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PPAR β facilitates the differentiation of insulin-producing cells derived from mouse embryonic stem cells

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The development of diabetes was due to accelerated loss of functional β cells. Peroxisome proliferator-activated receptors L (PPARs) are potential targets in diabetes therapy, but their direct roles in the differentiation of embryonic stem (ES) cells into insulin-producing (IP) cells remained unclear. Here, we established a three step protocol for the differentiation of mouse ES cells into IP cells, and investigated whether PPARs could modulate the differentiation process. The co-expression of PPARs and insulin was confirmed at the terminal stage of differentiation by flow cytometry. Western blot showed that PPAR β upregulation was paralleled with differentiation of mouse ES cells into IP cells in a time-dependent manner. Moreover, our data demonstrated that PPAR β agonist L165041 significantly increased the ratio of differentiated IP cells, whereas PPAR β antagonists GSK0660 had the opposite inhibitory effect. Quantity RT-PCR analysis demonstrated that the gene expression level of insulin-1 and insulin-2 could be dramatically induced by L165041 and suppressed by GSK0660. However, the expression of glucagon, somatostatin and pancreatic polypeptide did not alter by both of them in the differentiation. Concomitantly, glucose-stimulated insulin secretion (GSIS) function of the IP cells in the presence of L165041 was significantly elevated, whereas the function was not affected by GSK0660 treatment. Taken together, PPAR β play a critical role in promoting the specific differentiation of ES cells into IP cells and enhancing the GSIS function of differentiated IP cells

Biography

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