A Dissertation on

"THE CASE CONTROL STUDY OF RELATIONSHIP BETWEEN ARM LENGTH AND TYPE 2 DIABETES MELLITUS"

Submitted to
THE TAMILNADU DR. M.G.R. MEDICAL UNIVERSITY
CHENNAI – 600032.

In partial fulfilment of the Regulations
for the Award of the Degree of

M.D. BRANCH - I

GENERAL MEDICINE

DEPARTMENT OF GENERAL MEDICINE
GOVERNMENT KILPAUK MEDICAL COLLEGE AND HOSPITAL
CHENNAI – 600 010

APRIL 2017

BONAFIDE CERTIFICATE

This is to certify that the dissertation entitled **"RELATIONSHIP BETWEEN ARM LENGTH AND TYPE 2 DIABETES MELLITUS"** is a bonafide work done by **Dr.ELAYARAJA.P,** Post Graduate student in the Department of General Medicine, Kilpauk Medical College, Chennai-10, under our guidance and supervision in partial fulfilment of the rules and regulations of **The Tamilnadu Dr. M.G.R. Medical University** for the award of **M.D. Degree Branch I (General Medicine)** during the Academic period from **2014 to 2017.**

Prof. Dr. T.S.SANTHI, M.D,
Guide of the Study and
Professor of General Medicine,
Government Royapettah Hospital &
Kilpauk Medical College, Chennai -14.

Prof.Dr.E.SURESH , M.D.,D.DIAB.,
Co-Guide of the study and
Professor and Head of Department,
Department of diabetology,
Kilpauk Medical College, Chennai-10.

Prof. DR.S.USHA LAKSHMI MD.,F.I.M.M.C
Professor and Head of department
Dept of medicine
Kilpauk medical college Chennai-10

Prof. Dr. R. NARAYANA BABU, M.D., D.C.H
DEAN,
Kilpauk Medical College,
Chennai -10.

DECLARATION

I, solemnly declare that the dissertation entitled **"RELATIONSHIP BETWEEN ARM LENGTH AND TYPE2 DIABETES MELLITUS"** is done by me at Kilpauk Medical College, Chennai – 10 during the academic year of 2014 to 2017 under the guidance and supervision of **Prof. Dr. T. S. SANTHI, M.D.,** and **Prof. Dr. E. SURESH MD., D. Diab** to be submitted to **The Tamilnadu Dr. M.G.R. Medical University** towards the partial fulfilment of requirements for the award of **M.D. DEGREE IN GENERAL MEDICINE BRANCH – I.**

Place: Chennai

Date:

(Dr. ELAYARAJA.P)

ACKNOWLEDGEMENT

At the outset, I would like to thank the beloved Dean, **Prof. Dr. R. NARAYANA BABU, M.D., D.C.H** for permitting me to utilize the infrastructure and resources needed to conduct this study in Kilpauk Medical College.

I would like to express my sincere gratitude to **Prof. Dr. K. V. LEELA M.D., D.G.O.,** Vice Principal, for the timely help and support to this study. I would like to thank **DR.S.USHALAKSHMI M.D, F.I.M.M.C** Professor and HOD, Department of General Medicine, Kilpauk Medical College for permitting me to conduct this study.

I would like to thank wholeheartedly to my Guide**, Prof. Dr. T.S.SANTHI M.D.,** Professor , Department of General Medicine, Govt., Royapettah hospital and **Prof.Dr.E.SURESH M.D**.,**D.DIAB**, HOD dept of diabetology,kilpauk medical college,kilpauk for their valuable guidance and encouragement during this study.

It gives me immense pleasure to express my sincere and deep gratitude to Professors **Prof. Dr.S.MAYILVAHANAN M.D.,** Prof**. Dr. K. V. RAJALAKSHMI M.D.,** and **Prof. Dr. P. PARANTHAMAN M.D., DTCD.,** for their constant motivation and critical suggestions.

I sincerely thank the **Prof. Dr. KOMALA MD.,** dept of Biochemistry for their valuable guidance and support.

I am extremely thankful to the Registrar **Dr. S. KALAICHELVI M.D.,** Asst. Professors **Dr. I. ROHINI, M.D., Dr. JAYAKUMAR MD., DM, Dr. N. JAYAPRAKASH M.D.,** and **Dr. H. ANU RADHA M.D.,** and other Assistant Professors for their help and support.

I would always remember with extreme sense of thankfulness, the co-operation and criticism shown by my fellow post graduates colleagues and friends.

I extend my thanks to the Departments of Biochemistry, Diabetology for their cooperation and support.

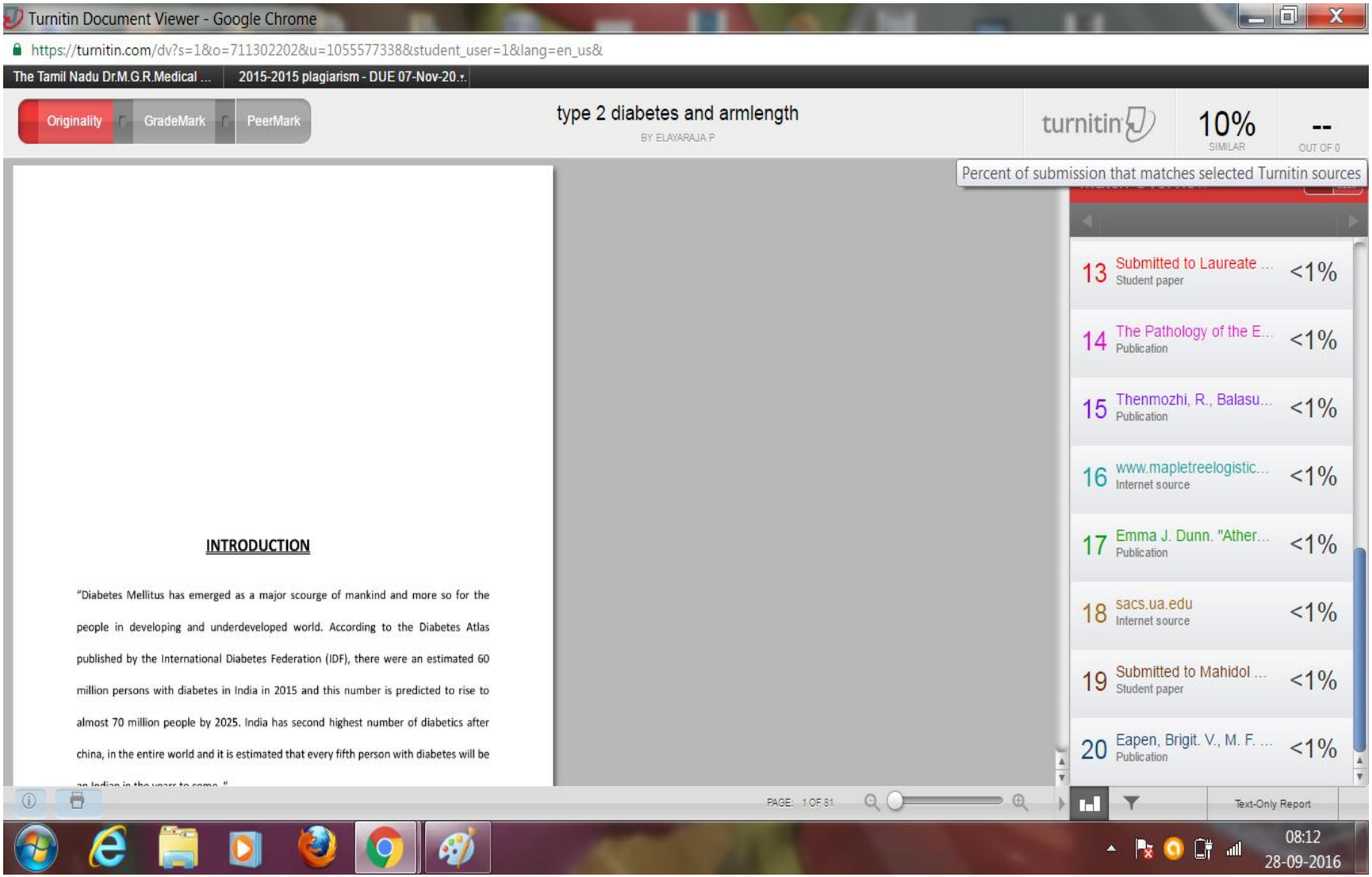
I wholeheartedly thank all my study people involved in this study and without them this would not have become a reality.

Last but not the least I would like to thank my cases and controls for giving full cooperation for my study. Without my patient I am not complete my study.

Finally I thank all of them those who helped me to finish this study in a successful manner.

ABBREVIATIONS

ADA	-	American Diabetes Association
AGEs	-	Advanced Glycosylation End Products
BMI	-	Body Mass Index
BUN	-	Blood Urea Nitrogen
CAD	-	Coronary Artery Disease
CKD	-	Chronic Kidney Disease
DM	-	Diabetes Mellitus
ESRD	-	End-Stage Renal Disease
e-GFR	-	Estimated Glomerular Filtration Rate
FPG	-	Fasting Plasma Glucose
GTT	-	Glucose Tolerance Test
HbA1C	-	Haemoglobin A1 C
IUGR	-	intra uterine growth retardation.
IDF	-	international diabetic federation.
PPPG	-	Post Prandial plasma Glucose


Turnitin Document Viewer - Google Chrome
https://turnitin.com/dv?s=1&o=711302202&u=1055577338&student_user=1&lang=en_us&
The Tamil Nadu Dr.M.G.R.Medical ...
2015-2015 plagiarism - DUE 07-Nov-20..
Originality
GradeMark
PeerMark
type 2 diabetes and armlength
BY ELAYARAJA P
turnitin
10%
SIMILAR
--
OUT OF 0
Percent of submission that matches selected Turnitin sources
INTRODUCTION
"Diabetes Mellitus has emerged as a major scourge of mankind and more so for the people in developing and underdeveloped world. According to the Diabetes Atlas published by the International Diabetes Federation (IDF), there were an estimated 60 million persons with diabetes in India in 2015 and this number is predicted to rise to almost 70 million people by 2025. India has second highest number of diabetics after china, in the entire world and it is estimated that every fifth person with diabetes will be
13 Submitted to Laureate ... Student paper <1%
14 The Pathology of the E... Publication <1%
15 Thenmozhi, R., Balasu... Publication <1%
16 www.mapletreelogistic... Internet source <1%
17 Emma J. Dunn. "Ather... Publication <1%
18 sacs.ua.edu Internet source <1%
19 Submitted to Mahidol ... Student paper <1%
20 Eapen, Brigit. V., M. F. ... Publication <1%
PAGE: 1 OF 81
Text-Only Report
08:12
28-09-2016

CONTENTS

INTRODUCTION

INTRODUCTION

Diabetes Mellitus has emerged as a major scourge of mankind and more so for the people in developing and underdeveloped world. According to the Diabetes Atlas published by the International Diabetes Federation (IDF), there were an estimated 60 million persons with diabetes in India in 2015 and this number is predicted to rise to almost 70 million people by 2025. India has second highest number of diabetics after china, in the entire world and it is estimated that every fifth person with diabetes will be an Indian in the years to come.

It has been observed over time that some ethnic groups are more predisposed to diabetes than the rest. This can be explained by the "thrifty gene hypothesis" proposed by geneticist James V. Neel in 1962. It postulates that a genetic predisposition to develop diabetes was adaptive to the feast and famine cycles of paleolithic human existence, allowing humans to fatten rapidly and profoundly during times of feast in order that they might better survive during times of famine. Fatter individuals carrying the thrifty genes would thus better survive times of food scarcity. However, in modern societies with a constant abundance of food, this genotype efficiently prepares individuals for a famine that never comes. It follows from this theory that ethnic groups with a history of food scarcity will have undergone a relatively high evolutionary pressure and hence may harbour more thrifty genes than other populations.

In 1992, Nicholas Hales and David Barker proposed the thrifty phenotype, they suggested that an individuals metabolic profile is determined not by their genetic composition, but rather by the environmental cues during the early periods of life[1]. This theory claims that the nutrition of a baby during fetal and early postpartum life determines the efficiency of metabolism in the adult life. The development of insulin resistance is postulated to be directly related to the body "predicting" a life of starvation for the developing fetus[2]. Given this metabolic profile, the child will have a greater chance of survival in a setting that is lacking adequate food resources or that undergoes bouts of famine. But, if at any point in their life their situation changes and they are in an environment of persistent nutritional affluence, their modified metabolism will prove detrimental in much the same way a thrifty genotype would.

The result of this mismatch between the environment in which the brain evolved, and the environment of today, is widespread chronic obesity and related health problems like diabetes, which is a major source of concern for societies undergoing a transition from sparse to better nutrition.

Although the relationship of diabetes with insulin resistance is clear at all the ages studied, but the relationship of diabetes to insulin secretion is less clear. The relative contribution of genes and environment to these relationships remains a matter of debate. Adverse early life environment, influences the development of beta cells (in terms of both mass and function) and insulin resistance, making an individual more prone to developing diabetes in later life[1].

Adult limb length and height reflect early life development, both in utero and during early childhood. They serve as surrogate measures of assessing nutritional environment and stress during early phases of life, and thereby are instrumental in determining the influence of early environment of an individual on the development of insulin resistance.

Since the hypothesis was proposed, many studies worldwide have confirmed the initial epidemiological evidence. A study done by Dr. Sanju Cyriac and Dr. Mohammed Ismail in 2007 showed that women with Gestational Diabetes had shorter leg length compared to normal pregnant women[3]. However only a single study has been done to test the hypothesis that arm length was inversely related to Type 2 Diabetes Mellitus. Hence the aim of this study is to analyze the correlation of arm length with Diabetes among the South Indian Population.

AIMS

&

OBJECTIVES

AIM OF THE STUDY

The aim of the study is to find the association of Type 2 Diabetes Mellitus with arm length as a marker for early life environment and development.

OBJECTIVES

1. To determine the relation of arm length with Diabetes Mellitus.
2. To determine whether arm length correlates better inverse relationship with Diabetes as compared to leg length.
3. Height waist ratio in relation to type 2 diabetes mellitus.

REVIEW OF LITERATURE

REVIEW OF LITERATURE

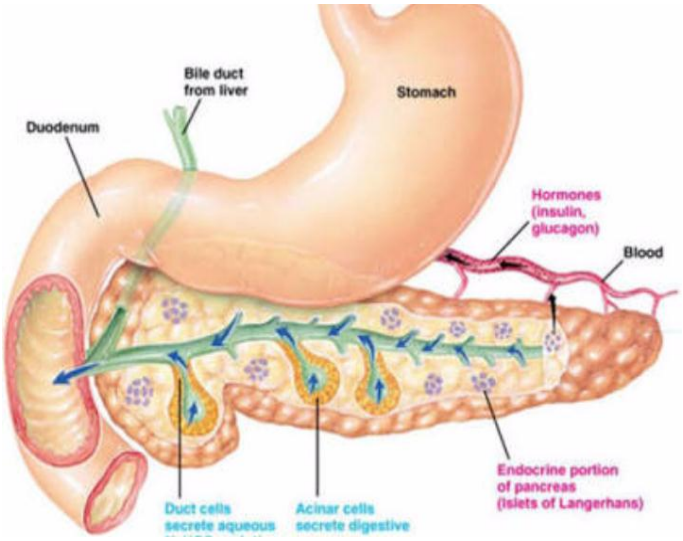
DIABETES MELLITUS

Diabetes mellitus is a group of metabolic disorders characrerised by hyperglycemia resulting from defects in insulin secretion , insulin action or both.

PANCREAS

FUNCTIONAL ANATOMY

The adult human pancreas is made up of numerous collections of cells called islets of langerhans. There are about 1-2 million islets and it makes up only 2% volume of pancreas. There are four major cell types in the islets of langerhans based on staining characteristics and appearance. They are:



Alpha cells: produces glucagon

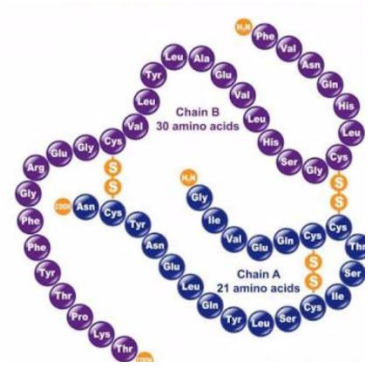
Beta cells: produces insulin, which is anabolic in nature. These cells are majority in islets of pancreas. About 60-70% .

