PREKURE CLINIC

THE PREKURE CLINIC APPROACH · INSULIN RESISTANCE AND DIABETES

Insulin resistance, prediabetes and type 2 diabetes

Get your metabolism working properly again.

Insulin resistance, prediabetes and type 2 diabetes are stages of one problem: a metabolism that is losing its ability to return to normal after a meal. The good news is that it can often be turned around, and the earlier you start, the easier it is.

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The PREKURE Clinic · Lifestyle medicine · Metabolic medicine · Behavioural medicine · Updated November 2026

PART 1

The PREKURE Clinic approach at a glance

What type 2 diabetes really is. A healthy metabolism copes with a meal and returns to normal within a few hours. Type 2 diabetes is the late stage of a metabolism that can no longer do this. For years before diagnosis, the body becomes resistant to insulin and makes more and more of it to keep glucose in range, while fat builds up in the liver and pancreas. High insulin also switches off fat burning, so the body stays stuck in storage mode, which helps explain the hunger, weight gain and low energy many people feel. Blood glucose is the last number to change. Prediabetes and type 2 diabetes are the stages where it finally does.

Why act before the glucose number changes. Insulin resistance and high insulin travel with heart and blood vessel disease, kidney disease, fatty liver, dementia, depression and several cancers. Most of that evidence is observational, but the mechanisms through high insulin are plausible, so we treat them as a shared pathway. About 1 in 4 New Zealand adults had prediabetes in the last national survey, and in the United States more than 8 in 10 people with prediabetes do not know they have it. Most people do little about it, because a slightly high number does not feel urgent. We think it is the best moment to act.

The good news: it can be reversed. Remission means a normal HbA1c without glucose-lowering medicines. In the DiRECT trial, 46 in 100 people diagnosed within the previous 6 years reached remission at one year with a structured diet programme, against 4 in 100 with usual care, and 86 in 100 of those who lost 15 kg or more. The earlier, the better. As glucose and insulin fall, many people find that hunger settles, energy and mental clarity improve, and weight comes down. Remission fades if weight returns, so ongoing support matters.

How we get there. Weight loss is the strongest driver of remission, and lower-carbohydrate eating is our first-line way to get there. It is an effective, guideline-endorsed option that lowers glucose, triglycerides and medicine use within months. Food is only part of the picture. Sitting for long periods, poor sleep, stress, late meals and low muscle mass push glucose and insulin up. Walking after meals, strength training and better sleep bring them down. We find the one or two changes that move your numbers most and that you can keep, test them on your own glucose, and adjust as we go.

Medicines. Most people diagnosed with type 2 diabetes start metformin alongside lifestyle change, as New Zealand guidance recommends. It is cheap, rarely causes low glucose and does not cause weight gain, and in overweight people in the UK Prospective Diabetes Study it cut heart attacks by 39% and deaths by 36%. Stomach upset is common at first, and long-term use can lower vitamin B12, which we check. Taking metformin does not stop anyone working towards remission: as glucose falls, we review whether it is still needed, and remission is confirmed 3 months after stopping it. SGLT2 inhibitors and GLP-1 medicines reduce heart attacks, heart failure, kidney failure and deaths in people at higher risk. These medicines do not produce remission, and their benefits last only while they are taken. Diet and medicines work together, not as alternatives.

Our stance

1. Type 2 diabetes is the late stage of a metabolism that cannot return to normal, and the earlier stages, insulin resistance and prediabetes, deserve action too.
2. Type 2 diabetes can often be put into remission, especially in the first 5 years, and we aim for it wherever it is realistic.
3. Lower-carbohydrate eating is our first-line dietary approach, supported by health coaches and started when people are ready. Total diet replacement and other approaches are also legitimate.
4. Plans are personalised to what drives each person's glucose and insulin and what they can sustain, then adjusted using their own results.
5. Cutting carbohydrate safely needs a medicine plan, and we use heart- and kidney-protecting medicines where they are indicated, whatever the diet. Insulin is not the default answer to insulin resistance: we aim to reduce the need for it, and use it when the pancreas is failing or glucose is dangerously high.
6. Remission is not a cure. Eye, kidney and HbA1c checks continue every year, and we offer support when weight or glucose rises.
7. We count everyone who starts, and we publish our results.

