SUPPLEMENTARY DATA



Suppl. Fig. 1. GE reduces viability of human prostate cancer PC-3 cells in a concentration- and time- dependent manner. **A**. Cells were plated at a density of $5,000/\text{cm}^2$ overnight and treated with either vehicle (DMSO) alone or GE at concentrations of 50 to 2000 µg/ml for 48h. After 48h, cells were collected upon a brief trypsinization and counted with a hemocytometer after trypan blue staining. **B**. PC-3 cells were treated with 250 µg/ml GE for 0, 6, 12, 24, 48 and 72h. Columns, mean total number of blue (dead) cells from three independent samples for each treatment; bars, SD. P<0.05 versus control.



Suppl. Fig. 2. GE inhibits the growth of various cancer cells from different tissue types. Cells were treated for 72h with GE concentrations ranging between 1-1000 μ g/ml followed by evaluation of cell proliferation using the Alamar Blue assay. Graph shows the percentage of viable cells (y-axis) versus GE concentrations (x-axis) for breast (MDA-MB-231, MCF-7) and cervical (HeLa) cancer cells.



Suppl. Fig. 3. GE affects cell-cycle progression kinetics by causing a G1/S phase arrest followed by an increase in sub-G1 cell population, suggesting apoptosis. **A** and **B** are bar-graph representation of relative percentage of cells in various cell-cycle phases over dose (0-1000 μ g/ml) and time (0-48h) of GE treatment, respectively.



Suppl. Fig. 4. 1000 μ g/ml GE treatment for 6h induces apoptosis at without any cell cycle arrest whereas, 50 μ g/ml for 72h induced G2/M arrest followed by enhanced apoptosis compared to 1000 μ g/ml.



Suppl. Fig. 5. Immunofluorescence micrographs showing that GE treatment at 250 μ g/ml for 24h causes translocation of cytochrome c from mitochondria to cytosol.



Suppl. Fig. 6. Treatment of androgen-responsive LNCaP cells for 24h with varying dose levels of GE (0-1000 μ g/ml) increase the sub-G1 cell population (**A**), indicative of apoptosis. **B**. Immunoblot analysis showed increased expression of cleaved caspase-3 over varying doses.

 β -actin was used as a loading control. C. Caspase-3/7 activity assay showed an increase over varying dose leevsl in LNCaP cells.



Suppl. Fig. 7. Quantitation of tumor weight from control and GE-treated groups. Columns, mean tumor weight; bars, SD. P<0.05 versus control.

Suppl. Fig. 8



Suppl. Fig. 8. 100 mg/kg GE administered daily by oral gavage does not cause any detectable pathologic abnormalities in normal tissues. Panels show H&E staining of paraffin-embedded 5- μ m-thick tissue sections of the gut, liver, adrenal, spleen, lung, brain, kidney, heart, testes and bone marrow from control and GE-treated groups of mice (magnification, 200X).

Suppl. Fig. 9



Suppl. Fig. 9. Immunohistochemical staining of paraffin-embedded colonic sections from vehicle-treated control and GE-treated groups, for Ki67, an indicator of proliferation index. Images were captured by a light microscope (Olympus BX40) using a 10X objective. The staining pattern was indistinguishable among the control and treated groups indicating absence of GE toxicity in tissue with rapidly proliferating tissues.



Suppl. Fig. 10. GE feeding did not cause any observable toxicity as observed by comparable serum biochemical profiles among control and GE-treated mice. No significant difference could be detected for both groups and the levels of alkaline phosphatase (Alk phos), alanine aminotransferase (ALT), Gamma-glutamyltransferase (GGT) (markers of hepatic function) and

blood urea nitrogen and creatinine (renal toxicity markers) were comparable for the treated and the control groups. Standard electrolyte panel (sodium, potassium, calcium, chloride, bicarbonate) also showed no abnormalities in electrolytes among the two groups. In addition, anion gap, a useful indicator of abnormalities in the acid-base balance, was also indistinguishable among the two groups.