



Harnessing dysglycaemia heterogeneity for precision type 2 diabetes prevention

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Purpose of review

Type 2 diabetes (T2D) is a heterogeneous, progressive metabolic disease and a major contributor to cardiovascular disease worldwide. Current prevention strategies, centred on population-level lifestyle recommendations, have had limited success, highlighting the need for more comprehensive approaches that account for interindividual variation in intervention response.

Recent findings

Although discrete T2D subtypes using genomics or clinical variables have been proposed for targeted diabetes prevention and care strategies, emerging evidence supports a more dynamic view of heterogeneity as a continuous spectrum of metabolic dysfunction. Continuous glucose monitoring and multiomics profiling have shown promises in detecting early metabolic dysfunction in individuals with normal glycemia.

Summary

In this review, we synthesize advances in modelling T2D phenotypic continuum and highlight how emerging technologies, including continuous glucose monitoring and multiomics profiling, can detect early metabolic dysregulation. We also discuss methodological challenges, such as the need for standardized deep phenotyping, robust analytic frameworks, and inclusion of diverse populations to ensure equity in translation. Finally, we outline future directions for translating these insights into scalable, mechanism-informed interventions that address glycaemic progression from subclinical changes to more advanced forms of the disease.

Keywords

continuous glucose monitoring, genomics, multiomics, precision prevention, type 2 diabetes continuum

INTRODUCTION

Diabetes, a chronic disease defined by progressive glycaemic decline, doubles the risk of cardiovascular complications, and it is the major contributor to year lost due to disability or early death [1]. Globally, 1 in 10 individuals suffers from diabetes, predominantly type 2 diabetes (T2D), projected to impact 1.31 billion people by 2050 [2]. Despite clinical guidelines emphasizing healthy diet, physical activity, and weight management, the global prevalence of T2D continues to rise, underscoring the limitations of current ‘one-size-fits-all’ approaches and the need to identify more effective prevention strategies.

A major challenge in T2D prevention is its heterogeneity. T2D arises from diverse, overlapping disrupted physiological processes, as evidenced by the large number of genes associated with T2D, interacting with behavioural, environmental, and social determinants of health [3,4]. The large number of potential variations in these interactions can be compared to a painter’s palette (the infamous ‘McCarthy palette’) [5], where hues and tones are

mixed, leading to individual differences in clinical presentation, varying treatment responses, and differing risks for complications [6,7], limiting the effectiveness of current strategies. Understanding and harnessing this heterogeneity is a main objective of precision diabetes medicine [8].

Early efforts to subtype T2D into discrete categories have provided valuable insights. These studies have, for example, revealed that individuals with impaired insulin sensitivity due to alterations in adipose tissue function are more likely to progress to cardiovascular complications, while those with

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KEY POINTS

- T2D heterogeneity exists as a continuous spectrum rather than discrete subtypes. Phenotypic continuum models that track metabolic dysfunction gradients demonstrate superior prediction of treatment responses and complications compared to traditional discrete clustering approaches, better reflecting the progressive nature of glucose dysregulation.
- CGM reveals hidden metabolic dysfunction in apparently healthy individuals. Up to 39% of individuals with severe glucose variability detected by CGM would not be diagnosed with prediabetes or diabetes using traditional tests (fasting glucose, OGTT, or HbA1c), highlighting the limitations of static measurements in capturing early metabolic dysfunction.
- Multiomics approaches identify early molecular changes and intervention targets. Metabolomics detects nitrogen metabolism disturbances early in disease progression; proteomics reveals immune alterations 1 year before clinical transition; and microbiome profiling enables prediction of personalized dietary responses and identifies modifiable therapeutic targets.
- Integration of CGM with omics data enables precision prevention strategies. Combining continuous glucose dynamics with molecular signatures transforms heterogeneity from an obstacle into a roadmap for personalized intervention, enabling mechanism-informed strategies that simultaneously address glycaemic progression, lipid profiles, and cardiovascular risk aligned with individual biology.

predominant insulin resistance due to liver/lipid fat alterations were more likely to develop renal dysfunction [9]. However, these frameworks may oversimplify the dynamic nature of dysglycaemia, failing to capture its continuous variation, temporal fluctuations, and overlapping pathophysiological processes. Recent evidence suggests that conceptualizing T2D as a continuum spectrum offers a more granular understanding of disease progression at individual level [10].