PART 2 · THE EVIDENCE

How a metabolism stops returning to normal

Insulin rises years before glucose. In the Whitehall II study, 6,538 British civil servants were followed for about 10 years, and 505 developed diabetes. In those people, insulin sensitivity fell steeply over the 5 years before diagnosis, and insulin output rose to compensate before falling. Glucose rose steeply only in the final 3 years (Tabák, Lancet 2009). A normal glucose test can hide years of rising insulin.

There is more than one path. A study of people at raised risk found six distinct groups. Some mainly made too little insulin, one had marked fatty liver and insulin resistance with a very high risk of diabetes, and one already had kidney damage

and higher death rates before diabetes was diagnosed (Wagner, Nat Med 2021). Glucose alone misses these differences.

Why high insulin matters. Insulin is the body's main storage signal. Even modest rises block the release of fat from fat stores, and high levels act as a growth signal in blood vessels and some tissues. That makes high insulin a plausible shared driver of weight gain, fatty liver, heart disease and some cancers (Crofts, Diabesity 2015; Janssen, Int J Mol Sci 2021). For eye, kidney and nerve damage, the causal case for high glucose is strong, because trials that lowered glucose prevented them. For heart disease, cancer and dementia, the human evidence is mostly observational, and insulin resistance, liver fat, high blood pressure and abnormal blood fats cluster together. The practical point does not depend on settling that debate: losing liver fat and lowering glucose and insulin improve the whole cluster at once.

What we measure. Alongside HbA1c and fasting glucose, we look at triglycerides and HDL cholesterol, liver tests, waist and blood pressure, which can show a metabolism under strain before glucose rises.

How common and how serious

Measure	Finding	Source
Diabetes in New Zealand	About 307,000 on the register (types 1 and 2); 5.5% of adults have type 2 diabetes	Health NZ register; Verster, PLoS One 2026
Prediabetes in New Zealand	25.5% of adults (2008/09)	Coppell, NZ Med J 2013
Life expectancy	About 14 years shorter if diagnosed at 30, 10 years at 40, 6 years at 50	Kaptoge, Lancet Diabetes Endocrinol 2023
Heart attack and stroke	About twice the risk with diabetes; 13% to 30% higher risk already with prediabetes	ERFC, Lancet 2010; Huang, BMJ 2016
Kidney failure in New Zealand	Diabetes caused 46% of new dialysis or transplant patients in 2024	ANZDATA 2025

Measure	Finding	Source
Fatty liver	About 55 in 100 people with type 2 diabetes	Younossi, J Hepatol 2019
Cancer and dementia	Robust links with breast, bowel and womb cancer; diabetes is one of 14 modifiable dementia risk factors	Tsilidis, BMJ 2015; Livingston, Lancet 2024
Eye, kidney and nerve damage	Each 1 point lower HbA1c linked to 37% fewer of these complications	UKPDS 35, BMJ 2000

Remission and how long it lasts

Remission means an HbA1c under 48 mmol/mol at least 3 months after stopping glucose-lowering medicines (Riddle, Diabetes Care 2021). It depends mostly on weight lost. In DiRECT at 12 months, remission was reached by none of those who gained weight, 7% of those who lost up to 5 kg, 34% at 5 to 10 kg, 57% at 10 to 15 kg and 86% at 15 kg or more (Lean, Lancet 2018). Quality of life also improved. It depends on duration too. Those whose insulin-producing cells recovered had had diabetes for a shorter time (Taylor, Cell Metab 2018), and in one practice remission was 77% within a year of diagnosis against 20% after 15 years (Unwin 2023).

