Type 2 Diabetes: Insulin Resistance, Remission, Hidden Sugars, and Long-Term Health

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Type 2 diabetes is not simply “type 1 diabetes that starts later.” Many people with insulin resistance initially make extra insulin—sometimes substantially elevated amounts—yet their bodies do not respond to it effectively enough. Blood glucose rises when insulin production can no longer meet that increased need. Nutrition and other sustained changes can substantially improve control; some people achieve medication-free remission. Neither a guaranteed cure nor inevitable deterioration is an accurate description. [1] [2] [3]

Do not stop insulin or other diabetes medicines to test a diet or try to qualify as “in remission.” Major changes in carbohydrate intake, calories, weight, or activity can change medication needs. Arrange a monitoring and medication-adjustment plan with your diabetes team. Type 1 diabetes requires insulin; nutrition cannot replace it. [4] [5]

This guide explains what is established, what is promising, and where popular claims go beyond the evidence. It is education, not a diagnosis, a personalized diet, or a medication-withdrawal plan.

Key Takeaways

• Type 1 usually involves autoimmune destruction of insulin-producing cells. Type 2 commonly combines insulin resistance with insulin production that is inadequate for the body's needs.

• Insulin can be high before or early in type 2, but not in everyone. Production can decline over time, and some people need insulin treatment.

• Stress hormones, inflammatory illness, steroid medicines, adipose-tissue function, and cardiovascular health can influence glucose; none alone explains every person's type 2 diabetes.

• Routine insulin blood testing is not recommended for most people with diabetes. That is different from saying insulin resistance is unimportant.

• Remission is real, but it is not a cure or a promise. ADA-convened experts addressed remission in 2009, with an updated international definition in 2021.

• Children can develop type 2 and adults can develop type 1. The old “adult-onset” and “juvenile” labels are unreliable.

• Added sugars appear under many ingredient names. Look at serving size, total carbohydrate, total sugars, and the separate Includes Added Sugars line.

• Diabetes can damage eyes, kidneys, nerves, the heart, brain, and limb circulation. Early treatment and ongoing screening matter even when you feel well or achieve remission.

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These points are explained and sourced below. The aim is practical action without blame, false reassurance, or exaggerated promises. [1] [4] [6] [3] [7] [8]

1. Type 1 and type 2: different underlying problems

Insulin is a hormone made by pancreatic beta cells. It helps regulate glucose use and storage. Diabetes means blood glucose is too high, but the mechanisms behind that result differ. Age, body size, and whether someone currently uses insulin are not sufficient to identify the type. [1]

Question	Type 1 diabetes	Type 2 diabetes
What is the usual underlying problem?	An autoimmune process damages beta cells, leading to severe insulin deficiency.	Insulin resistance and beta-cell dysfunction combine; insulin output becomes inadequate for the body's needs.
Is the person still making insulin?	Some secretion can remain, especially near diagnosis, but insulin treatment is necessary.	Often yes; levels can be elevated, normal, or low depending on disease stage and circumstances.
Who can develop it?	Children, teenagers, and adults—including older adults.	Adults and young people; childhood type 2 is a serious concern.
Can nutrition replace insulin?	No. Insulin is needed to live.	Nutrition can substantially improve glucose control, and some adults achieve remission, but medication or insulin may still be needed.
What did people used to call it?	“Juvenile diabetes” or “juvenile-onset diabetes.”	“Adult-onset diabetes.”

The table describes typical mechanisms, not a home classification test. There are also gestational diabetes, monogenic forms, and diabetes associated with pancreatic disease or certain medicines. A person labeled type 2 who develops unexplained weight loss, ketosis, or rapidly worsening glucose may need reassessment. An adult can have autoimmune type 1 even if they also have overweight or obesity. [1] [9] [10]

2. How can glucose be high when insulin is also high?

Insulin resistance means that tissues do not respond to insulin as effectively as they should. The pancreas may initially compensate by releasing more insulin. During this stage, glucose can remain within the usual range despite elevated insulin. In susceptible people, compensation becomes inadequate and glucose rises. [11] [2]

This resolves an apparent contradiction: a person can have more insulin than someone without insulin resistance, yet still not enough effective insulin action for their own needs. “High insulin” does not mean glucose is controlled, and it does not mean the person can safely stop prescribed treatment. [2] [1]

The process is not identical in everyone. Beta-cell function may already be impaired when type 2 is diagnosed, can deteriorate over time, and can improve to different degrees with treatment. It is too simplistic to say every pancreas merely “wears out” on the same schedule. Some people with type 2 ultimately need insulin because their own production is insufficient. Needing insulin is not a personal failure. [2] [5]

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Which comes first: insulin resistance or high insulin?

