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Isothiocyanates improve hepatic insulin sensitivity through inhibiting NLRP3 activation in Kupffer cells

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 \mathbf{N} LRP3 inflammasome plays an important role in innate immunity and is well known to be associated with the progression of numerous inflammatory diseases including type 2 diabetes, alcoholic and non-alcoholic steatohepatitis, atherosclerosis and gout. Benzyl isothiocyanate (BITC) and phenethyl isocyanate (PEITC), rich in cruciferous vegetables, possess antiinflammatory, antioxidant, hypoglycemic and anti-cancer properties. In this study, we intend to examine the role of Kupffer cells in BITC and PEITC improvement of hepatic insulin resistance. Primary kupffer cells and hepatocytes were isolated from Sprague-Dawley rats. Results showed that BITC and PEITC attenuated LPS/nigericin-induced NLRP3 inflammasome activation and IL-1 β secretion. Conditioned medium from Kupffer cells exposed to LPS/nigericin and recombinant IL-1 β downregulated hepatic insulin signaling as evidenced by inhibiting Akt phosphorylation as well as increased phosphoenolpyruvate carboxylase (PEPCK) expression. In the presence of BITC and PEITC, changes in hepatic Akt phosphorylation and PEPCK expression induced by IL-1 β were reversed. These findings suggest that the anti-diabetic potency of BITC and PEITC is likely to be associated with their inhibition of NLRP3 inflammasome activation in Kupffer cells, which leads to improve insulin sensitivity and suppress gluconeogenesis in hepatocytes.

Biography

Chong-Kuei Lii has completed his PhD from the Department of Food Science and Human Nutrition of Iowa State University, USA. He is a Professor of the Department of Nutrition of China Medical University in Taiwan. His research interests include exploring biological activities of herb and food components as well as herb and drug interactions. He has published more than 100 papers in reputed journals.

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