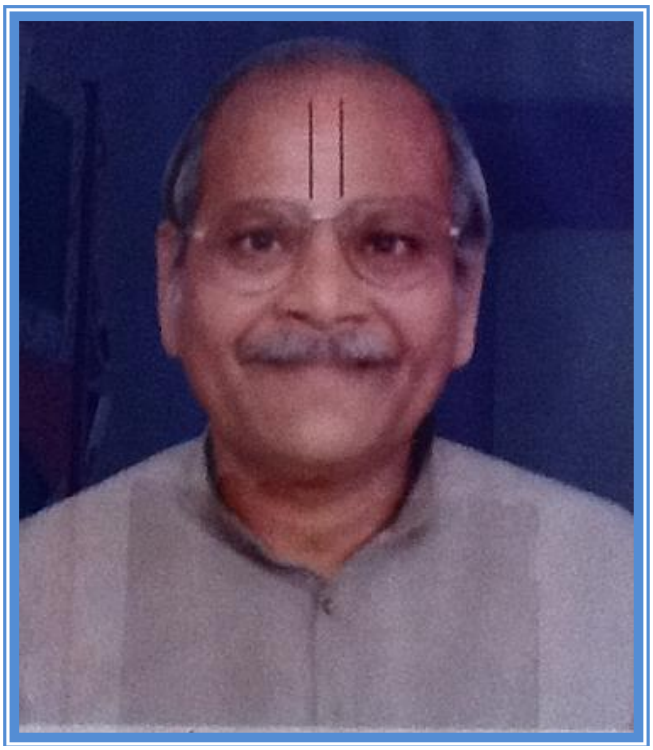
Date:
Place: Rajkot

Mr. Chirag Harshadkumar Shah
Reg. No. 3485
Department of Pharmaceutical Sciences, Saurashtra University, Rajkot 360005.

Affectionately Dedicated to,

The God,

Who empowers me every moment for doing the best



Dedicated to my beloved father

Who inspired me always.....

ACKNOWLEDGEMENTS

"Gratitude makes sense of our past, brings peace for today and creates a vision for tomorrow"

I solemnly owe the successful completion of this dissertation work to Almighty for keeping his everlasting blessing on me.

"Working hard, working sincerely, without expectation of results, success or appreciation is the key to reach greater goals" Especially in research, where results are neither easily available nor guaranteed, concentrating merely on results would prove to be worthless. And it is because of the blessing of almighty and my faith in him this work became success.

It is the matter of pride and great elation for me to acknowledge the contribution of many individuals, who have been inspirational, supportive and helpful throughout my work and endowed me with most precious knowledge to see delighting success in my endeavor.

It gives me great pleasure and profound sense of reverence to express my gratitude and thankfulness to my eminent & esteemed research guide Prof.(Dr.) Navin R.Sheth (Head,Dept.of Pharmaceutical Sciences,Saurashtra University, Rajkot) for his constant help, effective guidance, boundless enthusiasm, critical comments and for availing all the facilities required to complete this dissertation work. Despite an incredibly busy schedule, he always found time to provide the valuable feedback and suggestions.

My deepest heartfelt indebtness and gratefulness to former Vice Chancellor Dr.Kamlesh Joshipura and current Vice Chancellor Dr.Mahendra Padaliya, Saurashtra University, Rajkot for their able guidance till successful completion of work.

Parents are next to the God and they are balance of our life. It is my privilege to utilize this golden moment and bow myself to acknowledge my father late Mr.Harshadkumar Shah and my mother Ms.Arvindaben Shah, father in law Hasmukhbhai,mother in law Pushpaben for their blessings and moral support at all the stages of project right from its conceive to the execution.

My deepest heartfelt indebtness and gratefulness to Dr.Bhagirath Solanki for his able guidance, time to time suggestion, critical review and help throughout the dissertation work.

I am very much thankful to Dr.Bhagirath Patel,Dr.Parag Shah and Dr.Himanshu Parikh for rendering their help for conceptualizing the study design.

I must thank my wife Dr.Nimisha Shah for her continuous support and motivation during conduct of all the camps, data entry, analysis and through out dissertation work.

I must acknowledge the financial support provided by Gujarat Council of Science and Technology (GUJCOST), Gandhinagar.

To make this monumental task very easy, the help rendered by Dr.Mayank Joshi and his team mates at Sun Pathology Laboartory, Ahmedabad was remarkable.

This notable task would have not completed with out the sincere, timely and dedicated help from Mr.Pravin for data entry and Mr.Tushar Sakpal for statistical analysis.

It gives me an immense pleasure to acknowledge the valuable guidance of Dr.A.Sankarnarayanan, Dr.D.D.Santani, Dr.V.Addepalli and Dr.R.K.Goyal at every stage of project conduct.

I must acknowledge the support of Harishbhai, Dhamechabhai, Shabnamben and other teaching and nonteaching staff members of Department of Pharmaceutical Sciences, Saurashtra University, Rajkot for their support.

A special thanks to Dr.V.Mohan and Dr.V.Deepa (Dr.Mohan's Diabetes Centre, Chennai) for their help, guidance and suggestion for operational and scientific aspects.

The love, support and encouragement, I received from my daughter Archi and son Atman, can not be expressed in any words and the sacrifice they have given, the time they allowed me to utilize in my research work will be a lifelong and the best gift, I consider most invaluable.

I owe special thanks to my everlasting friends Manish, Devang, Gaurang, Ashish, Yatin, Shardul, Dr.Sujay, Dr.Mudgal, and Dr.Sachin for their all type of help, valuable suggestions and inspiration.

I have no words to express my immense feeling and thanks to my grandfather late shri Vallabhdas,grandmother shrimati Shantaben, brother Parag, sister in law Hetal, sister Shitalben, brother in law Jatinkumar, nephews Pranshu and Samip to their affection and lovable support.

I am also thankful to my previous employers Nicholas Piramal India Ltd and SIRO Clinpharm Ltd, Mumbai, and current employer Cliantha Research Limited, Ahmedabad for granting me the permission and help at every stage.

I also take an opportunity to appeal my profound gratitude to my all family members and relatives for their support in all the ways.

There are many more whose name flashes across my mind that, I am grateful in making this thesis success. It would not be possible to name them separately but I am conscious of my obligation and indebt to all of them collectively.

I have tried to follow this verse **"कर्मंयेवाधिकारास्ते माँ फलेषु कदाचन"**said by the Almighty himself in Bhagwad Gita throughout my life and this research work as well.

I must acknowledge the help of following friends, colleagues, relatives and well wishers, who have helped for the conduct of the camps at various locations.

Sr.No.	Location of Camp	Contributors
1.	Lunawada Dist: Panchmahal	Dr.Bhavin Mehta,Dr.Joeb Rampurwala (Sifa Hospital),Ms.Hemangi Mehta
2.	Sthapatya Apt,Gurukul,Ahmedabad	Mr.Parag Shah, Mr.Nihar Shah,Dr.Kalpeshkumar Patel,Dr.Himmat Jhula,Dr.Nehal Sadhu Mr.Tanay Shah,Mr.Jatin Shah,Ms.Shital Shah
3.	Bayad,Dist:Sabarkantha	Mr.Arpan Shah,Dr.Bipin Shah,Dr.Prakash Shah,Dr.Mahendra Patel
4.	Ubharan, Dist:Sabarkantha	Mr.Harshadbhai Shah,Mr.Dhirajlal Shah Dr.Ashwin Jaiswal,Dr.Sachin Patel,Dr.Bharat Patel, Mr.Gopal Shah
5.	Dhansuara, Dist:Sabarkantha	Mr.Rupesh Shah,Mr.Narendra Shah, Dr.Rakesh Shah Mr.Pravinbhai Shah,Mr.Prakash Desai
6.	Modasa-1 & Modasa -2 Dist:Sabarkantha	Mr.Alpesh Shah,Mr.Hasmukhbhai Shah Dr.Ashwin Patel,Dr.H.D.Shah,Dr.Jamil Khanji, Dr.Nehal Shah,Mr.Mukesh Bhatt,Mr.Mukund Shah,Mr.Prakash Shah, Ms.Alpa Shah
7.	Salal,Dist:Sabarkantha	Mr.Hasmukh Panchal, Mr.Mahesh Vyas
8.	Bapunagar,Ahmedabad	Dr.Shailesh Shah,Mr.Neel Shah,Dr.Vipul Vadera,Dr.Dindesh Desai,Dr.Bhavesh Tadani,Dr.Paresh Parsala,Dr.Jinit & Payal Shah
9.	Meghaninagar,Ahmedabad	Dr.Paresh Patel,Dr.Alpa Patel,Mr.Nihar Shah, Mr.Meet Shah, Mr.Naresh Shah
10.	Munjka & Rajkot	Dr.Navin Sheth,Dr.Sachin Parmar,Dr.Kamlesh Joshipura,M.Pharm Students of Pharmacy Dept.

CHIRAG HARSHADKUMAR SHAH

TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

LIST OF ABBREVIATIONS

ADA: American Diabetes Association

AIR: Acute Insulin Response

AIDS: Acquired Immunodeficiency Syndrome

BMI: Body Mass Index

CRF: Case Record Form

CVD: Cardiovascular Diseases

CODI: Cost of Diabetes in India

DCCT: Diabetes Control and Complications Trial Research Group

DECODA: Diabetes Epidemiology Collaborative Analysis of Diagnostic Criteria in Asia

DECODE: Diabetes Epidemiology Collaborative Analysis of Diagnostic Criteria in Europe

DPP-IV: Dipeptidyl Peptidase IV

DRI: Dietary Reference Intake

DSME: Diabetes Self-Management Education

EASD: European Association of Study in Diabetes

FFA: Free Fatty Acid

FPG: Fasting Plasma Glucose

GDP: Gross Domestic Product

GLP: Glucagon Like Peptide

HDL**:** High Density Lipoprotein

HIV: Human Immunodeficiency Virus

IFG: Impaired Fasting Glucose

IGT: Impaired Glucose Tolerance

LDL: Low Density Lipoprotein

MNT: Medical Nutrition Therapy

NIDDM: Non-Insulin Dependent Diabetes Mellitus

NHANES: National Health and Nutrition Examination Survey

OGTT: Oral Glucose Tolerance Test

QR: Quality Review

RDA: Recommended Dietary Allowance

SAS: Statistical Analysis Software

SD: Standard Deviation

UKPDS: United Kingdom Prospective Diabetes Study

VLDL**:** Very Low Density Lipoprotein

WHO: World Health Organization

WHR: Waist to Hip ratio

1. ABSTRACT:

Prevalence of Type II diabetes is increasing globally as well as in India day by day. Asian Indians have racial predisposition for Diabetes. Urban India is getting industrialized rapidly and Diabetes is now one of the most common non-communicable diseases globally. The country is already being dubbed as the **"Diabetes Capital of the World"**

Governments and medical fraternity across the world have acknowledged that the diabetes is increasing at epidemic rates and affecting all countries. For the first time non infectious disease has been seen as posing as serious a global health threat as infectious epidemics such as HIV/AIDS. Today, diabetes has affected 250 million people or around 6 % of the world's adult population.

As per Diabetes Atlas, population of diabetes is 194 millions in 2003 and projected to 333 millions by 2025.(22)The World Health Organization (WHO) has projected that the global prevalence of type 2 diabetes mellitus will be more than double from 135 millions in 1995 to 300 millions by 2025.

Prevalence of Diabetes in the world was 5.1 % in 2003 and by 2025 it will be around 6.5%. IGT prevalence in the world was 8.2 % in 2003 and projected to be 9.0% in 2025 (age group 20-79 years).

International Diabetes Federation estimates that the number of diabetic patients in India is more than doubled from 19 million in 1995 to 40.9 million in 2007.It is projected to increase by 69.9 million by 2025. This means by 2025 there are nearly around 7 crores people will have diabetes.

Currently up to 11 % of India's urban population and 3 % of rural population above the age of 15 have diabetes. It has been demonstrated, that industrialization and modernization lead to sedentary lifestyle, obesity and higher risk of metabolic disorders.

The risk variables associated with diabetes are almost similar in all nations, but its expressions and intensities vary widely between different races and countries.

The prevalence of diabetes mellitus differs in all the states of India across the rural & urban area. This is because of the different dietary pattern, physical activity and mental status.

In year 2007 & 2008, a cross sectional survey was conducted via mode of camps at various urban and rural part of Gujarat. After obtaining a proper consent form, comprehensive questionnaire was used to collect the various anthropological details, physical examination and blood collection was performed from around 1700 subjects $\geq$ 20 years of age from the different area of Gujarat. Chi square test was used for all categorical comparisons. Also multiple logistic regression was used for detailed exploratory analysis.

We have focused mainly on prediabetes covering both Impaired Fasting Glucose (IFG) and Impaired Glucose Tolerance (IGT). We also identified the relationship of Waist to Hip ratio(WHR),Body Mass Index(BMI),systolic blood pressure, Diastolic blood pressure, Serum Triglycerides, Serum Cholesterol, Serum LDL and Serum HDL on prediabets.We have also evaluated impact of family history of diabetes on the development of prediabetes.

We have also evaluated the correlation of prediabets with the age and found striking observation that the prevalence of prediabets is higher after age of 40 years.

The crude prevalence of IFG in Gujarati population is around 4.23 % and IGT is around 6.82 %.But the age adjusted prevalence of IFG is around 2.72% and IGT is around 4.67%.If we extrapolate these, it indicates that around 1.3 million people are having impaired fasting glucose and around 2.3 million people have impaired glucose tolerance. The prevalence of IGT found more after age of 40 years.

For IFG, there is increase after age of 40 years, but not significant statistically. Based on the findings and observations, we have provided the appropriate counseling to the subjects. We have also made them aware about the importance of diabetes management and the precautions to be taken for its progression.

High prevalence of IGT and IFG validates that, there are chances of the pandemic trend in Gujarat, as eventually IGT and IFG may get converted into Diabetics in near future. These result needs urgent attention to develop a public awareness programme.

2. INTRODUCTION:

Type 2 diabetes mellitus represents the final stage of a chronic and progressive syndrome representing a heterogeneous disorder caused by various combinations of insulin resistance and decreased pancreatic β-cell function caused by both genetic and acquired abnormalities (1-7). Diabetes is the most common metabolic disorder, which is still incurable even today.

Currently, type 2 diabetes mellitus is diagnosed when the underlying metabolic abnormalities consisting of insulin resistance and decreased β-cell function cause elevation of plasma glucose above 126 mg/dl (7 mmol/L) in the fasting state and/or above 200 mg/dl (11.1 mmol/L) 120 min after a 75-g glucose load (8). Although defined on the basis of elevated plasma glucose levels, it is clear that diabetes is characterized by many associated abnormalities. More significantly, they predispose affected individuals to severe serious chronic problems. However, it is imperative for medical fraternity to identify *"subclinical"* diabetes to intercept the disease process. (9)

However, the fact that many newly diagnosed type 2 diabetic subjects already suffer from so called "late complications of diabetes" at the time of diagnosis (10) indicates that the diagnosis may have been delayed and, in addition, that the prediabetic condition is harmful to human health and requires increased awareness by physicians and the general public. Thus, type 2 diabetes mellitus represents only the *"tip of the iceberg"* of long existing metabolic disturbances with deleterious effects on the vascular system, tissues, and organs. Consequently, urgent efforts are required to avoid the growing number of patients with this form of a *"silently killing"* metabolic disease. (11)

Sixteen million individuals in the United States with type 2 diabetes mellitus and an additional 30–40 million with impaired glucose tolerance result in health care costs exceeding 100 billion dollars annually (12).Treatment is predominantly directed at microvascular and macrovascular complications (13). In type 1 diabetes mellitus the relationship between glycemic control and microvascular complications has been well established (14). The relationship between tight glycemic control and microvascular

disease in type 2 diabetes mellitus appears to be established in the recently completed United Kingdom Prospective Diabetes Study (15-16). India is a vast country with populations differing in racial admixtures as well as in social and cultural habits. It is likely that, the prevalence of non insulin dependent diabetes (type 2 diabetes) could also be different in various races due to genetic and environmental factors.

An estimated 1 million new cases are identified each year in people aged ≥20 years (17). Two modern-day epidemics, HIV–AIDS and type 2 diabetes mellitus, have inspired impassioned calls for more effective interventions. In the 1980s, the rapid spread of HIV, with its associated severe acute illness and high mortality, prompted activist groups and others to call for the accelerated approval of medications that showed promise of efficacy. (18) Similarly, diabetes is recognized as a global health problem nowadays, and it has been projected that the number of diabetic patients are rising and will rise from an estimated 135 million in 1995 to 300 million in 2025. (19)

Diabetes is more common in African Americans, Latinos, Native Americans, and Asian Americans/Pacific Islanders, as well as the aged population. This means they are also at increased risk for developing pre-diabetes. Prevalence of Type 2 diabetes is increasing globally as well as rapidly in India also. Asian Indians have an ethnic susceptibility to type II diabetes. (20) Type 2 diabetes constitutes about 85 to 95% of all diabetes in developed countries, and accounts for an even higher percentage in developing countries (21). There is substantial evidence that, it is epidemic in many developing and newly industrialized nations (22).

Urban India is rapidly getting industrialized and is now one of the most common non-communicable diseases globally. It has been demonstrated that, industrialization and modernization lead to sedentary lifestyle, obesity and higher risk of metabolic disorders (23-24).

Prevalence of Diabetes in the world was 5.1 % in 2003 and by 2025 is around 6.5%. IGT prevalence in the world was 8.2 % in 2003 and to be 9.0% in 2025. (age group 20-79 years).

Complications from diabetes, such as coronary artery and peripheral vascular disease, stroke, diabetic neuropathy, amputations, renal failure and blindness are resulting in increasing disability, reduced life expectancy and enormous health costs for virtually every society. Diabetes is certain to be one of the most challenging health problems in the 21stcentury (22).The country is already being dubbed as the **"Diabetes Capital of the World."**

Despite the morbidity and mortality associated with retinopathy, nephropathy, and neuropathy, cardiovascular disease remains the leading cause of death in type 2 diabetes mellitus (25-26) consequently; the treatment of confounding risk factors of obesity, hypertension, and hyperlipidemia assumes major importance and must be coordinated with good glycemic control for reduction in total mortality in type 2 diabetes mellitus (26-30).

Currently up to 11 % of India's urban population and 3 % of rural population above the age of 15 have diabetes. The WHO estimates the mortality from diabetes and heart disease cost India about 9450 billion rupees (210 billion $) and expected to increase to 15075 billion rupees (335 billion $) in next 10 years (31).

The economic growth of India is impressive with GDP clocking at higher rate during the last few years. This contributes further augmentation of purchasing power and consumer spending in the country. This economic growth has also seen the increasing westernisation of the Indian population. Dietary patterns are changing. Spending on food items are increasing and lifestyle becoming sedentary.Earlier, infectious and parasitic diseases were very common in India. However, significant medical advancement has helped to control the disease and this has started the new era of non communicable diseases like diabetes, obesity, hypertension, cancer and heart diseases (32).

It has been reported that by year 2030, 46% of Indian population will live in cities.

Impaired glucose tolerance and impaired fasting glucose form an intermediate stage in natural history of diabetes mellitus.

The rising Impaired Glucose Tolerance (IGT) /Impaired Fasting Glucose (IFG) reflect not only the epidemic in India, but it is closely links to strong family history, social class and auxology. In fact, positive family history, changing socio-economic class, increasing waist circumference (WC), waist-hip ratio (WHR), weight and BMI are the most crucial drivers to rapidly rising diabetes in India (9).

As discussed above, IGT and IFG are not equivalent metabolically, and it is therefore not surprising that there are differences in their prevalence and in the people categorized as having one or the other. A key issue is also the poor short-term repeatability of the OGTT. In most populations, IGT is more prevalent than IFG. Furthermore, there is some degree of overlap between the categories but the majority of people with IGT do not have IFG, and the majority with IFG does not have IGT. Thus, IFG and IGT identify substantially different segments of the population with impaired glucose regulation (33).

The DECODE(34-36)and DECODA(37) studies, which include data from 13 European and 11 Asian studies, respectively, reported that the mean two-hour plasma glucose concentration increased linearly with age, but a similar trend was not observed with FPG.

The large increase in two-hour plasma glucose in the elderly resulted in the increase in the prevalence of undiagnosed diabetes.
Individuals with IGT manifest abnormalities in insulin action and early insulin secretion similar to those seen in patients with type 2 diabetes. Furthermore progression of IGT to diabetes is characterised by dramatic decline in early insulin secretion. Acute Insulin secretary responses to intravenous glucose are lower in subject with IGT and they are high risk for developing diabetes. Prospectively low Acute Insulin Release (AIR) predicts development of diabetes in several populations. That's why early insulin secretion is important for the rapid and efficient suppression of endogenous glucose production after meal (38).

IFG was commoner in men than women in virtually all age groups, typically being 1.5-3 times higher, but up to seven or eight times higher in Europeans aged 50-70 years. But the prevalence of IGT was higher in women than men in all age groups except over the age of 60 in Asian populations and over the age of 80 in the European.

Finally, the prevalence of IGT tends to increase across all age groups, but that of IFG tends to plateau in middle age and in European men in particular falls in older age groups. Similar observations have been made in the USA, Denmark and Australia (39-42).

Evidence from many prospective studies has indicated that subjects with IGT have markedly higher risk for the development of type 2 diabetes and for all cause and cardiovascular mortality than subjects with normal glucose. Many newly diagnosed IFG patients progress to diabetes in less than 3 years. Original IFG subjects converted at a rate of 5.56% per year after an average of 29 months (43).

The WHO report had shown that diabetes was more common among middle-aged women in the developing countries, and this might be due to a higher proportion of women getting exposed to risk factors such as unhealthy diet, physical inactivity and central obesity (44).

The major risk factors associated with diabetes in India were age and positive family history of diabetes. With the rising trend in the prevalence among the population, especially in the younger subjects, there is likelihood for further increase in the familial predisposition to the disease. Increasing prevalence of diabetes in younger age would imply a future escalation in the prevalence of diabetic complications also (44).

It has been demonstrated that industrialization and modernization lead to sedentary lifestyle, obesity and higher risk of metabolic disorders. The risk variables associated with diabetes are almost similar in all nations, but its expressions and intensities vary widely between different races and countries (45).

The prevalence of diabetes mellitus differs in all the states of India across the rural and urban area. This is because of the different dietary pattern, physical activity and mental status.

In this study, we have analyzed the prevalence of prediabetes covering both Impaired Fasting Glucose (IFG) and Impaired Glucose Tolerance (IGT). We also tried to identify the major risk factors for the increase in this incidence.

3. OBJECTIVE:

India is going to face a big challenge posed by the rising prevalence of diabetes and its complications unless steps are taken to implement the primary and secondary prevention in the diabetes. For this purpose it is quite essential to identify the risk factors for diabetes and for the vascular complications. The objectives of this study are following;

- To determine the prevalence of Prediabetes (IFG and IGT) in the urban and rural population of Gujarat.

- To also assess the attributable risk factors for the same in Gujarati population.

4. REVIEW OF LITERATURE:

4.1 Overview of Diabetes:

Diabetes *(diabetes mellitus)* is a disorder of the human body, which is characterized by high blood sugar levels, a condition known as *hyperglycemia*. It is caused either by an inadequate secretion of the glucose-regulating hormone, *insulin*, or an inadequate response by the body's cells to insulin.

Progressive in nature, type 2 diabetes begins with peripheral insulin resistance and ends in complete loss of insulin secretion if the disease progresses through its entire natural history (46-48). Initially, pancreatic beta cells are able to compensate for insulin resistance by increasing basal and postprandial insulin secretion (49).

In time, however, compensation fails as beta cells exhaust their capacity to react appropriately to rising glucose concentrations. A deterioration of glucose homeostasis ensues, and glucose intolerance develops (50). Left untreated, intolerance worsens, and the diabetic state is manifested.

4.2 Types of Diabetes:

Diabetes is classified into two categories; these are type 1 and type 2.

Diabetes Mellitus Type 1 – Also called juvenile onset diabetes or Insulin-Dependent Diabetes Mellitus (IDDM), Type 1 diabetes is characterized by a decreased or outright absence of production of insulin. This is due to a disorder in the autoimmune response of the person, causing his own antibodies to attack the insulin producing cells in the pancreas.

Diabetes Mellitus Type 2 – Also known as maturity onset diabetes, obesity-related diabetes, or Non-Insulin Dependent Diabetes Mellitus (NIDDM), Type 2 diabetes results from the inability of the body's cells to respond to insulin. As the disease progresses, the production of insulin in the body decreases.

Gestational Diabetes – This is often called Type 3 diabetes although the designation is rarely used in medical practice. Gestation diabetes occurs among women during pregnancy and is similar to Type 2 diabetes in that it is a result of the cell's resistance

to insulin. The consequence is often abnormal increased fetal weight, increased surrounding amniotic fluid caused by increased fetal urination (called *polyhydramnios*), fetal jaundice and low blood sugars after delivery. On rare occasions, the condition has also been said to cause intra-uterine death.

Irrespective of its type, diabetes represents an anomalous rise of glucose in a person's blood. This anomaly is as a result of the inadequate level of insulin or perhaps a misuse of it. If the suitable treatment is not given, this disease can be the cause of many severe complications (cardiac disease, amputations, blindness, and impotence). Although it normally occurs among older, inactive individuals, children and adolescents are increasingly identified, contributing to the growing pool of patients who require lifelong care (51–53).

Initially, pancreatic beta cells are able to compensate for insulin resistance by increasing basal and postprandial insulin secretion (54).

At this point in the disease, patients require therapy for glycemic control and have already developed an atherosclerotic burden. And hence, the strict attention is required.

4.3 Signs of Diabetes:

The onset of diabetes is varied, depending on its particular type. Most Type 2 diabetes cases have a slow onset, taking years before the signs start to appear. However, in Type 1 cases, particularly in children, the symptoms may appear rapidly, taking only months or even weeks.

The most obvious signs of diabetes include the following:

- Frequent thirst *(polydipsia)*
- Constant urination *(polyuria)*
- Rapid loss of weight
- Unusual hunger
- Obvious weakness and fatigue

4.4 Diagnosis of Diabetes:

There are many methods by which diabetes is diagnosed, but doctors commonly use the following approaches:

- Health screening
- Detection of hyperglycemia
- New signs and symptoms attributable to diabetes

Diagnosis is often prompted with the onset of the symptoms. Patients often undergo a diabetes screening test, the particulars of which often vary according to circumstances and local policy. Some may be made to undergo random glucose testing, fasting glucose and insulin, or glucose two hours after ingestion of 75g of glucose. Sometimes, doctors diagnose the disease through a formal glucose tolerance test.