Programme	Year 1	Later	Source
DiRECT (diet replacement)	46 in 100	36 in 100 at 2 years; about a quarter of 2-year remitters still in remission at 5 years	Lean 2019; Lean 2024
Look AHEAD (intensive lifestyle)	11.5 in 100	7.3 in 100 at 4 years	Gregg, JAMA 2012
Virta (ketogenic, non-randomised)		About 9 in 100 starters at 5 years	McKenzie 2024

Remission is still worth having. In Look AHEAD, it was linked to 40% less cardiovascular disease and 33% less kidney disease (observational; Gregg, Diabetologia 2024), and in DiRECT the diet group had under half the rate of serious adverse events over 5 years. Good glucose control early also has a lasting legacy: in the UK Prospective Diabetes Study, fewer heart attacks and

deaths persisted for decades after the trial ended (Holman, NEJM 2008; Adler, Lancet 2024).

Lower-carbohydrate and other diets

In trials, lower-carbohydrate usually means under about 130 g of carbohydrate a day, and ketogenic under about 50 g, low enough that the body runs mainly on fat and makes ketones for fuel (Goldenberg, BMJ 2021). Lower-carbohydrate eating reliably improves glucose and cuts medicine use in the short term. Trials do not show it beats other weight-loss diets beyond about a year.

Study	Finding	Quality
23 trials, 1,357 people	At 6 months, HbA1c under 6.5% in 57 against 31 in 100; at 12 months, HbA1c 0.23 points lower, LDL 0.14 mmol/L higher	Moderate (Goldenberg, BMJ 2021)
50 trials	Each 10% cut in carbohydrate share lowers HbA1c by 0.2 points at 6 months	Moderate (Jayedi, AJCN 2022)
Trials over 12 months	No HbA1c difference; better HDL and triglycerides	Moderate (Ichikawa, JDI 2024)
Adelaide, 2 years	Same weight and HbA1c; fewer medicines, steadier glucose and better blood fats on lower-carbohydrate	Moderate (Tay, DOM 2018)
Same weight loss, different diet (72 people, 6 weeks)	Lower-carbohydrate lowered HbA1c and triglycerides further at the same weight loss	Moderate (Thomsen, Diabetologia 2022)

Ketogenic diets also stop hunger rising as weight falls, which helps people keep going (Gibson, Obes Rev 2015).

Real-world programmes. Virta's coach-supported ketogenic model, which PREKURE's approach most resembles, lowered HbA1c from 7.6% to 6.3% with 12% weight loss at one year in a non-randomised, company-funded study (Hallberg, Diabetes Ther 2018). In Dr David Unwin's practice, 51% of the 186

patients who chose lower-carbohydrate eating reached drug-free remission over about 33 months. Neither had a randomised comparison group, and people chose the approach.

Guidelines and safety. The American Diabetes Association suggests considering lower carbohydrate intake for some adults (ADA 2026), and the New Zealand Society for the Study of Diabetes lists low-carbohydrate and ketogenic eating among acceptable options (NZSSD). Insulin and sulfonylureas usually need reducing before carbohydrate is cut. SGLT2 inhibitors with very low-carbohydrate eating raise the risk of ketoacidosis, so NZSSD advises against the combination. LDL cholesterol rises in some people and should be checked.

The other levers

Most of this evidence shows changes in glucose and insulin after meals or in diabetes risk, not remission on its own. In a person with several drivers, the effects add up.

Driver	What the evidence shows	What we do
Sitting for long periods	2 minutes of light walking every 20 minutes cut glucose after a meal by 24% and insulin by 23% (Dunstan 2012; Buffey 2022)	Walks after meals, movement breaks
Exercise training	Structured exercise lowered HbA1c by 0.67 points across 23 trials; advice alone did little (Umpierre, JAMA 2011)	Coached aerobic and strength training
Muscle mass	Each 10% more muscle linked to 11% lower insulin resistance (Srikanthan 2011)	Strength training and enough protein
Food order	Vegetables and protein before carbohydrate cut glucose after the meal by up to 37% (pilot) (Shukla 2015)	Vegetables and protein first
Individual responses	Glucose responses to identical meals varied widely between 800 people (Zeevi, Cell 2015)	Personal testing
Sleep and sleep apnoea	Short sleep raises diabetes risk by 28%, moderate to severe apnoea by 63% (Cappuccio 2010; Wang 2013)	Sleep coaching; screening for apnoea

