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Licarin A induces cell death by activation of autophagy and apoptosis in non-small cell lung cancer cells

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Erratum in

Correction to: Licarin A induces cell death by activation of autophagy and apoptosis in non-small cell lung cancer cells.

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Abstract

Lung cancer has a relatively poor prognosis with a low survival rate and drugs that target other cell death mechanism like autophagy may help improving current therapeutic strategy. This study investigated the anti-proliferative effect of Licarin A (LCA) from Myristica fragrans in non-small cell lung cancer cell lines-A549, NCI-H23, NCI-H520 and NCI-H460. LCA inhibited proliferation of all the four cell lines in a dose and time dependent manner with minimum IC50 of 20.03 ± 3.12, 22.19 ± 1.37 μM in NCI-H23 and A549 cells respectively. Hence NCI-H23 and A549 cells were used to assess the ability LCA to induce autophagy and apoptosis. LCA treatment caused G1 arrest, increase in Beclin 1, LC3II levels and degradation of p62 indicating activation of autophagy in both NCI-H23 and A549 cells. In addition, LCA mediated apoptotic cell death was confirmed by MMP loss, increased ROS, cleaved PARP and decreased pro-caspase3. To understand the role of LCA induced autophagy and its association with apoptosis, cells were analysed following treatment with a late autophagy inhibitor-chloroquine and also after Beclin 1 siRNA transfection. Data indicated that inhibition of autophagy resulted in reduced anti-proliferative as well as pro-apoptotic ability of LCA. These findings confirmed that LCA brought about autophagy dependent apoptosis in non-small cell lung cancer cells and hence it may serve as a potential drug candidate for non-small cell lung cancer therapy.

Keywords: Apoptosis; Autophagy; Chloroquine; Licarin A; Reactive oxygen species.

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