Supplementary Information

The spice-derived phenolic, malabaricone B induces mitochondrial damage in lung cancer via a p53-independent pathway

Mrityunjay Tyagi,1,2,# Biswanath Maity,1,# Bhaskar Saha,1,#

Ajay Bauri,1 Mahesh Subramanian,1,2 Subrata Chattopadhyay1,2 and Birija Sankar Patro,1,2,*

1Bio-Organic Division, Bhabha Atomic Research Centre, Mumbai-400085, India

2Homi Bhabha National Institute, Training School Complex, Anushakti Nagar, Mumbai-400094, India.
Figure S11. Mal B kills different cancer cells both time and dose-dependently. **A.** A549 cells, **B.** A375 cells, **C.** Jurkat cells, **D.** INT-407 cells, **E.** WI-38 cells. The cells were treated with vehicle
(0.1% DMSO) or increasing concentrations of mal B. Cell proliferation at different time points (24-72 h) was assessed by the MTT assay and the results are expressed in percentage considering that of the untreated control cells as 100. The experiments were repeated three times with similar results. All determinations were made in five replicates and the values are mean ± S. E. M. *p<0.05, **p<0.01 compared to vehicle control cells.

**Fig. S12**

![Graphs showing the effect of NAC on apoptosis and caspase-3 activity](image)

Figure S12. NAC dose-dependently reduces mal B-induced apoptosis and caspase-3 activation in A549 cells. **A.** Effect on apoptosis. **B.** Effect on caspase-3 activity. The cells were treated with vehicle (0.1% DMSO) or mal B (10 μM) for 48 h. Apoptosis in terms of enriched nucleosomes in cytoplasm, and caspase-3 the activity were estimated. The experiments were repeated three times with similar results. All determinations were made in five replicates and the values are mean ± S. E. M. *p<0.01 compared to vehicle control cells; **p<0.05 compared to only mal B (10 μM) treatment.