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A novel function of Fibroblast Growth Factor 21(FGF21) in combating inflammatory diseases

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FGF21 has been widely accepted as a regulator for glucose and lipid metabolism. A recent report indicated that circulating FGF21 levels are elevated in the serum and synovial fluid of patients with Rheumatoid Arthritis (RA), but the role of FGF21 in the development of RA is totally unknown. The aim of this study is to investigate efficacy of FGF21for treatment of RA and the molecular mechanisms underline the therapeutic effect on collagen-induced arthritis (CIA). Mice with CIA were subcutaneously administered with FGF21 (5, 2 or 1 mg·kg^{-1}·d^{-1}), IL-1β antibody (5 mg·kg^{-1}·d^{-1}), IL-17A antibody (5 mg·kg^{-1}·d^{-1}) and dexamethasone (DEX) (1 mg·kg^{-1}·d^{-1}), respectively. The effects of treatment were determined by arthritisseverity score, histological damage and cytokine production. The activation of NF-κB was analyzed by Western blotting. We also detected the levels of oxidative stress parameters. Our results showed that FGF21 had beneficial effects on clinical symptom and histological lesion of CIA mice. Similar to antibody and DEX, FGF21 treatment alleviated the severity of arthritis by reducing humoral and cellular immune responses and down-regulating the expression of pro-inflammatory cytokines. FGF21 treatment also reduced the expression of TNF-α, IL-1β, IL-6, IFN-γ and MMP-3 and increased level of IL-10 in the spleen tissue or the plasma of CIA mice in a dose-dependent manner. Furthermore, FGF21 inhibited IκBα degradation and NF-κB p65 nuclear translocation and induced significant changes of oxidative stress parameters (MDA, SOD, CAT, GSH-PX and GSH) in the plasma. We therefore conclude that FGF21 exerts the therapeutic efficacy for RA through antioxidant reaction and inhibiting NF-κB inflammatory pathway. This study provides evidence that FGF21 may be a promising therapeutic agent for RA patients.

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Risk factors associated with early postpartum abnormal glucose tolerance in Japanese women with Gestational Diabetes

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Objective: Gestational Diabetes Mellitus (GDM) is associated with a significantly increased risk of developing Diabetes later in life. We identified the risk factors associated with abnormal glucose tolerance (AGT) on the first postpartum glucose tolerance test (OGTT) in Japanese women with GDM.

Research Design & Methods: We included Gestational diabetic women who underwent their first postpartum OGTT at 6-8 weeks postpartum. We defined the combination of impaired glucose tolerance and Diabetes as postpartum AGT according to the WHO criteria. We investigated the association between postpartum AGT and risk factors including maternal age, prepregnancy BMI, insulin therapy during pregnancy, plasma glucose (PG) levels, HbA1c, fasting immunoreactive insulin (IRI), the insulinogenic index (II), homeostasis assessment model (HOMA)-insulin resistance, and HOMA-ß at diagnosis of GDM during pregnancy.

Results: We investigated 169 women with GDM, who underwent OGTT at 6.9±1.5 weeks postpartum. Fifty-eight women (34%) exhibited postpartum AGT. In a univariate analysis, 1-hour PG ($p<0.005$), HbA1c ($p<0.001$), II ($p<0.05$), and insulin therapy ($p<0.001$) were associated with postpartum AGT. After adjusting for confounding variables, II ($p<0.005$) and insulin therapy ($p<0.005$) were found to be independent risk factors associated with postpartum AGT. The adjusted odds ratios for postpartum AGT in women with II<0.4 and women treated with insulin therapy were 5.7 (95% confidence interval, 1.69-21.66) and 3.43 (1.03-12.55), respectively.

Conclusion: In Japanese Gestational diabetic women, a lower II, a marker of early-phase insulin secretion, and the use of insulin therapy during pregnancy were independent risk factors predicting early postpartum AGT.

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