Delta cells: produce somatostatin, which acts locally in a paracrine manner and inhibits secretion of pancreatic polypeptide, insulin and glucagon.

F(OR PP) cells: produce pancreatic polypeptide, which is shows absorbtion of food, but its physiological significance is uncertain.

INSULIN SYNTHESIS

Human insulin gene is located in region p13 of chromosome 11, which consists of 3 exons and 2 introns. There exists tissue selective expression of insulin gene – confined to beta cells of pancreas, with exception of yolk sac and fetal liver. Insulin is made up of 2 chains – A chain (21 amino acids) and B chain (30 amino acids) both linked by disulphide bridges.



Insulin biosynthesis occurs in the beta cells in two intermediate stages -

- Synthesis of preproinsulin (in endoplasmic reticulum), which is cleaved by protease into proinsulin.
- Conversion of proinsulin to insulin (in golgi apparatus), by trypsin and carboxypeptidase B-like activity, proinsulin is cleaved into insulin and c-peptide

INSULIN SECRETION

Insulin secretion occurs by the process of exocytosis. Insulin, which is stored as granules moves along the network of microtubules and micro filaments towards the plasma membrane. The contractile proteins, actin and myosin also play a role in secretion. The factors which regulate insulin secretion are:

- Nutrients
- Hormones
- Neural

NUTRIENTS

The most important physiological stimulus is glucose. In respose to oral glucose load of 12g, 1.4 units of insulin is secreted. Incretin effect explains the greater secretory response after oral glucose as compared to intravenous glucose administration. Glucose stimulates insulin release in 2 phases – the first is a rapid phase lasting 5-10 mins, followed by a prolonged second phase which persists during stimulus of the high glucose.

HORMONES

Gut peptides – glucagon and somatostatin affects insulin secretion by local paracrine action. Gastric inhibitory polypeptide and glucagon like peptide – 1 increases insulin secretion by increasing the intracellular cAMP

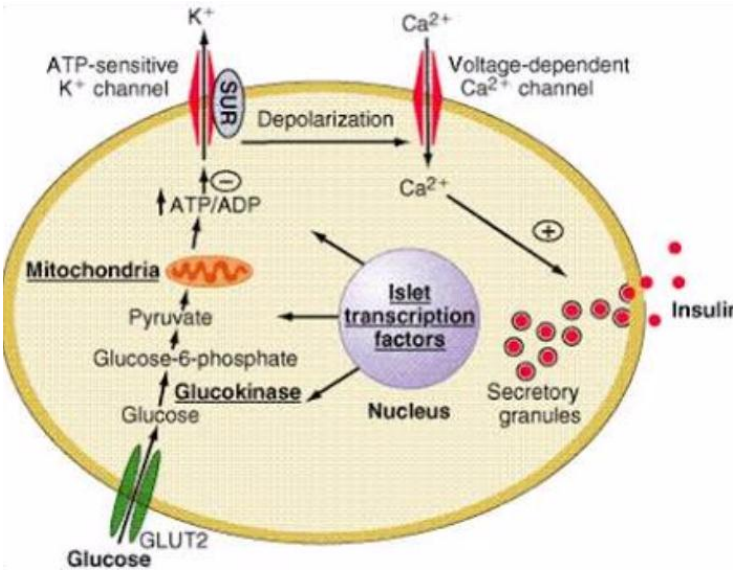
levels. Gut hormones augment insulin release following oral glucose administration – which is termed as ‘enteroinsular axis’.

NEURAL

Prior to food intake, the smell, sight and expectation of food lead to insulin secretion to minimize early rise in postprandial plasma glucose, which is known as cephalic phase of insulin secretion. Vagus nerve controls this ‘Hypothalamo entero insular axis’. Vagotomy and pancreatic transplantation (islet denervation) results in early rise of post prandial plasma glucose.

INSULIN RELEASE FROM B – CELLS:

In the basal state, ATP modulated K+ channels remain open and voltage dependant Ca++ channel is closed. With the entry to glues through GLUT – 2, depolarization of the membrane occurs and the K+ channel is closed and Ca++ channel is opened, which releases insulin stored in the cytosol by emicytosis. The released insulin, after entering the portal vein, in part gets metabolized by first pass metabolism. Insulin sercetogogues can be physiological or pharmacological. The initiators, also called as primary stimulants provoke insulin release, which include glucose, noncarbohydrates (like arginine, lysine, ketone bodies and free fatty acids) and drugs(sulphomylurease). The potentiators are ineffective by themselves, but increase the release in response to glucose or aminoacids, which include cAMP, acetylcholine and the gut peptides (GIP, glucagon, secretin)



MECHANISM OF ACTION OF INSULIN

Insulin receptor is a glycoprotein made of 2a and 2 b subunits lined by disulphide bridges. Insulin binds to a subunit, which is extracellular, which triggers tyrosine kinase activity in the b subunit and causes auto phosphorylation. This brings about phosphorylation or dephosphorylation of certain proteins and enzymes in the cytoplasm. Protein synthesis and growth promoting actions of insulin are mediated through phosphoinositol 3- kinase pathway.

Insulin resistant states can be due to

- Genetic disorders (Type A syndrome and its variants – Leprechaunism and Lipoatrophic diabetes)
- Immune disorders (Type B syndrome – Anti insulin antibodies and anti insulin rector antibodies)

- Metabolic conditions (Obesity, type 2 diabetes)
- Physiological (puberty, pregnancy, aging, cushings syndrome)

DIABETES MELLITUS – DIAGNOSTIC CRITERIA

- Glucose tolerance is normal when the fasting and 2-hr values and <100mg/dl and <140 mg respectively
- Diabetes is diagnosed if the fasting is >= 126mg or 2-hr plasma glucose is >=200 mg
- Imparied glucose tolerance is present when the 2-hr value is 140-199 mg/dl
- Impaired fasting glucose is present when the fasting level is >=100 and <= 125 and the 2 hr is <-140 mg/dl

CLASSIFICATION OF DIABETES MELLITUS

TYPE 1

Beta cells destruction usually leading to absolute insulin deficiency

- Autoimmune (islet cell antibody and GAD positive)
- Idiopathic

TYPE 2

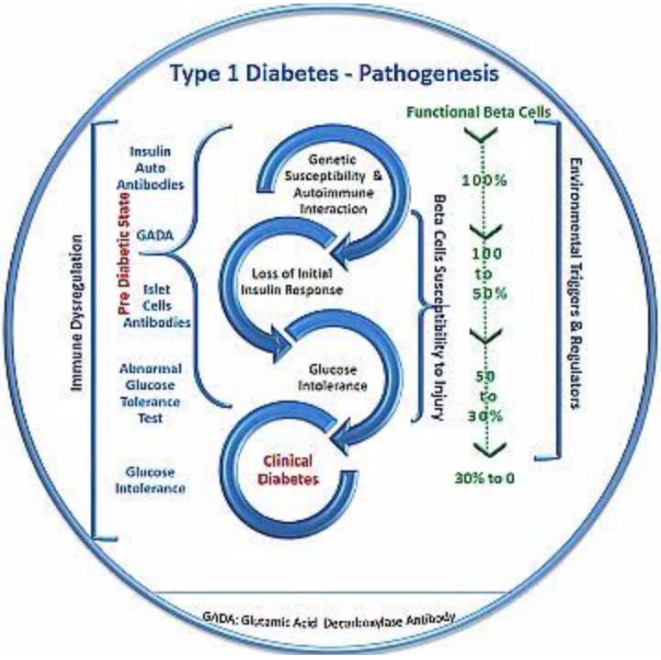
- Predominantly insulin resistance
- Predominantly insulin secretory defects

OTHER SPECIFIC TYPES OF DIABETES

- Genetic defects of beta cell dysfunction (MODY 1 to 6)
- Genetic defects in insulin action
- Disease of exocrine pancreas (e.g. Fibro calculus pancreatopathy)
- Endocrinopathies (e.g. Acromegaly, Cushings)
- Drugs or chemical – induced (e.g. glucocorticoids)
- Infections (e.g. congenital rubella)
- Uncommon forms (e.g. Stiff man syndrome)
- Other genetic syndromes

GESTATIONAL DIABETES

AETIOPATHOGENESIS OF TYPE 1 DIABETES MELLITUS



I. GENETIC FACTORS

A) Twin studies

About 10% of type 1 DM subjects have a sibling or parent with the disease. Among identical twins, if one of them develops type 1 DM, the co-twin has a 30-50% chance of developing the disease.

B) Histocompatibility antigen

Population studies show that the association of type 1 DM with HLA-B8 genes DR3-DQ2 and DR4-DQ8, which poses 14 times higher risk of developing diabetes mellitus is Caucasians. In Indian population, there is no association with HLA-B15. The empiric risk of developing type 1 diabetes in the general population is 0.4%. Around 1 in 20 first degree relatives of patients with type1 diabetes mellitus will develop this disorder.

C) Insulin Gene

Insulin gene, located in short arm of Chromosome 11, with nucleotide flanking the gene has been reported with type 1 DM

II. IMMUNOLOGICAL FACTORS

The feature which confirm the autoimmune pathogenic mechanism – 'insulitis' - is circulating antibodies to islet-cells and cell mediated abnormalities in type 1 DM. Islet cell antibodies (ICA) belonging to Ig G is

directed against the cytoplasm of these cells. 80=90% of the beta cells gets deranged before the cinical manifestation of diabetes. These islet cell antibodies are demonstrated by conventional direct immune fluorescence (IFL). Islet cell surface antibodies(ICSA) has also been detected. The ICSA show separate specificity specifically to the β, α and γ cells. Recently a protein of MW 64 kDa has been identified in human islet cells, which has been identified as glutamic acid decarboxylase (GAD65), which is beta cell specific unlike ICA, which is islet specific.

Insulin auto antibodies (IAA) to insulin molecule has been described recently in newly diagnosed type 1 DM. Lymphocytes from type1 DM subjects can kill cultured insulinoma cells. Inspite of total T cell population not being altered in type 1 DM subjects, the population of suppressor T cell is decreased.

III. VIRUSES/TOXINS

Epidemiological studies have linked Coxsackie B4 virus, congenital rubella, encephalomyocarditis virus, mumps virus, Venezuelan equine encephalitis virus, herpes virus and echo virus to be associated with type 1 DM. Viruses damage the beta cells by direct invasion or be triggering an auto immune response. Chemical agents like pentamidine and Vacor (rodenticide) have been linked with causation of type1 DM.

IV. DIETARY FACTORS

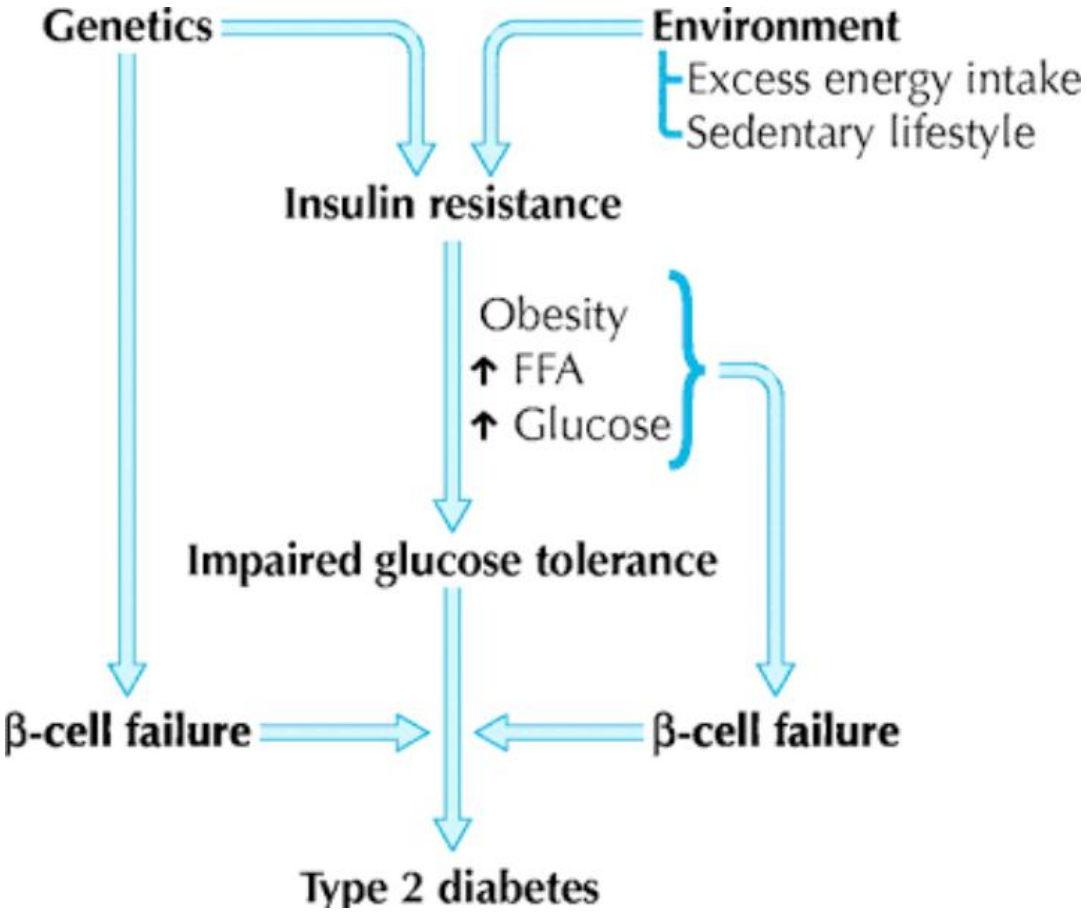
Consumption of cow's milk in early life is a contributory factor for development of type 1 DM. The Bovine Serum Albumin (BSA), an antigen may enter in an intact form through the gut of neonates and stimulate an immune response directed against β cells. Nitrosamines, found in smoked and cured meat has found to bear an association with causation of type1 DM.

HONEY MOON PHASE

At the onset of the disease, the beta cell response to secretogogues, being poor leads to high insulin requirement. After the correction of hyperglycemia, the endogenous insulin secretory capacity recovers and the exogenous insulin decreases. This is called as honeymoon phase, which lasts for few months to years. With total destruction of beta cells absolute deficiency of insulin occurs.

AETIOPATHOGENES OF TYPE 2 DIABETES MELLITUS

Diabetic genotype is influenced by various factors, the predominant one being central obesity, and others are physical indolence, dietary habits consumption off refined carbohydrates and reduced intake of fiber, urbanization with associated affluence and the stress of life.



The abnormalities in the genesis of hyperglycemia are:

1. Impaired pancreatic insulin secretion

2. Peripheral resistance to insulin action (liver and muscle)

3. Excessive hepatic glucose uptake

IMPAIRED PANCREATIC INSULIN SECRETION

Beta cell dysfunction falls into 2 distinct types: a) the pulsatile delivery is lost even when the glucose tolerance is normal and b) the loss of compensatory mechanisms. Insulin secretory abnormalities in type 2 diabetes mellitus include:

- Decreased glucose sensing
- Impaired ability to respond to elevations and reductions during glucose infusion
- Reduced or absent first-phase insulin secretion in response to intravenous glucose administration
- Reduced or absence early insulin secretory response to oral glucose
- Alterations in the rapid oscillations of insulin secretion
- Inadequate insulin secretion for the magnitude of hyperglycemia
- Reduced effect of gastrointestinal hormones in potentiating glucose-mediated insulin secretion.

