TYPE 2 DIABETES

Research Summary

High Blood Sugar Is the Result, Not the Cause

This document follows the final 52-slide presentation in presentation order. For each cited study, it summarizes the study design, the main findings, and how the study is being used in the corresponding slide. The interpretation sections distinguish what the study directly demonstrates from the broader biological synthesis used in the video.

Evidence framework: Direct human evidence, prospective association, mechanistic/animal evidence, condition-specific evidence, and clinical synthesis are not treated as interchangeable. A study can support biological plausibility without proving the entire food–inflammation–diabetes pathway.

Research studies in slide order

Slide	Slide title	Study
7	The pancreas isn't lazy. It's compensating.	Bregenzer et al., 2006
11	Inflammation and insulin resistance	Pradhan et al., 2001
12	Inflammation and insulin resistance	Spranger et al., 2003
15	Gut inflammation can add to the load	Mkumbuzi et al., 2020
16	Food can drive the inflammatory signals	Tetzlaff et al., 2017
17–18	Autoimmunity and insulin resistance	Winer et al., 2011
19	Treating inflammation dropped A1C	Larsen et al., 2007
20	Treating inflammation dropped A1C	Goldfine et al., 2013
22	Immune food reactions and Type 1 Diabetes	Karjalainen et al., 1992
23	Immune food reactions and Type 1 Diabetes	Pastore et al., 2003
28	Changing inflammation can change the outcome	Ridker et al. / CANTOS, 2017
29	Changing inflammation can change the outcome	Huang et al., 2021
33	Weight-loss helps, but isn't the whole story	Wu et al., 2024
34	Blood sugar can improve without weight loss	Sandforth et al., 2025
38	The 2025 IBS trial — individual reactivity studied	Singh et al., 2025

The pancreas isn’t lazy. It’s compensating.

Bregenzer N, Hartmann A, Strauch U, Schölmerich J, Andus T, Bollheimer LC. Increased insulin resistance and beta cell activity in patients with Crohn’s disease. Inflamm Bowel Dis. 2006;12(1):53–56.
DOI: 10.1097/01.MIB.0000195975.97673.f5

Evidence type: Small human comparative study in an inflammatory disease.

Study basics

- 17 patients with Crohn’s disease and 13 healthy controls underwent a 75-g oral glucose tolerance test.
- Glucose and insulin were measured for five hours; insulin resistance and beta-cell function were calculated from the responses.
- The study is used as a supporting reference on the slide showing that normal glucose can be maintained for a period despite insulin resistance.

Key results

- Fasting glucose and glucose-tolerance-test glucose values were similar in Crohn’s patients and controls.
- Insulin levels, calculated beta-cell activity, and insulin resistance were significantly higher in the Crohn’s group.
- The authors concluded that increased insulin secretion could compensate for insulin resistance in the setting of chronic inflammatory disease.

Interpretation for this slide: This provides a useful clinical example of compensation: tissues can be insulin resistant while fasting glucose remains normal because the pancreas is producing more insulin. It fits the slide’s message that rising insulin demand can precede abnormal fasting glucose or A1C.

Important limitation: This was a small Crohn’s-disease study and does not establish that inflammation was the only cause of insulin resistance. It is best used as a physiologic example rather than proof of the video’s full thesis.

Inflammation and insulin resistance

Pradhan AD, Manson JE, Rifai N, Buring JE, Ridker PM. C-reactive protein, interleukin 6, and risk of developing type 2 diabetes mellitus. JAMA. 2001;286(3):327–334.
DOI: 10.1001/jama.286.3.327

Evidence type: Prospective nested case-control study within the Women’s Health Study.

Study basics

- 27,628 women were free of diagnosed diabetes, cardiovascular disease, and cancer at baseline.
- 188 women who developed type 2 diabetes during approximately four years of follow-up were compared with 362 matched controls.
- Baseline CRP and IL-6 were measured before diabetes developed.

Key results

- Baseline CRP and IL-6 were significantly higher in women who later developed diabetes.
- Unadjusted highest-versus-lowest quartile relative risks were 15.7 for CRP and 7.5 for IL-6.
- After adjustment for BMI, family history, smoking, exercise, alcohol, and hormone therapy, CRP remained strongly predictive (RR 4.2); the adjusted IL-6 estimate was 2.3.

