

VITAL PULSE

VOL NO. 67

DIABETES

A-Z

SMALL CHANGES,
BIG IMPACT

YOUR DAILY
DIABETES
GUIDE

**WHAT IS
TYPE II
DIABETES**

THAT WILL CHANGE YOUR
LIFE

**THE RISE OF
NOVEL TREATMENT**

FEATURING

DR. NILUSNI

APRIL 2026



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What is Type II diabetes^{1,2}

Type 2 Diabetes (T2DM) is a metabolic disorder characterized by chronic hyperglycaemia, meaning persistently high levels of glucose in the blood. It occurs when the body becomes resistant to insulin and the pancreas cannot produce enough insulin to maintain normal blood glucose levels. The condition most often develops in middle and late adulthood due to impaired insulin action and regulation of blood glucose.

Persistently high blood sugar doesn't just affect energy, it also triggers chronic low-grade inflammation. Fat cells and blood vessel cells respond to excess glucose by releasing inflammatory cells and cytokines, which interfere with insulin's action and damage blood vessels.

What goes wrong in T2DM?²

- Decreased peripheral glucose uptake
- Delayed/reduced insulin secretion
- Inadequate suppression of glucagon after meals
- Defective suppression of endogenous glucose production in liver

Over time, this inflammatory cycle worsens insulin resistance and contributes to complications like heart disease, kidney damage, and nerve problems.

But, what does Diabetes Mellitus³ really mean?

The word “**diabetes**” comes from the Greek diabainein, meaning “to pass through” or “siphon,” to describe excessive urination.

The suffix “**mellitus**” is Latin for “honey-sweet” was used to describe the sweet-tasting urine.

6 WHYS TO INSULIN RESISTANCE

Insulin is a hormone that acts like a key, allowing glucose from bloodstream to enter cells and be stored. In T2DM, cells become resistant to insulin, and pancreas struggle to keep up with insulin demands, which leads to high blood sugars.

1. INSULIN SECRETION⁴

In Type 2 Diabetes, insulin is normally released in rhythmic pulses that help regulate blood glucose. When insulin is released in pulses, its concentration repeatedly rises and falls in the blood. These fluctuations are strongest in the portal vein going to the liver and smaller in the rest of the circulation.

Early in the disease, this pulsatile pattern becomes disrupted, and tissues also become less responsive to insulin. The combination of impaired insulin secretion and insulin resistance contributes to rising blood glucose levels.

2. GENETIC PREDISPOSITION⁵

Genetics plays an important role in the development of Type 2 Diabetes, although the disease results from a combination of genetic susceptibility and environmental factors such as diet, physical activity, and body weight.

Estimates suggest the heritability of T2DM ranges from 20–80%, indicating a strong inherited component. Individuals with one parent affected have about a 40% lifetime risk of developing the disease, which increases to around 70% if both parents are affected.

Large genetic studies have identified several genes associated with increased susceptibility to T2DM. Many of these genes influence β -cell function and insulin secretion rather than insulin signaling itself. Well-replicated examples include TCF7L2, KCNQ1, and KCNJ11, which are involved in glucose metabolism and insulin release. Despite these discoveries, much of the genetic basis of T2DM remains unexplained, highlighting the complex interaction between genetics and environmental factors in disease development.

3. OBESITY^{6,7}

Excess body weight is one of the strongest risk factors for Type 2 Diabetes. As body mass index (BMI) increases, the lifetime risk of developing T2DM rises dramatically—from approximately 7% to 70% in men and 12% to 74% in women. Conversely, weight loss has been shown to significantly reduce the incidence of diabetes among individuals at high risk.

Obesity⁶ results from the chronic consumption of calories in excess of the body's energy needs, leading to the excessive growth of adipose tissue (fat depots). However, not all fat affects metabolic health in the same way. Subcutaneous adipose tissue (SAT), located beneath the skin, can safely store excess lipids and is associated with a lower risk of insulin resistance. In contrast, visceral adipose tissue (VAT), which surrounds internal organs, is strongly associated with metabolic dysfunction.⁷



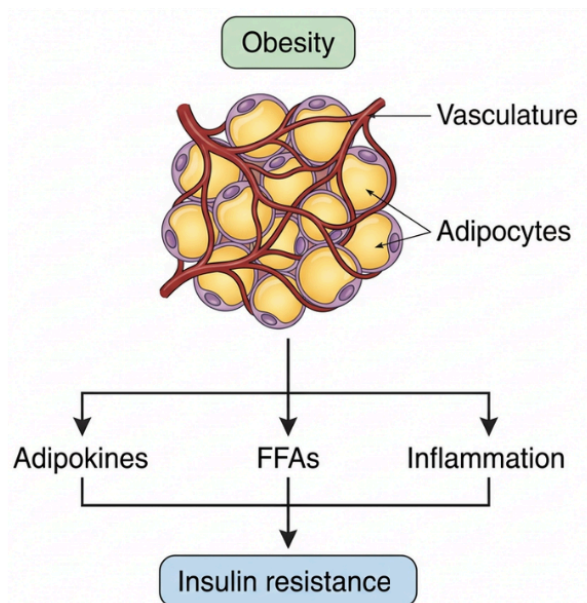


Fig. 1. Original pic from the slide⁸, Figure recreated using Gemini

4. INCRETIN DEFECT^{2,9}

After a meal, hormones called incretins are released from endocrine cells in the small intestine. These hormones, including Glucagon-like peptide-1 (GLP-1) and Glucose-dependent insulinotropic polypeptide (GIP), enhance insulin secretion from pancreatic β -cells and help regulate blood glucose levels.

In Type 2 Diabetes, the incretin effect is reduced due to impaired hormone release or decreased responsiveness of pancreatic β -cells. As a result, insulin secretion after meals is insufficient, contributing to elevated post-meal blood glucose levels.

How Visceral Fat Promotes Insulin Resistance⁷

1. Release of adipokines and inflammatory signals

Visceral adipose tissue releases signaling molecules known as adipokines, as well as inflammatory cytokines. These molecules interfere with insulin signaling in tissues such as muscle and liver, reducing the ability of insulin to stimulate glucose uptake.

