

SWEAT BASED GLUCOSE MONITORING WITH INBUILT DRUG DELIVERY SYSTEM

Submitted in partial fulfillment of the requirements for the award of
Master of Engineering Degree in
Biomedical Engineering

by

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BONAFIDE CERTIFICATE

This is to certify that this Project Report is the bonafide work of JINI JACOB.Y
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GLUCOSE MONITORING WITH INBUILT DRUG DELIVERY SYSTEM**”
under my supervision from September 2020 to April 2021.

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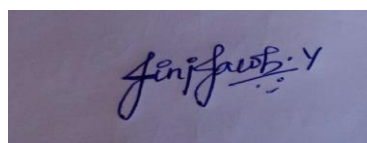
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DECLARATION

I JINI JACOB.Y (Reg. No.39440002) hereby declare that the Project Report entitled “**SWEAT BASED GLUCOSE MONITORING WITH INBUILT DRUG DELIVERY SYSTEM**” done by me under the guidance of Dr. S. KRISHNAKUMAR is submitted in partial fulfillment of the requirements for the award of Master of Technology degree in Medical Instrumentation.

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A rectangular box containing a handwritten signature in blue ink. The signature appears to be 'Jini Jacob.Y' written in a cursive style.

SIGNATURE OF THE CANDIDATE

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ABSTRACT

Electrochemical analysis of sweat using soft bio electronics on human skin provides a new route for noninvasive glucose monitoring without painful blood collection and estimate the blood glucose concentration .Diabetes is caused by hyperglycemia and hypoglycemia. High glucose concentration causes chronic diseases and low glucose concentration causes shock or death. Therefore it is very important to prevent both high glucose and low glucose concentration. Sweat contains glucose and the sweat glucose concentration has a quite good correlation with blood glucose concentration. To enhance accuracy we used different kind of sensors like temperature sensor ,humidity sensor, current sensor and we measure glucose as well as temperature simultaneously. After finding out the glucose concentration we need to control the high glucose concentration by delivering an appropriate amount of drug painlessly and rapidly .The system provides a novel closed loop solution for the noninvasive sweat based management of diabetes mellitus.

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LIST OF SYMBOLS AND ABBREVIATIONS

DKA	-	Diabetic Ketoacidosis
HHS	-	Hyperglycemia State
LADA	-	Latent autoimmune diabetic of adults
ACEs	-	Adverse childhood experiences
MODY	-	Maturity onset diabetes of young
ADA	-	American diabetic association
ACEIs	-	Angiotensin converting enzyme inhibitors
ADP-4	-	Dipeptidyl peptidase
ARBs	-	Angiotensin receptor blockers
IDF	-	International Diabetes Federation
ER	-	Emergency Room
NIDDM	-	Noninsulin -dependent diabetes mellitus
DCCT	-	Diabetes control and complications
NIDDK	-	National Institute of Diabetes and Digestive and Kidney
UKPDS	-	United kingdom prospective diabetes study

CHAPTER 1

INTRODUCTION

Diabetes mellitus (DM), commonly known as diabetes, is a social occasion of metabolic issues depicted by a high glucose level all through a deferred time period. Signs routinely consolidate progressive pee, extended thirst and extended desiring. At whatever point left untreated, diabetes can causes cause various challenges intricacy. Extraordinary entrapments can join diabetic ketoacidosis hyperosmolar hyper glycemia state, or destruction. Certifiable long stretch complexities join cardiovascular contamination, stroke, tireless kidney affliction, foot ulcers, mischief to the nerves, damage to the eyes and mental obstruction.

Diabetes is a result of either the pancreas not making adequate insulin, or the cells of the body not responding true to form to the insulin conveyed. There are three essential sorts of diabetes mellitus:

Type 1 diabetes results from frustration of the pancreas to make adequate insulin due to loss of beta cells. This construction was as of late implied as "juvenile diabetes". The insufficiency of beta cells is achieved by an invulnerable framework response. The purpose behind this safe framework response is dark.

Type 2 diabetes begins with insulin resistance, a condition where cells disregard to respond to insulin fittingly. As the disease progresses, a shortfall of insulin may similarly make. This construction was as of late suggested "grown-up starting diabetes". The most notable explanation is a blend of over the top body weight and inadequate exercise.

Gestational diabetes is the third standard construction, and happens when pregnant women without a previous history of diabetes make hyperglycemia levels. Type 1 diabetes ought to be managed insulin implantations. Countering and treatment of type 2 diabetes incorporates keeping a sound eating standard, standard real exercise, a normal body weight, and avoiding use of tobacco. Type 2 diabetes may be treated with drugs, for instance, insulin sensitizers with or without insulin. Control of heartbeat and keeping up authentic foot and eye care are critical for people with the contamination. Insulin and some oral prescriptions can cause hypoglycemia. Weight decrease operation in those with obesity is sometimes an effective measure in those with type 2 diabetes. Gestational diabetes usually resolves after the birth of the baby.

As of 2019, an estimated 463 million people had diabetes worldwide (8.8% of the adult population), with type 2 diabetes making up about 90% of the cases. Rates are similar in women and men. Trends suggest that rates will continue to rise. Diabetes at least doubles a person's risk of early death. In 2019, diabetes resulted in approximately 4.2 million deaths. It is the 7th leading cause of death globally. The global economic cost of diabetes related health expenditure in 2017 was estimated at 727 billion. In the United States, diabetes cost nearly 327 billion in 2017. Average medical expenditures among people with diabetes are about 2.3 times higher.

1.1 SIGNS AND SYMPTOMS

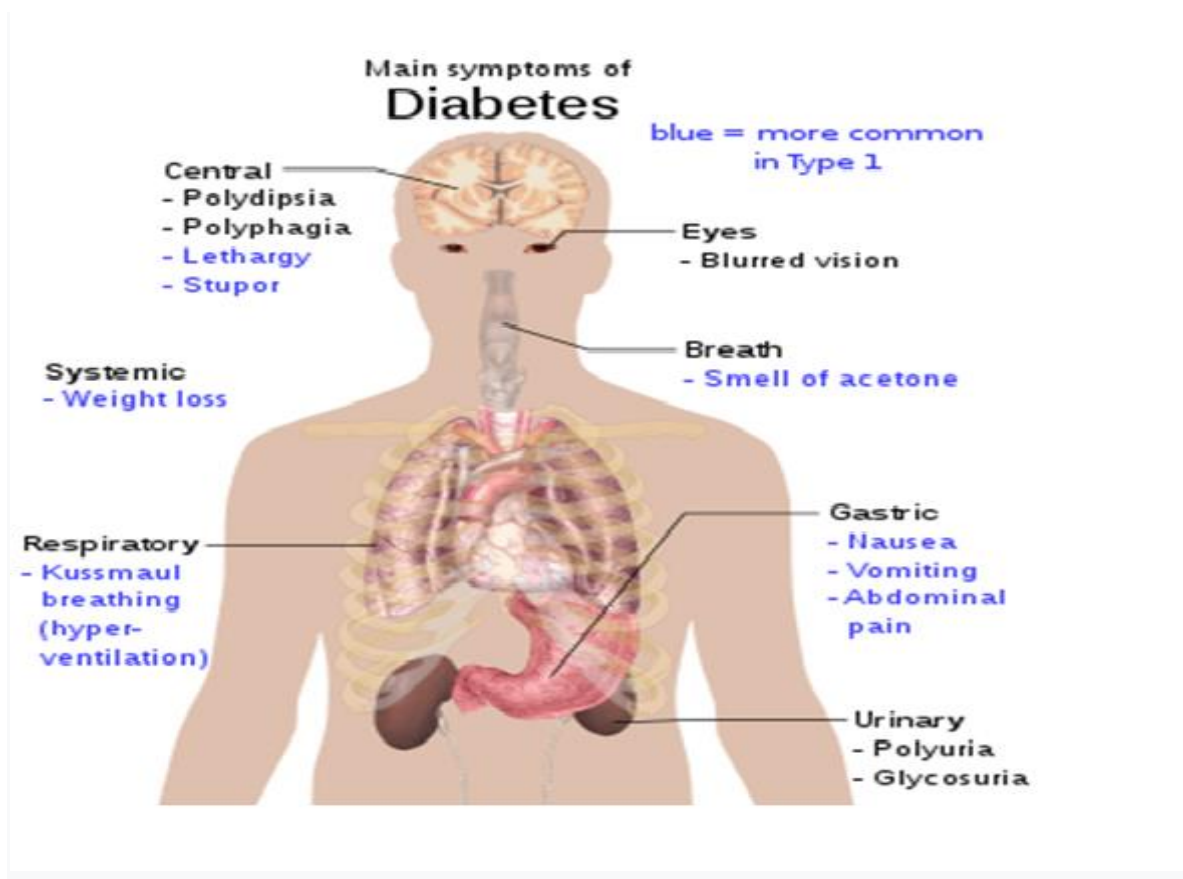


Fig:1.1 Symptoms of diabetics

The classic symptoms of untreated diabetes are unintended weight loss, polyuria (increased urination), polydipsia (increased thirst), and polyphagia (increased hunger). Symptoms may develop rapidly (weeks or months) in type 1 diabetes, while they usually develop much more slowly and may be subtle or absent in type 2 diabetes.

Several other signs and symptoms can mark the onset of diabetes although they are not specific to the disease. In addition to the known ones above, they include blurred vision, headache, fatigue, slow healing of cuts, and itchy skin. Prolonged high blood glucose can cause glucose absorption in the lens of the eye, which leads to changes in its shape, resulting in vision changes. Long-term vision loss can also be caused by diabetic retinopathy. A number of skin rashes that can occur in diabetes are collectively known as diabetic dermadromes.

1.2 DIABETIC EMERGENCIES

People with diabetes (usually but not exclusively in type 1 diabetes) may also experience diabetic ketoacidosis (DKA), a metabolic disturbance characterized by nausea, vomiting and abdominal pain, the smell of acetone on the breath, deep breathing known as Kussmaul breathing, and in severe cases a decreased level of consciousness. DKA requires emergency treatment in hospital. A rarer but more dangerous condition is hyperosmolar hyperglycemic state (HHS), which is more common in type 2 diabetes and is mainly the result of dehydration caused by high blood sugars.

Treatment-related low blood sugar (hypoglycemia) is common in people with type 1 and also type 2 diabetes depending on the medication being used. Most cases are mild and are not considered medical emergencies. Effects can range from feelings of unease, sweating, trembling, and increased appetite in mild cases to more serious effects such as confusion, changes in behavior such as aggressiveness, seizures, unconsciousness, and rarely permanent brain damage or death in severe cases. Rapid breathing, sweating, and cold, pale skin are characteristic of low blood sugar but not definitive. Mild to moderate cases are self-treated by eating or drinking something high in rapidly absorbed carbohydrates. Severe cases can lead to unconsciousness and must be treated with intravenous glucose or injections with glucagon.

