World Journal of Diabetes

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World J Diabetes 2025 July 15; 16(7): 106218

DOI: 10.4239/wjd.v16.i7.106218

ISSN 1948-9358 (online)

REVIEW

Illuminating diabetes *via* multi-omics: Unraveling disease mechanisms and advancing personalized therapy

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Specialty type: Endocrinology and metabolism

Provenance and peer review: Invited article; Externally peer reviewed.

Peer-review model: Single blind

Peer-review report's classification

Scientific Quality: Grade A, Grade B, Grade B, Grade B, Grade C

Novelty: Grade A, Grade B, Grade C

Creativity or Innovation: Grade A, Grade B, Grade C

Scientific Significance: Grade B, Grade B, Grade C

P-Reviewer: Adepoju VA; Alvarez M; Jiang W; Pappachan JM; Sun XD

Received: February 20, 2025

Revised: April 8, 2025

Accepted: May 27, 2025

Published online: July 15, 2025

Processing time: 146 Days and 2.3 Hours

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Abstract

Diabetes mellitus (DM) comprises distinct subtypes-including type 1 DM, type 2 DM, and gestational DM - all characterized by chronic hyperglycemia and substantial morbidity. Conventional diagnostic and therapeutic strategies often fall short in addressing the complex, multifactorial nature of DM. This review explores how multi-omics integration enhances our mechanistic understanding of DM and informs emerging personalized therapeutic approaches. We consolidated genomic, transcriptomic, proteomic, metabolomic, and microbiomic data from major databases and peer-reviewed publications (2015-2025), with an emphasis on clinical relevance. Multi-omics investigations have identified convergent molecular networks underlying β-cell dysfunction, insulin resistance, and diabetic complications. The combination of metabolomics and microbiomics highlights critical interactions between metabolic intermediates and gut dysbiosis. Novel biomarkers facilitate early detection of DM and its complications, while single-cell multi-omics and machine learning further refine risk stratification. By dissecting DM heterogeneity more precisely, multi-omics integration enables targeted interventions and preventive strategies. Future efforts should focus on data harmonization, ethical considerations, and real-world validation to fully leverage multi-omics in addressing the global DM burden.

Key Words: Diabetes mellitus; Metabolomics; Multi-omics; Precision medicine; Genomics; Transcriptomics; Proteomics; Biomarker discovery; Personalized therapy

Core Tip: Metabolomics offers key insights into the distinct metabolic pathways of each diabetes mellitus subtype. By integrating multiple omics layers-genomic, transcriptomic, proteomic, and microbiomic - researchers can refine disease classification, identify novel biomarkers, and develop personalized interventions, substantially enhancing the efficacy of diabetes management.

Citation: Song CM, Lin TH, Huang HT, Yao JY. Illuminating diabetes *via* multi-omics: Unraveling disease mechanisms and advancing personalized therapy. *World J Diabetes* 2025; 16(7): 106218
URL: https://www.wjgnet.com/1948-9358/full/v16/i7/106218.htm
DOI: https://dx.doi.org/10.4239/wjd.v16.i7.106218

INTRODUCTION

Diabetes mellitus (DM) is a common metabolic disorder typically categorized as type 1 DM (T1DM), type 2 DM (T2DM), or gestational DM (GDM) apart from various uncommon types such as monogenic diabetes, syndromic diabetes and secondary diabetes. T1DM frequently stems from autoimmune destruction of pancreatic β-cells[1-3], whereas T2DM is characterized by insulin resistance alongside varying degrees of β-cell dysfunction[4-9]. GDM, in turn, develops during pregnancy and adversely impacts both maternal and fetal health[10-16]. Despite their distinct etiologies, all DM subtypes share the feature - sustained hyperglycemia, culminating in micro-/macrovascular complications such as diabetic kidney disease (DKD) and diabetic retinopathy (DR)[17-21].