IMPAIRED PERIPHERAL ACTION OF INSULIN

Hyperinsulinemia antedates the development of type 2 DM, which is due to resistance in various tissues like liver, muscle, splanchnic. Post binding defects are of 3 types: a) impaired generation of insulin's second messenger b) diminished glucose transport into the cell and c) a post glucose transport abnormality in some critical step involved in glucose utilization. In

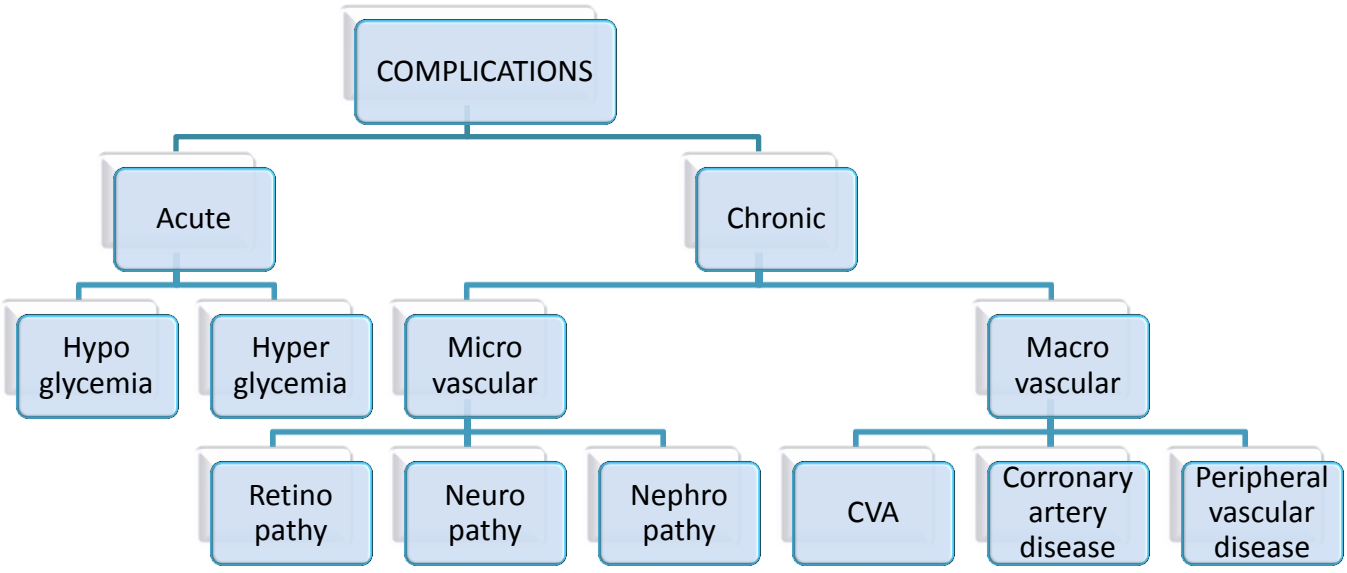
muscles, there is impaired insulin receptor tyrosine kinase activity, diminished glucose transporters and diminished glycogen synthetase and diminished glycogen synthetase and pyruvate dehydrogenase.

INSULIN RESISTANCE AS A PRIMARY DEFECT

Insulin resistance is an inherited defect that initiates the diabetic event. The hyperglycemia to insulin resistance occurs in 3 phases:

First phase	Plasma glucose remains normal inspite of reistance because insulin levels are increased
Second phase	Insulin resistance tends to worsen and post prandial hyperglycemia develops inspite of high insulin concentration
Third phase	Insulin resistance does not change but decreasing insulin secretion causes fasting hyperglycemia.

COMPLICATIONS OF DIABETES MELLITUS



PATHOGENESIS OF COMPLICATIONS

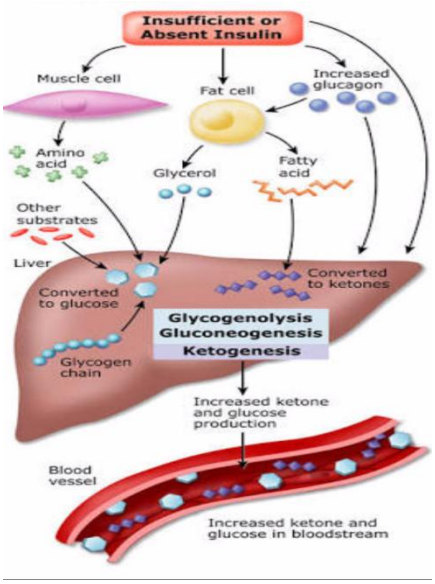
ACUTE COMPLICATIONS

Diabetes mellitus can lead to both hypo and hyperglycemia. Hypoglycemia while on treatment can be due excess insulin dosage or oral hypoglycemic agents. Symptoms of palpitations, giddiness, light headedness should arouse the suspicion of hypoglycemia and hence patient education in this aspect and its management is very necessary.

DIABETIC KETOACIDOSIS

Insufficient amount of insulin or absent insulin causes increased glucagon, breakdown of fatty acids and muscle breakdown into glucose formation, which in addition to increased glycogenolysis, causes increased free fatty acids and decreased malonyl coA and hepatic carnitine content leading to accelerated ketogenesis. Deficient conversion of blood glucose into glucose 6 phosphate and pyruvate and further breakdown, leads to accumulation of acetoacetyl co A, which forms acetoacetic acid, acetone and acetonemia and acetonuria.

Glycosuria leads to urinary losses of water and electrolytes, leading to dehydration and decreased extracellular fluid volume. Acetonemia leads to sodium depletion, loss of base and metabolic acidosis. Further, cellular catabolism and stress leads to potassium, chloride and phosphorus depletion.



CHRONIC COMPLICATIONS

Normal tissue

↓

Functional changes

↓

Early structural changes

↓

Progressive tissue damage - irreversible

↓

End stage disease

The chronic complications of diabetes mellitus is caused by hyperglycemia induced functional changes in the cellular level and further causing structural changes, which is progressive and irreversible leading to end stage disease.

A) VASCULAR CHANGES

To ensure adequate nutrition to the tissues, blood vessels must possess inherent mechanisms to regulate the flow and contractility, permeability, coagulation and regeneration following injury.

The changes that occur in diabetes are:

i) REDUCED CONTRACTILITY –

Endothelium dependant relaxation in response to acetyl choline is absent in diabetes.

ii) THICKENED BASEMENT MEMBRANE –

Leads to increased permeability, which precedes structural changes and hence is reversible

iii) ENDOTHELIAL DYSFUNCTION –

Reduction in relaxing factors and increase in constriction factors along with hyperglycemia, impaired insulin action, proatherogenic dyslipidemia, hypertension, obesity, vasoactive hormones, cytokines and VEGF lead to atherogensis.

iv) COAGULATION –

Elevated fibrinogen, VWF that complexes with factor VIII, reduced PGI2 lead to atherogenesis in diabetes.

v) Degeneration of pericyte leads to reduced contractility and increased endothelial proliferation further leading to microaneurysm formation.

vi) Increased CRP, hyper homocysteinemia and increased plasminogen activator inhibitor – 1 are the other factors leading to vascular complications in diabetes.

B) METABOLIC CHANGES

Mechanism of hyperglycemia induced tissue damage occurs due to

- Aldose reductase activity/ redox changes
- Diacylglycerol – protein kinase C activation
- Formation of advanced glycation end products
- Formation of reactive oxygen species.

<u>i) POLYOL PATHWAY</u>

This alternate pathway for glucose metabolism, glucose is converted to sorbitol by aldose reductase, which is then oxidized to fructose by the enzyme sorbitol dehydrogenase. Aldose reductase has low affinity (high kM) for glucose and thus under euglycemic conditions, AR is not very active.

ii) ADVANCED GLYCATION END PRODUCTS

Glycation, a chemical modification to proteins with reducing sugars, indicates a possible explanation for the association between hyperglycemia and tissue pathologies. Reducing sugars react with the amino groups of long-lived proteins to produce non-enzymatic crosslinks, which are the end-stage products of the Maillard reaction and are also known as advanced glycation end products (AGEs)

MORPHOLOGICAL CHANGES

EYES

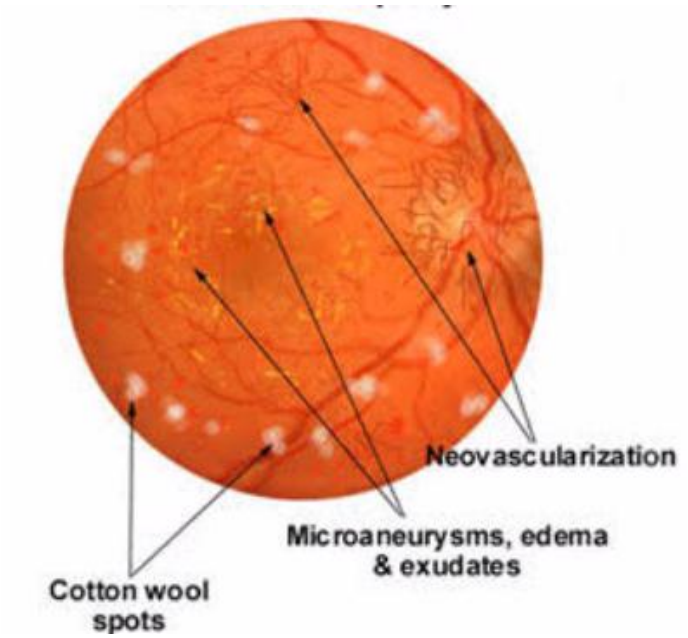
About 60 % of diabetics develop retinopathy within 15 to 20 years. Basement membrane thickening and pericyte degeneration leads to microaneurysm formation, which is the hallmark of retinopathy. Retinopathy can be classified as:

BACKGROUND RETINOPATHY

- Microaneuryms and dot hemorrhages are earliest abnormalities
- Leaks lead to blot hemorrhages
- Exudation of lipid and protein rich fluid causes hard exudates
- Associated macular edema leads to Maculopathy
- Diminished perfusion of capillaries causes retinal infarcts and edema, seen as cotton wool spots. (Preproliferative Retinopathy)

PROLIFERATIVE RETINOPATHY

Persistent hypoxia triggers neovascularisation from the veins which are friable and can cause hemorrhages into the retina and vitreous. When fibrosis follows it, it is called retinitis proliferans, which leads to increased traction resulting in in-retinal detachment.



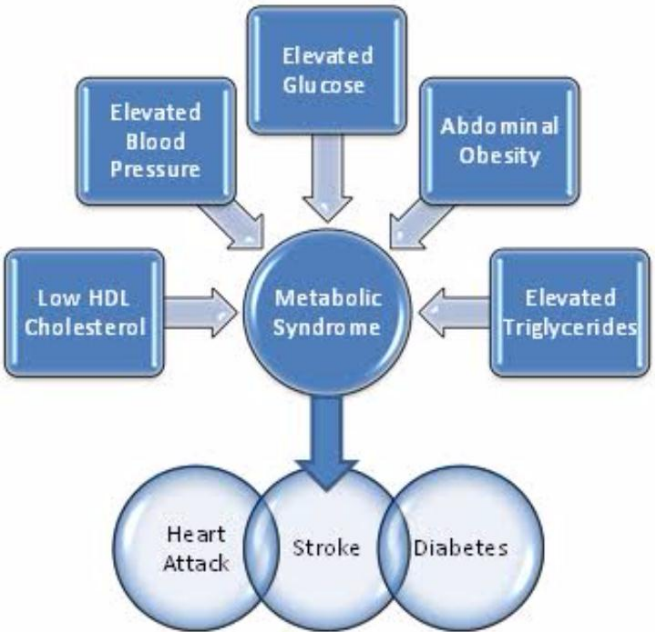
KIDNEY

The earliest abnormality is renal hypertrophy with raised GFR. Glomerular basement membrane thickens, mesangial cells proliferate to increase the mesangial matrix, which can even obliterate the glomerulus. This is called as diffuse glomerulosclerosis. When the glomerulus takes the form of peripherally situated ovoid hyaline mass, it is termed nodular glomerulosclerosis or Kimmelstein – Wilson disease, which is pathognomic of diabetes.

NERVES

The primary event to initiate nerve damage remains unclear. In diabetic mono neuropathies, the main mechanism appears to be occlusion of the vasa nervorum causing ischemic damage to the nerves. The commonest variety is the symmetric distal polyneuropathy, characterized by Schwann cell injury leading to myelin degeneration and axonal damage.

METABOLIC SYNDROME



According to The New International Diabetes Federation (IDF) definition, for a person to be defined as having the Metabolic syndrome, they must have:

CENTRAL OBESITY (defined as waist circumference >94 cm or Europid men and >80cm for Europid women, with ethnicity specific values for other groups)

Plus any two of the following:

- Raised **TGL** – 150 mg/dl, or specific treatment for this lipid abnormality
- Reduced **HDL Cholestrol** - <40 mg/dl in males and <50 mg/dl in females, or specific treatment for this lipid abnormality
- Raised **blood pressure** – Systolic BP >130 or diastolic BP >85 mm Hg, or treatment of previously diagnosed hypertension.
- Raised **fasting plasma glucose** >100 mg/dl or previously diagnosed type 2 diabetes

Insulin resistance is considered as the primary defect in the patho physiological mechanism underlying the development of the syndrome. However, inflammation and endothelial dysfunction has a role in the causation. Metabolic and pro-thrombotic cardiovascular risk factors influence the risk of developing athero-thrombotic vascular disease.

Targets for treatment of the components of metabolic syndrome are:

	High risk	Very low risk
BP	<135/85	<120/80
Fasting glucose	<110 mg/dl	<100 mg/dl
2h plasma glucose	<180 mg/dl	<144 mg/dl
TGL	<150 mg/dl	<132 mg/dl
HDL	>35mg/dl	>39mg/dl

What makes certain ethnic groups more predisposed to Diabetes?

Thrifty Gene Hypothesis

- "Thrifty genotype" modified the regulation of insulin release and glucose storage, may have provided a survival advantage for some of our hunter-gatherer predecessors[1].
- This metabolic profile would have permitted them to fatten more quickly during times of abundance, allowing them to efficiently store excess energy. Fatter individuals carrying thrifty genes would thus better survive through periods of food scarcity.
- Abundance these genes however predispose their carriers to diseases caused by excess nutritional intake, such as obesity.

Thrifty Phenotype Hypothesis

- Nutrition of a baby during fetal and early postpartum life determines the efficiency of metabolism in the adult life. The development of insulin resistance is postulated to be directly related to the body "predicting" a life of starvation for the developing fetus[3].
- Adverse early life environment, influences the development of beta cells (in terms of both mass and function) and insulin resistance, making an individual more prone to developing diabetes in later life.[2]

" THE BARKER HYPOTHESIS "

The "developmental origins of adult disease" hypothesis, often called "the Barker hypothesis" proposes that these diseases originate from adaptations of the fetus when it is malnourished[undernourished]. These adaptations may be cardiovascular, metabolic, or endocrine, and they can permanently alter the structure and function of the body, increasing coronary vascular disease risk factors, such as syatemic hypertension, type 2 diabetes mellitus, insulin resistance, and hyperlipidaemia[4-7]. This hypothesis originally involved from observation by Barker and colleagues that the regions in England with the highest rates of infant mortality in the early 20th century also had the highest rates of mortality from coronary heart disease decades later. As themost commonly registered cause of infant death at the start of 20th century was low birth weight, these observations led to the hypothesis that lowbirth- weight babies who survived infancy and childhood might be at increased risk of coronary heart disease in later life [8].

As Barker reported in several epidemiological and anthropological studies, in fetal period, tissues and organs go through the so-called "critical" periods of development. These may coincide with periods of rapid cell division. Although the fetal growth is influenced by its genes, several studies proved that it is usually influenced by intrauterine environment, in particular the nutrients and oxygen received from the mother. Influence linked to

fetal and placental growth have an important effect on the risk of coronary heart disease and stroke. Thus, this theory focusing on intrauterine life, offers a new point of departure for research in cardiovascular disease. According to the thrifty phenotype hypothesis, deficient fetal supply may be followed by a programming, which includes circulatory adjustment and insulin resistance in liver and muscular tissue in order to spare the brain [9-14].

Postnatal overnutrition following intrauterine growth restriction can be reason for the development of obesity and type 2 diabetes. whereas the elevated cardiovascular mortality may be associated with rapid postnatal catch-up growth in early infancy [15]. Many reports of correlation between birth size and glucose and insulin metabolism have widely been reviewed. In particular, fetal growth retardation has been associated with increased insulin resistance, more fasting insulin concentrations, and increased incidence of type 2 diabetes. Neonatal abdominal circumference has been shown to predict plasma cholesterol and fibrinogen levels in adults in later life, which are both risk factors for cardiovascular disease.