1.3 COMPLICATION

These typically develop after many years (10–20) but maybe the first symptom in those who have otherwise not received a diagnosis before that time.

The major long-term complications relate to damage to blood vessels. Diabetes doubles the risk of cardiovascular disease and about 75% of deaths in people with diabetes are due to coronary artery disease. Other macrovascular diseases include stroke, and peripheral artery disease.

The primary complications of diabetes due to damage in small blood vessels include damage to the eyes, kidneys, and nerves. Damage to the eyes, known as diabetic retinopathy, is caused by damage to the blood vessels in the retina of the eye, and can result in gradual vision loss and eventual blindness. Diabetes also increases the risk of having glaucoma, cataracts, and other eye problems. It is recommended that people with diabetes visit an eye doctor once a year. Damage to the kidneys, known as diabetic nephropathy, can lead to tissue scarring, urine protein loss, and eventually chronic kidney disease, sometimes requiring dialysis or kidney transplantation. Damage to the nerves of the body, known as diabetic neuropathy, is the most common complication of diabetes. The symptoms can include numbness, tingling, pain, and altered pain sensation, which can lead to damage to the skin. Diabetes-related foot problems (such as diabetic foot ulcers) may occur, and can be difficult to treat, occasionally requiring amputation. Additionally, proximal diabetic neuropathy causes painful muscle atrophy and weakness.

There is a link between cognitive deficit and diabetes. Compared to those without diabetes, those with the disease have a 1.2 to 1.5-fold greater rate of decline in cognitive function. Having diabetes, especially when on insulin, increases the risk of falls in older people.

1.4 CAUSES

Table 1.1 Type 1 and Type 2 Diabetes

Feature	Type 1 diabetes	Type 2 diabetes
Onset	Sudden	Gradual
Age at onset	Mostly in children	Mostly in adults
Body size	Thin or normal	Often obese

Ketoacidosis	Common	Rare
Autoantibodies	Usually present	Absent
Endogenous insulin	Low or absent	Normal, decreased or increased
Concordance in identical twins	50%	90%
Prevalence	~10%	~90%

Diabetes mellitus is classified into six categories: type 1 diabetes, type 2 diabetes, hybrid forms of diabetes, hyperglycemia first detected during pregnancy, "unclassified diabetes", and "other specific types". The "hybrid forms of diabetes" contains slowly evolving, immune-mediated diabetes of adults and ketosis-prone type 2 diabetes. The "hyperglycemia first detected during pregnancy" contains gestational diabetes mellitus and diabetes mellitus in pregnancy (type 1 or type 2 diabetes first diagnosed during pregnancy). The "other specific types" are a collection of a few dozen individual causes. Diabetes is a more variable disease than once thought and people may have combinations of forms. The term "diabetes", without qualification, refers to diabetes mellitus.

1.5 TYPE 1

Type 1 diabetes is characterized by loss of the insulin-producing beta cells of the pancreatic islets, leading to insulin deficiency. This type can be further classified as immune-mediated or idiopathic. The majority of type 1 diabetes is of an immune-mediated nature, in which a T cell-mediated autoimmune attack leads to the loss of beta cells and thus insulin. It causes approximately 10% of diabetes mellitus cases in North America and Europe. Most affected people are otherwise healthy and of a healthy weight when onset occurs. Sensitivity and responsiveness to insulin are usually normal, especially in the early stages. Although it has been called "juvenile

diabetes" due to the frequent onset in children, the majority of individuals living with type 1 diabetes are now adults.

"Brittle" diabetes, also known as unstable diabetes or labile diabetes, is a term that was traditionally used to describe the dramatic and recurrent swings in glucose levels, often occurring for no apparent reason in insulin-dependent diabetes. This term, however, has no biologic basis and should not be used. Still, type 1 diabetes can be accompanied by irregular and unpredictable high blood sugar levels, and the potential for diabetic ketoacidosis or serious low blood sugar levels. Other complications include an impaired counterregulatory response to low blood sugar, infection, gastroparesis (which leads to erratic absorption of dietary carbohydrates), and endocrinopathies (e.g., Addison's disease). These phenomena are believed to occur no more frequently than in 1% to 2% of persons with type 1 diabetes.

Type 1 diabetes is partly inherited, with multiple genes, including certain HLA genotypes, known to influence the risk of diabetes. In genetically susceptible people, the onset of diabetes can be triggered by one or more environmental factors, such as a viral infection or diet. Several viruses have been implicated, but to date there is no stringent evidence to support this hypothesis in humans. Among dietary factors, data suggest that gliadin (a protein present in gluten) may play a role in the development of type 1 diabetes, but the mechanism is not fully understood.

Type 1 diabetes can occur at any age, and a significant proportion is diagnosed during adulthood. Latent autoimmune diabetes of adults (LADA) is the diagnostic term applied when type 1 diabetes develops in adults; it has a slower onset than the same condition in children. Given this difference, some use the unofficial term "type 1.5 diabetes" for this condition. Adults with LADA are frequently initially misdiagnosed as having type 2 diabetes, based on age rather than a cause.

1.6 TYPE 2

Type 2 diabetes is characterized by insulin resistance, which may be combined with relatively reduced insulin secretion. The defective responsiveness of body tissues to insulin is believed to involve the insulin receptor. However, the specific defects are not known. Diabetes mellitus cases due to a known defect are classified separately. Type 2 diabetes is the most common type of diabetes mellitus. Many

people with type 2 diabetes have evidence of prediabetes (impaired fasting glucose and/or impaired glucose tolerance) before meeting the criteria for type 2 diabetes. The progression of prediabetes to overt type 2 diabetes can be slowed or reversed by lifestyle changes or medications that improve insulin sensitivity or reduce the liver's glucose production.

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Type 2 diabetes is primarily due to lifestyle factors and genetics. A number of lifestyle factors are known to be important to the development of type 2 diabetes, including obesity (defined by a body mass index of greater than 30), lack of physical activity, poor diet, stress, and urbanization. Excess body fat is associated with 30% of cases in people of Chinese and Japanese descent, 60–80% of cases in those of European and African descent, and 100% of Pima Indians and Pacific Islanders. Even those who are not obese may have a high waist–hip ratio.

Dietary factors such as sugar-sweetened drinks are associated with an increased risk. The type of fats in the diet is also important, with saturated fat and trans fats increasing the risk and polyunsaturated and monounsaturated fat decreasing the risk. Eating white rice excessively may increase the risk of diabetes, especially in Chinese and Japanese people. Lack of physical activity may increase the risk of diabetes in some people.

Adverse childhood experiences (ACEs), including abuse, neglect, and household difficulties, increase the likelihood of type 2 diabetes later in life by 32%, with neglect having the strongest effect.

1.7 GESTATIONAL DIABETES

Gestational diabetes resembles type 2 diabetes in several respects, involving a combination of relatively inadequate insulin secretion and responsiveness. It occurs in about 2–10% of all pregnancies and may improve or disappear after delivery. It is recommended that all pregnant women get tested starting around 24–28 weeks gestation. It is most often diagnosed in the second or third trimester because of the increase in insulin-antagonist hormone levels that occurs at this time. However, after pregnancy approximately 5–10% of women with gestational diabetes are found to have another form of diabetes, most commonly type 2. Gestational diabetes is fully treatable, but requires careful medical supervision throughout the pregnancy. Management may include dietary changes, blood glucose monitoring, and in some cases, insulin may be required.

Though it may be transient, untreated gestational diabetes can damage the health of the fetus or mother. Risks to the baby include macrosomia (high birth weight), congenital heart and central nervous system abnormalities, and skeletal muscle malformations. Increased levels of insulin in a fetus's blood may inhibit fetal surfactant production and cause infant respiratory distress syndrome. A high blood bilirubin level may result from red blood cell destruction. In severe cases, perinatal death may occur, most commonly as a result of poor placental perfusion due to vascular impairment. Labor induction may be indicated with decreased placental function. A caesarean section may be performed if there is marked fetal distress or an increased risk of injury associated with macrosomia, such as shoulder dystocia.

Maturity onset diabetes of the young (MODY) is a rare autosomal dominant inherited form of diabetes, due to one of several single-gene mutations causing defects in insulin production. It is significantly less common than the three main types, constituting 1–2% of all cases. The name of this disease refers to early hypotheses as to its nature. Being due to a defective gene, this disease varies in age at presentation and in severity according to the specific gene defect; thus there are at least 13 subtypes of MODY. People with MODY often can control it without using insulin.

Some cases of diabetes are caused by the body's tissue receptors not responding to insulin (even when insulin levels are normal, which is what separates it from

type 2 diabetes); this form is very uncommon. Genetic mutations (autosomal or mitochondrial) can lead to defects in beta cell function. Abnormal insulin action may also have been genetically determined in some cases. Any disease that causes extensive damage to the pancreas may lead to diabetes (for example, chronic pancreatitis and cystic fibrosis). Diseases associated with excessive secretion of insulin-antagonistic hormones can cause diabetes (which is typically resolved once the hormone excess is removed). Many drugs impair insulin secretion and some toxins damage pancreatic beta cells, whereas others increase insulin resistance (especially glucocorticoids which can provoke "steroid diabetes"). The ICD-10 (1992) diagnostic entity, *malnutrition-related diabetes mellitus* (MRDM or MMDM, ICD-10 code E12), was deprecated by the World Health Organization (WHO) when the current taxonomy was introduced in 1999. Yet another form of diabetes that people may develop is double diabetes. This is when a type 1 diabetic becomes insulin resistant, the hallmark for type 2 diabetes or has a family history for type 2 diabetes. It was first discovered in 1990 or 1991.