'Which multi-omics findings are most clinically translatable for T1DM, T2DM, and GDM?' Addressing this question demands clearer delineation among subtypes-T1DM is largely driven by autoimmunity, T2DM by metabolic overload and insulin resistance, and GDM by pregnancy-related hormonal imbalances. Metabolomics has been instrumental in uncovering each subtype's metabolic disruption[5,10-15,22,23]. However, no single omics layer can fully capture the disease's complexities. Consequently, multi-omics integration-encompassing genomics, transcriptomics, proteomics, and epigenomics - provides a more comprehensive lens through which gene-protein-metabolite-environment interactions are elucidated[1,4-9,24-29].

By combining multiple omics layers, researchers have greatly expanded our understanding of β-cell dysfunction, insulin resistance, and diabetic complications. For instance, coupling transcriptomics with metabolomics reveals how inflammatory pathways correlate with lipid metabolic shifts[30-38], while proteogenomic analyses highlight potential therapeutic targets in gluconeogenesis or glucose uptake[19,20,39-43]. Through these approaches, a refined understanding of DM's heterogeneity emerges, paving the way for novel biomarkers, individualized therapies, and translational research advances[2,18,21,44-50].

METHODOLOGY

We systematically searched PubMed and Web of Science for the period spanning January 2020 to December 2025. Our search strings included "(multi-omics OR genomics OR proteomics OR metabolomics OR microbiomics OR epigenomics) AND (diabetes OR T1DM OR T2DM OR GDM)". We included original studies, reviews, and meta-analyses offering mechanistic or translational insights into the main DM subtypes. Studies focusing solely on non-diabetic metabolic disorders or lacking clear multi-omics integration were excluded. We prioritized large-cohort or consortium-based evidence as well as innovative single-cell and machine learning (ML)-based studies.

METABOLOMICS IN DIABETES: CAPTURING THE BIOCHEMICAL FOOTPRINT

Metabolomics provides a critical view of small-molecule metabolites (*e.g.*, amino acids, lipids, carbohydrates), clarifying the metabolic differences in T1DM, T2DM, GDM, and advanced complications like DKD or DR[5,7,10-12,14,16,18,26,43,51,52].

T1DM: Autoimmune-related signatures

Autoimmune-mediated β-cell destruction and insulin deficiency yield specific pro-inflammatory metabolites[1-3].

T2DM: Insulin resistance and lipid dysregulation

T2DM is characterized by insulin resistance and elevated BCAAs or lipid imbalances[5,8,22,23,27-32,35-37,41,47,53-55], distinct from T1DM's primary autoimmunity.

GDM: Pregnancy-induced metabolic shifts

GDM results from pregnancy-related hormonal fluctuations that disrupt glucose/lipid homeostasis[10-16]. Metabolomic profiles often reveal dysregulated bile acids and lipids, potentially allowing earlier diagnosis.

Complications (DKD, DR)

Although chronic hyperglycemia predisposes all DM subtypes to renal or retinal damage, multi-omics suggests that T1DM, T2DM, and GDM may follow subtype-specific pathways. Recent metabolomic studies document amino acids, lipids, and inflammatory markers tied to complication severity[17-20].

With liquid chromatography-mass spectrometry, gas chromatography-mass spectrometry, nuclear magnetic resonance, and rigorous computational pipelines, metabolomics offers fresh insights into hyperglycemia's pathophysiology and pinpoints promising biomarkers or therapeutic targets[4,6,9,21,24-26,33,34,42-46,48,50,52,56,57]. By capturing each patient's metabolic "footprint", metabolomics provides a crucial step toward precision diabetes care (Figure 1).

BEYOND METABOLITES: INTEGRATING GENOMICS, TRANSCRIPTOMICS, PROTEOMICS, AND MICROBIOME

While metabolomics uncovers vital biochemical shifts, multi-omics integration extends to genomics, transcriptomics, proteomics, and microbiomics[58-61]. Genomics unearths hereditary predispositions; transcriptomics highlights gene-expression changes induced by hyperglycemia or insulin resistance[1-3,7-9,26-28]. Proteomics analyzes post-translational modifications, enzyme expression, and critical signaling events[5,6,19,20,24,25,29,43,52].

Microbiomics, focusing on 16S rRNA sequencing or shotgun metagenomics, further reveals how gut dysbiosis may exacerbate insulin resistance, particularly in T2DM and GDM[62-64]. Integrating these layers offers a holistic perspective on how DM progresses from normoglycemia to overt hyperglycemia.