These mechanisms can run in both directions. Resistant muscle, liver, and fat tissue commonly lead beta cells to release more insulin to compensate. Circulating levels also depend on how quickly insulin is cleared, not only how much is secreted. Experimental work raises the possibility that prolonged high-insulin exposure can further change tissue responses in some settings. But a high insulin measurement cannot establish which process began first in one person, and no evidence shows that every rise in insulin causes type 2 diabetes. Genetics, beta-cell capacity, fat distribution, medicines, and other exposures change the story. [2] [1] [12]

Insulin helps move glucose into tissues, supports nutrient storage, and restrains release of stored fuel. Body-fat gain also depends on energy intake and expenditure and the body's adaptations; insulin alone does not create fat regardless of food or energy balance. Conversely, severe insulin deficiency in untreated type 1 diabetes is a dangerous illness, not proof that a person without insulin cannot store body fat or a weight-control strategy. The useful clinical question is how to improve metabolic health safely, not how to drive insulin to zero. [13] [5]

Stress, illness, and steroid hormones

During acute physical or psychological stress, adrenaline (epinephrine) can increase liver glucose output; cortisol helps coordinate responses to stress. Glucose may rise during illness or mental stress, especially in someone already living with diabetes. An acute hormone response is not the same as permanently elevated hormone levels. Ongoing stress may affect glucose through sleep, activity, eating, and diabetes self-care as well as physiology; neither stress nor an isolated high glucose reading proves chronic cortisol excess or identifies the sole cause of diabetes. Discuss recurring patterns and support needs with your care team without self-blame. [14] [15] [16]

Cushing syndrome, in which cortisol is pathologically excessive, is a distinct condition that can reduce insulin sensitivity and worsen glucose. Systemic glucocorticoid medicines, such as prednisone, can also raise glucose, often particularly after meals; a large observational study in people with inflammatory diseases found a dose-related association with new diabetes, not proof that the medicine caused every case. If you are prescribed steroids, ask whether and when glucose monitoring or a treatment review is needed. Do not stop or taper steroids on your own; appropriate anti-inflammatory treatment may be essential. Routine cortisol testing is not warranted solely because someone has type 2 diabetes. [16] [17] [1]

Inflammation and autoimmunity: related but not interchangeable

Metabolic inflammation can contribute to insulin resistance, especially in dysfunctional adipose tissue, but not everyone with an autoimmune condition develops type 2 diabetes. Autoimmune type 1 destroys insulin-producing beta cells and requires different treatment. In someone with inflammatory or autoimmune illness, disease activity, reduced activity, body composition, sleep, and treatment (especially systemic steroids) can all affect glucose. Ask about monitoring in your own context rather than assuming a single inflammatory pathway explains your diagnosis. [13] [1] [17]

Prescribed insulin and weight: benefit and trade-offs

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Injected insulin can be necessary to treat marked or symptomatic high glucose in type 2, and is essential in type 1. It can sometimes be associated with weight gain as glucose loss in urine falls and energy is retained, and low-glucose episodes may prompt extra eating. This is a possible treatment effect to discuss—not a promise of weight gain or proof of inevitable, permanent insulin resistance. The 2026 ADA treatment guideline considers insulin in type 2 when hyperglycemia is symptomatic or especially high, and describes other or combination treatments according to clinical circumstances. Ask about your glucose pattern, hypoglycemia risk, weight concerns, and options; never stop or reduce prescribed insulin without an individualized clinician plan. [18] [5]

Some people feel pressure to omit insulin to affect weight. Intentional insulin restriction for weight control, sometimes called “diabulimia” colloquially, is dangerous disordered-eating behavior, not a treatment or a formal diagnosis by itself. It can lead to life-threatening diabetic ketoacidosis (DKA). If this is happening, tell your diabetes team promptly and ask for eating-disorder-informed support without shame. If vomiting, abdominal pain, rapid or difficult breathing, or severe illness develops, seek emergency care now. This concern is not limited to one age group or diabetes type. [19] [20]

