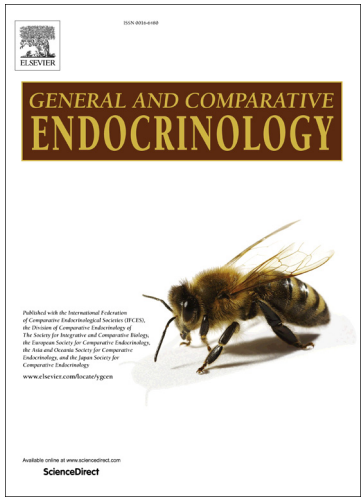

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Egr2 enhances insulin resistance via JAK2/STAT3/SOCS-1 pathway in HepG2 cells treated with palmitate

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Abstract

Insulin resistance is generally responsible for the pathogenesis of type 2 diabetes mellitus (T2DM). Early growth response proteins-2 (Egr2) has been reported to be able to increase the expression of the suppressors of cytokine signaling-1 (SOCS-1), and impair insulin signaling pathway through suppression of insulin receptor substrates (IRS), including IRS-1 and IRS-2. However, whether Egr2 is directly involved in the development of insulin resistance, and how its potential contributions to insulin resistance still remain unknown. Here, our present investigation found that the expression levels of Egr2 were up-regulated when insulin resistance occurs, and knockdown of Egr2 abolished the effect of insulin resistance in HepG2 cells induced with palmitate (PA). Importantly,