2. Increased free fatty acids and lipotoxicity

Visceral fat releases large amounts of free fatty acids (FFAs) into the circulation. These lipids can accumulate in organs such as the liver and skeletal muscle (ectopic fat deposition), disrupting cellular metabolism and impairing insulin signaling. This process is known as lipotoxicity.

3. Chronic inflammation

As visceral fat expands, immune cells, especially macrophages, accumulate in the tissue and release inflammatory cytokines. This chronic low-grade inflammation further disrupts insulin receptor signaling and contributes to systemic insulin resistance in Type 2 Diabetes.

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VITAL PULSE

5. ENHANCED GLUCOSE ABSORPTION²

In Type 2 Diabetes, a larger amount of glucose from ingested food enters the bloodstream than normal. This happens because the small intestine absorbs glucose more efficiently, partly due to increased activity of transporters such as SGLT-1 and GLUT-2, which move sugar from the gut into the blood. At the same time, the liver is less able to take up and store this glucose, so more remains in circulation. The result is higher blood sugar after meals (postprandial hyperglycaemia), which places additional strain on pancreatic β -cells to produce insulin, worsening the overall disruption of glucose control.

6. CIRCADIAN RHYTHM¹⁰

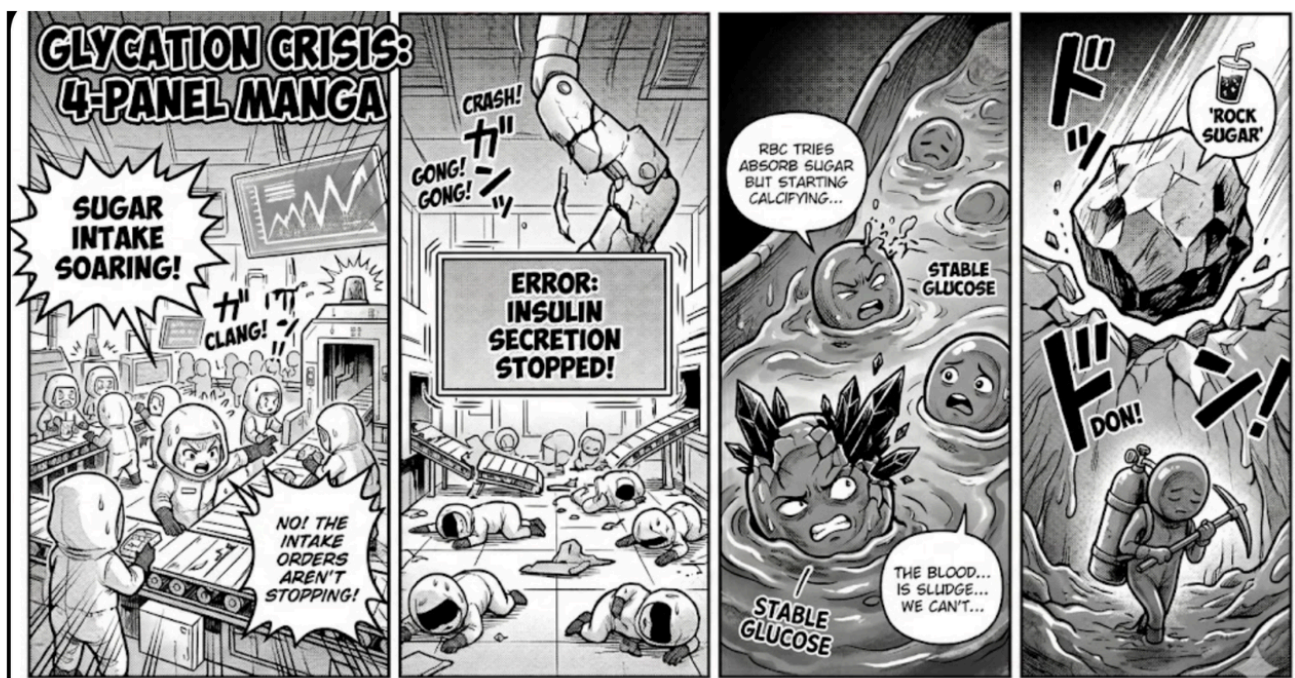
The body's internal biological clock regulates many metabolic processes, including glucose metabolism and insulin sensitivity. Disruption of the circadian misalignment occurs when the body's natural rhythm becomes out of sync with the external light-dark cycle. Irregular sleep patterns, shift work, and inconsistent eating schedules have been associated with an increased risk of Type 2 Diabetes. Adequate sleep and regular meal timing help maintain circadian regulation and support healthy glucose control.

CONNECTION TO ENDOCRINE¹¹

Endocrine disorders can interfere with insulin action and contribute to the development of Type 2 Diabetes.

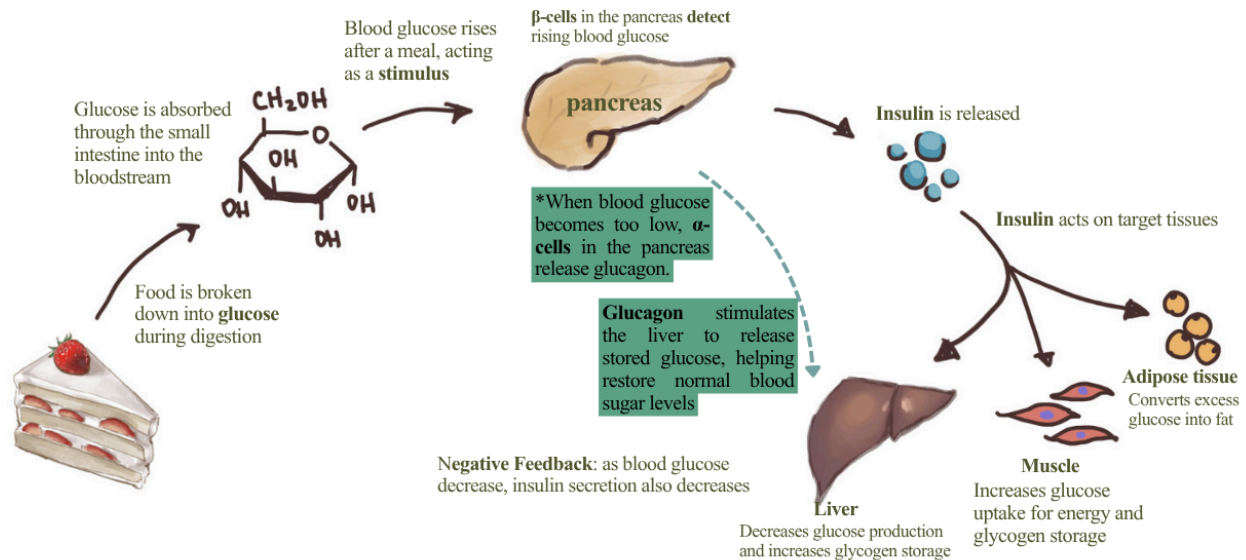
Excess hormones that oppose insulin can increase blood glucose levels and promote insulin resistance.

