



The Insulin Resistance: Analysing Divergent Physiological Dynamics and Remission Pathways in Prediabetes versus Overt Type 2 Diabetes using Large Language Model

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Abstract:

Insulin resistance (IR) is a metabolic pandemic affecting South Asia, where both genetic predisposition and high carbohydrate exposure combined with protein-deficient nutrition are key drivers. Standard laboratory markers measure static disease thresholds, but often do not teach patients about the systems-level physiological changes and daily fluctuations that drive metabolic decline. This study illustrates how Generative AI and Large Language Model (LLM) can create a novel paradigm to bridge the gap between complex biochemical diagnostics and individualized patient education by combining LLM with multi-system biomarker analysis to assess divergent physiological dynamics between two different metabolic phenotypes (early subclinical prediabetes [34-year-old female] vs. severe, decompensated overt Type 2 Diabetes Mellitus [49-year-old male]). The LLM pipeline synthesizes cell-state analyses of multi-system biomarker axes (e.g., glycemic dynamics, purine stress, atherogenic dyslipidemia, hematological parameters, micronutrients, and inflammation) that highlight the 1-to-3-year golden window of beta-cell reversibility in prediabetes, while establishing targeted non-dietary clinical imaging and cardiovascular risk stratification protocols for overt T2DM. Within the context of deterministic medical states, LLM frameworks transition endocrinological care from glucocentric symptom management to proactive, patient-engaged metabolic remission.

1. Introduction

Periodic laboratory monitoring of glycated hemoglobin (HbA1c) and fasting blood glucose are generally used to monitor insulin resistance and T2DM, but isolated glucose-centric measurements do not typically describe the multi-system biochemical crosstalk such as ectopic lipotoxicity, purine-mediated mitochondrial oxidation, and

endothelial dysfunction that can progressively increase metabolic disease [1,2]. This disconnect is a major contributor to lifestyle non-adherence and delayed therapeutic intervention.

In clinical practice, 25% to 30% of patients with prediabetes go on to develop overt T2DM in 3 to 5 years [3,4], and within 1 to 3 years of diagnosis, there is a "golden window" of cellular plasticity where dysfunctional pancreatic beta cells can be

repaired via weight loss (5% to 10%), nutritional restructuring, and cofactor repletion [5,6]. Moreover, in T2DM patients, ectopic fat loss from the liver and pancreas can restore beta-cell insulin secretion [7].

Recent advances in Transformer architectures and Large Language Models (LLMs) have opened applications in context-aware clinical decision support [8], zero-shot biochemical reasoning, and customized conversational health engagement [9,10,11]. Retrieval-Augmented Generation (RAG) architectures allow LLMs to translate complex, multi-system diagnostic panels into simple, literacy-adapted cell-state explanations that empower patients to alter their behavior. The Table 1 provides an overview of LLM based medical research

2. Generative AI & Prompting

Prompt engineering is used to perform intermediate biochemical calculations (e.g., checking the non-linearity of HOMA-IR under severe glucotoxicity, computing TG/HDL atherogenic ratios, and

assessing non-linear purine clearance) before arriving at clinical interpretations. Boundary were set for deterministic fallback, and pathological extreme values (e.g., serum uric acid > 15 mg/dL or fasting blood glucose > 200 mg/dL) were automatically flagged to trigger mandatory clinical verification alerts for secondary renal impairment or acute glucotoxicity. A comprehensive analysis is done on patient blood reports considering multi-system biomarker matrix and pathophysiological roles given in Table 2.

3. Comprehensive Multi-System Biomarker Matrix & Mechanistic Pathways

Metabolic syndrome is a self-reinforcing systems-level disorder [19] where nutrient oversupply, purine-mediated hyperuricemic oxidative stress, and atherogenic dyslipidemia converge on cellular insulin signaling pathways. To capture this crosstalk, our framework evaluates a multi-system biomarker matrix spanning six physiological axes.

