066-LIPS

Modulation of AMPK$_{\alpha 1}$ in PC3 Cells, Following Exposure to Triphenyl Methanol Derivatives

AMP-activated protein kinase (AMPK) is activated physiologically due to stresses such as low nutrients and prolonged exercise. Furthermore, AMPK may be activated pharmacologically by metformin (the most widely prescribed Type 2 diabetes drug), phenformin, AICAR (Acadesine/AICA riboside) and resveratrol. When active due to low nutrients, AMPK coordinates the control of cell growth and autophagy *via* suppression of the mammalian target of rapamycin complex 1 (mTORC1) pathway. We have recently synthesized novel flavonoids, namely, triphenylmethanol derivatives (TPMs), but the effectiveness of the TPMs on the activity of AMPK remains unclear. We hypothesized that the novel TPMs will inhibit cell proliferation through activation of AMPK isoforms in cells. The effects of TPMs, on phospho-AMPKα1 in prostate, PC-3, cells were investigated. Cells were exposed to TPMs for either 12 or 24 hr. at the respective doses of 0, 25, 50, 100 and 200 μM. Following the incubation, the AMPKα1 phospho-protein in cultured cells were analyzed by the in-cell ELISA (ICE) assay kit (Cat. #: ab151280, Fisher Scientific, GA). The results indicate that cells exposed to the respective doses of TPMs increased phospho-AMPKα1 in a dose-dependent manner. The effects of the increases for the phospho-AMPKα1 in cells was greater for the 24 compared to the 12-hr. incubation. We are currently embarking on studies are to investigate the specificities of the said insults in increasing phospho-AMPKα1 activities and for the other respective isoforms.