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Prediabetes is pre-nothing: Call it early type 2 diabetes

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Prediabetes is often a footnote in primary care, flagged inconsistently on lab reports, mentioned briefly or ignored for 'watchful waiting'. This passive approach fails to reflect the biological reality. Long before HbA1c reaches the diagnostic threshold for type 2 diabetes (T2D) (≥ 48 mmol/mol or ≥ 6.5%), the disease process is active: insulin resistance, hyperinsulinaemia and β-cell stress drive early microvascular and cardiovascular damage.[1,2]

Prediabetes (HbA1c 39–46 mmol/mol or 5.7% – 6.4%) is not benign; it is early T2D, and primary care must treat it as such.

Mechanistically, insulin resistance in muscle, liver and adipose tissue forces β-cells to overproduce insulin, leading to hyperinsulinemia, a hallmark of early metabolic dysfunction.[3] This initially masks rising glucose but stresses β-cells, causing defects mirroring T2D. Hyperinsulinaemia often precedes glucose abnormalities by up to 24 years, affecting up to 75% of those with normal glucose tolerance.[4] These early defects matter: 30% – 50% of individuals with prediabetes progress to T2D within 5–10 years.[2]

The consequences extend well beyond glycaemia. Prediabetes increases all-cause mortality and cardiovascular risk by 13% – 20%.[5] Endothelial dysfunction, a driver of atherosclerosis, is evident early, with insulin resistance and hyperinsulinaemia promoting vascular damage.[6,7] Retinal damage already affects ~10%,[8] neuropathy up to 20%,[9] kidney disease risk is doubled[10] and non-alcoholic fatty liver disease (NAFLD) occurs in up to 70% of cases.[6,11] Emerging evidence also links prediabetes to an increased risk of dementia and stroke.[12] In short, complications attributed to diabetes often begin during so-called 'prediabetes'.

The good news is that prediabetes is highly reversible. Therapeutic carbohydrate reduction (TCR) not only prevents progression but has also been shown to restore normal glucose regulation. Therapeutic carbohydrate reduction reduces insulin demand, improves β-cell function and normalises glucose levels, often within weeks, typically without medication.[13] Studies collectively show 77% – 97% of patients with prediabetes can return to normal HbA1c and dramatically improve metabolic health.[14,15,16,17] Early intervention not only prevents T2D but also reduces the risk of its comorbidities, with the potential to save healthcare systems billions.

Yet this opportunity is routinely wasted. Patients newly diagnosed with T2D often have years of documented prediabetes in their records that were never discussed. When this is understood, patients' frustration is justified: why wasn't action recommended when intervention would have been most effective? Failing to act on prediabetes is not just a missed opportunity; it is an ethical failure. Part of this inaction reflects institutional resistance. Reclassifying prediabetes as early T2D expands treatment populations, triggering concerns about medicalisation and resource allocation. Funders baulk at covering interventions for 'pre-disease'. But this short-term thinking ignores the economics: treating early T2D with lifestyle intervention costs substantially less than managing late-stage complications. The question isn't whether to treat, but when treatment is most effective and least expensive.

The term itself sabotages action. 'Prediabetes' signals a waiting room, not a treatment window. Primary care must reframe this as early T2D, establish concrete targets (HbA1c < 39 mmol/mol or 5.7%) and provide or refer for tailored dietary and lifestyle intervention.

Language determines response. 'Prediabetes' encourages surveillance; 'early T2D' demands treatment. Retiring the term 'prediabetes' aligns clinical language with pathophysiology and ensures intervention begins when it can prevent, rather than merely delay, irreversible complications.