Interpretation for this slide: The important point for this slide is timing: inflammatory markers were elevated before the diabetes diagnosis. That supports the possibility that inflammation can participate upstream in the development of dysregulated glucose control rather than being only a consequence of established diabetes.

Important limitation: This is prospective observational evidence. It shows prediction and temporality, not proof that CRP or IL-6 caused the diabetes. The 7.5- and 15.7-fold figures shown in the deck are the unadjusted estimates.

Inflammation and insulin resistance

Spranger J, Kroke A, Möhlig M, Hoffmann K, Bergmann MM, Ristow M, Boeing H, Pfeiffer AFH. Inflammatory cytokines and the risk to develop type 2 diabetes: results of the prospective population-based EPIC-Potsdam Study. Diabetes. 2003;52(3):812–817.

DOI: 10.2337/diabetes.52.3.812

Evidence type: Prospective nested case-control study within the EPIC-Potsdam cohort.

Study basics

- The underlying cohort included 27,548 adults initially free of diabetes.
- 192 people who later developed type 2 diabetes were compared with 384 controls.
- IL-6, TNF-alpha, and IL-1beta-related inflammatory patterns were assessed before diagnosis.

Key results

- IL-6 was higher in people who later developed type 2 diabetes.
- After multivariable adjustment, the highest versus lowest IL-6 quartile was associated with roughly 2.6-fold higher odds of future diabetes.
- The combination of detectable IL-1beta and high IL-6 was associated with about 3.3-fold higher odds.
- TNF-alpha was less independent of adiposity after adjustment.

Interpretation for this slide: Placed immediately after Pradhan, this study shows that the “inflammation before diabetes” finding was not confined to one cohort. Together, the two prospective studies strengthen the case that inflammatory signalling can precede dysglycaemia.

Important limitation: This remains observational evidence. It supports an upstream role for inflammation but does not identify the source of the inflammation and does not show that food reactions caused it.

Gut inflammation can add to the load

Mkumbuzi L, Mfengu MMO, Engwa GA, Sewani-Rusike CR. Insulin Resistance is Associated with Gut Permeability Without the Direct Influence of Obesity in Young Adults. Diabetes Metab Syndr Obes. 2020;13:2997–3008.

DOI: 10.2147/DMSO.S256864

Evidence type: Cross-sectional human study.

Study basics

- 151 young South African adults were evaluated.
- Zonulin was used as a circulating marker of gut permeability.
- Fasting insulin and glucose were used to calculate HOMA-IR, insulin sensitivity, and beta-cell function; measures of adiposity and inflammatory adipokines were also assessed.

Key results

- Zonulin was positively associated with BMI and visceral fat, but also with HOMA-IR and insulin concentration.
- In this cohort, insulin-resistance markers were not significantly associated with obesity itself.
- The authors concluded that insulin resistance was associated with gut permeability without a direct influence of obesity in these young adults.

Interpretation for this slide: This study supports the slide's narrower idea that gut-barrier/permeability biology can track with insulin resistance and may represent an inflammatory input that is not captured simply by body weight.

Important limitation: Cross-sectional data cannot establish direction of causation. Zonulin is a surrogate marker of permeability, and the study does not prove that gut permeability caused insulin resistance or that food reactions were responsible.

Food can drive the inflammatory signals

Tetzlaff WF, Meroño T, Menafra M, et al. Markers of inflammation and cardiovascular disease in recently diagnosed celiac disease patients. World J Cardiol. 2017;9(5):448–456.

DOI: 10.4330/wjc.v9.i5.448

Evidence type: Small cross-sectional human study in newly diagnosed celiac disease.

Study basics

- 20 untreated adults with newly diagnosed celiac disease were compared with 20 healthy controls matched for age, sex, and body size.
- Inflammatory markers, fasting glucose and insulin, HOMA-IR, QUICKI, and lipid measures were assessed.
- Celiac disease provides a defined example in which a food protein (gluten) drives an immune-mediated disease.

Key results

- The celiac group had higher inflammatory markers, including hsCRP.
- Fasting insulin was higher (about 7.2 vs 4.6 mU/L) and HOMA-IR was higher (about 1.45 vs 1.00).
- QUICKI insulin sensitivity was lower (about 0.33 vs 0.42).
- Fasting glucose was essentially the same between groups, illustrating metabolic compensation despite greater insulin resistance.