Programming of Noninsulin- Dependent Diabetes

Insulin has a main role in fetal growth, and alteration of glucose and insulin metabolism are therefore an obvious possible connection between early growth and cardiovascular disease [16]. Although obesity and a sedentary lifestyle are main role in the development of non-insulin dependent

diabetes, they seem to lead to the disease only in predisposed persons.. Family and twin studies have suggested familial predisposition, but the nature of this predisposition is unknown. The disease tends to be transmitted through the maternal rather than paternal side of the family [17].

Size at Birth and Noninsulin-Dependent Diabetes

A number of other studies have confirmed the relation between birth weight, impaired glucose tolerance, and noninsulin-dependent diabetes that was first reported in Hertfordshire [18-23] (**Table 1**). In the Health Persons Study in the United States, the odds ratio for diabetes, after adjusting for current body mass, was 1.9 among men whose birth weights were less than 5.5 lb compared with those who weighed 7-8.5 lb [24]. Among the Pima Indians in the United States, the odds ratio for diabetes was 3.8 in male and female who weighed less than 5.5 lb[25]. In Preston it was the thin babies who developed impaired glucose tolerance and diabetes. Lithell and colleagues confirmed the association with thinness in Uppsala, Sweden [22] (**Table 2**). The prevalence of diabetes was 3 times more among male in the lowest fifth of ponderal index at birth. This was a stronger association than that with birth weight, with the prevalence of diabetes only twice as high among male in the lowest fifth of birth weight. Among the Pima Indians, in whom diabetes in pregnancy is rare common, young men and women with birth weights over 9.9 lb had an increased prevalence of non insulin

dependent diabetes [25]. The association between birth weight and non-insulin dependent diabetes was therefore U-shaped. The increased risk of diabetes among babies with high birth weights was associated with maternal diabetes in pregnancy.

Table 1. Prevalence of noninsulin-dependent diabetes and impaired glucose tolerance in men 59-70 years of age

Birth weight lb (kg)	**No of men**	**% of impaired glucose tolerance or diabetes**
<5.5(2.50)	20	40
6.5(2.95)	47	34
7.5(3.41)	104	31
8.5(3.86)	117	22
9.5(4.31)	54	13
>9.5(4.31)	28	14

Table 2. Prevalence of noninsulin-dependent diabetes by ponderal index at birth among men 60 years of age in Uppsala, Sweden.

Ponderal index at birth (kg/m^3)	**No of men**	**% of diabetes**
<24.2	193	11.9
24.2	193	5.2
25.9	196	3.6
27.4	188	4.3
>29.4	201	3.5

Insulin resistance

The pathogenesis of noninsulin-dependent diabetes [26] mainly due to insulin deficiency and insulin resistance . There is evidence that both may be determined in fetal period . Male and female with low birth weight have a high prevalence of the insulin resistance syndrome [27] in which impaired glucose tolerance, hypertension, and raised serum triglyceride concentrations occur in the same patient. The patients are insulin resistant and have hyperinsulinaemia. **Table 3** shows results for a sample of the men in Hertfordshire. Phillips et al.[28] carried out insulin tolerance tests on 103 men and women in Preston. At any value of adult body mass index, insulin resistance was high in people who had a low ponderal index at birth. In addition, at each ponderal index, resistance was high in those with high body mass index. The greatest mean resistance was therefore in those with low ponderal index at birth but high body mass index as adults.

Table 3. Prevalence of the insulin resistance syndrome in men 59-70 years of age according to birth weight.

Birth weight lb (kg)	No of men	% with insulin resistant syndrome
<5.5(2.50)	20	30
6.5(2.95)	54	19
7.5(3.41)	114	17
8.5(3.86)	123	12
9.5(4.31)	64	6
>9.5(4.31)	32	6

Law et al. Reported relation between thinness at birth and raised 30-minute plasma glucose concentrations in 7-year old children in Salisbury, UK [29]. Whincup et al. Studied British children 10-11 years of age and found that plasma insulin concentrations both fasting and after oral glucose [30] in low birth weight babies. This is consistent with the relationship between low birth weight and insulin resistance. Among these children, however, the plasma glucose concentrations of those who had low birth weight were unaltered, which implies that despite being insulin resistant they were able to maintain glucose homeostasis. In contrast Yajnik and colleagues found that Indian children 4 years of age who had low birth weights had raised plasma glucose and insulin concentrations, suggesting that at the levels of low fetal growth and insulin resistance that prevail in India, even young children are unable to maintain glucose homeostasis [31]. Forrester and colleagues found an relation between reduced glucose tolerance and shortness at birth among children in Jamaica, in whom the serum glycated hemoglobin levels rose progressively between those who were 52 cm or more in length at birth and those who were 46 cm or less[32]. These findings in children provide further support for the hypothesis that impaired development in utero induce type 2 diabetes and that the seeds of diabetes in the next generation have already been sown and are apparent in today's children.

MECHANISM: INSULIN RESISTANCE

The processes that connection between thinness at birth with insulin resistance in adult life are not known. Reduced mid-arm circumference present in babies delivered at term with low ponderal index, whichimplies that they have a low muscle bulk as well as less subcutaneous fat[33]. It is therefore possible that thinness at birth is associated with abnormalities in the structure and function of muscle that develop in midgestation and persist into adult life, interfering with insulin's ability to promote glucose uptake. Magnetic resonance spectroscopy studies show that people who were thin at birth have lower rates of glycolysis and glycolytic ATP production during exercise [34] reduce its metabolic dependence on glucose and increase oxidation of other substrates, including amino acids and lactate seen in under nutrition fetus. This has led to the hypothesis that a glucose-sparing metabolism persists into adult life, and that insulin resistance arises as a consequence of similar processes, possibly because of reduced rates of glucose oxidation in insulin-sensitive peripheral tissues.

Concentrations of anabolic hormones including insulin and IGF-I fall ,when the fetus is restricted to nutrition, while catabolic hormones, including glucocorticoids rise. insulin resistance development also due to underlying persistant hormone changes.. Bjorntorp has postulated that glucocorticoids, growth hormone, and sex steroids may play a major role in the evolution of the metabolic syndrome[35].

INSULIN DEFICIENCY

Fewer beta cells [36] seen in infants ,who have small for dates. There are conflicting reports on whether the reduced P-cell mass in subjects with noninsulin-dependent diabetes[37]. As a working hypothesis it seems reasonable to Propose that **"the size and function of the adult pancreatic P-cell complement is influenced by nutritional and other factors determining fetal and infant growth".** Whether and when noninsulin dependent diabetes supervenes will be determined by the rate of attrition of D cells with aging, and by the development of insulin resistance, of which obesity is an important determinant [38].

In Mysore, South India, male and female showed signs of both insulin resistance and insulin deficiency [39,] those present with type 2 diabetes mellitus. People from South India living in Britain has been observed [40,41] high prevalence of central obesity, insulin resistance and type 2 diabetes mellitus. The study of men and women in Mysore again showed this. Those who had noninsulin-dependent diabetes also had a low insulin increment after a standard challenge, indicating that they were insulin deficient as well as resistant. However, whereas insulin resistance was associated with low birth weight, type 2 diabetes was associated with shortness at birth in relation to birth weight (i.e., a high ponderal index) and with maternal adiposity.

These findings led to a novel explanation for the epidemic in urban and migrant Indian populations [39] type 2 diabetes mellitus.. Widespread predisposition of Indian population to insulin resistance due to fetal under nutrition. Their levels of physical activity diminished When these people move to cities . Young women, no longer required to do agricultural work or walk long distances to fetch water and firewood, become fatter and more insulin resistant. They are therefore not able to maintain glucose homeostasis during pregnancy, even at relatively low levels of obesity, and become hyperglycemic, though not necessarily diabetic. It is known that high plasma glucose concentrations within the normal range influence fetal growth and lead to macrosomia.

MATERIALS & METHODS

MATERIALS AND METHODS:

This study was conducted in Government Royapettah hospital, Chennai for a duration of 6 months from April 2016 to Sep 2016. A proper ethical approval was obtained from the Institutional Ethical Committee .The study was conducted after getting informed consent from all the Subjects involved in this study.

Study Design : case control study

Collaborating Depts. : Diabetology, Biochemistry,
And Master Health Check up

Study Period : 6 months (April 2016 to Sep 2016)

Conflict of Interest : Nil

Study population:

Patients attending Diabetology outpatient department will be included in the study. An equal number of Healthy, age and sex matched subjects without diabetes or its complications, who are undergoing master Health Check up will be included in the study for control.

Inclusion Criteria:

Case

- Fasting plasma glucose (FBS) of >125 mg/dl, or
- Postprandial plasma glucose at 2Hr (PPBS) > 200mg/dl.
- Who are k/c/o t2dm an regular treatment with controlled blood sugar value.

Controls

- Fasting plasma glucose (FBS) of <110 mg/dl, or
- Postprandial plasma glucose at 2Hr (PPBS) < 140 mg/dl.

Exclusion Criteria:

- Type I Diabetes Mellitus
- Patients with Impaired Glucose Tolerance
- Limb deformities due to any cause

Sample size: 134 (67cases, 67 controls) for each male and females.

Sample size calculated with G* POWER 3.1.3 VERSION, by using standard error, power of the study.

T test –means: difference between two independent means(two group)

Analysis: a priori : compute required sample size

Input:	trail(s)	=	two
	Effect size d	=	0.49
	Alpha error	=	0.05
	Power(1-beta error)	=	0.80
	Allocation ratio N2/N1	=	1
Output:	noncentrality parameter	=	2.836
	Critical t	=	1.978
	Df	=	132
	Sample size group 1	=	67
	Sample size group 2	=	67
	Total sample size	=	134
	Actual power	=	0.80384

Methodology:

After obtaining informed written consent, basic demographic details, detailed clinical history and physical examination was done. Base line Fasting and post prandial Blood sample for Blood Glucose has been collected.

Measurements:

Total arm length was defined as the distance in centimeters between the superior border of the acromion process and the tip of the middle finger, when the arm and hand were fully extended

Upper arm length: distance between the superior border of the acromion process to the posterior surface of olecranon process of ulna, with arm flexed at 90 degrees

Forearm length: With arm flexed at the elbow, measured from the head of the olecranon process to the tip of the styloid process of ulna.

Height: defined as the maximum distance from the floor to the vertex of the head, with patient standing erect with heels and toes together and the arms hanging by the sides.

Total leg length: from the standing surface to the trochanteric landmark

Lower leg length: with the patient sitting with slightly flexed knee, and with the leg crossed over the opposite leg; measured from the medial border of the proximal tibia to the distal tip of the medial malleolus .

Waist circumference: Perimeter measured at the approximate midpoint between the lower margin of the last palpable rib and the top of the iliac crest, in standing position after normal expiration

Hip circumference: This is the perimeter at the level of the greatest posterior protuberance of the gluteals.

BMI: Weight in kgs/ (Height in metres)^2 x 100

Data collection: Data was collected using a pre- designed Proforma, after obtaining the Ethics committee approval. Written informed consent of participants was taken.

Data Analysis:

The collected data were analysed with IBM.SPSS statistics software 23.0 Version.To describe about the data descriptive statistics frequency analysis, percentage analysis were used for categorical variables and the mean & S.D were used for continuous variables. To find the significant difference between the bivariate samples in Independent groups (Cases & Controls) the Unpaired sample t-test was used.In the above statistical tool the probability value .05 is considered as significant level. .Continuous variables such as arm length, height waist ratio, leg length will be presented as mean with standard deviation.

- Compare the mean value between cases and controls.

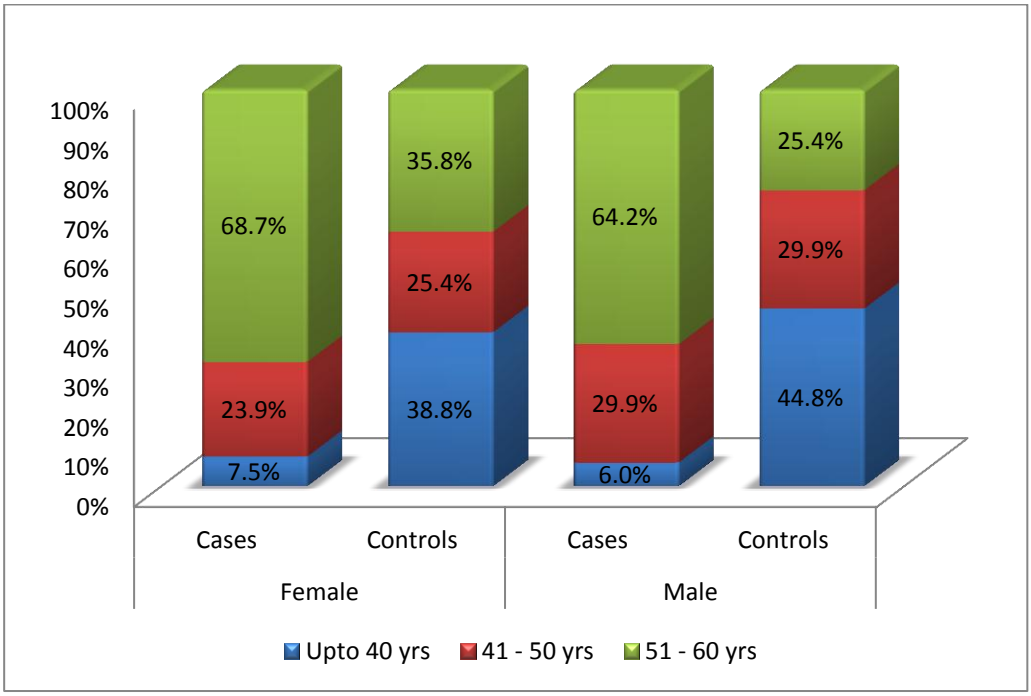
RESULTS & ANALYSIS

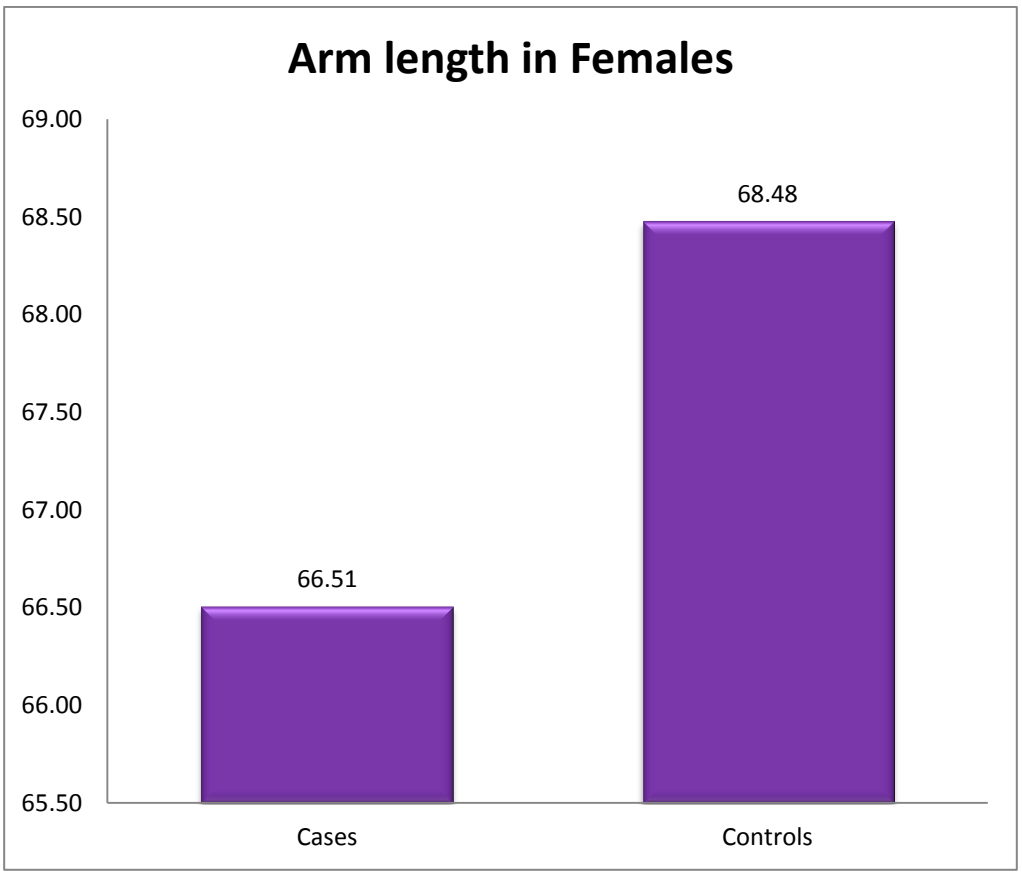
RESULTS AND OBSERVATIONS

In our study, the maximum number of female cases belonged to the age group 51-60 yrs and that was about 68.7 %. The maximum number of male cases belonged to age group 51-60 yrs and that was about 64.2%.