- **Hyperthyroidism**: increases metabolism and glucose production
- **Acromegaly**: excess growth hormone reduces insulin sensitivity
- **Hyperthyroidism** – increases metabolism and glucose production



Insulin in action: Normal vs T2DM⁸

Normal: Homeostasis reached



Homeostasis maintained by pancreas:
β-cells → Insulin → lowers blood glucose
α-cells → Glucagon → raises blood glucose

T2DM: Insulin resistance⁸

Insulin resistance

Cells in muscle, liver, and adipose tissue become less responsive to insulin. As a result, glucose is not taken up efficiently by cells.

Blood glucose remains elevated

Because tissues do not absorb glucose properly, glucose accumulates in the bloodstream. At the same time, the liver continues producing glucose, further increasing blood sugar levels.

Pancreatic compensation (normoglycemia)

The pancreas attempts to overcome insulin resistance by releasing larger amounts of insulin, leading to hyperinsulinemia. At this stage, glucose levels may appear relatively normal

Progression of Type 2 Diabetes as Pancreas Fails¹²

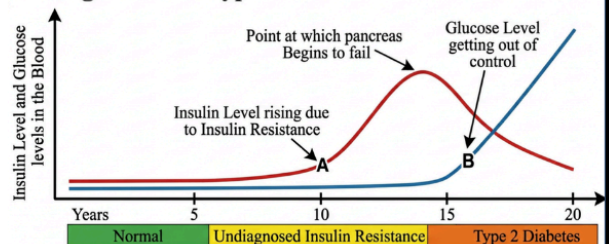


Fig 2 The chart illustrates the progression of Type 2 Diabetes, showing the relationship between glucose intake, insulin level and diabetes progress¹², figure recreated by Gemini

Early β-cell dysfunction (impaired glucose tolerance)

Over time, β-cells become stressed and gradually lose their ability to produce insulin effectively

Late β-cell dysfunction (Chronic hyperglycaemia)

Over time, β-cells become stressed and gradually lose their ability to produce insulin effectively

Test yourself!

During the "Pancreatic Compensation" phase of Type 2 Diabetes, why might a standard glucose test show "normal" results?

- The body has stopped absorbing glucose from food.
- The liver has stopped producing glucose entirely.
- The pancreas is overproducing insulin to overcome cell resistance.
- Muscle tissues have become more sensitive to insulin.

Correct Answer: C This is often called the "honeymoon" phase of diabetes because high insulin levels mask the underlying resistance by keeping the sugar levels temporarily stable.

The Thrifty Phenotype:

A Survival Strategy Gone Wrong

13

The thrifty phenotype hypothesis suggests that poor nutrition during fetal development or early infancy can increase the risk of developing Type 2 Diabetes later in life. When nutrients are limited during early development, the body adapts by becoming more efficient at conserving and storing energy.

While this adaptation may help survival in environments where food is scarce, it can become harmful if the individual is later exposed to high-calorie diets and abundant food. In such conditions, these metabolic adaptations may increase the risk of insulin resistance and T2DM.

Be the Doctor: The T2DM Clinic Challenge

Dr. Reader! the waiting room is full! Put on your white coat and use your knowledge of Type 2 Diabetes to help these patients.

Scenario 1: The Birthday Check-up

The Patient: Marcus, a healthy 36-year-old with no family history of diabetes, comes in for a routine physical. He says, "I feel fine, but my friend said I should start getting checked for diabetes now that I'm older."

The Clinical Question: Based on the 2026 American Diabetes Association (ADA) standards, how should you advise Marcus?

- A. Tell him he's too young and to come back when he's 45.
- B. Confirm that since he is over 35, he should begin regular screening.
- C. Only test him if he starts showing symptoms like extreme thirst.

Scenario 2: Reading the Lab Results

The Patient: Sarah's blood work just arrived on your desk. Her Fasting Plasma Glucose (FPG) is 7.2 mmol/L and her HbA1c is 6.8%.

The Clinical Question: What is your official diagnosis for Sarah based on the thresholds in our research?

- A. She is in the clear; these levels are normal.
- B. She has "Prediabetes" and needs lifestyle changes only.
- C. She meets the criteria for a Type 2 Diabetes diagnosis ($HbA1c \geq 6.5$ and $FPG \geq 7.0$ mmol/L).

Scenario 3: The "Tingling" Complaint

The Patient: An elderly patient who has lived with T2DM for 15 years complains of a "pins and needles" sensation in his feet and blurry vision.

The Clinical Question: You suspect damage to the small blood vessels. Which category of complications is this?

- A. Macrovascular complications —affecting the heart and brain.
- B. Microvascular complications —affecting the eyes, kidneys, and nerves.
- C. Acute etiology—a temporary side effect of too much exercise.

**Want more questions?
Check our website for
more interactive
questions!**

<https://ecsepheto.github.io/PathologySchoolWork/PULSE>



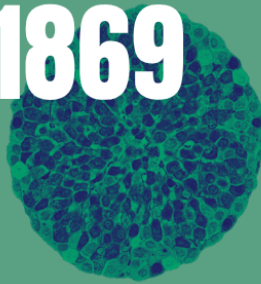
**Check
Your
Answer!**

Answer Key for the "Junior Doctor":
1. B (The ADA now recommends screening start at age 35).
2. C (Both her FPG and HbA1c are above the diagnostic threshold).
3. B (Damage to nerves and eyes are classic microvascular issues).

