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Review

Circadian Etiology of Type 2 Diabetes Mellitus

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Abstract

Type 2 diabetes mellitus (T2DM) is a growing global health issue, strongly associated with the obesity epidemic. People with T2DM face a high risk of both microvascular complications (such as retinopathy, nephropathy, and neuropathy) and macrovascular complications (including cardiovascular diseases), due to high blood sugar levels and various aspects of insulin resistance syndrome. Both environmental factors like obesity, poor diet, and lack of physical activity and genetic factors contribute to the multiple physiological disruptions that lead to impaired glucose regulation in T2DM. The main problems in T2DM are insulin resistance and reduced insulin secretion, but at least six other physiological abnormalities also play a role in glucose metabolism dysfunction. Because of these multiple underlying issues, managing T2DM typically requires a combination of several antidiabetic drugs to maintain normal blood sugar levels. Treatment should be not only effective and safe but also enhance patients' quality of life. While several new drugs are being developed, the most urgent need is for treatments that improve insulin sensitivity, stop the progressive failure of pancreatic β-cells characteristic of T2DM, and prevent or reverse microvascular complications.

Keywords: Type 2 Diabetes Mellitus, Circadian Rhythm, Insulin Resistance, β-cell Dysfunction, Obesity, Genetic Factors, Lifestyle Modification, Pharmacotherapy, Emerging Therapies, Phytotherapy

INTRODUCTION

Type 2 diabetes mellitus (T2DM) is marked by abnormal regulation of carbohydrate, fat, and protein metabolism, resulting from defective insulin secretion, insulin resistance, or both. Among the three main types

of diabetes, T2DM is the most prevalent, accounting for over 90% of cases, compared to type 1 diabetes mellitus (T1DM) and gestational diabetes. In recent decades, our knowledge of how T2DM develops and progresses has advanced significantly. The primary cause is the gradual decline in insulin secretion by pancreatic β cells, typically occurring alongside existing insulin resistance in skeletal muscle, liver, and fat tissue (see BOX 1). Before the onset of overt high blood sugar, individuals often experience prediabetes, a condition that increases the risk of developing T2DM (see TABLE 1). Prediabetes is defined by one or more of the following: impaired fasting glucose (IFG), impaired glucose tolerance (IGT), or elevated glycated hemoglobin A1C (HbA1c) levels. People with IFG have fasting plasma glucose levels above normal but not high enough to be diagnosed with diabetes. IGT involves insulin resistance in muscle and reduced late-phase (second-phase) insulin secretion after eating, while those with IFG show liver insulin resistance and impaired early-phase (first-phase) insulin secretion. Individuals with prediabetes have HbA1c levels ranging from 5.7% to 6.4% and represent a diverse group in terms of underlying mechanisms and clinical presentation. The annual rate at which rediabetes progresses to T2DM varies between 3% and 11%.

The clinical symptoms, underlying pathophysiology, and disease progression in diabetes patients can differ significantly between individuals, and sometimes unusual symptom presentations make it challenging to definitively classify type 2 diabetes mellitus (T2DM). At diagnosis, many T2DM patients show no symptoms, while others may have severe hyperglycemia or even diabetic ketoacidosis. Conditions like latent autoimmune diabetes in adults and maturity-onset diabetes of the young can mimic T2DM.

Table 1: Diagnostic reference values

Parameters	Normal*	Pre diabetes	T2DM
Hemoglobin A1c	<5.7%‡	5.7–6.4%‡	≥6.5%
	<6.0%§	6.0–6.4%§	
Fasting plasma glucose	<100 mg per dl‡	100–125 mg per dl‡	≥126 mg per dl
	<110 mg per dl§	110–125 mg per dl§	
Two hour plasm OGTT	<140 mg per dl	140–199 mg per dl	≥200 mg per dl

