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Benzyl isothiocyanate (BITC) improves insulin resistance induced by palmitic acid and reduces gluconeogenic gene expression in mice primary hepatocytes

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Insulin resistance is closely associated with type 2 diabetes, obesity and a number of chronic diseases such as hyperlipidemia, hypertension and cardiovascular diseases. Palmitic acid (PA) is the most abundant dietary and plasma saturated fatty acid. Increased plasma free fatty acid, especially the PA is known to be one of the key risk factors in the induction of insulin resistance. BITC is organosulfur phytochemical rich in cruciferous vegetables and has been shown to possess anti-cancer, anti-inflammatory and antioxidant properties. However, it remains unclear whether BITC improves insulin resistance. This study aimed to examine the protection of BITC against PA-induced insulin resistance in primary mouse hepatocytes. Results showed that BITC reversed PA-induced insulin resistance by decreasing the phosphorylation of IRS (Tyr632) and increasing the phosphorylation of Akt (Thr308). BITC increases glycogen synthesis by increasing phosphorylation of GSK3β(Ser9) and reducing GS(Ser641) phosphorylation. In addition, BITC increases the phosphorylation of FOXO1 (Thr24), which leads to inhibiting glucose-6-phosphatase (G6pc) and phosphoenolpyruvate carboxykinase 1 (Pck1) mRNA expression, respectively. These findings suggest that BITC improves PA-induced hepatic insulin resistance and this effect is likely to dependent on the IRS/AKT/GSK3β/GS and IRS/AKT/FOXO1 signal pathway. Consequently, BITC promoted glycogenesis and suppressed gluconeogenesis in hepatocytes.

Biography

Wei-Ting Tsai is currently pursuing his Masters in the Department of Nutrition from the China Medical University in Taiwan.

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