Genes encode the proteins which dictate cellular functions within organisms. Each step in the flow of information from DNA to RNA to protein provides the cell with integral information that works to regulate functionality by adjusting the amount and type of proteins manufactured. The functionality of genes has been found to be unique among individual samples, with one study stating gene expression levels were variable based on the “underlying genetics of the [samples] they were collected from” (1). Gene expression also gives essential information regarding cellular responses during diseased states and the communication of these genes to the cells to increase organismal immunity. Immune responses are heighted by a combination of genes when the cell is under distress, and understanding the unique integration of genes in molecular pathways can thoroughly improve the efficacy of combination treatments (2). By profiling the expression of genes based on these genetically inherited differences, disease treatments can be crafted on an individual, patient basis.

Studies behind benign and malignant tumor formation are of increased interest for gene therapy studies, since they offer a control mechanism for the up and down regulation of essential genes under normal or uncontrolled cellular growth conditions. “Clients with very advanced cancers refractory to conventional treatment indicate that [gene therapy] can specifically mediate tumor regression with low toxicity” (3). Therefore, understanding how the organism responds to the inheritance of a single functionally defective gene through the regulation of defense genes is imperative to stopping tumor growth in patients prior to the removal of the defective gene through vector therapy (3).

Lung cancer has traditionally been associated with smoke inhalation from carcinogenic substances which activate a downstream pathway to accelerate the development of cancer (5). Lung cancer is specifically caused by epidermal growth factor receptor genes, a receptor tyrosine kinase that can be modulated through inhibition with EGFR synthetic inhibition therapy (getfitnib) (4). However, these methods are not conducive to long term treatments, due to the sensitivity predictions and high mutation rates of cancerous cells (4). Within this study, we evaluate the representative genes discovered within smoking and non-smoking patient tumors and their associations with the human body and specifically EGFR.

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