Peroxisome proliferator-activated receptor gamma

Peroxisome proliferator-activated receptor gamma (**PPAR-γ** or **PPARG**), also known as the **glitazone receptor**, or **NR1C3** (nuclear receptor subfamily 1, group C, member 3) is a type II nuclear receptor that in humans is encoded by the *PPARG* gene.[2][3][4]

1 Tissue distribution

PPARG is mainly present in adipose tissue, colon and macrophages. Two isoforms of PPARG are detected in the human and in the mouse: PPAR-γ1 (found in nearly all tissues except muscle) and PPAR-γ2 (mostly found in adipose tissue and the intestine).[5]

2 Function

PPARG regulates fatty acid storage and glucose metabolism. The genes activated by PPARG stimulate lipid uptake and adipogenesis by fat cells. PPARG knockout mice fail to generate adipose tissue when fed a high-fat diet.[6]

This gene encodes a member of the peroxisome proliferator-activated receptor (PPAR) subfamily of nuclear receptors. PPARs form heterodimers with retinoid X receptors (RXRs) and these heterodimers regulate transcription of various genes. Three subtypes of PPARs are known: PPAR-alpha, PPAR-delta, and PPAR-gamma. The protein encoded by this gene is PPAR-gamma and is a regulator of adipocyte differentiation. Alternatively spliced transcript variants that encode different isoforms have been described.[7]

Many naturally occurring agents directly bind with and activate PPAR gamma. These agents include various polyunsaturated fatty acids like arachidonic acid and arachidonic acid metabolites such as certain members of the 5-Hydroxyicosatetraenoic acid and 5-oxo-eicosatetraenoic acid family, e.g. 5-oxo-15(S)-HETE and 5-oxo-ETE or 15-Hydroxyicosatetraenoic acid family including 15(*S*)-HETE, 15(*R*)-HETE, and 15(*S*)-HpETE.[8][9][10] The activation of PPAR gamma by these and other ligands may be responsible for inhibiting the growth of cultured human breast, gastric, lung, prostate and other cancer cell lines.[11]

3 Interactions

Peroxisome proliferator-activated receptor gamma has been shown to interact with:

- EDF1[12][13][14]
- EP300[15][16]
- HDAC3[15][17]
- MED1[16]
- NCOA3[16]
- NCOA4[18]
- NCOA2[16]
- NR0B2[19]
- PPARGC1A[20][21]
- RB1.[15]

4 Clinical relevance

PPAR-gamma has been implicated in the pathology of numerous diseases including obesity, diabetes, atherosclerosis, and cancer. PPAR-gamma agonists have been used in the treatment of hyperlipidaemia and hyperglycemia.[22] PPAR-gamma decreases the inflammatory response of many cardiovascular cells, particularly endothelial cells.[23] PPAR-gamma activates the PON1 gene, increasing synthesis and release of paraoxonase 1 from the liver, reducing atherosclerosis.[24]

Many insulin sensitizing drugs (namely, the thiazolidinediones) used in the treatment of diabetes target PPARG as a means to lower serum glucose without increasing pancreatic insulin secretion. Different classes of compounds which activate PPARgamma weaker than thiazolidinediones (the so-called "partial agonists of PPARgamma") are currently studied with the hope that such compounds would be still effective hypoglycaemic agents but with fewer side effects.[25][26]

A fusion protein of PPAR-γ1 and the thyroid transcription factor PAX8 is present in approximately one-third of follicular thyroid carcinomas, to be specific those cancers with a chromosomal translocation of

t(2;3)(q13;p25), which permits juxtaposition of portions of both genes.[27][28]

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