	Female		Male	
	Cases	Controls	Cases	Controls
Upto 40 yrs	7.5%	38.8%	6.0%	44.8%
41 - 50 yrs	23.9%	25.4%	29.9%	29.9%
51 - 60 yrs	68.7%	35.8%	64.2%	25.4%

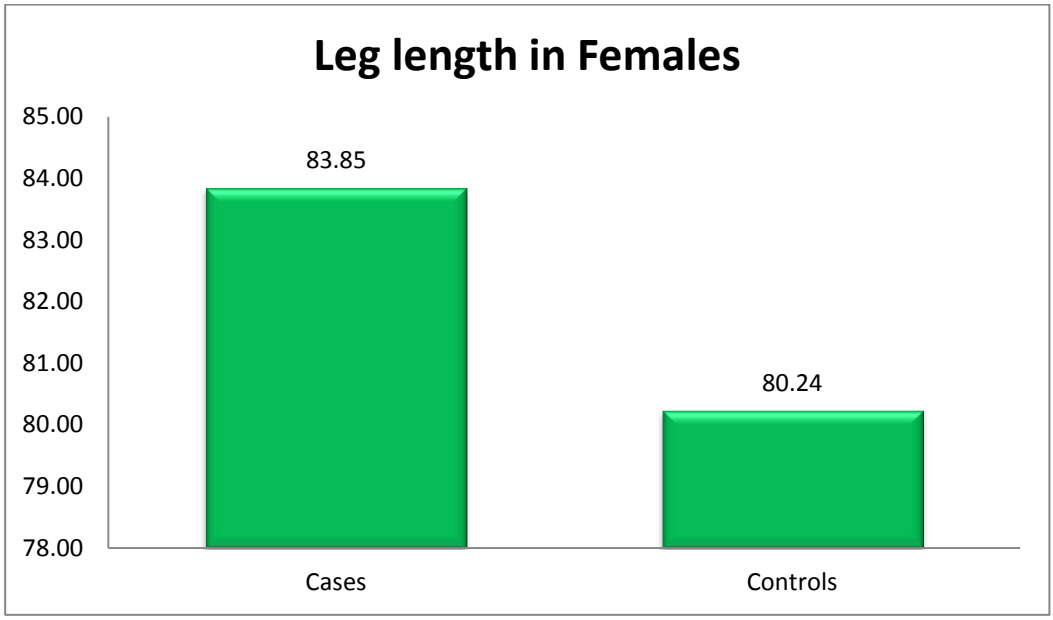
In this study group study female controls are maximum below the age group 40 yrs. they have 38%.male controls are also maximum below the age group of 40yrs, they have 44.8%.


100%
90%
80%
70%
60%
50%
40%
30%
20%
10%
0%
68.7%
23.9%
7.5%
35.8%
25.4%
38.8%
64.2%
29.9%
6.0%
25.4%
29.9%
44.8%
Cases
Controls
Cases
Controls
Female
Male
Upto 40 yrs
41 - 50 yrs
51 - 60 yrs



In our study females mean arm length in case group are 66.51 cm (SD 4.7).

Mean arm length in control group are 68.48 cm(SD 3.2)

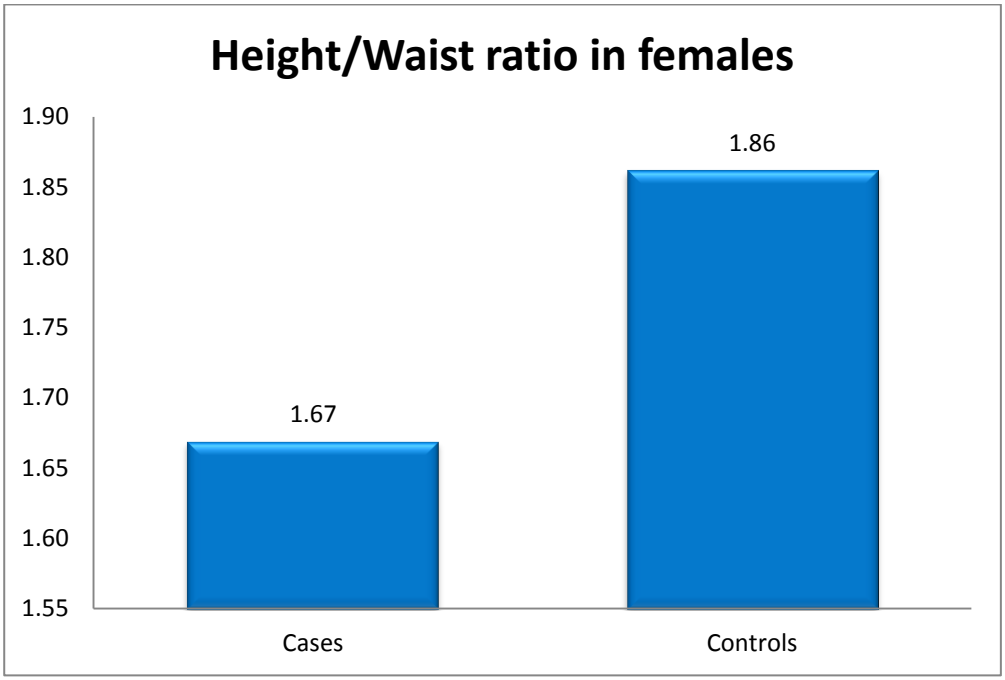


In this study shows female case group mean leg length are 83.85cm (SD 6.9).

female control group mean leg length are 80.24 cm(SD 7.0)

Study group: female

Group Statistics[a]				
EC		Mean	Std. Deviation	Std. Error Mean
ARM LENGTH	Cases	66.51	4.711	.576
	Controls	68.48	3.254	.398
LEG LENGTH	Cases	83.85	6.931	.847
	Controls	80.24	7.069	.864
HW RATIO	Cases	1.67	.227687	.027816
	Controls	1.86	.307209	.037532



In this study female case group have height/waist ratio showed 1.67(SD 0.22) and female control group have height/waist ratio 1.86 (SD 0.30)

Independent Samples Tests

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
ARM LENGTH	Equal variances assumed	.794	.375	-3.041	132	.003	-2.000	.658	-3.301	-.699
	Equal variances not assumed			-3.041	130.215	.003	-2.000	.658	-3.301	-.699
LEG LENGTH	Equal variances assumed	8.152	.005	3.895	132	.000	4.284	1.100	2.108	6.459
	Equal variances not assumed			3.895	120.089	.000	4.284	1.100	2.106	6.461
HW RATIO	Equal variances assumed	5.440	.021	-4.836	132	.000	-.205009	.042391	-.288863	-.121155
	Equal variances not assumed			-4.836	123.278	.000	-.205009	.042391	-.288918	-.121100

In this study in the female group , the difference in arm length between cases and control was statistically significant (p value 0.003)

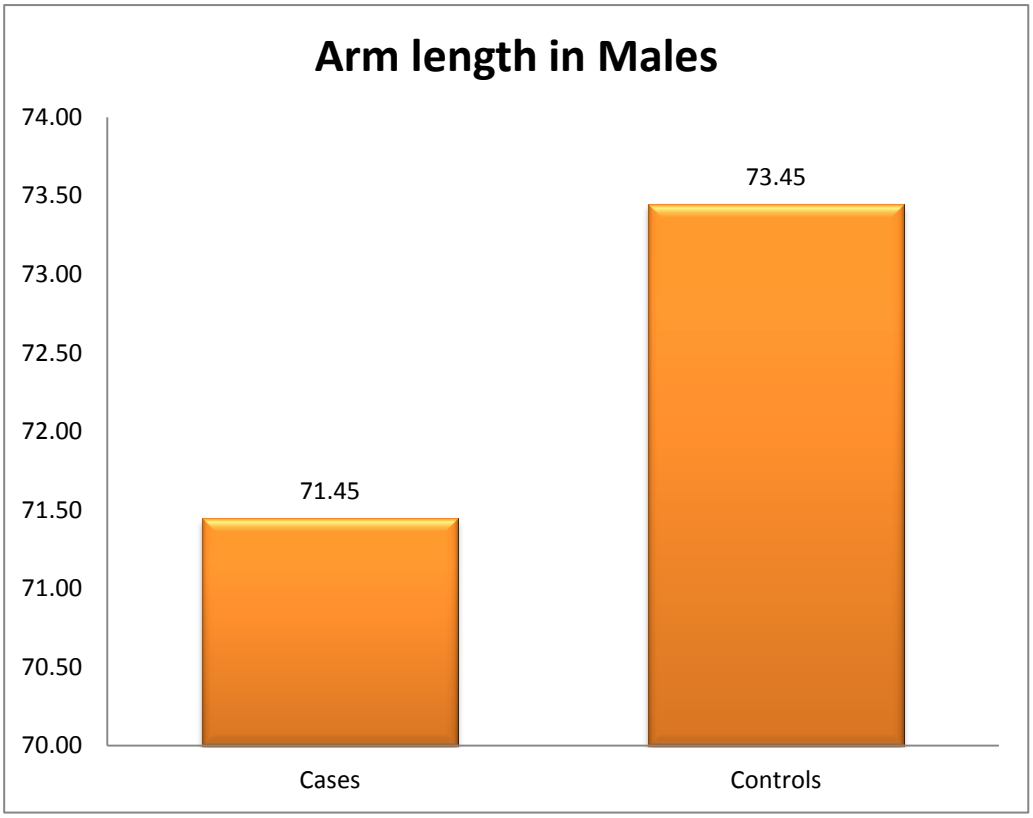
In this study in the female group , the difference in height/waist ratio between cases and control was statistically significant (p value 0.000)

Independent Samples Test[a]

		Levene's Test for Equality of Variances		t-test for Equality of Means						
		F	Sig.	t	df	Sig. (2-tailed)	Mean Difference	Std. Error Difference	95% Confidence Interval of the Difference	
									Lower	Upper
ARM LENGTH	Equal variances assumed	3.317	.071	-2.817	132	.006	-1.970	.699	-3.354	-.587
	Equal variances not assumed			-2.817	117.295	.006	-1.970	.699	-3.355	-.585
LEG LENGTH	Equal variances assumed	.196	.658	2.986	132	.003	3.612	1.209	1.219	6.004
	Equal variances not assumed			2.986	131.949	.003	3.612	1.209	1.219	6.004
HW RATIO	Equal variances assumed	6.042	.015	-4.122	132	.000	-.192579	.046716	-.284988	-.100171
	Equal variances not assumed			-4.122	121.701	.000	-.192579	.046716	-.285060	-.100099

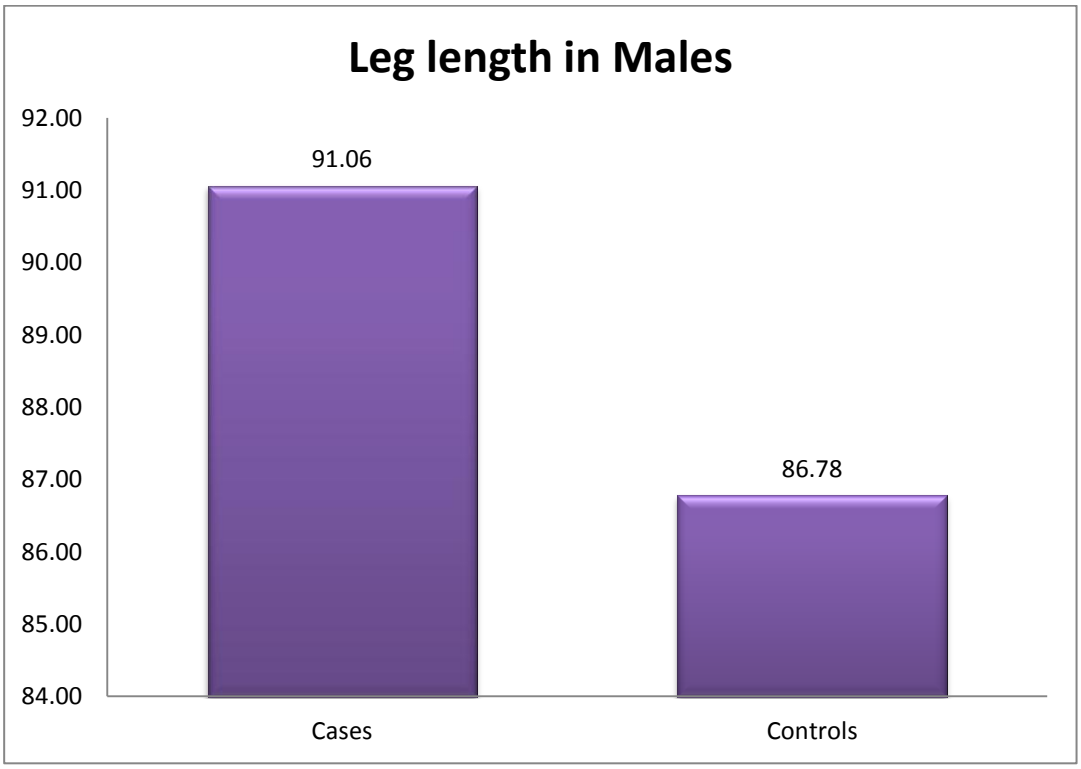
In this study in the male group , the difference in arm length between cases and control was statistically significant (p value 0.006)

In this study in the male group , the difference in height/waist ratio between cases and control was statistically significant (p value 0.000)

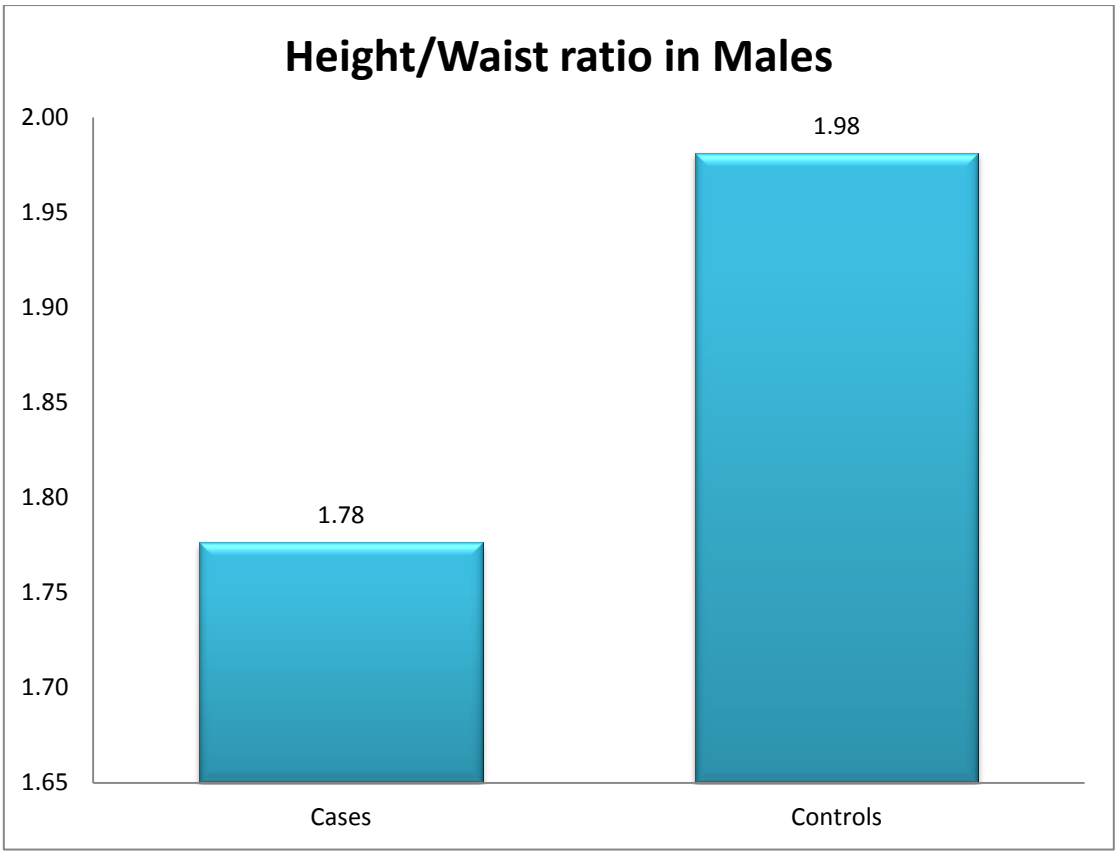


In our study males mean arm length in case group are 71.45 cm (SD 4.02). Mean arm length in control group are 73.45 cm(SD 3.5) Study group: male