Technological advances are making this perspective increasingly actionable. Continuous glucose monitoring (CGM) and multiomics platforms are enabling high-resolution characterization of metabolic and molecular phenotypes under real-world conditions. These tools can reveal subtle glycaemic deviations years before clinical onset by providing phenotypically and biologically grounded insights into disrupted mechanisms.

In this review, we synthesize progress in modeling diabetes heterogeneity, examine how emerging technologies, including CGM, multiomics, and artificial intelligence-driven analytics, can uncover early

metabolic dysfunction, and outline opportunities for translating these insights into precision prevention strategies. We also discuss key methodological and implementation challenges, including the need for standardized deep phenotyping, rigorous analytic frameworks, and equitable inclusion of diverse populations. Finally, we discuss opportunities for translating this growing knowledge base into scalable interventions that address both glycaemic progression and cardiovascular risk, bridging the gap between conceptual advances in diabetes heterogeneity and their clinical application.

UNDERSTANDING T2D HETEROGENEITY

A central question when approaching T2D heterogeneity is which features best define subgroups of individuals who may benefit most from targeted interventions. Early efforts have largely relied on clinical characteristics at or near T2D diagnosis to classify individuals into discrete clusters. One widely cited approach used six clinical measures – age at diagnosis, BMI, HbA1c, HOMA2-B, HOMA2-IR, and GADA antibodies – to define five clusters of individuals with different clinical presentation: severe autoimmune diabetes, severe insulin-deficient diabetes, severe insulin-resistant diabetes, mild obesity-related diabetes, and mild age-related diabetes [11]. These clusters have been replicated across multiple cohorts and consistently associated with differential risks of microvascular and macrovascular complications.

However, cluster-based clinical models assume stability in subgroup membership, whereas in reality, an individual's metabolic profile can shift over time. This static framework limits the ability to capture dynamic disease trajectories. Genetic studies have attempted to refine these approaches by grouping variants into 'partitioned' polygenic scores (pPSs) that reflect distinct biological axes, such as beta-cell dysfunction, adverse fat distribution, or liver-mediated insulin resistance [9]. While these scores illuminate causal mechanisms – linking, for example, beta-cell dysfunction to stroke risk or adiposity-driven risk to coronary artery disease – they have shown limited clinical utility. Dietary interventions tailored to genetic endophenotypes have yielded null results, likely because of overlapping biological processes and the difficulty of accurately categorizing individuals into single subgroups [12,13].

These limitations have motivated a paradigm shift from discrete subtypes toward continuum-based models. Rather than defining T2D as a set of mutually exclusive endotypes, which does not recapitulate T2D pathophysiology, continuum models conceptualize metabolic dysfunction as a graded spectrum, reflecting variable contributions of multiple overlapping

processes. Innovative methods have begun to operationalize this perspective.

In 2022, Nair *et al.* [14] applied reverse graph embedding to nine clinical variables from 23 137 individuals in Scotland with recent-onset T2D to generate a tree-like structure that mapped phenotypic variation at diagnosis. The study found that distinct phenotypic trajectories along the resulting ‘diabetes continuum’ are associated with divergent risks of insulin requirement, chronic kidney disease, retinopathy, and cardiovascular events. They further validated these findings in the UK Biobank and the ADOPT clinical trial, showing that different phenotypes respond differently to monotherapy drugs. The study underscores that underlying phenotypic variation influences both disease onset and long-term outcomes.

Dynamic phenotyping studies further reinforce the utility of this approach. In the German Diabetes Study, intensive metabolic profiling revealed gradients of insulin sensitivity and secretion that mapped to distinct clinical liabilities: low insulin sensitivity correlated with hepatic steatosis, systemic inflammation, and cardiovascular risk, while impaired insulin secretion predicted earlier insulin dependence and neuropathy [15²²]. Longitudinal follow-up showed that insulin secretion declined more rapidly than insulin sensitivity, with about 20% of participants shifting position along the continuum over 5 years. Continuum frameworks have since been replicated in ethnically diverse cohorts of individuals with T2D [16] and in individuals with T1D [17], demonstrating their generalizability. However, none of these studies have addressed whether this approach can be used for targeted T2D preventive strategies and the extent to which is superior than current population-wide applications.