For asymptomatic people, the timing and frequency of screening for prediabetes or T2DM depend on the presence of risk factors. Screening at-risk individuals is crucial because prediabetes is common, and about 30% of people with T2DM remain undiagnosed. Preventing diabetes involves identifying those with prediabetes and implementing lifestyle changes such as weight loss and exercise, along with antidiabetic and anti-obesity medications. The American Diabetes Association (ADA) Consensus Conference recommended treating high-risk individuals (HbA1c >6.5%; BMI ≥30 kg/m²; age ≤60 years) with impaired glucose tolerance or impaired fasting glucose using medications like metformin. Pioglitazone and combined low-dose metformin and rosiglitazone reduce both acute and long-term microvascular complications (including retinopathy, nephropathy, and neuropathy) and macrovascular complications (such as heart attack and stroke). T2DM should be considered and managed as a heterogeneous disorder with multiple pathophysiological abnormalities, differing risks for complications, and variable responses to treatment. Ultimately, achieving a true cure for T2DM will require understanding its molecular causes and developing effective strategies to address the obesity epidemic. This Primer covers the epidemiology, diagnosis, pathophysiology, and current and future management of T2DM.

Table 2: Multi factorial risk reduction outpatient goals of therapy in T2DM

Parameter	ADA	AACE	IDF (WDF)
Glucose			
Fasting glucose (mg per dl)	70–130*	<110	115
2 hour postprandial glucose (mg per dl)	<180*	<140	<160
Hemoglobin A1c (%)	<7	≤6.5	<7.0
Lipids			
LDL cholesterol (mg per dl)	<70‡	70‡	<70‡
Non-HDL cholesterol (mg per dl)	NR	<130 <100‡	<97
HDL cholesterol (mg per dl)	>40 in men >50 in women	>40 in men >50 in women	>39

Triglycerides (mg per dl)	<150	<150	<200
Blood pressure			
Systolic pressure/ diastolic)	<140/80*	<130/80	≤130/80 pressure (mm Hg)

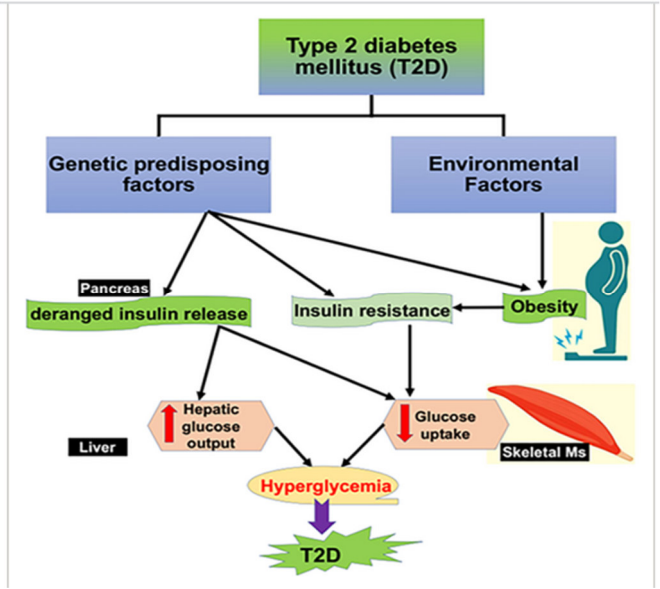
ETIOLOGY

Type 2 diabetes mellitus (T2DM) has become a significant global public health issue. According to the International Diabetes Federation, in 2013, 382 million adults aged 20 to 70 worldwide were living with T2DM, with 80% of these individuals residing in low- and middle-income countries. This figure is projected to increase to 592 million by 2035. China and India are particularly affected, experiencing a sharp rise in T2DM prevalence despite relatively low obesity rates.

Asians tend to have a higher body fat percentage, more abdominal fat, and less muscle mass compared to others with the same body mass index (BMI), which may contribute to their greater susceptibility to T2DM. Additionally, poor nutrition during fetal development and early life, followed by overnutrition later on, can accelerate the spread of T2DM, especially in populations undergoing rapid dietary and lifestyle changes, including altered eating habits and decreased physical activity. The prevalence of T2DM is slightly higher in men than in women.

Epidemiological research has enhanced our understanding of the behavioral, lifestyle, and biological risk factors for T2DM. Increased body fat, as indicated by higher BMI, is the most significant risk factor. Certain dietary choices are linked to a lower risk of T2DM regardless of body weight, such as consuming more whole grains, green leafy vegetables, nuts, and coffee; eating less refined grains, red and processed meats, and sugary drinks; and drinking alcohol in moderation. Physical inactivity is a major behavioral risk factor, while both aerobic exercise and resistance training offer benefits. Sedentary behaviors, like extended periods of watching television, are associated with higher risk. Both short (five hours or less) and long (nine hours or more) sleep durations, as well as rotating shift work, increase the risk. Smoking is also a significant risk factor for developing T2DM, independent of body weight and other factors.