Discover Islets of Langerhans¹⁴

Paul Langerhans identified cells that came to be known as the “islets of Langerhans”

1869



Pancreas Involvement¹⁴

Von Mering & Minkowski found that the removal of pancreas lead to diabetes, establishing hypothesis about diabetes etiology.

1889



Naming of Insulin¹⁴

De Mayer & Schaefer later named the Islet of Langerhans' secretion “insulin,” drawing from the Latin “insula”, meaning “island.”

1909



Insulin Extraction¹⁴

At Macleod's lab, Banting, Best, and Collip tied off the pancreatic duct so the digestive part of the pancreas would shrink while the islets stayed intact. Using extracts from these dogs to reverse the diabetes they created, they showed that diabetes happens when the body lacks insulin.

1921



A BRIEF HISTORY OF

From the first discovery in 1869 to today

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1922

First Insulin Injection¹⁴

The first insulin injection was given to a 14-year-old boy and produced a modest drop in blood sugar. Similar encouraging results were soon seen in six other patients.



INSULIN



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1996

Short Acting Insulin¹⁴

Short-acting insulins work fast, reach their strongest effect early, and wear off quickly. The first one, Lispro, was approved in 1996, followed by Aspart in 2000 and Glulisine in 2004.



1993

Complications Control¹⁴

The Diabetes Control and Complications Trial demonstrated a clear linear relationship between improved glycemic control and reduced complications. Because hypoglycemia limits intensive therapy, researchers sought physiologic insulin regimens designed to mimic the body's natural basal and meal-related insulin secretion.

TCGGATT
CAGGTTT
CTTACCA
TTGGCTT

1978

Insulin in Genetic Tech¹⁴

David Goeddel and his team at Genentech produced the first human insulin using recombinant DNA technology joining the A and B chains made in *E. coli*. Later, Graham Bell sequenced human insulin in 1980, and by 1982 the first recombinant insulin was on the market.



1930s

Long- Acting Animal Insulin¹⁴

To make insulin last longer in the body, H. C. Hagedorn added protamine, and Scott and Fisher later added zinc. These changes produced longer-acting animal insulins that could work for about 24–36 hours.

SPECIAL Ed. The Medical Revolution of

Frederick Banting

In the early 1920s, the Canadian surgeon Frederick Banting came up with a bold plan to solve Type II Diabetes disease.¹⁵

He believed the key lay in isolating a specific substance from the pancreas. By carefully tying off a duct in the organ, he allowed certain parts to fade away while keeping the life-saving cells he needed intact.¹⁵

To test his theory, Banting teamed up with Professor John Macleod at the University of Toronto, who provided a laboratory and a talented assistant named Charles Best. Their work began in the spring of 1921, and by late summer, they achieved a breakthrough: they proved that their new extract could rapidly lower blood sugar levels.¹⁶

Soon after, biochemist J.B. Collip joined the team to refine the extract so it could be safely used on people. Their discovery was so revolutionary that it earned the team a Nobel Prize in 1923, forever changing diabetes from a death sentence into a manageable condition.¹⁶

Portrait of Frederick Banting [16].

Insulin Today

Doctors and patients often delay starting insulin for months or years due to fear of needles or outdated myths. However, insulin is still necessary when the pancreas stops making enough insulin and pills are no longer enough.¹⁷

TAILORED TREATMENT ¹⁷

Long-Acting Insulin

Also known as the “background insulin”. Think of this as your safety net. Taken just once or twice a day, it provides a steady, flat stream of insulin that works behind the scenes to keep your sugar levels stable overnight and between your meals.

Fast-Acting Insulin

Also known as the “mealtime supporter”, these versions are designed for speed. Taken right before you eat, they tackle the sudden surge of sugar that follows a meal. For those who dread needles, options like Afrezza—a fast-acting inhaled powder—offer a needle-free alternative.

Pre-mixed Insulin

For those seeking simplicity, pre-mixed insulin provided an “all-in-one” shots, which combines both background and mealtime insulin into a single dose, reducing the number of daily injections.



BEYOND SYRINGE ¹⁷

Digital Smart Pens

These pens do the thinking for you. They record the timing of your last dose, calculate exactly how much you need for your next meal, and sync all that data directly to your smartphone.

Patch Pumps

These small devices stick directly to your skin. They provide constant background insulin and allow you to deliver mealtime doses with a simple button click through your clothing.

Automated Systems

The “gold standard” of care, these devices talk to your glucose monitor 24/7. They automatically adjust your insulin levels in real-time, significantly reducing the risk of sudden sugar crashes or spikes.

MANAGE TRADE-OFFS ¹⁷

Weight Control

While insulin can cause weight gain and low blood sugar, modern formulas significantly reduce the frequency of low blood sugar episodes. Additionally, weight gain can be effectively managed by pairing the lowest effective insulin dose with a focused diet and exercise routine.

Do You Know the Signs?

THE 3 P'S

1. POLYURIA¹⁸

When the blood glucose exceeds the kidney's reabsorption threshold, it spills into urine and drags water with it, causing osmotic diuresis.

2. POLYDIPSIA¹⁹

The fluid lost through polyuria triggers persistent thirst. The body attempts to replace what the kidneys are expelling.

3. POLYPHAGIA¹⁹

Glucose floods the bloodstream but cannot enter insulin-resistant cells in muscle and fat.

INSULIN RESISTANCE^{19,20}

Cells in muscle, liver, and adipose tissue become less responsive to insulin. Glucose is not taken up efficiently by cells.

FATIGUE & WEAKNESS^{18,21}

Muscle and fat cells cannot extract glucose for fuel.

BLURRED VISION^{18,22}

High glucose draws fluid out of the lens of the eye by osmosis causing distortion of its shape and reducing its ability to focus.

WEIGHT LOSS²³

Inability to use glucose causes the body to break down fat and muscle as fuel

SLOW WOUND HEALING^{19,21}

Elevated glucose impairs immune cell function and creates a sugar rich environment where pathogens thrive. Poor circulation impacts healing especially in the lower limbs.