For example, genome-wide association studies identify DM susceptibility loci, while transcriptomics verify gene-expression alterations in islet, muscle, or liver tissues[4,8,27]. Proteomics further confirm protein-level changes relevant to insulin pathways[19,20,24]. Together with metabolomics, these integrated data illuminate regulators of gluconeogenesis, adipogenesis, and inflammation (Figure 2)[65-67].

KEY MULTI-OMICS FINDINGS: BIOMARKER DISCOVERY AND MECHANISTIC INSIGHTS

Table 1 outlines representative multi-omics investigations in DM. Beyond familiar markers such as BCAAs and certain lipid profiles, integrated omics efforts uncover new signatures distinguishing DM subtypes or correlating with disease severity. For instance, elevated glycerophospholipids correlate with insulin resistance in T2DM, whereas atypical amino acid pathways may foreshadow DKD or cognitive decline[21,30,32,38,45,47,54,55].

Mechanistically, these analyses link genotypes or gene-expression changes to imbalances in lipid and glucose metabolism, clarifying how genetic risk factors translate into clinical phenotypes. Proteomics corroborates the enzyme-level or post-translational modifications that shape metabolic flux. In T1DM, multi-omics shows how autoimmune-associated gene variants converge with metabolic dysfunction, identifying potential early interventions[1-3]. Meanwhile, GDM-focused metabolomics surveys serum and placental samples to detect abnormal energy substrates predictive of future diabetic complications[10,12-15]. Multi-omics approaches also spotlight pathophysiological networks underlying DR, diabetic cardiomyopathy (DCM), and microvascular damage, enabling more precise patient stratification and potential disease-modifying strategies[24,25,36,52,54].

By bridging molecular perturbations and clinical endpoints, multi-omics surpasses single-marker paradigms. These findings (Table 1) further support biomarker discovery and highlight new therapeutic levers for DM detection, risk stratification, and treatment.

NEW TOOLS AND FUTURE DIRECTIONS: SINGLE-CELL MULTI-OMICS AND ML

Single-cell multi-omics applications

Single-cell multi-omics deciphers the cellular heterogeneity masked by bulk analyses[68-70]. In DR or DCM, single-cell profiling reveals specific endothelial or immune cell subsets that drive tissue injury[71,72]. Such detailed views may identify novel therapeutic avenues, including immunomodulation in T1DM or targeted interventions for T2DM-related cardiovascular complications.

ML in multi-omics integration

ML excels at integrating and analyzing large-scale multi-omics datasets[73,74]. Techniques ranging from graph neural networks to autoencoders improve biomarker identification and DM classification accuracy[75,76]. However, model optimization varies across T1DM, T2DM, and GDM due to their distinct disease drivers. Collaborations among data-sharing platforms (*e.g.*, MetaboLights) facilitate reproducibility and drive multi-omics biomarkers toward clinical implementation[77-79].

Table 1 Representative multi-omics studies across different diabetes subtypes and complications

Dm subtype/focus	Omics approach(s)	Primary objective/key findings	Ref.
T1DM (human cohorts)	Genomics (HLA region); metabolomics (LC-MS); epigenomics (RRBS)	Investigate autoimmune-driven β-cell destruction and identify early T1DM biomarkers	[1-3]
T2DM (human & animal)	Transcriptomics (RNA-Seq); proteomics (LC-MS/MS); metabolomics (NMR)	Elucidate insulin resistance, islet dysfunction; discover novel therapeutic targets	[8,9]
GDM (human studies)	Metabolomics (GC-MS); microbiomics (16S rRNA)	Uncover pregnancy-specific metabolite and microbiome signatures predictive of GDM	[10,12-15]
DKD/DR complications	Proteomics (targeted); metabolomics (untargeted); ML-based integration	Correlate inflammatory and fibrotic biomarkers with organ damage; early detection in both T1DM and T2DM	[17-20]
GI complications	Multi-omics synergy (transcript + metabolite)	Reveal changes in gut motility, microbiome composition, disease progression in T2DM	[25,36,42]
Maternal hyperglycemia	Metabolomics (LC-MS); lipidomics	Assess how hyperglycemia in pregnant sows influences neonatal hepatic metabolism	[43]
Microbiome in T1DM/ T2DM/GDM	Microbiomics (16S rRNA, shotgun metagenomics); Metabolomics (SCFAs)	Examine gut dysbiosis related to insulin resistance, inflammation, and distinct metabolic phenotypes	[62-64]