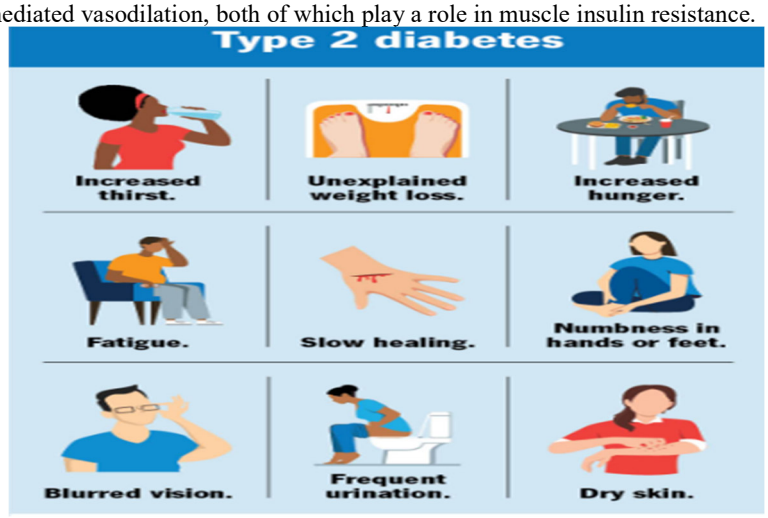
Although genetics play a role in T2DM development, the current diabetes epidemic cannot be attributed to new genetic mutations but is largely driven by the obesity epidemic. Nonetheless, genetic factors influence how individuals respond to environmental changes, and vice versa.



PATHOPHYSIOLOGY

Type 2 diabetes mellitus (T2DM) is a complex disease influenced by both genetic and environmental factors. Its pathophysiology is marked by β-cell dysfunction, insulin resistance, and chronic inflammation, all of which gradually disrupt blood glucose regulation and result in microvascular and macrovascular complications. Regarding hyperglycemia, at least eight distinct pathophysiological defects contribute to impaired glucose balance, and these abnormalities are well recognized early in the progression of T2DM. In addition to this

"ominous octet," two more pathophysiological issues can be included: activation of inflammatory pathways and reduced insulin-mediated vasodilation, both of which play a role in muscle insulin resistance.



GENETIC FACTORS

Type 2 diabetes mellitus (T2DM) tends to run in families and is inheritable. Siblings of a person with T2DM have about a 2 to 3 times higher risk of developing the disease compared to families where no siblings have it; this risk rises to 30 times if two siblings are affected. The likelihood of developing T2DM is greater if the mother has the disease rather than the father. Additionally, having a body mass index (BMI) of 30 or higher or a fasting glucose level above 5.5 mmol/L significantly increases the risk. In comparison, the relative risk for type 1 diabetes mellitus (T1DM) is around 15, and for maturity-onset diabetes of the young (MODY), it is about 50. Identifying the genes responsible for complex polygenic diseases like T2DM has been challenging. A major advancement occurred in 2007 with genome-wide association studies (GWAS) that identified common genetic variants linked to T2DM. The strongest association was found with a single nucleotide polymorphism (SNP) in the TCF7L2 gene, previously reported through gene linkage analysis. Other genes with SNPs associated with T2DM include SLC30A8, FTO, CDKAL1, CDKN2A, CDKN2B, HHEX, IGF2BP2, and GCKR, among others. Later GWAS expanded this list to over 100 common variants related to T2DM. Most of these variants are located in introns, so it is more accurate to refer to genetic loci rather than specific genes. Consequently, understanding how these loci increase T2DM risk is complex. Exceptions include a few variants in exons that affect gene function, such as SLC30A8 (which encodes a zinc transporter essential for insulin storage), KCNJ11 (which encodes an ATP-dependent potassium channel), and GCKR (which encodes a glucokinase regulatory protein). Intronic variants may influence the expression of nearby genes (in cis) or distant genes (in trans), but this has been confirmed for only a few genes, like a variant in MTNR1B, which encodes a melatonin receptor. Studies of cultured human islets with risk alleles have shown decreased β-cell function and survival. However, gaining mechanistic understanding of these SNPs has been difficult, and animal studies have provided limited insights. For example, a human mutation in SLC30A8 offers protection against T2DM, whereas in mice, it increases susceptibility. Other animal research has been more informative. In a polygenic T2DM model using GK rats, impaired insulin secretion was linked to a variant in the ADRA2A gene, causing overexpression of the α2A-adrenergic receptor in islets. Pretreating human islet cells with yohimbine, an inhibitor of this receptor, restored insulin secretion. Subsequently, treating human carriers of this variant with yohimbine led to a dose-dependent improvement in insulin secretion. Reduced β cell function has also been linked to epigenetic changes50 and microRNA profiles51, which probably raise the proportion of cases where genetic inheritance plays a significant role in disease development42.