ACANTHOSIS NIGRICANS¹⁸

A visible skin sign where skins near folds thicken and become dark and velvety.

CLINICAL MANIFESTATIONS



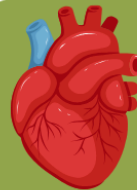
Microvascular- Retinopathy^{18,25,26}
sustained hyperglycemia leads to retinal blood vessel damage. It causes blurry vision and can be detected by routine dilated eye examination



Increase risk of stroke, cerebralvascular diseases.¹⁸



Microvascular - Nephropathy^{18,25,26}
Damaged to glomerular capillaries leading to renal disease that can be worsened by hypertension. A urine albumin to creatinine ratio and eGFR can be used to detect nephropathy.



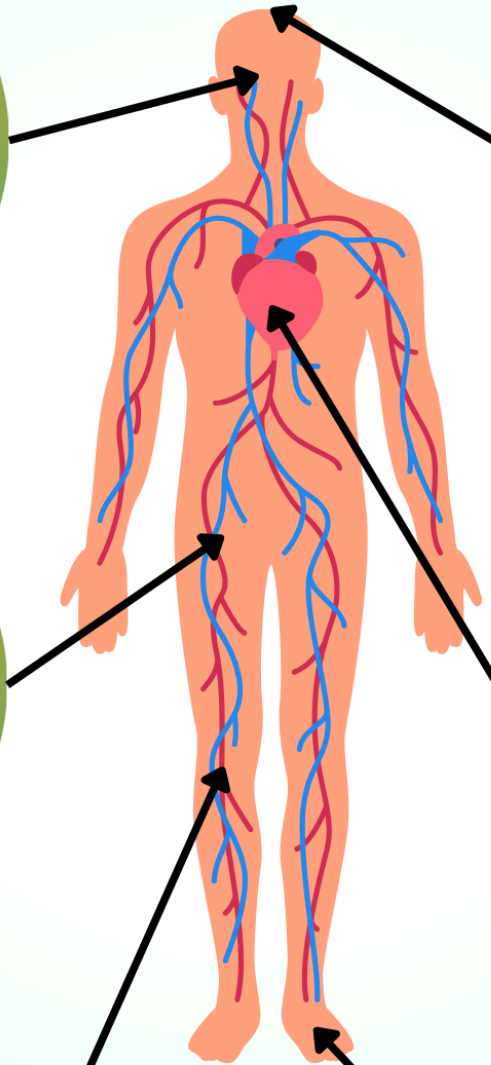
Macrovascular- heart and blood vessels¹⁸
Increased risk of coronary artery disease



Neuropathy^{18,25,26}
Tingling and numbness in the body especially the lower extremities. Pulses, reflexes and pinprick sensation are used to detect neuropathy



Extremities¹⁸
The combination of neuropathy and poor circulation leads to delayed wound healing and foot ulcers. Foot examinations are essential for T2DM patients



T2DM RISK: ATHEROSCLEROSIS

HOW IT HAPPENS

THE BUILD UP OF
PLAQUES INSIDE
THE WALLS OF THE
ARTERIES, THE
LEADING KILLER OF
PEOPLE WITH T2D



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ENDOTHELIAL DAMAGE²⁷

high blood sugar injures the inner lining of the blood vessels causing it to become leaky. Fats and immune cells are able to seep into the artery wall and start plaque formation

AGES- STICKY SUGAR COMPOUNDS²⁸

Prolonged elevated glucose levels causes the glucose to stick to proteins forming advanced glycation end products (AGEs). They convert immune cells into “foam cells” which are a key component of plaques

OXIDATIVE STRESS²⁹

Excess glucose in cells causes the mitochondria to overproduce reactive oxygen species (ROS). They go on to continue damage to vessel walls, oxidize LDL cholesterol into a harmful form and cause plaques to become more fragile and prone to rupture

DIABETIC DYSLIPIDAEMIA²⁷

The increase levels of harmful blood fats and decrease of HDL causes more plaque formation

CHRONIC INFLAMMATION³⁰

Insulin resistance triggers a low grade inflammatory state that drives plaque growth.

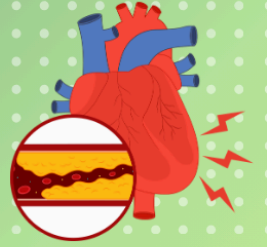
INSULIN RESISTANCE IN BLOOD VESSELS²⁸

Vascular cells become resistant to insulin’s protective effects which impairs normal vessel function, raises blood pressure, and creates the ideal conditions for plaques to grow

SO WHAT HAPPENS?

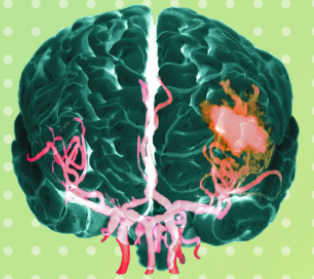
CORONARY ARTERY DISEASE³¹

Plaques narrow the arteries that supply the heart raising the risk of chest pain and heart attack



STROKE³¹

Plaques in vessels that supply the brain can block blood flow and trigger a stroke



PERIPHERAL ARTERY DISEASE³²

Narrowed arteries in the legs can cause pain when walking, poor wound healing, and sometimes lead to amputation



UNSTABLE PLAQUES²⁹

Plaques in some patients tend to be more vulnerable to rupture than in non-diabetics, leading to severe cardiac events.



01 INSULIN RESISTANCE^{33,34}

The insulin receptor signaling cascade is impaired when cells from the muscles, liver, and adipose tissue stop responding normally to insulin. Glucose cannot enter the cells even when insulin is present. In the liver, insulin stimulates the suppression of glucose release, but insulin resistance prevents the suppression, leading to more glucose being poured into the bloodstream. Excess visceral fat from obesity accelerates this process by flooding the circulation with free fatty acids (FFAs) and inflammatory cytokines that interfere with insulin signaling.

03 BETA-CELL DYSFUNCTION AND FAILURE^{36,37}

After prolonged exposure to glucotoxicity and lipotoxicity, beta-cells are placed in a state of oxidative stress. Pro-inflammatory cytokines accumulate and drive beta-cell apoptosis. By the time T2DM is diagnosed, more than 50% of beta-cells are lost. This leads to increased blood glucose levels.