Interpretation for this slide: This is the deck's clearest condition-specific human example linking a known food-triggered immune disease with greater inflammation and poorer insulin sensitivity. It supports biological plausibility that food-driven immune activity can influence glucose regulation without the food itself directly raising glucose.

Important limitation: Celiac disease is a specific autoimmune condition and cannot be generalized to ordinary food sensitivity. The sample was small and cross-sectional, so the study shows association rather than causation.

Autoimmunity and insulin resistance

Winer DA, Winer S, Shen L, et al. B cells promote insulin resistance through modulation of T cells and production of pathogenic IgG antibodies. Nat Med. 2011;17(5):610–617.

DOI: 10.1038/nm.2353

Evidence type: Combined human association and animal mechanistic study.

Study basics

- Human component: 32 overweight/obese adults of similar body size were compared according to insulin sensitivity.
- The insulin-resistant group showed a distinct IgG autoantibody pattern against self-proteins; the strongest identified signal included antibodies against GOSR1.
- Mouse component: B-cell-deficient obese mice, B-cell/IgG transfer experiments, and B-cell depletion with anti-CD20 were used to test causality.

Key results

- Human insulin resistance was associated with a unique profile of IgG autoantibodies despite similar obesity.
- Diet-induced obese mice lacking B cells were protected from insulin resistance despite weight gain.
- Transfer of B cells or IgG from obese insulin-resistant mice worsened glucose metabolism in recipient mice.
- B-cell depletion improved glucose abnormalities and reduced inflammatory immune activity.

Interpretation for this slide: These two slides deliberately use the same paper for two different levels of evidence. The human data show an immune signature associated with insulin resistance independent of simple body size; the animal experiments go further by showing that changing B cells and IgG can change insulin resistance. Together they support adaptive immune involvement as more than a passive marker.

Important limitation: The human antibodies were autoantibodies against the body's own proteins, not food antibodies. The animal work supports mechanism but does not prove that food-triggered immunity causes insulin resistance in humans.

Treating inflammation dropped A1C

Larsen CM, Faulenbach M, Vaag A, et al. Interleukin-1-Receptor Antagonist in Type 2 Diabetes Mellitus. N Engl J Med. 2007;356:1517–1526.

DOI: 10.1056/NEJMoa065213

Evidence type: Double-blind randomized placebo-controlled human trial.

Study basics

- 70 adults with type 2 diabetes were randomized to anakinra (an IL-1 receptor antagonist) or placebo for 13 weeks.
- The study assessed HbA1c, beta-cell secretory function, insulin sensitivity, and inflammatory markers.

Key results

- At 13 weeks, HbA1c was 0.46 percentage points lower in the anakinra group than in placebo after adjustment.
- Beta-cell secretory function improved and the proinsulin-to-insulin ratio improved.
- CRP and IL-6 fell with treatment.
- Insulin sensitivity did not significantly differ between groups, suggesting much of the glycaemic benefit was related to beta-cell function.

Interpretation for this slide: This is direct human intervention evidence: changing one inflammatory pathway changed glucose control and beta-cell function. It supports the deck's claim that inflammatory signalling can be biologically active in type 2 diabetes rather than simply accompanying it.

Important limitation: This does not mean IL-1 blockade is a general diabetes treatment or that inflammation is the only driver. The most accurate published result is the 0.46 percentage-point between-group HbA1c difference.

Presentation note: The slide translates that effect into a simple 8.0% → 7.54% example for viewers; that example is illustrative, not the literal mean baseline-to-endpoint trajectory reported for the anakinra group.

Treating inflammation dropped A1C

Goldfine AB, Fonseca V, Jablonski KA, et al. Salicylate (Salsalate) in Patients With Type 2 Diabetes: A Randomized Trial. Ann Intern Med. 2013;159(1):1–12.

DOI: 10.7326/0003-4819-159-1-201307020-00003

Evidence type: Randomized, double-blind, placebo-controlled human trial.

Study basics

- 286 adults with type 2 diabetes were randomized to salsalate 3.5 g/day or placebo for 48 weeks; 283 were included in analysis.
- Salsalate is an anti-inflammatory salicylate and was used here to test whether dampening inflammatory signalling could improve glycaemia.