PREVALENCE

The age-specific prevalence rates for the years 1996, 2003, 2010, and 2017 are presented separately for type 1 diabetes (T1D) and type 2 diabetes (T2D), with detailed figures broken down by sex and calendar year. Throughout the study period, the crude prevalence of T1D (ages 0–99) remained relatively stable at approximately 0.5% for men and 0.4% for women. In contrast, the crude prevalence of T2D tripled during this time, rising from 1.2% to about 4.5%, with a slightly higher increase observed in men compared to women (see table esm3). This corresponds to an annual growth rate of 5.5% (table 1). Consequently, the proportion of T1D cases among all diabetes patients decreased from around 25% on January 1, 1996, to 10% on January 1, 2017 (table esm2). Regarding age-specific prevalence, T1D rates increased up to approximately age 40 in men and

age 30 in women. For T2D, the highest age-specific prevalence on January 1, 2017, was observed at age 80, reaching 19% in men and 16% in women.

During the study period from 1996 to 2016, there were 363,664 new diabetes cases recorded, of which 19,712 (5.4%) were T1D (table esm4). Individuals over 100 years old and those not residing in the area at diagnosis were excluded. For T1D, incidence rates showed a slight increase among younger age groups but decreased in older age groups, resulting in an overall average annual decline of 3.5%. In contrast, T2D incidence patterns were similar across different ages, with an increase until 2011, a decline until 2014, followed by a rise during the final two years of the study. Age–period–cohort models indicated that men had higher incidence rates than women and that the age-related incidence patterns for T1D differed somewhat by sex. In men, incidence increased up to about age 18, plateaued, and then slightly rose again until age 40, whereas in women, incidence increased until around age 15 before declining. Although direct comparison of absolute incidence rates between T1D and T2D is challenging due to differences in age at diagnosis, broadly speaking, T2D occurs at rates 20 to 30 times higher than T1D.

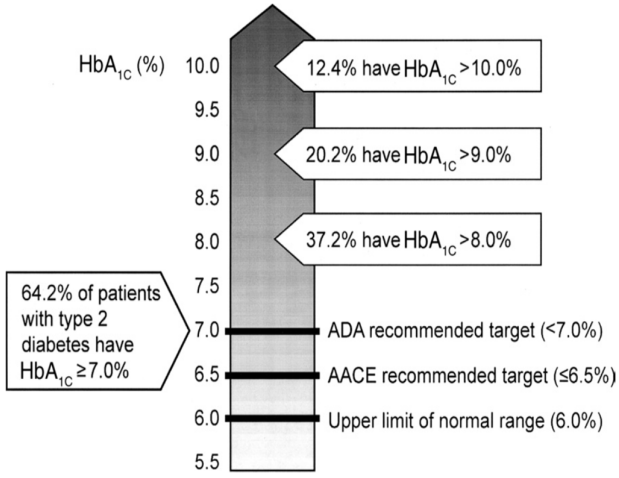
CURRENT TREATMENT STRATEGIES

Contemporary management strategies for type 2 diabetes emphasize preventing the disease, screening individuals at high risk, and providing intensive treatment during the prediabetic phase. Central to effective management in all cases is lifestyle modification. Even modest weight loss (around 4 kg) can positively impact fasting blood glucose levels, and when combined with increased physical activity, it can also improve other cardiovascular risk factors such as high blood pressure, atherogenic dyslipidemia, endothelial dysfunction, and elevated plasma viscosity.