Fat tissue: location and function, not one cell-size rule

Fat tissue can expand through both enlargement of existing adipocytes and creation of new ones. Where fat is stored—including visceral and liver fat—and how the tissue adapts matter beyond weight, cell count, or one biopsy measure of cell size. Both large and small cells can appear in metabolically unfavorable settings; small cells are not categorically noninflammatory. A small overfeeding study in men even found that smaller baseline fat cells were associated with a greater decline in insulin sensitivity; this does not predict any individual patient's outcome. [13] [21]

Liposuction removes subcutaneous fat for contouring, but it is not a diabetes or insulin-resistance treatment. In one small human study, substantial abdominal fat removal did not significantly improve insulin sensitivity 10–12 weeks later. A separate small randomized study observed some return and redistribution of removed fat after a year. These studies differ in participants and procedures; neither proves remaining cells inevitably enlarge, become inflamed, or deposit fat viscerally in every patient. Changing fat mass surgically is not interchangeable with sustained metabolic changes from nutrition, activity, and clinical care. [22] [23] [13]

Blood pressure and sexual health can be clues, not diagnoses

Insulin resistance and high blood pressure have a meaningful human evidence link, not just a theoretical overlap. In a 1987 study using the euglycemic insulin clamp, 13 lean, normal-glucose-tolerant adults with untreated essential hypertension had less insulin-stimulated glucose uptake than 11 matched controls (3.80 versus 6.31 mg/min/kg); the investigators also related uptake to blood-pressure severity. This shows resistance can accompany hypertension even without obesity or diabetes in a selected group. It does not establish that resistance caused their high pressure or that high insulin causes most hypertension. [24]

Several pathways make the overlap biologically plausible. Insulin can influence kidney sodium retention and sympathetic activity; metabolic and vascular stress may also affect blood-vessel relaxation, arterial stiffness, and renin–angiotensin–aldosterone signaling. Within endothelial cells, insulin-related PI3K–Akt–eNOS signaling can promote nitric oxide (NO), which helps vessels relax. Researchers propose that this route may be impaired in some insulin-resistant states while other, including MAPK-linked and endothelin-related, constricting signals persist. This is a model involving multiple tissues and pathways—not proof of a single universal cause in human patients. Kidney disease, salt sensitivity, genetics, medicines, and other factors influence pressure; severe insulin resistance can occur without hypertension. Measure and treat blood pressure on its own merits as well as glucose. [25] [26] [27] [28]

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Erectile dysfunction (ED) and metabolic dysfunction also have a substantial studied association. A 2024 systematic review pooled 17 studies (3,810 men with ED and 8,252 without). The ED groups had higher measures related to insulin resistance: HOMA-IR standardized mean difference 0.59 (95% CI 0.15–1.03), triglyceride-glucose index 0.53 (0.31–0.75), and visceral adiposity index 0.45 (0.25–0.64). These are differences between study groups, not percentages of men who will develop ED. Variation between studies was high (I^2 82%, 69%, and 76%, respectively), and pooled per-unit odds analyses did not establish reliable prediction for an individual. Most included evidence was observational. [29]

The NO pathway offers a concrete possible link: erections require blood-vessel relaxation in penile tissue; diminished insulin-related endothelial NO signaling may contribute to impaired flow alongside other factors. In a rat experiment, insulin relaxed penile arteries through an endothelium-, NO-, and PI3K-dependent process in lean animals, whereas obese insulin-resistant rats had impaired relaxation and a MAPK-related constricting response. This supports a mechanism but does not demonstrate the same sequence or an endothelin-blocking treatment in every human. In one clinic-selected study of 192 men under 40 with otherwise unexplained ED and 33 controls, ED was associated with higher HOMA-IR, higher systolic pressure, and poorer endothelial dilation. ED can thus be an early cardiometabolic clue in some younger men before known diabetes, but it is not necessarily the earliest symptom or a diagnostic test for insulin resistance, nor does it affect all men. [30] [31] [32]

A small randomized pilot tested adding metformin to sildenafil in 30 selected, nondiabetic men with insulin resistance and a poor response to sildenafil: erectile-function scores improved in the 17 receiving metformin versus the 13 receiving placebo, but reported adverse events were more common (61.5% versus 7.7%). This is a promising limited result, not a recommendation to start metformin for ED or proof that all ED arises from insulin resistance. Persistent difficulties also can involve medicines, nerves, hormones, stress, and other conditions. Ask a clinician to assess sexual health and cardiovascular risk without embarrassment; do not self-prescribe or change diabetes or blood-pressure medicines based on a mechanism alone. [33] [32] [29]