LC-MS: Liquid chromatography-mass spectrometry; GC-MS: Gas chromatography-mass spectrometry; NMR: Nuclear magnetic resonance; RRBS: Reduced representation bisulfite sequencing; ML: Machine learning; SCFAs: Short-chain fatty acids; T1DM: Type 1 diabetes mellitus; T2DM: Type 2 diabetes mellitus; HLA: Human leukocyte antigen; GDM: Gestational diabetes mellitus; GI: Gastrointestinal; MS: Mass spectrometry; DKD: Diabetic kidney disease; DR: Diabetic retinopathy.

FROM BENCH TO BEDSIDE: TRANSLATIONAL APPLICATIONS AND PERSONALIZED THERAPIES

Multi-omics research is shifting from conceptual breakthroughs to real-world clinical utility[80,81]. By jointly examining genomic, transcriptomic, and metabolic data in patient samples, clinicians can more accurately identify subphenotypes beyond basic metrics (*e.g.*, body mass index, fasting glucose)[77-79,82-87]. In T2DM, multi-omics biomarkers can flag individuals at elevated risk for nephropathy or cardiomyopathy, enabling earlier, more focused interventions[21,24,27,29,43-46,48,53,88,89]. Rare metabolic disorders such as maple syrup urine disease also benefit from multi-omics, which locates hidden epigenetic or transcriptomic anomalies[90,91]. The same logic applies to autoimmune diseases and cancers, where integrated cell-free DNA methylation and fragmentation analyses provide noninvasive diagnostic options with higher specificity[77-79,83,85,92-94].

ML-based clinical decision tools

ML-based clinical decision tools can now predict drug responses or therapy resistance by merging transcriptomic, metabolomic, and proteomic data[32,34,57,95]. In non-alcoholic fatty liver disease/metabolic dysfunction-associated fatty liver disease and hepatocellular carcinoma, multi-omics uncovers asymptomatic molecular changes and druggable pathways[86,88,89]. Collectively, these advances highlight how multi-omics can tailor interventions across DM subtypes, immunomodulation in T1DM, metabolic-targeting strategies in T2DM, or fetal-protective regimens in GDM (Table 2).

CHALLENGES AND FUTURE PERSPECTIVES IN MULTI-OMICS INTEGRATION

Despite significant progress, implementing multi-omics in routine practice faces several hurdles:

Data complexity

Analyzing heterogeneous datasets (DNA, epigenetics, RNA, proteins, metabolites) demands expert bioinformatics, secure data management, and specialized hardware[83,95,96]. Large or rare cohorts produce massive, high-dimensional data requiring advanced ML algorithms[82,83,95].

Standardization

Discrepancies in sample collection, instrumentation, and preprocessing hamper reproducibility. Although attempts to harmonize data exist, cross-study consistency remains a concern[89,91,97,98].

Cost and logistical burden

Generating multi-omics data for sizable cohorts is expensive and time-intensive, especially for acute or rare diseases[84,87,97]. Specialized instrumentation and expertise further restrict broader adoption.

Table 2 Translational insights from multi-omics studies in diabetes

Focus/theme	Key multi-omics finding	Potential clinical application
T1DM: Early autoimmune risk assessment	Genomics/epigenomics highlight HLA variants & methylation changes; Metabolomics reveals pro-inflammatory signatures	Identifying high-risk individuals for early intervention[1-3]
T2DM: Insulin resistance & metabolic overload	Elevated BCAAs/lipids from metabolomics [5,8,23,30-38]; Transcriptomics pinpoints insulin signaling defects [24,25]	Improved patient stratification; Tailored dietary or pharmacological interventions targeting dysregulated pathways
GDM: Pregnancy-specific biomarkers & interventions	Metabolomics + microbiomics [10,12-15] reveal distinct lipid/bile acid profiles, gut flora changes	Early screening of at-risk mothers; Nutritional or probiotic therapies to minimize fetal impact
Diabetic complications (DKD, DR)	Proteomics + metabolomics identify inflammation/fibrosis [17-20]; Single-cell multi-omics links endothelial dysfunction to hyperglycemia	Risk stratification for DKD, DR; Earlier monitoring and targeted therapy

BCAAs: Branched-chain amino acids; HLA: Human leukocyte antigen; DKD: Diabetic kidney disease; DR: Diabetic retinopathy; T1DM: Type 1 diabetes mellitus; T2DM: Type 2 diabetes mellitus; GDM: Gestational diabetes mellitus; HLA: Human leukocyte antigen.

