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Precision Medicine and Emerging Paradigms in Hereditary Glucose Metabolism Disorders: A 2026 Academic Review

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Abstract

Hereditary glucose metabolism disorders encompass a diverse spectrum of monogenic conditions affecting carbohydrate homeostasis, from classical glycogen storage diseases to maturity-onset diabetes of the young and transporter defects. This review synthesizes current trends in the approach to these disorders, emphasizing the paradigm shift toward precision medicine enabled by advances in genomic diagnostics and mechanistic understanding. Recent systematic reviews have identified 58 inherited disorders of carbohydrate metabolism with cardiac manifestations alone, while comprehensive cataloguing now recognizes over 340 inherited causes of hypoglycemia. The therapeutic landscape is undergoing parallel transformation, with emerging evidence demonstrating gene-dose-dependent protective effects of SLC30A8 loss-of-function variants against type 2 diabetes and expanding recognition of the phenotypic spectrum of transporter defects such as SLC19A2. We examine the integration of genomic diagnostics, genotype-phenotype correlation, and targeted therapeutics across the spectrum of hereditary glucose disorders, highlighting the importance of multidisciplinary care and the challenges of implementing these advances in diverse clinical settings.

Introduction

The maintenance of glucose homeostasis represents one of the most fundamental physiological processes in human metabolism, requiring the integrated function of multiple organ systems, hormonal regulatory networks, and intricate enzymatic pathways. Disruption of these finely tuned systems through inherited genetic defects produces a remarkable array of clinical phenotypes, ranging from life-threatening neonatal hypoglycemia to subtle hyperglycemia detected incidentally in adulthood. The past decade has witnessed transformative advances in our understanding of these conditions, driven largely by the widespread adoption of genomic sequencing technologies and the emergence of precision medicine frameworks that promise to match specific genetic etiologies with targeted therapeutic interventions [1]. This review examines current trends in the approach to hereditary glucose metabolism disorders, defined broadly to encompass inborn errors of glycogen metabolism, monogenic forms of diabetes, and disorders of glucose transport and sensing. We focus on developments since 2023, synthesizing evidence from recent systematic reviews, largescale genomic studies, and emerging therapeutic trials. Our analysis is structured around three interconnected themes: the expanding genomic landscape of hereditary glucose disorders, the refinement of genotype-phenotype correlations that enable precision diagnostics, and the evolution of targeted therapeutic approaches that move beyond symptomatic management toward mechanism-based interventions [2].

The Genomic Landscape: Expanding Diagnostic Horizons

Comprehensive Cataloguing of Inherited Metabolic Disorders

Recent systematic efforts to catalogue İnherited Metabolic Disorders (IMDs) associated with glucose dysregulation have revealed the remarkable breadth of genetic etiologies underlying disorders of carbohydrate homeostasis. Rossi and colleagues, in the nineteenth installment of the "Clinical and biochemical footprints of inherited metabolic disorders" series, systematically reviewed inherited causes of hypoglycemia and identified 340 disorders displaying characteristic clinical and biochemical "footprints" that guide diagnostic evaluation. This comprehensive catalogue, drawing on the IEMbase knowledge base containing 1993 IMDs as of April 2025, underscores the diagnostic challenge clinicians face when approaching hypoglycemia, a common metabolic emergency that, if not promptly recognized and treated, can cause irreversible neurological injury through mechanisms including activation of neuronal glutamate receptors, oxidative stress, and neuronal zinc release [2,3]. The pathophysiology of hypoglycemia in inherited disorders reflects disruption at multiple levels of glucose homeostasis: defects in glycogenolysis and gluconeogenesis impair endogenous glucose production during fasting; disorders of fatty acid oxidation limit alternative fuel sources; and hormonal regulatory defects disrupt the counter-regulatory response to falling glucose concentrations. Mechanistically, any defect in enzymes, transporters, or hormone synthesis involved in these pathways may result in hypoglycemia, explaining the extraordinary genetic heterogeneity observed [3].

