Long chain fatty acid induce energy inbalance and enhance AMPK/ULK1 pathway to cause autophagy in H9C2 cardiomyoblast cells

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Abstract

Metabolic syndrome is a combination of the medical disorders that, when occurring together, increase the risk of developing cardiovascular disease and diabetes. Recent study demonstrated that accumulation of saturated free fatty acids increased energy metabolism imbalance in the heart. Autophagy is critical for cell survival during states of energy crisis. Recently study show that Palmitic acid induces autophagy, but the mechanism is still unclear. In this study, we focus on how PA induces autophagy and the relationship between Palmitic acid-induced autophagy and apoptosis in H9c2 cells. We found that Palmitic acid induced autophagy via compensational increasing of phospho-AMPK and phospho-ULK1 futher to cause apoptosis. Cardiomyocytes use fatty acid as well as glucose for ATP production. These substrates are transported into the cell by fatty acid transporter CD36 and the glucose transporter 4 (GLUT4). These two receptors have shift phenomenon ,After treatment Palmitic acid induced lipotoxin in H9c2 cells. So, we focus on how PA induces two receptors shift in H9c2 cells.