The following is a list of disorders that may increase the risk of diabetes:

- Genetic defects of β -cell function
 - Maturity onset diabetes of the young
 - Mitochondrial DNA mutations
- Genetic defects in insulin processing or insulin action
 - Defects in proinsulin conversion
 - Insulin gene mutations
 - Insulin receptor mutations
- Exocrine pancreatic defects
 - Chronic pancreatitis
 - Pancreatectomy
 - Pancreatic neoplasia
 - Cystic fibrosis
 - Hemochromatosis
 - Fibrocalculous pancreatopathy

1.8 PATHAPHYSIOLOGY

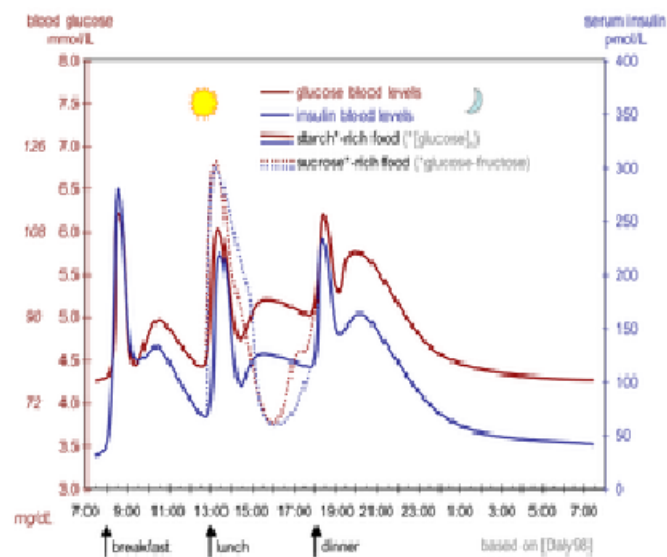


Fig:1.2 Blood Sugar Level

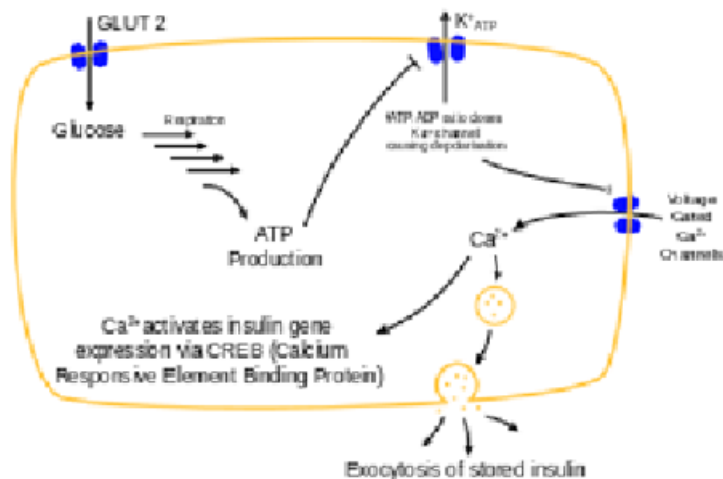


Fig:1.3 Mechanism of Insulin

Insulin is the principal hormone that regulates the uptake of glucose from the blood into most cells of the body, especially liver, adipose tissue and muscle, except smooth muscle, in which insulin acts via the IGF-1. Therefore, deficiency of insulin or the insensitivity of its receptors play a central role in all forms of diabetes mellitus.

The body obtains glucose from three main sources: the intestinal absorption of food; the breakdown of glycogen (glycogenolysis), the storage form of glucose found in the liver; and gluconeogenesis, the generation of glucose from non-carbohydrate substrates in the body. Insulin plays a critical role in regulating glucose levels in the body. Insulin can inhibit the breakdown of glycogen or the process of

gluconeogenesis, it can stimulate the transport of glucose into fat and muscle cells, and it can stimulate the storage of glucose in the form of glycogen.

Insulin is released into the blood by beta cells (β -cells), found in the islets of Langerhans in the pancreas, in response to rising levels of blood glucose, typically after eating. Insulin is used by about two-thirds of the body's cells to absorb glucose from the blood for use as fuel, for conversion to other needed molecules, or for storage. Lower glucose levels result in decreased insulin release from the beta cells and in the breakdown of glycogen to glucose. This process is mainly controlled by the hormone glucagon, which acts in the opposite manner to insulin.

If the amount of insulin available is insufficient, or if cells respond poorly to the effects of insulin (insulin resistance), or if the insulin itself is defective, then glucose is not absorbed properly by the body cells that require it, and is not stored appropriately in the liver and muscles. The net effect is persistently high levels of blood glucose, poor protein synthesis, and other metabolic derangements, such as metabolic acidosis in cases of complete insulin deficiency.

When glucose concentration in the blood remains high over time, the kidneys reach a threshold of reabsorption, and the body excretes glucose in the urine (glycosuria). This increases the osmotic pressure of the urine and inhibits reabsorption of water by the kidney, resulting in increased urine production (polyuria) and increased fluid loss. Lost blood volume is replaced osmotically from water in body cells and other body compartments, causing dehydration and increased thirst (polydipsia). In addition, intracellular glucose deficiency stimulates appetite leading to excessive food intake (polyphagia).

Diabetes mellitus is characterized by recurrent or persistent high blood sugar, and is diagnosed by demonstrating any one of the following:

- Fasting plasma glucose level ≥ 7.0 mmol/L (126 mg/dL)
- Plasma glucose ≥ 11.1 mmol/L (200 mg/dL) two hours after a 75 gram oral glucose load as in a glucose tolerance test (OGTT)
- Symptoms of high blood sugar and casual plasma glucose ≥ 11.1 mmol/L (200 mg/dL)
- Glycated hemoglobin (HbA_{1c}) ≥ 48 mmol/mol (≥ 6.5 DCCT %).

Table 1.2 Diabetes Criteria

Condition	2-hour glucose	Fasting glucose	HbA_{1c}			
<i>Unit</i>	<i>mmol/L</i>	<i>mg/dl</i>	<i>mmol/L</i>	<i>mg/dL</i>	<i>mmol/mol</i>	<i>DCCT %</i>
Normal	< 7.8	< 140	< 6.1	< 110	< 42	< 6.0
Impaired fasting glycaemia	< 7.8	< 140	6.1–7.0	110–125	42–46	6.0–6.4
Impaired glucose tolerance	≥ 7.8	≥ 140	< 7.0	< 126	42–46	6.0–6.4
Diabetes mellitus	≥ 11.1	≥ 200	≥ 7.0	≥ 126	≥ 48	≥ 6.5

A positive result, in the absence of unequivocal high blood sugar, should be confirmed by a repeat of any of the above methods on a different day. It is preferable to measure a fasting glucose level because of the ease of measurement and the considerable time commitment of formal glucose tolerance testing, which takes two hours to complete and offers no prognostic advantage over the fasting test. According to the current definition, two fasting glucose measurements above 7.0 mmol/L (126 mg/dL) is considered diagnostic for diabetes mellitus.

Per the WHO, people with fasting glucose levels from 6.1 to 6.9 mmol/L (110 to 125 mg/dL) are considered to have impaired fasting glucose. People with plasma glucose at or above 7.8 mmol/L (140 mg/dL), but not over 11.1 mmol/L (200 mg/dL), two hours after a 75 gram oral glucose load are considered to have impaired glucose tolerance. Of these two prediabetic states, the latter in particular is a major risk factor for progression to full-blown diabetes mellitus, as well as cardiovascular disease. The American Diabetes Association (ADA) since 2003 uses a slightly different range for impaired fasting glucose of 5.6 to 6.9 mmol/L (100 to 125 mg/dL). Glycated hemoglobin is better than fasting glucose for determining risks of cardiovascular disease and death from any cause.

1.9 PREVENTION

Type 2 diabetes—which accounts for 85–90% of all cases worldwide—can often be prevented or delayed by maintaining a normal body weight, engaging in physical activity, and eating a healthy diet. Higher levels of physical activity (more than 90 minutes per day) reduce the risk of diabetes by 28%. Dietary changes known to be effective in helping to prevent diabetes include maintaining a diet rich in whole grains and fiber, and choosing good fats, such as the polyunsaturated fats found in nuts, vegetable oils, and fish. Limiting sugary beverages and eating less red meat and other sources of saturated fat can also help prevent diabetes. Tobacco smoking is also associated with an increased risk of diabetes and its complications, so smoking cessation can be an important preventive measure as well.

The relationship between type 2 diabetes and the main modifiable risk factors (excess weight, unhealthy diet, physical inactivity and tobacco use) is similar in all regions of the world. There is growing evidence that the underlying determinants of diabetes are a reflection of the major forces driving social, economic and cultural change: globalization, urbanization, population aging, and the general health policy environment.

1.10 MANAGEMENT

Learning about the disease and actively participating in the treatment is important, since complications are far less common and less severe in people who have well-managed blood sugar levels. Per the American College of Physicians, the goal of treatment is an HbA_{1C} level of 7-8%. Attention is also paid to other health problems

that may accelerate the negative effects of diabetes. These include smoking, high blood pressure, metabolic syndrome obesity, and lack of regular exercise. Specialized footwear is widely used to reduce the risk of ulcers in at-risk diabetic feet although evidence for the efficacy of this remains equivocal.

1.10.1 Lifestyle

People with diabetes can benefit from education about the disease and treatment, dietary changes, and exercise, with the goal of keeping both short-term and long-term blood glucose levels within acceptable bounds. In addition, given the associated higher risks of cardiovascular disease, lifestyle modifications are recommended to control blood pressure.

Weight loss can prevent progression from prediabetes to diabetes type 2, decrease the risk of cardiovascular disease, or result in a partial remission in people with diabetes. No single dietary pattern is best for all people with diabetes. Healthy dietary patterns, such as the Mediterranean diet, low-carbohydrate diet, or DASH diet, are often recommended, although evidence does not support one over the others. According to the ADA, "reducing overall carbohydrate intake for individuals with diabetes has demonstrated the most evidence for improving glycemia", and for individuals with type 2 diabetes who cannot meet the glycemic targets or where reducing anti-glycemic medications is a priority, low or very-low carbohydrate diets are a viable approach. For overweight people with type 2 diabetes, any diet that achieves weight loss is effective.

1.11 MEDICATIONS

1.11.1 Glucose control

Most medications used to treat diabetes act by lowering blood sugar levels through different mechanisms. There is broad consensus that when people with diabetes maintain tight glucose control – keeping the glucose levels in their blood within normal ranges – they experience fewer complications, such as kidney problems or eye problems. There is however debate as to whether this is appropriate and cost effective for people later in life in whom the risk of hypoglycemia may be more significant.

There are a number of different classes of anti-diabetic medications. Type 1 diabetes requires treatment with insulin, ideally using a "basal bolus" regimen that

most closely matches normal insulin release: long-acting insulin for the basal rate and short-acting insulin with meals. Type 2 diabetes is generally taken with medication that is taken by mouth (e.g. metformin) although some eventually require injectable treatment with insulin or GLP-1 agonists.

Metformin is generally recommended as a first-line treatment for type 2 diabetes, as there is good evidence that it decreases mortality. It works by decreasing the liver's production of glucose. Several other groups of drugs, mostly given by mouth, may also decrease blood sugar in type 2 diabetes. These include agents that increase insulin release (sulfonylureas), agents that decrease absorption of sugar from the intestines (acarbose), agents that inhibit the enzyme dipeptidyl peptidase-4 (DPP-4) that inactivates incretins such as GLP-1 and GIP (sitagliptin), agents that make the body more sensitive to insulin (thiazolidinedione) and agents that increase the excretion of glucose in the urine (SGLT2 inhibitors). When insulin is used in type 2 diabetes, a long-acting formulation is usually added initially, while continuing oral medications. Doses of insulin are then increased until glucose targets are reached.

1.11.2 Blood pressure lowering

Cardiovascular disease is a serious complication associated with diabetes, and many international guidelines recommend blood pressure treatment targets that are lower than 140/90 mmHg for people with diabetes. However, there is only limited evidence regarding what the lower targets should be. A 2016 systematic review found potential harm to treating to targets lower than 140 mmHg, and a subsequent systematic review in 2019 found no evidence of additional benefit from blood pressure lowering to between 130 - 140mmHg, although there was an increased risk of adverse events.

