AMPK*α1* IN PC-3 CELLS, FOLLOWING EXPOSURE TO TRIPHENYL METHANOL DERIVATIVES (TMPS).

Epidemiological studies indicate that treatment with metformin, an AMP-activated protein kinase (AMPK) activator, reduces the incidence of cancers. Activation of AMPK has also been reported to oppose tumor progression in types of cancers and offers promising cancer therapy. Furthermore, AMPK is a master regulator of energy metabolism and has also been implicated in cell cycle progression, angiogenesis, cell transformation, migration, and cancer. 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 cancer cell proliferation through activation of AMPK isoforms in cells. The effects of TPMs, on 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 based on the cell viability studies by the (3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide, a tetrazole) (MTT) assay. The results indicate that cells exposed to the respective doses of TPMs increased both phospho- and total-AMPK$_{\alpha}$ in a dose- and time-dependent manner. The effects of the increases for the *phospho*- and *total*-AMPKα in cells was greater for the 24 compared to the 12-hr. incubation. Further studies are currently going on to elucidate the specificities of the said insults in increasing the *phospho*- and *total*-AMPKα activities and for the other respective isoforms.