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Activation of PPARalpha and PPARgamma by the GLP-1 receptor agonist, exendin-4, in HL-60 cells. The glucagon-like peptide-1 (GLP-1) agonist, exendin-4, is a potent modulator of lipoprotein metabolism and insulin secretion from pancreatic beta cells. One of the actions of exendin-4 is the stimulation of adipogenesis in preadipocytes. It is known that ligands for the peroxisome proliferator-activated receptors (PPARs) are potent activators of adipocyte differentiation. Therefore, we have investigated the activation of the PPARs by exendin-4. Exendin-4 caused a dose-dependent accumulation of nuclear protein complexes containing PPARalpha and PPARgamma as well as the coactivator, SRC-1, in HL-60 promyelocytic cells. These

complexes were displaceable by antiserum for human PPARalpha and PPARgamma but not by antiserum for human SRC-1. Treatment of HL-60 cells with exendin-4 up-regulated mRNA for PPARalpha and PPARgamma but not for SRC-1. Exendin-4-induced activation of PPARs and accumulation of SRC-1 could be inhibited by BAY j11-7384, an antagonist of the GLP-1 receptor. In contrast, BAY j10350, an inactive analogue of BAY j11-7384, did not inhibit these actions of exendin-4. Thus, exendin-4 is able to activate PPARalpha and PPARgamma in HL-60 cells and may be a useful tool to elucidate the role of PPARs in adipogenesis and other biological processes that are related to exendin-4.

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