EC		N	Mean	Std. Deviation	Std. Error Mean
ARM LENGTH	Cases	67	71.45	4.024	.492
	Controls	67	73.45	3.577	.437
LEG LENGTH	Cases	67	91.06	7.299	.892
	Controls	67	86.78	5.268	.644
HW RATIO	Cases	67	1.78	.210209	.025681
	Controls	67	1.98	.276065	.033727



In this study shows male case group mean leg length are 91.06cm (SD 7.2). male control group mean leg length are 86.78 cm(SD 5.2)

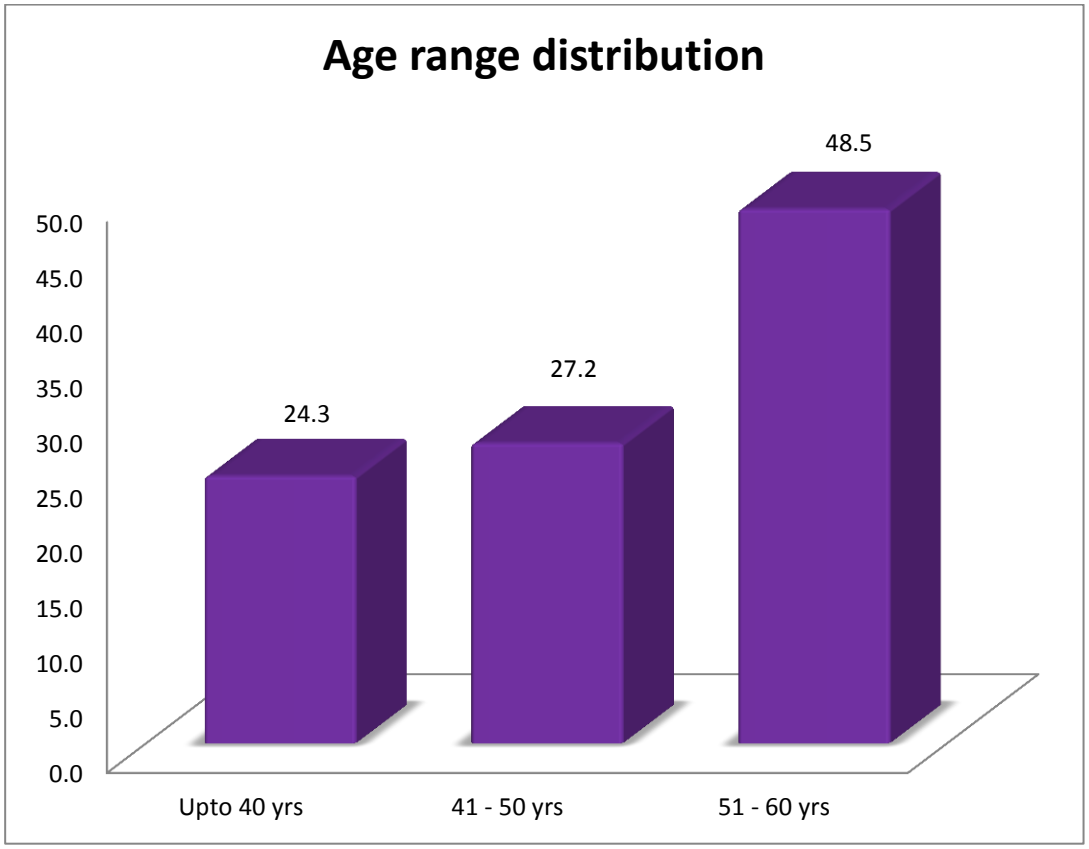


In this study male case group have height/waist ratio showed 1.78 (SD 0.21) and male control group have height/waist ratio 1.98 (SD 0.27)

Descriptive Statistics[a] : FEMALE					
	N	Minimum	Maximum	Mean	Std. Deviation
AGE	134	30	60	49.65	9.586
HT	134	130	175	154.39	9.018
WT	134	36	108	62.04	12.077
WC	134	62	119	89.28	12.393
HC	134	67	126	95.90	12.713
Valid N (listwise)	134				

Descriptive Statistics[a:] : MALE					
	N	Minimum	Maximum	Mean	Std. Deviation
AGE	134	30	60	48.41	9.755
HT	134	150	186	165.37	5.775
WT	134	38	107	64.43	12.824
WC	134	66	122	89.81	13.352
HC	134	66	120	89.42	11.216
Valid N (listwise)	134				

BOTH MALE AND FEMALE



In this study , participation of both cases and control group in both sexes , 24.3% belong to age group 31-40 yrs. 27.2% belong to age group 41-50 yrs.48.5% belong to age group of 51-60 yrs.

Age range: both male and female					
		Frequency	Percent	Valid Percent	Cumulative Percent
Valid	Upto 40 yrs	65	24.3	24.3	24.3
	41 - 50 yrs	73	27.2	27.2	51.5
	51 - 60 yrs	130	48.5	48.5	100.0
	Total	268	100.0	100.0	

DISCUSSION

DISCUSSION

In our study, the maximum number of female cases belonged to the age group 51-60 yrs and that was about 68.7 %. The maximum number of male cases belonged to age group 51-60 yrs and that was about 64.2%.

In our study female controls were maximum below the age group of 40 yrs.And Was about 38% . male controls were also maximum below the age group of 40yrs, about 44.8%.

In our study females mean arm length in cases are 66.51 cm (SD 4.7). Mean arm length in controls are 68.48 cm(SD 3.2).

This study shows female case groups mean leg length are 83.85cm (SD 6.9). female controls mean leg length are 80.24 cm(SD 7.0).

In this study female case group have height/waist ratio showed 1.67(SD 0.22) and female control have height/waist ratio 1.86 (SD 0.30).

In this study in the female group , the difference in arm length between cases and control was statistically significant (p value 0.003)

In this study in the female group , the difference in height/waist ratio between cases and control was statistically significant (p value 0.000)

In this study in the male group , the difference in arm length between cases and control was statistically significant (p value 0.006)

In this study in the male group , the difference in height/waist ratio between cases and control was statistically significant (p value 0.000).

In our study males mean arm length in cases are 71.45 cm (SD 4.02). Mean arm length in controls are 73.45 cm(SD 3.5).

In this study shows male cases mean leg length are 91.06cm (SD 7.2). male controls mean leg length are 86.78 cm(SD 5.2).

In this study male case group have height/waist ratio showed 1.78(SD 0.21) and male control have height/waist ratio 1.98 (SD 0.27).

In this study , participation of both cases and control group in male & females , 24.3% belong to age group 31-40 yrs. 27.2% belong to age group 41-50 yrs.48.5% belong to age group of 51-60 yrs.

In our study total arm length in female diabetic patients having lower than normal female people in the age group of 30-60 years. Which correlates with previous study done by m.m. smith et al[42].

In our study total arm length in male diabetic patients having lower than normal male people in the age group of 30-60 years. Which correlates with previous study done by m.m. smith et al[42].

In our study total leg length in female diabetic patients having higher than normal female people in the age group of 30-60 years. Which correlates with previous study done by m.m. smith et al[42].

In our study total leg length in male diabetic patients having higher than normal male people in the age group of 30-60 years. this result is conforms with previous study done by m.m. smith et al[42].

In our study compared height waist ratio in female diabetic patients and control groups. Female diabetic patients having lower height waist ratio than control group.

- In our study compared height waist ratio in male diabetic patients and control groups. male diabetic patients having lower height waist ratio than control group. IUGR babies , poor nutrition in early childhood period are more prone for obesity, insulin resistance in adult period . This leads to type 2 diabetes mellitus in these people more commonly than normal people.

- Preventing IUGR births by providing good AN care, also avoiding under nutrition in early childhood period helps in achieving the main goal to prevent type 2 diabetes in this group of people.

- The main causes of IUGR in India is maternal anemia, UTI, PIH, antenatal hemorrhage . Among these maternal anemia is the main cause of anemia in India.

- Correction of anemia is the important aspect to prevent IUGR not only in pregnant females, but also in all the females in child bearing age group (21-35).

- Correction of anemia in pregnant females can be done by early registration of pregnancy in nearby health centre, which is provided by Govt of india or private health sector. Periodical follow up of blood hemoglobulin, weight chart, BP monitoring, USG for assessing fetal weight by measuring abdominal circumference and femur length, has also a role.

- If anemia is detected early in pregnant female as the diagnostic criteria provided by Govt of india , patient treated with iron tablets, I/V iron sucrose and blood transfusion as guide lines.

- poor nutrition in early childhood period is prevented by giving health education to parents and frequent weight monitoring during immunization visit. weight is monitored by marking weight in growth chart in immunization card , which is provided by Govt of india.

CONCLUSION

CONCLUSION:

- Total arm length has inverse relationship with type 2 diabetes mellitus. It is observed in this study for both sexes individually. .

- Height waist ratio has also inverse relationship with type 2 diabetes mellitus patients. Diabetes patients having lower height waist ratio , compared with normal people. This is observed in this study both male and female individually.

- Total leg length has positive relationship with type 2 diabetes mellitus patients. Type 2 diabetes mellitus patients having higher leg length compared to normal people. The above is confirmed in this study both male & female separately.

SUMMARY

Diabetes mellitus is the major non communicable disease in the world. India has second highest number of diabetics after china . It has been observed over time that some ethic groups are more predisposed to diabetes than others. This can be explained by “thirfty gene hypothesis” proposed by geneticist james v.neel in 1962.This explains IUGR babies and poor nutrition in early childhood leads to obesity, insulin resistance in adult period . This people are more prone for type 2 diabetes mellitus . This people have short armlength compare to normal person. So our aim of the study is relationship between type 2 diabets mellitus and height waist ratio in relation diabetes . sample calculated from previous study incidence. 67 case and controls taken both males females separately. Final conclusion of our case control study is armlength is inversely proportional to type 2 diabetes mellitus. Height waist ratio is also inversely proportional to type 2 diabetes mellitus. In this result correlate with previous study. Based on this result pregnant females in india improve nutritional status to prevent IUGR babies which lead to type 2 diabetes in future.

BIBLIOGRAPHY

BIBLIOGRAPHY

1. Chakravarthy MV and Booth FW. 2004. Eating, exercise, and "thrifty" genotypes: connecting the dots to- ward an evolutionary understanding of modern chronic diseases. *J ApplPhysiol, 96: 3-10.*

2. Hales CN and Barker DJP. Type 2 (non-insulin dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologica. 1992; 35: 595-601.

3. Watve MG, Yajnik CS. "Evolutionary origins of insulin resistance: a behavioral switch hypothesis". BMC Evol. Biol. 2007; **7**: 61

4. D. J. P. Barker, "Adult consequences of fetal growth restriction," *Clinical Obstetrics and Gynecology*, vol. 49, no. 2, pp. 270–283, 2006

5. D. J. P. Barker, "In utero programming of cardiovascular disease," *Theriogenology*, vol. 53, no. 2, pp. 555–574, 2000.

6. P. Bateson, D. Barker, T. Clutton-Brock et al., "Developmental plasticity and human health," *Nature*, vol. 430, no. 6998, pp. 419–421, 2004.

7. C. Osmond, E. Kajantie, T. J. Fors´en, J. G. Eriksson, and D. J. P. Barker, "Infant growth and stroke in adult life: the Helsinki birth cohort study," *Stroke*, vol. 38, no. 2, pp. 264–270, 2007.

8. H. A. De Boo and J. E. Harding, “The developmental origins of adult disease (Barker) hypothesis,” *Australian and New Zealand Journal of Obstetrics and Gynaecology*, vol. 46, no. 1, pp. 4–14, 2006.

9. I. C. McMillen and J. S. Robinson, “Developmental origins of the metabolic syndrome: prediction, plasticity, and programming,” *Physiological Reviews*, vol. 85, no. 2, pp. 571–633, 2005.

10. C. J. Stocker andM. A. Cawthorne, “The influence of leptin on early life programming of obesity,” *Trends in Biotechnology*, vol. 26, no. 10, pp. 545–551, 2008.

11. D. D. Briana and A. Malamitsi-Puchner, “Intrauterine growth restriction and adult disease: the role of adipocytokines,” *European Journal of Endocrinology*, vol. 160, no. 3, pp. 337– 347, 2009.

12. S. C. Langley-Evans, “Nutritional programming of disease: unravelling the mechanism,” *Journal of Anatomy*, vol. 215, no.1, pp. 36–51, 2009.

13. C. S. Yajnik, “Nutrient-mediated teratogenesis and fuelmediated teratogenesis: two pathways of intrauterine programming of diabetes,” *International Journal of Gynecology and Obstetrics*, vol. 104, supplement, pp. S27–S31, 2009

14. C. N. Hales and D. J.P. Barker, “Type 2 (non-insulindependent) diabetes mellitus: the thrifty phenotype hypothesis,” *Diabetologia*, vol. 35, no. 7, pp. 595–601, 1992.

15. D. J. P. Barker, J. G. Eriksson, T. Fors´en, and C. Osmond, “Fetal origins of adult disease: strength of effects and biological basis,” *International Journal of Epidemiology*, vol. 31, no. 6, pp. 1235–1239, 2002.

16. Fowden AL. The role of insulin in prenatal growth. J Dev Physiol 12:173-182 (1989).

17. Mitchell BD, Valdez R, Hazuda HP, Haffner SM, Monterrosa A, Stern MP. Differences in prevalence of diabetes and impaired glucose tolerance according to maternal or paternal history of diabetes. Diabetes Care 16:1262-1267 (1993).

18. Osmond C, Barker DJP, Winter PD, Fall CHD, Simmonds SJ. Early growth and death from cardiovascular disease in women. Br Med J 307:1519-1524 (1993).

19. Hales CN, Barker DJP, Clark PMS, Cox LJ, Fall C, Osmond C, Winter PD. Fetal and infant growth and impaired glucose tolerance at age 64. Br Med J 303:1019-1022 (1991).

20. Fall CHD, Osmond C, Barker DJP, Clark PMS, Hales CN, Stirling Y, Meade TW. Fetal and infant growth and cardiovascular risk factors in women. Br Med J 310:428-432 (1995).

21. Phipps K, Barker DJP, Hales CN, Fall CHD, Osmond C, Clark PMS. Fetal growth and impaired glucose tolerance in men and women. Diabetologia 36:225-228 (1993).

22. Lithell HO, McKeigue PM, Bergland L, Mohsen B, Lithell UB, Leon DA. Relation of size at birth to non-insulin dependent diabetes and insulin concentrations in men aged 50-60 years. Br Med J 312:406-410 (1996).

23. Olah KS. Low matemal birthweight - an association with impaired glucose tolerance in pregnancy. J Obstet Gynaecol 16:5-8 (1996).

24. Curhan GC, Willett WC, Rimm EB, Stampfer MJ. Birth weight and adult hypertension and diabetes mellitus in US men [Abstract]. Am J Hypertens 9:11 A (1996)

25. McCance DR, Pettitt DJ, Hanson RL, Jacobsson LTH, Knowler WC, Bennett PH. Birth weight and non-insulin dependent diabetes: thrifty genotype, thrifty phenotype, or surviving small baby genotype? Br Med J 308:942-945 (1994).

26. DeFronzo RA. The triumvirate: beta cell, muscle, liver. A collusion responsible for NIDDM. Diabetes 1988;37:667-687.

27. Barker DJP, Hales CN, Fall CHD, Osmond C, Phipps K, Clark PMS. Type 2 (non-insulin-dependent) diabetes mellitus, hypertension and hyperlipidaemia (syndrome X): relation to reduced fetal growth. Diabetologia 36:62-67 (1993).

28. Phillips DIW, Hirst S, Clark PMS, Hales CN, Osmond C. Fetal growth and insulin secretion in adult life. Diabetologia 37:592-596 (1994).

