Background/aim: Lung cancer is one of the leading cause of cancer death in all over the World. Therefore, there is an urgent need to develop effective therapies for this cancer. Rottlerin, a Kmala tree-derived anticancer compound, exerts number of therapeutic effects on cell survival, invasion, apoptosis and autophagy. In this study, we evaluated the effects of rottlerin on cell proliferation and cell cycle pathways in human lung cancer cells.

Material Methods: A549 human lung cancer cell line was cultured in RPMI 1640 medium (Gibco BRL) supplemented with 10% fetal bovine serum and 1% penicillin–streptomycin. Rottlerin was purchased from (Sigma-Aldrich, St. Louis, MO). The viability and/or proliferation of cells treated with different concentrations of rottlerin (2–20 ?M) were detected by colorimetric MTT assay (Promega, Madison, WI, USA). Colony formation capability of the cells under rottlerin treatment was evaluated with in vitro clonogenic assay. Mitogen Activated Protein Kinase/Extracellular-Signal-Regulated kinases (MAPK/ERK) phosphorylation and Cyclin D1 protein expression were also assessed by westernblot analysis.

Results: We found that rottlerin inhibits cell proliferation and colony formation with dose depending manner in A549 cells. MAPK/ERK phosphorylation and Cyclin D1 protein expression were downregulated by rottlerin at 5?M concentration.

Conclusion: Rottlerin inhibits proliferation of highly aggressive lung cancer cells by blocking key signaling pathways promoting tumorigenesis. Further understanding the underlying mechanisms of rottlerin-induced cancer cell death is an important issue for its development as a novel targeted therapies.