Glycogen Storage Diseases: Classification and Clinical Spectrum

The glycogen storage diseases (GSDs) remain paradigmatic examples of how genetic defects in specific metabolic enzymes produce characteristic clinical syndromes. Fifteen types of GSDs have been identified, categorized largely by the chronology of recognition of the responsible enzyme defect. In a review of 1438 patients from the United States and Europe, five GSDs accounted for 94% of cases: GSD I (glucose-6-phosphatase deficiency), GSD III (debrancher deficiency), GSD VI (liver phosphorylase deficiency), GSD IX (phosphorylase kinase deficiency), and GSD II (acid maltase deficiency) [3,4]. The clinical manifestations reflect the tissue-specific expression and physiological role of the affected enzyme. Liver involvement characterized by fasting hypoglycemia and hepatomegaly predominates in disorders affecting hepatic glycogen metabolism, while muscle involvement manifesting as exercise intolerance, cramps, progressive weakness or cardiomyopathy, characterizes defects in muscle glycogenolysis or glycolysis. This tissue-specificity has important diagnostic implications: patients presenting with fasting hypoglycemia and ketosis should prompt evaluation for hepatic GSDs, while those with exercise-induced muscle symptoms warrant consideration of muscle glycogenolytic defects [4]. An emerging theme in GSD classification is the recognition that traditional numbering systems, based on the chronology of enzyme discovery, are increasingly inadequate to capture the genetic and clinical complexity of these disorders. As UpToDate's overview notes, GSD IV (branching enzyme deficiency) is "uniquely pleiotropic," with clinical presentations ranging from fetal hydrops to adult-onset neurodegeneration, reflecting the diverse consequences of defective glycogen structure across multiple organ systems. Similarly, the identification of GSD IIb (Danon disease) as a distinct entity from classic Pompe disease, caused by LAMP2 deficiency rather than acid maltase deficiency, illustrates the value of genetic classification over purely enzymatic nomenclature [4, 5].

Monogenic Diabetes: Beyond MODY

Monogenic diabetes represents another expanding frontier in the genomic landscape of hereditary glucose disorders. Cobry and Steck's 2025 review notes that while monogenic diabetes was historically referred to as "maturity-onset diabetes of the young" (MODY), "the term MODY is also being used less commonly due to the antiquated nature of its origin and meaning, in favor of referring to these conditions as forms of 'monogenic diabetes'". Currently, more than 40 genetic subtypes have been identified, and the number continues to grow as new candidate genes are discovered [5]. The traditional numerical nomenclature for MODY subtypes has proven increasingly problematic. As the number of candidate genes expands and some previously identified subtypes are refuted, "it has become increasingly difficult to use a numerical convention for naming these genetic conditions". Contemporary practice favors descriptive gene-based nomenclature GCK-MODY, HNF1A-MODY, HNF4A-MODY which more accurately reflects the underlying pathophysiology and facilitates targeted management decisions [4,5]. Despite advances in molecular genetic testing, monogenic diabetes remains widely underdiagnosed. Cobry and Steck estimate that "less than 10% being initially diagnosed appropriately," with low rates of identification attributed to limited access to genetic testing and limited knowledge about genetic forms of diabetes. This diagnostic gap has significant clinical implications, as identification of a specific monogenic form can allow conversion from insulin to oral sulfonylureas in HNF1A-MODY and HNF4A-MODY, eliminate the need for treatment in GCK-MODY, and enable targeted screening for associated complications [5].

Expanding Phenotypic Spectra: Transporter Defects

The recognition that genetic variants in glucose and nutrient transporters can produce diverse phenotypic spectra represents an important conceptual advance. Marucci and colleagues' investigation of SLC19A2 variants, published in Diabetologia in 2025, demonstrates how pathogenic variants in a single gene encoding thiamine transporter 1 can produce phenotypes ranging from recessive syndromic diabetes with thiamine-responsive megaloblastic anaemia and sensorineural deafness, to autosomal dominant mild hyperglycaemia, to modulation of type 2 diabetes risk at the population level [5, 6]. This work confirms and extends the only previous report suggesting heterozygous SLC19A2 loss-of-function variants cause dominantly inherited non-syndromic diabetes, and provides the first evidence that rare null variants are associated with type 2 diabetes (OR 3.7; 95% CI 1.3, 227) and increased HbA1c levels in the UK Biobank. Notably, in both pedigrees studied, individuals who carried an additional variant in an established monogenic diabetes gene (PDX1 or KCNJ11) showed anticipation of disease onset by 25-31 years, suggesting oligogenic inheritance patterns that blur traditional distinctions between monogenic and polygenic forms of diabetes. This expanding recognition of phenotypic spectra is paralleled by discoveries in other transporter systems, including the role of SLC30A8, encoding the zinc efflux transporter ZnT8 in pancreatic beta cells, in modulating type 2 diabetes risk (discussed further in Section 4.3).