Key results

- Mean HbA1c over 48 weeks was about 0.37 percentage points lower with salsalate than placebo.
- Fasting glucose and triglycerides were lower, and several inflammatory blood-cell measures decreased.
- Weight and LDL cholesterol increased, and albuminuria increased during treatment but reversed after drug withdrawal.

Interpretation for this slide: This second randomized human trial reinforces the previous slide: a different anti-inflammatory strategy also improved glucose control. The pair makes the argument stronger than relying on a single drug or inflammatory pathway.

Important limitation: The study also shows why the conclusion should stay narrow: anti-inflammatory treatment improved glycaemia, but it had mixed metabolic and safety effects. It is not evidence that all anti-inflammatory treatments are beneficial or appropriate.

Presentation note: As with the anakinra slide, the published result is best stated as the between-group HbA1c difference. The deck's 7.87% → 7.51% example is a viewer-friendly translation rather than a literal individual trial trajectory.

Immune food reactions and Type 1 Diabetes

Karjalainen J, Martin JM, Knip M, et al. A Bovine Albumin Peptide as a Possible Trigger of Insulin-Dependent Diabetes Mellitus. N Engl J Med. 1992;327(5):302–307.

DOI: 10.1056/NEJM199207303270502

Evidence type: Human observational immunology study.

Study basics

- 142 children with newly diagnosed type 1 diabetes were compared with healthy children and adult blood donors.
- Researchers measured antibodies to bovine serum albumin (BSA), especially the ABBOS peptide fragment.
- They also tested whether those antibodies could recognize a pancreatic beta-cell surface protein.

Key results

- Children with new type 1 diabetes had markedly elevated anti-BSA/ABBOS antibodies compared with controls.
- The ABBOS-reactive antibodies also recognized a beta-cell surface protein (p69), providing a molecular-mimicry/cross-reactivity mechanism.

Interpretation for this slide: The slide uses this study to show that a food-protein-directed immune response can, at least in a defined autoimmune setting, cross-react with proteins associated with insulin-producing cells. That makes food-protein molecular mimicry biologically plausible.

Important limitation: The study does not prove that cow's milk causes type 1 diabetes. It is an observational cross-reactivity study, and later prevention research has not established cow's-milk avoidance as a proven strategy for preventing type 1 diabetes.

Immune food reactions and Type 1 Diabetes

Pastore MR, Bazzigaluppi E, Belloni C, Arcovio C, Bonifacio E, Bosi E. Six months of gluten-free diet do not influence autoantibody titers, but improve insulin secretion in subjects at high risk for type 1 diabetes. J Clin Endocrinol Metab. 2003;88(1):162–165.

DOI: 10.1210/jc.2002-021177

Evidence type: Small human dietary intervention in people at high risk for type 1 diabetes.

Study basics

- 17 first-degree relatives with at least two islet autoantibodies entered the study.
- Participants followed a gluten-free diet for six months, followed by six months of a normal gluten-containing diet.
- The main measures were islet-autoantibody levels and acute insulin response to intravenous glucose.

Key results

- Autoantibody levels did not significantly change.
- After six months gluten-free, acute insulin response improved in 12 of 14 evaluable participants (P=0.04).
- After gluten was reintroduced, insulin response fell in 10 of 13 participants (P=0.07).
- Insulin sensitivity also moved in a favourable direction during gluten avoidance and worsened after reintroduction, but the initial improvement was not statistically robust.

Interpretation for this slide: This study is used as a human proof-of-concept that changing exposure to a food protein can alter pancreatic insulin-response physiology in a highly selected immune-risk population, even when autoantibody levels themselves do not change.

Important limitation: The study was very small, nonblinded, sequential rather than parallel-controlled, and involved people already at high autoimmune risk. It is hypothesis-generating and should not be generalized to type 2 diabetes or to routine gluten avoidance.

Changing inflammation can change the outcome

Ridker PM, Everett BM, Thuren T, et al.; CANTOS Trial Group. Antiinflammatory Therapy with Canakinumab for Atherosclerotic Disease. N Engl J Med. 2017;377:1119–1131.

DOI: 10.1056/NEJMoa1707914

Evidence type: Large randomized placebo-controlled cardiovascular outcomes trial.

Study basics

- 10,061 people with a previous myocardial infarction and persistent inflammation (hsCRP ≥2 mg/L) were randomized to placebo or one of three canakinumab doses.
- Canakinumab specifically blocks IL-1beta and reduces downstream inflammatory signalling.
- The trial tested cardiovascular outcomes rather than glucose lowering.

