

Abstract

During my diploma thesis I was working on the investigation of a newly identified gene. This gene was found in our lab using transcriptome analysis comparing preadipocytes and fully differentiated fat cells. During the differentiation of adipocytes in vitro the expression of this gene is strongly increasing similar to other genes known to be important for adipogenesis like PPAR (Peroxisome Proliferator Activated Receptor), C/EBP (CCAAT/Enhancer Binding Protein), ATGL (Adipose Triglyceride Lipase), KLF4 (Kruppel Like Factor 4) and many more. The search for homologies showed, that this gene might act as a Strictosidine synthase or be of importance in carbohydrate transport and metabolism. According to this information, we measured the glucose transport/import into adipocytes and in addition we also measured the fatty acid uptake of these cells, but our newly identified gene neither seems to be important for glucose nor for fatty acid transport into fat cells. Silencing of this newly identified gene in 3T3-L1 cells led to strongly impaired differentiation capacity, reduced amount of lipid droplets and decreased TG-content in this cells. Additionally, the expression of many genes relevant in adipogenesis was significantly decreased, such as PPAR, C/EBP, ATGL, aP2 (Adipocyte Protein 2), Fasn (Fatty Acid Synthase) and many more. MICO (Mutual Information and Correlation) analysis showed that there might be a correlation between the expression of our gene and PPAR. Using in vitro experiments, this prediction could be confirmed. PPAR agonists increased the expression of our gene whereas a PPAR antagonist inhibits the agonist induced increase of our gene. These results suggest that PPAR is a regulator of our newly identified gene. Experiments with mice showed that our gene is not regulated by the feeding condition, but strongly increased in obese mice. Taken together, these results show that our newly identified gene seems to be of high importance in the development of fat cells in vivo and in vitro.