Driver	What the evidence shows	What we do
Meal timing and shift work	Late dinners worsened night-time glucose; shift work linked to 9% higher odds of diabetes (Gu 2020; Gan 2015)	Earlier, regular meals
Stress, low mood and smoking	Depression raises diabetes risk by 60%, smoking by 37% (Mezuk 2008; Pan 2015)	Mental fitness support; quit support
Medicines	Steroids cause high glucose in about a third of users; statins add about 1 case per 255 people over 4 years, far outweighed by heart protection (Liu 2014; Sattar 2010)	Medicine review; statins are not stopped for this

A finger-prick meter or continuous glucose monitor lets people see what a walk, a late dinner or a poor night does to their own glucose. In type 2 diabetes without insulin, monitors lowered HbA1c by about 0.3 points (Jancev, Diabetologia 2024). We use them as a short-term learning tool. They are not funded for type 2 diabetes in New Zealand.

Medicines and New Zealand access

Medicine (who was studied)	Out of 100 people	Time	Source
Metformin, early treatment	About 5 fewer deaths and 6 fewer heart attacks	About 17 years	UKPDS 91, Lancet 2024
Empagliflozin (heart disease)	1.6 fewer major cardiovascular events; 2.6 fewer deaths	3.1 years	EMPA-REG, NEJM 2015
Dapagliflozin (kidney disease)	5.3 fewer major kidney events	2.4 years	DAPA-CKD, NEJM 2020
Liraglutide (high cardiovascular risk)	1.9 fewer major cardiovascular events; 1.4 fewer deaths	3.8 years	LEADER, NEJM 2016

Medicine (who was studied)	Out of 100 people	Time	Source
Semaglutide (kidney disease)	4.5 fewer major kidney events	3.4 years	FLOW, NEJM 2024
Vildagliptin and other DPP-4 inhibitors (sitagliptin, heart disease)	No change in heart attacks, strokes or heart failure; they lower HbA1c modestly, do not cause weight gain and rarely cause low glucose	3 years	TECOS, NEJM 2015
HARMS			
Metformin	Stomach upset common at first; long-term use can lower vitamin B12	Ongoing	ADA 2026
Empagliflozin	About 3 more genital yeast infections	Pooled trials	FDA label 2023
SGLT2 inhibitors	Ketoacidosis about 1 in 3,000, higher with very low-carbohydrate eating		NZSSD
Liraglutide	About 1 more stops because of nausea; about 1 more has gallstones	3.8 years	LEADER
Insulin	About 1 more major low-glucose episode each year; about 4 kg weight gain	Per year	UKPDS 33, Lancet 1998

NZSSD recommends an HbA1c under 53 mmol/mol for most people, lower for younger people at low risk of low glucose, and higher for frail people. Someone in remission who has heart or kidney disease may still benefit from an SGLT2 inhibitor or GLP-1 medicine, matched safely to their diet.

Insulin: when it is needed, and when we try to avoid it. Insulin is essential when the pancreas cannot make enough, when glucose is very high with symptoms or weight loss, in pregnancy, and during serious illness. But when high glucose comes mainly from insulin resistance and a high-carbohydrate, highly processed diet, adding insulin treats the number, not the cause. It brings weight gain and low-glucose episodes (UKPDS 33), and in a body already flooded with insulin it

adds to the load. In the ACCORD trial, people with long-standing diabetes were given medicines to push glucose down hard, and three-quarters of them were on insulin. This brought more low-glucose episodes, more weight gain and more deaths, and that part of the trial was stopped early. Why more people died is not known (Gerstein, NEJM 2008). Guidelines now prefer a GLP-1 medicine before insulin for most people who need an injection (ADA 2026). We aim to reduce the need for insulin by eating less carbohydrate and moving glucose into muscle through activity and strength training. In the Virta programme, 94% of people using insulin had reduced or stopped it by one year (Hallberg 2018). Large trials have not shown that insulin itself causes heart disease or cancer (ORIGIN, NEJM 2012), so this is a reason to need less insulin, not a reason to refuse it when it is needed.

