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Vitamin C impact on genistein-induced cell death in prostate cancer

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Abstract

Prostate cancer is the second leading cause of cancer-related death in America. An estimated 220,800 new cases and 27,540 cancer-related deaths are expected in 2015. Reactive Oxygen Species (ROS) can promote cancer cell proliferation when they reach elevated levels. Vitamin C (Vit C) is a water-soluble antioxidant, capable of inhibiting the formation of ROS. Genistein(Gn), an isoflavone found in plants, also possesses the ability to inhibit ROS formation. The purpose of this study was to investigate the impact of vitamin C on genistein-induced apoptosis in LNCaP cells and the potential pathways involve, using cell-based assays including: MTT assay to determine the effect of the various treatments (Gn, Vit C and Gn+Vit C combination) on LNCaP; Nitroblue tetrazolium assay (NBT) to assess treatment-induced intracellular ROS levels; Fluorescence microscopy to determine the mode of treatment-induced cell death. Briefly, LNCaP cells were exposed to varying concentrations of genistein (Gn10-70 uM) and vitamin C (C10-70uM) as single treatments, and Gn-VitC combination. For Gn-VitC combination regiment, the IC50 (40uM) of the Vit C (previously determined), was used with each concentration of the genistein. Post-treatment effects on the cells were assessed after 48 hr using the assays listed above.

The overall data from the result revealed a dose-dependent effect in all the three treatments, and apoptosis as the major mode of cell death and that vitamin C significantly augmented the effects of genistein. The combination treatment showed the most dramatic effect, causing most apoptosis. Details of the overall data implicates ROS in the treatment-induced apoptosis and significant positive impact of vitamin C on genistein treatment: an indication of the potential chemo/phyto-preventive significance of the nutrients. Further studies are in progress.

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