02 COMPENSATORY HYPERINSULINAEMIA^{35,36}

The pancreas detects rising glucose levels and responds by producing more insulin. This boost works for a while, but over time the beta-cells become strained.

PATHO *GE*

04 HEPATIC GLUCOSE OVERPRODUCTION^{33,38}

With ongoing insulin resistance, the liver's glucose output stops responding properly. Gluconeogenesis remains active, and free fatty acids promote non-alcoholic fatty liver disease, which blocks insulin signals and pushes glucose levels higher.

08 GENETICS AND ENVIRONMENT³⁸

T2DM has a strong heritable component. Genes affecting beta cell function and insulin receptor signaling contribute to the risk of developing T2DM. Epigenetic factors also allow the same genetic backgrounds to produce very different outcomes depending on lifestyle. A high-calorie diet, physical inactivity, obesity, and aging all contribute to the susceptibility for T2DM.

NESIS

05 GLUCAGON DYSREGULATION³⁸

In healthy metabolism, glucagon is suppressed after meals. Glucagon instructs the liver to release glucose, so in T2DM this suppression fails. Glucagon remains elevated even when blood sugar levels are high, leading to a continuing increase in glucose production.

07 GUT MICROBIOTA DYSBIOSIS⁴⁰

The gut microbiome is reduced in T2DM, especially the butyrate-producing species that are important in GLP-1 pathway activation. This leads to reduced GLP-1 activation and impairs gut barrier integrity, allowing bacterial lipopolysaccharides to leak into the bloodstream, causing inflammation. This poses a worsening of insulin resistance.

06 INCRETIN DEFECT³⁹

Gut cells release incretin hormones GLP-1 and GIP after meals. These secretions are reduced in T2DM, and beta cells become resistant to the little incretin still produced. This affects insulin secretion and glucagon suppression.

WHO IS AFFECTED?

WHO SHOULD BE SCREENED?

DIAGNOSTICS

TAKING NEXT STEPS

Diagnosis^{41,42}

HbA1c (Glycated Haemoglobin)

- An HbA1c level of $>6.5\%$ meets the threshold for a diagnosis.

Fasting plasma glucose

- Diabetes is diagnosed at a fasting blood glucose of greater than or equal to 126 mg/dL. The patient must fast for at least 8 hours prior to the test

Oral glucose tolerance test

- This 2 hour test checks blood glucose levels before and after the patient drinks a special sweet drink. It tells clinicians how the body processes sugar. diabetes is diagnosed at a 2 hour blood glucose of greater than or equal to 200mg/dL

Prediabetes⁴³

Pre-T2DM, many patients pass through a stage of prediabetes. It is defined by the presence of impaired fasting glucose and/or impaired glucose tolerance. Prediabetes is a significant risk factor for progression to diabetes as well as cardiovascular disease.

Who Should Be Screened?⁴³

Individuals without risk factors, testing should commence at the age of 35. Testing for prediabetes should be considered for adults that are overweight or have more additional risk factors. Risk factors include hypertension, dyslipidaemia, family history, prior gestational diabetes, polycystic ovary syndrome, and certain racial/ethnic backgrounds.

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If you are prepared to make the
sacrifices, think and work hard enough
and long enough.

— Frederick Banting



THE FRONT-LINE LIFESTYLE CHANGES ⁴⁴

Weight Management

The most effective method to prevent Type 2 diabetes involves aiming for a five to seven percent reduction in body weight. This is best paired with over 150 minutes of moderate weekly exercise, such as brisk walking.

Dietary Choices

Adopting eating patterns like the Mediterranean, plant-based, or low-carbohydrate diets yields strong results. The primary focus must remain on consuming whole foods while strictly minimizing processed items.

Prevention Programs

Enrolling in a structured diabetes prevention program provides necessary guidance. These frameworks are readily accessible in-person, online, or through dedicated digital applications.

Sleep Quality

Securing approximately seven hours of high-quality sleep is critical. Identifying as a night owl increases diabetes risk by 2.5 times. Furthermore, sleeping fewer than six hours or more than nine hours can spike the risk by up to 50 percent.

THE PRIMARY DEFENSE MEDICATIONS ⁴⁵

Metformin

This is the most highly recommended preventive medication, proven to reduce diabetes development by 31 percent. It is explicitly advised for high-risk individuals, such as those under 60, those with high body weight, or those who experienced diabetes during pregnancy. Long-term use requires regular Vitamin B12 checks, as the drug can cause a deficiency. It is also used proactively to prevent blood sugar spikes in patients requiring high doses of steroids or specific cancer medications.

Weight Management Drug

Modern medications designed for weight management, including semaglutide, tirzepatide, liraglutide, and phentermine / topiramate, are also effective preventative measures against diabetes.

ALTERNATIVE INTERVENTIONS - P1 ⁴⁵

Carbohydrate Blockers

Medications that slow the digestion of carbohydrates in the gut are effective for blood sugar control. However, they frequently cause stomach upset and gas, which often makes patients reluctant to maintain long-term use.

Insulin Sensitizers

These drugs help the body respond better to insulin, temporarily relieving the pancreas. Their use is currently highly restricted due to severe safety concerns, including liver toxicity, heart issues, and bladder cancer.

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ALTERNATIVE INTERVENTIONS - P2 ⁴⁴

Blood Pressure Medications

Certain common blood pressure medications surprisingly aid the body in utilizing insulin more efficiently. Clinical studies indicate they can lower the risk of developing new-onset diabetes by approximately 25 percent.

Vitamins and Gut Health

Low Vitamin D levels strongly correlate with a higher risk of diabetes, making proper maintenance beneficial. Additionally, improving gut bacteria through prebiotics and probiotics is currently being researched for its potential to enhance overall metabolism and reduce diabetes-causing inflammation.

COMPLICATION MANAGEMENT ⁴⁴

Cardiovascular Risks

Pre-diabetes significantly elevates the risk of heart disease. It is mandatory to proactively manage blood pressure, lower cholesterol, and immediately cease smoking. While cholesterol-lowering drugs can cause a slight increase in blood sugar, the cardiovascular protection they offer far outweighs the diabetes risk. Patients should not discontinue these medications without medical authorization.