2015 American Diabetes Association recommendations are that people with diabetes and albuminuria should receive an inhibitor of the renin-angiotensin system to reduce the risks of progression to end-stage renal disease, cardiovascular events, and death. There is some evidence that angiotensin converting enzyme inhibitors (ACEIs) are superior to other inhibitors of the renin-angiotensin system such as angiotensin receptor blockers (ARBs), or aliskiren in preventing cardiovascular disease. Although a more recent review found similar effects of

ACEIs and ARBs on major cardiovascular and renal outcomes. There is no evidence that combining ACEIs and ARBs provides additional benefits.

1.11.3 Aspirin

The use of aspirin to prevent cardiovascular disease in diabetes is controversial. Aspirin is recommended by some in people at high risk of cardiovascular disease, however routine use of aspirin has not been found to improve outcomes in uncomplicated diabetes. 2015 American Diabetes Association recommendations for aspirin use (based on expert consensus or clinical experience) are that low-dose aspirin use is reasonable in adults with diabetes who are at intermediate risk of cardiovascular disease (10-year cardiovascular disease risk, 5–10%). National guidelines for England and Wales by the National Institute for Health and Care Excellence (NICE) recommend against the use of aspirin in people with type 1 or type 2 diabetes who do not have confirmed cardiovascular disease.

1.11.4 Surgery

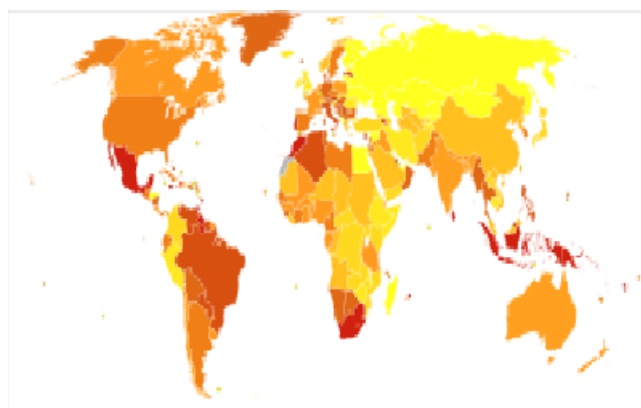
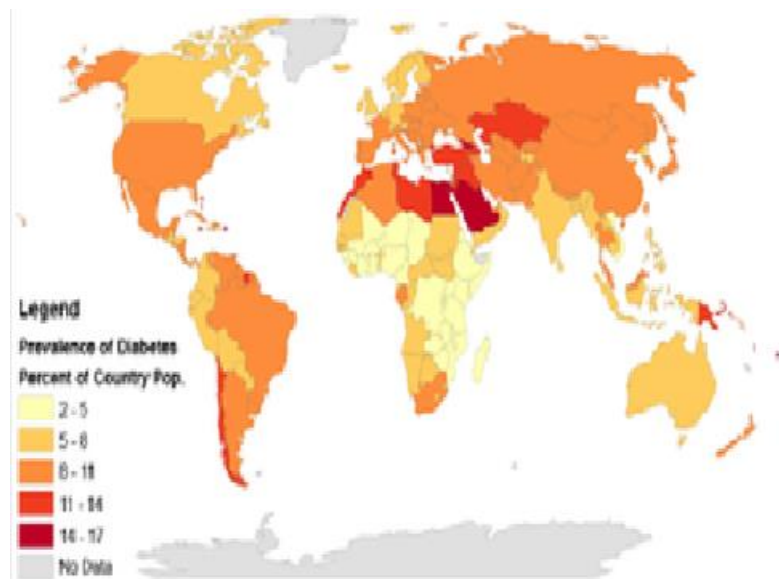
Weight loss surgery in those with obesity and type 2 diabetes is often an effective measure. Many are able to maintain normal blood sugar levels with little or no medications following surgery and long-term mortality is decreased. There is, however, a short-term mortality risk of less than 1% from the surgery. The body mass index cutoffs for when surgery is appropriate are not yet clear. It is recommended that this option be considered in those who are unable to get both their weight and blood sugar under control.

A pancreas transplant is occasionally considered for people with type 1 diabetes who have severe complications of their disease, including end stage kidney disease requiring kidney transplantation.

1.11.5 Support

In countries using a general practitioner system, such as the United Kingdom, care may take place mainly outside hospitals, with hospital-based specialist care used only in case of complications, difficult blood sugar control, or research projects. In other circumstances, general practitioners and specialists share care in a team approach. Home telehealth support can be an effective management technique.

1.11.6.Epidiomology



Mortality rate of diabetes worldwide in 2012 per million inhabitants



Fig:1.7 Rates of diabetes

In 2017, 425 million people had diabetes worldwide, up from an estimated 382 million people in 2013 and from 108 million in 1980. Accounting for the shifting age structure of the global population, the prevalence of diabetes is 8.8% among adults, nearly double the rate of 4.7% in 1980. Type 2 makes up about 90% of the cases. Some data indicate rates are roughly equal in women and men, but male excess in diabetes has been found in many populations with higher type 2 incidence, possibly due to sex-related differences in insulin sensitivity, consequences of obesity and regional body fat deposition, and other contributing factors such as high blood pressure, tobacco smoking, and alcohol intake.

The WHO estimates that diabetes resulted in 1.5 million deaths in 2012, making it the 8th leading cause of death. However another 2.2 million deaths worldwide were attributable to high blood glucose and the increased risks of cardiovascular disease and other associated complications (e.g. kidney failure), which often lead to premature death and are often listed as the underlying cause on death certificates rather than diabetes. For example, in 2017, the International Diabetes Federation (IDF) estimated that diabetes resulted in 4.0 million deaths worldwide, using modeling to estimate the total number of deaths that could be directly or indirectly attributed to diabetes.

Diabetes occurs throughout the world but is more common (especially type 2) in more developed countries. The greatest increase in rates has however been seen

in low- and middle-income countries, where more than 80% of diabetic deaths occur. The fastest prevalence increase is expected to occur in Asia and Africa, where most people with diabetes will probably live in 2030. The increase in rates in developing countries follows the trend of urbanization and lifestyle changes, including increasingly sedentary lifestyles, less physically demanding work and the global nutrition transition, marked by increased intake of foods that are high energy-dense but nutrient-poor (often high in sugar and saturated fats, sometimes referred to as the "Western-style" diet) The global number of diabetes cases might increase by 48% between 2017 and 2045.

1.12 HISTORY

Diabetes was one of the first diseases described, with an Egyptian manuscript from c. 1500 BCE mentioning "too great emptying of the urine." The Ebers papyrus includes a recommendation for a drink to take in such cases. The first described cases are believed to have been type 1 diabetes. Indian physicians around the same time identified the disease and classified it as *madhumeha* or "honey urine", noting the urine would attract ants.

The term "diabetes" or "to pass through" was first used in 230 BCE by the Greek Apollonius of Memphis. The disease was considered rare during the time of the Roman empire, with Galen commenting he had only seen two cases during his career This is possibly due to the diet and lifestyle of the ancients, or because the clinical symptoms were observed during the advanced stage of the disease. Galen named the disease "diarrhea of the urine" (diarrhea urinosa).

The earliest surviving work with a detailed reference to diabetes is that of Aretaeus of Cappadocia (2nd or early 3rd century CE). He described the symptoms and the course of the disease, which he attributed to the moisture and coldness, reflecting the beliefs of the "Pneumatic School". He hypothesized a correlation between diabetes and other diseases, and he discussed differential diagnosis from the snakebite, which also provokes excessive thirst. His work remained unknown in the West until 1552, when the first Latin edition was published in Venice.

Two types of diabetes were identified as separate conditions for the first time by the Indian physicians Sushruta and Charaka in 400–500 CE with one type being associated with youth and another type with being overweight. Effective treatment

was not developed until the early part of the 20th century when Canadians Frederick Banting and Charles Herbert Best isolated and purified insulin in 1921 and 1922. This was followed by the development of the long-acting insulin NPH in the 1940s.

1.13 ETIOLOGY

The word *diabetes* comes from Latin *diabētēs*, which in turn comes from Ancient Greek, which literally means "a passer through; a siphon". Ancient Greek physician Aretaeus of Cappadocia (fl. 1st century CE) used that word, with the intended meaning "excessive discharge of urine", as the name for the disease. Ultimately, the word comes from Greek (*diabainein*), meaning "to pass through," which is composed of (*dia-*), meaning "through" and (*bainein*), meaning "to go". The word "diabetes" is first recorded in English, in the form *diabete*, in a medical text written around 1425.

The word *mellitus* comes from the classical Latin word *mellītus*, meaning "mellite" (i.e. sweetened with honey; honey-sweet). The Latin word comes from *mell-*, which comes from *mel*, meaning "honey"; sweetness;, whose meaning is the same as that of the English suffix "-ite". It was Thomas Willis who in 1675 added "mellitus" to the word "diabetes" as a designation for the disease, when he noticed the urine of a person with diabetes had a sweet taste (glycosuria). This sweet taste had been noticed in urine by the ancient Greeks, Chinese, Egyptians, Indians, and Persians.

1.14 SOCIETY AND CULTURE

St. Vincent Declaration" was the result of international efforts to improve the care accorded to those with diabetes. Doing so is important not only in terms of quality of life and life expectancy but also economically – expenses due to diabetes have been shown to be a major drain on health – and productivity-related resources for healthcare systems and governments.

Several countries established more and less successful national diabetes programmes to improve treatment of the disease.

People with diabetes who have neuropathic symptoms such as numbness or tingling in feet or hands are twice as likely to be unemployed as those without the symptoms.

In 2010, diabetes-related emergency room (ER) visit rates in the United States were higher among people from the lowest income communities (526 per 10,000 population) than from the highest income communities (236 per 10,000 population). Approximately 9.4% of diabetes-related ER visits were for the uninsured.

1.15 NAMING

The term "type 1 diabetes" has replaced several former terms, including childhood-onset diabetes, juvenile diabetes, and insulin-dependent diabetes mellitus (IDDM). Likewise, the term "type 2 diabetes" has replaced several former terms, including adult-onset diabetes, obesity-related diabetes, and noninsulin-dependent diabetes mellitus (NIDDM). Beyond these two types, there is no agreed-upon standard nomenclature.

Diabetes mellitus is also occasionally known as "sugar diabetes" to differentiate it from diabetes insipidus.

The Burmese, Russian Blue, Abyssinian, and Norwegian Forest cat breeds are at higher risk than other breeds. Overweight cats are also at higher risk.

The symptoms may relate to fluid loss and polyuria, but the course may also be insidious. Diabetic animals are more prone to infections. The long-term complications recognized in humans are much rarer in animals. The principles of treatment (weight loss, oral antidiabetics, subcutaneous insulin) and management of emergencies (e.g. ketoacidosis) are similar to those in humans.