Table 1. State-of-the-Art Scientific Literature at the AI-Endocrinology Nexus

Author(s) & Year	Key Focus & Research Domain	Primary Findings & Methodological Contribution
Bak & Chin (2024) [12]	LLMs & Behavior Change	Analyzed LLM efficacy in identifying motivational states to facilitate cardiometabolic health behavior change.
Furtado et al. (2025) [13]	Conversational Agents	Evaluated user experience and clinical efficacy of conversational AI assistants in T2DM self-management.
Gaitán-Guerrero et al. (2025) [14]	IoT & Sensor Fine-Tuning	Introduced LLM-RawDMeth methodology for fine-tuning LLMs on raw IoT and continuous glucose monitoring data.
Healey et al. (2025) [15]	CGM Trace Interpretation	Demonstrated capability of LLMs to parse, interpret, and contextualize complex continuous glucose monitoring traces.
Lee et al. (2024) [9]	Dual-RAG & Hallucination Guardrails	Implemented dual retrieval-augmented generation grounded in clinical guidelines to eliminate generative hallucinations.
Mashatian et al. (2024) [16]	Biomedical Extraction	Designed trustworthy generative AI extraction frameworks tailored for diabetic limb preservation and decision support.
Ong et al. (2025) [10]	Clinical Decision Support	Evaluated LLM clinical decision support across 16 medical specialties, improving medication safety and diagnostics.
Sridhar & Gumpeny (2025) [11]	Generative AI in Diabetes	Analyzed clinical utility, conversational engagement benefits, and algorithmic safeguards for deploying ChatGPT in diabetes management.
Summers (2018) [17]	Lipotoxicity & Sphingolipids	Elucidated ceramide-mediated inhibition of Akt signaling and mitochondrial fission in skeletal muscle and liver.
Zhao et al. (2026) [18]	Behavioral Interventions	Reviewed content design and implementation of LLM-driven conversational agents targeting cardiometabolic modification.

Table 2. Multi-System Biomarker Matrix and Pathophysiological Roles

Physiological Axis	Key Biomarkers & References	Primary Pathophysiological Role
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Glycemic Dynamics	HbA1c, Fasting Glucose, Fasting Insulin, C-Peptide, HOMA-IR Target: HOMA-IR < 1.0, HbA1c 4.0-5.6%	Quantifies peripheral receptor desensitization versus compensatory beta-cell strain and secretory reserve capacity.
Purine & Metabolic Stress	Serum Uric Acid (SUA) Target: 3.5-7.2 mg/dL (Metabolic target ≤ 5.5 mg/dL)	Pro-oxidant generated by hepatic fructose catabolism; activates NADPH oxidase (NOX4) and quenches endothelial nitric oxide (eNOS).
Cardiovascular / Lipid	Total Cholesterol, TG, HDL-C, LDL-C, VLDL-C, Lp(a), TG/HDL Ratio Target: TG < 150 mg/dL, TG/HDL < 3.0	Evaluates hepatic de novo lipogenesis, atherogenic small dense LDL (Pattern B sdLDL), and genetically fixed thrombotic risk.
Hematology	RBC Count, Packed Cell Volume (PCV), RDW Target: PCV 40-50%, RDW 11.6-14.0%	Identifies secondary compensatory erythrocytosis (chronic hypoxia/OSA), whole-blood hyperviscosity, and systemic metabolic strain.
Micronutrients	25-OH Vitamin D, Serum Iron Target: Vit D 30-100 ng/mL, Iron 70-180 µg/dL	Nuclear VDR transcription cofactors for insulin receptor expression and mitochondrial respiratory chain electron transport.
Metainflammation	High-Sensitivity CRP (hs-CRP) Target: < 5.0 mg/L	Differentiates systemic acute-phase hepatic response from silent, local tissue metainflammation (metabolic endotoxemia).