From 1 September 2026, Pharmac widened access to empagliflozin, dulaglutide and liraglutide for anyone with an HbA1c above 53 mmol/mol despite other funded treatment (Pharmac). Vildagliptin (Galvus, or Galvumet with metformin) is also funded, and funded GLP-1 medicines usually require metformin, empagliflozin and vildagliptin to have been tried for at least 6 months first. Combining an SGLT2 inhibitor and a GLP-1 medicine is funded only when the SGLT2 inhibitor is prescribed for heart failure, so in practice for people with type 2 diabetes and heart failure. Semaglutide and tirzepatide are available privately but not funded.

How the PREKURE Clinic chooses a plan

Situation	Focus
Insulin resistance or prediabetes, normal or near-normal glucose	Get the metabolism working again: lower-carbohydrate eating, movement, strength and sleep; track HbA1c, triglycerides, liver tests and waist
Diagnosed in the past few years, overweight	Aim for remission: lower-carbohydrate eating or total diet replacement, targeting 10 to 15 kg weight loss

Situation	Focus
Longer duration or on insulin	Better control and fewer medicines, including reducing insulin where it is mainly treating insulin resistance; remission is less likely but possible; a low threshold for working with the person's diabetes specialist team
Heart disease, heart failure or kidney disease	SGLT2 inhibitor or GLP-1 medicine for protection, matched safely to the diet
In remission	Keep the weight off; HbA1c, eye and kidney checks at least yearly

Questions to ask: What is driving my glucose and insulin? Is remission realistic for me? Which way of eating can I sustain? Which medicines need changing before I cut carbohydrate? How will we know it is working?

What the PREKURE Clinic does

1. Medical assessment with a lifestyle-medicine-trained doctor: HbA1c, lipids, liver and kidney tests, blood pressure, heart and kidney risk, and a full medicine review.
2. A safe medicine plan before diet change, reducing insulin and sulfonylureas and reviewing SGLT2 inhibitors before carbohydrate is cut, with a low threshold for working with the person's diabetes specialist team when insulin has been used for years.
3. A matched PREKURE health coach for lower-carbohydrate eating or other approaches, family meals, sleep, stress, movement after meals, strength training and personal glucose testing.
4. Heart and kidney protection with funded medicines where indicated, in partnership with your GP.
5. Regular monitoring, with deprescribing as glucose improves, and re-engagement when weight or glucose rises, not blame.
6. Outcome tracking, counting everyone who starts: remission, HbA1c, medicines and weight at 1 and 2 years.

What the PREKURE Clinic will not claim: that type 2 diabetes is cured; that lower-carbohydrate eating is proven better than other diets long term; that most people will reach remission; that high insulin is proven to cause every chronic disease; that diet makes heart and kidney medicines unnecessary; or that medicines make diet irrelevant.

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This page is general information based on published evidence as at November 2026. It is not personal medical advice. Talk with your doctor or a PREKURE Doctor about what is right for you.

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Selected references

Figures were checked against primary sources on 30 October 2026. "Per 100" figures are our arithmetic from published rates.

How metabolism goes wrong

- Wagner R et al. Pathophysiology-based subphenotyping of individuals at elevated risk for type 2 diabetes. Nat Med 2021. doi:10.1038/s41591-020-1116-9
- Crofts CAP, Zinn C, Wheldon MC, Schofield GM. Hyperinsulinemia: a unifying theory of chronic disease? Diabesity 2015. Record
- Janssen JAMJL. Hyperinsulinemia and its pivotal role in aging, obesity, type 2 diabetes, cardiovascular disease and cancer. Int J Mol Sci 2021. doi:10.3390/ijms22157797
- Tabák AG et al. Trajectories of glycaemia, insulin sensitivity and insulin secretion before diagnosis of type 2 diabetes (Whitehall II). Lancet 2009. doi:10.1016/S0140-6736(09)60619-X
- Taylor R et al. Remission of human type 2 diabetes requires decrease in liver and pancreas fat. Cell Metab 2018. doi:10.1016/j.cmet.2018.07.003
- Gibson AA et al. Do ketogenic diets really suppress appetite? A systematic review and meta-analysis. Obes Rev 2015. doi:10.1111/obr.12230