1.16 MAJOR CLINICAL TRIALS

The Diabetes Control and Complications Trial (DCCT) was a clinical study conducted by the United States National Institute of Diabetes and Digestive and Kidney Diseases (NIDDK) that was published in the *New England Journal of Medicine* in 1993. Test subjects all had type 1 diabetes and were randomized to a tight glycemic arm and a control arm with the standard of care at the time; people were followed for an average of seven years, and people in the treatment had dramatically lower rates of diabetic complications. It was as a landmark study at the time, and significantly changed the management of all forms of diabetes.

The United Kingdom Prospective Diabetes Study (UKPDS) was a clinical study conducted by Z that was published in *The Lancet* in 1998. Around 3,800 people with

type 2 diabetes were followed for an average of ten years, and were treated with tight glucose control or the standard of care, and again the treatment arm had far better outcomes. This confirmed the importance of tight glucose control, as well as blood pressure control, for people with this condition

1.17 LITERATURE SURVEY

Diabetes mellitus is perceived just like a condition, an assortment of issues that have hyperglycaemia and glucose prejudice as their trademark, due either to insulin inadequacy or to the impeded viability of insulin's activity, or to a blend of these. To comprehend diabetes it is important to comprehend the ordinary physiological cycle happening during and after a dinner. Food goes through the stomach related framework, where supplements, including proteins, fat and starches are consumed into the circulation system. The presence of sugar, a starch, signs to the endocrine pancreas to emit the chemical insulin. Insulin causes the take-up and capacity of sugar by practically all tissue types in the body, particularly the liver, musculature and fat tissues Islam (1998).

Lamentably, there is no solution for diabetes yet except for by controlling glucose levels through a sound eating regimen, exercise and drug the danger of long haul diabetes confusions can be diminished. Long haul confusions that can be capable are: - eyes – waterfalls and retinopathy (continuous harming of the eye) that may prompt visual impairment - kidneys – kidney sickness and kidney disappointment - nerves – neuropathy (progressive harming of nerves) - feet – ulcers, contaminations, gangrene, and so forth - cardiovascular framework – solidifying of veins, coronary illness and stroke . Corvelli (2003)

The reformist idea of the illness requires steady reassessment of glycaemic control in individuals with diabetes and proper change of remedial regimens. When glycaemic control is not, at this point kept up with a solitary specialist, the expansion of a second or third medication is generally more powerful than changing to another single specialist. Medicinal plants which have showed against diabetic action during prior examinations incorporate *Panax* species, *Phyllanthus* species, *Acacia arabica*, *Aloe vera*, *Aloe barbadensis*, *Artemisia pallens*, *Momordica charantia*, *Alium cepa*, *Trigonella foenum-graecum* and so forth Khawaja (2006).

Not many South-African plants have been logically investigated for their enemy of diabetic qualities. The latest work was finished by Yardimci (2008).

A significant prerequisite for precise epidemiologic and clinical exploration on and for the administration of diabetes mellitus is a fitting order. Moreover the way toward understanding the etiology of an infection and contemplating its common history includes the capacity to recognize and separate between its different structures and spot them into a judicious etiopathologic system Wepasnick (1997).

The contemporary characterization of diabetes and different classifications of glucose prejudice, in light of exploration on this heterogeneous condition, was created in 1979 by the National Diabetes Data Group. Two significant types of diabetes are perceived in Western nations; insulin subordinate diabetes mellitus (IDDM, type I diabetes) and non-insulin dependant diabetes (NIDDM, type II diabetes). The proof of this heterogeneity is overpowering and incorporates the accompanying: a) there are numerous particular issues, the vast majority of which are exclusively uncommon, in which glucose prejudice is a component; b) there are huge contrasts in the predominance of the significant types of diabetes among different racial or ethnic gatherings around the world; c) glucose resilience presents variable clinical highlights, for instance, the contrasts between meager ketosis-inclined, insulin dependant diabetes and hefty, non-ketotic insulin safe diabetes; d) hereditary, immunologic and clinical investigations show that in Western nations, the types of diabetes with their beginning basically in youth or in adulthood are unmistakable elements; e) the kind of non-insulin requiring diabetes in youngsters, which is acquired in an autosomal prevailing design is obviously not the same as the exemplary intense diabetes of adolescents; and f) in tropical nations, a few clinical introductions happen, including fibrocalcific pancreatitis and hunger related diabetes. This and other aggregate proof have been utilized to isolate diabetes mellitus into four particular sorts to be specific; • insulin dependant diabetes, • non-insulin dependant diabetes, • hunger related diabetes, • different kinds of diabetes. The arrangement features the checked heterogeneity of the diabetic condition. Such heterogeneity has significant ramifications for clinical administration of diabetes as well as for biomedical examination Elisseeff (1997).

In this examination the attention was primarily on type II diabetes while type I diabetes was talked about momentarily to bring up the contrasts between the two kinds of diabetes. Insulin dependant diabetes mellitus (IDDM) The subclass of diabetes, type I diabetes, is for the most part described by the sudden beginning of serious side effects, reliance on exogenous insulin to support life and inclination to

ketosis even in the basal express, which is all brought about by total insulin inadequacy. IDDM is the most common kind of diabetes among kids and youthful grown-ups in agricultural nations, and was officially named adolescent diabetes Liu (1997).

It is a catabolic problem wherein circling insulin is practically missing, plasma glucagon is raised, and the pancreatic B cells neglect to react to all insulinogenic boosts McDonnell (2001).

Type I diabetes is thought to result from an irresistible or harmful natural possibility in individuals whose invulnerable frameworks are hereditarily inclined to build up a fiery immune system reaction against pancreatic B cell antigens. Outward factors that may influence cell working incorporate harm brought about by infections like the mumps infection and coxsackie infection B4, by substance specialists, or by ruinous cytotoxins and antibodies delivered from sharpened immunocytes. A fundamental hereditary imperfection identifying with pancreatic B cell replication or capacity may incline an individual to the improvement of B cell disappointment after viral contaminations. Also, explicit HLA qualities may build vulnerability to a diabetogenic infection or might be connected to certain invulnerable reaction qualities that incline patients to a dangerous immune system reaction against their own islet cells (autoaggression). Perceptions that pancreatic B cell harm has all the earmarks of being decreased when immunosuppressive medications, for example, cyclosporine or azathioprine are given at the underlying appearance of type I diabetes support the significance of auto-hostility by the sinsusceptible framework as a central point in the pathogenesis of this sort of diabetes

Type II diabetes extraordinarily out numbers any remaining types of diabetes. Patients with NIDDM are not dependant on exogenous insulin for avoidance of ketonuria and are not inclined to ketosis. Nonetheless, they may require insulin for the rectification of fasting hyperglycaemia if this can't be accomplished with the utilization of diet or oral specialists, and they may create ketosis under unique conditions, for example, serious pressure hastened by contaminations or injury Zhang (1997).

The pathogenesis in type II diabetes is that t The pathogenesis in type II diabetes is that the pancreas produces insulin yet the body doesn't use the insulin accurately. This is fundamentally because of fringe tissue insulin opposition where insulin-receptors or different intermediates in the insulin flagging pathways inside

body cells are obtuse toward insulin and therefore glucose doesn't promptly enter the tissue prompting hyperglycaemia or raised blood glucose focuses (1997).

Weight, which by and large outcomes in debilitated insulin activity, is a typical danger factor for this kind of diabetes, and most patients with type II diabetes are will at last require various enemy of diabetic specialists to keep up satisfactory glycaemic control Mater (2001).

The main depiction of diabetes is credited to Arataeus of Cappadocia in Asia Minor in the principal century AD. The principal endeavors for treating diabetes, when no a greater number of was thought about it than the polyuria, were made by Dr. John Rollo, Surgeon General to the Royal Artillery, 1796 through dietary limitations. The revelation of insulin in 1921 by Dr Frederick Banting was a significant discovery throughout the entire existence of medication and the treatment of diabetes Jaroch (1997).

Diabetes mellitus (DM) is the most well-known endocrine issue, and influences in excess of 100 million individuals around the world (6% of the populace) and in the following 10 years it might influence multiple times a greater number of individuals than it does now (World Health Organization and American Diabetes Association). The World Health Organization has brought up that the avoidance of diabetes and its confusions isn't just a significant test for the future, however fundamental if wellbeing for everything is to be an achievable objective, and unequivocally accentuate the ideal, normal utilization of customary and regular native prescriptions Singh (1994).

In spite of the fact that the improvement of present day medication brought about the approach of current drugs including insulin, biguanides, sulfonylureas and thiazolidinediones there is as yet a need to search for new medications as no medication (aside from severe glycaemic the executives with insulin) has been appeared to control diabetic inconveniences viably. Corresponding to plants additionally, excepting a couple of studies, the majority of the investigations have not evaluated the effect of these plants on the course of diabetic inconveniences. It is important to assess plant species generally utilized against diabetes mellitus to find new mixtures that can be utilized adequately against this illness just as diminish diabetic complexities.

The human pancreas is fundamentally made out of two sorts of secretory cells that are both associated with supplement taking care of: 98% of the cells-the exocrine

kind – emit a food handling chemical bicarbonate combination into the duodenum, while the excess 2% - the endocrine kind have a metabolic capacity and discharge a combination of supplement created chemicals into the gateway vein. This little endocrine part is of essential significance in keeping up glucose homeostasis through the activity of the 51-amino corrosive peptide insulin. Four endocrine cell types can be recognized: A cells (alpha), B cells (beta), D cells (delta) and PP cells Bandodkar (1997).

These endocrine cells are appropriated all through the pancreas in zones known as islets.

The islet changes, from a morphological perspective, related with different sorts of diabetes can be isolated into those with and without serious beta-cell misfortune. Serious beta-cell misfortune is found in type I diabetes and some extraordinary types of diabetes, for example, infection related diabetes and inborn diabetes. Islets without extreme loss of beta-cells are experienced in type II diabetes and in the auxiliary types of diabetes (Klöppel and In't Veld, 1997) Insulin The beta-cells of the pancreatic islets integrate insulin from a solitary chain antecedent of 110 amino acids named preproinsulin. After movement through the film of the unpleasant endoplasmic reticulum, the 24-amino-corrosive N-terminal sign peptide of preproinsulin is quickly divided off to shape proinsulin. Here the particle folds and the disulfide bonds are framed. On the transformation of human proinsulin to insulin in the Golgi-unpredictable, four essential amino acids and the excess connector or C peptide are eliminated by proteolysis. This brings about the two-peptide chains (An and B) of the insulin atom, which contains one intra-subunit and two between subunit disulfide bonds. The A chain for the most part is made out of 21 amino acids and the B chain 30. The two chains of insulin structure a profoundly requested design with a few helical districts in both the An and B chains . Two particles of Zn^{2+} are composed in a proinsulin hexamer and this type of insulin apparently is put away in the granules of the pancreatic β cells. It is accepted that Zn^{2+} has a 21 Chapter 1 utilitarian job in the arrangement of gems and that crystallization encourages the transformation of proinsulin to insulin, just as the capacity of the chemical Rogers (1996).