Table 3. Side-by-Side Baseline Laboratory & Biomarker Comparison

Physiological Axis	Biomarker / Laboratory Test	Diabetic Subject (Male, 49)	Prediabetic Subject (Female, 34)	Biological Reference / Target
Glycemic Dynamics	HbA1c (HPLC)	10.4% (Severely High)	5.8% (Prediabetic)	4.0 - 5.6%
Glycemic Dynamics	Fasting Glucose	242.0 mg/dL (High)	96.0 mg/dL (Normal)	70 - 100 mg/dL
Glycemic Dynamics	Fasting Insulin	6.0 µIU/mL (Relative Deficiency)	6.6 µIU/mL (Optimal)	2.0 - 25.0 µIU/mL
Glycemic Dynamics	HOMA-IR (Calculated)	3.59 (Severe Glucotoxicity)	1.56 (Mild Resistance)	< 1.0 (Optimal)
Glycemic Dynamics	C-Peptide	2.60 ng/mL (Preserved Secretion)	2.14 ng/mL (Preserved Secretion)	1.1 - 4.4 ng/mL
Purine Stress	Serum Uric Acid	17.9 mg/dL (Extreme Hyperuricemia)	4.5 mg/dL (Normal)	3.5 - 7.2 mg/dL
Lipid / Vascular	Total Cholesterol	309.0 mg/dL (High)	182.0 mg/dL (Normal)	< 200 mg/dL
Lipid / Vascular	Triglycerides (TG)	339.0 mg/dL (High)	94.0 mg/dL (Optimal)	< 150 mg/dL
Lipid / Vascular	HDL / VLDL Cholesterol	HDL: 56 / VLDL: 67.8 mg/dL	HDL: 48 / VLDL: 18.8 mg/dL	HDL > 40 / VLDL < 30
Lipid / Vascular	Lipoprotein(a) [Lp(a)]	> 90.0 mg/dL (High Genetic Risk)	18.0 mg/dL (Normal)	< 30.0 mg/dL

Lipid / Vascular	TG / HDL Ratio	6.05 (Pattern B sdLDL)	1.96 (Normal Pattern A)	< 3.0 (Optimal)
Hematology	RBC / Packed Cell Vol.	RBC: 5.76 / PCV: 51.0%	RBC: 4.21 / PCV: 38.5%	RBC: 4.5-5.5 / PCV: 40-50%
Hematology	RDW	14.3% (Elevated Strain)	12.1% (Normal)	11.6 - 14.0%
Micronutrients	25-OH Vitamin D	14.20 ng/mL (Deficient)	18.50 ng/mL (Deficient)	30.0 - 100.0 ng/mL
Inflammation	hs-CRP	< 5.0 mg/L (Normal Systemic)	< 1.2 mg/L (Normal Systemic)	< 5.0 mg/L

Table 4: Advanced Diagnostic & Clinical Follow-Up Stratification Panel for overt diabetic patient

Diagnostic Category	Recommended Investigation	Physiological Target & Clinical Rationale
Cardiovascular Imaging	Coronary Artery Calcium (CAC) Scan	Quantifies calcified coronary plaque burden to accurately calibrate 10-year absolute cardiovascular risk in high Lp(a) profiles (Kronenberg et al., 2022).
Vascular Assessment	Carotid Intima-Media Thickness (CIMT)	Screens for subclinical carotid atheromatous plaques and accelerated endothelial remodeling driven by Pattern B sdLDL.
Valvular Screening	Transthoracic Echocardiogram	Screens for early aortic valve calcification and stenosis, strongly driven by genetically high Lp(a) (> 90 mg/dL).
Hepatic Stratification	Transient Elastography (FibroScan)	Quantifies hepatic fat fraction (CAP score) and detects early bridging fibrosis associated with NAFLD/NASH and SREBP-1c lipogenesis.
Renal Monitoring	Urinary Albumin-to-Creatinine Ratio (UACR)	Screens for early microvascular glomerular barrier breakdown, accelerated by severe hyperuricemia (17.9 mg/dL) and intraglomerular hypertension.
Nocturnal Screening	Overnight Polysomnography (Sleep Study)	Confirms Obstructive Sleep Apnea (OSA) as the root cause of secondary compensatory erythrocytosis (PCV 51.0%, RBC 5.76).