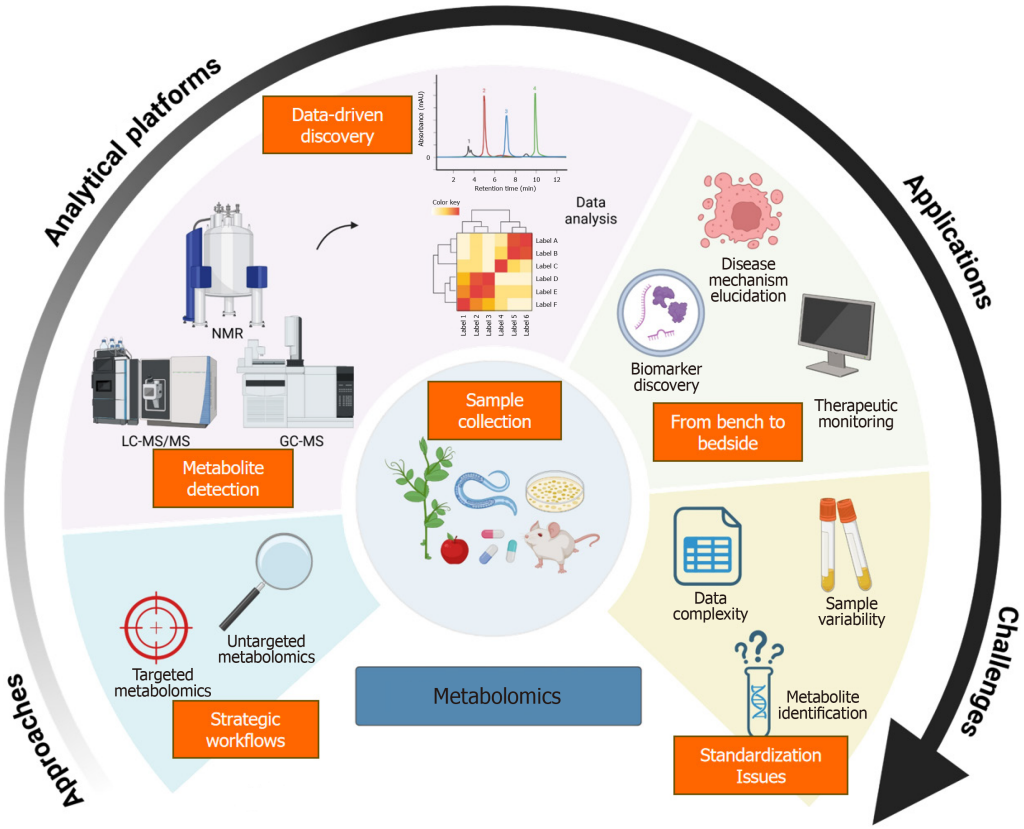

Figure 1 Overview of metabolomics approaches, analytical platforms, applications, and challenges. Samples (biofluids, tissues, cells) are collected from individuals with distinct diabetes subtypes (type 1 diabetes mellitus, type 2 diabetes mellitus, gestational diabetes mellitus) and related complications (*e.g.*, diabetic kidney disease, diabetic retinopathy). Researchers can choose targeted *vs* untargeted metabolomics strategies depending on the objectives (biomarker discovery, mechanism elucidation). Key analytical platforms include liquid chromatography-mass spectrometry, gas chromatography-mass spectrometry and nuclear magnetic resonance, each capturing unique metabolite classes. The figure also highlights the main challenges-such as data complexity, sample variability, and metabolite identification-along with the resulting potential applications in diagnosis, risk stratification, and therapeutic monitoring. LC-MS: Liquid chromatography-mass spectrometry; GC-MS: Gas chromatography-mass spectrometry; NMR: Nuclear magnetic resonance.