For adults aged 40-50, healthcare givers recognize standard screening tests for diabetes with earlier screening tests for those with potential risk factors, such as obesity, family history of diabetes, and high risk ethnicity (Hispanic, American Indian, African, American, Pacific Island, and South Asian).

4.5 Pathophysiology of Type 2 Diabetes Mellitus:

Type 2 diabetes mellitus is a heterogeneous disorder with varying prevalence among different ethnic groups. In the United States most affected populations are native Americans, particularly in the desert Southwest, Hispanic-Americans, and Asian-Americans (55).

Even in India, also the prevalence is highly variable in the several part of the country. Various studies have conducted to know the prevalence of the Diabetes in various part of the country. Recent population based studies showing the prevalence of type 2 diabetes in different part of India (56).

Fig 1: Prevalence of Diabetes in various parts of India.

(Adapted from Mohan V et al *Diabetologia*, *49:1175*-8; 2006)

The pathophysiology of type 2 diabetes mellitus is characterized by peripheral insulin resistance, impaired regulation of hepatic glucose production and declining β -cell function, eventually leading to β-cell failure.

The primary events are believed to be an initial deficit in insulin secretion and, in many patients, relative insulin deficiency in association with peripheral insulin resistance (64-65).

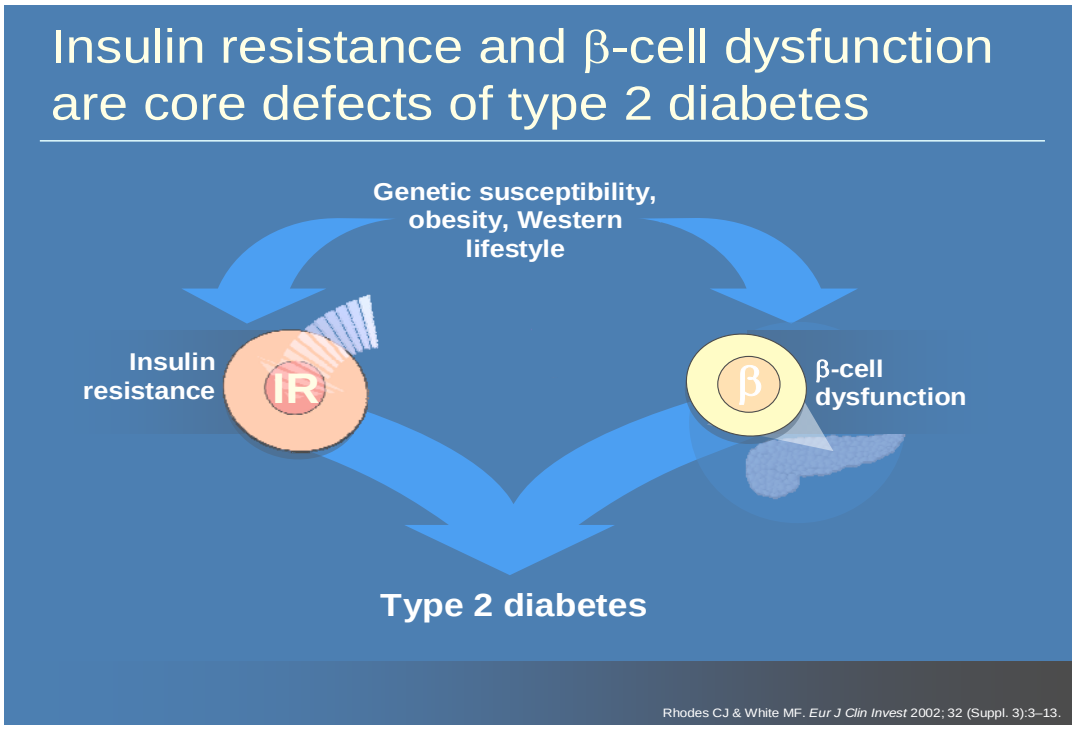


Fig 2: Insulin resistance and β cell dysfunction

4.5.1 The Role of β-cell in Insulin Resistance:

The β-cell is critical in the maintenance of glucose tolerance and a marked decrease in β -cell function is clearly associated with the development of hyperglycemia in type 2 diabetes. β -Cell dysfunction is initially characterized by impairment in the first phase of insulin secretion during glucose stimulation and may antedate the onset of glucose intolerance in type 2 diabetes (66).

Initiation of the insulin response depends upon the transmembranous transport of glucose and coupling of glucose to the glucose sensor. The glucose/glucose sensor complex then induces an increase in glucokinase by stabilizing the protein and impairing its degradation. The induction of glucokinase serves as the first step in linking intermediary metabolism with the insulin secretary apparatus. Glucose transport in β-cells of type 2 diabetes patients appears to be greatly reduced, thus shifting the control point for insulin secretion from glucokinase to the glucose transport system (67-68).

Later in the course of the disease, the second phase release of newly synthesized insulin is impaired, an effect that can be reversed, in part at least in some patients, by restoring strict control of glycemia. This secondary phenomenon, termed desensitization or β -cell glucotoxicity, is the result of a paradoxical inhibitory effect of glucose upon insulin release and may be attributable to the accumulation of glycogen within the β-cell as a result of sustained hyperglycemia (69).

Other defects in β-cell function in type 2 diabetes mellitus include defective glucose potentiation in response to nonglucose insulin secretagogues, asynchronous insulin release, and a decreased conversion of proinsulin to insulin (70-71).

Glucokinase is absent within the β-cell in some families with maturity onset diabetes of young (72). However, deficiencies of glucokinase have not been found in other forms of type 2 diabetes (73-74).

In some early onset patients with type 2 diabetes (perhaps as many as 20%) there may be a deficiency in insulin secretion that may or may not be due to autoimmune destruction of the β -cell and is not due to a deficiency in the glucokinase gene.

In the great majority of patients with type 2 diabetes (80%), the delay in immediate insulin response is accompanied by a secondary hypersecretory phase of insulin release as a result of either an inherited or acquired defect within the β-cell or a compensatory response to peripheral insulin resistance. Over a prolonged period of time, perhaps years, insulin secretion gradually declines, possibly as a result of intra islet accumulation of glucose intermediary metabolites (75).

In view of the decline in β-cell mass, sulfonylureas appear to serve a diminishing role in the long term management of type 2 diabetes (76).

4.5.2 Insulin Resistance:

Emanating from the prismatic demonstration by Yalow and Berson,the presence of hyperinsulinemia in type 2 diabetes, insulin resistance has been considered to play an integral role in the pathogenesis of the disease (77).

Recent clinical reviews, however have questioned the primacy, specificity, and contribution of insulin resistance to the disease state (78-79).

As chronic hyperinsulinemia inhibits both insulin secretion (80) and action (81), and hyperglycemia can impair both the insulin secretary response to glucose (82) as well as cellular insulin sensitivity (83-84), the precise relation between glucose and insulin level as a surrogate measure of insulin resistance has been questioned.

Lean type 2 diabetic patients over 65 year of age have been found to be as insulin sensitive as their age matched nondiabetic controls (85).

Moreover, in the majority of type 2 diabetic patients who are insulin resistant, obesity is almost invariably present (86-87).

Additionally, insulin resistance is found in hypertension, hyperlipidemia, and ischemic heart disease, entities commonly found in association with diabetes (88-90) again raising the question as to whether insulin resistance results from different pathogenetic disease processes or is unique to the presence of type 2 diabetes (88, 91-92).

Prospective studies have demonstrated the presence of either insulin deficiency or insulin resistance before the onset of type 2 diabetes. Two studies have reported the presence of insulin resistance in nondiabetic relatives of diabetic patients at a time, when their glucose tolerance was still normal (93-94).

Other studies in this high risk group have failed to demonstrate insulin resistance, and in the same group, impaired early phase insulin release and loss of normal oscillatory pattern of insulin release have been described (95-96).Based upon these divergent studies, it is still impossible to dissociate insulin resistance from insulin deficiency in

the pathogenesis of type 2 diabetes. However, both entities unequivocally contribute to the fully established disease.

4.5.3 Metabolic abnormalities associated with Insulin Resistance: (97)

Increase	Decrease
Small dense low density lipoprotein	Triglyceride elimination
Central body fat	High density lipoprotein
Free fatty acids	Vasodilatory response
Triglyceride synthesis	
Insulin	
Proinsulin	
Plasminogen Activator inhibitor-1	
Sympathomimetic activity	
Sodium retention	
Blood pressure	

Table: 1 Metabolic abnormalities associated with insulin resistance.

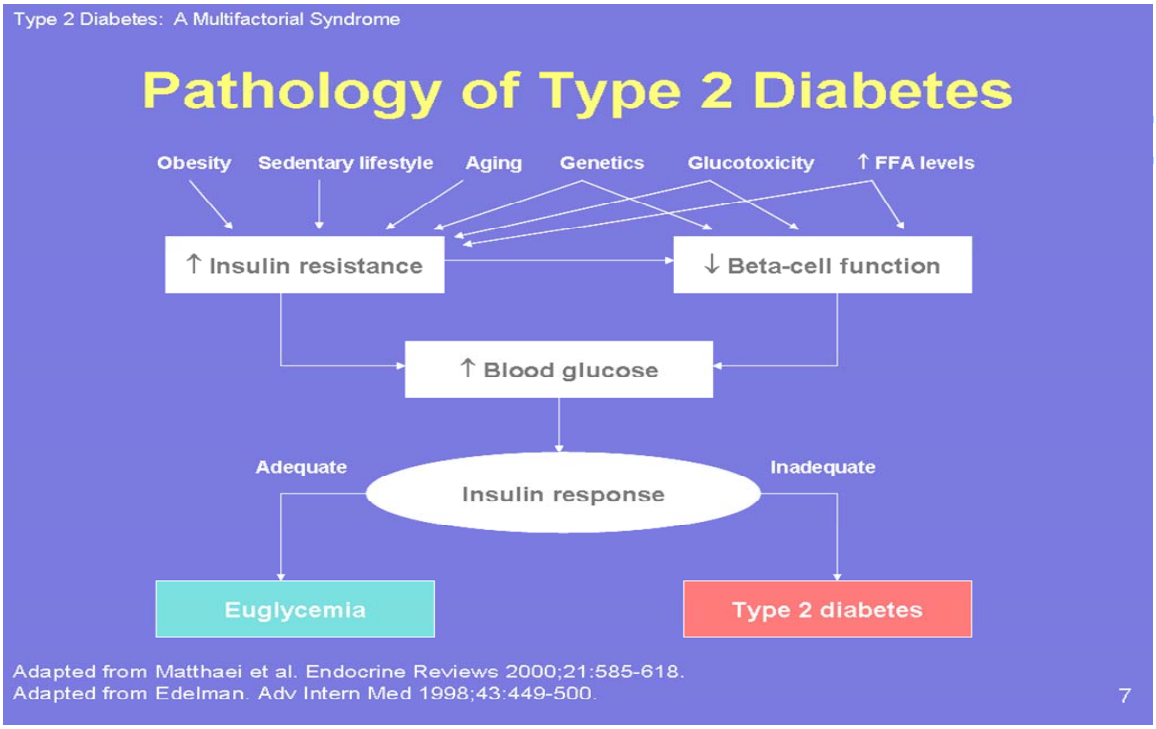


Fig 3: Pathology of diabetes

Understanding the importance of insulin sensitivity in modulating β-cell function has demonstrated that the β-cell is capable of dramatically increasing insulin release in response to increased secretary demand. In individuals, who are at increased risk of

developing type 2 diabetes, this adaptive response is inadequate and results in deteriorating glucose tolerance.

In fact, even within the normal range of fasting glucose, β-cell function declines as plasma glucose increases. There has been a resurgence of interest in changes in β-cell morphology in type 2 diabetes. It is clear that the numbers of β-cells are decreased in the disease, and this is in part due to the deposition of islet amyloid. In addition to reductions in β-cell mass, functional changes in secretary function also occur independently.

In humans acute elevations in free fatty acids are associated with decreased β-cell function. Thus, the reduced insulin release observed in type 2 diabetes and in high-risk states is clearly multifactorial, and is related to both genetics and environmental changes (98).

4.6 Risk factors of Diabetes:

Type 2 diabetes is known as a metabolic cum vascular disease. The American Heart Association has recently stated that "From the point of view of cardiovascular medicine, it may be appropriate to say diabetes is a cardiovascular disease". It is well known that diabetic vascular disease is not just limited to the macro vasculature, but also affect the microcirculation with devastating results, like Neuropathy, Nephropathy and Retinopathy.

Following are at higher risk factors for diabetes:

- Age greater than 45 years
- Diabetes during a previous pregnancy
- Excess body weight (especially around the waist)
- Family history of diabetes
- Given birth to a baby weighing more than 4.5 kg
- HDL cholesterol under 35 mg/dL
- High blood levels of triglycerides(250 mg/dL or more)
- High blood pressure (greater than or equal to 140/90 mmHg)

- Impaired glucose tolerance
- Low activity level (exercising less than 3 times a week)
- Metabolic syndrome
- Polycystic ovarian syndrome
- A condition called *acanthosis nigricans*, which causes dark, thickened skin around the neck or armpits

4.7 Management of Diabetes:

Because more than 75% of adult patients with type 2 diabetes die of macro vascular events such as myocardial infarction and stroke,(99) afflicted patients require comprehensive management to control their diabetes as well as any other metabolic disorders that contribute to cardiovascular risk and disease related morbidity and mortality.

4.7.1 Type 2 Diabetes Treatment Goals:

The goal of treatment in type 2 diabetes is to keep blood sugar levels at normal or near normal levels. Careful control of blood sugars can help to prevent the long-term effects of poorly controlled blood sugar (diabetic complications of the eye, kidney, and cardiovascular system).

In people with type 2 diabetes, home blood sugar testing might be recommended, especially in those who take oral diabetes medicines or insulin shots. Home blood sugar testing is not usually necessary for people who are diet controlled.

A normal fasting blood sugar is less than 100 mg/dL(5.6 mmol/L), although some people will have a different goal.

Blood sugar control can also be estimated with a blood test called A1C. The A1C blood test measures your average blood sugar level during the past two to three months. The goal A1C for most people with type 2 diabetes is 7 percent or less, which corresponds to an average blood sugar of 150 mg/dL (8.3 mmol/L).

Person's average blood sugar (150 mg/dl or 8.3 mmol/L) is higher than their fasting blood sugar goal (100 mg/dL or 5.6 mmol/L) for several reasons.

- Blood sugar goes up after eating
- How much and how fast your blood sugar goes up depends on the type and amount of food you eat at a particular meal
- The amount your blood sugar goes up also depends on what diabetes treatment(s) you use and your activity level

The most common long-term complication of type 2 diabetes is cardiovascular (heart) disease, which can lead to heart attack, chest pain, stroke, and even death. People with type 2 diabetes have twice the risk of heart disease as those without diabetes. However, you can substantially lower your risk of cardiovascular disease by:

- Quitting smoking
- Managing high blood pressure and high cholesterol with diet, exercise, and medicines
- Taking a low-dose aspirin every day, if indicated

4.7.2 Type 2 Diabetes Medicines:

A number of oral medicines are available for treatment of type 2 diabetes as given below:

Metformin - Most people who are newly diagnosed with type 2 diabetes will immediately put on a medicine called Metformin. Metformin improves how your body responds to insulin to reduce high blood sugar levels.

Common side effects of metformin include nausea, diarrhea and flatulence. These are usually not severe, especially if you take metformin along with food.

Patients with certain types of kidney, liver, and heart disease, and those who drink alcohol excessively should not take metformin.

If the blood sugar levels are still high after two to three months, but your A1C is close to the goal (between 7 and 8.5 percent), a second medicine might be added. The best

second medicine depends upon individual factors, including the person's weight, other medical problems, and preferences regarding use of injections.

The following are general recommendations:

- The most commonly recommended second medicine is a short acting sulfonylurea, such as glipizide.
- A thiazolidinedione, such as pioglitazone, is an alternative to sulfonylureas, but only for people who are not at increased risk of heart failure or bone fracture.
- A GLP-agonist, such as Exenatide, is an option for patients who are overweight and who want to avoid developing low blood sugar.
- A meglitinide, such as Repaglinide, is an option for people who cannot take a sulfonylurea or prefer to avoid injections.

Sulfonylureas — Sulfonylureas have been used to treat type 2 diabetes for many years. They work by increasing the amount of insulin what your body makes, and can lower blood sugar levels by approximately 20 percent. However, they stop working over time.

Sulfonylureas are generally used, if metformin does not adequately control blood sugar levels when taken alone. One should not take a sulfonylurea if he/she is allergic to sulfa drugs.

If you take a sulfonylurea, you can develop low blood sugar, known as hypoglycemia. Low blood sugar symptoms can include:

- Sweating
- Shaking
- Feeling hungry
- Feeling anxious

Low blood sugar must be treated quickly by eating 10 to 15 grams of fast-acting carbohydrate (eg, fruit juice, hard candy, glucose tablets).

Insulin - In the past, insulin treatment was reserved for patients with type 2 diabetes whose blood sugars were not controlled with oral medicines and lifestyle changes.

However, there is increasing evidence that using insulin at earlier stages may improve overall diabetes control and help to preserve the pancreas's ability to make insulin.

Insulin injections may be used as a first-line treatment in some people with type 2 diabetes, or it can be added to or substituted for oral medicines.

Thiazolidinediones - This class of medicines includes Pioglitazone, which work to lower blood sugar levels by increasing the body's sensitivity to insulin.

Common side effects of thiazolidinediones include:

- Weight gain and swelling of the feet and ankles.
- A small but serious increased risk of developing or worsening heart failure. People who take thiazolidinediones should monitor for swelling.
- A small increased risk of bone fractures

GLP-agonists - The GLP-agonists, Exenatide and Liraglutide, are injectable medicines. They are not a first line treatment, but might be considered for people whose blood sugar is not controlled on the highest dose of one or two oral medicines.They may be especially helpful for overweight patients, who are gaining weight on oral medicine.

GLP-agonists do not usually cause low blood sugar. They promote weight loss, but can also cause bothersome side effects, including nausea, vomiting, and diarrhea. Pancreatitis has been reported rarely in patients taking GLP-agonists, but it is not known if the drugs caused the pancreatitis. You should stop taking exenatide or liraglutide, if you develop severe abdominal pain. Exenatide should not be used in patients with abnormal kidney function.

DPP-IV Inhibitors - This class of medicines includes Sitagliptin, Saxagliptin, and Vildagliptin. They lower blood sugar levels by increasing insulin release from the pancreas in response to a meal. They are not a first-line treatment, but they can be given alone in patients who can’t tolerate the first-line medicines (metformin, sulfonylureas), or they can be given with other oral medicines if blood sugars are still higher than goal. These medicines do not cause hypoglycemia or changes in body weight. However, they may cause some nausea and diarrhea. There have been rare

reports of pancreatitis and skin reactions. DPP-IV inhibitors are expensive, and the long-term risks and benefits are unknown.

Meglitinides - Meglitinides include Repaglinide and Nateglinide. They work to lower blood sugar levels, similar to the sulfonylureas, and might be recommended in people who are allergic to sulfa-based drugs.

Alpha-glucosidase inhibitors-which include Acarbose and Miglitol, work by interfering with the absorption of carbohydrates in the intestines. This helps to lower blood sugar levels, but not as well as metformin or the sulfonylureas. They can be combined with other medicines if the first medicine does not lower blood sugar levels enough.

The main side effects of alpha-glycosidase inhibitors are gas (flatulence), diarrhea, and abdominal pain; starting with a low dose may minimize these side effects. The medicine is usually taken three times per day with the first bite of each meal (100).

5. COMMON MYTHS AND FACTS ABOUT DIABETES:

Myth 1: The greatest misconception regarding diabetes is that, it is a disease of affluence and rich.

Fact: This is totally untrue, as the disease affects rich as well as poor alike and seen in all strata of the population.

Myth 2: The belief is that it is contagious.

Fact: It is untrue as the disease is not transmitted by any germs but runs in families as it is transmitted to the offspring by genetic transmission.

Myth 3: It is believed that once diabetes is controlled one can take liberties regarding diet as medicines are controlling diabetes.

Fact: Diet and exercise is cornerstone in diabetes.

Myth 4: Once the Insulin is started in patients it becomes lifelong treatment.

Fact: This is especially not true with diabetes.

6. PREDIABETES:

Pre-diabetes (comprise of IFG and IGT) is the state that occurs when a person's blood glucose levels are higher than normal but not high enough for a diagnosis of diabetes.

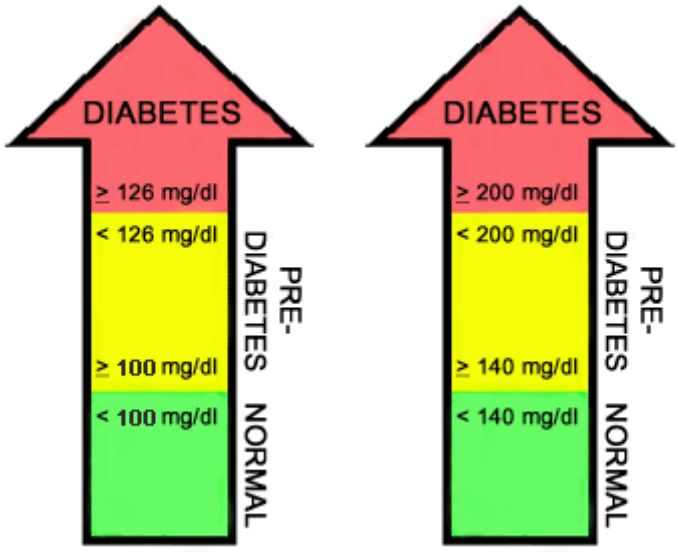
The terms IFG and IGT refer to an intermediate metabolic stage between normal glucose and diabetes. The concept of IFG was introduced by Charles et al. to refer at fasting plasma glucose levels ≥ 6.1 and < 7.8 mmol/L (101).

Impaired glucose tolerance is a more advanced stage of alteration in the glucose metabolism than impaired fasting glucose (102).

These fasting plasma glucose limits correspond to the level above which acute phase insulin secretion is lost in response to intravenous administration of glucose (103).

About 11 percent of people with pre-diabetes in the Diabetes Prevention Program standard or control group developed type 2 diabetes each year during the average three years of follow-up. Other studies show, that many people with pre-diabetes develop type 2 diabetes in 10 years.

Recently, the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus (1999) proposed a reduction in both upper and lower fasting glucose values for diagnosis of IFG to correspond at the new diagnosis criteria for diabetes (104-106).



FPG **OGTT**

Fig 4: Blood glucose ranges in IFG and IGT patients

However, recent reports have shown a lack of agreement between the 2-h post glucose category of IGT and the fasting glucose category of IFG among subjects of different ethnic populations.

Among US adults, 40-74 years of age, 15.6 percent (14.9 million) have IGT, and 9.7 percent (9.6 million) have IFG (107-109).

Indeed, an analysis on the 7-year follow-up Funagata Diabetes Study showed that IGT is a risk factor for cardiovascular disease, whereas IFG is not (110).

Moreover, longitudinal population-based studies have demonstrated a higher sensitivity of IGT over IFG for predicting the progression to type 2 diabetes (111).

People with pre-diabetes are at higher risk of cardiovascular diseases. People with pre-diabetes have a 1.5-fold risk of cardiovascular disease compared to people with normal blood glucose. People with diabetes have 2 to 4 fold increased risk of cardiovascular diseases.

People who are overweight and age 45 or older, they should be checked for pre-diabetes during their next routine medical visit. If their weight is normal and they are over age 45, they should ask their doctor during a routine office visit, if testing is appropriate.

For adults younger than 45 and overweight, your doctor may recommend testing if you have any other risk factors for diabetes or pre-diabetes. These include high blood pressure, low HDL cholesterol and high triglycerides, a family history of diabetes, a history of gestational diabetes or giving birth to a baby weighing more than 4 kg (~ 9 pounds) or belonging to an ethnic or minority group at high risk for diabetes.

To maintain serum glucose under 7.8 mmol/L, subjects with IFG show a significant hyperinsulinemia post glucose load, whereas the IGT subjects exhibit low insulin secretion unable to maintain the serum glucose within the normal referenced range values, findings that suggest different stages of impaired insulin secretion.

On the same way, our findings show that IGT is a more advanced stage of alteration in the glucose metabolism than the IFG. On the basis of this statement, the lack of concordance between IFG and IGT criteria that has been reported (112-116).

IFG and IGT are metabolically distinct disorders with limited overlap, among those who had IFG and/or IGT, 16 percent had both IFG and IGT, 23 percent had IFG alone and 60 percent had IGT alone (117).

IGT is a common condition that greatly increases risk for the subsequent development of type 2 diabetes. Individuals with IGT manifest abnormalities in both insulin action and early insulin secretion similar to those seen in patients with type 2 diabetes. These abnormalities not only precede diabetes, they predict it as well. Furthermore, the progression from IGT to diabetes is characterized by a dramatic decline in early insulin secretion. It is now evident that early insulin secretion plays an important role in the rapid and efficient suppression of endogenous glucose production following a meal. Loss of early insulin secretion initially leads to postprandial hyperglycemia which, as the disease progresses, worsens to clinical hyperglycemia.

Studies have shown that people with pre-diabetes can prevent or delay the development of type 2 diabetes by up to 58 percent through changes to their lifestyle that include modest weight loss and regular exercise. The expert panel recommends, that people with pre-diabetes reduce their weight by 5-10 percent and participate in some type of modest physical activity for 30 minutes daily. For some people with pre-diabetes, intervening early can actually turn back the clock and return elevated blood glucose levels to the normal range.

Symptoms of Insulin Resistance and Pre-diabetes:

Insulin resistance and pre-diabetes usually have no symptoms. If you have a severe form of insulin resistance, you may get dark patches of skin, usually on the back of your neck. Sometimes people get a dark ring around their neck. Other possible sites for these dark patches include elbows, knees, knuckles, and armpits. This condition is called acanthosis nigricans.

6.1 Pathogenesis of Prediabetes:

The progression from normal glucose tolerance to type 2 diabetes is characterized by dual defects that include insulin resistance and an insulin secretary defect caused by beta-cell dysfunction.