3.1 Molecular Mechanisms of Metabolic Disruption

In hyperinsulinemic states, DAGs and ceramides accumulate intracellularly and activate stress kinases (PKC θ , PKC ϵ , JNK), inhibitory IRS-1 serine phosphorylation, and preventing Akt membrane translocation [17].

Purine stress and endothelial dysfunction: Fructose metabolism in the liver leads to the production of uric acid via ketohexokinase (KHK), which is not subject to feedback inhibition, and uses ATP for the process. Uric acid stimulates mitochondrial NOX4, resulting in ROS, activation of the NLRP3 inflammasome (IL-1 β , IL-18 release), and eNOS uncoupling, leading to vasoconstriction.

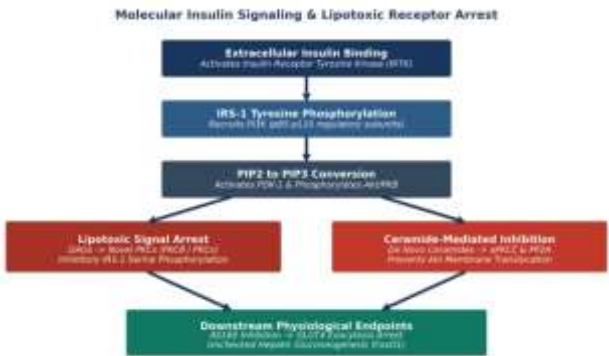


Figure 1. Molecular insulin signaling and lipotoxic receptor arrest

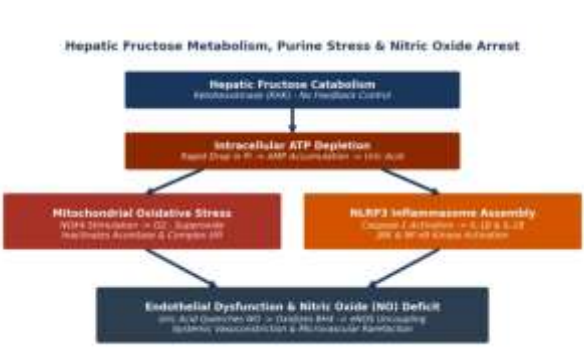


Figure 2. The Hepatic Fructose Metabolism that influence purine stress and nitric oxide arrest

Pattern B sdLDL remodeling: Hyperinsulinemia triggers hepatic SREBP-1c, which increases de novo lipogenesis and VLDL overproduction. Cholesteryl ester transfer protein (CETP) exchanges TG from VLDL with LDL and HDL, and hepatic lipase hydrolysis reduces LDL particle volume, resulting in highly atherogenic small dense LDL (sdLDL / Pattern B) [20].

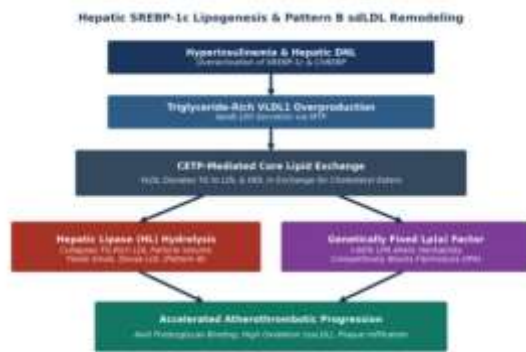


Figure 3. Hepatic SREBP-1c Lipogenesis and Pattern B sdLDL Remodeling

4. Comparative Clinical Case Analyses

To demonstrate the analytical precision of the LLM pipeline across divergent clinical presentations, two representative metabolic cases were processed: a 49-year-old male presenting with overt decompensated Type 2 Diabetes Mellitus, and a 34-year-old female presenting with early subclinical prediabetes (Table 3).