Key results

- The 150-mg dose reduced the primary cardiovascular event rate by about 15% versus placebo (hazard ratio 0.85).
- CRP and IL-6 fell substantially.
- LDL cholesterol was not lowered, showing that the cardiovascular benefit occurred through a pathway distinct from lipid lowering.
- Fatal infections were more frequent with canakinumab, and all-cause mortality was not reduced.

Interpretation for this slide: Placed after the “What if diabetes isn’t causing everything else?” slide, CANTOS supports the common-soil concept: changing an inflammatory cytokine pathway changed cardiovascular outcomes without changing LDL. This makes it plausible that inflammation can be an upstream contributor to more than one metabolic/vascular outcome rather than every condition simply being downstream from diabetes.

Important limitation: CANTOS was not a diabetes-treatment trial and does not identify food as the inflammatory trigger. It supports the broader causal importance of inflammation in vascular disease, not the specific food-reaction hypothesis.

Presentation note: The left side of this slide also briefly revisits the Larsen anakinra trial from slide 19 to connect glucose and cardiovascular outcomes through inflammatory pathways.

Changing inflammation can change the outcome

Huang Y, Lin C, Cheng N, et al. Effect of a Novel Macrophage-Regulating Drug on Wound Healing in Patients With Diabetic Foot Ulcers: A Randomized Clinical Trial. JAMA Netw Open. 2021;4(9):e2122607.

DOI: 10.1001/jamanetworkopen.2021.22607

Evidence type: Multicenter phase 3 randomized clinical trial.

Study basics

- 236 adults with Wagner grade 1 or 2 diabetic foot ulcers were randomized to topical ON101 cream or a standard absorbent dressing.
- Treatment continued for up to 16 weeks, with complete wound healing as the primary outcome.
- ON101 was developed to alter dysregulated macrophage behaviour in the diabetic wound environment.

Key results

- Complete healing occurred in 60.7% of the ON101 group versus 35.1% of the comparator group.
- The odds ratio for complete healing was 2.84 (95% CI 1.66–4.84; P<0.001).
- The benefit was observed despite persistent diabetes, demonstrating that changing local inflammatory biology could change the wound outcome.

Interpretation for this slide: This study supports the slide's key point that inflammation is not merely a laboratory marker. In a diabetes-related complication, changing the inflammatory wound environment substantially changed healing. It fits the idea that inflammatory pathways can contribute directly to parallel disease outcomes.

Important limitation: This was a topical wound-healing trial. It does not show that systemic inflammation caused the ulcers, and it does not show that food reactions were the source of inflammation.

Weight-loss helps, but isn't the whole story

Wu H, Yang A, Lau ESH, et al. 1-year weight change after diabetes diagnosis and long-term incidence and sustainability of remission of type 2 diabetes in real-world settings in Hong Kong: An observational cohort study. PLoS Med. 2024;21(1):e1004327.

DOI: 10.1371/journal.pmed.1004327

Evidence type: Large population-based observational cohort study.

Study basics

- 37,326 people with newly diagnosed type 2 diabetes were followed in routine care in Hong Kong.
- Median follow-up was 7.9 years.
- The study examined how weight change in the first year after diagnosis related to later diabetes remission and relapse.

Key results

- Only 6.1% achieved remission during follow-up.
- Among those who achieved remission, 67.2% later returned to hyperglycaemia over a median of 3.1 years.

- Greater weight loss clearly increased the chance of remission: ≥10% weight loss was associated with more than threefold higher remission probability compared with weight gain.
- Greater weight loss also reduced the risk of returning to hyperglycaemia.

Interpretation for this slide: This slide is intentionally not anti-weight-loss. The study shows that weight loss materially improves the odds of remission, but it also shows that body weight alone does not guarantee durable glucose normalization in real-world care. That supports the video's broader message that the regulatory biology still matters.

Important limitation: This was observational real-world data, not a randomized weight-loss trial. It would be incorrect to use it to claim that weight loss is ineffective; the study actually shows a strong dose-response relationship between greater weight loss and better remission outcomes.

Blood sugar can improve without weight loss

Sandforth A, Arreola EV, Hanson RL, et al. Prevention of type 2 diabetes through prediabetes remission without weight loss. Nat Med. 2025;31:3330–3340.

