Investigation of the gene expression pattern and the regulation of stearoyl-CoA desaturase 5 (SCD5)

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V. Zámbó[I], L. Szabó[I], M. Csala[I], É. Kereszturi[I]

[I]Semmelweis University, Department of Molecular Biology, Budapest, Hungary

Elevation of free fatty acid (FA) levels is a key component in the development of severe diseases. The cellular stress caused by saturated FA overload can be reduced by local desaturation. Thus, the stearoyl-CoA desaturase (SCD1) enzyme is an important member in the cellular defense mechanism against lipotoxicity. The function and the regulation of Scd1 are well characterized, but SCD5, the other human isoform is barely been studied yet. The present work aimed to reveal, whether both SCD5 transcriptional variants (A and B) described in NCBI database were transcribed in human tissues and, if so, in what extent. We also aimed to identify the promoter region of SCD5 gene.The total SCD5 gene expression of hepatic and renal cell lines and eight different human tissues was assessed by RT-PCR. Specific primer pairs were designed to quantify the two transcriptional variants separatelly by qPCR. To analyze SCD5 promoter region, four fragments of different length were amplified from human genomic DNA and cloned into the pGL3-Basic luciferase reporter vector. Promoter activities were measured by luciferase assays from transiently transfected HEK293T or HepG2 cells. The SCD5 mRNA was detected in HEK293T cell line, whereas it is not present in HepG2 cells. The 1000 bp length region 5' upstream from start codon has been shown to be the most transcriptionally active in luciferase reporter system, however in cell line specific manner. The transcriptional variant A of SCD5 turned out to be the most abundant in the brain, while the highest expression level of variant B was measured in the pancreas. Variant A was present 10-100 times higher than B in all tissues. Although both transcriptional variants are expressed, the significantly lower expression of the B isoform cannot be explained by the common promoter. Further research is needed to elucidate the mechanism of the observed cell type specificity of SCD5 promoter activity, as well as its potential contribution in human diseases.