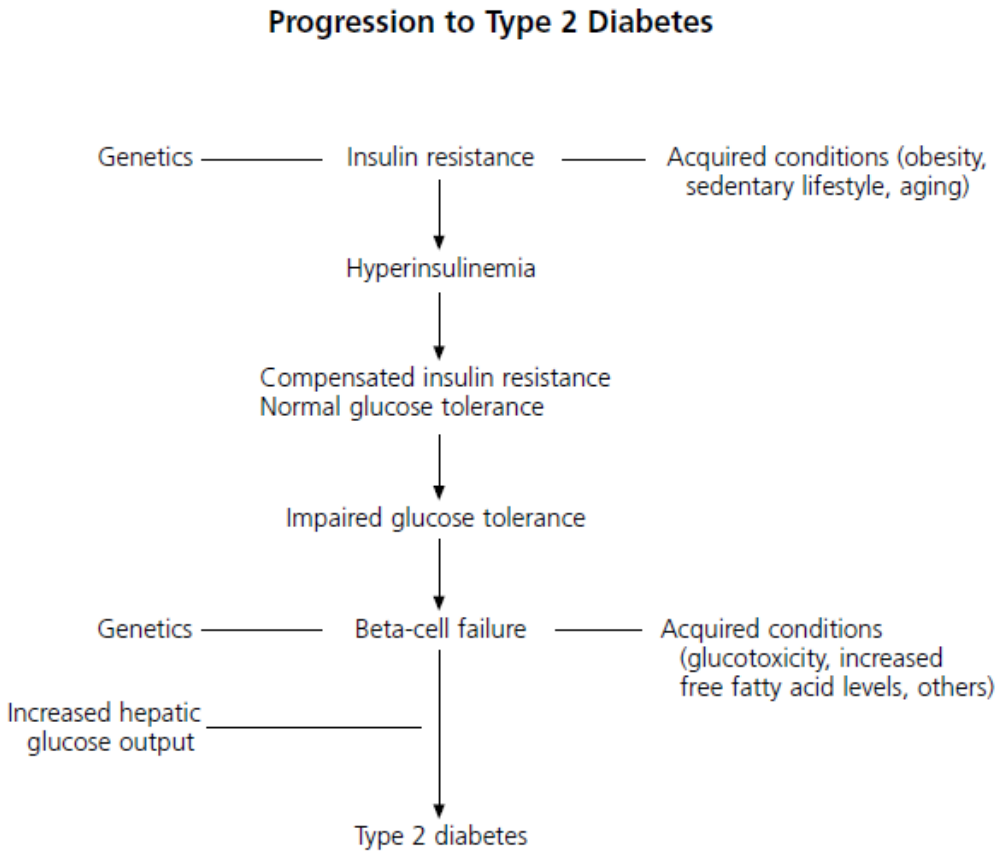


Fig 5: Etiologic sequence for the development of type 2 diabetes.

Insulin resistance is characterized by decreased tissue sensitivity to insulin and marked compensatory hyperinsulinemia. Initially, plasma glucose levels are maintained in the normal range. In patients, who will eventually develop diabetes, there is a decline in beta-cell secretary capacity.

Insulin resistance precedes the onset of type 2 diabetes mellitus by several years. During this time a prediabetic state of compensated hyperinsulinemia exists characterized by impaired glucose tolerance and associated with dyslipidemias and other metabolic abnormalities of the insulin resistance state.

The existence of long history of an atherogenic, prediabetic state emphasizes the importance of long term strategy for primary prevention of type 2 diabetes.

The first glucose abnormality that is detected is a rise in the postprandial glucose levels because of reduced first-phase insulin secretion. With time, further decline in beta cell function leads to elevation of the fasting glucose levels. Eventually, diabetes occurs, with more insulin secretary loss.

6.2 Focus on Prevention:

IGT is the first easily identifiable step in the pathophysiology of type 2 diabetes mellitus. It is associated with high risk for type 2 diabetes mellitus and subsequent vascular morbidity and mortality. It is currently unknown, whether treating IGT will reduce the incidence of macro vascular complications, as studies addressing this issue have yet to be conducted. Therefore, the main reason to identify and treat IGT is to prevent or delay the onset of type 2 diabetes mellitus.
Recent studies of patients with IGT have shown success for lifestyle interventions in delaying or preventing the development of diabetes (118-120).

There is strong evidence that a structured programme of diet and exercise can reduce the risk of progression to type 2 diabetes in patients with IGT. Patients with IFG and IGT should be advised on the benefits of modest weight loss, good dietary habits and regular physical activity.

IGT is characterized by an increase in postprandial glucose levels, which is considered the earliest metabolic abnormality in type 2 diabetes mellitus. It is one of a series of risk factors for CVD (hypertension, high triglyceride levels, low high-density lipoprotein-cholesterol and central obesity), known as the metabolic syndrome. The different factors making up this syndrome are intimately related. An impaired lipid profile can contribute to insulin resistance, as IGT may play a pathogenic role on other cardiovascular risk factors.

Macronutrients (MNT) is an integral component of diabetes prevention, management, and self-management education. In addition to its role in preventing and controlling

diabetes, ADA recognizes the importance of nutrition as an essential component of an overall healthy lifestyle.

Clinical trials/outcome studies of MNT have reported decreases in A1C at 3–6 months ranging from 0.25% to 2.9% with higher reductions seen in type 2 diabetes of shorter duration.

7. DIABETES SELF-MANAGEMENT EDUCATION (DSME):

People with diabetes should receive diabetes self-management education (DSME) according to national standards when their diabetes is diagnosed and as needed thereafter. Effective self-management and quality of life are the key outcomes of DSME and should be measured and monitored as part of care (121–126).
DSME is an essential element of diabetes care and national standards for DSME are based on evidence for its benefits.

Education helps people with diabetes initiate effective self-management and cope with diabetes when they are first diagnosed. Ongoing DSME and support also help people with diabetes maintain effective self-management throughout a lifetime of diabetes as they face new challenges and treatment advances become available.DSME helps patients optimize metabolic control, prevent and manage complications, and maximize quality of life in a cost-effective manner (127).

DSME is the ongoing process of facilitating the knowledge, skill, and ability necessary for diabetes self-care. This process incorporates the needs, goals, and life experiences of the person with diabetes. The overall objectives of DSME are to support informed decision-making, self-care behaviors, problem-solving, and active collaboration with the health care team to improve clinical outcomes, health status, and quality of life in a cost-effective manner (128).

Current best practice of DSME is a skills-based approach that focuses on helping those with diabetes to make informed self-management choices.

- Individuals who have prediabetes or diabetes should receive individualized medical nutrition therapy (MNT) as needed to achieve treatment goals.
- In overweight and obese insulin-resistant individuals, modest weight loss has been shown to reduce insulin resistance. Thus, weight loss is recommended for all overweight or obese individuals who have or are at risk for diabetes.
- For weight loss, either low-carbohydrate, low-fat calorie-restricted, or Mediterranean diets may be effective in the short-term (up to 2 years).

- For patients on low-carbohydrate diets, monitor lipid profiles, renal function, and protein intake (in those with nephropathy), and adjust hypoglycemic therapy as needed.
- Physical activity and behavior modification are important components of weight loss programs and are most helpful in maintenance of weight loss.
- Among individuals at high risk for developing type 2 diabetes, structured programs that emphasize lifestyle changes that include moderate weight loss (7% body weight) and regular physical activity (150 min/week), with dietary strategies including reduced calories and reduced intake of dietary fat, can reduce the risk for developing diabetes and are therefore recommended.
- Individuals at high risk for type 2 diabetes should be encouraged to achieve the U.S. Department of Agriculture (USDA) recommendation for dietary fiber (14 g fiber/1,000 kcal) and foods containing whole grains (one-half of grain intake) (121-126).

7.1 Macronutrients in Diabetes Management:

- The best mix of carbohydrate, protein, and fat may be adjusted to meet the metabolic goals and individual preferences of the person with diabetes.
- For individuals with diabetes, the use of the glycemic index and glycemic load may provide a modest additional benefit for glycemic control over that observed when total carbohydrate is considered alone.
- Saturated fat intake should be <7% of total calories.
- Reducing intake of *trans* fat lowers LDL cholesterol and increases HDL cholesterol.
- Routine supplementation with antioxidants, such as vitamins E and C and carotene, is not advised because of lack of evidence of efficacy and concern related to long-term safety.
- Individualized meal planning should include optimization of food choices to meet recommended dietary allowance (RDA)/dietary reference intake (DRI) for all micronutrients.

8. PHARMACOECONOMICS OF DIABETES:

The average cost of Diabetes management in India is around INR 20000/- per annum.In this, ambulatory care constitutes 65% of cost, while the hospitalization cost is 35%. Cost of medications is 31% of which specific diabetes drug costs are only 17%. Ambulatory care including monitoring and doctor visits constitute 34% of costs.

Description	Cost(INR)
Clinician Visit	853.00
Monitoring and Laboratory expenses	1609.00
Treatment cost	2262.00
Hospitalization	2434.00
Mean total direct cost	7158.00
Mean indirect cost	12756.00
Total estimated annual cost	**19914.00**

Table: 2 Cost of Diabetes management in India

8.1. Factors influencing Costs of Care:

8.1.1. Late vs Early Diagnosis:

Diabetes is often diagnosed late, perhaps too late. 50% of people with diabetes even in developed countries have complications at diagnosis. Untreated or improperly managed diabetes leads to serious and often life-threatening complications.

Complications requiring multiple therapies and prolonged hospitalization are responsible for most of the diabetes-related direct costs. Among people with diabetes who are hospitalized, the average annual direct costs are more than double those for people with diabetes who are not hospitalized. Complications are also responsible for indirect costs in terms of productivity loss and absenteeism.

8.1.2 Education:

Many socio-economic factors affect the time of diagnosis and thus the outcome of diabetes. Consequently they also affect the costs. The level of education appears to be important.

Diagnosis can be delayed by 3-7 years in the less-educated and uneducated sections of the population. The age of diagnosis was directly related to the level of education: college-educated people were on average diagnosed seven years before people with no literacy.

Despite a longer average duration of diabetes, those with a college education had a considerably lower rate of diabetes complications (45% complication-free) compared with people with low or no literacy (20% complication-free).

8.1.3. Unemployment:

Type 2 diabetes produces few symptoms and acutely is not life threatening. Often, weakness and tiredness are the only manifestations of the condition. While it is common for inactive unemployed people to ignore these symptoms (consciously or otherwise), those who are working are more likely to notice the signs as these influence the capacity to work. In the CODI study, compared to working people in urban areas, people in lower-income groups were diagnosed on average 4 years later as were people living in remote rural areas. People who are aware of diabetes before diagnosis or those with a family member with diabetes may be diagnosed earlier.

8.1.4 Complications:

The factors that influence delay in diagnosis also determine the rate of complications. Place of residence seems to play an indirect role: people with diabetes living in the semiurban or rural areas have higher rates of complications; despite less duration of diabetes; than those in urban settings. This would appear to reflect delayed diagnosis and the availability of less-than-optimum or indeed the total lack of care.

A similar trend is noted with regard to employment and socioeconomic status. Employed and working people with diabetes have fewer complications compared to those not working or those in rural areas engaged in agricultural labor. Among people with similar diabetes duration, larger proportions from the higher socio-economic strata are free of or have fewer diabetes complications (54% complication free; 8% with three complications), compared to the lower socioeconomic group (22% complication free; 26% with three complications).As might be expected, education appears to play a role in the development of diabetes complications.

For people with a similar duration of diabetes, 45% of those who finished higher education had no complications, compared to 20% for the no-literacy group. While awareness alone cannot overcome the socioeconomic barriers to health, within the same socio-economic groups, people who are aware of the problem suffer fewer complications than those who are not aware. Similar findings have been reported from the USA.

While the average annual direct cost for out-patient care for all people with diabetes was 4724 INR, the cost of care for those without complications was 18% lower, but 48% higher for those with three or more complications. As with ambulatory care, the cost of hospitalization increased with the number of complications.

8.2. Conclusions:

Uneducated, unemployed people with diabetes who cannot afford or do not have access to even minimum healthcare facilities especially those living in semi-urban or rural areas are likely to be diagnosed late. They are at increased risk of developing diabetes related complications due to delays in diagnosis and/or improper treatment.

Effective treatment of diabetes is not costly; however, in both human and economic terms, not treating the condition is extremely costly.

Also due to current rise in inflation and raising the cost of medications, it is anticipated that the cost of therapy will rise exponentially. The present cost of INR 20000/-(128-131) may be touch to INR 25,000 – INR 30,000.

9. MATERIALS AND METHODS:

The present cross functional study was carried out in various locations of Gujarat. We followed the American Diabetic Association (ADA) criteria for defining the IFG and IGT.

Impaired Fasting Glucose (IFG) was defined as a fasting plasma glucose value of100-125 mg/dl (5.6-6.9 mmol/L) in the absence of a previous diagnosis of diabetes (132).

Impaired Glucose Tolerance (IGT) was defined as a plasma glucose concentration of 140-200 mg/dl (7.8 to 11.0 mmol/L) two hours after oral administration 75 gm of glucose in subjects, whose plasma glucose concentration after overnight fasting was less than 140 mg/dl (132).

9.1 Study documents preparation:

Protocol, ICF and CRF were prepared in consultation with the experts, Dr.N.R.Sheth and Dr.Bhagirath Solanki. In the process Declaration of Helsinki principle was followed.After the preparation, quality check was performed. The required copies were prepared and submitted to the ethics committee of B.J.Medical College and Civil Hospital, Ahmedabad for its review.An Informed consent form was prepared as per the regulatory requirement.*(Appendix-I)*

9.2 Case Record Form:

A survey questionnaire was administered to all the participating subjects to collect the detailed information. *(Appendix-II)*

9.2.1 Demographic Details:

Following demographic details were collected from all the participating subjects.
a) Age b) Sex c) Height d) Weight e) BMI f) Waist to Hip ratio g) Systolic Blood pressure h) Diastolic Blood pressure

9.2.2 Inclusion & Exclusion Criteria:

Inclusion Criteria:

- Men and Women of age ≥ 20 years
- Having family history of Diabetes mellitus
- Subjects who are willing to participate in the study

Exclusion Criteria:

- Patients who are diabetics and pursuing diet, exercise and Oral hypoglycemic agents and Insulin also.

We have focused on the family members of diabetic patients because they are very much keen to undergo risk stratification and they comply with the camp activities.

To get around 1650 eligible subjects, approximately 1700 individuals (considering the drop out ratio of around 15%, for any reasons) more than ≥ 20 years of age were screened from the different areas of Gujarat. We also ensured that, we get the equal distribution from all the socio economic class, education class, field workers and office workers.

9.2.3 Laboratory Investigations:

Following laboratory investigations were performed for all the individuals at the central laboratory. A qualified and trained doctor/phlebotomist collected the blood samples.

a. Fasting blood glucose
b. 2 h Blood Glucose
c. Total cholesterol
d. Serum Triglycerides
e. Serum HDL cholesterol
f. Serum LDL cholesterol

9.3. Ethics Committee Approval: The study documents were approved by the ethics committee of B.J Medical College and Civil Hospital, Ahmedabad. *(Appendix-III)*

9.4 Methodology followed during the Screening:

9.4.1. Activities carried out before camps (2 weeks before):

- Meeting with Local Lions Club/Society/Community group/School management.
- Meeting with Local Family Physicians
- Registration of subjects

9.4.2. Activities carried out during Camps:

- Validate the registration of subjects
- Obtain the consent from subjects
- Collect the blood for investigations (Fasting stage)
- Administration of 75 gm Glucose dissolved in water
- Meet the physician (for physical examination)
- Collection of all demographic details in the CRF
- After 2 hours blood collection
- At the end, awareness was created in community about, How to Prevent Diabetes?

9.4.2.1 Anthropometric Measurement:

Body weight was measured (to the nearest 100 gm) with the subject standing still on weighing scale and weight equally distributed on each leg. Subjects were instructed to wear minimum outerwear and no footwear, while their weight was being measured.

Height was measured using a nonstretchable tape (to the nearest 0.1 cm) with the subject in an erect position against a vertical surface and the head positioned so that the top of the external auditory meatus was in the level with the inferior margin of the body orbit.

Body mass index was calculated by dividing the weight (in kilograms) with the square of height (in meters).

Waist circumference (to the nearest 0.1 cm) was measured using a tailor's tape at a point mid way between tip of iliac crest and last costal margin in the back and at umbilicus in the front.

After each subject had been seated for 5 min, blood pressure was measured twice to the nearest 2 mm Hg from the left arm of the participant using a standard sphygmomanometer. The average of the two measurements was used for all analyses. Diastolic blood pressure (DBP) was recorded at the fifth Korotkoff sound.

The physicians completed the interview questionnaire, which included questions about past medical history, family history of diabetes, history of medical treatment, smoking habits, occupation and education. Smoking status included current smoker and non-smoker.

9.4.2.2 Laboratory Analysis:

After 10–12 hr of an overnight fast, each subject voided, and then the fasting blood sample was collected. A 75 gm anhydrous glucose dissolved in 250 ml of water was given orally over the course of 5 min and a second blood sample was drawn exactly 2 h later for glucose estimation. Blood for glucose determination was collected into tubes containing fluoride and EDTA.Through out the study only one technician was allocated to avoid interpersonal error.

These blood samples were immediately centrifuged and processed further. Lipid profile samples were analyzed by Erba EM 360 (Germany).

9.4.3 Recruitment Status:

In this randomly selected study, we could manage to collect the data for 1652 subjects in the eleven camps. The details of conducted screening camps are as follows;

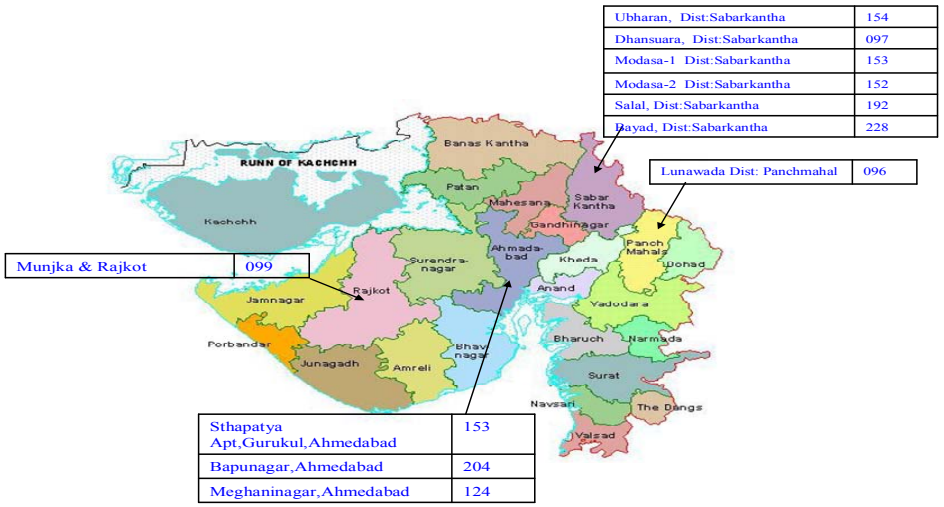


Fig 6: Location of the camps and recruitment status

Overall 12 screening camps were conducted at various Rural and Urban areas of Gujarat.

Sr.No	Place of Camp	Area	Date of Camp	No. of Subjects
1.	Lunawada, Dist: Panchmahal	Rural	17/05/2007	096
2.	Sthapatya Apt,Gurukul,Ahmedabad	Urban	20/05/2007	153
3.	Bayad,Dist:Sabarkantha	Rural	27/05/2007	228
4.	Ubharan, Dist:Sabarkantha	Rural	03/06/2007	154
5.	Dhansuara, Dist:Sabarkantha	Rural	10/06/2007	097
6.	Modasa-1, Dist:Sabarkantha	Urban	07/10/2007	153
7.	Modasa-2, Dist:Sabarkantha	Urban	14/10/2007	152
8.	Salal,Dist:Sabarkantha	Rural	17/11/2007	192
9.	Bapunagar,Ahmedabad	Urban	09/12/2007	204
10.	Meghaninagar,Ahmedabad	Urban	23/12/2007	124
11.	Munjka, Dist:Rajkot	Rural	19/03/2008	050
12.	Rajkot	Urban	19/03/2008	049
	Total			**1652**

Table: 3 Recruitment status during camps.

9.4.3.1 Schedule of Events:

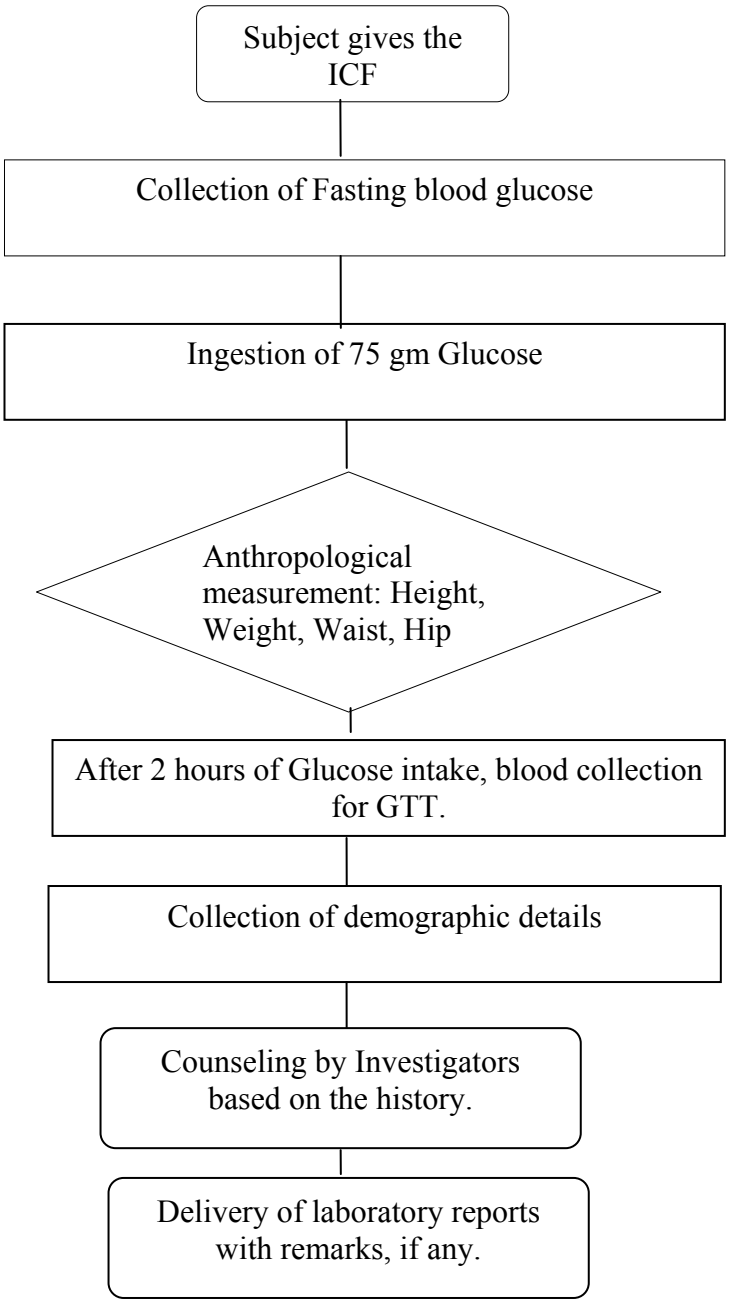


Fig 7: Schedule of events of the trial.

All the subjects were assessed for the Inclusion and Exclusion criteria as per the Protocol. No subjects were enrolled inadvertently and by practicising that highest quality standard was maintained.

9.4.4 Activities carried out after Camps:

- Discussion with Dr. N.R. Sheth
- Issue resolution and learning for the other camps
- Data entry
- Data validation and QC checks of the data
- Data cleaning & analysis

9.5 Methodology followed during Data entry:

During each camp, data in the CRFs were collected by trained team members. The time available between fasting glucose collection and 2 h post glucose collection was utilised to gather complete and appropriate demographic information. Each CRF was also checked for its completeness and correctness. Each team members was also provided detailed training to avoid missing out the vital and important information.

CRFs were collected and stored at secured and access controlled place. After that, all the CRFs were reviewed second time for any discrepancies. The once the check is over, the data entry was performed. Data entry was performed using Microsoft Excel 2003.

Manual review of CRFs were performed to identify the inconsistencies, which cannot be recognized by an electronic check.

9.6 Methodology followed during Data cleaning and Data analysis:

Quality review (QR) was performed to ensure data integrity and quality of the data deliverables. Initial QR was performed on received CRFs and lab reports. Wherever, we felt the data are doubtful or outliers, we omitted those data from analysis.

9.7 Statistical Analysis:

All statistical tests were considered significant at 5% level of significance. All data was analyzed by using Chi square test using SAS version 8.2.

Demographic, background and baseline data was presented descriptively. All continuous variables like age, height, weight and laboratory data was represented by mean ±SD (Standard Deviation). All the categorical variables were presented as counts and percentages.

Missing Data and Omitted Data: At various stages of the study, we did observe missing of the data. We also noticed that some data was not captured and CRFs were kept blank for those particular fields.

10. RESULTS:

We have collected data from 1652 subjects during the period of May 2007 to March 2008 from Urban and Rural area of Ahmedabad, Sabarkantha, Rajkot and Panchmahal districts of Gujarat in Western India.

10.1. Overall prevalence of IFG and IGT:

		IFG		IGT	
Gender	**n=**	**1454**	**100(%)**	**1453**	**100(%)**
	Male	30	2.06	45	3.16
	Female	10	0.69	44	2.95
Total Crude Prevalence		**40**	**2.75**	**89**	**6.11**
Age adjusted prevalence			2.72%		4.67%

Table 4: Gender wise distribution of IFG and IGT population.