Insulin is appeared as the concealed peptide chains, An and B (1996). Insulin emission Insulin is delivered from pancreatic β -cells at a low basal rate and at a lot higher rate in light of an assortment of upgrades, particularly glucose.

Hyperglycaemia brings about expanded intracellular ATP (adenosine triphosphate) levels, which close the ATP-subordinate potassium channels. Diminished outward potassium current through this divert brings about depolarization of the β -cell and the launch of voltage-gated calcium channels. The subsequent expanded intracellular calcium triggers the emission of the chemical Insulin discharge Insulin is delivered from pancreatic β -cells at a low basal rate and at a lot higher rate in light of an assortment of improvements, particularly glucose. Hyperglycaemia brings about expanded intracellular ATP (adenosine triphosphate) levels, which close the ATP-subordinate potassium channels. Diminished outward potassium current through this divert brings about depolarization of the β -cell and the launch of voltage-gated calcium channels. The subsequent expanded intracellular calcium triggers the emission of the chemical .Insulin corruption The liver and kidney are the two primary organs that eliminate insulin from dissemination, probably by hydrolysis of the disulfide association between the An and B chains through the activity of glutathione insulin transhydrogenase (insulinase). After this reductive cleavage further debasement by proteolysis happens. The liver ordinarily gets the blood free from Insulin granules Glucose carrier Glucose Myosin fiber Metabolism ATP (Closes depolarization) K^+ Channel Ca^{2+} Channel (Depolarization opens) . Model of the control of insulin discharge from the pancreatic β -cells by glucose Hibbard(2001).

Roughly 60% of the insulin delivered from the pancreas by righteousness of its area as the terminal site of the entry vein blood stream, with the kidneys eliminating 35 – 40% of the endogenous chemical. Be that as it may, in insulin-treated diabetics getting subcutaneous insulin infusions, this proportion is switched, with 60% of exogenous insulin being cleared by the kidney and the liver eliminating close to 30-40%. The half-existence of flowing insulin is 3-5.

CHAPTER 2

AIM AND SCOPE

2.1 AIM

Electrochemical investigation of sweat utilizing delicate bio hardware on human skin gives another course to noninvasive glucose observing without agonizing blood assortment and gauge the blood glucose fixation. Diabetes is brought about by hyperglycemia and hypoglycemia. High glucose fixation causes constant sicknesses and low glucose focus causes stun or demise. Thusly it is vital to forestall both high glucose and low glucose fixation. Sweat contains glucose and the perspiration glucose focus has a very decent relationship with blood glucose fixation. To improve precision we utilized distinctive sort of sensors like temperature sensor ,mugginess sensor, current sensor and we measure glucose just as temperature at the same time. Subsequent to discovering the glucose fixation we need to control the high glucose focus by conveying a proper measure of medication easily and quickly . The framework gives a novel shut circle answer for the noninvasive perspiration based administration of diabetes mellitus.

2.2 SCOPE

To screen the glucose screen framework.

However, patients regularly don't follow this suggestion due to the torment and going with extreme pressure of monotonous blood assortment and insulin shots.

This regularly prompts different serious diabetic intricacies, like cardiovascular and kidney illnesses, stroke, visual deficiency, and nerve degeneration.

furthermore, insulin overtreatment causes a sudden drop in the blood glucose fixation, which may cause seizures, obviousness, and even passing.

Therefore, a novel technique for easy and tranquil glucose observing and exact support of homeostasis through controlled medication conveyance is profoundly attractive.