A researcher's perspective versus clinical evidence

Benjamin Bikman, PhD, is a Brigham Young University professor whose lab studies insulin resistance, adipose development, and metabolism. His BYU-listed qualification is a PhD in bioenergetics, not an MD or a documented clinical practice credential. He also offers public education through his self-branded YouTube channel and Metabolic Classroom playlist. These are places to hear a researcher's interpretation, not independently verified clinical advice or evidence that BYU endorses the channel. His personal site and HLTH Code product site advertise paid memberships, coaching, and nutritional products; consider commercial interests when evaluating claims. [34] [35] [36] [37]

In a peer-reviewed mouse experiment, Bikman and colleagues studied a lipid-related inflammation pathway linked to insulin resistance. Animal findings can generate useful human research questions but cannot establish that a particular diet treats human type 2 diabetes or that insulin is its one universal cause. The current clinical recommendations here rely instead on human research and professional guidance. Never change medicine because of a video or an animal experiment. [38] [18]

The role of insulin measurement

Insulin levels often are not measured because joint laboratory guidance for diabetes does not recommend routine insulin or proinsulin testing in most people with diabetes or at risk. A single insulin result is affected by food timing, concurrent glucose, the test method, insulin clearance, and treatment. It is not a standardized stand-alone measure of someone's insulin resistance. [4]

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Different tests answer different questions:

Test	Main question it helps answer
HbA1c and plasma glucose	Is glucose elevated, and how well is it being managed? These —not insulin levels—are central to diagnosing diabetes.
Islet autoantibodies	Is an autoimmune process likely? These may help when type 1 and type 2 features overlap.
C-peptide, interpreted with concurrent glucose and clinical context	How much insulin is the pancreas producing? It can help resolve uncertain classification in insulin-treated people.
Insulin concentration	Useful for selected specialist questions, including some hypoglycemia evaluations, but not a routine diabetes screening or management test.

C-peptide is produced alongside the body's own insulin and is not part of injected insulin. Its interpretation depends on the circumstances; neither one reassuring value nor a negative antibody result is permission to stop insulin. When an insulin-treated person's diagnosis is uncertain, ask whether a supervised classification review would change care. [1] [4]

3. Remission is possible—but “reversal” needs a definition

There is an important, hopeful distinction between controlling glucose and meeting a formal definition of remission. Both are worthwhile outcomes. Good glucose control while taking medicine can protect health even if the person does not meet an off-medication definition. [3]

The 2021 international consensus ordinarily defines type 2 diabetes remission as HbA1c below 6.5%, persisting for at least three months without usual glucose-lowering medication. When HbA1c is unreliable, clinicians may use an appropriate alternative assessment. One good glucose reading or an improved HbA1c while still taking medication does not establish remission under this definition. [3]

Remission is not cure. Glucose can rise again with weight regain, illness, changes in treatment, or progression of the underlying condition. Prior exposure to high glucose may also leave continuing complication risk. The consensus recommends at least annual glycemic testing and continued eye, kidney, foot, blood-pressure, and weight surveillance; there is no evidence that these checks can safely be abandoned. [3]

How remission guidance developed

In 2009, a consensus statement initiated by the ADA proposed remission categories, while explicitly stating that its opinions were not the ADA's official position. In 2021, an ADA-convened international expert group proposed the current usual definition. Current 2026 ADA Standards continue to recognize that substantial, sustained weight loss can produce remission in some people. The 2009 expert statement should not be mislabeled an official ADA policy. [6] [3] [39]

Type 2 diabetes is not always permanently medication-dependent, and meaningful improvement is possible. No guideline establishes that every person—or most people across the entire type 2 population—can achieve durable medication-free remission with diet alone. [3] [40] [41] [42]

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4. Findings from dietary-intervention studies

Reported remission percentages depend on who participated, how intensive the intervention was, how remission was defined, and how long people were followed. These are not interchangeable estimates of one person's chances.