References

1. American Diabetes Association. Standards of medical care in diabetes – 2023. Diabetes Care. 2023;46(Suppl. 1):S1–291. https://doi.org/10.2337/dc23-SINT
2. Tabák AG, Herder C, Rathmann W, Brunner EJ, Kivimäki M. Prediabetes: A high-risk state for diabetes development. Lancet. 2012;379(9833):2279–2290. https://doi.org/10.1016/S0140-6736(12)60283-9
3. Abdul-Ghani MA, DeFronzo RA. Pathophysiology of prediabetes. Curr Diab Rep. 2006;6(3):193–198. https://doi.org/10.1007/s11892-009-0032-7
4. Crofts C, Zinn C, Wheldon M, Kraft JR. Identifying hyperinsulinaemia in the absence of impaired glucose tolerance: An examination of the Kraft database. Diabetes Res Clin Pract. 2016;118:81–88. https://doi.org/10.1016/j.diabres.2016.06.007
5. Cai X, Zhang Y, Li M, et al. Association between prediabetes and risk of all-cause mortality and cardiovascular disease: Updated meta-analysis. BMJ. 2020;370:m2297. https://doi.org/10.1136/bmj.m2297
6. Crofts C, Zinn C, Wheldon M, Schofield G. Hyperinsulinemia: A unifying theory of chronic disease? Diabesity. 2015;1(4):102–111. https://doi.org/10.15562/diabesity.2015.19
7. Gupta AK, Ravussin E, Johannsen DL, Stull AJ, Cefalu WT, Johnson WD. Endothelial dysfunction: An early cardiovascular risk marker in asymptomatic obese individuals with prediabetes. Br J Med Med Res. 2012;2(3):413–423. https://doi.org/10.9734/bjmmr/2012/1479
8. Kirthi V, Nderitu P, Alam U, Evans JR, Sivaprasad S. The prevalence of retinopathy in prediabetes: A systematic review. Surv Ophthalmol. 2022;67(4):1332–1345. https://doi.org/10.1016/j.survophthal.2022.04.002
9. Ziegler D, Rathmann W, Dickhaus T, Meisinger C, Mielck A, KORA Study Group. Prevalence of polyneuropathy in pre-diabetes and diabetes is associated with abdominal obesity and macroangiopathy: The MONICA/KORA Augsburg Surveys S2 and S3. Diabetes Care. 2008;31(3):464–469. https://doi.org/10.2337/dc07-1796
10. Echouffo-Tcheugui JB, Narayan KM, Weisman D, Golden SH, Jaar BG. Association between prediabetes and risk of chronic kidney disease: A systematic review and meta-analysis. Diabetes Med. 2016;33(12):1615–1624. https://doi.org/10.1111/dme.13113
11. Younossi ZM, Koenig AB, Abdelatif D, Fazel Y, Henry L, Wymer M. Global epidemiology of nonalcoholic fatty liver disease – Meta-analytic assessment of prevalence, incidence, and outcomes. Hepatology. 2016;64(1):73–84. https://doi.org/10.1002/hep.28431
12. Han S, Naderi E, Wang K, Ma Y, Biessels GJ, Ahmadizar F. Pre-diabetes as a critical stage for risk of dementia and stroke: Evidence from the UK Biobank and Mendelian randomization [preprint]. *medRxiv*. 2025. https://doi.org/10.1101/2025.09.30.25336879
13. Hallberg SJ, McKenzie AL, Williams PF, et al. Effectiveness and safety of a novel care model for the management of type 2 diabetes at 1 year: An open-label, non-randomized, controlled study. Diabetes Ther. 2018;9(2):583–612. https://doi.org/10.1007/s13300-018-0373-9
14. Hawkins M, Zinn C, Delon C. The application of carbohydrate-reduction in general practice: A medical audit. J Metab Health. 2023;6(1):1–11. https://doi.org/10.4102/jmh.v6i1.86
15. Glandt M, Ailon NY, Berger S, Unwin D. Use of a very low carbohydrate diet for prediabetes and type 2 diabetes: An audit. J Metab Health. 2024;7(1):a87. https://doi.org/10.4102/jmh.v7i1.87
16. Unwin D, Khalid AA, Unwin J, et al. Insights from a general practice service evaluation supporting a lower carbohydrate diet in patients with type 2 diabetes mellitus and prediabetes: A secondary analysis of routine clinic data including HbA1c, weight and prescribing over 6 years. BMJ Nutr Prev Health. 2021;3(2):285–294. https://doi.org/10.1136/bmjnph-2020-000072
17. Stentz FB, Brewer A, Wan J, et al. Remission of pre-diabetes to normal glucose tolerance in obese adults with high protein versus high carbohydrate diet: Randomized control trial. BMJ Open Diabetes Res Care. 2016;4(1):e000258. https://doi.org/10.1136/bmjdrc-2016-000258