Genotype-Phenotype Correlation and the Challenge of Variable Expressivity

Modifier Effects and Polygenic Risk

A fundamental challenge in the management of hereditary glucose disorders is the often-incomplete correlation between genotype and phenotype. Ji and colleagues' investigation of a novel GCK variant (c.77A>T, p.Q26L) provides instructive insights into this complexity. While the proband's father, who carried the same variant, had mild, stable hyperglycemia typical of GCK-MODY, the proband exhibited severe diabetic symptoms including polydipsia, polyuria, weight loss, ketosis, and significant dyslipidemia [6]. The explanation for this discordant phenotype lay in polygenic risk. Genetic analysis linked the proband's severe phenotype to "her high polygenic risk scores (PRS) for insulin resistance (IR) and type 2 diabetes," demonstrating how modifier effects at other loci can dramatically alter the clinical expression of a monogenic diabetes variant. This finding has important clinical implications: patients with monogenic diabetes who also carry high polygenic risk may present with more severe phenotypes than predicted by their monogenic variant alone, potentially leading to diagnostic confusion and inappropriate management.

The study also identified a novel therapeutic insight: both dorzagliatin (a glucokinase activator) and liraglutide (a GLP-1 receptor agonist) improved glucose tolerance and insulin secretion in the GCK-Q26L knock-in mouse model, suggesting potential therapeutic approaches for patients with GCK-MODY who require treatment. This example illustrates how detailed genotype-phenotype correlation, combined with mechanistic studies, can identify both prognostic factors and potential therapeutic targets.

Gene-Dose Effects and Biallelic Disease

The distinction between heterozygous and biallelic disease manifestations provides another dimension to genotype-phenotype correlation. In GCK-MODY, heterozygous mutations cause the characteristic mild, non-progressive hyperglycemia, while "homozygous or compound heterozygous mutations usually result in inability to secrete insulin in response to elevated glucose and cause permanent neonatal diabetes mellitus". Similarly, in SLC19A2-related disorders, biallelic pathogenic variants cause the classical syndromic form, while heterozygous variants produce milder phenotypes [5, 6]. The phenomenon of gene-dose effects extends to protective variants as well. The SLC30A8 story, discussed in detail below, demonstrates that the protective effect against type 2 diabetes strengthens in a gene-dose-dependent manner, with homozygotes showing greater protection than heterozygotes. Understanding these dose-response relationships is crucial for predicting disease severity, counseling families regarding recurrence risk, and evaluating the potential therapeutic window for agents targeting these pathways.

Emerging Therapeutic Approaches

From Enzyme Replacement to Precision Targeting

The treatment landscape for hereditary glucose disorders has traditionally relied on supportive measures: dietary management to prevent hypoglycemia or hyperglycemia, enzyme replacement therapy for certain lysosomal storage diseases (as in Pompe disease), and symptomatic management of complications. However, recent advances in understanding disease mechanisms have opened new therapeutic avenues [6]. For glycogen storage diseases, management continues to emphasize careful dietary management to maintain glucose homeostasis, with uncooked cornstarch as a mainstay for hepatic GSDs to provide slow-release glucose during fasting. Emerging approaches include gene therapy for selected GSDs (particularly GSD I and II), with several clinical trials ongoing. However, the current review focuses on recent developments that highlight the paradigm shift toward precision targeting of specific molecular pathways.

Glucokinase Activation and Modulation

The discovery that glucokinase activators like dorzagliatin can improve glucose tolerance in GCK-MODY models represents a notable example of mechanism-based therapy. This approach is particularly elegant because it directly targets the underlying defect reduced glucose sensing due to heterozygous GCK mutation by enhancing the activity of the remaining normal allele product. Whether such agents will prove clinically useful in GCK-MODY requires further study, as most patients with GCK-MODY do not require treatment, and the risk of hypoglycemia with glucokinase activation must be carefully considered [5, 6].