Study	Who and what were studied	Main result	Important limitation
DiRECT, one year	Adults aged 20–65, diabetes for less than six years, BMI 27–45, not using insulin; closely supported weight management with an initial formula-diet phase.	68/149 participants, or 46%, in the intervention arm achieved the trial's remission endpoint, versus 4% of controls.	Selected adults; not simply advice to “eat better.” The original endpoint required at least two months off diabetes medicines, before the later three-month consensus definition. [40]
DiRECT, larger achieved weight loss	Participants who actually lost at least 15 kg—about 33 lb—at one year.	31/36, or 86%, were in remission.	This was a small subgroup defined by achieved weight loss, not 86% of everyone with type 2 diabetes. It does not guarantee the same result for everyone who loses that amount. [40]
DiRECT, five-year extension	Selected participants offered continued maintenance support after the original trial; 85 had five-year data.	11/85, or 13%, were in remission at five years.	This was a selected extension cohort, not all original intervention participants. Durability and relapse matter. [41]
DIADEM-I, one year	Selected adults aged 18–50 in Qatar, of Middle Eastern/ North African background, BMI at least 27, diabetes duration no more than three years; intensive diet/activity support.	Reported remission was 61% versus 12% with usual care.	A young, early-disease cohort. The verified original abstract does not provide the full operational remission definition; do not silently substitute the 2021 definition. [43]
Look AHEAD	A broader US trial cohort of adults with type 2 and overweight/obesity; intensive lifestyle intervention versus diabetes support/education.	Any off-medication remission was 11.5% at year one and 7.3% at year four in the intervention group, versus 2% in controls at both times.	Different participants and intervention; lower rates show why one high-response study cannot stand for everyone. [42]

DiRECT also reported 36% remission at two years in its original intervention arm. The fall in remission over longer follow-up does not erase the earlier benefits; it demonstrates why sustained support is part of treatment rather than an optional extra. Regaining weight or needing medicine again should prompt help, not shame. [41]

How ketogenic diets fit the evidence

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DiRECT tested a structured weight-management program, not a universal ketogenic prescription. A 2021 BMJ review of low-carbohydrate trials found favorable six-month results when “remission” allowed continued medication, but the off-medication findings were less certain and a clear benefit was not established at twelve months. Definitions and follow-up length change the interpretation. [40] [44]

Reducing carbohydrate can be useful for some people, but carbohydrate amount, food quality, total energy intake, sustainability, nutritional adequacy, and medication safety all matter. The right plan is one that is safe for the individual and can be maintained—not necessarily the strictest plan advertised. [39] [44]

5. Making dietary change practical

For adults with type 2 and overweight or obesity, the 2026 ADA Standards report that 5–7% weight loss improves glucose and cardiovascular risk factors, while sustained loss above 10% can have greater disease-modifying effects and may produce remission. These are clinical findings, not weight-loss assignments for every reader. People without excess weight, pregnant people, children, frail adults, and people with eating disorders need different considerations. [39] [19]

Useful starting questions are:

• What do I actually drink each day, including café drinks, juice, sweetened tea, soda, sports drinks, and flavored milk?

• How large are my portions compared with the label's serving size?

• Where can I replace sweetened products with unsweetened options I enjoy?

• Am I concentrating only on sugar while overlooking large portions of digestible starch?

• What nutrition changes are affordable, culturally acceptable, and realistic in my household?

• Can a registered dietitian or diabetes education program help me make a plan and coordinate medication adjustments?

These questions turn a broad instruction to “eat better” into choices that can be reviewed with the care team. There is no single required eating pattern for every person with type 2 diabetes. Activity and long-term support also matter. [7] [45] [39] [5]

Do not copy a very-low-calorie research diet on your own. Such programs involve patient selection, medication review, monitoring, food reintroduction, and maintenance support. Adult remission trial protocols should not be imposed on growing children. [40] [39] [19]

6. “Hidden sugar”: names, labels, and serving sizes

Sugar is not always listed simply as “sugar.” Ingredient names can make it less obvious, and repeated sweetened foods and drinks can add up before a person realizes how much they are consuming. The solution is not to memorize every possible name: use both the ingredient list and the Nutrition Facts panel. [7] [46]

Names or descriptions you may see	What to understand
Sugar, table sugar, sucrose, dextrose	These are examples of sugars used as ingredients; a chemical name does not make them sugar-free.

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Corn syrup, high-fructose corn syrup, other syrups	Syrup-based sweeteners can contribute added sugar.
Honey, maple syrup, agave syrup	“Natural” does not mean non-sugar. These still count as added/free sugars in the relevant definitions.
Fruit-juice concentrate used as a sweetener	Fruit-related wording does not necessarily mean the same thing as eating intact fruit.

Check labels on cereals, granola, flavored yogurt, breads, sauces, dressings, snack products, and drinks rather than assuming that a health-oriented name means little added sugar. Formulations differ; not every product in these categories is sweetened. [7] [46]

Read four parts of the label

1. Serving size and servings per container. If you consume two servings, you consume twice the listed carbohydrate and sugar.