When starting medication, it is crucial to consider the patient's current blood sugar control. For patients with HbA1c levels above 8.5%, medications like sulfonylureas or metformin should be initiated. Metformin, unless contraindicated, helps improve blood sugar control and supports weight loss without causing hypoglycemia. It is generally well tolerated, widely covered by formularies, and cost-effective. The American Diabetes Association recommends starting metformin alongside lifestyle changes at the time of type 2 diabetes diagnosis. For patients with HbA1c around 7.5%, drugs with a slower onset, such as thiazolidinediones (TZDs), may be appropriate, although their use can be limited by weight gain and high cost. Recently, safety concerns have emerged regarding rosiglitazone and pioglitazone, as meta-analyses indicate these TZDs may increase the risk of heart failure.

Combination oral antidiabetic therapy can be an effective initial treatment for patients new to diabetes management because it targets both insulin resistance and insulin deficiency. However, when HbA1c levels exceed 8.5%, combination therapy alone is unlikely to achieve target HbA1c levels,since oral agents typically reduce HbA1c by only 1% to 2%.

Beyond these traditional medications, several new drug classes are now used to enhance diabetes treatment. Many of these work by targeting glucagon-like peptide-1 (GLP-1), a natural hormone produced by L-cells in the small intestine. After eating, GLP-1 stimulates pancreatic insulin production and secretion. This "incretin effect" refers to the increased insulin response to oral glucose compared to intravenous glucose. GLP-1 also suppresses glucagon secretion, helps regulate gastric emptying to stabilize blood glucose levels, and reduces appetite, leading to weight loss of 2 to 3 kg over six months. Since individuals with type 2 diabetes have a deficiency in GLP-1, they cannot adequately suppress glucagon after meals or control postprandial blood sugar spikes. Clinical studies of exenatide (Byetta; Eli Lilly & Co., Indianapolis, IN), a synthetic version of exendin-4 approved in the U.S. for type 2 diabetes not adequately controlled by metformin and/or sulfonylureas, show that administering 10 µg of exenatide twice daily for six months can lower HbA1c by 1% and reduce body weight by 2 kg. Long-term studies demonstrate a sustained HbA1c reduction of 1.1% after 18 months, along with a gradual average weight loss of 4.4 kg. Up to 45% of patients may experience temporary side effects such as nausea, vomiting, or diarrhea. Exenatide is most effective when used in the early stages of type 2 diabetes; since it lowers HbA1c by about 1%, patients with an HbA1c of 8% on combination oral therapy are unlikely to reach the ADA target of 7% by simply adding exenatide. Most patients with HbA1c levels above 8% will still need insulin therapy to achieve their target goals.



EMERGING TRENDS

There is growing recognition that traditional methods of diabetes care and education need to be customized to address the distinct pathophysiological, behavioral, and psychosocial traits of young individuals with type 2 diabetes. Managing type 2 diabetes at a younger age requires adjustments from the standard treatment used for older adults, although within this younger group, there is considerable diversity in life circumstances, education, and employment status. This diversity includes personal factors—such as being in full-time education, working age, experiencing high diabetes-related distress, or having poor mental health—that can influence engagement with healthcare providers and personal motivation. Additionally, medical considerations may call for different strategies, like the need for contraception and preconception counseling in pregnant women with type 2 diabetes. Since early-onset type 2 diabetes can coincide with the typical age of onset for monogenic and type 1 diabetes, diagnostic testing may be necessary. Young people may be just as likely, or even more so, to have diabetes types other than type 2. Pediatric guidelines generally advise pancreatic autoantibody testing for all children with high blood sugar and testing for monogenic diabetes if antibodies are negative. In contrast, adults are usually tested for pancreatic autoantibodies only when type 1 diabetes is suspected, not for all diabetes types. Although there is limited evidence to support routine antibody testing for all young adults newly diagnosed with diabetes, healthcare providers should carefully evaluate patient history for signs that might indicate an alternative diagnosis, such as type 1 or monogenic diabetes in adults with early-onset diabetes.