CHAPTER 3

MATERIALS AND METHODOLOGY

3.1 BLOCK DIAGRAM

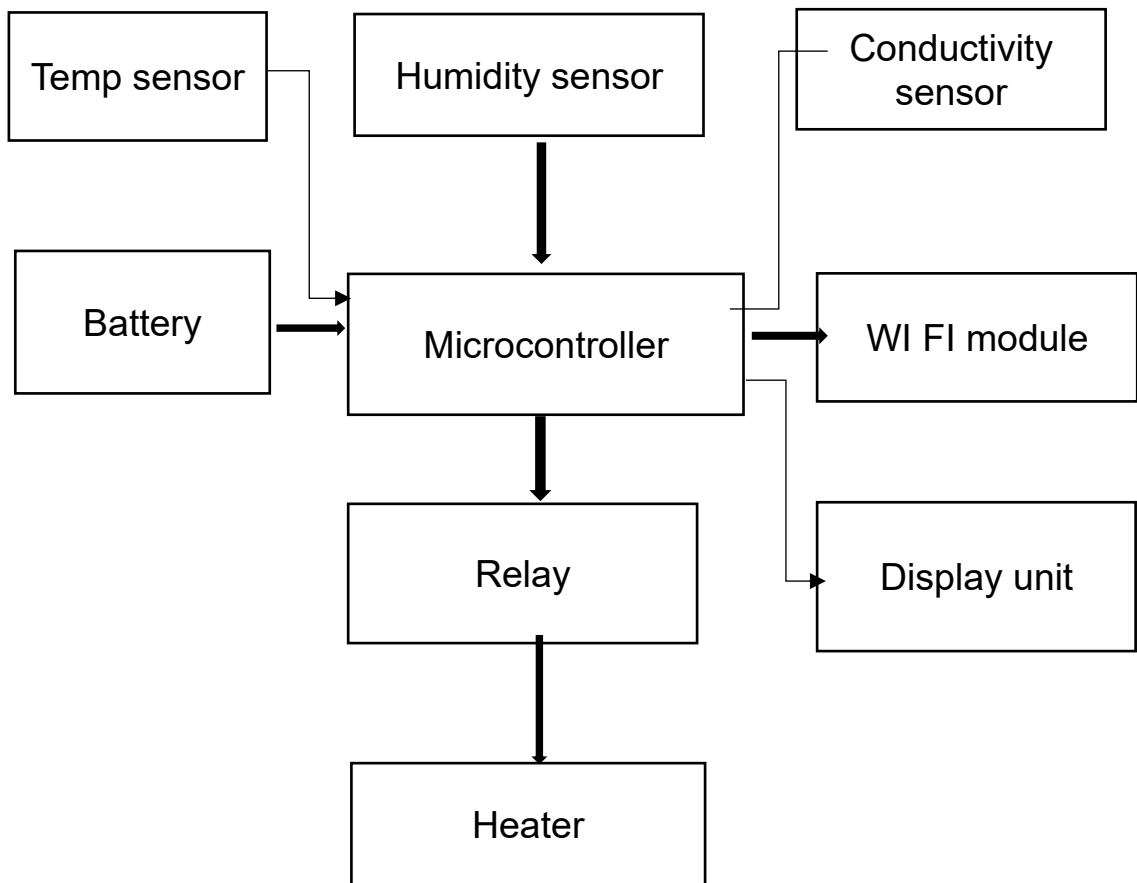


Fig:3.1 Block diagram of sweat based system

3.2 FLOWCHART

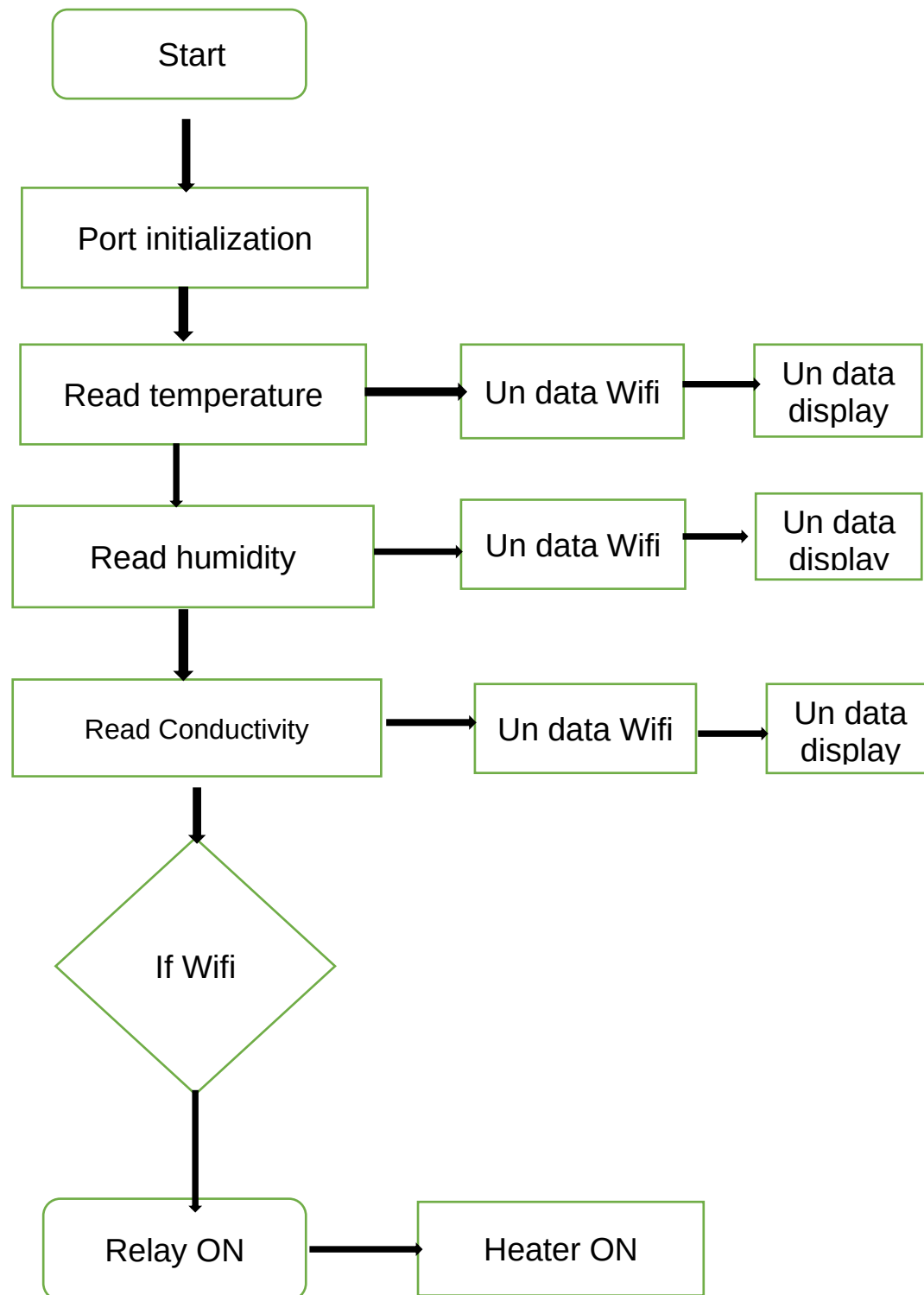


Fig:3.2 Flowchart of sweat based system

3.3 CIRCUIT DIAGRAM

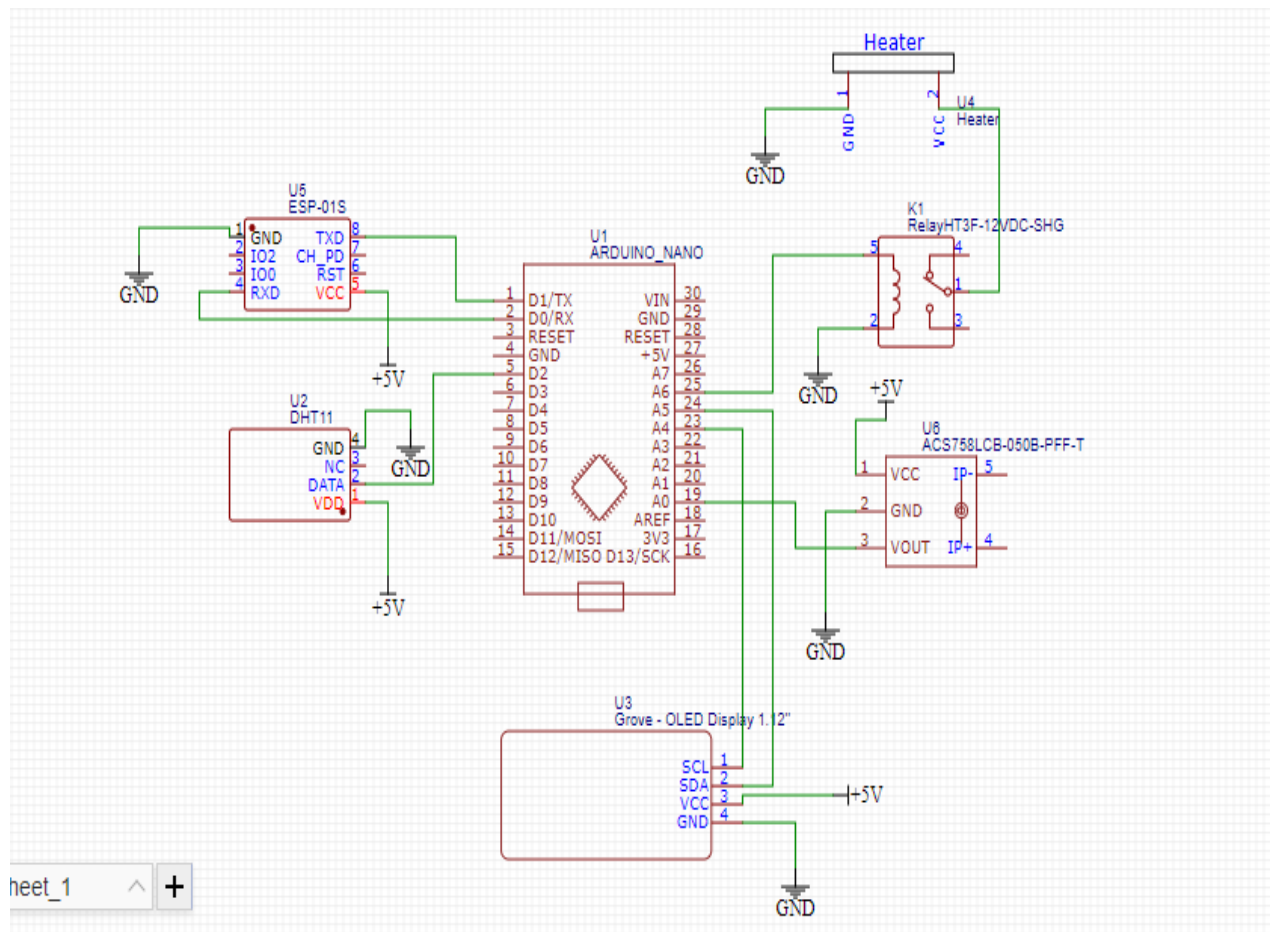


Fig:3.3 Circuit diagram of sweat based system

3.4 MATERIALS

3.3.1 Arduino nano

The Arduino Nano, as the name proposes is a minimal, complete and bread-board well disposed microcontroller board. The Nano board weighs around 7 grams with measurements of 4.5 cms to 1.8 cms (L to B). This article examines about the specialized specs in particular the pinout and elements of every single pin in the Arduino Nano board.

Table 3.1 Arduino nano

Model Type	<i>Nano</i>
Color	<i>Blue</i>
Microcontroller	<i>ATmega328</i>
Operating Voltage (VDC)	<i>5</i>
Input Voltage(Recommended)	<i>7-12 V</i>
Power Consumption (Watt)	<i>1</i>
Analog I/O Pins	<i>8</i>
Digital I/O Pins	<i>22 (6 of which are PWM)</i>
Clock Speed	<i>16 MHz</i>
Flash Memory	<i>32 KB</i>
EEPROM	<i>1 KB</i>
Architecture	<i>AVR</i>

3.3.2 Humidity sensor

Humidity is the convergence of water fume present noticeable all around. Water fume, the vaporous condition of water, is for the most part imperceptible to the natural eye. Stickiness demonstrates the probability for precipitation, dew, or mist to be available. Dampness relies upon the temperature and pressing factor of the arrangement of interest. A similar measure of water fume brings about higher stickiness in cool air than warm air.

Table 3.2 Humidity Sensor

<i>Humidity Measuring Range(%)</i>	<i>20 to 90</i>
<i>Temperature Measuring Range(°C)</i>	<i>0 to +50</i>
<i>Humidity Measurement Accuracy(%)</i>	<i>±5.0</i>
<i>Temperature Measurement Accuracy(°C)</i>	<i>±2.0</i>
<i>Response Time(s)</i>	<i><5</i>
<i>Input Supply Voltage (VDC)</i>	<i>5</i>

3.3.3 Temperature sensor

A temperature sensor is a gadget, ordinarily, a thermocouple or opposition temperature identifier, that gives temperature estimation in a comprehensible structure through an electrical sign. A thermometer is the most essential type of a temperature meter that is utilized to gauge the level of hotness and coolness.

Table 3.3 Temperature Sensor

<i>Model</i>	<i>DS18B20</i>
<i>Operating Voltage (VDC)</i>	<i>3 ~ 5.5</i>
<i>Operating Temperature (°C)</i>	<i>-55 to 125</i>
<i>Accuracy</i>	<i>±0.5°C</i>
<i>Cable Length (Meter)</i>	<i>1</i>

3.3.4 current sensor module

A flow sensor is a gadget that distinguishes electric flow in a wire and creates a sign corresponding to that flow. The created sign could be simple voltage or current or a computerized yield.

Table 3.4 Current Sensor

<i>Current sensor chip</i>	<i>ACS712ELC-5A</i>
<i>Operating Voltage (V)</i>	<i>4.5V ~ 5.5V DC</i>
<i>Measure Current Range</i>	<i>-5 ~ +5A</i>
<i>Sensitivity</i>	<i>100mV/A</i>
<i>Length (mm)</i>	<i>32</i>
<i>Width (mm)</i>	<i>13</i>
<i>Height (mm)</i>	<i>13.5</i>

3.3.5 0.96" OLED Display Module – SPI/I2C

The SSD1306 Oled show Module has a sum of 4 pins named as GND, VCC, SCL, and SDA. This Oled show Module can be effortlessly fueled up utilizing 3.3V to 5V. This is a low force show module and can be effortlessly utilized with the Arduino sheets utilizing 5 volts, and can likewise be utilized with 3.3V regulator sheets like ESP8266 and ESP32, and so on.

Table 3.5 Display Module

<i>Driver IC</i>	<i>SSD1306</i>
<i>Pixel Resolution</i>	<i>128 x 64</i>
<i>Operating Voltage (VDC)</i>	<i>3.3 ~ 5</i>
<i>Operating Temperature (°C)</i>	<i>-30 to 60</i>
<i>Character Color</i>	<i>Blue</i>
<i>Background Color</i>	<i>Black</i>
<i>Length (mm)</i>	<i>27</i>
<i>Width (mm)</i>	<i>27</i>
<i>Height (mm)</i>	<i>4.2</i>

3.3.6 Wi-fi module

It is comprised of three components which are fundamental for its working. The radio signs are sent by receiving wire and switches and they are gotten by Wi-Fi collector like PCs. Wi-Fi module has 8 pin outs which are organized in 2 lines. Keeping the incorporated side confronting you and pins nearer to your end. Lower line from left to right is

Table 3.6 Wi-fi module

Package/Case	<i>DIP-18</i>
Antenna Type	<i>PCB</i>
Transmit Power (dBm)	<i>802.11b: 16±2 dBm (@11Mbps) 802.11g: 14±2 dBm (@54Mbps) 802.11n: 13±2 dBm (@HT20, MCS7)</i>
Receiving Sensitivity	<i>54 Mbps (3/4 64-QAM): -70dBm 6 Mbps (1/2 BPSK): -88dBm CCK, 1 Mbps : -90dBm CCK, 11 Mbps: -85dBm HT20, MCS7 (65 Mbps, 72.2 Mbps): -67dBm</i>

UART Baudrate	Support 300 ~ 4608000 bps 'Default 115200 bps
Frequency Range	2412 ~ 2484MHz
IO Port	11
Security	WEP/WPA-PSK/WPA2-PSK
Storage Environment	-40 º ~ 90 º , < 90%RH
Interface	UART/GPIO/PWM/ADC
Operating temperature	-20 º ~ 85 º
SPI Flash	built-in 1Mbyte
Power Supply Voltage	3.0V ~ 3.6V'Current >300Ma

3.3.7 Small heater

If strip heaters are used as a radiant heater, they have can have fins to maximize surface area and heat transfer to the air. Strip heaters can be clamped or bolted onto objects or solid surface contact heating over large areas and are primarily used in indoor applications.

Table 3.7 Heater

Resistance Range (Ω)	4.2 to 5.7
Diameter (mm)	6
Length (mm)	20
Weight (gm)	28
Cable Length (Meter)	1

3.3.8 RELAY MODULE

Transfer Module A hand-off is essentially a switch which is worked by an electromagnet. The electromagnet requires a little voltage to get actuated which we will give from the Arduino and whenever it is initiated, it will pull the contact to make the high voltage circuit. The hand-off module we will utilize is the SRD-05VDC-SL-C.

Table 3.8 Relay Module

No. of Channels	1
Trigger Voltage (VDC)	5
Trigger Current (mA)	20

<i>Switching Voltage (VAC)</i>	250@10A
<i>Switching Voltage (VDC)</i>	30@10A
<i>Length (mm)</i>	38
<i>Width (mm)</i>	26
<i>Height (mm)</i>	18
<i>Weight (gm)</i>	13

CHAPTER 4

RESULTS AND DISCUSSION

4.1 RESULTS

In the wake of discovering the glucose fixation we need to control the high glucose focus by conveying a fitting measure of medication easily and quickly .The framework gives a novel shut circle answer for the noninvasive perspiration based administration of diabetes mellitus.