29. Law CM, Gordon GS, Shiell AW, Barker DJP, Hales CN. Thinness at birth and glucose tolerance in seven year old children. Diabet Med 12:24-29 (1995)

30. Whincup PH, Cook DG, Adshead F, Taylor SJC, Walker M, Papacosta D, Alberti KGMM. Childhood size is more strongly related than size at birth to glucose and insulin levels in 10-11- year-old children. Diabetologia 40:319-326 (1997).

31. Yajnik CS, Fall CHD, Vaidya U, Pandit AN, Bavdekar A, Bhat DS, Osmond C, Hales CN, Barker DJP. Fetal growth and glucose and insulin metabolism in four-year-old Indian children. Diabet Med 12:330-336 (1995).

32. Forrester TE, Wilks RJ, Bennett Fl, Simeon D, Osmond C, Allen M, Chung AP, Scott P. Fetal growth and cardiovascular risk factors in Jamaican schoolchildren. Br Med J 312:156-160 (1996).

33. Robinson SM, Wheeler T, Hayes MC, Barker DJP, Osmond C. Fetal heart rate and intrauterine growth. Br J Obstet Gynaecol 98:1223-1227 (1996).

34. Taylor DJ, Thompson CH, Kemp GJ, Barnes PRJ, Sanderson AL, Radda GK, Phillips DIW. A relationship between impaired fetal growth and reduced muscle glycolysis revealed by 31p magnetic resonance spectroscopy. Diabetologia 38:1205-1212 (1995).

35. Bjorntorp P. Insulin resistance: the consequence of a neuroendocrine disturbance? Int J Obesity 19 (suppl 1:S6-S10 (1995) 36.Van Assche FA, Aerts L. The fetal endocrine pancreas. Contrib Gynecol Obstet 5:44-57 (1979).

37. Hellerstrom C, Swenne I, Andersson A. Islet cell replication and diabetes. In: The pathology of the endocrine pancreas in diabetes (Lefebvre PJ, Pipeleers DG, eds.) Heidelberg:Springer, 3888;141-170.

38. Hales CN, Barker DJP. Type 2 (non-insulin-dependent) diabetes mellitus: the thrifty phenotype hypothesis. Diabetologia 35:595-601 (1992).

39. Fall CHD, Stein CE, Kumaran K, Cox V, Osmond C, Barker DJP, Hales CN. Size at birth, maternal weight, and non-insulin dependent diabetes in South India. Diabet Med 15:220-227 (1998).

40. Mather HM, Keen H. The Southall diabetes survey: prevalence of known diabetes in Asians and Europeans. Br Med J 291:1081-1084 (1985).

41. McKeigue PM, Shah B, Marmot MG. Relation of central obesity and insulin resistance with high diabetes prevalence and cardiovascular risk in South Asians. Lancet 337:382-386 (1991).

42. Smits, M. M., et al. "Arm length is associated with type 2 diabetes mellitus in Japanese-Americans." *Diabetologia* 55.6 (2012): 1679-1684.

ANNEXURE

INSTITUTIONAL ETHICS COMMITTEE
GOVT.KILPAUK MEDICAL COLLEGE,
CHENNAI-10
Protocol ID. No. 11/2016 Dt: 20.06.2016
CERTIFICATE OF APPROVAL

The Institutional Ethical Committee of Govt. Kilpauk Medical College, Chennai reviewed and discussed the application for approval "A CASE CONTROL STUDY OF RELAIONSHIPBETWEEN ARMLENGTH AND TYPE 2 DIABETES MELLITUS" - For Project Work submitted by Dr.P.Elayaraja, Post Graduate in Internal Medicine, Govt. Kilpauk Medical College, Chennai-10.

The Proposal is APPROVED.

The Institutional Ethical Committee expects to be informed about the progress of the study any Adverse Drug Reaction Occurring in the Course of the study any change in the protocol and patient information /informed consent and asks to be provided a copy of the final report.

DEAN
Govt.Kilpauk Medical College,
Chennai – 10.

PROFORMA

1. Name: 2. Age: 3. Sex:

4. Occupation: 5. Education:

6. History of smoking: 7. Consumption of alcohol:

8. Chief Complaint:

9. Past History:

10. History of Diabetes: Yes / No

If Diabetic:

11. Duration: 12. Family History:

13. Treatment:

Investigations:

14. FBS: 15. PPBS:

Anthropometry:

17. Height:

18. Weight:

19. Body Mass Index:

20. Total arm length

21. Upper arm:

22. Forearm:

23. Mid-arm circumference:

24. Total lower limb length:

25. Upper leg length:

26. Lower leg length

27. Hip circumference

28. Waist circumference:

PATIENT CONSENT FORM

Study detail : "Relation between arm length and Type2 Diabetes Mellitus, a case control study at a tertiary care hospital in Chennai"

Study centre : GRH, KILPAUK MEDICAL COLLEGE, CHENNAI

Patients Name :

Patients Age :

Identification Number :

Patient may check (✓) these boxes

I confirm that I have understood the purpose of procedure for the above study. I have the opportunity to ask question and all my questions and doubts have been answered to my complete satisfaction. ☐

I understand that my participation in the study is voluntary and that I am free to withdraw at any time without giving reason, without my legal rights being affected. ☐

I understand that sponsor of the clinical study, others working on the sponsor's behalf, the ethical committee and the regulatory authorities will not need my permission to look at my health records, both in ☐

respect of current study and any further research that may be conducted in relation to it, even if I withdraw from the study I agree to this access. However, I understand that my identity will not be revealed in any information released to third parties or published, unless as required under the law. I agree not to restrict the use of any data or results that arise from this study. ☐

I agree to take part in the above study and to comply with the instructions given during the study and faithfully cooperate with the study team and to immediately inform the study staff if I suffer from any deterioration in my health or well-being or any unexpected or unusual symptoms. ☐

I hereby consent to participate in this study.

I hereby give permission to undergo complete clinical examination and diagnostic tests including hematological, biochemical, radiological tests. ☐

Signature/thumb impression:

Patients Name and Address: place date

Signature of investigator :

Study investigator's Name : place date

சுயஒப்புதல் படிவம்

ஆய்வு செய்யப்படும் தலைப்பு: கை நீளம் மற்றும் வகை 2

நீரிழிவு நோய் இடையே உள்ள உறவு.

இடம்: பொது மருத்துவத்துவ துறை.
இராயப்பேட்டை மருத்துவமனை
அரசு கீழ்பாக்கம் மருத்துவ கல்லூரி மருத்துவமனை.
சென்னை

பங்குபெறுபவரின் பெயர் :

பங்குபெறுபவரின் வயது : **பங்குபெறுபவரின் எண் :**

மேலே குறிப்பிட்டுள்ள மருத்துவ ஆய்வின் விவரங்கள் எனக்கு ☐ விளக்கப்பட்டது. நான் இவ்வாய்வில் தன்னிச்சையாக பங்கேற்கிறேன். எந்த காரணத்தினாலோ எந்த சட்டசிக்கலுக்கும் உட்படாமல் நான் இவ்வாய்வில் இருந்து விலகிக்கொள்ளல்லாம் என்றும் அறிந்து கொண்டேன்.

இந்த ஆய்வு சம்பந்தமாகவோ, இதை சார்ந்து மேலும் ஆய்வு ☐ மேற்கொள்ளும்போதும் இந்த ஆய்வில்பங்கு பெறும் மருத்துவர் என்னுடைய மருத்துவ அறிக்கைகளை பார்ப்பதற்கு என் அனுமதி தேவையில்லை என அறிந்து கொள்கிறேன். இந்த

ஆய்வின் மூலம் கிடைக்கும் தகவலையோ, முடிவையோ பயன்படுத்திக்கொள்ள மறுக்க மாட்டேன்.

இந்த ஆய்வில் பங்கு கொள்ள ஒப்புக்கொள்கிறேன். இந்த ☐ ஆய்வை மேற்கொள்ளும் மருத்துவ அணிக்கு உண்மையுடன் இருப்பேன் என்றும் உறுதியளிக்கிறேன்.

பங்கேற்பவரின் கையொப்பம் ஆய்வாளரின் கையொப்பம்

இடம் :

தேதி :

ஆராய்ச்சி தகவல் தாள்

சென்னை இராயபேட்டை அரசு மருத்துவமனையில் நீரிழிவு நோய் குறித்த ஆராய்ச்சி நடைபெற்று வருகிறது.

நீரிழிவு நோய் வகை 2 உள்ளவர்களுக்கு கை நீளம் குறைவாக உள்ளது. இது நோய் இல்லாதவறை விடவும் சற்று குறைவாக உள்ளது. இதற்கு காரணம் அந்த நபர்கள் கருவில் உள்ளபோதோ அல்லது பிறந்த இரண்டு வருடங்களில் உணவு பற்றாகுறையாக இருந்தாலோ அவர்கள் எதிர்காலத்தில் உடல் பருமன் வந்து நீரிழிவு நோய் வகை 2 ஏற்பட வாய்ப்பு உண்டு. அவர்களுக்கு எலும்பு வளர்ச்சி குறைவாக இருப்ப்தால் கை நீளம் குறைவாக உள்ளது.

ஆகவே பெண்கள் கர்ப்பமாக இருக்கும்போது உணவு பற்றாகுறை இல்லாமல், ஆரோக்கியமான உணவுடன் இருந்தால் ஆரோக்கியமான குழந்தை பெற்று எதிர்காலத்தில் நீரிழிவு நோய் வருவதை குறைப்பதே இந்த ஆராய்ச்சியின் நோக்கமாகும்.

நீங்களும் இந்த ஆராய்ச்சியில் பங்கேற்க நாங்கள் விரும்புகிறோம். முடிவுகளை அல்லது கருத்துக்களை வெளியிடும் போதோ அல்லது ஆராய்ச்சியின் போதோ தங்களது பெயரை வெளியிடமாட்டோம் என்பதை தெரிவித்து கொள்கிறோம்.

இந்த ஆராய்ச்சியில் பங்கேற்பது தங்களுடைய விருப்பத்தின் பேரில் தான் இருக்கிறது. மேலும் நீங்கள் எந்நேரமும் இந்த ஆராய்ச்சியிலிருந்து விலகிக்கொள்ளலாம் என்பதையும் தெரிவித்துக்கொள்கிறோம். இந்த சிறப்பு பரிசோதனையின் முடிவுகளை ஆராய்ச்சியின் போது அல்லது ஆராய்ச்சியின் முடிவில் தங்களுக்கு அறிவிப்போம் என்பதையும் தெரிவித்து கொள்கிறோம்.

ஆராய்ச்சியாளர் கையொப்பம்

பங்கேற்பாளர் கையொப்பம்

தேதி:

MALE PATIENTS

S. NO	NAME	AGE	SEX	ARM LENGTH (cms)	HEIGHT (cms)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cms)	HIP CIRCUM FERENCE (cms)	LEG LENGTH (cms)	HEIGHT/ WAIST RATIO
1	AYYANATHAN	60	M	69	160	58	90	81	84	1.777777778
2	ARUL	40	M	68	155	38	66	66	80	2.348484848
3	VASUDEVVAN	45	M	70	167	55	79	80	86	2.113924051
4	RAJENDRAN	60	M	71	160	48	76	74	76	2.105263158
5	SANJEEVI	48	M	64	160	55	80	79	78	2
6	KARUPAIYA	60	M	71	170	55	83	82	80	2.048192771
7	ARUMUGAM	58	M	66	155	45	72	76	78	2.152777778
8	KUMAR	40	M	61	150	45	78	74	76	1.923076923
9	MUTHU	53	M	69	168	62	85	83	86	1.976470588
10	BOJAN	35	M	69	165	55	78	77	88	2.115384615
11	MUJAFIR AHMED	50	M	62	162	65	88	83	84	1.840909091
12	VELU	56	M	68	160	64	86	88	84	1.860465116
13	RAJAMANI	58	M	65	157	83	104	102	78	1.509615385
14	MURUGAN	55	M	77	163	84	112	106	83	1.455357143
15	PRABAKARAN	59	M	73	168	57	76	78	90	2.210526316
16	MUTHU	60	M	67	160	74	108	106	92	1.481481481
17	KANNIYAPPAN	47	M	81	185	86	102	100	98	1.81372549
18	SEBASTIN	60	M	75	168	58	84	82	93	2
19	MUTHURASU	55	M	74	160	63	87	86	100	1.83908046
20	JAYAKUMAR	60	M	70	161	56	87	80	89	1.850574713
21	SENTHAMARAI	60	M	73	165	55	88	85	92	1.875

S. NO	NAME	AGE	SEX	ARM LENGTH (cms)	HEIGHT (cms)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cms)	HIP CIRCUM FERENCE (cms)	LEG LENGTH (cms)	HEIGHT/ WAIST RATIO
22	KASI	60	M	71	162	62	97	92	80	1.670103093
23	KUMAR	57	M	72	160	56	89	86	98	1.797752809
24	MURUGAN	45	M	72	164	63	89	86	92	1.842696629
25	SADASIVAM	59	M	74	165	69	110	100	94	1.5
26	NARAYANAN	49	M	72	174	107	122	118	102	1.426229508
27	PRABAKAR	57	M	75	174	86	106	106	100	1.641509434
28	SURAMANI	60	M	79	180	74	93	93	104	1.935483871
29	PERUMAL	58	M	72	165	57	86	84	92	1.918604651
30	SAMUVEL	58	M	74	172	82	102	100	100	1.68627451
31	GANESAN	60	M	70	170	76	103	99	94	1.650485437
32	MARIYAPPAN	48	M	74	170	94	120	112	100	1.416666667
33	SIVAKUMAR	50	M	72	164	60	88	87	94	1.863636364
34	GEORGE	46	M	77	173	91	121	108	93	1.429752066
35	SEKAR	49	M	72	170	74	99	94	98	1.717171717
36	VENKATARAMAN	59	M	72	170	55	88	89	95	1.931818182
37	SAMUVEL	60	M	69	164	60	94	93	94	1.744680851
38	ARUMUGAM	56	M	76	172	58	87	84	93	1.977011494
39	LOGANATHAN	59	M	79	170	59	81	87	103	2.098765432
40	CHOKALINGAM	60	M	72	160	62	93	91	93	1.720430108
41	CHANDRAN	48	M	72	160	78	102	108	95	1.568627451
42	RAJENDRAN	60	M	69	168	74	101	100	98	1.663366337
43	KUMAR	49	M	76	178	77	94	94	107	1.893617021
44	SAFEED AHMED	59	M	71	163	65	100	95	94	1.63

S. NO	NAME	AGE	SEX	ARM LENGTH (cms)	HEIGHT (cms)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cms)	HIP CIRCUM FERENCE (cms)	LEG LENGTH (cms)	HEIGHT/ WAIST RATIO
45	TAMEEM BASHA	37	M	73	165	87	97	94	91	1.701030928
47	VASUDEVAN	51	M	71	165	69	100	97	88	1.65
48	VINOTH KUMAR	49	M	63	158	64	93	93	86	1.698924731
49	ARUMUGAM	57	M	74	168	65	102	96	91	1.647058824
50	CHANDRAKUMAR	59	M	74	169	70	102	98	93	1.656862745
51	SIVAGURUNATHAN	55	M	72	158	50	86	84	87	1.837209302
52	KABALI	54	M	69	171	95	118	112	92	1.449152542
53	MANIVEL	49	M	77	173	61	97	90	93	1.783505155
54	THIYAGARAJA	50	M	65	157	52	84	83	84	1.869047619
55	PONNUVELU	60	M	71	162	70	104	97	85	1.557692308
56	CHELLAMUTHU	46	M	63	162	58	94	98	84	1.723404255
57	CHANDRAKUMAR	58	M	74	169	70	102	98	93	1.656862745
58	MUTHURASU	55	M	74	160	63	87	86	100	1.83908046
59	JAYAKUMAR	60	M	70	161	56	87	80	89	1.850574713
60	SENTHAMARAI	58	M	73	165	55	88	85	92	1.875
61	KASI	60	M	71	162	62	97	92	80	1.670103093
62	KUMAR	57	M	72	160	56	89	86	98	1.797752809
63	GANESAN	60	M	70	170	76	103	99	94	1.650485437
64	MARIYAPPAN	48	M	74	170	94	120	112	100	1.416666667
65	SIVAKUMAR	50	M	72	164	60	88	87	94	1.863636364
66	GEORGE	46	M	77	173	91	121	108	93	1.429752066
67	SEKAR	49	M	72	170	74	99	94	98	1.717171717
	AVERAGE			**71.45454545**					**91.106061**	**1.778238573**