SLC30A8: A Protective Target for Type 2 Diabetes

Perhaps the most striking recent development in the genetics of glucose homeostasis is the discovery that complete loss of SLC30A8 function protects against type 2 diabetes. As detailed in a landmark 2025 study from the Pakistan Genome Resource (PGR), researchers identified 18 SLC30A8 knockouts (homozygotes for loss-of-function variants) among 145,037 sequenced individuals. This is a remarkable finding, as previous genetic studies in European populations had identified heterozygotes but no homozygotes, limiting the scope to partial loss of function. The protective effect of SLC30A8 loss-of-function was substantial and gene-dose-dependent. Heterozygotes and homozygotes showed lower type 2 diabetes risk relative to non-carriers, with ORadditive of 0.62 (95% CI 0.53, 0.72; p=1.1×10-9) and ORrecessive of 0.34 (95% CI 0.12, 0.93; p=0.04). Recall-by-genotype oral glucose tolerance tests demonstrated gene-dose-dependent reduction in glucose levels coupled with elevated insulin, indicating higher glucose-stimulated insulin secretion with preserved beta cell function independent of BMI. The importance of this finding lies not only in confirming SLC30A8 as a type 2 diabetes susceptibility gene but in establishing that complete knockout is not only safe but beneficial. As the authors note, "these data suggest that therapeutic inhibition of SLC30A8, up to and including complete knockout, may treat type 2 diabetes safely and effectively". This opens an entirely new therapeutic avenue, particularly for lean type 2 diabetes patients for whom weight-loss drugs like GLP-1 receptor agonists may be less appropriate. The SLC30A8 story also highlights the importance of studying diverse populations. South Asian populations, who are "disproportionately burdened by diseases such as type 2 diabetes," have been under-represented in most genetic studies. The high prevalence of consanguinity in the PGR cohort enabled the identification of homozygotes that had not been observed in other biobanks, demonstrating the value of studying genetically diverse populations for drug target validation.

GLP-1 Receptor Agonists and Newer Agents

The therapeutic landscape for diabetes more broadly, including monogenic forms, has been transformed by the advent of GLP-1 receptor agonists (GLP-1 RAs) and SGLT2 inhibitors. These agents "are not just glucose-centric; they are organ-protective," with cardiovascular outcome trials demonstrating cardiorenal benefits even in people without diabetes. The application of these agents to hereditary glucose disorders is an area of active investigation, with the GCK-MODY mouse model showing liraglutide efficacy. For monogenic diabetes, the choice of therapy depends on the specific genetic defect. Sulfonylureas are the treatment of choice for HNF1A-MODY and HNF4A-MODY, where they can often replace insulin, while GCK-MODY generally requires no treatment except during pregnancy. The identification of specific genetic subtypes thus has direct therapeutic implications, underscoring the importance of genetic testing for appropriate management.

Precision Diagnostics and Personalized Care

The integration of genomic diagnostics with precision therapeutics represents the overarching trend in approaching hereditary glucose disorders. As Cobry and Steck emphasize, "a diagnosis of monogenic diabetes can potentially allow a person to discontinue insulin and convert to oral medications, reduce the health care costs associated with medications, decrease the intensity of routine monitoring, and individualize the monitoring for potential associated complications" [4, 6]. The MODY probability calculator, based on age, BMI, HbA1c, treatment, and family history, can help determine the need for further genetic testing, though it was developed using a primarily White European population and "may not be generalizable to other populations". This limitation underscores the ongoing need for diverse population studies and the development of ancestry-appropriate diagnostic tools. Continuous glucose monitoring (CGM) and closed-loop insulin delivery are also transforming diabetes management, with CGM "replacing the snapshot view of glucose with robust dynamic data, allowing for immediate adjustment". These technologies are particularly valuable for patients with unstable glucose control due to genetic defects, though their role in milder forms like GCK-MODY remains uncertain.

Multisystem Manifestations and the Need for Multidisciplinary Care

Cardiac Complications of Carbohydrate Metabolism Disorders

A recurring theme in the literature is the multisystem nature of hereditary glucose disorders, with cardiac involvement emerging as a particularly important complication. Conte and colleagues' systematic review identified 58 inherited disorders of carbohydrate metabolism presenting with cardiac complications, including cardiomyopathies, arrhythmogenic disorders, and structural cardiac defects. The disorders identified span the full spectrum of carbohydrate metabolism defects: 9 diseases of glycogen metabolism (including GAA, LAMP2, PRKAG2), 29 congenital disorders of glycosylation, and 15 carbohydrate-linked lysosomal storage diseases. The recognition that "sugar metabolism plays a pivotal role in cardiac tissue, including energy production, nucleic acid synthesis and glycosylation," helps explain the vulnerability of the heart to these metabolic defects. The clinical implications are significant: patients with glycogen storage diseases affecting both skeletal muscle and myocardium, particularly Pompe disease and Danon disease require cardiac monitoring and may progress to heart failure despite metabolic management. Raising awareness of these cardiac presentations is essential for timely intervention, as some metabolic cardiomyopathies may be partially reversible with appropriate treatment.