LIFESTYLE MANAGEMENTS

Lifestyle changes and the possibility of remission Similar to type 2 diabetes that develops later in life, promoting healthy lifestyle habits is considered fundamental to treatment. The DiRECT trial, a randomized controlled study involving 306 mostly White participants with an average age of 54, evaluated the effectiveness of very-low calorie diets over 12 weeks compared to standard care. It showed an average adjusted weight loss of 8.8 kg (95% CI 7.3 to 10.3 kg) and remission in 36% of participants after two years. Two additional randomized controlled trials have also tested very-low calorie diets. In adolescents, the TODAY study examined a lifestyle intervention consisting of 200–300 minutes of moderate exercise weekly and a daily diet of 1200–1500 kcal alongside metformin treatment. This intervention did not lead to significant differences in weight loss or HbA1c levels compared to metformin alone or metformin combined with rosiglitazone. The DIADEM-1 trial enrolled 158 participants from the Middle East and North Africa region, aged 18 to 50 years (73% men), with type 2 diabetes of less than three years' duration and a BMI of 27 or higher. The intervention involved very-low calorie diets of 800–820 kcal per day for three months, followed by gradual food reintroduction, physical activity, and structured lifestyle support during maintenance. After 12 months, the intervention group lost an average of 11.98 kg compared to 3.98 kg in the control group (adjusted mean difference of 6.08 kg, 95% CI 3.79 to 8.37 kg). Remission occurred in 61% of the intervention group versus 12% of controls (odds ratio 12.03, 95% CI 5.17 to 28.03). To date, DIADEM-1 is the only randomized controlled trial to investigate remission in young adults and non-White populations. The results from DIADEM-1 are encouraging, showing similar weight loss to the DiRECT study in a younger group with an even higher remission rate, possibly due to shorter diabetes duration. However, these findings have not yet been replicated in other young adult groups, and evidence in children is limited to the TODAY study and small observational studies with mixed outcomes. It remains uncertain whether very-low calorie diets can produce lasting remission in early-onset type 2 diabetes across diverse ethnicities and socioeconomic backgrounds. Adjustments to protocols, such as intermittent very-low

calorie diets, may be necessary to address the significant lifestyle factors driving early-onset type 2 diabetes. Regardless of remission potential, focused dietary guidance to support weight loss should remain a central component of management.

COMORBID CONDITIONS

Comorbid conditions were evaluated using all available data up to and including the index date. The conditions assessed included congestive heart failure (CHF), chronic kidney disease (CKD), retinopathy, neuropathy, cardiovascular disease (CVD), urinary tract infections (UTI), genital mycotic infections, hypoglycemia, pancreatitis, liver disease, hypertension (HTN), hyperlipidemia, and overweight/obesity. CVD was classified based on ICD-9-CM codes for myocardial infarction, ischemic heart disease, peripheral artery disease, or cerebrovascular disease. CKD was identified either by an ICD-9-CM diagnosis code or, if absent, by an estimated glomerular filtration rate (eGFR) below 60 mL/min/1.73m², using the most recent measurement before the index date. If eGFR was not already calculated in the database, it was computed using the Modification of Diet in Renal Disease (MDRD) Study equation. Hypoglycemia was determined using a modified version of the algorithm by Ginde et al., excluding ICD-9-CM codes 270.3, 775.0, 775.6, and 962.3 due to their irrelevance to the study population. Hypertension was defined by the presence of an ICD-9-CM diagnosis code, a systolic blood pressure (SBP) of 140 mmHg or higher, a diastolic blood pressure (DBP) of 90 mmHg or higher from the most recent measurement before the index date, or the use of antihypertensive medications. Hyperlipidemia was identified through ICD-9-CM codes, a low-density lipoprotein cholesterol (LDL-c) level of 160 mg/dL or higher from the latest measurement before the index date, or the use of cholesterol-lowering drugs. Overweight or obesity was defined by either an ICD-9-CM code or a body mass index (BMI) of 25 kg/m² or greater from the most recent measurement. CHF, retinopathy, neuropathy, UTI, genital mycotic infections, pancreatitis, and liver disease were all identified solely through ICD-9-CM diagnosis codes (refer to the appendix for specific codes). Additional variables considered included age, gender, race, residential location, duration of diabetes, and the most recent values for HbA1c, blood pressure, eGFR, lipid profiles, and BMI.