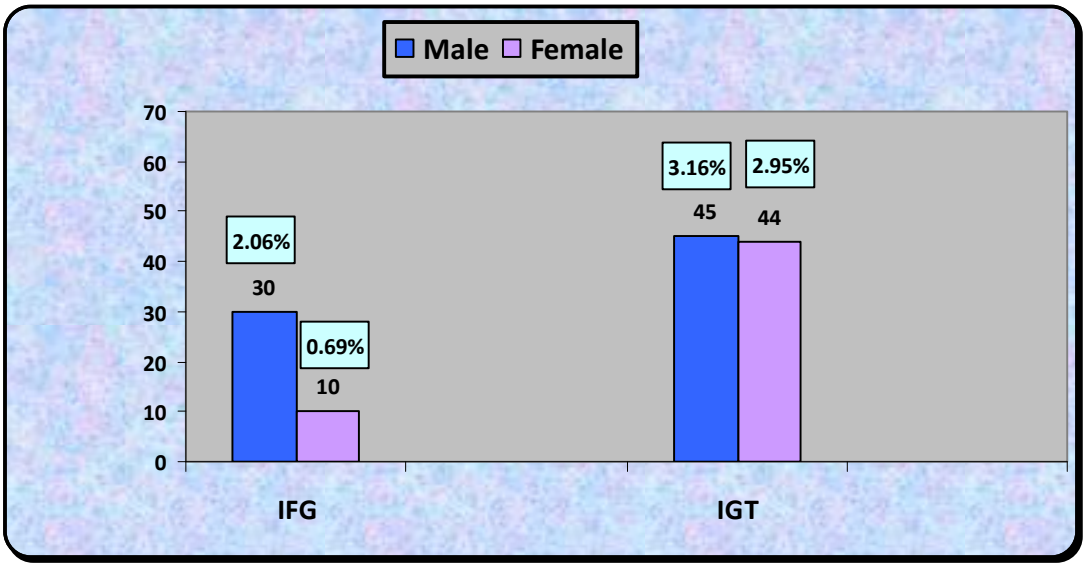


Fig: 8 Graphical presentation of IFG and IGT population

The collected data suggest that, overall prevalence of IFG in Gujarat is around 2.76% and IGT is around 6.12%.But when we applied age related prevalence (133), we found the prevalence of IFG is 2.72% and IGT is around 4.67%.

There are around 10 subjects, who were common in both IFG and IGT groups.

10.2 Gender wise presentation of IFG and IGT:

Upon further evaluation, we found that the prevalence of IFG (Impaired Fasting Glucose) in male is around 2.06 % and in female population is around 0.69%.

The prevalence of IGT (Impaired Glucose Tolerance) in male is around 3.09% and in female is around 2.94%.

10.3 Distribution of IFG and IGT population in Urban and Rural area:

Area	IFG	IGT
Rural (n= 822)	38 (4.62%)	34 (4.32%)
Urban (n= 855)	02 (0.23%)	50 (5.84%)
Total	**40**	**84**

Based on the above information, we can conclude that the prevalence of IFG and IGT is more in urban and rural area.

10.4 Age wise prevalence of IFG and IGT:

Category	Age Group (in years)	IGT (N=984)	IFG(N=985)
I	<= 30	1	2
II	31 – 40	3	2
III	41 – 50	34	9
IV	51 – 60	19	9
V	>= 60	10	9

Table 5: Age wise prevalence of IFG & IGT

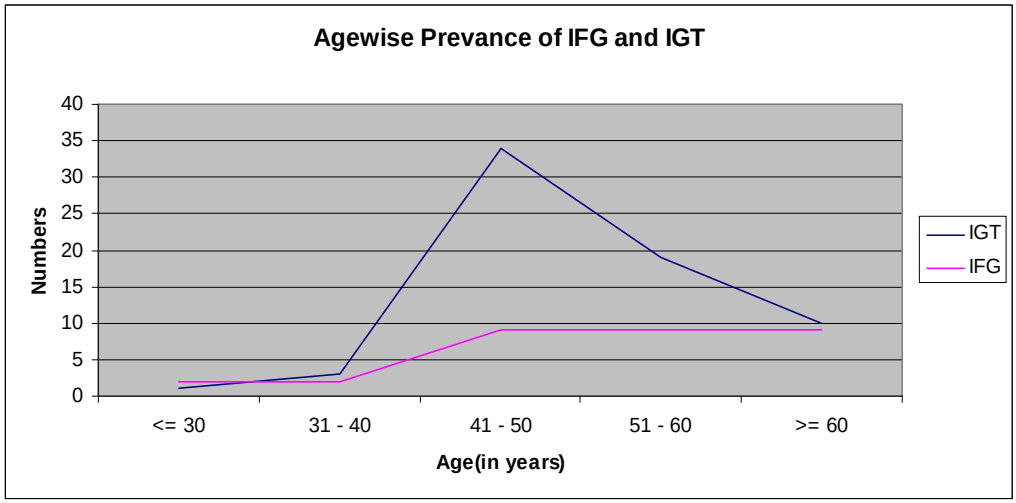


Fig 9: Agewise prevalence of IFG and IGT

From the above data, we can conclude that the prevalence of IFG and IGT increases, as the age progresses. From these data, it can be also proven that, prediabetes prevalence significantly start increasing after the age more than 41 years and after that it starts reducing. It means the and alertness for prevention of prediabetes is required age of 40 years to prevent prediabetes development and later on diabetes.

10.5 Characteristics of Population:

In this study, we characterised the population based on their BMI, WHR, Systolic and Diastolic blood pressure for both the sub groups (IFG and IGT)as given in below table.

Table 6: Characteristics of IFG Population:

Category	Statistical tool	BMI (kg/m^2)	Waist/Hip Ratio	Systolic Blood Pressure (mm Hg)	Diastolic Blood Pressure (mm Hg)
Normoglycemic (< 110 mg/dl)	N	1246	1208	1240	1239
	MIN-MAX	13.76-57.67	0.54-1.62	70.00-220.00	48.00-170.00
	MEAN±SD	**25.66±5.22**	**0.91± 0.09**	**123.27 ± 15.03**	**81.44 ± 9.61**
IFG(>110 - <126 mg/dl)	N	38	35	39	39
	MIN-MAX	19.95-35.44	0.80-1.13	100.00-196.00	60.00-100.00
	MEAN±SD	**26.81± 4.21**	**0.96 ± 0.07**	**135.54 ± 21.45**	**82.92 ± 9.97**
Newly Diagnosed Diabetes(>126 mg/dl)	N	79	68	77	77
	MIN-MAX	17.15-36.00	0.82-1.60	96.00-200.00	58.00-110.00
	MEAN±SD	**26.62 ± 3.61**	**0.97± 0.11**	**132.39 ± 19.69**	**83.94 ± 9.60**

In IFG population, the mean value of BMI is 26.81 $\pm$ 4.21 kg/m^2, WHR is 0.96 $\pm$ 0.07, Systolic blood pressure is 135.54 $\pm$ 21.45 mm Hg and Diastolic blood pressure 82.92 $\pm$ 9.97 mm Hg.

This proves that BMI and WHR is highly significant statistically in IFG population. The result proves that IFG population is more prone to develop central obesity.

Similarly, mean systolic and diastolic blood pressure is more in IFG population as compare to normoglycemic population. This proves that blood pressure is also risk factor for development of prediabetes.

The observation in IFG population was correlated with newly diagnosed subjects. It means the probability of risk for prediabetes is as high as diabetes population.

Table 7: Summary of normoglycemic, IFG and newly diagnosed diabetes subjects by BMI, WHR, Systolic and Diastolic blood pressure:

Category	BMI (kg/m2)			Waist to Hip Ratio						Systolic Blood Pressure (mm Hg)			Diastolic blood Pressure (mm Hg)		
				Male			Female								
	Total	**< 25**	**≥ 25**	Total	**< 0.95**	**≥ 0.95**	**Total**	**< 0.85**	**≥ 0.85**	**Total**	**<120**	**≥120**	**Total**	**<80**	**≥80**
Fasting Blood Glucose	1363 (100)	652 (47.84)	711 (52.16)	795 (100)	501 (63.02)	294 (36.98)	516 (100)	232 (44.96)	284 (55.04)	1356 (100)	708 (52.21)	648 (47.79)	1355 (100)	801 (59.11)	554 (40.89)
Normoglycemic (<110 mg/dl)	1246 (91.42)	614 (45.05)	632 (46.37)	730 (91.82)	477 (60.00)	253 (31.82)	478 (92.64)	223 (43.22)	255 (49.42)	1240 (91.45)	670 (49.51)	570 (42.04)	1239 (91.44)	742 (54.76)	497 (36.68)
IFG >110 <126 mg/dl	38 (2.79)	13 (0.95)	25 (1.83)	26 (3.27)	10 (1.26)	16 (2.01)	9 (1.74)	2 (0.39)	7 (1.36)	39 (2.88)	14 (1.03)	25 (1.84)	39 (2.88)	21 (1.55)	18 (1.33)
Newly Diagnosed Diabetes (>126 mg/dl)	79 (5.80)	25 (1.83)	54 (3.96)	39 (4.91)	14 (1.76)	25 (3.14)	29 (5.62)	7 (1.36)	22 (4.26)	77 (5.68)	24 (1.77)	53 (3.91)	77 (5.68)	38 (2.80)	39 (2.88)
P-value	0.0023			<0.0001			0.0233			<.0001			0.1502		

* 42 subjects data were missing and hence not considered for analysis.

Note: P-value obtained using chi-square test.

Above table shows that;

BMI: subjects with BMI greater than equal to 25 have more chances of becoming diabetic, 25 (1.83%) versus 13 (0.95%). Also in this category, more no. of newly diagnosed diabetes has been identified 54 (3.96%) versus 25 (1.83%).

A p-value of 0.0023, obtained using chi-square test, indicates this association is statistically significant.

Waist to hip ratio: Male subjects with waist to hip ratio $\geq$ 0.95 have more chances of becoming diabetic, 16 (2.01%) versus 10 (1.26%). A p-value of <0.0001, obtained using chi-square test, indicates this association is statistically significant.

For female subjects with waist to hip ratio $\geq$ 0.85 have more chances of becoming diabetic, 7 (1.36%) versus 2 (0.39%). A p-value of 0.0233, obtained using chi-square test, indicates this association is statistically significant.

Blood Pressure: Subjects with systolic blood pressure greater than 120 mm Hg have more chances of becoming diabetic, 25 (1.84%) versus 14 (1.03%). A p-value of <0.0001, obtained using chi-square test, indicates this association to be statistically significant.

While for diastolic blood pressure, there is inverse relationship noted.

Table 8: Characteristics of IGT Population:

Category	Statistical tool	BMI (kg/m^2)	Waist/Hip Ratio	Systolic Blood Pressure (mm Hg)	Diastolic Blood Pressure (mm Hg)
I:2h Post Glucose <140 mg/dl *	N	1229	1189	1220	1219
	MIN-MAX	13.76-85.22	0.54-1.60	44.00-368.00	10.00-170.00
	MEAN±SD	**25.57± 5.21**	**0.91±0.09**	**123.94 ± 17.18**	**81.43 ± 9.96**
II: Impaired Glucose Tolerance(IGT) (2 h post glucose 140-200 mg/dl)	N	84	74	83	83
	MIN-MAX	16.23-57.67	0.55-1.62	86.00-196.00	60.00-100.00
	MEAN±SD	**29.36± 6.49**	**0.94±0.12**	**130.19± 17.66**	**83.64± 9.43**

Category I: Subjects, 2 hr post glucose value < 140 mg/dl

Category II: Subjects with 2 hr post glucose value 140-200 mg/dl (Impaired glucose Tolerance)

* 78 subjects with blood glucose value more than 200 mg/dl were excluded from the analysis.

In IGT population, the mean value of BMI is 29.36 $\pm$ 6.49 kg/m^2, WHR is 0.94 $\pm$ 0.12, Systolic blood pressure is 130.19 $\pm$ 17.66 mm Hg and Diastolic blood pressure 83.64 $\pm$ 9.43 mm Hg.

This proves that BMI and WHR is highly significant statistically in IGT population. This is suggestive that, IGT population is more prone to develop obesity.

Similarly, mean systolic and diastolic blood pressure is more in IGT population as compare to category I population. This proves that blood pressure is also risk factor for development of prediabetes.

And hence, the above findings strongly propose that the BMI, WHR, systolic blood pressure and diastolic blood pressure are risk factors in IFG and IGT population.

Table 9: Summary of IGT subjects by BMI, WHR, Systolic and Diastolic blood pressure:

Category	BMI(kg/m2)			Waist to Hip Ratio						Systolic Blood Pressure (mm of Hg)			Diastolic blood Pressure (mm of Hg)		
				Male			Female								
	Total	**< 25**	**≥ 25**	Total	**< 0.95**	**≥ 0.95**	**Total**	**< 0.85**	**≥ 0.85**	**Total**	**<120**	**≥120**	**Total**	**<80**	**≥80**
2 h Post Glucose	1313 (100)	624 (47.52)	689 (52.48)	757 (100)	479 (63.28)	278 (36.72)	506 (100)	226 (44.66)	280 (55.34)	1303 (100)	681 (52.26)	622 (47.74)	1302 (100)	765 (58.86)	537 (41.24)
2h Post Glucose (<140 mg/dl)	1229 (93.60)	605 (46.08)	624 (47.52)	717 (94.72)	467 (61.69)	250 (33.03)	472 (93.28)	213 (42.09)	259 (51.19)	1220 (93.63)	649 (49.81)	571 (43.82)	1219 (93.63)	728 (55.91)	491 (37.71)
Impaired Glucose Tolerance(IGT) (2 h post glucose 140-200 mg/dl)	84 (6.40)	19 (1.45)	65 (4.95)	40 (5.28)	12 (1.59)	28 (3.70)	34 (6.72)	13 (2.57)	21 (4.15)	83 (6.37)	32 (2.46)	51 (3.91)	83 (6.37)	37 (2.84)	46 (3.53)
P-value	< 0.0001			< 0.0001			0.2003			0.0098			0.0067		

Above table shows that;

BMI: Subjects with BMI $\geq$ to 25 have more chances for development of Impaired Glucose Tolerance 65 (4.95%) versus 19 (1.45%).

A p-value of <0.0001, obtained using chi-square test, indicates this association to be statistically significant.

Waist to hip ratio: Male subjects with waist to hip ratio greater than equal 0.95 have more chances of development of Impaired Glucose Tolerance 28 (3.70%) versus 12 (1.59%). A p-value of <0.0001, obtained using chi-square test, indicates this association is statistically significant.

For female subjects with waist to hip ratio greater than equal 0.85 have more chances of development of Impaired Glucose Tolerance 21 (4.15%) versus 13 (2.57%). A p-value of 0.2003, obtained using chi-square test, indicates this association is not statistically significant.

Blood Pressure: Subjects with systolic blood pressure greater than 120 have more chances of development of Impaired Glucose Tolerance , 51 (3.91%) versus 32 (2.46%).

A p-value of 0.0098 obtained using chi-square test, indicates this association to be statistically significant.

Subjects with diastolic blood pressure greater than 80 have more chances of development of Impaired Glucose Tolerance , 46 (3.53%) versus 37 (2.84%).

A p-value of 0.0067 obtained using chi-square test, indicates this association to be statistically significant.

10.6 Lipid Analysis in Prediabetes Population:

Table 10: Summary of Lipid Analysis for Normoglycemic, IFG and newly diagnosed diabetes population:

Category	Statistical tool	Serum Cholesterol (mg/dl)	HDL Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)	Serum Triglycerides (mg/dl)
Normoglycemic (< 110 mg/dl)	N	1330	1329	1329	1330
	MIN- MAX	31.40-360.50	14.00-57.20	66.90-225.30	59.30-870.10
	MEAN±SD	**203.02±38.24**	**44.55±4.68**	**131.55±25.56**	**141.66±70.83**
IFG(>110 <126 mg/dl)	N	40	40	40	40
	MIN- MAX	129.90-293.70	38.10-56.90	67.60-193.90	70.90-489.70
	MEAN±SD	**206.83±51.28**	**45.77±5.95**	**130.47±35.61**	**178.44±94.18**
Newly Diagnosed Diabetes(>126 mg/dl)	N	89	89	89	89
	MIN- MAX	104.10-310.40	37.00-57.10	65.80-192.70	73.70-612.90
	MEAN±SD	**206.23±45.49**	**45.05±5.21**	**126.35±32.23**	**193.21±96.52**

The above mentioned data suggest that, Sr.tryglycerides is highly significant statistically in IFG population as compare to normoglycemic.It also matches with the findings from various studies in the Indian population.

IFG population has more Sr.tryglycerides, as compare to normoglycemic population. The result from the data proves our assumptions that, IFG population is more prone to develop dyslipidemia.

Similarly for other lipid parameters such as Sr.Cholesterol,HDL-C and LDL-C is also elevated in IFG population but not highly significant statistically.

Table 11: Profile of normoglycemic, IFG and newly diagnosed diabetes population by their lipids:

Category	Serum Cholesterol(mg/dl)			HDL Cholesterol (mg/dl)						LDL Cholesterol(mg/dl)			Serum Triglycerides (mg/dl)		
				Male			Female								
	Total	**< 200**	**≥ 200**	Total	**< 40**	**≥ 40**	**Total**	**< 50**	**≥ 50**	**Total**	**<130**	**≥130**	**Total**	**<150**	**≥150**
Fasting Blood Glucose	1442 (100)	713 (49.85)	729 (50.55)	855 (100)	718 (83.98)	137 (16.02)	586 (100)	65 (11.09)	521 (88.91)	1441 (100)	724 (50.24)	717 (49.76)	1442 (100)	921 (63.87)	521 (36.13)
Normoglycemic (<110 mg/dl)	1316 (91.26)	650 (45.08)	666 (46.19)	781 (91.35)	655 (76.37)	128 (14.97)	534 (91.13)	55 (9.39)	479 (81.74)	1315 (91.26)	652 (45.25)	663 (46.01)	1316 (91.26)	866 (60.06)	450 (31.21)
IFG >110 <126 mg/dl	40 (2.77)	20 (1.39)	20 (1.39)	30 (3.51)	25 (2.92)	5 (0.58)	10 (1.71)	4 (0.68)	6 (1.02)	40 (2.78)	22 (1.53)	18 (1.25)	40 (2.77)	20 (1.39)	20 (1.39)
Newly Diagnosed Diabetes (>126 mg/dl)	86 (5.96)	43 (2.98)	43 (2.98)	44 (5.15)	40 (4.68)	4 (0.47)	42 (7.17)	6 (1.02)	36 (6.14)	86 (5.97)	50 (3.47)	36 (2.50)	86 (5.96)	35 (2.43)	51 (3.54)
P-value	0.8950			0.4364			0.0098			0.02545			<0.001		

Above table shows that, subjects with Sr.tryglycerides greater than equal to 150 have more chances for development of Impaired Fasting Glucose.

A p-value of <0.0001, obtained using chi-square test, indicates this association to be statistically significant.

Table 12: Summary of Lipid Analysis for IGT population:

Category	Statistics	Serum Cholesterol (mg/dl)	Serum HDL Cholesterol (mg/dl)	LDL Cholesterol (mg/dl)	Serum triglycerides (mg/dl)
I :2h Post Glucose (<140 mg/dl)*	N	1369	1368	1368	1369
	MIN- MAX	31.40-360.50	14.00-57.20	65.80-225.30	59.30-870.10
	MEAN±SD	**203.59±39.10**	**44.64±4.78**	**131.70±26.16**	**143.91±74.01**
II:Impaired Glucose Tolerance(IGT) (2 h post glucose >140-200 mg/dl)	N	89	89	89	89
	MIN- MAX	129.90-87.40	37.00-56.10	67.60-188.40	72.10-489.70
	MEAN±SD	**199.68±39.07**	**44.34±4.36**	**124.02±28.07**	**175.23±77.12**

Category I: Subjects, 2 hr post glucose value < 140 mg/dl.

Category II: Subjects with 2 hr post glucose value 140-200 mg/dl (Impaired glucose Tolerance)

* 59 subjects with 2 hr blood glucose value more than 200 mg/dl were excluded from the analysis.

The above mentioned data suggest that, there is significant difference in Sr.triglycerides value in Category I compared to IGT population.

IGT population also has more Sr.triglycerides, as compare to category I. The above result proves our assumption that IGT population is more prone to develop diabetic dyslipidemia. This finding could be unique for Gujarati population that, they have higher triglycerides. Other laboratory parameters may be found abnormal but our study has

highlighted significantly about triglycerides and genetically match with Indian population, where they have been labelled with hypertriglyceridemia.We can also hypothesize that, other laboratory parameters may be found abnormal after the age of 60 years.

Table 13: Profile of IGT population by their lipids:

Category	Serum Cholesterol(mg/dl)			HDL Cholesterol (mg/dl)						LDL Cholesterol(mg/dl)			Serum Triglycerides(mg/dl)		
				Male			Female								
	Total	< 200	≥ 200	Total	< 40	≥ 40	Total	< 50	≥ 50	Total	<130	≥130	Total	<150	≥150
2 h Post Glucose	1377 (100)	680 (49.38)	697 (50.62)	808 (100)	681 (84.28)	127 (15.72)	568 (100)	63 (11.09)	505 (88.91)	1376 (100)	689 (50.07)	687 (49.93)	1377 (100)	886 (64.34)	491 (35.66)
2h Post Glucose (<140 mg/dl)	1288 (93.54)	634 (46.04)	654 (47.49)	762 (94.31)	642 (79.46)	120 (14.85)	525 (92.43)	55 (9.68)	470 (82.75)	1287 (91.26)	636 (46.22)	651 (47.31)	1288 (93.54)	850 (61.73)	438 (31.81)
Impaired Glucose Tolerance(IGT) (2 h post glucose 140-200 mg/dl)	89 (6.46)	46 (3.34)	43 (3.12)	46 (5.69)	39 (4.83)	7 (0.87)	43 (7.57)	8 (1.41)	35 (6.16)	89 (6.47)	53 (3.85)	36 (2.62)	89 (6.46)	36 (2.61)	53 (3.85)
P-value	0.8905			0.9235			0.1027			0.2713			<0.001		

Above table shows that, subjects with triglycerides greater than 150 have more chances for development of Impaired Glucose Tolerance 53 (3.85%) versus 36 (2.61%).

A p-value of <0.0001, obtained using chi-square test, indicates this association is statistically significant.

For HDL-C, in the group of male subjects with HDL-C less than 40 have more chances of development of Impaired Glucose Tolerance 39 (4.83%) versus 7 (0.87%). A p-value of 0.9235, obtained using chi-square test, indicates this association is statistically significant.

10.7 Family History and Prediabetes:

In this study, we also tried to correlate the prevalence of IFG and IGT in the population with the family history of diabetes. The below mentioned table provides the details on the same.

Table 14: Correlation of Family History of Diabetes with IFG.

Category	**Family history of Diabetes**		
IFG	**Positive**	**Negative**	**Total**
Yes	14(3.47%)	389	403(100%)
No	24	986	960
Total	**38**	**1325**	**1363**
Odds ratio	1.5279	p value	0.2962

From the above analysis, we can conclude that, in our study, out of 403(100%) subjects of IFG, 14(3.47%) subjects have the family history of diabetes.

Table 15: Correlation of Family History of Diabetes with IGT.

Category	**Family history of Diabetes**		
IGT	**Positive**	**Negative**	**Total**
Yes	31(8.13%)	350	381(100%)
No	53	877	930
Total	**84**	**1227**	**1311**
Odds ratio	1.5852	p value	0.0683

Similarly, out of 381(100%) subjects with IGT, 31(8.13%) has the family history of diabetes. Which means the family history plays very important role in the development of prediabetes especially for IGT.

Interpretation of Odds Ratio:

In IFG population, odds ratio of 1.5279 suggest that the persons, who have family history of diabetes are having 1.5 times more chance to develop IFG compare to the persons, who do not have family history of Diabetes.

In IGT population, odds ratio of 1.5852 suggest that the persons who have family history of diabetes are having 1.6 times more chance to develop IGT compare to the persons, who do not have family history of Diabetes.

11. ETHICAL REQUIREMENT:

Prior to initiation of the study, protocol, informed consent form, case record form and other relevant study documents were submitted to the Institutional ethics committee of B.J.Medical College and Civil Hospital, Ahmedabad. And after receiving the approval, study was initiated. The study was conducted as per Schedule Y and ICH GCP guidelines. We have also followed the Declaration of Helsinki guidelines.

11.1 Patient Information and Consent:

No patient is subjected to any study-related examination or activity before that patient and/or legal guardian has given informed consent. Written informed consent was obtained from all the subjects.

12. DISCUSSION:

Diabetes is the main cause of kidney failure, limb amputations and new onset blindness in adults and is a major cause of heart disease and stroke. Many clinical trials have proven that, these complications can be dramatically reduced with tight control of blood glucose, blood pressure and cholesterol. National campaigns such as the National Diabetes Education Program's "Be Smart about your Heart. Control the **ABC**s of Diabetes" (Hb**A**1c, **B**lood Pressure, and **C**holesterol) have led to a wider awareness of the need to control the risk factors for diabetes complications (134).

Only less than 12 percent of people diagnosed with diabetes meet the recommended goals for blood glucose, blood pressure, and cholesterol despite a great deal of research showing that controlling these conditions dramatically delays or prevents diabetes complications. Moreover, the percentage of people who achieve these targets has changed little in the last decade (134).

Epidemiological analyses of the Diabetes Control and Complications Trial Research Group (DCCT) and UKPDS demonstrate a curvilinear relationship between A1C and microvascular complications (136-137).

Such analysis suggest that, on a population level, the greatest number of complications will be prevented by transferring patients from very poor control to fair or good control. These analyses also suggest that further lowering of A1C from 7 to 6% is associated with further reduction in the risk of microvascular complications, though the absolute risk reductions become much smaller.

Many epidemiologic studies and meta-analyses have clearly shown a direct relationship between A1C and CVD, the potential of intensive glycemic control to reduce CVD has been less clearly defined.

In the DCCT, there was a trend toward lower risk of CVD events with intensive control.

However, 9-year post-DCCT follow-up of the cohort has shown that participants previously randomized to the intensive arm had a 42% reduction (P = 0.02) in CVD

outcomes and a 57% reduction (P = 0.02) in the risk of nonfatal myocardial infarction (MI), stroke, or CVD death compared with those previously in the standard arm (138-140).

The ADA and the EASD published an expert consensus statement on the approach to the management of hyperglycemia in individuals with type 2 diabetes (141). Highlights of this approach are: intervention at the time of diagnosis with metformin in combination with lifestyle changes (MNT and exercise) and continuing timely augmentation of therapy with additional agents (including early initiation of insulin therapy) as a means of achieving and maintaining recommended levels of glycemic control (i.e., A1C <7% for most patients). Whenever A1C targets are not achieved, treatment intensification is based on the addition of another agent from another class.

People with diabetes should be advised to perform at least 150 min/week of moderate-intensity aerobic physical activity (50–70% of maximum heart rate).In the absence of contraindications, people with type 2 diabetes should be encouraged to perform resistance training three times per week.
Structured exercise interventions of at least 8 weeks duration have been shown to lower A1C by an average of 0.66% in people with type 2 diabetes, even with no significant change in BMI (142).