Functional validation

Findings from omics-based analyses demand laboratory confirmation (*e.g.*, CRISPR, siRNA)[1,20,43,90]. In DR studies, single-cell transcriptomics plus metabolomics may identify key endothelial subsets behind vascular dysfunction, but robust *in vitro*/*in vivo* validation is crucial[24,25,36,52,54].

Limitations and outlook

Applicability of this review is limited by the intrinsic heterogeneity of multi-omics studies, which vary in methodology, cohort size, and data standardization. Moreover, routine multi-omics adoption encounters barriers involving cost,

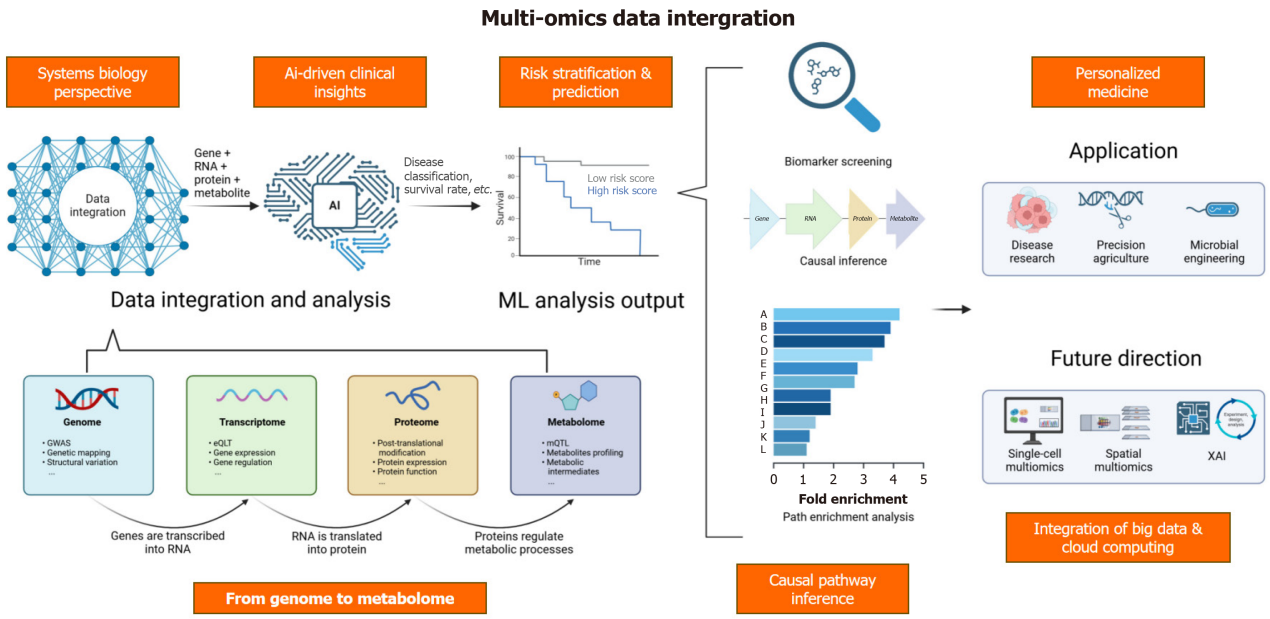

Figure 2 Multi-omics data integration and machine learning in disease research. This schematic illustrates how multi-layered omics data-genomic (*e.g.*, genome-wide association study), transcriptomic (gene expression), proteomic (protein abundance/modifications), metabolomic (small-molecule metabolites), and microbiomic-are consolidated into a unified analysis pipeline. Machine learning models (*e.g.*, neural networks, random forests, or autoencoders) can uncover hidden patterns, classify diabetes subtypes, and predict disease progression or therapy response. The figure underscores how single-cell/spatial omics approaches enhance resolution by identifying rare cell populations, while explainable artificial intelligence techniques clarify the basis of predictive models, ultimately guiding personalized interventions. AI: Artificial intelligence; XAI: Explainable artificial intelligence; ML: Machine learning.

infrastructure, and ethical concerns around large-scale data sharing. Future research should focus on establishing unified protocols, recruiting diverse populations, and leveraging emerging single-cell/spatial omics and artificial intelligence (AI) frameworks for maximal clinical impact.