DOI: 10.1038/s41591-025-03944-9

Evidence type: Post hoc analysis of a multicenter randomized lifestyle-intervention study, replicated in the US Diabetes Prevention Program.

Study basics

- Researchers specifically examined people with prediabetes who did not lose weight (or gained weight) during lifestyle intervention.
- Responders who returned to normal glucose regulation were compared with nonresponders despite similar lack of weight loss.
- The findings were reproduced in an independent DPP dataset.

Key results

- Prediabetes remission without weight loss was accompanied by improved insulin sensitivity and beta-cell function.
- Responders had more favourable fat distribution: nonresponders gained more visceral fat, whereas responders had relatively more subcutaneous fat deposition.
- Future type 2 diabetes risk was 71% lower in the PLIS analysis and 73% lower in the DPP replication among those who achieved remission without weight loss.

Interpretation for this slide: This is strong evidence for the slide's central point: glucose regulation can improve substantially without scale weight falling. It supports shifting attention from weight alone toward insulin sensitivity, beta-cell function, and where fat is stored.

Important limitation: This study does not directly support inflammation as the mechanism. Circulating inflammatory-marker trajectories did not clearly distinguish responders from nonresponders. It supports the regulation-over-weight concept, while the inflammation mechanism must be supported by the other studies in the deck.

The 2025 IBS trial — individual reactivity studied

Singh P, Chey WD, Takakura W, et al. A Novel, IBS-Specific IgG ELISA-Based Elimination Diet in Irritable Bowel Syndrome: A Randomized, Sham-Controlled Trial. Gastroenterology. 2025;168(6):1128–1136.e4.

DOI: 10.1053/j.gastro.2025.01.223

Evidence type: Multicenter randomized, double-blind, sham-controlled dietary trial.

Study basics

- 238 adults with IBS were randomized at eight centers; 223 were included in the modified intention-to-treat analysis.
- Participants had reactivity to at least one food on a novel 18-food IBS-specific IgG assay.
- The experimental group followed an antibody-guided elimination diet for eight weeks; the control group followed a matched sham diet.

Key results

- The primary abdominal-pain response occurred in 59.6% of the guided group versus 42.1% of the sham group (P=0.02).
- The effect was larger in constipation-predominant IBS: 67.1% versus 35.8%.
- In mixed-bowel-habit IBS, response was 66.0% versus 29.5%.
- The study demonstrated that individualized IgG-guided dietary selection could outperform a sham dietary strategy under blinded randomized conditions.

Interpretation for this slide: This is the deck's strongest controlled evidence for the narrower concept of individualized food reactivity: a specific antibody-guided selection method produced different clinical outcomes from a matched sham approach. It supports the idea that individualized immune-related food selection can be tested rigorously rather than being assumed from generic "anti-inflammatory diet" lists.

Important limitation: This was an IBS trial, not a diabetes trial. It does not prove that food-specific IgG causes insulin resistance, does not validate every commercial IgG panel, and does not establish IgG-guided elimination as a diabetes treatment.

Overall evidence summary

What is directly supported: Prospective human studies show inflammatory markers can precede type 2 diabetes; mechanistic animal studies show B cells/IgG can alter insulin resistance; randomized human trials show that modifying inflammatory pathways can improve glycaemia or other diabetes-related outcomes.

What is biologically plausible but less direct: Gut permeability, celiac disease, type 1 food-protein cross-reactivity, and the IBS antibody-guided diet trial show several ways in which immune and food-related biology can interact with metabolic or inflammatory physiology. These studies support plausibility and coherence, but they are not direct proof that food reactions are a primary cause of type 2 diabetes.

What the weight studies add: Weight loss clearly improves the probability of remission, but durable remission is not guaranteed; conversely, some people can normalize glucose regulation without losing weight. Together these studies support focusing on regulation and physiology rather than using body weight as the only explanation.

The synthesis used in the video: The combined evidence supports chronic inflammation as one contributor to dysregulated glucose control. In a susceptible individual, a persistent inflammatory food reaction is biologically plausible as one possible source of that inflammatory load. No single study in this document proves that full pathway from food protein → immune response → chronic inflammation → type 2 diabetes; the video's thesis is a synthesis of prospective, mechanistic, interventional, and condition-specific evidence.