AN EVALUATION OF GROWTH AND VIABILITY OF THE A549 AND MRC-5 CELL LINES UPON EXPOSURE TO SELECTIVE ORGANIC INHIBITORS OF GLYCOLYSIS

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Abstract: Lung cancer is one of the most prevalent and deadly cancers in United States. In 2007, the United States reported over 213,000 new lung cancer diagnoses and 160,390 deaths caused by lung cancer. Research has shown that cancer cells exhibit higher glycolytic rates than normal cells. We hypothesize that exposure of A549 cells to organic inhibitors of glycolysis would have a negative impact on their survival and viability due to a vast decrease in glycolytic rates and the resultant ATP production. Therefore, the objective of this research was to assess the differential role of eleven organic glycolytic inhibitors in controlling lung cancer in vitro. These organic reagents include fructose diphosphate (FDP), sodium citrate, ascorbic acid, crude honey, sodium bicarbonate, D-glucose, oxalic acid, glycerol, zinc acetate, pyruvic acid, and sodium ascorbate. The human lung fibroblast cell line (MRC-5) was selected to represent the normal human lung and the human alveolar epithelial cell line A549 was selected to represent lung cancer in vitro. These cells were maintained and exposed to different organic reagents at concentration levels ranging from 31.3-2,000 µg/ml in 96 well plates in triplets using MTT, Alamar blue and cell counting (T4 cellometer) assays as well as phase-contrast photo-imaging. Data was analyzed statistically to determine the survival and death parameters. Our results indicate that exposure of A549 cells to organics resulted in concentration dependent cell destruction / cell proliferation depending on the cell line exposed. Eight of the eleven organics used namely zinc acetate, oxalic acid, honey, FDP, pyruvic acid, sodium citrate, ascorbic acid and glycerol showed statistically significant (p<0.05) differential negative effects on the A549 line in comparison to its control as well as to their effects on the MRC-5 cell line. Viability using the T4 cellometer counting chamber ranged between 60-76% for the A549 compared to 96- >100% (proliferation) for its control as well as the exposed MRC-5 cell line. LC50 using MTT and Alamar blue assays ranged from 3-10 µg/ml. We conclude that eight of the tested organics impaired glycolysis which is crucial to the generation of cellular energy and survival of the A549 cell line. This study is very promising and warrants further investigations towards exploiting this differential response aiming at an effective cancer therapeutic potential role for these natural organics.

Keywords: Lung cancer, A549, MRC-5, Organic inhibitors, Glycolysis, Cell viability

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