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Novel effect of Acetyl-11-Keto- Boswellic acid (AKBA) on mitophagy-induced apopotosis using cellular proteomic profiling

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Terpenoids and their potential analogues have attracted recent attention to their anti-cancer activity with lower adverse effects. Acetyl-11-Keto- Boswellic acid (AKBA) is a derivative of boswellic acid that exerts anti-cancer properties against different types of cancer cells. AKBA known to induce apoptosis via activation of caspase 8 and also induction of epigenetic pathways via regulation of histone deacetylase gene expressions. However, molecular target and pathways are not identified. In this study we implemented in-depth non-labelled proteomic profiling of breast cancer cells MCF-7 treated with AKBA. Proteomic signal ratios were normlized using variance stablizing normalization and corrected for false detection ratio. Otal of 137 proteins were signifcanlty up and down regulated. Set of mitochondrial proteins were downregulated and set of autophagy protein pathways were up-regulated. Further confirmation of mitophagy pathway was studied using western blot of SQTRM, PLINK, PINK-1 proteins. Smiliarly, mitochondrial content were measured to confirm mitochondrial degradation. To assess if mitochondrial loss is due to autophagy pathwya, we performed dual staining for LC3 and mitochondrial markers. Conclusion, our result show for first time that AKBA induced apoptosis via mitophagy pathway which explains previously known AKBA induction of caspase-8 pathway. AKBA provide a novel therapeutic agent that is capable of induction of apoptosis to cancer cells with less toxicity to normal cells.

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