Assessment of psychological and social situation should be included as an ongoing part of the medical management of diabetes.

Psychological and social problems can impair the individual's or family's ability to carry out diabetes care and therefore compromise health status. There are opportunities for the clinician to assess psychosocial status in a timely and efficient manner, so that referral for appropriate services can be accomplished (143-145).

The present study provides the first representative**,** population based estimates of Prediabetes (IFG & IGT) in Urban & Rural Gujarati community.

This study was designed to give the reliable state wise estimates of diabetes prevalence, as commonly people believes that Gujarati community have high affinity to Diabetes because of their food habit and affluent lifestyle.

Various epidemiological studies conducted in India during year 2000 to 2005 have shown the varied prevalence of Diabetes in Indian population. Out of these if, we review the studies conducted in western part of India (Yagnik et al from Pune in 2004 & Iyer et al from Dombivali in 2004, as per WHO guidelines) has shown the prevalence of 4.3% and 4.01% respectively (146).

Our study has also shown the crude prevalence of IFG in Gujarati population is around 2.76% and IGT is around 6.12%. But the age adjusted prevalence of IFG is around 2.72% and IGT is around 4.67%, which is comparable to the prevalence observed in other studies conducted in the past across the globe.

A recent survey in a rural area in 2003 showed indications of transition in the lifestyle of rural population and striking increase in the rate of prevalence of diabetes was noted (6.3%)(148-149).Our study shows that the prevalence of IFG in Rural area is around 4.62 % and IGT is around 4.32%.

Lifestyle changes due to urbanisation and aging of the population are two important determinant of rising prevalence of prediabetes (IFG & IGT) in developing countries such as India.

In an analysis of six prospective studies, the risk of developing diabetes was found to be approximately 3.6 to 8.7 percent per year in patients with IGT (147).

IFG and IGT frequently are associated with metabolic syndrome. The Adult Treatment Panel III of the National Cholesterol Education Program has identified metabolic syndrome as a constellation of lipid and non lipid risk factors for coronary artery disease (148).

The syndrome is characterized by insulin resistance, atherogenic dyslipidemia (high triglyceride level, low high-density lipoprotein cholesterol level, and small, dense low-density lipoprotein cholesterol particles), hypertension, abdominal obesity and prothrombotic and proinflammatory states.

As obesity or an increase in intra abdominal adipose tissue is associated with insulin resistance in the absence of diabetes, it is believed by some that, insulin resistance in type 2 diabetes is entirely due to the coexistence of increased adiposity (149).

We also noticed higher BMI, WHR, higher systolic blood pressure and diastolic pressure in IFG and IGT population in our study.

Compared with normoglycemic persons, patients with IGT are at substantially greater risk of developing cardiovascular disease (150).

We have also counseled all the subjects to have control diet mainly to curtail the carbohydrate to reduce the serum try glycerides.Changes in serum triglyceride and HDL cholesterol were more favorable with the low-carbohydrate diets. In one study, those subjects with type 2 diabetes demonstrated a greater decrease in A1C with a low-carbohydrate diet than with a low-fat diet (151).

Plasma levels of LDL-C generally do not differ significantly from those in patients without diabetes, but qualitative changes in LDL-C particles make them highly atherogenic (152-157). In our study,we also found that LDL-C is not much altered.However,we have noticedmore changes in Sr.Tryglyceride. In IFG the mean Sr.Tryglyceride is 178.44 $\pm$ 94.18 mg/dl and in IGT it is 175 $\pm$ 77.12 mg/dl.This is highly correlated with other facts in Indian population.

The primary goal with respect to dietary fat in individuals with diabetes is to limit saturated fatty acids, *trans* fatty acids, and cholesterol intake, so as to reduce risk for CVD.

We have also concluded that men have higher prevalence of IFG and IGT than female. This also matches with the observations from other international studies. The probable hypothesis for this development could be disturbances in glucose metabolism, accelerate the process of endothelial dysfunction.

Studies have also confirmed that prevalence of IFG and IGT is increasing as the age progresses. Most common in adults over age 40, this form of diabetes is strongly linked to obesity, inactivity, family history of diabetes, and racial or ethnic background. Our study has also proven the same and hence we are strongly recommending the control and/or precautions to be taken after age 40.

Obesity has an effect of on insulin resistance and hence weight loss is an important therapeutic objective for overweight or obese individuals with prediabetes or diabetes (158).

Obesity and a high fat diet may contribute to the development of both insulin resistance and insulin secretary dysfunction in susceptible individuals. Strategies that improve insulin resistance and enhance early insulin secretion may prevent the progression from IGT to diabetes. Already, there is substantial evidence the weight loss and exercise may reduce the risk of developing diabetes by up to 58% (159).

In our study, we have also noticed the higher mean BMI 26.81 $\pm$ 4.21 kg/m^2 and waist to hip ration 0.96 $\pm$ 0.07 in IFG population. Similarly for IGT population, where mean BMI is 29.36 $\pm$ 6.49 kg/m^2, WHR is 0.94 $\pm$ 0.12. This is more than the normoglycemic population. Hence, it proves that IFG and IGT population is more prone to develop obesity.
Patients with impaired glucose tolerance or impaired fasting glucose have a significant risk of diabetes and thus are important target for primary prevention (160).

At present, India is in the verge of a rapid epidemiological transition with increased urbanization. Present urbanization rate is 35% compared to 15% in the 1950's and this could have major implications on the present and future disease patterns in India with

context to diabetes and coronary artery disease. Socio-economic development over the last 40-50 years has resulted in a dramatic change in lifestyle towards more westernization, leading to physical inactivity due to technological advancement, affluence leading to consumption of fat rich diet, sugar and high level of mental stress. All these could adversely influence insulin sensitivity and lead to obesity (161).

Since 1970, several studies have been done comparing urban and rural populations in India, which have shown higher prevalence of diabetes among urban residents compared to their rural counterparts both in southern and northern parts of India (162).In this study, we found the prevalence of IFG in urban area is 4.32% and in rural area is 2.18%. But the prevalence of IGT in urban area is 5.84% and in rural is also 4.26%. We found prevalence of IFG and IGT is more in urban area.

In diabetic subjects, some surrogate measures of vascular pathology, such as endothelial dysfunction, are negatively affected by postprandial hyperglycemia (163). Studies have also confirmed that circulating endothelium factors (mainly PAI-I) are more in IGT and IFG population. This leads to the damage in the endothelial cells (164-165).

Currently, since last decade the percentage of people with diagnosed diabetes taking blood pressure medication has grown, around 36 percent of participants in the most recent NHANES met ADA's current blood pressure goal of less than 130/80 mmHg and 40 percent had high blood pressure. Around ~23% of Americans aged 20 to 74 years have high blood pressure (i.e, systolic blood pressure >140 mm Hg or diastolic blood pressure >90 mm Hg); in addition, 25% to 45% of these hypertensive patients are insulin resistant and dysmetabolic (166-168).

In our study, we also found the subjects with IFG and IGT had a high blood pressure. In IFG population mean systolic blood pressure was 135.54 $\pm$ 21.45 mm Hg and Diastolic blood pressure 82.92 $\pm$ 9.97 mm Hg, which was higher than normoglycemic population. Similarly, for IGT population systolic blood pressure was 130.19 $\pm$ 17.66 mm Hg and Diastolic blood pressure 83.64 $\pm$ 9.43 mm Hg.

We have counseled the subjects for control of blood pressure by non pharmacological measures first and then opt for pharmacological measures.

The link between hypertension and atherosclerosis, myocardial infarction and stroke has been well established through longitudinal observational research and clinical trials of antihypertensive drugs (169).

In nutshell, prediabetes population has high probability for getting converted into Diabetes in the long run (may be 3-5 years) (170). In any given year, 5% to 10% of glucose-intolerant patients progress to type 2 diabetics (171-173).

And prediabetes also develop diabetes dyslipidemia and lead to morbidity and mortality. Therefore, public attention to the high prevalence of IFG and Diabetes is requested.

In addition, first degree relatives of patients with type 2 diabetes have been found to have impaired insulin action upon skeletal muscle glycogen synthesis due to both decreased stimulation of tyrosine kinase activity of the insulin receptor and reduced glycogen synthase activity (174-175).

In our study, we have also correlated that the person who have the history of diabetes has 1.52 times more possibility of the development of IFG. Similarly, the person who has the history of diabetes has 1.58 times more possibility of developing IGT.

We also confirmed that, WHR is statistically significant in both the groups compare to the normoglycemic, it is said to be have correlation with Diabetes and Obesity.

HDL has the inverse relation, states that HDL is more in IFG and IGT population as compare to the normoglycemic. In the contrary, other lipid parameters such as LDL, serum cholesterol and serum triglycerides are elevated in IFG and IGT population and have the direct relationship with dyslipidemia and prediabetes. And may lead to diabetes dyslipidmia in the long run.

The management plan should be formulated as a collaborative therapeutic alliance among the patient and family, the physician and other members of the health care team. A variety of strategies and techniques should be used to provide adequate education and development of problem-solving skills in the various aspects of prediabetes management early stage.

There may be possible limitation of the study due to the sample size.

13. SUMMARY AND CONCLUSION:

This was the first representative study conducted to evaluate the prevalence of Prediabetes in Gujarat. Because of that, it has led highest significance, as till today we are relying on the various publications from other part of India and abroad.

From this study we conclude that, overall prevalence of IFG in Gujarat is around 2.72% and IGT is around 4.67%. And significantly observed that, the prediabetes prevalence is increasing day by day in urban and rural area because of the change in the lifestyle, food habit, working culture, affluence.

If we extrapolate with the total population of the state, Gujarat will have be around 1.31 million people of IFG and 2.30 million people with IGT are present currently. And if, we assume the same quantum of rise, than in next 10 years these numbers may become more than double i.e. around 4 million of IFG and 5 million of IGT alone. Along with that, if we assume the conversion rate from IFG and IGT to diabetes, than every 2-3 years around 80,000-140,000 new diabetes are added in to the current population. This is an impediment in the economy of the state and nation, as young population is converted into diabetes.

Lipid abnormality is also reported in this population along with glucose abnormality. This clearly indicates that, in this population along with glucose, we must focus on lipid abnormality corrections. Clinicians generally focus only on glucose, but we must focus on lipid too, to avoid the development of diabetes dyslipidemia. May be the stage can be classified as a pre dyslipidic/metabolic syndrome.

We also need to motivate the population, who are approaching at 40 years because in this age we found maximum prevalence of IGT. Even clinically also after this age, the micro vascular changes starts and may be precipitate with morbidity. As the morbidity increases to this age group it has a significant impact on pharmacoeconomical burden on an individual and at nation level.

We also evidently correlated, that the person who has family history of diabetes, in that group around 3.47% of IFG and 8.13% of IGT are found. Hence as an important point, we can first counsel the population, who has history of diabetes. Because this population, after age of 35 should start taking precautions to delay their diabetes development.

Also, we counseled the subjects for the diet restrictions mainly for transfat and carbohydrate consumption. Along with that the main emphasis was given on the physical exercise average around 150 minutes in a week.

As blood pressure is also one of the factors correlated in this population, people must focus on distressing exercise to avoid the development of blood pressure.

Gujarat has started rapid urbanization and industrialization after year 2005-06. Our study was conducted in 2007 and 2008, when the economic growth of the state was on the pick and affluence was also increasing. And hence to maintain the same economics of the state and nation, we must focus on the prevention of this deadly disease.

This study has given the new direction in the society and medical fraternity to make aware about the status of prediabetes and especially to Endocrinologists, Physicians, Healthcare workers, Medical practioners and Common men. We as a group also tried to educate to the people through media campaign. Some of the media coverage in national English and Gujarati dailies during on 14 November 2011 on the occasion of World Diabetes Day is attached herewith. *(Appendix-VI).*

Striking common attributable risk factors for the same in urban and rural areas are higher BMI, Waist to hip ratio, higher systolic and diastolic blood pressure, lipid abnormalities mainly higher triglycerides.

Our study also established the fact that risk factors, such as ageing and hypertension in western Indian population has strong relationship and concurs with the studies in the western populations.

Summarising, present study shows that IFG and IGT are associated for developing diabetes and they are associated with cardiovascular risk factors in the Western Indian population.
Thus, the findings of present study have important public health implications. Both IFG and IGT is not a clinical entity but rather a risk factor for future diabetes and cardiovascular disease.

The prediction by WHO, that India is likely to be the country having the largest number of diabetic subjects by the year 2025 seems to be true.

And ultimately the message of **"Prevention is better than Cure"** should be followed to avoid the situation.

14. REFERENCES:

1. Gerich JE: The genetic basis of type 2 diabetes mellitus: impaired insulin secretion *vs.* impaired insulin sensitivity. Endocr Rev 19:491–503; 1998.
2. DeFronzo RA: Pathogenesis of type 2 (non-insulin dependent) diabetes mellitus: a balanced overview. Diabetologia 35:389–397:1992.
3. Yki-Ja¨rvinen: H. Pathogenesis of non-insulin-dependent diabetes mellitus. Lancet 343:91–95; 1994.
4. Ferrannini E: Insulin resistance *vs.* insulin deficiency in noninsulin- dependent diabetes mellitus: problems and prospects. Endocr Rev 19:477–490; 1998.
5. Kahn CR Banting Lecture: Insulin action, diabetogenes, and the cause of type II diabetes. Diabetes 4:1066–1084; 1994.
6. Olefsky JM:Insulin resistance and the pathogenesis of noninsulin-dependent diabetes mellitus:cellular and molecular mechanisms. Adv Exp Med Biol 334:129–150; 1993.
7. Ha¨ring HU: Pathogenesis of type II diabetes: are there common causes for insulin resistance and secretion failure? Exp Clin Endocrinol Diabetes 107[Suppl.2]:S17–S23; 1999.
8. Report of the Expert Committee on the Diagnosis and Classification of Diabetes Mellitus Diabetes Care 22[Suppl 1]:S5–S19; 1999.
9. Shashank Joshi: Subclinical Diabetes: Vascular threat to Asian Indians. Editorial article of Journal of Physicians of India (JAPI) Vol.51; Aug 2003.
10. Beck Nielsen H, Groop LC: Metabolic and genetic characterization of prediabetic states.Sequence of events leading to noninsulin- dependent diabetes mellitus. J Clin Invest 94:1714–1721; 1994.
11. Stephan Matthaei, Michael Stumvoll, Monika Kellerer and Hans-Ulrich Häring: Pathophysiology and Pharmacological Treatment of Insulin Resistance Endocr. Rev. 21: 585-618; 2000.
12. Harris MI, Couric CC, Reiber G, Boyko E, Stern M, Bennet P, eds**:** Diabetes in America, 2nd ed. Washington DC: U.S. Printing Office; NIH publication 95–1468;1995.

13. Roman SH, Harris MI.Management of diabetes from a public health perspective. Endocrinal Metab Clin North Am. 26:443– 447; 1997.
14. Diabetes Control and Complications Trial Research Group**:** The effect of intensive treatment of diabetes in the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med. 329:683–689;1993.
15. U.K. Prospective Diabetes Study Group**:** Intensive blood glucose control with sulphonylureas or insulin compared with conventional treatment and risk of complications in patients with type 2 diabetes (UKPDS 33). Lancet. 352:837–853;1998.
16. U.K. Prospective Diabetes Study Group**:**Effect of intensive bloodglucose control with metformin on complications in overweight patients with type 2 diabetes (UKPDS 34). Lancet. 352:854–865;1998.
17. Centers for Disease Control and Prevention.National Diabetes Fact Sheet: National Estimates and General Information on Diabetes in the United States, 2000. Atlanta, Ga: US Department of Health and Human Services, Centers for Disease Control and Prevention; 2003.
18. David M.Nathan: Finding New Treatments for Diabetes -How Many, How Fast . How Good? NEJM, February 2007.
19. King H, Aubert RE, Herman WH: Global burden of Diabetes 1995-2025: prevalence, numerical estimates and projections Diabetes Care 21:1414-1431, 1998.
20. Ramchandra A,Snehlata C: Problem specific to developed countries and effect of westernization.: Hitman GA(ed) Type 2 Diabetes – Prediction and Prevention, Wiley,London,325-345,1999.
21. World Health Organization:Prevention of diabetes mellitus. Technical Report Series no. 844. WHO, Geneva, 1994.
22. Diabetes Atlas 2nd edition, 2003.
23. P.Z. Zimmet.Diabetes epidemiology as a tool to trigger diabetes research and care, Diabetologia 42, 499-518; 1999.

24. A.Ramachandran, C.Snehalatha, E.Latha, M.Manoharan,V.Vijay: Impacts of urbanization on the life style and on the prevalence of diabetes in native Asian Indian population, Diabetes Res. Clin. Pract. 44; 207-213; 1999.

25. U.K.Prospective Diabetes Study Group**:** Tight blood pressure control and risk of macrovascular and microvascular complications in type 2 diabetes (UKPDS 38).Br Med J. 317:703–713; 1998.

26. Wingard DL, Barrett-Connor E**:** Heart disease and diabetes.In: Harris MI, Couric CC, Reiber G, Boyko E, Stern M, Bennet P, eds. Diabetes in America, 2nd ed. Washington DC: U.S. Printing Office; NIH publication 95–1468; 429;1995.

27. Pyorala K, Pedersen TR, Kjekshus J, et al**:** Cholesterol lowering with simvastatin improves prognosis of diabetic patients with coronary heart disease. A subgroup analysis of the Scandinavian Simvastatin Survival Study (4S). Diabetes Care. 20:614–620; 1997.

28. Eisenberg DA**:** Cholesterol lowering in the management of coronary artery disease: the clinical implications of recent trials. Am J Med. 104:2S–5S;1998.

29. Koskinen P, Manttari M, Manninen V, Huttunen JK, Heinonen OP, Frick MH**:** Coronary heart disease in NIDDM patients in the Helsinki heart study. Diabetes Care. 15:820–825; 1992.

30. Mahler RJ**:** Diabetes and hypertension. Horm Metab Res. 22:599–607;1990.

31. P.A.Fransis: Fighting Diabetes Threat, Pharma Pulse, August 2009.

32. Ramchandran A, Mary S,Yamuna A,Murugesan N,Snehalatha C: High Prevalance of Diabetes and Cardiovasular risk factors associated with Urbanisation in India,Diabetologia.31(5):893:898;2008.

33. Moulinath Banerjee; J. Kennedy Cruickshank; Prediabtes is the term useful? British Journal of Diabetes and Vascular Disease.4(4);2004.

34. Will new diagnostic criteria for diabetes mellitus change phenotype of patients with diabetes? Reanalysis of European epidemiological data. DECODE Study Group on behalf of the European Diabetes Epidemiology Study Group. *BMJ*. 317:371-375; 1998.

35. Qiao Q, Hu G, Tuomilehto J *et al*: Age- and sex-specific prevalence of diabetes and impaired glucose regulation in 11 Asian cohorts. *Diabetes Care*; 26:1770-1780; 2003.

36. Qiao Q, Tuomilehto J, Borch-Johnsen K:Post-challenge hyperglycaemia isassociated with premature death and macrovascular complications. *Diabetologia*; 46(suppl 1):M17-M21;2003.

37. Qiao Q, Nakagami T, Tuomilehto J *et al*. Comparison of the fasting and the 2-h glucose criteria for diabetes in different Asian cohorts. *Diabetologia.* 43:1470-1475. 2000.

38. Pratley RE, Weyer C: Progression from IGT to type 2 diabetes mellitus: the central role of impaired early insulin secretion. Curr Diab Rep. 2(3):242-252; Jun 2002.

39. Prevalence of Diabetes and Impaired Fasting Glucose in Adults - United States, 1999-2000. 52(35). Atlanta, CDC. MMWR; 2004.

40. Harris MI, Flegal KM, Cowie CC *et al*: Prevalence of diabetes, impaired fasting glucose, and impaired glucose tolerance in U.S. adults. The Third National Health and Nutrition Examination Survey, 1988-1994. *Diabetes Care*; 21:518-524; 1998.

41. Glumer C,Jorgensen T, Borch-Johnsen K.Prevalences of diabetes and impaired glucose regulation in a Danish population: the Inter 99 study. *Diabetes Care*.26:2335-2340;2003.

42. Dunstan DW, Zimmet PZ, Welborn TA *et al*.The Rising Prevalence of Diabetes and Impaired Glucose Tolerance: The Australian Diabetes, Obesity and Lifestyle Study. *Diabetes Care*; 25:829-834;2002.

43. Gregory A Nicholas,Teresa A.Hillier and Jonathan B.Brown;Progression from newly acquired impaired fasting glucose to type 2 diabetes. Diabetes Care. 30:228-233,2007.

44. A.Ramachandran, C.Snehalatha, V. Vijay: Temporal changes in prevalence of type 2 diabetes and impaired glucose tolerance in urban southern India Diabetes Research and Clinical Practice. 58,55-60; 2002.

45. DNA, English daily Newspaper, Mumbai edition; 14 November 2006.

46. Hayden MR: Islet amyloid, metabolic syndrome, and the natural progressive history of type 2 diabetes mellitus. *JOP;* 3:126–138; 2002.

47. Bonora E, Kiechl S, Willeit J, et al: Prevalence of insulin resistance in metabolic disorders:The Bruneck Study. *Diabetes.* 47:1643–1649;1998.

48. Bloomgarden ZT.Insulin resistance: Current concepts. *Clin Ther.*20:216–231 ; 1998.

49. Kahn SE, Prigeon RL, McCulloch DK, et al: Quantification of the relationship between insulin-sensitivity and beta-cell function in human subjects. Evidence for a hyperbolic function. *Diabetes.*42:1663–1672;1993.

50. Kahne SE. The importance of the beta-cell in the pathogenesis of type 2 diabetes mellitus. Am J Med.108 (Suppl 6A):2S–8S; 2000.

51. Rosenbloom AL, Joe JR, Young RS, Winter WE. Emerging epidemic of type 2 diabetes in youth. *Diabetes Care.*22:345–354;1999.

52. Sinha R, Fisch G, Teague B, et al: Prevalence of impaired glucose tolerance among children and adolescents with marked obesity. *N Engl J Med.* 346:802–810; 2002.

53. Rowell HA, Evans BJ, Quarry-Horn JL,Kerrigan JR: Type 2 diabetes mellitus in adolescents.*Adolesc Med.*13:1–12;2002.

54. Kahn SE, Prigeon RL, McCulloch DK, et al: Quantification of the relationship between insulin-sensitivity and beta-cell function in human subjects. Evidence for a hyperbolic function. *Diabetes.*; 42:1663–1672;1993.

55. Harris MI, Couric CC, Reiber G, Boyko E, Stern M, Bennet P, eds: Diabetes in America, 2nd ed. Washington DC: U.S. Printing Office; NIH publication,95–1468;1995.

56. Mohan et al: Epidemiology of Type 2 Diabates; Indian J Med Research 125, pp 217-230; March 2007.

57. Raman Kutty V, Joseph A, Soman CR: High prevalence of type 2 diabetes in an urban settlement in Kerala, India. *Ethn Health*; *4* : 231-9;1999.

58. Shah SK, Saikia M, Burman NN, Snehalatha C,Ramachandran A: High prevalence of type 2 diabetes in urban population in north eastern India. *Int J Diabetes Dev Countries*; *19:* 144-147;1999.

59. Zargar AH, Khan AK, Masoodi SR, Laway BA, Wani AI,Bashir MI, *et al*: Prevalence of type 2 diabetes mellitus and impaired glucose tolerance in the Kashmir Valley of the Indian subcontinent. *Diabetes Res Clin Pract.47* :135-146;2000.

60. Ramachandran A, Snehalatha C, Kapur A, Vijay V, Mohan V, Das AK, *et al*.Diabetes Epidemiology Study Group in India (DESI): High prevalence of diabetes and impaired glucose tolerance in India: National Urban Diabetes Survey. *Diabetologia*. 44: 1094-1101; 2001.

61. Gupta A, Gupta R, Sarna M, Rastogi S, Gupta VP, Kothari K: Prevalence of diabetes, impaired fasting glucose and insulin resistance syndrome in an urban Indian population. *Diabetes Res Clin Pract. 61:* 69-76;2003.

62. Menon VU, Kumar KV, Gilchrist A, Sugathan TN, Sundaram KR, Nair V, *et al*: Prevalence of known and undetected diabetes and associated risk factors in central Kerala - ADEPS. *Diabetes Res Clin Pract*. 74: 289-294;2006.

63. Mohan V, Deepa M, Deepa R, Shantirani CS, Farooq S,Ganesan A, *et al*:Secular trends in the prevalence of diabetes and glucose tolerance in urban South India - the Chennai Urban Rural Epidemiology Study (CURES-17). *Diabetologia*.49:1175-1178;2006.

64. Reaven GM: The role of insulin resistance in human disease.Diabetes. 37:1595–1607;1998.

65. Olefsky JM DeGroot LJ, Besser GM, Cahill JC eds: Pathogenesis of non-insulin dependent diabetes (type 2). In. Endocrinology, 2nd ed. Philadelphia: Saunders; 1369–1388; 1989.

66. Ward WK, Beard JC, Porte D: Clinical aspects of islet B cell function in non-insulin dependent diabetes mellitus.Diabetes Metab Rev.2:297–313;1986.

67. Leahy JL: Natural history of B-cell dysfunction in NIDDM. Diabetes Care.13:992–1010,1991.

68. Porte D: B cells in type 2 diabetes mellitus. Diabetes.40:166–180;1991.

69. Malaisse W: Metabolic signaling of insulin secretion. Diabetes Rev.4:145–159. 1996.

70. Porte D, Kahn SE: Hyperproinsulinemia and amyloid in NIDDM: clues to etiology of islet B cell dysfunction? Diabetes. 38:1333–1336;1989.

71. O'Rahilly S, Turner RC, Matthews DR: Impaired pulsatile secretion of insulin in relatives of patients with non-insulin dependent diabetes mellitus. N Engl J Med. 318:1225–1230. 1988.

72. Nicholls JSD,Chan SP,Ali K,Beard RW, Dornhorst A: Insulin secretion and sensitivity in women fulfilling WHO criteria for gestational diabetes mellitus. Diabetes Med. 12:56–60; 1995.

73. Matchinsky F. Liang Y, Kesavan P, et al: Glucokinase as pancreatic B cell glucose sensor and diabetes gene. J Clin Invest. 92:2092–2098; 1993.

74. Byrne MM, Sturis J, Clement K, et al.Insulin secretory abnormalities in subjects with hyperglycemia due to glucokinase mutations. J Clin Invest. 93:1120 –1130; 1994.