2. Total Carbohydrate. Sugar is not the only carbohydrate relevant to glucose. Digestible starch also breaks down into glucose.

3. Total Sugars. This includes naturally occurring sugars and added sugars.

4. Includes X g Added Sugars. This amount is already included in Total Sugars—do not add the two numbers together.

For example, a hypothetical label with 20 g Total Sugars, including 15 g Added Sugars, contains 20 g total—not 35 g. If you consume two servings, that becomes 40 g total, including 30 g added. [7]

The FDA's 50 g Added Sugars Daily Value for a 2,000-calorie diet is a reference used for label percentages, not a personal daily goal and not an amount everyone should try to consume. There is no FDA Daily Value for Total Sugars. The American Heart Association gives lower general adult added-sugar limits: 25 g/day for women and 36 g/day for men. These are general limits, not individualized diabetes meal plans. [7] [46]

“No added sugar” is not “no effect on glucose”

Plain milk and intact fruit contain naturally occurring sugars and should not automatically be treated as nutritionally identical to candy. They also are not carbohydrate-free. Likewise, bread, rice, pasta, and potatoes can affect glucose through starch even when little sugar appears on the label. Portion size and the overall eating pattern matter. [7] [45]

WHO's free sugars category is broader than the FDA's Added Sugars category: it also includes sugars naturally present in fruit juice, honey, and syrups, while excluding sugars inside intact fruit/vegetables and milk. Thus a 100% juice with no added sugar is not equivalent to water, and whole fruit is not interchangeable with juice. [47]

7. That café drink may be a substantial sugar source

The sugar content of a sweetened café drink can be easy to underestimate if it is thought of simply as “coffee.” Check the exact drink, size, standard recipe, and customizations.

Verified example: the US Starbucks menu for a Grande, 16-fl-oz Mocha Frappuccino Blended Beverage lists 370 calories, 54 g total carbohydrate, and 51 g total sugars, accessed September 22, 2026. That is roughly 13 teaspoons of total sugar by weight, using about 4 g per teaspoon. [48]

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The distinction matters: 51 g is total sugar, not a verified 51 g of added sugar. Milk contributes naturally occurring lactose, while syrups and sauces contribute added sugars. Starbucks does not provide an added-sugar breakdown for this example. Do not compare its total directly with the FDA's 50 g Added Sugars Daily Value or claim all 13 teaspoons were added during preparation. [48] [7]

This is one standard drink, not a claim that all coffee drinks have the same content. Size and customization change the result. Useful questions include: “Is this sweetened by default? What is in the base? Can I order a smaller size or an unsweetened option?” Removing one topping does not necessarily remove sugar already in the base or sauce. [48]

Sugar-sweetened drinks are associated with type 2 diabetes risk and can contribute substantial calories. But one drink does not prove what caused an individual's diabetes. Genetics, physiology, activity, body composition, medicines, food access, and social circumstances also matter. [49] [11] [19]

8. Why “adult-onset” no longer describes type 2

Type 1 was historically called “juvenile diabetes,” and type 2 “adult-onset diabetes.” These names were never reliable biological rules. Adults can develop type 1, and children can develop type 2; current names describe disease types rather than age groups. The terminology change cannot be attributed to one recent year or one cause. [10] [50] [1]

The increase in youth type 2 is real. In the US SEARCH surveillance study, incidence among 10–19-year-olds rose from 9.0 per 100,000 in 2002–2003 to 17.9 per 100,000 in 2017–2018. These are annual incidence rates, not percentages. The study used five-center surveillance and did not establish rates in children under ten. [51]

Elementary-school-age cases are possible and have been documented historically. Type 2 below age ten is uncommon. A case series published in 1992 described diagnoses at ages 7–14 during 1984–1990. That documents earlier cases; it does not establish how common type 2 is among elementary-school children today. [19] [52]

Diet and activity are important modifiable factors, but it is inaccurate and unfair to reduce childhood type 2 to “the child ate sugar.” Puberty-related insulin resistance, genetics, family history, maternal diabetes, adiposity, food and activity environments, and social conditions contribute. Family-centered care should support growth and mental health rather than shame or punitive restriction. [19]