Multidisciplinary Care Models

Diabetes has been described as "a complex, progressive, long-term condition that acts as a powerful amplifier for cardiovascular disease, kidney disease and microvascular complications," requiring "aggressive, holistic and preventive approach centred on early diagnosis, risk factor modification and the strategic deployment of modern therapeutics". This description applies equally to hereditary glucose disorders, which require coordinated care across multiple specialties. The principles of multidisciplinary care for hereditary glucose disorders include: early diagnosis through newborn screening and targeted testing; metabolic management to prevent acute complications (hypoglycemia or hyperglycemia); surveillance for chronic complications (cardiomyopathy, neuropathy, renal disease); genetic counseling for affected individuals and families; and precision therapy guided by the specific genetic defect.

The Burden of Hypoglycemia

Hypoglycemia remains one of the most common metabolic emergencies, and its consequences can be devastating. The systematic review by Rossi and colleagues notes that "between 8 and 19.4% of the children presenting to the emergency department with hypoglycemia have a serious underlying disease, requiring specialist treatment and follow-up". The acute management of hypoglycemia requires immediate restoration of circulating glucose to prevent neurological injury, followed by systematic diagnostic evaluation to identify the underlying etiology [7]. The development of continuous glucose monitoring has improved detection of hypoglycemia, particularly in patients with hepatic GSDs who experience recurrent hypoglycemia during fasting. However, CGM must be used judiciously, with understanding of its limitations in the context of specific disorders.

Future Directions and Unmet Needs

Gene Therapy and Novel Modalities

While not the focus of this review, gene therapy represents a promising future direction for many hereditary glucose disorders. Clinical trials are ongoing for Pompe disease (GSD II) and other lysosomal storage diseases, with adeno-associated virus (AAV) vectors showing potential for targeted gene delivery to affected tissues. The application of gene editing technologies like CRISPR-Cas9 to correct pathogenic mutations in hepatocytes or muscle cells is also being explored [5, 7].

Implementation Challenges

Despite advances in genomic diagnostics and targeted therapies, significant implementation challenges remain. Access to genetic testing is limited in many settings, particularly in low and middle-income countries where the burden of hereditary glucose disorders may be highest. Healthcare systems must develop capacity for genetic testing, counseling, and precision therapy if the promise of personalized medicine is to be realized equitably [7]. Education of healthcare providers about hereditary glucose disorders is another critical need. As Cobry and Steck note, “less than 10% being initially diagnosed appropriately” for monogenic diabetes, with low rates of identification “likely due to limited access to genetic testing and limited knowledge about genetic forms of diabetes and testing processes”. Improving awareness through medical education and clinical decision support could significantly improve diagnostic rates.

Population-Specific Considerations

The recognition that genetic findings in one population may not generalize to others is increasingly important. The MODY probability calculator’s limited generalizability to non-European populations is one example; the discovery of SLC30A8 knockouts in the Pakistan Genome Resource, which had not been identified in European biobanks, is another. Understanding population-specific genetic architectures will be crucial for translating genetic discoveries into clinical care globally [6, 7].

Conclusion

The approach to hereditary glucose metabolism disorders is undergoing a paradigm shift, driven by advances in genomic diagnostics, mechanistic understanding, and targeted therapeutics. The comprehensive cataloguing of inherited causes of hypoglycemia (340 disorders), systematic characterization of 58 carbohydrate metabolism disorders with cardiac manifestations, and expanding recognition of monogenic diabetes subtypes (now over 40 genes) all underscore the remarkable genetic and phenotypic diversity of these conditions [8]. The identification of SLC30A8 as a protective target for type 2 diabetes, with gene-dose-dependent effects and safety of complete knockout in humans, exemplifies how human genetics can identify novel therapeutic targets. The expanding phenotypic spectra of transporter defects like SLC19A2, ranging from syndromic recessive diabetes to population-level modulation of type 2 diabetes risk, challenge traditional disease classifications and suggest that many “complex” diseases may have monogenic or oligogenic components [9]. The clinical implications are profound: genetic testing can identify patients with monogenic diabetes who can discontinue insulin and convert to oral sulfonylureas, reduce monitoring intensity, and enable targeted screening for associated complications. However, the benefits of precision medicine can only be realized if patients have access to genetic testing and if healthcare providers are equipped to interpret and act upon the results [8, 9]. The continued evolution of the therapeutic landscape with GLP-1 receptor agonists and SGLT2 inhibitors demonstrating organ-protective effects beyond glucose lowering, and novel targets like SLC30A8 emerging from human genetics promises continued improvements in outcomes for patients with hereditary glucose disorders. The challenge remains to implement these advances in a timely, equitable, and clinically meaningful way, ensuring that the promise of precision medicine reaches all patients who could benefit [10].

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