CONTINUOUS GLUCOSE MONITORING AND THE PHENOTYPIC CONTINUUM

Since its introduction in 2000, CGM has transformed diabetes care by enabling real-time assessment of interstitial glucose levels. Unlike single time-point or average-based measures, CGM captures the full temporal dynamics of glycemia, revealing fluctuations, excursions, and variability that traditional metrics fail to capture. CGM-derived measures such as time-in-range, glycaemic variability, and nocturnal nadirs are now integral to therapeutic decision-making [18].

Importantly, CGM has shown that substantial glycaemic variability exists even in individuals without diagnosed diabetes. In a 2018 study, Hall and colleagues applied clustering algorithms to CGM traces from 57 adults without diabetes, identifying three ‘glucotypes’ of low, moderate, and severe

glycaemic variability [19]. Strikingly, up to 39% of individuals in the ‘severe variability’ group were missed by fasting glucose, OGTT, or HbA1c testing, yet spent as much as 15% of the time in prediabetic ranges and 2% in diabetic ranges. These findings suggested that CGM could reveal hidden subphenotypes of dysglycaemia that precede clinical diagnosis. However, replication efforts in larger cohorts, such as the Maastricht Study, demonstrated limited reproducibility [20], underscoring the challenges of generalizing discrete glucotype classifications.

Large-scale initiatives have since advanced the field by linking CGM-derived metrics to broader clinical phenotypes. For example, in the 10K study, average glucose measures such as time-in-range correlated most strongly with adiposity, whereas variability measures were more closely linked to liver function [21²³]. These findings not only highlight CGM’s potential to identify distinct pathophysiological domains but also illustrate the limitations of relying on summary metrics or clustering, which may oversimplify the complex and dynamic nature of glucose regulation.

Recent advances in machine learning provide a way forward. The GLUFORMER model applies transformer-based generative architectures to CGM data, learning latent embeddings that capture the full temporal structure of glucose dynamics [22²⁴]. Unlike traditional clustering, these embeddings can interpolate between states, generate synthetic trajectories, and model the space between observed phenotypes. This capability aligns directly with the concept of T2D as a phenotypic continuum, where individuals span gradations of metabolic dysfunction rather than fitting neatly into discrete subtypes.

Crucially, GLUFORMER embeddings outperform standard CGM metrics for stratification and prediction, demonstrating that representation learning can uncover biologically meaningful patterns overlooked by conventional approaches. Beyond glucose dynamics, such embeddings could be integrated with genomics, metabolomics, microbiome profiles, and lifestyle data to construct unified, multimodal phenotypic spaces. These spaces would not only improve individualized risk prediction but also enable counterfactual simulations – for example, predicting how an individual’s glycaemic trajectory might shift under dietary modification or pharmacologic treatment.

MULTI-OMICS APPROACHES FOR PRECISION TYPE 2 DIABETES PREVENTION ALONG THE CONTINUUM SPECTRUM

Genetic and molecular profiling provide stable, biologically anchored dimensions for characterizing diabetes heterogeneity. Recent studies have shown that

partitioned polygenic scores (pPS) for beta-cell dysfunction, proinsulin processing, adiposity, lipodystrophy, and liver/lipid metabolism can be overlaid on phenotypic continuum models of newly diagnosed T2D [14]. While individual genetic effects are modest compared to clinical variation, regression analyses revealed meaningful structure: obesity pPS aligned with one dimension of the continuum, whereas beta-cell, proinsulin, and liver pPS aligned with another. Specific regions of the continuum enriched for lipodystrophy pPS were linked to poor durability of thiazolidinedione (TZD) therapy, illustrating how genetic architecture can map onto both disease trajectories and therapeutic response [14]. These findings underscore the potential of genetics to anchor continuum-based models in causal biology, while also highlighting limitations related to modest effect sizes, partial overlaps between endotypes, and limited predictive utility for targeted prevention.