Youth-onset type 2 can progress aggressively. The TODAY studies documented difficulty maintaining glucose control and substantial complications by young adulthood in the studied cohort. This is a reason for prompt pediatric diabetes care and long-term support—not a prediction that every child will have the same outcome. Adult remission trial percentages should not be presented as pediatric cure rates. [53] [54] [19]

9. What uncontrolled diabetes can damage

Type 2 diabetes may be relatively quiet while injury develops. Persistently elevated glucose and associated cardiovascular risks can affect small blood vessels, large arteries, and nerves. Complications are serious possibilities, not inevitable outcomes, and glucose control is only one part of prevention. [8] [28]

Area	Possible consequences	What to discuss with the care team

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Eyes	Retinal blood-vessel damage and macular edema can impair vision or cause blindness.	A dilated comprehensive eye examination at type 2 diagnosis and follow-up based on findings; do not wait for blur. [55]
Heart and coronary arteries	Coronary disease and heart attack; some people need stents or coronary artery bypass surgery. Diabetes also increases heart-failure risk.	Blood pressure, cholesterol, smoking, symptoms, and treatments that reduce cardiovascular risk. Bypass is a selected treatment, not an inevitable stage. [28]
Brain	Stroke and related disability.	Blood-pressure and lipid management, smoking cessation, and emergency recognition of stroke symptoms. [28]
Legs and feet: circulation	Peripheral artery disease reduces arterial blood flow, often to the legs and feet; poor healing can contribute to limb-threatening wounds.	Foot examinations, pulses/circulation assessment, and prompt evaluation of wounds or concerning symptoms. [28] [55]
Peripheral nerves	Burning, tingling, electric or stabbing pain; sometimes loss of sensation rather than pain.	Neuropathy assessment and foot protection. Numbness can allow injuries to go unnoticed. [55]
Feet and limb loss	Nerve damage, poor circulation, ulcers, and infection can combine and lead to amputation.	Regular foot care and urgent assessment of new ulcers, spreading redness, or other signs of infection. Amputation is not unavoidable. [55]
Kidneys	Chronic kidney disease can progress to kidney failure requiring dialysis or transplantation.	At least annual urine albumin-to-creatinine ratio and estimated kidney filtration testing in type 2, with closer follow-up when disease is present. [56]

Diabetes-associated kidney disease is the leading cause of kidney failure in the United States, according to the 2026 ADA Standards. This burden statement concerns diabetes overall, not type 2 alone. About one in three adults with diabetes has kidney disease, according to NIDDK; that statistic does not mean one in three has kidney failure. Early disease may have no symptoms, which is why urine and blood tests matter. [56] [57]

The circulation problem in the limbs is peripheral arterial disease. It is distinct from coronary disease involving the heart's arteries. Both deserve attention alongside glucose—not after glucose management has “failed.” [28]

10. Protecting organs is broader than lowering HbA1c

Ask about an individualized glucose target, blood pressure, cholesterol, smoking, activity, nutrition, and recommended screening. Lowering HbA1c is important, but it is not a substitute for treating high blood pressure or high cardiovascular risk. [28]

For selected people with type 2 and cardiovascular or kidney disease, medicines such as SGLT2 inhibitors or GLP-1 receptor agonists have benefits beyond additional glucose lowering. Other treatments, including appropriate blood-pressure medicines and finerenone in selected kidney disease, may also be indicated. Which treatment fits depends on kidney function, other conditions, risks, access, and clinical assessment. [28] [56]

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Being medication-free is not more important than being protected. If a drug remains appropriate for heart, kidney, or weight management, discuss that benefit rather than stopping it to earn a remission label. The consensus notes that continued glucose-lowering drugs can make drug-independent remission impossible to determine, even when they are prescribed for another reason. [3] [28] [56]

11. Make major changes safely—and recognize emergencies

If you use insulin or a medicine that stimulates insulin release, such as a sulfonylurea, a major reduction in carbohydrate or calories can lead to low glucose unless treatment is reviewed. Arrange a glucose-monitoring plan, know your low-glucose action plan, and ask who to contact for dose adjustments. Do not improvise medication changes. [5] [58]

People taking an SGLT2 inhibitor need specific advice before fasting or starting a ketogenic diet, plus an individualized plan for illness and procedures. Current prescribing information identifies reduced caloric intake, ketogenic diets, missed or reduced insulin, illness, surgery, volume depletion, and alcohol misuse as ketoacidosis triggers. If symptoms that could signal ketoacidosis develop—nausea, vomiting, abdominal pain, unusual tiredness or malaise, or difficult/rapid breathing—stop the SGLT2 inhibitor and seek immediate medical care, even if glucose is not very high. Do not reduce or omit insulin on your own. [59] [20]