DOWNSIDE OF PILL USAGE ⁴⁴

Patient Adherence

The primary challenge in preventative care is adherence. It is difficult to convince individuals who feel perfectly healthy to commit to a daily preventative pill, particularly when those medications cause disruptive physical side effects.

Counterproductive Medications

Common medications prescribed for entirely different conditions can unintentionally elevate blood sugar levels. This includes certain water pills, steroids, beta-blockers, and specific cholesterol-lowering medications.



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EDUCATION

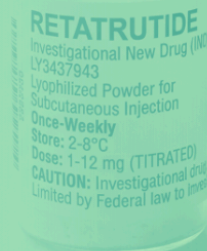
Established in 2021 PYERTX is dedicated to enhancing mental health awareness and providing essential services for minority youth. With a network of over 80 members, they have successfully organized 15+ events, directly impacting over 3,000 individuals.



The Next-Generation for Treatment

RETATRUTIDE

■ As a next-generation medication for Type-2 diabetes, Retatrutide is a next-generation "three-in-one" medication that treats Type 2 diabetes and obesity by simultaneously targeting three metabolism-regulating hormones. This approach reduces blood sugar and body weight significantly better than current standard treatments.⁴⁶



Images generated by gemini

BEYOND BLOOD SUGAR

■ *Goal / Focus for Future Medications*

Healthy Weight Loss⁴⁶

Because the medications often trigger such extreme weight loss, A primary goal for doctors moving forward is ensuring that patients shed excess fat rather than losing essential muscle mass and bone density.

Concurrent Conditions⁴⁶

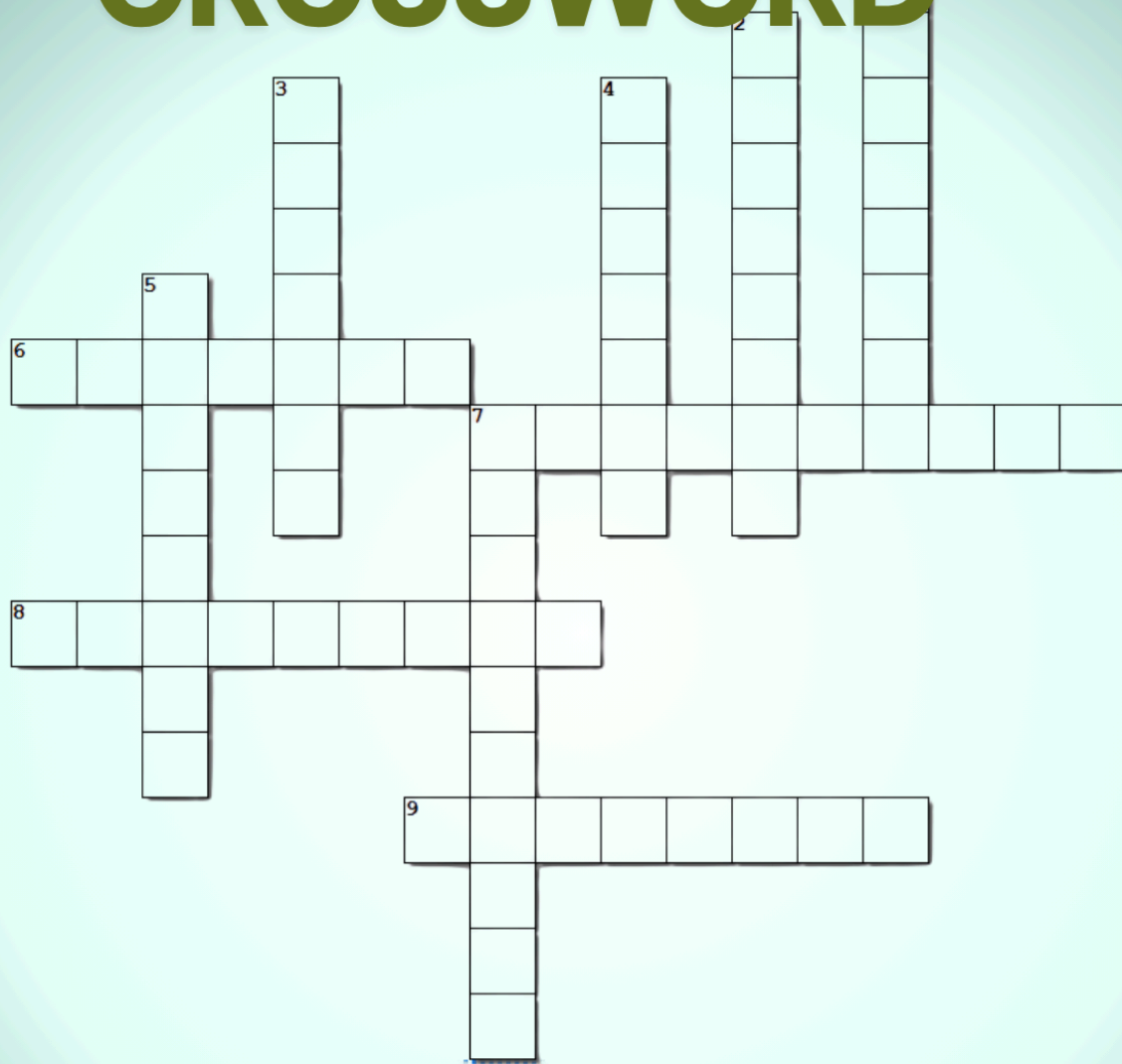
Modern treatments aim to tackle diabetes alongside the heavy health burdens that often accompany it. Modern medication aims to aid diabetic patients suffering from concurrent conditions like heart disease, sleep apnea, or joint pain caused by osteoarthritis.

Cost Barrier⁴⁶

While these advanced medications prove incredibly effective in clinical settings, there is a major, ongoing concern that they will remain far too expensive for the average patient to readily access and afford for long-term management.