75. Kruszynska Y, Olefsky JM: Cellular and molecular mechanism of non-insulin dependent diabetes mellitus. J Invest Med. 44:413– 428; 1996.

76. Jeng CY, Hollenbeck CB, Wu MS, et al: How does glibenclamide lower glucose concentration in patients with type 2 diabetes? Diabetes Med.6:303–308;1989.

77. Yalow RS, Berson SA: Immunoassay of endogenous plasma insulin in man. J Clin Invest. 39:1157–1162;1960.

78. Ferrannini E:Insulin resistance *vs.* insulin deficiency in non-insulin dependent diabetes mellitus: problems and prospects. Endocr Rev. 19:477–490;1998.

79. Gerich JE: The genetic basis of type 2 diabetes mellitus: impaired insulin secretion *vs.* impaired insulin sensitivity. Endocr Rev. 19:491–503;1998.

80. DeFronzo RA, Binder C, Wahren J, Felig P, Ferrannini E, Faber O: Sensitivity of insulin secretion to feedback inhibition by hyperinsulinemia. Acta Endocrinol (Copenh). 98:81– 84. 1981.

81. Del Prato S, Leonetti F, Simonson DC, Sheehan P, Matsuda M, DeFronzo RA: Effect of sustained physiologic hyperinsulinemia and hyperglycemia on insulin secretion and insulin sensitivity in man. Diabetologia. 37:1025–1035;1994.

82. Unger RH, Grundy S: Hyperglycemia as an inducer as well as a consequence of impaired islet cell function and insulin resistance: implications for the management of diabetes. Diabetologia. 28:119 –125;1985.

83. Fink RI, Wallace P, Brechtel G, Olefsky JM: Evidence that glucose transport is rate-limiting for in-vivo glucose uptake. Metabolism. 41:897–902;1992.

84. Yki-Jarvinen H: Glucose toxicity. Endocr Rev. 13:415–431;1992.

85. Arner P, Pollace T, Lithell H: Different etiologies of type 2 (non-insulin dependent) diabetes mellitus in obese and non obese subjects. Diabetologia.34:483– 487;1991.

86. Kissebah A, Freedman D, Peiris A: Health risk of obesity. Med Clin North Am. 73:111–138;1989.

87. Bjorntop P: Abdominal obesity and the development of non-insulin dependent diabetes mellitus. Diabetes Metab Rev. 4:615– 622; 1988.

88. Reaven GM: The role of insulin resistance in human disease. Diabetes.37:1595–1607; 1998.

89. Ferrannini E, Stern MP**:**Primary insulin resistance: a risk syndrome. In: Leslie RDG, Robbins DC, eds. Diabetes: clinical science in practice. Cambridge University Press; 200–220;1995.

90. Nabulsi AA, Folsom AR, Heiss G, Weir SS, Chambless LE, Wadson RL,Eckfeldt JH:Fasting hyperinsulinemia and cardiovascular risk factors in non-diabetic adults: stronger associations in lean vs. obese subjects. Metabolism. 44:914 –922;1995.

91. Charles MA, Eschwege E, Thibult N, et al: The role of non-esterified fatty acids in the deterioration of glucose tolerance in Caucasian subjects: results of the Paris prospective study. Diabetologia. 40:1101–1106;1997

92. Fagan TC, Deedwania PC: The cardiovascular dysmetabolic syndrome. Am J Med. 105:775–825;1998.

93. Gelding SV, Goldham N, Nithbyananthan R, Anyaoku V, Johnston DG: Insulin resistance with respect to lipolysis in non-diabetic relative of European patients with type 2 diabetes. Diabetes Med. 12:66 –73; 1995.

94. Gulli G, Ferrannini ETL, Stern M, Haffner S, DeFronzo R: The metabolic profile of NIDDM is fully established in glucose-tolerant offspring of two Mexican-American NIDDM parents. Diabetes. 41:1575–1587; 1992.

95. Pimenta W, Korytkowski M, Mitrakou A, et al: Pancreatic b-cell dysfunction as the primary genetic lesion in NIDDM. JAMA. 273:1855–1861; 1995.

96. O'Rahilly S, Turner R, Matthews D: Impaired pulsatile secretion of insulin in relatives of patients with non-insulin dependent diabetes. N Engl J Med. 318:1225–1230;1988.

97. Chapter titled "Insulin Resistance,Dyslipedemia and Coronary Artery Disease in Type 2 Diabetes Mellitus-The Indian Perspective" from Book "Coronary Artery Disease in Indians-A Global Perspective" by Dr.Kamal K.Shethi

98. www.cmeondiabetes.com. As per the last assessed on June,2010.

99. Laakso M. Epidemiology of diabetic dyslipidemia. Diabetes Rev; 3:408–422;1995.

100. www.uptodate.com/contents/patient-information-diabetes-mellitus-type-2-treatment. As per the last assessed on February 28,2011.

101. Charles, M. A., Fontboune, A., Thibult, N., Warnet, J. M., Rosselin, G. E. & Eschwege, E. Risk factors for NIDDM in white population. Paris prospective study. Diabetes 40, 796-799; 1991.

102. Fernando Guerrero-Romero, Martha RodrõÂguez-MoraÂn: Journal of Diabetes and its complications; 15, 34-37;2001

103. Brunzell, J. D., Robertson, R. P., Lerner, R. L., Hazzard, W. R., Ensink, J. W., Biewrman, E. L., & Porte, D. Jr: Relationship between fasting plasma glucose levels and insulin secretion during intravenous glucose tolerance test. J Clin Endocrinol Metab 42, 222- 229; 1976.

104. Gniuli, D., Mingrone, G., Strollo, F. Strollo, G. & Grevo, A: Do the new screening and diagnostic criteria of diabetes proposed by the American Diabetes Association really match? Diabetes Care 22,367-368; 1999.

105. Ollerton, R., Playle, R., Luzio, S., & Owens, D: Under diagnosis of type 2 diabetes by use of American Diabetes Association. Diabetes Care 22, 649, 1999 (letter).

106. Fornengo, P., Bruno, A., Grassi, G., Vineis, P., & Pagano, G: Concordance between American Diabetes Association and World Health Organization criteria in a Northwestern Italian population. Diabetes Care 22, 652, 1999 (letter).

107. Unwin N,Shaw J,Zimmet P,Alberti KG: Impaired glucose tolerance and impaired fasting glycemia the current status on definition and intervention. Diabet Med, 19:708-723; 2002.

108. Harris MI, Eastman RC, Cowie CC, Flegal KM,Eberhardt MS: Comparison of diabetes diagnostic categories in the U.S. population according to the 1997 American Diabetes Association and 1980-1985 World Health Organization diagnostic criteria. Diabetes Care; 20:1859-1862; 1997.

109. National Center for Health Statistics. The Third National Health and Nutrition Examination Survey (NHANES III, 1988-94) reference manuals and reports. Hyattsville, Md.: U.S. Dept. of Health and Human Services, Centers for Disease Control and Prevention, National Center for Health Statistics, 1996.

110. Tominaga et al**.**Impaired Glucose Tolerance and Mortality from Cardiovascular Diseases: Funagata Diabetes Study. Yamagata Med J; Vol: 18:37-44;2000.

111. Sicree, R. A., Zimmet, P. Z., King, H. O. & Coventry, J. S: Plasma insulin response among Nauruans: prediction of deterioration in glucose tolerance over 6 years. Diabetes 36.779-786; 1987.

112. Gomez-Perez, F. J., Aguilar-Salinas, J. C., Lopez-Alveranga, J. C., Perez-Jauregui, J., Guillen-Pineda, L. E., & Rull, J. A: Lack of agreement between the World Health Organization category of impaired glucose tolerance and the American Diabetes Association category of impaired fasting glucose. Diabetes Care 21. 1886-1888; 1998.

113. Gimeno, S. G. A., Ferreira, S., Franco, L., & Iunes, M:Comparison of glucose tolerance categories according to World Health Organization and American Diabetes Association diagnostic criteria in a population based study in Brazil. Diabetes Care 21, 1889-1892; 1998.

114. Vegt, F., Dekker, J. M., Stehouwer, C., Nijpels, G., Bouter, L., & Heine, R: The 1997 American Diabetes Association criteria versus the 1985 World Health Organization criteria for the diagnosis of abnormal glucose tolerance. Diabetes Care 21. 1686- 1690; 1998.

115. Guerrero-Romero, F., Rodriguez-Moran, M., & Alvarado-Ruiz, R:Concordance between the 1997 fasting ADA criteria and the WHO criteria in healthy Mexican subjects. Diabetes Care 22. 527; 1999 (letter).

116. Lal Somani, B., Bangar, S. S., & Bhalwar, R: American Diabetes Association criteria for diabetes diagnosis. Diabetes Care. 22,366; 1999 (letter).

117. Genuth S, Alberti KG, Bennett P, Buse J, Defronzo R, Kahn R, et al: Follow-up report on the diagnosis of diabetes mellitus. Diabetes Care. 26: 3160-7; 2003.

118. Knowler WC, Barrett-Connor E, Fowler SE, Hamman RF, Lachin JM, Walker EA, et al:Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. N Engl J Med.346:393-403;2002.

119. Tuomilehto J, Lindstrom J, Eriksson JG, Valle TT,Hamalainen H, Ilanne-Parikka P, et al:Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. N Engl J Med. 344:1343-1350; 2001.

120. Pan XR, Li GW, Hu YH, Wang JX, Yang WY, An ZX, et al: Effects of diet and exercise in preventing NIDDM in people with impaired glucose tolerance. The Da Qing IGT and Diabetes Study. Diabetes Care. 20:537-44; 1997.

121. Norris SL, Engelgau MM, Narayan KM: Effectiveness of self-management training in type 2 diabetes: a systematic review of randomized controlled trials. Diabetes Care.24:561–587;2001.

122. Norris SL, Lau J, Smith SJ, Schmid CH, Engelgau MM:Self-management education for adults with type 2 diabetes: a meta analysis of the effect on glycemic control. Diabetes Care. 25:1159–1171; 2002.

123. Gary TL, Genkinger JM, Guallar E, Peyrot M, Brancati FL: Meta-analysis of randomized educational and behavioral interventions in type 2 diabetes. Diabetes Educ; 29:488–501; 2003.

124. Steed L, Cooke D, Newman S:A systematic review of psychosocial outcomes following education, self-management and psychological interventions in diabetes mellitus. Patient Educ Couns; 51:5–15;2003.

125. Ellis SE, Speroff T, Dittus RS, Brown A, Pichert JW, Elasy TA:Diabetes patient education: a meta-analysis and meta-regression. Patient Educ Couns; 52: 97–105; 2004.

126. Warsi A, Wang PS, LaValley MP, Avorn J, Solomon DH:Self-management education programs in chronic disease: a systematic review and methodological critique of the literature. Arch Intern Med. 164:1641–1649;2004.

127. Mulcahy K, Maryniuk M, Peeples M,Peyrot M, Tomky D, Weaver T, Yarborough P: Diabetes self-management education core outcomes measures. Diabetes Educ;29:768–784;2003.

128. Funnell MM, Brown TL, Childs BP, Haas LB, Hosey GM, Jensen B, Maryniuk M, Peyrot M, Piette JD, Reader D, Siminerio LM, Weinger K, Weiss MA: National standards for diabetes self-management education. Diabetes Care.30: 1630–1637;2007.

129. Wild S, Roglic G, Green A, Sicree R, King H:Global prevalence of diabetes. Estimates for the year 2000 and projections for 2030. *Diabetes Care*. 27: 1047-1053;2004.

130. Kapur A: Cost of Diabetes in India. The CODI Study Paper presented at the Novo Nordisk *Diabetes Update*, Bangalore, February 2000.

131. Björk S, Kapur A, Kelkar S, Nair JD, Ramachandran A:Aspects of diabetes in India: A nationwide survey. *Research and Clinical Forums*. 25(1): 5-34;2003.

132. American Diabetes Association website; www.ada.org. As per the last assessed on May 2007.

133. www.censusindia.net As per the last assessed on December 2009.

134. www.ndep.nih.gov. As per the last assessed on June 2010.

135. Sharon H. Saydah,,Judith Fradkin,,Catherine C. Cowie: Poor Control of Risk Factors for Vascular Disease Among Adults With Previously Diagnosed Diabetes Journal of American Medical Association. January 21, Vol.291, 335-342, 2004.

136. The Diabetes Control and Complications Trial Research Group. (DCCT). The effect of intensive treatment of diabetes on the development and progression of long-term complications in insulin-dependent diabetes mellitus. N Engl J Med;329:977–986;1993

137. Stratton IM, Adler AI, Neil HA, Matthews DR, Manley SE, Cull CA, Hadden D,Turner RC, Holman RR:Association of glycaemia with macrovascular and microvascular complications of type 2 diabetes (UKPDS 35): prospective observational study. BMJ. 321:405–412;2000

138. Selvin E, Marinopoulos S, Berkenblit G,Rami T, Brancati FL, Powe NR, Golden H:Meta-analysis: glycosylated hemoglobin and cardiovascular disease in diabetes mellitus. Ann Intern Med.141:421–431;2004

139. Stettler C, Allemann S, Ju¨ ni P, Cull CA, Holman RR, Egger M, Kra¨henbu¨ hl S, Diem P: Glycemic control and macrovascular disease in types 1 and 2 diabetes mellitus: Meta-analysis of randomized trials. Am Heart J. 152:27–38; 2006.

140. Nathan DM, Cleary PA, Backlund JY, Genuth SM, Lachin JM, Orchard TJ, Raskin P, Zinman B:Diabetes Control and Complications Trial/Epidemiology of Diabetes Interventions and Complications (DCCT/EDIC) Study Research Group. Intensive diabetes treatment and cardiovascular disease in patients with type 1 diabetes. N Engl J Med.353:2643–2653 ;2005

141. Nathan DM, Buse JB, Davidson MB, Ferrannini E, Holman RR, Sherwin R, Zinman B: American Diabetes Association, European Association for Study of Diabetes. Medical management of hyperglycemia in type 2 diabetes: a consensus algorithm for the initiation and adjustment of therapy: a consensus statement of the American Diabetes Association and the European Association for the Study of Diabetes. Diabetes Care. 32:193–203; 2009.

142. Boule´ NG, Haddad E, Kenny GP, Wells GA, Sigal RJ:Effects of exercise on glycemic control and body mass in type 2 diabetes mellitus: a meta-analysis of controlled clinical trials. JAMA. 286:1218–1227;2001.

143. Anderson RJ, Grigsby AB, Freedland KE, de Groot M, McGill JB, Clouse RE, Lustman PJ:Anxiety and poor glycemic control:a meta-analytic review of the literature. Int J Psychiatry Med.32:235–247;2002.

144. Delahanty LM, Grant RW, Wittenberg E,Bosch JL, Wexler DJ, Cagliero E, Meigs JB:Association of diabetes-related emotionaldistress with diabetes treatment in primary care patients with Type 2 diabetes.Diabet Med.24:48–54;2007.

145. Anderson RJ, Freedland KE, Clouse RE, Lustman PJ:The prevalence of co morbid depression in adults with diabetes: a meta-analysis. Diabetes Care.24:1069–1078;2001.

146. Yagnik CS:Early life origin of Insulin resistance and type 2 diabetes in India and other Asian Countries. J Nutr. 134:205-210; 2004.

147. Edelstein SL, Knowler WC, Bain RP, Andres R, Barrett-Connor EL, Dowse GK, et al:Predictors of progression from impaired glucose tolerance to NIDDM: an analysis of six prospective studies. Diabetes.46:701-710;1997.

148. Executive Summary of the third report of the National Cholesterol Education Program (NCEP) Expert Panel on Detection, Evaluation, and Treatment of High Blood Cholesterol in Adults (Adult Treatment Panel III). JAMA. 285:2486-2497;2001.

149. Carey, D, Jenkins A, Campbell L, Freund J, Chisholm D:Abdominal fat and insulin resistance in normal and overweight women: direct measurements reveal a strong relationship in subjects at both low and high risk of NIDDM. Diabetes. 45:633–638. 1996

150. Tominaga M, Eguchi H, Manaka H, Igarashi K, Kato T, Sekikawa A:Impaired glucose tolerance is a risk factor for cardiovascular disease, but not impaired fasting glucose. The Funagata Diabetes Study. Diabetes Care. 22:920-924; 1999.

151. Stern L, Iqbal N, Seshadri P, Chicano KL, Daily DA, McGrory J, Williams M, Gracely EJ, Samaha FF:The effects of low-carbohydrate versus conventional weight loss diets in severely obese adults: one-year follow-up of a randomized trial. Ann Intern Med. 140:778–785; 2004.

152. Steinberg D, Parthasarathy S, Carew TE, et al:Beyond cholesterol. Modifications of low-density lipoprotein that increase its atherogenicity. *N Engl J Med.*320: 915–924, 1989

153. Austin MA: Plasma triglyceride as a risk factor for coronary heart disease. The epidemiologic evidence and beyond. *Am J Epidemiol.* 129:249–259; 1989.

154. Cominacini L, Garbin U, Pastorino AM, et al: Increased susceptibility of LDL to in vitro oxidation in patients with insulin-dependent and non-insulin-dependent diabetes mellitus. *Diabetes Res.*26:173–184;1994

155. Chait A, Brazg RL, Tribble DL, Krauss RM:Susceptibility of small, dense, low-density lipoproteins to oxidative modification in subjects with the atherogenic lipoprotein phenotype, pattern B. *Am J Med.* 94:350–356;1993.

156. de Graaf J, Hak-Lemmers HL, Hectors MP, et al:Enhanced susceptibility to in vitro oxidation of the dense low density lipoprotein subfraction in healthy subjects. *Arterioscler Thromb.* 11:298–306; 1991.

157. Brown V:Detection and management of lipid disorders in diabetes. *Diabetes Care*. 19(Suppl 1):S96–S102; 1996.

158. Norris SL, Zhang X, Avenell A, Gregg E, Bowman B, Schmid CH, Lau J: Longterm effectiveness of weight-loss interventions in adults with prediabetes: A review. Am J Prev Med. 28:126–139;2005.

159. Pratley RE,Weyer C: aCurr Diab Rep. Jun; 2(3):242-248. 2002.

160. Shobha S.Rao,Philip Disraeli and Tamar McGregor: Impaired Glucose Tolerance and Impaired Fasting Glucose, Am Fam Physician, 69, 1961-1968, 1971-1972; 2004.

161. V.Mohan: Why Indians are more prone to Diabetes? Journal of Physicians of India (JAPI) Vol.52, June 2004.

162. Deepa M, Pradeepa R, Rema M, Mohan A, Deepa R, Shanthiarani S, *et al*: The Chennai Urban Rural Epidemiology Study (CURES) study design and Methodology. Urban Component (CURES-1),JAPI Vol.51,863-870,September 2003.

163. Ceriello A, Taboga C, Tonutti L, Quagliaro L, Piconi L, Bais B, Da Ros R, Motz E: Evidence for an independent and cumulative effect of postprandial hypertriglyceridemia and hyperglycemia on endothelial dysfunction and oxidative stress generation: effects of short- and long-term simvastatin treatment.Circulation.106:1211–1218;2002.

164. Collier A, Rumley A, Rumley AG, Paterson JR, Leach JP, Lowe GDO, Small M: Free radical activity and haemostatic factors in NIDDM patients with and without microalbuminuria, Diabetes. 41:909-913, 1992.

165. Paul B Leurs, Ronald P.Stolk, Karly Hamulyak, Rene Van Oerle, Diederick E Grobbee, Bruce H.R.Wolffenbuttel: Tissue Factor Pathway Inhibitor and Other Endothelium-Dependent Haemostatic Factors in Elderly Individuals with Normal and Impaired Glucose Tolerance and Type 2 Diabetes. Diabetes Care. Vol.25, 1340-1345, November 8, August 2002.

166. Lind L,Berne C,Lithell H: Prevalence of insulin resistance in essential hypertension.*J Hypertens.*13:1457–1462;1995

167. Vanhala MJ, Pitkajarvi TK, Keinanen-Kiukaanniemi SM, et al: Hyperinsulinaemia in hypertensive subjects: Validity of a test for the detection of insulin resistance in clinical practice. *J Hum Hypertens.* 12:463–467;1998.

168. Hyattsville, Md. National Center for Health Statistics. *Health, United States, 2002 With Chartbook on Trends in the Health of Americans:* National Center for Health Statistics:2002.

169. MacMahon S, Peto R, Cutler J, et al: Blood pressure, stroke, and coronary heart disease. Part 1, Prolonged differences in blood pressure: Prospective observational studies corrected for the regression dilution bias. Lancet. 335:765–774; 1990.

170. Jaako Tuomilehto along with The Finish Diabetes Prevention Study Group. Prevalence of Type 2 Diabetes Mellitus by changes in lifestyle among subjects with Impaired Glucose Tolerance. NEJM, 344, 1343-50; 2001.

171. Tuomilehto J, Lindstrom J, Eriksson JG, et al:Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *N Engl J Med*. 344:1343–1350; 2001.

172. Weyer C, Bogardus C, Mott DM, Pratley RE: The natural history of insulin secretory dysfunction and insulin resistance in the pathogenesis of type 2 diabetes mellitus.*J Clin Invest.* 104:787–794;1999.

173. Knowler WC, Barrett-Connor E, Fowler SE, et al.Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *N Engl J Med.*;346:393–403; 2002.

174. Vaag A, Henriksen JE, Beck-Nielsen H:Decreased insulin activation of glycogen synthase in skeletal muscles of young non-obese Caucasian first degree relatives of patients with non-insulin dependent diabetes mellitus. J Clin Invest. 89:782–788. 1992.

175. Handberg A, Vaag A, Vinten J: Decreased tyrosine kinase activity in partially purified insulin receptors from muscle of young, non-obese first degree relatives of patients with type 2 (non-insulin dependent) diabetes mellitus. Diabetologia. 36:668–674. 1993

15. APPENDICES:

Appendix I : Informed Consent Form

Patient Information

Diabetes is now one of the most common non-communicable diseases globally. This is virtually true with reference to Indian subcontinent, which has been declared by the World Health Organisation as the "Diabetes Capital of the World". Prevalence of type 2 diabetes is rising globally and impact is most marked in developing countries like India. Asian Indians have a racial predisposition to develop diabetes. By year 2025 there will be more than 75 million (7.5 cr) Indians will be suffering from Diabetes. As per year 2003 information in Gujarat alone 45 lakh people were suffering from Impaired Glucose Tolerance (IGT). Also there will be increase in the population, which will have problem of Insulin secretion and Insulin action.

The objectives of the study to determine the prevalence of prediabetes in the rural and urban population of Gujarat and also to identify the major risk factors for the increase in this incidence.

You will be asked to give blood for the following laboratory investigations.

- Fasting blood glucose
- 2 h Blood Glucose (2 hours after taking 75 gm glucose)
- Below mentioned tests will be done to assess your lipid profile
    - Total cholesterol
    - Serum Tryglycerides
    - Serum HDL cholesterol
    - Serum LDL cholesterol

Patient Consent Form

I …… exercising my free power of choice, hereby give my consent to be included as a subject in the epidemiological study for diabetes. I have been informed to my satisfaction, by the attending physician/care taker the purpose of the study and the importance of blood withdrawal and follow up including the laboratory investigation to monitor and safeguard my body function.

I am also aware of my right to opt out of the trial at any time during the course of the trial without having to give the reasons for doing so.

Signature of the patient/volunteer_____ Date:_____

Thumb impression of patient, if applicable

Date: Signature of the attending physician/care taker

Appendix II: Case Record Form

Case Record Form

Name of the Individual : _____

Sex: Male: ☐ Female: ☐

Date of Birth: _____ **Age:** _____ years

Race: Indian_____ Others:_____

Address:_____
_____
_____

Area: Urban☐ Rural☐

Telephone: STD Code_____ (R) _____ (O) _____

Mobile: _____

Email Address:_____

Marital Status:_____

Family Details:

- No.of Family members: _____
- Spouse:_____
- Details of Children(s): _____ Age: _____
- Family History of Diabetes: Yes___ No___

Details of Diabetes:_____
_____
_____

Previous History of Cardiac Disease:

Education Level:

High school ☐ Graduate ☐ Post Graduate ☐ Others ☐

Occupation:

Business ☐ Office Job ☐ Field Job ☐ Farming ☐ Others ☐

Physical activity per Day:

< 30 min/24 hr	*Sedentary*
30 min-59 min/24 hr	Mild
> 60 min – 90-min/24 hr	Moderate
> 90 min/24 hr	Heavy

Type of Activity:

Cycling ☐ Walking ☐ Plowing ☐ Digging ☐ Gardening ☐

Crop carrying ☐ Manual irrigation ☐

Smoking: Smoker ☐ Nonsmoker ☐

Economic Classification *(as per Modified B.G.Prasad):*

Per capital Monthly income limit (in Rupees)	**Approximately equivalent economic status**
2310 and above	Upper class -I
1155-2309	Upper middle-II
690-1154	Upper Lower-III
346-689	Lower Middle IV
≤ 345	Lower Lower V

Monthly Income: in Rs. _____

Details about Meals:

a) Vegeterian ☐ Nonvegetarian ☐

b) Morning Meal:

_____
_____
_____

c) Evening Meal:

_____
_____
_____

d) Snacks:

_____
_____
_____

Oil consumption:_____ ml in a day

Milk product consumption:_____ ml in a day

- **Carbohydrate:**_____
- **Proteins:**_____
- **Fat:**_____
- **Approx.Cal:**_____/day

Demographic Details:

a) **Height: _____ in cm _____ meter**

b) **Weight:_____ in kg**

c) **Waist: _____ in cm**

d) **Hip:_____ in cm**

e) **Waist/Hip ratio:_____**

f) **BMI: Weight in kg/Height (m^2) _____**

Laboratory Investigations

a) Fasting Blood Glucose_____mg/dl

b) 2 h Blood Glucose_____ mg/dl

c) Serum Cholesterol_____mM /L

d) Serum Triglycerides_____ mM/L

e) Serum HDL Cholesterol_____mM/L

f) Systolic Blood Pressure_____ mm Hg

g) Diastolic Blood Pressure_____ mm Hg

Comments:

_____
_____
_____

Name of Investigator:_____

Signature: _____ Date:_____

Appendix III: Ethics Committee Approval Letter

The Ethics Committee
B J Medical College & Civil Hospital, Ahmedabad

Chairman
Dr M M Prabhakar
Medical Superintendent
Civil Hospital
Ahmedabad 380 016
Ph: 22683721 Ext 1001

Member-Secretary
Dr R K Dikshit
Professor/Head, Dept of Pharmacology
B J Medical College
Ahmedabad 380 016
Ph: 22683721 Ext 1275

(Please address all communications to Member-Secretary)

Ref No. EC/A/08/07

CERTIFICATE OF APPROVAL

The Ethics Committee met on 16.5.07 at 3.00 PM at the Office of the Medical Superintendent, Civil Hospital, Ahmedabad. Dr. M. M. Prabhakar, Medical Superintendent & Chairman, The Ethics Committee, presided over the meeting. Following members attended:

1. Dr R K Dikshit, MD, Professor/Head, Department of Pharmacology, B J Medical College, Ahmedabad - Member-Secretary.
2. Dr. G. K. Vankar, MD, Professor/Head, Department of Psychiatry, B J Medical College & Civil Hospital, Ahmedabad- Member
3. Dr B D Mankad, MD, Professor/Head, Department of Medicine, B J Medical College and Civil Hospital, Ahmedabad - Member
4. Dr B R Leuva, MD, Professor, Department of Obstetrics & Gynecology, B J Medical College & Civil Hospital, Ahmedabad - Member (in lieu of Dr Malini Desai)
5. Dr (Mrs) Aruna Dewan, MD, Consultant, NIOH, Ahmedabad
6. Mr Amar Vyas, MSW, Social Worker, Department of Community Medicine, B J Medical College, Ahmedabad - Member
7. Mr P K Yadav, LI B, Advocate, Gujarat High Court - Member

The Committee reviewed the following project:

Title: To assess the prevalence and attributable risk factors of diabetes mellitus in western Indian population
Site where the project will be conducted: Department of Medicine, B J Medical College & Civil Hospital, Ahmedabad
Principal Investigator: Dr. Bhagirath B Solanki, Associate Professor of Medicine, B J Medical College & Civil Hospital, Ahmedabad
Sponsors : None

Documents reviewed:

1. Study synopsis (Protocol)
2. Product Summary
3. Informed Consent Form
4. Case Record Form
5. Relevant References (Full Text)

Subsequent to the fulfillment of the procedural requirements and other conditions imposed by the Committee the project is hereby granted an approval. The approval is for the entire duration of the study. Permission to start the work on the same is also given. The Principal Investigator and the Sponsors are required to read and comply with the following:

1. The Committee has approved the ethical aspects of the proposed work. However, all other concerns related to the work (e.g. scientific, procedural, legal, financial and regulatory etc) remain the sole responsibility of the Principal Investigator and/or the Sponsor(s).