Seek emergency care now for marked confusion, extreme drowsiness, deep or rapid breathing, persistent vomiting, inability to keep fluids down, or severe dehydration. Type 2 does not make someone immune to diabetic ketoacidosis or hyperosmolar hyperglycemic state. Follow your prescribed sick-day/ketone plan, but do not delay emergency care while waiting for a routine response. [20]

Severe low glucose with seizure, unconsciousness, or inability to swallow safely is an emergency: call emergency services and give glucagon if available, as directed. Do not give food or drink by mouth to an unconscious person or someone unable to swallow safely. Ask your team in advance about rescue treatment and teach household members how to use it. [60] [58]

These safety considerations make coordination important so that treatment keeps pace with dietary changes and improvement.

12. What about “type 3 diabetes” and Alzheimer's?

Researchers sometimes use “type 3 diabetes” to discuss possible links between impaired insulin signaling in the brain and Alzheimer's disease. It is a proposed research label, not an established clinical diabetes diagnosis or an official replacement name for Alzheimer's disease. Shared mechanisms and associations do not mean that everyone with Alzheimer's has diabetes, that everyone with type 2 will develop Alzheimer's, or that dietary glucose control cures dementia. [61] [1]

13. Questions to take to your diabetes team

• How certain are we about my diabetes type? Would selective antibody or C-peptide testing change management?

• What are my HbA1c, glucose pattern, kidney filtration, urine albumin, blood pressure, and cholesterol results?

• What dietary changes are likely to help me most, and can I see a dietitian or diabetes educator?

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• Am I a reasonable candidate for a supported remission program? What outcomes are realistic given disease duration and treatment?

• If I reduce carbohydrate or calories, who will adjust my medicines and how should I monitor glucose?

• Could stress, inflammatory disease, steroid treatment, blood pressure, or changes in sexual health affect my care plan?

• Do my medicines require precautions with illness, fasting, a ketogenic diet, or surgery?

• Which medicines are protecting my heart or kidneys, not just lowering glucose?

• When are my eye and foot examinations due?

• If I achieve remission, what follow-up continues and what would signal relapse?

• If this concerns a child, how will the plan support growth, nutrition, mental health, and the whole family?

Use these questions to make a shared plan, not to request every test or stop treatment independently. [1] [3] [19] [28]

Frequently Asked Questions

If my insulin is high, why would I ever need insulin treatment?

A level that is high compared with a reference population may still be inadequate for the body's resistance. Some people also develop much lower insulin production over time or need insulin during particular illnesses or periods of marked hyperglycemia. The treatment decision depends on the whole clinical situation, not a belief that all type 2 is “too much insulin.” [1] [2] [5]

Can I improve my health even if I never reach remission?

Yes. Better glucose control, sustainable nutrition and activity changes, and treatment of cardiovascular and kidney risks are worthwhile without an off-medication label. Needing medication does not erase the benefits of lifestyle changes. [39] [28] [56]

Must I eliminate all fruit or all carbohydrates?

No universal rule requires that. Intact fruit, milk, sweets, and starches differ in composition even though carbohydrate from each can matter to glucose. Portions, the overall pattern, nutritional adequacy, preferences, and medication safety should guide the plan. A restrictive adult diet is not an appropriate default for a child. [7] [47] [45] [19]

If my glucose improves, can I stop eye and kidney checks?

No. Remission does not erase past exposure or all future risk. Continue the recommended diabetes follow-up, including glycemic testing and complication surveillance. [3]

Evidence and limitations

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This guide draws on current ADA Standards, original remission and youth studies, the international remission consensus, laboratory guidance, human observational and intervention research, government patient information, and an official product nutrition page. Source-review notes distinguish full text from abstract-only records and search-discovered links. Animal mechanisms and researcher lectures are not human treatment trials. Where only an original abstract was verified, conclusions are limited to the abstract. Trial populations, remission definitions, and follow-up periods differ; the percentages should not be pooled into a universal success rate.

The evidence supports meaningful dietary intervention and the possibility of remission. Remission guidance predates 2025; remission is not a guaranteed cure; childhood disease cannot be attributed to one food; and a proposed research label is not a dementia diagnosis. The goal is informed, supported action—and continued protection of eyes, kidneys, nerves, heart, brain, and feet.

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