4.1 Molecular Mechanisms of Metabolic Disruption analysis using LLM

A 49-year-old male patient with chronic hyperglycemia (HbA1c 10.4%, fasting blood glucose 242 mg/dL), a calculated HOMA-IR of 3.59 (indicating severe insulin resistance, but which standard linear Homeostatic Model Assessment [21] underestimates when acute glucotoxicity occurs), a fasting insulin level of 6.0 IU/mL (within standard laboratory reference limits, 2.0–25.0 IU/mL) indicating severe relative beta-cell exhaustion (with ambient glucose levels above 240 mg/dL), and preserved C-peptide concentrations (2.60 ng/mL) confirming intact endogenous insulin secretory capacity, preventing primary beta-cell failure or autoimmune destruction, and demonstrating that beta-cell function can be recovered with relief of glucolipotoxicity, is decompensated type 2 diabetes mellitus (chronic hyperglycemia: HbA1c 10.4%, fasting blood glucose 242 mg/dL), and a preserved C-peptide concentration of 2.60 ng/mL (within

standard laboratory reference limits, 2.0–25.0 IU/mL) indicating severe relative beta-cell exhaustion (with ambient glucose levels above 240 mg/dL) and demonstrating that beta-cell function can be recovered with relief of glucolipotoxicity.

At the same time, the patient has severe hyperuricemia (17.9 mg/dL) well above the saturation threshold for monosodium urate crystallization (6.8 mg/dL), strongly suggesting that renal underexcretion is involved (uric acid concentrations this high are typically a consequence of both hepatic de novo purine synthesis driven by unregulated fructose catabolism [ketohexokinase pathway] and secondary to hyperinsulinemia-stimulated renal proximal tubular URAT1/OAT4 reabsorption or early subclinical diabetic nephropathy. Uric acid is also an effective intracellular pro-oxidant that activates mitochondrial NADPH oxidase (NOX4), reduces ATP, and activates the NLRP3 inflammasome (IL-1 β , IL-18 release), which directly leads to uncoupling of endothelial nitric oxide synthase (eNOS) and increased vascular shear strain, increasing systemic insulin resistance.

Dyslipidemia is atherogenic (TG/HDL = 6.05 [highly predictive of Pattern B sdLDL predominance], VLDL = 67.8 mg/dL), and there is a very high Lipoprotein(a) [Lp(a) > 90 mg/dL], a genetically determined, highly atherogenic particle that competes with plasminogen to inhibit fibrinolysis [20]. Secondary compensatory erythrocytosis (high RBC [5.76 mil/cumm] and PCV [51.0%]) is typical of nocturnal hypoxia from OSA or systemic hemoconcentration.

4.2 Mechanistic Analysis of Prediabetic Cellular Plasticity (Female, 34)

For example, a 34-year-old woman with early subclinical insulin resistance (HbA1c 5.8%, fasting glucose 96 mg/dL, HOMA-IR 1.56), preserved lipid dynamics (TG 94 mg/dL, TG/HDL ratio 1.96, Lp(a) 18 mg/dL), and serum uric acid (4.5 mg/dL) but severe Vitamin D deficiency (18.50 ng/mL), which inhibits nuclear Vitamin D Receptor (VDR) transcription for insulin receptor expression and Calbindin-D28k beta-cell buffering, is an example of the 1-to-3-year golden window of prediabetes: the pancreatic beta-cell mass and insulin sensitivity are fully recoverable with dietary carbohydrate reduction, resistance exercise, and Vitamin D3 repletion before irreversible glucolipotoxic beta-cell apoptosis sets in.

4.2 Clinical Decision Support & Advanced Investigation Framework

For advanced T2DM presenting with severe metabolic collapse and unmodifiable genetic risk (Lp(a) > 90 mg/dL), the LLM framework shifts from routine glucose tracking to multi-system risk stratification. The following non-dietary clinical imaging and follow-up protocol is automatically synthesized by the LLM pipeline as presented in Table 4.