ETHICAL, REGULATORY, AND EQUITY CONSIDERATIONS

Ethical and regulatory challenges

Accessing large-scale patient omics data typically requires stringent institutional review board approvals and robust informed consent. Varying privacy regulations across institutions or countries can complicate data harmonization, underscoring the need for standardized ethical frameworks and global data-sharing strategies.

Potential risks of AI-driven data integration

AI offers powerful analytics but also poses risks such as unauthorized access or commercialization of sensitive patient data. Transparent governance and robust cybersecurity measures are paramount to protect patient confidentiality.

Equity and representation

Many multi-omics databases underrepresent certain ethnic or socioeconomic groups, leading to potential biases in disease diagnosis, risk prediction, and treatment. Incorporating diversity in cohort recruitment and cross-institutional collaborations can help minimize disparities and enhance the real-world relevance of multi-omics findings.

TOWARD A NEW ERA OF DIABETES MANAGEMENT

Although multi-omics results are largely consistent across diverse data layers, variations in patient ethnicity, sample size constraints, and diverging analytical pipelines introduce inconsistencies. Validating these findings demands large-scale longitudinal studies that explore causality and reduce confounders. Major open questions include how epigenetic modifications differ among T1DM, T2DM, and GDM at single-cell resolution; which multi-omics signatures best predict complications like DCM; and whether cost-effective ML pipelines can be deployed for widespread clinical screening.

By harmonizing genomics, transcriptomics, proteomics, and metabolomics, multi-omics has substantially reshaped our view of DM's complexity. Compared to single-omics, multi-omics supplies a more granular depiction of disease heterogeneity, enabling earlier biomarker discovery and improved therapeutic targeting-ranging from T2DM-specific interventions to T1DM immunomodulation and noninvasive metabolic assessments for GDM[1,2,10,12,16,26,28,43,50,82,83,85,88]. Personalized interventions, once aspirational, are now coming within reach, aided by robust multi-omics models

that refine patient stratification and novel treatment strategies for disorders extending beyond DM, including leukemia and autoimmune diseases[77,78,86,87,89,91-93,96,97,99,100].

Even with these advancements, fully embedding multi-omics into diabetes care mandates sustained interdisciplinary collaboration, standardized methodologies, and powerful computational tools. Data-sharing consortia, open-science initiatives, and single-cell/spatial omics innovations promise to illuminate cellular heterogeneity, especially in diabetic complications. Overcoming these challenges can usher in a new era of diabetes management-one anchored by mechanistic clarity, sophisticated diagnostics, and genuinely patient-specific care.

As multi-omics evolves, it yields unparalleled opportunities for transforming diabetes management into a predictive, preventive, and patient-centered framework. With concerted efforts in biomarker development, therapeutic innovation, and translational science, the vision of personalized diabetes treatment moves ever closer to reality.

CONCLUSION

By uniting genomics, transcriptomics, proteomics, and microbiomics, multi-omics research has revolutionized our understanding of DM's pathophysiology and reshaped its clinical management. In the near term, AI-enhanced pipelines that integrate multi-omics data are poised to improve early detection and enable precision therapies. Establishing standardized protocols and more diverse patient cohorts will be essential to ensure a seamless transition of these breakthroughs from the laboratory to everyday diabetes care. With sustained progress in biomarker discovery, therapeutic innovation, and translational research, a future defined by truly individualized diabetes interventions appears increasingly achievable.

FOOTNOTES

Author contributions: Song CM and Lin TH contributed equally; Yao JY supervised all aspects, made critical revisions, and is responsible for correspondence; Song CM, Lin TH, and Huang HT carried out the literature review, interpreted the data, and drafted the original manuscript; All authors prepared the draft and approved the submitted version.

Conflict-of-interest statement: All the authors report no relevant conflicts of interest for this article.

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S-Editor: Li L
L-Editor: A
P-Editor: Xu ZH

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