Artichoke: An Artist to Choke Cervical Cancer by Downregulation of Cyclin D and Bcl-2

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Background

- Cervical cancer (CC) continues to be one of the leading causes of mortality worldwide. It is estimated that 90% of deaths from CC occur in low- and middle-income countries.[1] Artichoke is a commonly consumed plant that has been studied more recently with increasing interest for its abundance in antioxidants. Our previous study showed that artichoke extract (AE) is a potent inhibitor of melanoma.[2] To extend that study, this study was designed to assess the potential antitumor effects of AE on the SiHa CC cell line.

Methods

- Clonogenic survival assay, cell proliferation, and caspase-3 activity kits were used to evaluate the effects of AE on cell survival, proliferation, and apoptosis of SiHa CC cells. Molecular mechanisms were further assessed by using RT-PCR and IHC.

Results

- SiHa CC cell colony count significantly decreased in the presence of AE. A decrease in the OD value of CC cells was also found in the presence of AE. The relative caspase-3 activity in SiHa CC cells increased significantly in the presence of AE. The anti-proliferative effect of AE on SiHa CC cells correlated with decreased expression of cyclin D. The pro-apoptotic effect of AE on SiHa CC cells correlated with decreased expression of Bcl-2.

Conclusion

- Artichoke inhibits growth of CC through inhibiting proliferation and promoting apoptosis by downregulation of cyclin D and Bcl-2. These findings extend the anti-tumor effect of artichoke from melanoma to CC, supporting the concept that artichoke exerts powerful anti-tumor property in not only one cancer. Such a study may be useful to develop natural treatments for many types of cancers.

Fig. 1. AE inhibits growth and proliferation of CC cells

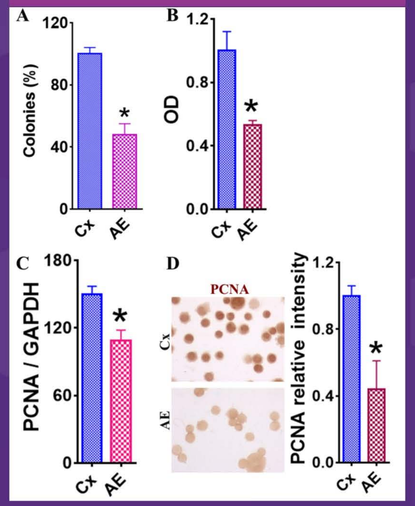

Fig. 2. Effect of AE on pro- and anti-apoptotic molecules by RT-PCR

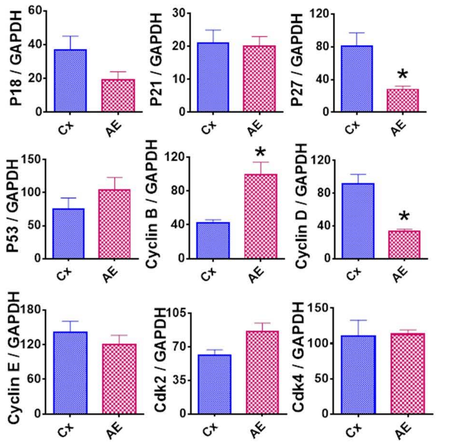

Fig. 3. AE downregulates anti-apoptotic molecule Bcl-2

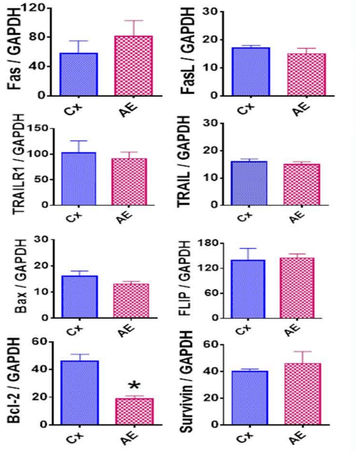

Fig. 4. AE promotes apoptosis of CC cells

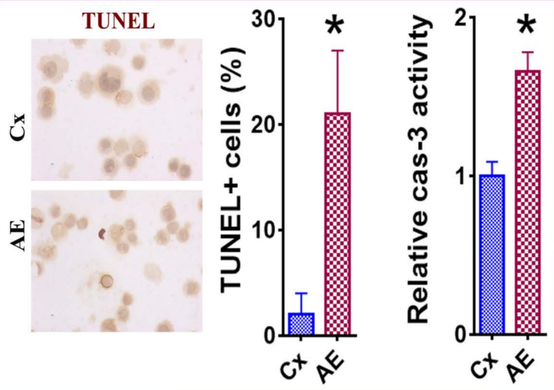

Fig. 5. AE downregulates pro-proliferative molecule Cyclin D