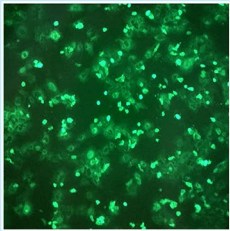




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Mouse Primary Liver Cells  
Expressing GFP-BirA

### **Translocation of GPAT4 from the Endoplasmic Reticulum to the Mitochondria May Alter Efficiency of TAG Synthesis in Liver Cells.**

Synthesis of triacylglycerol (TAG) is critical for the formation of lipoproteins and lipid droplets and is the main constituent of mammalian fat. The rate of TAG synthesis is controlled by the enzyme glycerol-3-phosphate acyltransferase (GPAT). GPAT4, an isoform of GPAT, is found predominantly in the ER, while GPAT1 is found in the mitochondria. Although GPAT4 and GPAT1 both produce lysophosphatidic acid (LPA), it is not known whether the outputs of phospholipids whose synthesis was initiated in the outer-mitochondrial membrane (OMM) differ from those that are initiated in the ER. It was hypothesized that GPAT4 and GPAT1 have non-interchangeable outputs within the cell and that the introduction of GPAT4 to the mitochondria would not affect TAG accumulation. To determine if there was a difference between the outputs of GPAT, primary liver cells were infected with adenovirus to express GPAT4 on the mitochondria (Ad-Tom20-GPAT4, "mtGPAT4"). We measured total enzyme activity in total particulate prepared from primary mouse hepatocytes infected with this virus. Introduction of mtGPAT4 had nearly a two-time increase in specific activity in these cells as compared to the GFP control adenovirus (Ad-T20-GFP-BirA, "control virus"). These results indicate that mtGPAT4 is functional in producing a greater output of PA (phosphatidic acid) as compared to non-infected cells and control virus infected cells. Furthermore, this increase in PA production may be indicative of a greater efficiency of TAG production within the cell. Understanding what affects the rate of TAG synthesis is important for providing a better understanding of diseases that are influenced by TAG production and maintenance, such as diabetes mellitus, lipodystrophy, and heart disease.