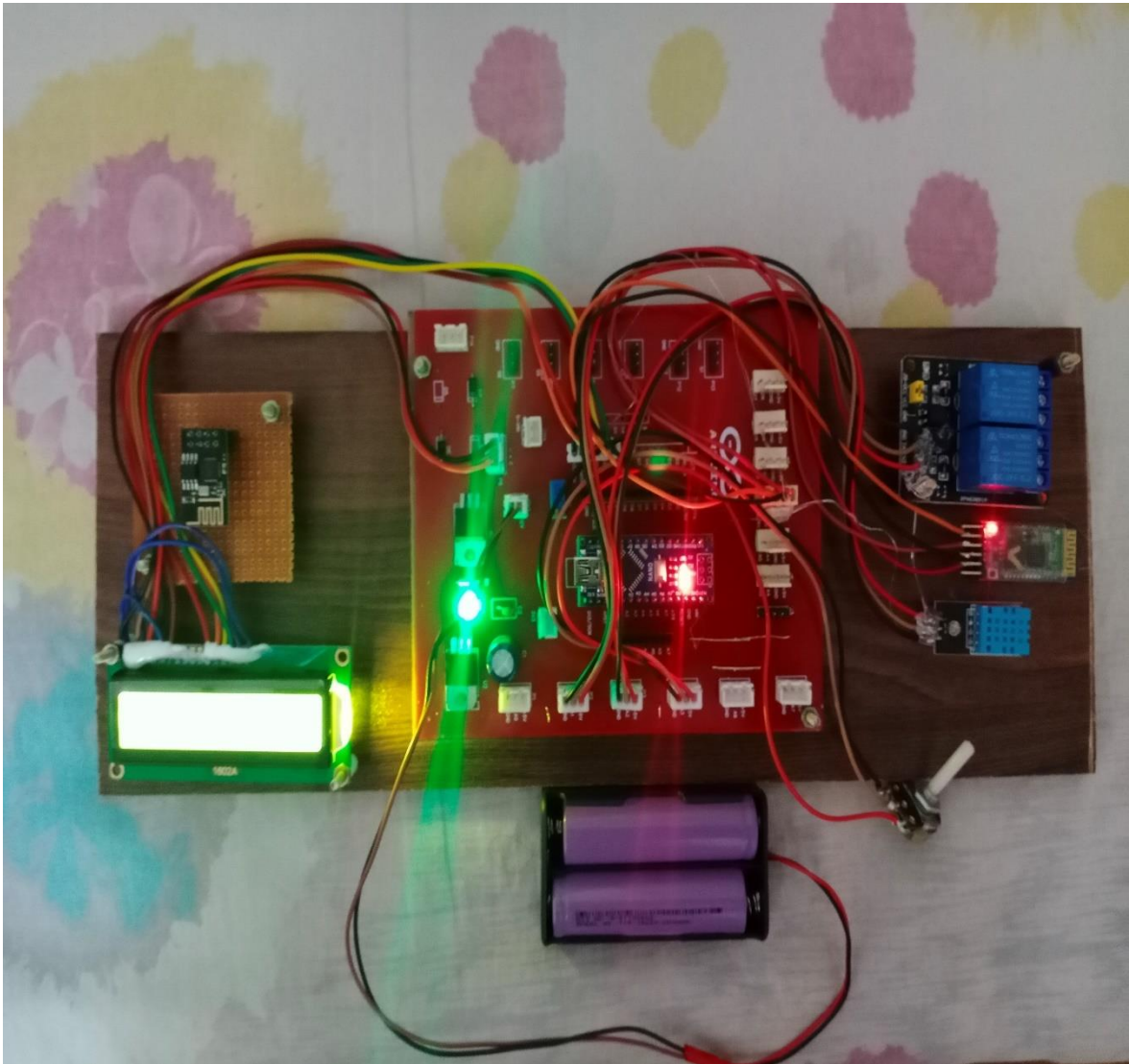


Fig:4.1 Prototype of sweat based system

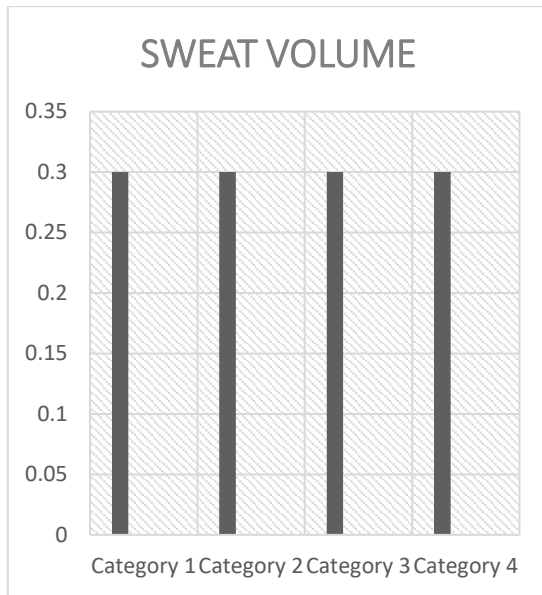


Fig:4.2 Sweat Volume of Patient

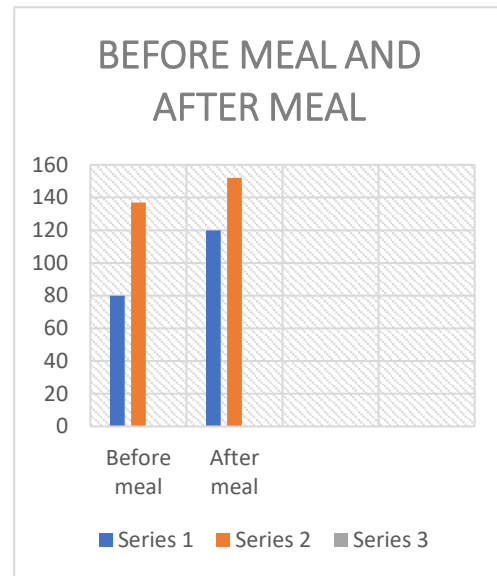


Fig:4.3 Before meal and After meal

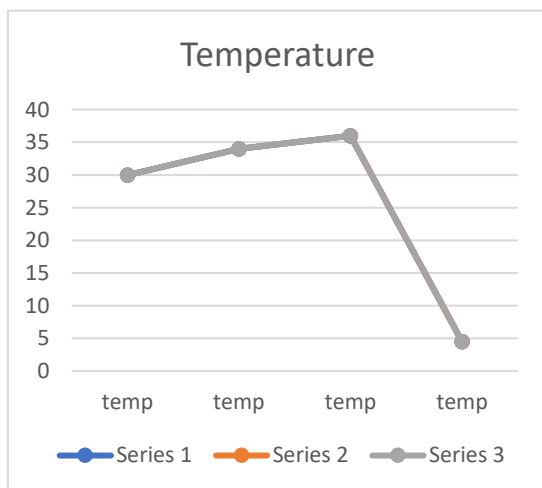


Fig:4.4 Temperature of patient

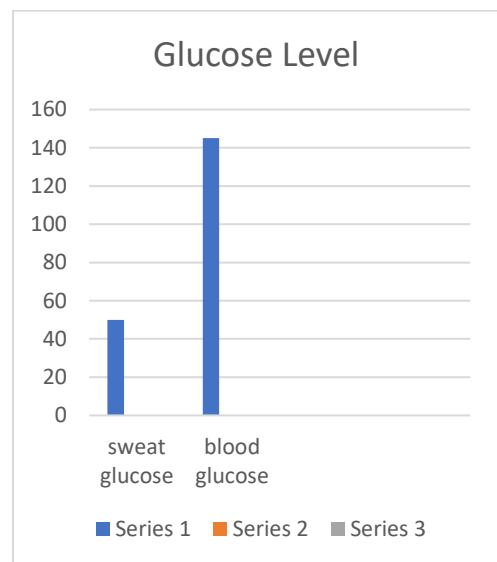


Fig:4.5 Glucose and Sweat level

Table 4.1 Data Collections of Sweat Based Glucose Monitoring

AGE	GENDER	TEMPERATURE	HUMIDITY	GLUCOSE CONCENTRATION
47	Female	34	67	131
49	Female	34.6	82	147
53	Male	37.3	71	149
55	Male	36.9	78	155
57	Male	35.2	57	123
16	Female	36.9	69	140
39	Male	38.5	52	121
66	Female	36.6	98	167
37	Female	37.2	51	119
58	Female	38.1	44	113
33	Female	35.8	59	125
66	Male	36.1	64	130
63	Female	37.2	82	149
59	Female	36.5	59	126

4.2 DISCUSSION

Here, I report a material construction, gadget plan, and framework combination procedure for a perspiration glucose checking gadget coordinated with criticism transdermal medication conveyance microneedles. Contingent upon the plan, the gadget can be either wearable-fix type or dispensable strip type. For effective perspiration control and detecting, the perspiration observing patch is gathered with different perspiration take-up and waterproof layers, and sensor sizes are scaled down to the point that ~1 ml of sweat is adequate for solid estimation. The estimations of sweat glucose levels with ongoing adjustment dependent on pH, temperature, and mugginess detecting are precise under different climate changes. The perspiration glucose information are very much associated with the blood glucose levels. For exact and conven .The thermo responsive microneedles constrained by multichannel warmers empower the multistage and spatially designed transdermal medication discharge because of the deliberate perspiration glucose level. For down to earth use of the current framework, there are a few things to improve. The drawn out strength and consistency of sensors are especially critical

to make the framework essentially material to human subjects with least recalibrations. Further investigations about the relationship between's glucose levels of blood and sweat are required before application to diabetic patients. Despite the fact that metformin utilized in the framework shows moderate concealment of the blood glucose levels because of its working component , another sort of medication (for instance, chlorpropamide) can be stacked for synergistic treatment or quick guideline of blood glucose level. With additional upgrades in these focuses, clinical interpretation can be sought after for sweat-based detecting and criticism treatment. The current framework gives significant new advances toward the easy and tranquil place of-care treatment of diabetes mellitus

CHAPTER 5

SUMMARY AND CONCLUSION

5.1 SUMMERY

Thus, for I have presented a technique that incorporates sweat based glucose observing with inbuilt medication conveyance framework alongside blood to such an extent that I am ready to discover glucose level. The current strategy included obtrusive technique which was not a simple technique. Likewise, there were numerous disadvantages for the current techniques.

In my proposed technique I have closed than it is effortless strategy and non intensive technique. Here I measure glucose just as temperature at the same time Furthermore, the dampness sensor screens the perspiration age measure

Subsequent to discovering the glucose focus we need to control the high glucose fixation by conveying a fitting measure of medication easily and quickly .The framework gives a novel shut circle answer for the noninvasive perspiration based administration of diabetes mellitus.

5.2 CONCLUSION

In the wake of discovering the glucose fixation we need to control the high glucose focus by conveying a fitting measure of medication easily and quickly .The framework gives a novel shut circle answer for the noninvasive perspiration based administration of diabetes mellitus.

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