Conclusions
CONCLUSION 1

2,4-Dichlorophenoxy acetic acid induced toxicity in lung cells by disrupting tubulin-microtubule network

2,4-Dichlorophenoxy acetic acid WHO recognised Class II 'moderately hazardous' herbicide commonly used in agriculture. Several reports on accident of poisoning damage and/or deaths caused by this herbicide represent it as real hazard in human and animal health. Due to it is sprayed over the crop field as aerosol; lung damage by this 2,4-D contaminated air is very common scenario. But the exact molecular mechanism of toxic action of 2,4-D is still unknown. The present study investigate the molecular mechanism of lung damage induced by 2,4-D, where it is demonstrated that 2,4-D decrease the cell viability of A549 cells by dose dependent manner and further study indicates that the mode of cell death is apoptosis. Furthermore, it also targets crucial cellular protein like tubulin microtubule network and that may cause cellular apoptosis. So by knowing molecular mechanism of 2,4-D induced lung damage, a policy should be made to reducing this type of toxicity, as a preventive measure.

CONCLUSION 2

Morin protects oral cells from smoke-less tobacco extract induced cytotoxic autophagy and apoptosis by modulating expression of Nrf2.

We found regulation of nrf2 plays a critical role in STE induced toxicity. STE reduced the level of Nrf2 and as a consequence of ROS dependent cytotoxic apoptosis and autophagy occur in SCC25 cell line. Morin effectively protects SCC25 cells from STE induced
Conclusions

Cytotoxicity by restoration of Nrf2 level. Hence, morin was found to be effective in ameliorating ROS dependent apoptosis and autophagy caused by STE induced toxicity in SCC cell line and that make it as a reliable option for antioxidant therapy in treatment of STE induced toxicity, pending further in vivo and clinical investigations.

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CONCLUSION 3

Inhibition of autophagy by chloroquine potentiates synergistically anticancer property of artemisinin by promoting ROS dependent apoptosis.

We found that autophagy was induced by Artemisinin along with apoptotic cell death in a variety of cancer cells, and inhibition of this autophagy by late autophagy inhibitors Chloroquine increased ROS dependent apoptotic cell death. So the appropriate manipulation of autophagy by using Chloroquine provides a powerful strategy to increase the efficacy of selective anticancer property of Artemisinin. Therefore our data suggest that CQ is a drug which already in clinical usage and repurposing it as a new adjuvant in selective anticancer therapy by using ART is useful, practical and cost effective.