2. The Committee must be informed from time to time about the progress of the work and a full report including the audited statement of accounts should be submitted within one month of the end of this work.

3. Any serious/unexpected side effects must be communicated to the Committee and any further instructions thereupon should be duly followed.

Dr. R. K. Dikshit
Member-Secretary
The Ethics Committee
B. J. Medical College & Civil Hospital
Ahmedabad-380 016

Ahmedabad
Dated, May, 16, 2007

Copy to:

Dr Bhagirath B Solanki, Associate Professor of Medicine, B J Medical College & Civil Hospital, Ahmedabad

Appendix IV: Photographs of the Camps

Appendix V: Publications

"To determine the pevalence of prediabetes in the rural population of Gujarat, India"

1116638

The Objective of the Study to Determine the Prevalence of Prediabetes in the Rural Population of Gujarat, India

Chirag H. Shah

Dept.of Pharmaceutical Sciences, Saurashtra University, Rajkot, India

Statement of Purpose, Innovation or Hypothesis: The purpose is to find out the prevalence of Prediabetes population which covers both Impaired Fasting Glucose and Impaired Glucose Tolerance in the Rural population as the westernization is increasing day by day.

Description of Methods and Materials: In year 2007 & 2008, a cross sectional survey was conducted via mode of camps at various rural part of Gujarat. After obtaining a proper consent form, comprehensive questionnaire was used to collect the various anthropological details, Physical examination and blood collection was done from around 822 subjects > 20 years of age from the different area of Gujarat. Chi square test was used for all categorical comparisons. Also multiple logistic regression was used for detailed exploratory analysis.

Data and Results: The crude prevalence of IFG in rural Gujarati population is around 4.62 % and IGT is around 4.32 %. If we extrapolate these to Gujarat's population, it indicates that around 2.31 million people are having impaired fasting glucose and around 2.16 million people have impaired glucose tolerance. The prevalence of IGT found more after age of 40 years. For IFG, there is increase after age of 40 years, but not significant statistically.

Interpretation, Conclusion or Significance: High prevalence of IFG and IGT validates that there are chances of the pandemic trend in Gujarat, as eventually IGT may gets converted into Diabetics in near future. These result needs urgent attention to develop a public awareness programme.

Prevalance of IFG and IGT in Rural Indian Population

		IFG		IGT	
Gender	n=	822	100%	786	100%
	Male	27	3.28%	22	2.81%
	Female	11	1.33%	12	1.53%

39TH RSSDI 2011 » PROCEEDINGS

Mumbai ▪ 4-6 November, 2011

7 Dyslipidemia in Pre Diabetes population in the rural area of Gujarat, India.

Chirag.H. Shah*, Navin R.Sheth, Bhagirath Solanki***, Nimisha Shah@**

*DEPT.OF PHARMACEUTICAL SCIENCES, SAURASHTRA UNIVERSITY, RAJKOT; INDIA; **PROF.& HEAD, DEPT.OF PHARMACEUTICAL SCIENCES, SAURASHTRA UNIVERSITY, RAJKOT, INDIA; ***ASSO.PROF.& HEAD OF THE UNIT, DEPT.OF MEDICINE,B.J.MEDICAL COLLEGE & CIVIL HOSPITAL, AHMEDABAD, INDIA; @FREELANCE CONSULTANT, AHMEDABAD, INDIA.

OBJECTIVE:

The purpose is to find out the prevalence of Dyslipidemia in prediabetes population (IFG and IGT) in the rural area of Gujarat.

METHODS:

In year 2007 & 2008, a cross sectional survey was conducted via mode of camps at various rural parts of Gujarat. After obtaining a proper consent form, comprehensive questionnaire was used to collect the various anthropological details. Physical examination and blood collection for glucose and lipid profile was done from around 822 subjects > 20 years of age from the different areas of Gujarat. Chi square test was used for all categorical comparisons.

RESULTS:

Table: 1 IFG and IGT prevalence.

	IFG	(%)	IGT	(%)
n=	822	100	786	100
Male	27	3.28	22	2.81
Female	11	1.33	12	1.53

The crude prevalence of IFG in rural Gujarati population is around 4.62 % and IGT is around 4.32%.

In IFG population, the mean value of serum cholesterol is 227.32 ± 48.67, serum triglycerides is 189.83 ± 109.98, LDL cholesterol is 144.94 ± 31.84 and HDL cholesterol is 47.66 ± 6.38.

In IGT population, the mean value of serum cholesterol is 198.54 ± 35.98, serum triglycerides is 191.47 ± 92.02, LDL cholesterol is 122.20 ± 25.99 and HDL cholesterol is 44.35 ± 4.35.

The level of all lipid parameters is significantly on higher side in prediabetes population as compare to normoglycemic. It proves that, the IFG& IGT population also has the dyslipidemia along with the abnormality in glucose values. Among this, serum triglycerides are significantly higher in IFG and IGT population.

CONCLUSION:

High prevalence of IFG and IGT validates that, there are chances of the pandemic trend in Gujarat, as eventually IGT may gets converted into Diabetics in near future.

Generally, we focus only on glucose management in this population. However from this study, we conclude that along with glucose, we must monitor the lipid parameters from beginning. This may help to prevent diabetic dyslipidemia.

These result needs urgent attention to develop a public awareness programme preferably by health department, medical fraternity and NGO working in this area.

Letter from the Hon.editor

--- On **Thu, 26/5/11, Siddharth Shah** ***<drsns@in.com>*** wrote:

From: Siddharth Shah <drsns@in.com>
Subject: japi
To: shahhchirag@yahoo.com
Date: Thursday, 26 May, 2011, 3:56 PM

Jr.No: 152/2011
Dr. Chirag H. Shah
E/3, Sthapatya Apartement,
Nr. Sterling Hospital,
Opp. Gurukul Road, Memnagar,
Ahmedabad 380 052.
Gujarat

Re.: Original Article entitled "To Assess the Prevalence of Impaired Glucose Tolerance and Impaired Fasting Glucose in Western India Population"

Dear Dr. Shah,

Your above article has been received for consideration for publication in JAPI. The article is being sent to the board of referees for scrutiny. We shall let you know about its suitability as soon as we receive the comments from the board referees.

With warm personal regards,
Yours sincerely,
Dr. Siddharth N. Shah
Hon.Editor, JAPI

Appendix VI: Media Coverage

પ્રિડાયાબીટીસ (ડાયાબીટીસ થતા પહેલાનું સ્ટેજ) તપાસ કેમ્પ

વર્ષ ૨૦૦૩ ની માહિતીના આધારે ભારતમાં ૩.૬ કરોડ લોકો ડાયાબીટીસથી પીડાતા હતા, જે વર્ષ ૨૦૨૫ સુધીમાં ૭.૫ કરોડ થવાની શકયતા છે. (Ref : WHO)

જો તમે,

- ૨૦ વર્ષ કે તેથી વધુ ઉંમરના સ્ત્રી કે પુરૂષ હો.....
- તમને ડાયાબીટીસ **ના** હોય.....
- તમારા નજીકના સગામાં કોઇને ડાયાબીટીસ છે કે નહી,તેની ખબર ન હોય.....

તો તેના નિદાન માટેના એક મેડીકલ કેમ્પનું આયોજન કરેલ છે. તો નીચે જણાવેલ સ્થળે અને સમયે ભૂખ્યા પેટે આવવા વિનંતી.

સ્થળ :

તારીખ : સમય :

કેમ્પ દરમિયાન નીચે મુજબની લોહીની તપાસ કરવામાં આવશે :

૧. ભૂખ્યા પેટે લોહીમાં ગ્લુકોઝની તપાસ ૨. ૭૫ ગ્રામ ગ્લુકોઝ લીધા પછી ના બે કલાક પછી લોહી ની તપાસ ૩. લોહીમાં ચરબીની તપાસ

કેમ્પ દરમિયાન ધ્યાનમાં રાખવાની અગત્યની બાબતો :

૧. સવારે ભૂખ્યા પેટે આવવું (ફકત પાણી લઇ શકાય)
૨. ગ્લુકોઝ લીધા પછી ના બે કલાક પછી પણ લોહી તપાસ કરવાની હોય બરાબર બે કલાક પછી ફરીથી લોહી આપવા વિનંતી (તે બે કલાક દરમિયાન ફકત પાણી જ લેવું.)

નામ નોંધાવવાના સ્થળ:

(૧) (૨)

નોંધ: મર્યાદિત સભ્યોનું જ રજીસ્ટ્રેશન કરવાનું હોઇ, વહેલી તકે રજીસ્ટ્રેશન કરાવી સમયસર સ્થળે પહોંચીને લાભ લેવા વિનંતી.

1. Advertisement used for registration of Subjects:

2. Sandesh: Leading Gujarati daily, Sabarkantha Edition,30 May 2007.

SANDESH : SABARKANTHA | WEDNESDAY, 30 MAY 2007

બાયડમાં યોજાયેલો પ્રિ-ડાયાબિટીસ કેમ્પ

(સં.ન્યુ.સ.)

બાયડ, તા.૨૯

સમગ્ર વિશ્વમાં કેન્સરની માફક વિસ્તરી રહેલા ડાયાબિટીસના દર્દને પ્રારંભીક તબક્કે જ શોધી કાઢવા માટે બાયડના પૂર્વ ધારાસભ્ય ડૉ. મહેન્દ્રભાઈ પટેલના સ્વર્ગીય પુત્ર આદિત્યના સ્મરણાર્થે આજે બાયડ ખાતે યોજાયેલ પ્રિ-ડાયાબિટીસ કેમ્પમાં ૨૦૯ દર્દીઓએ લાભ લીધો હતો. જ્યારે લાયન્સ-લીઓ કલબ-બાયડ અને સન પેથોલોજી લેબોરેટરીએ કેમ્પનું સફળ સંચાલન કર્યું હતું.

આ અંગે મળતી માહિતી અનુસાર બાયડ ખાતે અનેક યુવાનો અને મોટેરાઓ ડાયાબિટીસના પ્રાથમિક લક્ષણોથી પીડાતા હોવાની વિગતો તબીબી અવલોકનમાં બહાર આવ્યા બાદ ડાયાબિટીસના

૨૦૦ ઉપરાંત દર્દીઓએ લાભ લીધો : ડાયાબિટીસના અન્ય દર્દીઓએ પણ મોટી સંખ્યામાં લીધેલો લાભ

ચેક-અપ કેમ્પમાં પ્રજાને માતબર ખર્ચમાંથી મુક્તિ અપાવવા આજે સ્વ. આદિત્ય પટેલ અને લાયન્સ-લીઓ કલબ-બાયડના સંયુક્ત ઉપક્રમે સંજીવની હોસ્પિટલ ખાતે પૂર્વ ધારાસભ્ય ડૉ. મહેન્દ્રભાઈ પટેલ, પૂર્વ રીજીયન ચેરમેન ડૉ. પ્રકાશભાઈ શાહ, લીઓ ચેરમેન ભાવેશ પટેલ, કાન્તિ રાઠી, લાયન્સ પ્રમુખ રમેશભાઈ પટેલ, મંત્રી ગીરીશભાઈ પટેલ, લીઓ પ્રમુખ ધર્મેન્દ્ર બ્રહ્મભટ્ટની ઉપસ્થિતિમાં આજે તા.૨૭ના રોજ સવારના ૯-૦૦ કલાકથી કેમ્પ શરૂ થતાં જ બાયડનગર ઉપરાંત દૂર દૂરથી અનેક લોકો ચેક-અપ માટે ઉમટી પડતાં સંજીવની હોસ્પિટલ આગળ મોટી કતાર ખડકાઈ હતી અને બપોરના ૧૨-૦૦ કલાક સુધીમાં ૨૦૯ દર્દીઓએ પ્રિ-ડાયાબિટીસ કેમ્પનો લાભ લઈ પોતાના શરીરમાંની સુગરના રીપોર્ટ મેળવ્યા હતા. જ્યારે સન લેબોરેટરી-અમદાવાદના ચિરાગ શાહ, ડૉ. નિમિષા શાહ, નિહાર શાહે તમામ દર્દીઓના લોહીના નમૂના મેળવ્યા હતા. સમગ્ર પ્રિ-ડાયાબિટીસ કેમ્પને સફળ બનાવવા લાયન્સ કલબ-બાયડના ભૂતપૂર્વ પ્રમુખ એમ.બી. પટેલ, લીઓ શૈલેષ ભાટીયા, અર્પણ શાહ સહિત લાયન્સ-લીઓ પરિવારે ભારે જહેમત ઉઠાવી હતી.

3. Daily News and Analysis (DNA) Leading English Daily, Ahmedabad edition, on the eve of World Diabetes Day 14 Nov 2011.

AHMEDABAD
MONDAY, NOVEMBER 14, 2011
DNA
DAILY NEWS AND ANALYSIS

● World Diabetes Day Today

Pre-diabetes widespread in state, pateints unaware

According to a study, 14-22% of the population across India falls in the pre-diabetic category

Kinjal Desai

Too much of a good thing could be dangerous, particularly for diabetics. But what is causing concern to doctors and diabetologists is the pre-diabetic condition found in youngsters above 20 years. This could lead to future health complications and may be fatal too.

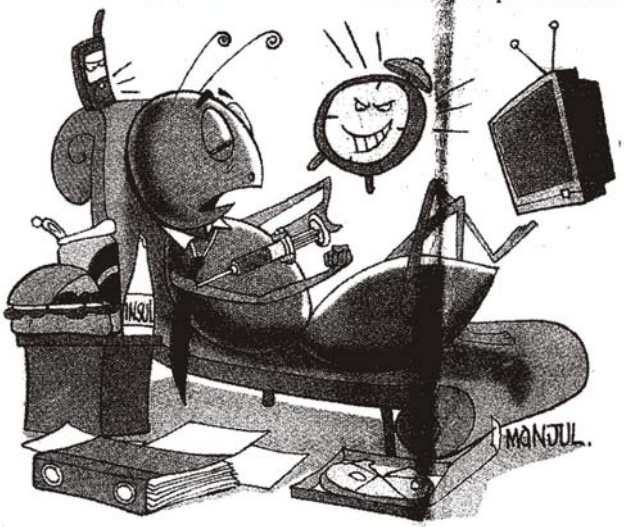
Dr Banshi Saboo is the key investigator for Gujarat for ICMR's 'India-D' study and president, Diabetes Care India. He said, "According to our study, so far nearly 14-22% of the population across India fall in the pre-diabetic condition. These people could get diabetes within a decade."

Dr Saboo explained that there are three known reasons for acquiring diabetes - family history, abdominal obesity and sedentary lifestyle. However, it can be prevented with timely tests and proper care.

According to an eight-month study conducted by Dr Chirag Shah and others in 12 camps, youth with a history of diabetes in the family were getting more prone to pre-diabetic incidence.

Also, people in the age group of 41-50 years are highly prone to develop diabetes.

Dr Chirag Shah along with Dr Nimisha Shah have formulated a chart for diabetic patients to both reduce their weight and also have a healthy life. According to the International Diabetes Federation data of 2009, 51 million people in India were found suffering from diabetes. But as per the 'India-D' study of ICMR, which is currently going on across 25 states and 8 major cities including Ahmedabad, 65 million people are diabetic.



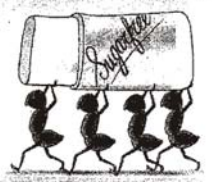
Dr Saboo's study done in a small village, Bareja, near Ahmedabad, revealed that one-third of the population above 55 years was found to be diabetic and of this population, 50% were not aware about their condition.

Agreeing that the pre-diabetic condition was more prominent in the Indian population than diabetes, Dr Mayur Patel, project mentor for 'Stop Diabetes programme - a movement for diabetes free India' said, "Pre-diabetic incidence is more common today, nearly 140% more than patients found suffering from diabetes. The percentage of Impaired Glucose Tolerance (IGT) found in people of Gujarat is very high". Owing to this, the conversion ratio from pre-diabetic to diabetic is very high.

Dr Patel continued, "The data till September 2011 reveals that of the 9,342 patients undergoing sugar testing, 1,849 were found to have diabetes and on further screening they were found to be suffering from complications."

Furthermore, 45.82% of urban population, 41.25% of the urban slum population and 25.49% of the rural population were found to be diabetic. Yet, 50% of this population was not aware about their diabetic condition, revealed Dr Patel.

EVENTS ON DIABETES DAY



On the occasion of World Diabetes Day on Monday, 'Life for child' programme supported by International Diabetes Federation (IDF) and International Society for Pediatric and Adolescent Diabetes (ISAPD) will be launched by Dia Care. Under the programme, children with type 1 diabetes, will be provided with glucometer strips, insulin and other diabetes products at subsidised rates.

Screening camps for diabetes will be held at prominent city club, Prahaladnagar Garden, Parimal Garden, Law Garden and also at IOC petrol pumps. There will also be free diabetes treatment camps at 56 family clinics. An awareness seminar will also be held at GLS auditorium by Endocrinologist Dr Ramesh Goyal.

ભારત ડાયાબિટીસની રાજધાની !

હરખાવા જેવું નથી, ભવિષ્ય કમકમાટી ઉપજાવે તેવું છે, ચિરાગ શાહ દ્વારા પ્રિ-ડાયાબિટીક સ્થિતિ અંગે સંશોધન

રાજકોટ, તા. ૧૩: સૌરાષ્ટ્ર યુનિવર્સિટીના ફાર્મસી ભવનમાં ભવનના વડા પ્રો. નવીન શેઠના માર્ગદર્શન નીચે અમદાવાદના ચિરાગ શાહ ડાયાબિટીસમાં ખાસ કરીને પ્રી-ડાયાબિટીક પરિસ્થિતિ અંગે છેલ્લા ત્રણ વર્ષથી સંશોધન કરે છે. તેઓના સંશોધન પ્રોજેકટને ગુજરાત સરકારના સાયન્સ અને ટેકનોલોજી વિભાગ દ્વારા સંચાલિત ગુજકોસ્ટ દ્વારા રૂ. ૨,૫૫,૦૦૦/- (બે લાખ પંચાવન હજાર)નું અનુદાન પણ મળ્યું છે. ગુજરાતમાં આ પ્રકારની સૌ પ્રથમ વખત સ્ટડી ચિરાગ શાહ કરી રહ્યા છે. 'તા. ૧૪ નવેમ્બર વિશ્વભરમાં 'વર્લ્ડ ડાયાબિટીસ ડે' તરીકે ઉજવાય છે. તેના સંદર્ભમાં ડાયાબિટીસ અંગે સંશોધનની વિગતો આપતા તેમણે જણાવ્યું હતું કે, દુનિયામાં ભારતને 'ડાયાબિટીસની રાજધાની' ગણવામાં આવે છે.

ચિરાગ શાહ

ડાયાબિટીસ એ એક બહુ જ ખતરનાક રોગ છે, જે શરીરના દરેક અવયવને અસર કરે છે, એમ

આજે વર્લ્ડ ડાયાબિટીસ-ડે

જણાવતા તેમણે કહ્યું કે, આજના દિવસોમાં ડાયાબિટીસ સર્વવ્યાપી તબીબ સમસ્યા છે. ડાયાબિટીસના દર્દીઓની સંખ્યા દિવસે ને દિવસે કૂદકે ને ભૂસકે વધતી જાય છે. સન ૧૯૯૫માં વિશ્વમાં ડાયાબિટીસના અંદાજીત દર્દીઓ ૧૩.૫ કરોડ હતા, જે વધીને સને ૨૦૨૫માં અંદાજિત ૩૦ કરોડ થવાની શકયતા છે.

વિશ્વ આરોગ્ય સંસ્થા (WHO) ના પ્રમાણે ભારતમાં સન-૨૦૦૩માં આશરે ૩.૬ કરોડ લોકો ડાયાબિટીસથી પીડાતા હતા. જે સન-૨૦૨૫માં આશરે ૭.૫ કરોડ થશે એટલે જ ભારતને દુનિયાની 'ડાયાબિટીસ રાજધાની' ગણવામાં આવે છે. ભવિષ્ય બહુ જ કમકમાટી ઉપજાવે તેવું લાગે છે. કારણ કે અત્યારે ભારત આંધળી ઝડપથી આધુનિકરણ અને શહેરીકરણ તરફ સૂસવાટાભેર આગળ વધી રહ્યું છે.

આપણી સોસાયટીને વેસ્ટોક્સીફીલેશન, કોકાકોલાનાઈઝેશન, પેપ્સીફીકેશન કે મેકડૉનાલ્ડાઈઝેશન કહેતા આપણે ધિક્કારની લાગણી અનુભવીએ છીએ.

ડાયાબિટીસના લોકોને સામાન્ય માણસ કરતા કિડનીના રોગો થવાની શકયતા ૨૫ ગણી, અંગછેદનની શકયતા ૩૦-૪૦ ગણી, હૃદયરોગના હુમલાની શકયતા ૨-૪ ગણી અને પક્ષઘાતની (લકવો) શકયતા બે ગણાથી વધારે છે. આખા વિશ્વમાં ડાયાબિટીસ, **(જુઓ પાંનું ૧૦)**

ડાયાબિટીસ વિશે ખોટી માન્યતા અને સાચી હકીકત

ખોટી માન્યતા-૧

ડાયાબિટીસ અંગે સૌથી મોટી ખોટી માન્યતા એ છે કે તે પૈસાદાર અને સમૃદ્ધ લોકોનો રોગ છે.

સાચી હકીકત

આ સાવ જ ખોટી વાત છે કારણ કે રોગ પૈસાદાર અને ગરીબ બને વર્ગના લોકોને સરખી રીતે અસર કરે છે.

ખોટી માન્યતા-૨

એવી માન્યતા છે કે તે એક ચેપી રોગ છે.

સાચી હકીકત

આ માન્યતા ખોટી છે-કારણ કે રોગ એ કોઈ જંતુ દ્વારા ફેલાતો નથી પણ એ જનનિક (આનુવંશિક) રીતે પેઢી દર પેઢી દ્વારા આગળ પ્રસરે છે.

ખોટી માન્યતા-૩

એવી માન્યતા છે કે ડાયાબિટીસ એકવાર કંટ્રોલમાં આવી ગયું હોય પછી ખોરાક અને દવાઓમાંથી મુક્તિ થઈ શકે.

સાચી હકીકત

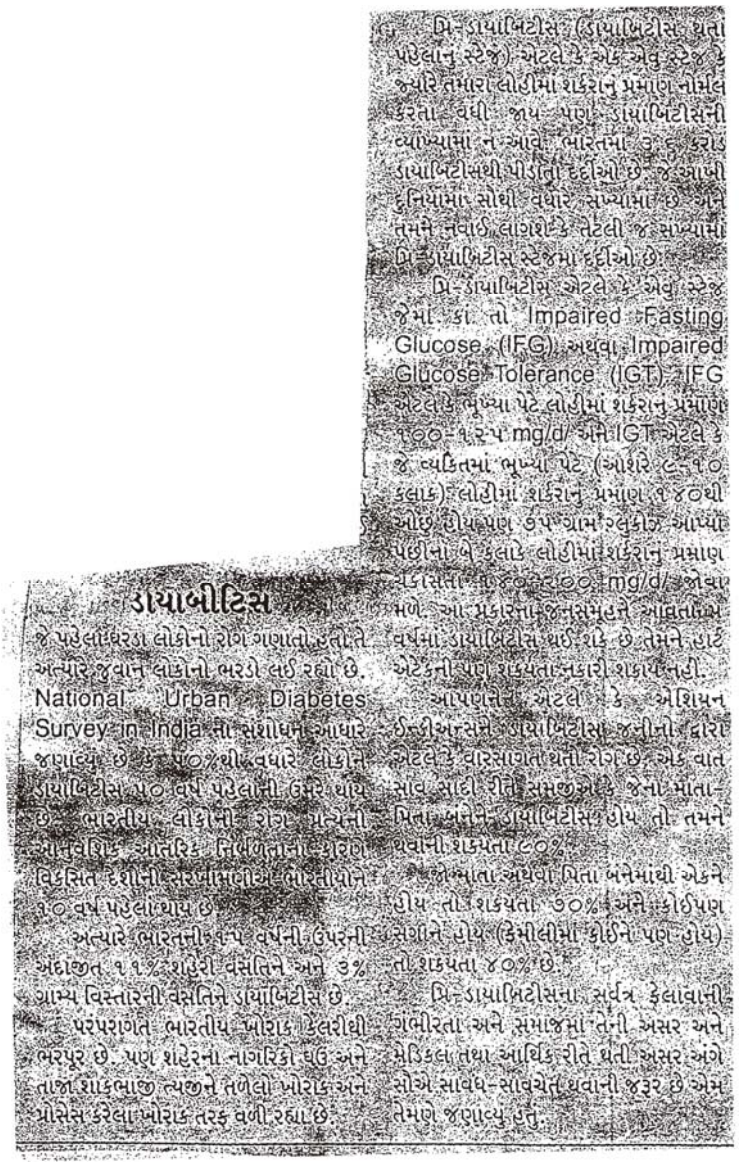
ડાયાબિટીસના મેનેજમેન્ટ માટે ખોરાક અને દવાઓ એ મહત્ત્વશીલ બાબત છે.