Daily

CROSSWORD



ACROSS

6. One of the strongest risk factors for T2DM often involving excess visceral fat
7. Microvascular damage to the eye caused by sustained high blood sugar
8. The most recommended preventive medication for high risk individuals
9. The organ containing beta cells that acts as the body's glucose "thermostat"

DOWN

1. One of the 3P's referring to excessive urination
2. The hormone that instructs the liver to release stored glucose
3. The Canadian surgeon who earned a Nobel Prize for discovering insulin
4. The hormone that acts like a "key" to let glucose into cells
5. Latin for "honey-sweet" used to describe the urine of affected patients
7. The condition where cells stop responding appropriately to insulin

Daily

WORD SEARCH

M E G L I T I N I D E S X Y Z
E S T A R C H P U M P A B C D
T P I O G L I T A Z O N E E F
F A F R E Z Z A Q R S T U V W
O M I N C R E T I N N C R E A
R Y L I N S U L I N G H I J K
M L D I A B E T E S L M N O P
I I N G L U C O S E Q R S T U
N N G L I P I Z I D E V W X Y
A B C A M Y L I N Z A B C D E
C O L E S E V E L A M F G H I
B R O M O C R I P T I N E J K
H O R M O N E L M N O P Q R S
P A N C R E A S T U V W X Y Z
L Q W E R T Y U I O P A S D F

Call for Submissions: Share Your Puzzles

We invite readers to submit original word searches, crosswords, and mini-games for a chance to be featured in our upcoming issues.

How to Participate:

- Submit your original puzzle design along with the full answer key.
- Selected designs will be published in future editions. We look forward to seeing your creativity.

1. AFREZZA: A fast-acting inhaled insulin powder.
2. AMYLIN: A hormone released with insulin to slow digestion.
3. BROMOCRIPTINE: A medication acting on dopamine to help metabolism.
4. COLESEVELAM: A cholesterol-lowering drug that also lowers blood sugar.
5. DIABETES: The metabolic condition focused on sugar management.
6. GLIPIZIDE: A medication that stimulates the pancreas to release insulin.
7. GLUCOSE: The primary sugar found in the blood
8. HORMONE: Chemical messengers like insulin and GLP-1.
9. INCRETIN: Natural gut hormones that boost insulin production.
10. INSULIN: The hormone responsible for helping cells absorb sugar.
11. MEGLITINIDE: Fast-acting medications taken before meals.
12. METFORMIN: A first-line treatment that stops the liver from overproducing sugar.
13. PANCREAS: The organ responsible for producing insulin.
14. PIOGLITAZONE: A medication used to improve insulin sensitivity.
15. PUMP: A device used for continuous insulin delivery.
16. STARCH: Carbohydrates that are broken down into sugar during digestion.

VITAL PULSE

ISSUE NO. 67

DIABETES

A-Z

**SPECIAL
EDITION:
TALK WITH A
DIABETES
EXPERT**

**HOW IS
DIABETES'
IMPACT**

**FROM
NILUSNI MEDICAL UNIVERSITY**

FEATURING

DR. NILUSNI

GEMINI GENERATED
IMAGE

FACTS

What makes Type II diabetes problematic

Type 2 diabetes is essentially a metabolic disorder characterized by a chronic state of having high levels of glucose in your blood. Normally, when you eat, your pancreas acts as a thermostat, detecting the rising blood sugar and releasing a hormone called insulin. Insulin acts as a key that binds to receptors on your cells, unlocking your muscle, liver, and fat tissues so they can absorb that glucose to use as energy or store it for later. However, in type 2 diabetes, a cellular communication breakdown occurs, which we call insulin resistance. Your cells simply stop responding appropriately to the insulin keys. The pancreas tries to compensate by overworking and flooding your system with even more insulin. Eventually, the insulin factory inside the pancreas, specifically the beta cells, becomes completely exhausted and begins to fail.

What is the meaning behind the name: diabetes mellitus?

The name diabetes mellitus comes directly from the Greek word diabetes, which means a siphon or to pass through, and the Latin word mellitus, which translates to sweet. The word diabetes was actually coined around 100 AD by a man named Aretaeus of Cappadocia. He used this term because he observed that patients seemed to pass fluids straight through their bodies, as if great masses of their flesh were being liquefied into urine. Much later, in the year 1798, a British physician named John Rollo officially added the word mellitus, meaning honey-sweet in Latin. He added this specific term to distinguish this sugary condition from other diseases that caused entirely tasteless urine. The extraordinarily sweet nature of the urine in this condition was actually discovered

independently by ancient Greek, Indian, and Egyptian civilizations. In fact, as early as 400 to 500 AD, ancient Indian physicians named Sushruta and Charaka referred to the condition as honey urine, specifically noting that the sugar content was so high it literally attracted ants.

Why do we call it Type II?

As for why we specifically call it Type 2, the idea of classifying the disease into different categories actually goes way back to those same ancient Indian physicians, who were the first to identify two distinct types of diabetes. In modern medicine, when we give a patient a Type 2 diagnosis, we are identifying a unique metabolic disorder characterized by chronic high blood sugar that almost always begins in middle and late adulthood. We use this classification to describe a very specific, vicious biological cycle involving two main problems: insulin resistance and beta-cell dysfunction. Insulin resistance occurs when the cells in your muscles, fat, and liver simply stop responding appropriately to the insulin your body is already making. This creates a massive strain on the pancreas, leading to beta-cell dysfunction, which is the sheer inability of your pancreatic beta cells to secrete enough extra insulin to overcome that cellular resistance.

How is diabetes treated before

In the early twentieth century, doctors named Allen and Joslin essentially put their patients on a strict metabolic lockdown. They prescribed intense fasting and heavily calorie-restricted diets. The primary strategy was to drastically slash sugar intake and force weight loss to reduce the overall burden on the body. This harsh approach yielded measurable results for patients at the time. It successfully reduced the leakage of sugar into the urine and lowered dangerous blood acidity. It also decreased the occurrence of diabetic comas and delayed death among affected children.

During this same period, scientists made a crucial educated guess about the root cause of the disease. They hypothesized that the pancreas acts as an internal chemical factory, releasing a specific unseen substance to control how the body processes carbohydrates. Driven by this central idea, researchers began attempting to create crude pancreatic extracts. They hoped to use these early preparations to manually drive down dangerously high blood glucose levels.