Conclusion: GDEs facilitate the transfer of key lipids and proteins between the gut and liver, potentially detoxifying harmful lipids by directing them to KCs. In prediabetes, impaired KC detoxification may increase the risk of hepatic metabolic disturbances. Additionally, our findings suggest that treatment with metformin and pioglitazone improves metabolic health by modifying GDEs' content, highlighting their potential as early biomarkers and underscoring their role in evaluating the effectiveness of antidiabetic therapies.

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Research overview on FOXO1 and its role in gestational diabetes mellitus through epigenetic lens: bibliometric analysis

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Background: Epigenetic studies showed various gene methylation in the placenta of women with gestational diabetes mellitus (GDM), which were connected to pathways for the metabolism of glucose and energy including FOXO1. Disruption of its expression has a profound effect on the pathogenesis of metabolic disorder. However, a global review of the development of FOXO1 methylation research in relation to GDM has not been conducted.

Aim: To explore FOXO1 methylation research and the occurrence of GDM by using bibliometric analysis.

Method: Bibliometric approach guided by PRISMA framework was applied to map the articles published in 2008-2023 from Scopus database using the keywords "methylation AND ("pregnant women" OR pregnancy) AND (diabetes OR "gestational diabetes")" and "FOXO1 AND Diabetes" (accessed May 20, 2024).

Results: A significant increase in the number of articles discussing DNA methylation and gestational diabetes (441 articles), FOXO1 and diabetes (887 articles) observed since 2008 to 2023. Network visualization of 441 articles revealed 4 clusters: epigenetic, pregnancy, gestational diabetes, and DNA methylation, while the 887 articles produced 4 clusters: type 2 diabetes, FOXO1, diabetes, and gluconeogenesis. DNA methylation closely tied to GDM, but no studies explored FOXO1 role in this process. FOXO1 strongly linked to diabetes but indirectly associated with GDM. Research on methylation has shifted toward cell-free DNA, neonatal outcomes, and macrosomia, while FOXO1 studies remain focused on cell signaling. These finding suggested further research potential.

Conclusion: Increasing studies on the association of DNA methylation with GDM, but no research has specifically linked FOXO1 methylation with GDM. Current trends show growing interest examining the long-term impact of GDM on offspring.

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Exploring the role of urokinase on exercise-induced irisin expression in obese and type 2 diabetes mouse model

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Background: Our previous research identified urokinase plasminogen activator (uPA) as a key factor in type 2 diabetes. Exercise promotes metabolic health by maintaining biochemical balance, reducing adipocyte size, and increasing irisin, a myokine that enhances insulin sensitivity and induces the browning of white adipose tissue (WAT).

Aim: This study investigated the role of uPA in muscle and adipose tissue responses during exercise in an obese and type 2 diabetes mouse model.

Method: Male wild-type and uPA-/- BLAB/c mice were fed either a chow or high-fat diet (HFD) from 5 weeks old. The type 2 diabetes group received streptozotocin and nicotinamide after high-fat feeding. Mice were split into exercise and sedentary groups. The exercise group performed treadmill running (40 min/day at 20 m/min, 5 days/week). At 12 weeks, blood and tissues (pancreas, BAT, WAT, and muscle) were collected.

Results: HOMA-IR increased in sedentary HFD wild-type mice but decreased after exercise, with no reduction in diabetic wild-type mice. In uPA-/- mice, HOMA-IR decreased, indicating lower insulin secretion. Serum irisin levels increased with exercise in all groups but less so in uPA-/- mice. In wild-type mice, muscle PGC1α and FNDC5 expression increased post-exercise, but not in diabetic mice, suggesting hyperglycemia inhibits these pathways. In BAT, PGC1α, FNDC5, and UCP-1 expression increased in exercised wild-type mice but not in diabetics. In uPA-/- mice, FNDC5 and UCP-1 increased in HFD BAT, but PGC1α and GLUT4 did not change. In WAT, PGC1α, FNDC5, and UCP-1 increased in type 2 diabetic uPA-/- mice. Visceral WAT in wild-type mice showed poor response to exercise, and uPA-/- mice had no significant marker changes.