PHYTOTHERAPHY IN THE TREATMENT OF DIABETES MELLITU

Regrettably, all anti-diabetic medications have side effects and tend to be costly. Consequently, developing new antidiabetic treatments that are both more affordable and have fewer adverse effects remains a significant challenge for researchers.

Polyphenols: Natural products rich in polyphenols—such as blackberries, red grapes, apricots, eggplant, coffee, cocoa, and green tea—can influence glucose metabolism through various mechanisms. These include restoring beta-cell function, enhancing insulin secretion, and increasing cellular glucose uptake, which collectively help improve insulin resistance.

Smart insulin patch: A novel smart insulin patch has been developed, featuring a thin square covered with over 100 tiny needles. Made from biocompatible materials, this patch acts quickly and is user-friendly. The microscopic needles contain insulin and glucose-sensitive enzymes that are released when blood glucose levels rise. In mouse studies, the patch lowered glucose levels for up to nine hours. It is anticipated that the patch may have an even longer effect in humans, who are more sensitive to insulin than mice.

Dual-acting peptide: GLP-1 and GIP are the primary incretin hormones released from the intestine after eating, both stimulating insulin secretion in a glucose-dependent manner. Animal studies indicate that combining GLP-1 with GIP enhances anti-obesity effects. Finan et al. demonstrated that an acylated dual agonist targeting both GLP-1 and GIP receptors led to greater reductions in weight (-18.8% vs. -8.8%), food intake, fat mass, and blood glucose compared to liraglutide. It also increased plasma insulin and C-peptide levels more significantly. However, no differences in glycemic control improvements were observed between the dual agonists and liraglutide. In patients with type 2 diabetes, dose-dependent HbA1c reductions were noted: -0.53% with 4 mg and -1.11% with 30 mg of the dual agonist, compared to -0.16% with placebo. The pharmacokinetic and pharmacodynamic profiles of GLP-1 and GIP receptor co-activation are promising for treating obese type 2 diabetes patients and may lead to the development of once-weekly dual agonist drugs.

GLP-1 and glucagon receptor dual agonism: Glucagon and GLP-1 have distinct but structurally related receptors. Glucagon raises blood glucose by stimulating gluconeogenesis and glycogenolysis in the liver, whereas GLP-1 lowers blood glucose by promoting insulin production and secretion in the pancreas. Administration of oxyntomodulin, a dual agonist of GLP-1 and glucagon receptors, in rodents and humans has shown improved glucose metabolism by reducing food intake and body weight while increasing energy expenditure, with effects more pronounced than those seen with GLP-1 alone. Additionally, giving PEGylated peptides once a week helped reduce body fat and enhanced how well the body processes sugar in mice that became obese from their diet. Scientists also found that ongoing treatment with compounds that work on both GLP1 and glucagon pathways can actually reverse obesity in these diet-induced obese mice. These dual-action

treatments also brought glucagon, glucose, and fat processing back to normal levels while reducing fatty buildup in the liver. This represents a promising new way to treat obesity in people who have type 2 diabetes.

CONCLUSION

Although changing your lifestyle and taking metformin are still the main ways doctors start treating type 2 diabetes, there are now many more medication options available as backup treatments. Today, patients have access to various types of pills and injections to help manage their diabetes. The medication families include sulfonylureas, meglitinides, insulin, TZD, and alpha-glucosidase inhibitors. More recently, doctors can also prescribe RA-GLP1 receptor agonists, iDPP4, and iSGLT2 inhibitors. Scientists have also created improved insulin versions that work more like the body's natural insulin production. For most people with type 2 diabetes, metformin is still the go-to first treatment. When doctors consider other treatments or add a second medication, they look at each person's specific situation - things like how high their blood sugar is, what other health problems they might have, what the patient prefers, and whether they can actually get the treatment. They also think about how well each medication works, how long it stays effective, whether it might cause dangerously low blood sugar, if it helps prevent diabetes complications, how it affects weight, and what side effects or health risks it might have. While we probably won't find a cure for diabetes anytime soon, researchers are working on new medications that are both safe and effective, which should help people with type 2 diabetes live better lives.

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