MALE –CONTROL

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
1	VENGADESH	50	M	79	178	52	73	79	94	2.438356164
2	ETTIYAPPAN	43	M	72	158	65	91	88	84	1.736263736
3	VINAYAGAM	48	M	71	158	48	73	84	86	2.164383562
4	PRADEEP	30	M	74	168	58	76	78	90	2.210526316
5	CHANDRAN	46	M	73	167	72	90	91	87	1.855555556
6	SAKTHIVEL	34	M	73	164	54	72	74	88	2.277777778
7	NAGARAJ	43	M	76	170	58	78	81	92	2.179487179
8	RANJITH	30	M	76	168	57	90	90	89	1.866666667
9	VIJAY	30	M	74	165	55	84	86	86	1.964285714
10	JEGADEESH	45	M	75	163	58	85	84	80	1.917647059
11	THANGARAJ	40	M	69	165	55	80	78	87	2.0625
12	RAVI	35	M	73	167	65	92	82	85	1.815217391
13	VIKRAM	30	M	72	160	48	69	71	84	2.31884058
14	RAJ	54	M	68	157	42	74	74	83	2.121621622
15	RAJARAM	41	M	72	168	83	100	92	92	1.68
16	KANNAN	32	M	81	186	86	96	92	100	1.9375
17	RAMESH	46	M	64	153	61	84	82	82	1.821428571
18	MEERAN	45	M	73	157	51	77	78	85	2.038961039
19	ROSAIAH	53	M	75	171	83	108	102	86	1.583333333
20	RAJAMANICKAM	48	M	73	168	52	72	75	90	2.333333333
21	JAMBU	56	M	74	163	72	67	70	86	2.432835821
22	DELIP	56	M	74	167	86	106	102	88	1.575471698

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
23	RADAKRISHNAN	54	M	77	167	60	77	79	89	2.168831169
24	MURUGAN	45	M	78	163	84	112	106	85	1.455357143
25	ARAVIND	57	M	77	171	59	78	84	87	2.192307692
26	VIJAYAKUMAR	36	M	69	157	52	83	81	80	1.891566265
27	ARUMUGAM	48	M	75	168	55	73	75	89	2.301369863
28	BALU	41	M	75	160	45	67	73	84	2.388059701
29	DURAI PANDY	56	M	74	159	55	77	83	78	2.064935065
30	RAJENDRAN	58	M	76	159	70	110	110	93	1.445454545
31	RAJASEKAR	60	M	75	165	58	81	84	99	2.037037037
32	VIJI	36	M	74	162	61	85	87	93	1.905882353
33	AHMED BASHA	56	M	75	165	56	80	86	87	2.0625
34	KALIYAN	56	M	74	166	74	107	104	96	1.551401869
35	KRISHNA	58	M	70	162	67	97	98	98	1.670103093
37	MANOJ	36	M	70	168	71	96	90	87	1.75
38	MURUGAN	32	M	73	163	74	110	120	88	1.481818182
39	SELVAN	35	M	69	162	47	74	84	80	2.189189189
40	RAJESH	43	M	68	163	67	82	89	79	1.987804878
41	RAFIQ	38	M	75	170	46	82	95	90	2.073170732
42	MOHAN	32	M	68	164	45	71	79	82	2.309859155
43	SUDAKAR	36	M	72	165	62	92	97	85	1.793478261
44	GOPAL	38	M	76	178	67	91	101	89	1.956043956
45	SRIRAM	35	M	79	172	75	90	102	82	1.911111111
46	KADHER	30	M	88	174	52	70	81	78	2.485714286
47	PAKEER	58	M	76	162	57	71	79	76	2.281690141

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
48	RAVI	32	M	71	164	62	80	89	78	2.05
49	PRAVEEN	35	M	73	172	75	85	92	88	2.023529412
50	TAMIL	48	M	76	164	59	86	89	79	1.906976744
51	RANJITH	30	M	76	168	57	90	90	80	1.866666667
52	VIJAY	30	M	73	165	55	84	86	87	1.964285714
53	JEGADEESH	45	M	72	163	58	85	84	86	1.917647059
54	THANGARAJ	40	M	69	165	55	80	78	86	2.0625
55	RAVI	35	M	73	167	65	92	82	90	1.815217391
56	RAJAMANICKAM	48	M	73	168	52	72	75	89	2.333333333
57	JAMBU	56	M	74	163	72	67	70	88	2.432835821
58	DELIP	56	M	73	167	86	106	102	89	1.575471698
59	RADAKRISHNAN	54	M	77	167	60	77	79	90	2.168831169
60	MURUGAN	45	M	78	163	84	112	106	88	1.455357143
61	KRISHNA	60	M	71	162	67	97	98	98	1.670103093
62	MANOJ	36	M	72	168	71	96	90	87	1.75
63	MURUGAN	32	M	72	163	74	110	120	88	1.481818182
64	SELVAN	35	M	69	162	47	74	84	80	2.189189189
65	RAJESH	43	M	68	163	67	82	89	79	1.987804878
66	CHANDRAN	46	M	73	167	72	90	91	90	1.855555556
67	SAKTHIVEL	34	M	72	164	54	72	74	88	2.277777778
	AVERAGE			**73.46969697**					**86.75758**	**1.976842116**

FEMALE –PATIENT

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (Kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
1	CHANDRALEKA	58	F	69	155	70	82	88	77	1.890243902
2	NAXEMA	56	F	59	140	59	73	84	75	1.917808219
3	VITTA	60	F	65	147	62	77	85	78	1.909090909
4	MEERA	48	F	65	157	75	90	110	80	1.744444444
5	SARASWATHY	40	F	64	154	70	96	100	79	1.604166667
6	KANNAMMAL	58	F	65	146	44	78	81	78	1.871794872
7	BANUMATHY	60	F	71	159	60	69	72	82	2.304347826
8	PADMA	55	F	63	148	63	90	93	78	1.644444444
9	ESWARI	43	F	67	160	55	66	69	84	2.424242424
10	JAGANAYAGI	36	F	63	140	90	119	122	75	1.176470588
11	RADHA	30	F	70	158	74	94	98	84	1.680851064
12	CHOKKAMMA	58	F	64	155	58	84	87	78	1.845238095
13	GOVINDAMMAL	60	F	67	150	59	84	90	76	1.785714286
14	GANTHI	48	F	62	152	61	82	85	79	1.853658537
15	MURUGESWARI	42	F	63	149	63	87	99	78	1.712643678
16	KURSHIT BEGAM	60	F	66	155	55	89	91	79	1.741573034
17	JEEVA	53	F	68	154	74	109	117	80	1.412844037
18	PANJALI	55	F	50	130	63	104	110	68	1.25
19	MALLIKA	55	F	70	161	56	83	88	85	1.939759036
20	AMMAKANNU	58	F	66	155	57	87	90	80	1.781609195
21	MUNIYAMMAL	59	F	72	165	50	84	90	89	1.964285714
22	ANJANA	60	F	69	158	60	73	77	85	2.164383562

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (Kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
23	DEVI	50	F	65	154	54	88	90	80	1.75
24	SARALA	52	F	66	157	62	94	103	85	1.670212766
25	BEEVI	50	F	64	153	60	95	100	80	1.610526316
26	NACHIYAR	45	F	67	155	84	106	117	84	1.462264151
27	LALITHA	56	F	64	152	60	96	101	80	1.583333333
28	SARASWATHY	60	F	66	155	57	97	101	92	1.597938144
29	CHANDRA	42	F	68	144	56	90	99	80	1.6
30	GAJALAKSHMI	47	F	77	158	65	97	104	99	1.628865979
31	MALARVIZHI	55	F	74	154	65	89	104	93	1.730337079
32	INDIRANI	47	F	67	150	63	93	105	89	1.612903226
33	LALITHA	60	F	74	155	54	89	104	93	1.741573034
34	AMARAVATHI	60	F	71	152	55	92	105	84	1.652173913
35	MAHALAKSHMI	57	F	64	138	50	95	101	79	1.452631579
36	VADIVELAMMAL	58	F	72	140	57	100	102	86	1.4
37	MALLIGA	60	F	66	144	68	95	103	97	1.515789474
38	MANIMEGALAI	45	F	61	137	46	83	91	76	1.65060241
39	PADMA	60	F	65	149	47	84	96	85	1.773809524
40	SAROJA	60	F	67	147	54	94	104	83	1.563829787
41	LAKSHMI	59	F	73	165	77	105	115	95	1.571428571
42	SHYAMALA	41	F	66	154	57	87	104	88	1.770114943
43	RAJESHWARI	58	F	68	150	60	97	115	88	1.546391753
44	NEELAVATHY	60	F	70	155	67	97	122	95	1.597938144
45	LILLY	60	F	66	155	54	84	103	89	1.845238095
46	KARPAGAM	47	F	73	160	60	103	102	102	1.553398058

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (Kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
47	NASIBEGAM	60	F	67	157	70	104	118	91	1.509615385
48	DEVIKA	53	F	73	160	74	98	122	99	1.632653061
49	KUPPAMMAL	60	F	71	155	59	92	102	86	1.684782609
50	LAKSHMI	60	F	65	152	56	93	104	91	1.634408602
52	MUNIYAMMAL	35	F	62	142	55	79	91	76	1.797468354
53	ANNAMMAL	32	F	67	152	60	106	90	84	1.433962264
54	GEETHA	60	F	69	167	50	87	75	90	1.91954023
55	JESINTHA	49	F	59	150	63	97	87	85	1.546391753
56	KALAIYARASI	52	F	69	162	72	92	85	85	1.760869565
57	VIJAYA	59	F	72	158	75	103	92	84	1.533980583
58	SASIKALA	58	F	73	162	65	108	100	86	1.5
59	KAMALA	58	F	67	158	57	90	95	86	1.755555556
60	VANAJA	59	F	66	155	47	76	86	89	2.039473684
61	VALLIYAMMAL	56	F	64	140	55	98	96	86	1.428571429
62	PUSHPA	60	F	63	141	70	112	114	85	1.258928571
63	JEEVA	53	F	68	154	74	109	117	85	1.412844037
64	PANJALI	55	F	50	130	63	104	110	67	1.25
65	BEEVI	50	F	64	153	60	95	100	79	1.610526316
66	NACHIYAR	45	F	67	155	84	106	117	79	1.462264151
67	LALITHA	56	F	64	152	60	96	101	78	1.583333333
	AVERAGE			**66.54545455**					**83.93939**	**1.670546732**

FEMALE –CONTROL

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
1	SAROJA	54	F	69	146	68	66	75	84	2.212121212
2	SELVI	43	F	63	149	64	65	69	80	2.292307692
3	VEERAMMAL	55	F	66	140	52	62	68	74	2.258064516
4	NOORIBEGAM	54	F	69	154	52	85	88	80	1.811764706
5	RANI	55	F	69	153	42	64	67	76	2.390625
6	DEVAKI	45	F	67	150	53	83	92	80	1.807228916
7	RENUKA	34	F	66	151	56	82	85	82	1.841463415
8	THANGAM	50	F	70	161	50	80	84	85	2.0125
9	SUBALAKSHMI	60	F	63	130	56	88	92	68	1.477272727
10	KARPAGAM	48	F	74	175	86	85	93	84	2.058823529
11	rajeswari	34	F	69	163	67	87	91	86	1.873563218
12	VIJAYA	40	F	70	155	62	88	91	83	1.761363636
13	SUNDARI	57	F	66	150	54	89	94	78	1.685393258
14	MEGALA	50	F	64	149	49	76	85	78	1.960526316
15	SHANMUGAVALLI	50	F	67	152	49	68	75	80	2.235294118
16	GANAPATHY	60	F	65	151	54	89	96	79	1.696629213
17	KANDIYAMMA	47	F	67	149	61	87	95	80	1.712643678
18	KALAIVANI	30	F	69	162	40	67	76	85	2.417910448
19	SAMSA	53	F	68	159	73	97	102	86	1.639175258
20	BHUVANESHWARI	35	F	58	132	36	76	79	70	1.736842105
21	SAIYALLAMA	60	F	69	153	62	94	101	76	1.627659574
22	SUBALAKSHMI	30	F	67	154	52	72	82	78	2.138888889

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
23	VILVASUDHA	35	F	73	170	65	87	92	80	1.954022989
24	KALA	58	F	68	165	40	65	77	89	2.538461538
25	UMARANI	42	F	72	158	97	118	122	84	1.338983051
26	BHAVANI	38	F	68	156	65	83	94	78	1.879518072
27	SHELLA	36	F	67	157	108	112	126	78	1.401785714
28	KALA	47	F	72	155	49	67	79	80	2.313432836
29	PANCHAVARNAM	59	F	72	158	69	95	100	86	1.663157895
30	AMBIKA	40	F	70	165	89	105	111	90	1.571428571
31	PRIYA	58	F	72	140	62	99	97	76	1.414141414
32	VIJAYA	40	F	69	150	60	94	98	78	1.595744681
33	KAMALA	60	F	64	150	60	95	104	84	1.578947368
34	MANIYAMMAL	59	F	67	150	60	105	103	91	1.428571429
35	AMUTHA	44	F	71	164	54	80	87	95	2.05
36	MARIYA	40	F	68	155	45	62	78	69	2.5
37	MOHANAPRIYA	32	F	70	162	64	87	95	72	1.862068966
38	MUNIYAMMAL	60	F	68	165	85	100	109	70	1.65
39	KAVITHA	42	F	69	152	70	98	105	71	1.551020408
40	SANGEETHA	52	F	71	162	80	95	102	73	1.705263158
41	GOWSEEBI	60	F	74	170	65	92	101	76	1.847826087
42	RAJESHWARI	54	F	69	155	70	102	108	71	1.519607843
43	CHITRA	34	F	72	167	75	92	103	74	1.815217391
44	GAYATHRI	36	F	67	160	56	85	77	69	1.882352941
45	VIJAYALAKSHMI	50	F	72	162	66	85	98	74	1.905882353
46	SAMUNDESWARI	60	F	70	170	55	86	94	79	1.976744186

S. NO	NAME	AGE	SEX	ARM LENGTH (cm)	HEIGHT (cm)	WEIGHT (kg)	WAIST CIRCUM FERENCE (cm)	HIP CIRCUM FERENCE (cm)	LEG LENGTH (cm)	HEIGHT/ WAIST RATIO
47	MUNIYAMMAL	55	F	65	174	65	92	105	85	1.891304348
48	DEVI	42	F	60	155	75	96	103	70	1.614583333
49	MAHESHWARI	35	F	68	162	60	100	109	78	1.62
50	MUTHU	32	F	74	170	62	80	94	82	2.125
51	SIVAGAMI	35	F	71	162	60	89	92	79	1.820224719
52	SUBALAKSHMI	60	F	63	130	56	88	92	68	1.477272727
53	KARPAGAM	48	F	74	175	86	85	93	95	2.058823529
54	rajeswari	34	F	69	163	67	87	91	88	1.873563218
55	VIJAYA	40	F	70	155	62	88	91	84	1.761363636
56	SUNDARI	57	F	66	150	54	89	94	79	1.685393258
57	MEGALA	50	F	64	149	49	76	85	78	1.960526316
58	SUBALAKSHMI	30	F	67	154	52	72	82	83	2.138888889
59	VILVASUDHA	35	F	73	170	65	87	92	90	1.954022989
60	KALA	58	F	68	165	40	65	77	90	2.538461538
61	UMARANI	42	F	72	158	97	118	122	86	1.338983051
62	BHAVANI	38	F	68	156	65	83	94	86	1.879518072
63	MANIYAMMAL	59	F	67	150	60	105	103	91	1.428571429
64	AMUTHA	44	F	71	164	54	80	87	95	2.05
65	MARIYA	40	F	68	155	45	62	78	69	2.5
66	MOHANAPRIYA	32	F	70	162	64	87	95	72	1.862068966
67	AMBIKA	40	F	70	165	89	105	111	89	1.571428571
	AVERAGE			**68.47761194**					**80.23881**	**1.861824461**