ખોટી માન્યતા-૪

એક વાર ઈન્સ્યુલીન ચાલુ કર્યા પછી દર્દી માટે તે આજીવન બની જાય છે.

સાચી હકીકત

તે ડાયાબિટીસ માટે તદ્દન રીતે સાચું નથી.



ડાયાબીટિસ

જે પહેલાં ૫૨૨ા લોકોનો રોગ ગણાતો હતો તે અત્યારે જુવાન લોકોનો ભરડો લઈ રહ્યો છે. National Urban Diabetes Survey in India ના સંશોધન આધાર જણાવ્યું છે કે ૫૦%થી વધારે લોકોને ડાયાબિટિસ ૫૦ વર્ષ પહેલાની ઉંમરે થાય છે. ભારતીય લોકોનો રોગ પ્રત્યેનો આનુવંશિક આંતરિક નિર્બળતાના કારણે વિકસિત દેશોની સરખામણીએ ભારતીયોને ૧૦ વર્ષ પહેલાં થાય છે.

અત્યારે ભારતનો ૧૫ વર્ષની ઉપરની અંદાજીત ૧૧% શહેરી વસતિને અને ૩% ગ્રામ્ય વિસ્તારની વસતિને ડાયાબિટિસ છે.

પરંપરાગત ભારતીય ખોરાક કેલરીથી ભરપૂર છે. પણ શહેરના નાગરિકો ઘઉ અને તાજા શાકભાજી ત્યજીને તળેલા ખોરાક અને પ્રોસેસ કરેલા ખોરાક તરફ વળી રહ્યા છે.

પ્રિ-ડાયાબિટીસ (ડાયાબ્રિટીસ થતાં પહેલાનું સ્ટેજ) એટલે કે એક એવું સ્ટેજ કે જ્યારે તમારા લોહીમાં શર્કરાનું પ્રમાણ નોર્મલ કરતા વધી જાય પણ ડાયાબિટીસની વ્યાખ્યામાં ન આવે. ભારતમાં ૬ કરોડ ડાયાબિટીસથી પીડાતા દર્દીઓ છે. જે આખી દુનિયામાં સૌથી વધારે સંખ્યામાં છે. અને તમને નવાઈ લાગશે કે તેટલી જ સંખ્યામાં પ્રિ-ડાયાબિટીસ સ્ટેજમાં દર્દીઓ છે.

પ્રિ-ડાયાબિટીસ એટલે કે એવું સ્ટેજ જેમાં કા. તો Impaired Fasting Glucose (IFG) અથવા Impaired Glucose Tolerance (IGT). IFG એટલે કે ભૂખ્યા પેટે લોહીમાં શર્કરાનું પ્રમાણ ૧૦૦-૧૨૫ mg/d/ અને IGT એટલે કે જે વ્યકિતમાં ભૂખ્યા પેટ (ઓશરે ૮-૧૦ કલાક) લોહીમાં શર્કરાનું પ્રમાણ ૧૪૦થી ઓછું હોય પણ ૭૫ ગ્રામ ગ્લુકોઝ આપ્યા પછીના બે કલાકે લોહીમાં શર્કરાનું પ્રમાણ ૧૪૦-૨૦૦ mg/d/ જોવા મળે. આ પ્રકારના જનસમૂહને આવતાં પાંચ વર્ષમાં ડાયાબિટિસ થઈ શકે છે. તમને હાર્ટ એટેકનો પણ શકયતા જણાવી શકાય નહીં.

આપણને એટલે કે અશિમન ઈન્ડીઆનને ડાયાબિટીસ જનીનો દ્વારા એટલે કે વારસાગત થતો રોગ છે. એક વાત સાવ સાદી રીતે સમજીએ કે જેના માતા-પિતા બંનેને ડાયાબિટીસ હોય તો તમને થવાની શકયતા ૮૦%.

જો માતા અથવા પિતા બનેમાંથી એકને હોય તો શકયતા ૩૦% અને કોઈપણ સગીને હોય (ફેમીલીમાં કોઈને પણ હોય) તો શકયતા ૪૦% છે.

પ્રિ-ડાયાબિટીસના સર્વત્ર ફેલાવાની ગંભીરતા અને સમાજમાં તેની અસર અને મેડિકલ તથા આર્થિક રીતે થતી અસર અંગે સૌએ સાવધ-સાવચેત થવાની જરૂર છે એમ તેમણે જણાવ્યું હતું.

અમેરિકામાં ડાયાબિટીસ અને આવક વચ્ચે ઊલટી રીતે સામ્યતા છે. ચરબીવાળો ખોરાક બહુ જ સસ્તો હોવાથી ગરીબ લોકો શ્રીમંત કરતા બહુ જ મેદસ્વી બને છે અને એ લોકો શારીરિક કસરત ઓછી કરે છે અને નીચલા સ્તરની સારવાર મળે છે. જ્યારે ભારતમાં અમેરિકા કરતા એકદમ ઊલટું છે. આ કેવો વિચિત્ર વિરોધાભાસ !!!

ભારતમાં ડાયાબિટીસની સારવાર માટેનો સરેરાશ ખર્ચ અંદાજે રૂ. ૨૦,૦૦૦/- પ્રતિ વર્ષ છે અને જે દિવસે દિવસે વધતો જાય છે. ડાયાબિટીસનો રોગ ભારતીયોના બેક બેલેન્સને ધીમે ધીમે ચાઉ કરી જાય છે. મધ્યમવર્ગીય ભારતીય અંદાજે તેમની ચોથા ભાગની કમાણી ડાયાબિટીસની દવાઓ અને તેની સારવાર પાછળ ખર્ચી કાઢે છે.

ડાયાબિટીસ પર સંશોધન કરતા વૈજ્ઞાનિકોએ સાબિત કર્યું છે કે ગામડામાં રહેતા ત્રણ ચતુર્થાંશ (૩/૪) લોકોને ખબર જ નથી હોતી કે તમને ડાયાબિટીસ થયું છે.

ડાયાબીટીસની આડઅસરો

ડાયાબીટીસના દર્દીઓમાં દરેકને એકસરખા ચિન્હો જોવા મળતા નથી. પણ અલગ અલગ જોવા મળે છે. પણ મહત્ત્વની વાત એ છે કે જેની આપણે અવગણના કરતા હોઈએ છીએ કે ડાયાબિટીસથી પીડાતા ૫૦% દર્દીઓને કોઈપણ ચિન્હ જોવા મળતા નથી એટલે કે asymptomatic હોય છે.

ડાયાબીટીસ એક એવો રોગ છે જે શરીરના બધા જ અગત્યના અવયવો પર અસર કરે છે અને તેની કાર્યક્ષમતા બિલકુલ ઘટાડીને નકામા બનાવી દે છે. WHO એ ભારતને ડાયાબિટીસની રાજધાની તરીકે જાહેર કરેલ છે. જેમાં આપણે બધા ગુજરાતી ભાઈ-બહેનોનો તો તેની સાથે ઘનિષ્ઠ સંબંધ છે. કારણ કે આપણા દરેક ખોરાકમાં ખાંડ અથવા ગોળ હોય જ છે.

અંદાજે ૧૦-૧૫ વર્ષ પછી બધા જ અવયવોને નુકસાન થાય છે.

5. Akila, Rajkot edition on the eve of World Diabetes Day 14 Nov 2011.

સવારે ચા, સાંજે...અકિલા | ૧૬ | સોમવાર તા.૧૪-૧૧-૨૦૧૧

આજે વર્લ્ડ ડાયાબીટીસ ડે

વૃધ્ધો જ નહી યુવાઓ પણ ડાયાબીટીસથી સાવધ રહે

રેસીયો બદલાયો છે, પહેલા ૫૦ વર્ષ બાદ લાગુ થતો આ રોગ હવે ૫૦ વર્ષ પૂર્વે જ થવા લાગ્યો છે: આનુવંશિક આંતરીક નિર્બળતા જવાબદાર: ખોટી માન્યતામાંથી બહાર આવી જીવનશૈલીમાં બદલાવ લાવવો અતિ જરૂરી: વર્લ્ડ ડાયાબીટીસ દિને રસપ્રદ માહિતી વર્ણવતા ચિરાગ શાહ અને ડો. નવીનશેઠ

ડાયાબીટીસએ એક બહુ ખતરનાક રોગ છે. જે શરીરના દરેક અવયવને અસર કરે છે. આજના દિવસોમાં ડાયાબીટીસ પણ સર્વવ્યાપી તબીબી સમસ્યા છે. ડાયાબીટીસના દર્દીઓની સંખ્યા દિવસે ને દિવસે કુદકે ભૂસકે વધતી જાય છે. સન-૧૯૯૫માં ડાયાબીટીસના અંદાજીત દર્દીઓ ૧૩.૫ કરોડ હતા. જે વધીને સને-૨૦૨૫ માં અંદાજીત ૩૦ કરોડ થવાની શકયતા છે. તેમ આજે વિશ્વ ડાયાબીટીસ દિને "આકિલા" સાથેની મુલાકાતમાં આ વિષયના સંશોધક ચિરાગ શાહ અને પ્રો.નવીન શેઠે જણાવ્યું હતું.

વિશ્વ આરોગ્ય સંસ્થા (WHO) ના પ્રમાણે ભારતમાં સન-૨૦૦૩માં આશરે ૩.૬ કરોડ લોકો ડાયાબીટીસથી પીડાતા હતા.

જ્યારતને દુનિયાની 'ડાયાબીટીસ રાજધાની' ગણવામાં આવે છે.

ડાયાબીટીસના લોકોને સામાસન્ય માણસ કરતા કિડનીના રોગો થવાની શકયતા ૨૫ ગણી, અંગછેદનની શકયતા ૩૦-૪૦ ગણી, હદયરોગના હુમલાની શકયતા ૨-૪ ગણી અને પક્ષાઘાતની (લકવા શકયતા બે ગણાથી વધારે છે.

ચિરાગભાઈએ જણાવે કે આખા વિશ્વમાં ડાયાબીટીસ જે પહેલા ધરઠા લોકોનો રોગ ગણાતો હતો તે અત્યારે જુવાન લોકોને ભરડો લઈ રહ્યો છે. National Urbad Diabetes Survey In indin ના સંશોધન આધારે જણાવ્યું છે કે ૫૦% થી વધારે લોકોને ડાયાબીટીસ ૫૦ વર્ષ પહેલાની ઉંમરે થાય છે. ભારતીય લોકોની રોગ પ્રત્યેની આનુવંશિક આંતરિક નિબળતાના કારણે વિકસિત દેશોની સરખામણીએ ભારતીયોને ૧૦ વર્ષ પહેલા થાય છે. અત્યારે ભારતની ૧૫ વર્ષ ઉપરની અંદાજીત ૧૨% શહેરી વસ્તીને અને ૩% ગ્રામ્ય વિસ્તારની વસ્તીને ડાયાબીટીસ છે.

ડાયાબીટીસ પર સંશોધન કરતા વૈજ્ઞાનિકોએ સાબિત કર્યું છે કે ગામડામાં રહેતા ત્રણ ચર્તુથાંશ (3/4) લોકોને ખબર જ નથી હોતી કે તેમને ડાયાબીટીસ થયું છે.

ડાયાબીટીસ વિશે ખોટી માન્યતા અને સાચી હકીકતો પ્રવર્તી રહી છે. જેમ કે ડાયાબીટીશ અંગે સૌથી મોટી ખોટી માન્યતા એ છે કે ને પૈસાદાર અને સમૃધ્ધ લોકોનો રોગ છે. સાચી હકીકત એ છે કે રોગ પૈસાદાર અને ગરીબ બંને વર્ગના લોકોને સરખી રીતે અસર કરે છે.

એક એવી માન્યતા છે કે તે એક ચેપી રોગ છે. પરંતુ આ માન્યતા ખોટી છે કારણ કે રોગ એ કોઈ જંતુ દ્વારા ફેલાતો નથી પણ એ જનીનક (આનુવંશિક) રીતે પેઢી દર પેઢી દ્વારા આગળ પ્રસરે છે. એજ રીતે એવી માન્યતા છે કે ડાયાબીટીસ એકવાર કંટ્રોલમાં આવી ગયું હોય પછી ખોરાક અને દવાઓમાંથી મુકિત થઈ શકે. પરંતુ સાચી હકીકત મુજબ ડાયાબીટીસના મેનેજમેન્ટ માટે ખોરાક અને દવાઓ મહત્વશીલ બાબત છે.

એક વાર ઈન્સ્યુલીન આપ્યુ કર્યા પછી કદી માટે તે આજીવન બની જાય છે તે પણ ખોટી માન્યતા છે. તે ડાયાબીટીસ માટે તદ્દન રીતે સાચુ નથી.

ડાયાબીટીસની આડઅસરો

ડાયાબીટીસના દર્દીઓમાં દરેકને એકસરખા ચિન્હો જોવા મળતા નથી. પણ અલગ અલગ જોવા મળે છે. પણ મહત્વની વાત એ છે કે જેની આપણે અવગણના કરતા હોઈએ છીએ કે ડાયાબીટીસથી પીડાતા ૫૦% દર્દીઓને કોઈપણ ચિન્હ જોવા મળતા નથી એટલે કે asymptomatic હોય છે.

ડાયાબીટીસ એક એવો રોગ છે જે શરીરના બધા જ અગત્યના અવયવો પર અસર કરે છે અને તેની કાર્યક્ષમતા બિલકુલ ઘટાડીને નકામા બનાવી દે છે. WHO એ ભારતને ડાયાબીટીસની રાજધાની તરીકે જાહેર કરેલ છે. જેમાં આપણે બધા ગુજરાતી ભાઈ-બહેનોને તો તેની સાથે વધિષ્ઠ સંબંધ છે. કારણ કે આપણા દરેક ખોરાકમાં ખાંડ અથવા ગોળ હોય છે.

અંદાજે ૧૦-૧૫ વર્ષ પછી બધા જ અવયવોને નુકસાન થાય છે. જેમ કે પગની સાંધાને મૂત્ર માર્ગને પાચન તંત્ર શરીરની નર્સોઓમાં કરે છે.

પ્રિ ડાયાલીટીસ

પ્રિ.ડાયાબીટીસ (ડાયાબીટીસ થતાં પહેલાનું સ્ટેજ) એટલે કે એક એવું સ્ટેજ કે જ્યારે તમારા લોહીમાં શર્કરાનું પ્રમાણ નોર્મલ કરતા વધી જાય પણ ડાયાબીટીસની વ્યાખ્યામાં ન આવે. ભારતમાં ૩.૬ કરોડ ડાયાબીટીસથી પીડાતા દર્દીઓ છે. જે આખી દુનિયામાં સૌથી વધારે સંખ્યામાં છે અને તમને નવાઈ લાગશે કે તેટલી જ સંખ્યામાં પ્રી-ડાયાબીટીસ સ્ટેજમાં દર્દીઓ છે.

પ્રી-ડાયબીટીસ એટલે કે એવું સ્ટેજ જેમાં કાં તો Impaire Fasting Glucose (IFG) અથવા Impaired Glucose Tolerance (IGT). IFG એટલે કે ભૂખ્યા પેટે લોહીમાં શર્કરાનું પ્રમાણ ૧૦૦-૧૨૫ mg/d અને IGT એટલે કે જે વ્યકિતમાં ભુખ્યા પેટે (આશરે ૯.૧૦ કલાક) લોહીમાં શર્કરાનું પ્રમાણ ૧૪૦ થી ઓછું હોય પણ ૭૫ ગ્રામ ગ્લુકોઝ આપ્યા પછીના બે કલાકે લોહીમાં શર્કરાનું પ્રમાણ ચઢાસતા ૧૪૦-૨૦૦ mg/dl જોવા મળે.

આ પ્રકારના જનસમુહને આવતા પ-વર્ષમાં ડાયાબીટીસ થઈ શકે છે અને તેમને હાર્ટએટેકની પણ શકયતા નકારી સકાય નહીં.

આપણને એટલે કે Alsan Indians ને ડાયાબીટીસ જનીનો દ્વારા એટલે કે વારસાગત થતો રોગ છે એક વાત સાવ સાદી રીતે સમજીએ કે જેના માતા-પિતા બન્નેને ડાયબીટીસ હોય તો તેમને થવાની શકયતા ૯૦%

વર્લ્ડ ડાયાબીટીસ ડે અંતર્ગત કેટલીક લોકોપયોગી માહિતી 'અકિલા' કાર્યાલયે વર્ણવતા ચિરાગ શાહ અને ડો. નવિન શેઠ તસ્વીરમાં નજરે પડે છે. (તસ્વીરઃ સંદીપ બગથરીયા) (૬.૨)

પણ હોય) તો શકયતા ૪૦% છે.

સંશોધનના ધ્યેયને પૂર્ણ કરવા ગુજરાતના જુદા જુદા શહેરી અને ગ્રામ્ય વિસ્તારમાં ૧૨ પ્રી-ડાયાબીટીસ કેમ્પ કરવામાં આવતા આ પ્રકારનું સંશોધન ગુજરાતમાંથી સૌ પ્રથમ વાર થયું અને જે વિખ્યાત મેડિકલ જર્નલમાં પ્રકાશિત પણ થયું છે. આ કેમ્પમાં આ ટીમ દ્વારા ૧૩૦૦ લોકોના વિશેની માહિતી એકત્રિત કરવામાં આવી હતી. જેમાં મળેલા મહત્વના પરિણામો મુજબ છે. (૧) ifgનો ફેલાવો (prev-ß.7ß,, IGT નો ફેલાવો ૪.૬૦% નાની ઉંમરે પ્રી-ડાયાબીટીસ અથવા ડાયાબીટીસ થાય તો માનસિક શારીરિકની સાથે આપણને આર્થિક રીતે પણ પાયમાલ કરી દે છે. તેની સારવાર દવાઓ તથા તપાસના ખર્ચાઓ દિવસે દિવસે વધતા જાય છે. આથી જનતાને એક સામાન્ય મંત્ર ગ્રહણ કરવો જરૂરી છે.

ઓછું ખાવું સમયસર ખાવું ચાલવુ, સમયસર સુઈ જવુ અને હસતા રહેવું.

ડાયાબીટીસના દર્દીઓ માટે ખોરાક અંગે સલાહ આપતા ચિરાગભાઈએ જણાવેલ કે તેને ત્રણ ભાગમાં વહેંચી શકાય પુષ્કળ પ્રમાણમાં લઈ શકાય તેવો ખોરાક, મર્યાદિત પ્રમાણમાં લઈ શકાય તેવો ખોરાક., કયારેય ન લઈ શકાય તેવો ખોરાક

(૧) પુષ્કળ પ્રમાણમાં લઈ શકાય તેવો ખોરાક

- કાચા શાકાભાજીને સલાડના રૂપમાં રાયતુ બનાવી લઈ શકાય.
- સ્કીમ મિલ્ક (મલાઈ વગરનું દુધ) માંથી બનાવેલ ચા, કોફી (ખાંડ વગરની), પાતળી છાશ, ખાંડ વગરનું લીંબુનુ પાણી અને શાકભાજીમાંથી બનાવેલ પાળતો સુપ લઈ શકાય.

(૨) મર્યાદિત પ્રમાણમાં લઈ શકાય તેવો ખોરાક

- ઘઉં, બાજરી, જુવાર, મકાઈ, રાગી (નાચણી), ચોખા વગેરે ધાન્ય પદાર્થમાંથી બનાવેલ વસ્તુ.
- બધા કઠોળ તથા દાળ.
- ફળ (નીચે જણાવેલ માત્રા મુજબ)
- મલાઈ વગરના દુધમાંથી બનાવેલ દહી, પનીર,

(૩) કયારે ન લઈ શકાય તેવો ખોરાક

- direct ખાંડ, ગોળ, મધ અને બજારમાં મળતી વસ્તુ જેવી કે જામ, જેલી, આઈસ્ક્રીમ, કેન્ડી, કેક, પેસ્ટ્રી અને મીઠાઈ.
- મેંદો અને તેમાંથી બનાવેલ વસ્તુ તથા બેકરીની આઈટમ જેવી કે બ્રેડ, ચિકકીટ ટોસ્ટ, ખારી અને સમોસા,
- કોઈપણ જાતના પીણા.
- સુકો મેવો જેવા કે બદામ, કાજુ, ખજુર, દ્રાક્ષ, ટોપરૂ, સીંગ.
- આથો આવેલ વસ્તુ અને સોડા હોય તેવી કોઈપણ વસ્તુ દા.ત. પાપડ, અથાણા.
- ક્રિમ બિસ્કીટ, નુડલ્સ, મેગી.

નોંધ

૧. રોટલીના લોટને મેંદો જેવો ન વાપરવો એટલે કે ચાળવો નહી.
૨. પુષ્કળપાન ન કરવું કારણ કે તે હૃદય, ફેફસા અને આંખને નુકસાન કરે છે.
૩. એક સાથે વધારે ખોરાક ન લેવો અને બે ખોરાકની વચ્ચે લાંબો ગાળો ન રાખવો. ડાયાબીટીસના દર્દીએ દર બે કલાકે મર્યાદિત પ્રમાણમાં ખોરાક લેવો જોઈએ.
૪. ઉપવાસ ન રાખવો.
૫. ડાયાબીટીસ થઈ જાય તેના ચિન્હો જેવા કે પરસેવો વળવો, ધુંધળું દેખાવુ, થકકારા થવા, માથુ ચકવુ, નબળાઈ લાગવી, બેચેની લાગવી. ઉપરના બધા તથા કોઈપણ ચિન્હો લાગે કે તરત જ ૧ ચમચી ખાંડ અથવા ૧-ટુકડો ગોળ લેવો. (મીઠાઈ ન લેવી).
૬. બાકેલો કે શેકેલો ખોરાક લેવાનો આગ્રહ રાખવો. બને ત્યાં સુધી તળેલો ખોરાક ન લેવો.
૭. રોજ સવારે અડધો કલાક કે એક કલાક ઝડપથી (Brisk) ચાલવું.
૮. કોઈપણ ફળ (લિસ્ટમાં બતાવેલ) જમવાના ૨-કલાક પહેલા કે બે કલાક પછી લેવું.
૯. રાંધેલ ખોરાકમાં મીઠું, ખાંડ કે ગોળ ઉપરથી ન લેવા.
૧૦. તેલનો વપરાશ ઘટાડવા નોન-સ્ટીક વાસણોનો ઉપયોગ કરવો જોઈએ છે

ફળની માત્રા નકકી કરતું લિસ્ટ

૪-૫ આંબળા, ૧-સફરજન, ૫-૧૦ જાંબુ, ૧-મોસંબી, ૭-૧૦ દ્રાક્ષ, ૧-ઈલાયચી કેળા, ૧-ચીકુ, ૧-ટુકડો કેરી, ૧-ટુકડો પાઈનેપલ, ૨-ટુકડા સક્કર ટેટી, ૧૫૦ ગ્રામ રાસબેરી, ૧૫૦ ગ્રામ સ્ટ્રોબેરી, ૨-૩ ટુકડા પપૈયા, ૨-૩ ટુકડા જામફળ

શાકભાજી

બધા જ પાંદડાવાળા શાકભાજી, દૂધી, તુરીયા, ટીન્સા પુષ્કળ પ્રમાણમાં, બટાકા, શકકરીયા ન


Prediabetes Camp
Lunawada,Dist:Panchmahal
17/05/2007


WELCOMES
You
- CHIRAG SHAH
- PARAG SHAH
Prediabetes Camp
Sthapatya Apt,Ahmedabad
20/05/2007


Prediabetes Camp
Sthapatya Apt. Ahmedabad
20/05/2007


Prediabetes Camp
Bayad,Dist:Sabarkantha
27/05/2007


Prediabetes Camp
Bayad,Dist:Sabarkantha
27/05/2007
The International Association of Lions Clubs
લાયન્સ ક્લબ-બાયડ


Prediabetes Camp
Ubharan,Dist:Sabarkantha
03/06/2007


Prediabetes Camp
Dhansura,Dist:Sabarkantha
10/06/2007


Prediabetes Camp
Modasa, Dist:Sabarkantha
07/10/2007


Prediabetes Camp
Modasa-2,Dist:Sabarkantha
14/10/2007


Prediabetes Camp
Salal, Dist:Sabarkantha
17/11/2007


Prediabetes Camp
Bapunagar,Ahmedabad
09/12/2007
WEL-COMES YOU
Bapunagar Homoeopathic Dr. Friend Circle
(All B.H.M.S. & D.H.M.S.)
A Part of :- General Medical Practitioner's Assoctation
[Gujarat State]


Assurance Cell (IQAC)
a University, Rajkot.
comes You
Department of Phar
Prediabets Camp
Rajkot
19/03/2008


Internal Quality Assurance Cell (IQAC)
Saurashtra University, Rajkot.
Welcomes You
Department of Pharmaceutical Sciences
Saurashtra University
Rajkot-360005
Prediabetes Camp
Munjka, Dist:Rajkot
19/03/2008

ABSTRACT

INTRODUCTION

OBJECTIVE

REVIEW OF LITERATURE

COMMON MYTHS AND FACTS ABOUT DIABETES

PREDIABETES

DIABETES SELF MANAGEMENT EDUCATION

PHARMACOECONOMICS OF DIABETES

MATERIALS AND METHODS

RESULTS

ETHICAL REQUIREMENT

DISCUSSION

SUMMARY AND CONCLUSION

REFERENCES

APPENDICES