5. Conclusions

This breakthrough in endocrinology relies on Large Language Models (LLM), such as Gemini 2.0, which transcends single axis glucocentric monitoring by evaluating multi-system biomarker crosstalk across glycemic, purine, lipid, hematological, micronutrient, and inflammatory axes to facilitate the critical communication between complex biochemical data and actionable patient understanding. For the early prediabetes stage, LLM-based cell-state synthesis highlights the 1-to-3-year window of beta-cell reversibility, which drives disease remission through targeted lifestyle modification. For advanced, decompensated T2DM, the pipeline provides clinicians with expert decision support to identify the hidden drivers of disease progression, including relative beta-cell exhaustion, hyperuricemic renal strain, and high genetic cardiovascular risk [Lp(a)], and automatically generates comprehensive non-dietary imaging protocols. Next steps include prospective trial validation to assess patient behavioural adherence and long-term cardiometabolic outcome improvements.

Author Statements:

- **Ethical approval:** The conducted research is not related to either human or animal use.
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References

- [1] Johnson, R. J., Sánchez-Lozada, L. G., Andrews, P., & Lanaspa, M. A. (2017). Perspective: A historical and scientific perspective of sugar and its relation with obesity and diabetes. *Advances in Nutrition*, 8(3), 412–422. <https://doi.org/10.3945/an.116.014654>
- [2] Matthews, D. R., Hosker, J. P., Rudenski, A. S., Naylor, B. A., Treacher, D. F., & Turner, R. C. (1985). Homeostasis model assessment: insulin resistance and beta-cell function from fasting plasma glucose and insulin concentrations in man. *Diabetologia*, 28(7), 412–419. <https://doi.org/10.1007/BF00280883>
- [3] Tabák, A. G., Herder, C., Rathmann, W., Brunner, E. J., & Kivimäki, M. (2012). Prediabetes: a high-risk state for diabetes development. *The Lancet*, 379(9833), 2279–2290. [https://doi.org/10.1016/S0140-6736\(12\)60283-9](https://doi.org/10.1016/S0140-6736(12)60283-9)
- [4] Knowler, W. C., Barrett-Connor, E., Fowler, S. E., Hamman, R. F., Lachin, J. M., Walker, E. A., & Nathan, D. M. (2002). Reduction in the incidence of type 2 diabetes with lifestyle intervention or metformin. *New England Journal of Medicine*, 346(6), 393–403. <https://doi.org/10.1056/NEJMoa012512>
- [5] Tuomilehto, J., Lindström, J., Eriksson, J. G., Valle, T. T., Hämäläinen, H., Ilanne-Parikka, P., ... & Uusitupa, M. (2001). Prevention of type 2 diabetes mellitus by changes in lifestyle among subjects with impaired glucose tolerance. *New England Journal of Medicine*, 344(18), 1343–1350. <https://doi.org/10.1056/NEJM200105033441801>
- [6] Lean, M. E., Leslie, W. S., Barnes, A. C., Brosnahan, N., Thom, G., McCombie, L., ... & Taylor, R. (2018). Primary care-led weight management for remission of type 2 diabetes (DiRECT): an open-label, cluster-randomised trial. *The Lancet*, 391(10120), 541–551. [https://doi.org/10.1016/S0140-6736\(17\)33102-1](https://doi.org/10.1016/S0140-6736(17)33102-1)
- [7] Taylor, R., Al-Mrabeh, A., Zhyzhneuskaya, S., Peters, C., Barnes, A. C., Aribisala, B. S., Hollingsworth, K. G., Mathers, J. C., Sattar, N., & Lean, M. E. (2018). Remission of human Type 2 diabetes requires decrease in liver and pancreas fat content but is dependent upon capacity for β cell recovery. *Cell Metabolism*, 28(4), 547–556. <https://doi.org/10.1016/j.cmet.2018.07.003>
- [8] Thirunavukarasu, A. J., Ting, D. S. J., Elangovan, K., Gutierrez, L., Tan, T. F., & Ting, D. S. W. (2023). Large language models in medicine. *Nature Medicine*, 29(8), 1930–1940. <https://doi.org/10.1038/s41591-023-02448-8>
- [9] Lee, J., Cha, H., Hwangbo, Y., & Cheon, W. (2024). Enhancing large language model reliability: Minimizing hallucinations with dual retrieval-augmented generation based on the latest diabetes guidelines. *Journal of Personalized Medicine*, 14(12), 1131. <https://doi.org/10.3390/jpm14121131>
- [10] Ong, J. C. L., Liyuan, J., Elangovan, K., Lim, G. Y. S., Lim, D. Y. Z., Sng, G. G. R., ... & Ting, D. S. W. (2025). Large language model as clinical decision

