Eur J Pharmacol. 2008 Dec 3;599(1-3):152-8. Epub 2008 Oct 9.

Glycine regulates the production of pro-inflammatory cytokines in lean and monosodium glutamate-obese mice.

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Source

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Abstract

Fat tissue plays an important role in the regulation of inflammatory processes. Increased visceral fat has been associated with a higher production of cytokines that triggers a low-grade inflammatory response, which eventually may contribute to the development of insulin resistance. In the present study, we investigated whether glycine, an amino acid that represses the expression in vitro of pro-inflammatory cytokines in Kupffer and 3T3-L1 cells, can affect in vivo cytokine production in lean and monosodium glutamate-induced obese mice (MSG/Ob mice). Our data demonstrate that glycine treatment in lean mice suppressed TNF-alpha transcriptional expression in fat tissue, and serum protein levels of IL-6 were suppressed, while adiponectin levels were increased. In MSG/Ob mice, glycine suppressed TNF-alpha and IL-6 gene expression in fat tissue and significantly reduced protein levels of IL-6, resistin and leptin. To determine the role of peroxisome proliferator-activated receptor-gamma (PPAR-gamma) in the modulation of this inflammatory response evoked by glycine, we examined its expression levels in fat tissue. Glycine clearly increased PPAR-gamma expression in lean mice but not in MSG/Ob mice. Finally, to identify alterations in glucose metabolism by glycine, we also examined insulin levels and other biochemical parameters during an oral glucose tolerance test. Glycine significantly reduced glucose tolerance and raised insulin levels in lean but not in obese mice. In conclusion, our findings suggest that glycine suppresses the pro-inflammatory cytokines production and increases adiponectin secretion in vivo through the activation of PPAR-gamma. Glycine might prevent insulin resistance and associated inflammatory diseases.

PMID: **18930730** [PubMed - indexed for MEDLINE]