How common and how serious

- Coppell KJ et al. Prevalence of diagnosed and undiagnosed diabetes and prediabetes in New Zealand: 2008/09 Adult Nutrition Survey. N Z Med J 2013;126(1370):23-42.
- Centers for Disease Control and Prevention. Prediabetes awareness: more than 8 in 10 adults with prediabetes do not know they have it. Report
- Health NZ. Virtual Diabetes Register
- Verster F et al. Life expectancy with and without diabetes in Aotearoa New Zealand. PLoS One 2026. doi:10.1371/journal.pone.0345892
- Kaptoge S et al. Life expectancy by age at diagnosis of type 2 diabetes. Lancet Diabetes Endocrinol 2023. doi:10.1016/S2213-8587(23)00223-1
- Emerging Risk Factors Collaboration. Diabetes, fasting glucose and risk of vascular disease. Lancet 2010. doi:10.1016/S0140-6736(10)60484-9
- Huang Y et al. Prediabetes and cardiovascular disease and mortality. BMJ 2016. doi:10.1136/bmj.i5953
- Forcey D et al. ANZDATA 48th Report, chapter 1: incidence of kidney failure with replacement therapy. 2025. ANZDATA Annual Report 2025
- Younossi ZM et al. Fatty liver disease in type 2 diabetes. J Hepatol 2019. doi:10.1016/j.jhep.2019.06.021
- Tsilidis KK et al. Type 2 diabetes and cancer: umbrella review. BMJ 2015. doi:10.1136/bmj.g7607
- Livingston G et al. Dementia prevention, intervention and care: 2024 Lancet Commission. Lancet 2024. doi:10.1016/S0140-6736(24)01296-0
- Stratton IM et al. UKPDS 35: glycaemia and complications. BMJ 2000. doi:10.1136/bmj.321.7258.405

Remission and diet

- Riddle MC et al. Definition of remission. Diabetes Care 2021. doi:10.2337/dci21-0034
- Lean MEJ et al. DiRECT 1 year. Lancet 2018. doi:10.1016/S0140-6736(17)33102-1; 2 years, Lancet Diabetes Endocrinol 2019. doi:10.1016/S2213-8587(19)30068-3; 5 years, 2024. doi:10.1016/S2213-8587(23)00385-6
- Gregg EW et al. Look AHEAD remission. JAMA 2012. doi:10.1001/jama.2012.67929
- Gregg EW et al. Remission and complications in Look AHEAD. Diabetologia 2024. doi:10.1007/s00125-023-06048-6
- Goldenberg JZ et al. Low-carbohydrate diets meta-analysis. BMJ 2021. doi:10.1136/bmj.m4743
- Jayedi A et al. Carbohydrate dose-response. Am J Clin Nutr 2022. doi:10.1093/ajcn/nqac066
- Ichikawa T et al. Long-term low-carbohydrate diets meta-analysis. J Diabetes Investig 2024. doi:10.1111/jdi.14271
- Tay J et al. Two-year low-carb trial. Diabetes Obes Metab 2018. doi:10.1111/dom.13164
- Thomsen MN et al. Carbohydrate restriction at equal weight loss. Diabetologia 2022. doi:10.1007/s00125-021-05628-8
- Hallberg SJ et al. Virta 1 year. Diabetes Ther 2018. doi:10.1007/s13300-018-0373-9
- McKenzie AL et al. Virta 5 years. Diabetes Res Clin Pract 2024. doi:10.1016/j.diabres.2024.111898
- Unwin D et al. Norwood practice. BMJ Nutr Prev Health 2023. doi:10.1136/bmjnph-2022-000544
- American Diabetes Association. Standards of Care 2026, section 5. doi:10.2337/dc26-S005
- NZSSD. Type 2 diabetes management guidance