- support system augments medication safety in 16 clinical specialties. SSRN Electronic Journal. <https://doi.org/10.2139/ssrn.5100060>
- [11] Sridhar, G. R., & Gumpeny, L. (2025). Prospects and perils of ChatGPT in diabetes. *World Journal of Diabetes*, 16(3), 98408. <https://doi.org/10.4239/wjd.v16.i3.98408>
- [12] Bak, M., & Chin, J. (2024). The potential and limitations of large language models in identification of the states of motivations for facilitating health behavior change. *Journal of the American Medical Informatics Association*, 31(9), 2047–2053. <https://doi.org/10.1093/jamia/ocae057>
- [13] Furtado, V., Araujo, J., Furtado, E. S., Bomfim, R., Silva, V., Mororó, N., ... & Freitas, A. B. (2025). Assessing the user experience of an LLM-based conversational assistant in diabetes mellitus care. *Journal of Healthcare Informatics Research*, 10(1), 116–153. <https://doi.org/10.1007/s41666-025-00217-5>
- [14] Gaitán-Guerrero, J. F., Martínez-Cruz, C., Espinilla, M., Díaz-Jiménez, D., & López, J. L. (2025). A novel fine-tuning and evaluation methodology for large language models on IoT raw data summaries (LLM-RawDMeth): A joint perspective in diabetes care. *Computer Methods and Programs in Biomedicine*, 269, 108878. <https://doi.org/10.1016/j.cmpb.2025.108878>
- [15] Healey, E., Tan, A. L. M., Flint, K. L., Ruiz, J. L., & Kohane, I. (2025). A case study on using a large language model to analyze continuous glucose monitoring data. *Scientific Reports*, 15(1), 1143. <https://doi.org/10.1038/s41598-024-84003-0>
- [16] Mashatian, S., Armstrong, D. G., Ritter, A., Robbins, J., Aziz, S., Alenabi, I., ... & Tavakolian, K. (2024). Building trustworthy generative artificial intelligence for diabetes care and limb preservation: A medical knowledge extraction case. *Journal of Diabetes Science and Technology*, 19(5), 1264–1270. <https://doi.org/10.1177/19322968241253568>
- [17] Summers, S. A. (2018). Could ceramides become the new cholesterol? *Cell Metabolism*, 27(2), 276–280. <https://doi.org/10.1016/j.cmet.2017.12.003>
- [18] Zhao, Y., Guo, R., Miao, Y., Luo, Y., Wang, H., & Wu, Y. (2026). Behavior change content and implementation of large language model-driven conversational agents in cardiometabolic care: Scoping review. *Journal of Medical Internet Research*, 28, e89190. <https://doi.org/10.2196/89190>
- [19] Petersen, M. C., & Shulman, G. I. (2018). Mechanisms of insulin action and insulin resistance. *Physiological Reviews*, 98(4), 2133–2223. <https://doi.org/10.1152/physrev.00063.2017>
- [20] Kronenberg, F., Mora, S., Stoes, E. S., Ference, B. A., Arsenault, B. J., Berglund, L., ... & Catapano, A. L. (2022). Lipoprotein(a) in clinical practice: European Atherosclerosis Society consensus statement. *European Heart Journal*, 43(39), 3925–3946. <https://doi.org/10.1093/eurheartj/ehac361>
- [21] Wallace, T. M., Levy, J. C., & Matthews, D. R. (2004). Use and abuse of HOMA modeling. *Diabetes Care*, 27(6), 1487–1495. <https://doi.org/10.2337/diacare.27.6.1487>