Metabolomics adds a dynamic layer to this framework by identifying circulating signatures of dysglycaemia. Elevated branched-chain and aromatic amino acids, alongside reduced glycine and glutamine, consistently precede incident T2D, marking early inflection points in metabolic deterioration [23,24]. Lipoprotein subclass profiling has revealed heterogeneous associations with both diabetes and cardiovascular outcomes [25,26], while intervention studies suggest that metabolomic signatures can help identify individuals most likely to benefit from dietary modification or regress to normoglycemia [27]. Similarly, proteomic analyses have uncovered immune and inflammatory alterations detectable years before clinical onset [28¹¹], and the gut microbiome has emerged as an additional source of variability, shaping both glycaemic responses to meals [29] and cardiometabolic risk through metabolites such as short-chain fatty acids [30], tryptophan [31], and cholesterol [32].

Together, this integration has the potential to link molecular pathways to temporal glycaemic patterns, bridging descriptive phenotypes with mechanistic biology.

FUTURE DIRECTIONS

Advancing precision T2D prevention requires a shift from population-level recommendations toward deeply individualized strategies grounded in an integrated understanding of metabolic heterogeneity. Central to this effort is the creation of large, deeply phenotyped, and longitudinally followed cohorts that capture continuous glucose metrics, multiomics data, microbiome profiles, and behavioural and environmental variables. Standardization of data collection and harmonization across cohorts will be essential to

ensure reproducibility and comparability of findings. Equitable representation of underrepresented populations is also critical to avoid exacerbating existing health disparities and to develop prevention strategies applicable across diverse ethnic, socioeconomic, and geographic contexts.

The convergence of CGM, wearable sensors, and multiomics profiling provides unprecedented granularity in capturing dynamic physiological states. When combined with generative artificial intelligence and advanced machine learning, these datasets can be translated into latent phenotypic embeddings that capture the continuum of metabolic dysfunction. Unlike traditional static clusters, these embeddings allow for modelling individual trajectories, identifying early inflection points, and simulating the effects of interventions in a counterfactual framework. This enables personalized predictions of dietary, pharmacologic, or lifestyle interventions, potentially allowing clinicians to proactively modify disease progression before irreversible β -cell decline or cardiovascular complications occur.

Despite these opportunities, several challenges must be addressed. Many existing datasets are large but shallow, limiting the ability to capture nuanced metabolic and molecular variation. Furthermore, the signal-to-noise ratio of genetic, metabolomic, or microbiome markers remains modest, necessitating rigorous statistical frameworks and validation across independent, ethnically diverse populations. The integration of multimodal data streams also raises complex questions around data governance, privacy, and ethical artificial intelligence use, requiring transparent policies and stakeholder engagement to maintain public trust.

Implementation science will be pivotal in translating these insights into scalable interventions. This includes evaluating the feasibility, acceptability, and cost-effectiveness of embedding CGM, multiomics, and artificial intelligence-driven analytics into routine clinical care [33]. Early-onset and high-risk populations offer particular opportunity for preventive impact, as interventions may be more effective before substantial β -cell decline occurs. Digital health tools, including apps and wearable devices, can support patient engagement and real-time feedback [34], reinforcing behaviour change while collecting actionable phenotypic data.

Looking forward, a multilayered, continuum-based framework integrating CGM, genomics, metabolomics, proteomics, microbiome, and behavioural data can move T2D prevention from a reactive, one-size-fits-all approach toward dynamic, predictive, and precision-guided care. The ultimate goal is to identify high-risk individuals early, understand the underlying biological mechanisms driving heterogeneity, and

implement targeted interventions that reduce both hyperglycaemia and downstream cardiovascular risk. Investments in deep phenotyping, algorithmic transparency, and equitable cohort design are essential to translate the conceptual promise of precision diabetes prevention into clinically actionable and globally relevant strategies.

CONCLUSION

Conceptualizing T2D as a phenotypic continuum rather than discrete subtypes, combined with high-resolution data from CGM, multiomics, and microbiome profiling, provides a powerful framework for precision diabetes prevention. Generative artificial intelligence approaches like GLUFORMER offer scalable tools to extract latent embeddings that capture dynamic metabolic states and predict responses to interventions. Integrating these approaches can improve early detection, risk stratification, and the design of personalized interventions, ultimately advancing precision prevention and mitigating the global burden of T2D and its cardiovascular complications.

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Conflicts of interest

There are no conflicts of interest.

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