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Antitumor action of Amygdalin on human breast cancer cells through selective sensitization to oxidative stress


The cytotoxic effects of amygdalin natural product on cultures of breast cancer cells were investigated in vitro. We used the cell lines MCF-7 and T47D, which are derived from luminal A subtype of breast tumors carcinoma. Though they vary in some molecular properties, these tumor cells share the presence of positive estrogen, progesterone receptors and lack of human epidermal growth factor 2. Our data demonstrated a growth suppression of MCF-7 and T47D by amygdalin in concentration and time-dependent manners. This growth suppression was concomitantly linked with an increase in the generation of malondialdehyde (MDA) and oxidized glutathione together with a decline in the total glutathione concentration and glutathione reductase activity. The proportional cell survival of these tumor cells was correlated positively with the total glutathione and inversely with the amygdalin or MDA levels ($p < 0.001$). In MCF-7 cells, the treatment with amygdalin showed 6 times less production of total glutathione as compared to the untreated matched tumor cells, whereas a similar amygdalin treatment of T47D cells yielded only 2.1 times difference in total glutathione

generation between the amygdalin treated and untreated tumor cells. Similarly, the amygdalin treatment of MCF-7 cells exhibited 2.4 times higher production of MDA than in the untreated tumor cells, while such difference in MDA formation between the amygdalin treated and untreated T47D tumor cells was dropped to 1.3 times. These data support an in vitro mechanism of amygdalin antitumor action against breast cancer cells potentiated by the induction of oxidative stress. The cells of MCF-7 originated from a highly proliferating breast cancer tumors seem to be more vulnerable to the oxidative stress mediated amygdalin cytotoxicity than T47D cells which derived from a slowly proliferating breast cancer tumor.

Speaker Biography

Muayad M Abboud has done PhD in clinical biochemistry, Medical and biological school, University of Southampton, England, followingly his Postdoctorate Fellow in medical research unit, University of Sussex, Brighton, England.

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