Other drivers of glucose and insulin

- Dunstan DW et al. Breaking up prolonged sitting. Diabetes Care 2012. doi:10.2337/dc11-1931
- Buffey AJ et al. Interrupting sitting with standing or light walking: meta-analysis. Sports Med 2022. doi:10.1007/s40279-022-01649-4
- Umpierre D et al. Structured exercise and HbA1c in type 2 diabetes. JAMA 2011. doi:10.1001/jama.2011.576

- Srikanthan P, Karlamangla AS. Muscle mass and insulin resistance. J Clin Endocrinol Metab 2011. doi:10.1210/jc.2011-0435
- Shukla AP et al. Food order and glucose after meals. Diabetes Care 2015. doi:10.2337/dc15-0429
- Zeevi D et al. Personalised nutrition by prediction of glycaemic responses. Cell 2015. doi:10.1016/j.cell.2015.11.001
- Cappuccio FP et al. Sleep and incident type 2 diabetes: meta-analysis. Diabetes Care 2010. doi:10.2337/dc09-1124
- Wang X et al. Sleep apnoea and type 2 diabetes: meta-analysis. Respirology 2013. doi:10.1111/j.1440-1843.2012.02267.x
- Gu C et al. Late dinner and glucose. J Clin Endocrinol Metab 2020. doi:10.1210/clinem/dgaa354
- Gan Y et al. Shift work and diabetes: meta-analysis. Occup Environ Med 2015. doi:10.1136/oemed-2014-102150
- Mezuk B et al. Depression and type 2 diabetes: meta-analysis. Diabetes Care 2008. doi:10.2337/dc08-0985
- Pan A et al. Smoking and type 2 diabetes: meta-analysis. Lancet Diabetes Endocrinol 2015. doi:10.1016/S2213-8587(15)00316-2
- Liu XX et al. Glucocorticoid-induced hyperglycaemia: meta-analysis. Ann Nutr Metab 2014. doi:10.1159/000365892
- Sattar N et al. Statins and incident diabetes. Lancet 2010. doi:10.1016/S0140-6736(09)61965-6
- Jancev M et al. Continuous glucose monitoring in type 2 diabetes: meta-analysis. Diabetologia 2024. doi:10.1007/s00125-024-06107-6

Medicines

- Adler AI et al. UKPDS 91 legacy effect. Lancet 2024. doi:10.1016/S0140-6736(24)00537-3
- Holman RR et al. UKPDS 80: 10-year follow-up of intensive glucose control. NEJM 2008. doi:10.1056/NEJMoa0806470
- UK Prospective Diabetes Study Group. UKPDS 33: intensive glucose control with sulfonylureas or insulin. Lancet 1998. doi:10.1016/S0140-6736(98)07019-6
- Zinman B et al. EMPA-REG OUTCOME. NEJM 2015. doi:10.1056/NEJMoa1504720
- Heerspink HJL et al. DAPA-CKD. NEJM 2020. doi:10.1056/NEJMoa2024816
- Marso SP et al. LEADER. NEJM 2016. doi:10.1056/NEJMoa1603827
- Perkovic V et al. FLOW. NEJM 2024. doi:10.1056/NEJMoa2403347
- US Food and Drug Administration. Jardiance (empagliflozin) prescribing information, 2023. FDA label
- Pharmac. Widened access to diabetes medicines, 2026
- Gerstein HC et al. Effects of intensive glucose lowering in type 2 diabetes (ACCORD). NEJM 2008. doi:10.1056/NEJMoa0802743
- ORIGIN Trial Investigators. Basal insulin and cardiovascular and other outcomes in dysglycemia. NEJM 2012. doi:10.1056/NEJMoa1203858
- American Diabetes Association. Standards of Care 2026, section 9: pharmacologic approaches to glycemic treatment. doi:10.2337/dc26-S009
- UK Prospective Diabetes Study Group. UKPDS 34: intensive glucose control with metformin in overweight patients with type 2 diabetes. Lancet 1998. doi:10.1016/S0140-6736(98)07037-8
- Green JB et al. Effect of sitagliptin on cardiovascular outcomes in type 2 diabetes (TECOS). NEJM 2015. doi:10.1056/NEJMoa1501352