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Abstracts

Posters Session 6: Lipid sensing and lipid sensors (PO 97–107)

PO 97

Adipose differentiation related protein (ADRP) and the secretion of triglyceride-rich lipoproteins in intestine

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Small intestine ensures the absorption of dietary lipids and the subsequent secretion of triglycerides (TG). In mice, we observed that a two-day high fat diet (HFD) leads to an increased intestinal postprandial secretion of triglycerides and the accumulation of cytosolic lipid droplets (LD) in enterocytes. We thus analyzed the expression of the various members of the lipid droplets-associated PAT family proteins. We found that only two proteins of PAT family, namely ADRP and TIP47, are expressed in the small intestine of mice, as well as in human Caco-2/TC7 enterocytes. In HFD fed mice as well as in Caco-2/TC7 cells supplied with postprandial lipid micelles, the amount of ADRP protein increases whereas TIP47 is not modulated. Moreover, in the intestinal epithelium, HFD induces the appearance of a lower molecular weight (MW) form of ADRP, and the two ADRP forms have a distinct cellular localisation as shown by subcellular fractionation experiments. The low MW ADRP is associated with the membrane fraction, while the high MW ADRP is associated with the cytoplasmic fraction. By immunofluorescence, we show that ADRP is localized at endoplasmic reticulum and around LD.

Our results show for the first time that, in intestine, an increased ADRP level correlates with an increased triglyceride secretion. Moreover, our results suggest that the subcellular localisation of ADRP depends on post-translational modifications which could possibly participate to the control of TG partitioning between secretion and cytosolic storage.

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PO 98

NF-Y and Sp1 but no SREBP have a role in transcriptional regulation of the rat SND P102 gene

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Background: Staphylococcal nuclease and Tudor domain containing protein of 102 kDa (SNDp102) is a protein encoded by the rat Snd1 gene, firstly described as transcriptional coactivator that has been recently associated to lipid bodies in a model of hepatocellular esteatosis and adenovirus-driven differential expression of the protein modifies phospholipid secretion into lipoproteins in rat hepatocytes. A sequence of 1688 bp corresponding to SNDp102 gene promoter (AY957585) was isolated, cloned and the basal transcriptional activity determined in both rat McA-RH7777 and human HepG2 hepatoma cells. Our own group demonstrated the functional implication of nuclear factor-Y (NF-Y) and specificity protein 1 (Sp1) in the SNDp102 gene transcription.

Hypothesis: Since bioinformatic analysis of the promoter sequence has predicted the presence of sterol regulatory element (SRE) binding sites, we have studied the role of these transcription factors in the transcriptional regulation of the gene.

Methods and results: Electrophoretic mobility shift and supershift assays (EMSA) using atorvastatin-stimulated nuclear extract from rat McA-RH7777 and human HepG2 cells demonstrated no specific binding of the SREBPs to the regions located at [−185, −151], [−240, −211], [−253, −226] and [−265, −232] of the promoter sequence. Promoter activity of 5′ deletion fragments determined by luciferase reporter gene was unchanged when overexpressing SREBP-1c in HepG2 cells by transitory transfection.

Conclusion: These results suggest that SREBPs by themselves have irrelevant role in the SNDp102 gene expression. However, further studies are still necessary considering that SREBP-1c regulation pathway may be dependent of other close elements such as NF-Y and/or Sp1.

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