**ABSTRACT**

Current therapies for cancer are limited due to considerable side effects and there is need for combinations of new therapeutic agents that are less toxic to normal cells and produce an enhanced antitumor effect. Hence, the present study was aimed toinvestigate the influence of α-TOS on anticancer and adverse effects of Ara-C.Ara-C (50, 100, 150 ng/ml), α-TOS (20, 40, 60 μg/ml) and their combinations on HL-60 cell line produced significant (P<0.001) dose and time dependent decrease in percentage cell viability as measured by MTT, Trypan blue exclusion assay, dosedependent decrease in percentage colony growth in Clonogenic assay as compared tocontrol. The determination of mechanisms of Ara-C, α-TOS and their combinations in decreasing percentage cell viability of HL-60 cell line indicated that Ara-C andα-TOS alone involve caspase-3 enzyme and DNA fragmentation but in combinations, along with the involvement of caspase-3 enzyme and DNA fragmentation there are other mechanisms involved in decreasing percentage cell viability of HL-60 cell line which are to be further investigated. In-vivo studies in mice with Ara-C (100 mg/kg/day), α-TOS (100, 150 mg/kg/day) and their combinations for 8 and 16 days post-treatment revealed a significant increase in nitric oxide level and a significant decrease in reduced glutathione and LDH levels, volume of peritoneal cell, packed cell volume as compared to cancer (DLA) induced group. The multi-treatment of Ara-C and α-TOS maintained the normal levels of hemoglobin content, leukocytes and platelet count and also increased the mean survival time of cancer mice as compared to Ara-C alone treatment. Thus the results indicate that α-TOS has significant anticancer effect and also potentiated anticancer effect and reduced adverse effects of Ara-C.

**Key words:** Ara-C, α-TOS, HL-60, caspase-3, DLA.