



## Identification Studies of Drug Resistance and the Tumor Microenvironment

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### ABOUT THE STUDY

The most frequent cancer diagnosis worldwide is lung cancer. New lung cancer cases are expected to make up 12.42% of all cancer diagnoses in 2021, and lung cancer deaths are expected to account for 21.67% of all cancer-related deaths, making it the cancer with the highest fatality rate. According to histology, non-small cell lung cancer makes up about 80% of lung cancer cases. Gefitinib and erlotinib are two examples of the EGFR Tyrosine Kinase Inhibitors (TKIs) that have been developed as a result of earlier genomics studies that identified several high-frequency genetic variants in NSCLC that regulate or contribute to the occurrence and development of NSCLC. These variants include KRAS mutations and EGFR mutations.

There have been few research on the connection between the Tumor Microenvironment (TME) and treatment resistance, despite the fact that there is now a lot of information showing how closely the TME is tied to tumour growth. The TME contains both tumour cells and other cell types, including immune cells, mesenchymal cells, and fibroblasts, and it offers the circumstances necessary for tumour survival. The development of drug resistance to anticancer therapies was formerly thought to be primarily caused by genetic changes in tumour cells; however, there is now evidence that the TME plays a critical part in this process.

Numerous earlier research have demonstrated that genetic changes in tumour cells are what lead to treatment resistance. However, the emergence of drug resistance is a multifaceted process that, in our opinion, also entails modifications to the TME that support the growth of tumour cells in a vicious cycle.

Significant tumour heterogeneity is also thought to contribute to the development of treatment resistance. Previous research using individual gene macro-level sequencing was unable to adequately explain tumour heterogeneity. However, by sequencing each cell individually, scRNA-seq can significantly advance cancer research.

The term "intratumor heterogeneity" describes the many traits that various tumour cells may exhibit inside the same overall lesion. Our findings demonstrated that even in identical growing circumstances, PC9 cells produce various clusters. Given that the other three clusters were centred in the area where D0, D4, and D11 cells overlap, clusters 2 and 4 of these five detected clusters were regarded as drug-sensitive and drug-resistant clusters, respectively. These groups of cells might be cells that are halfway between being drug-sensitive and drug-resistant. We unexpectedly discovered that MGs were barely expressed in the other clusters, despite the fact that MGs were characterised as differently expressed genes between clusters 2 and 4. This supported our hypothesis that clusters 2 and 4 constituted clusters that were drug-sensitive and resistant, respectively.

Collagen VI expression was higher in the resistant group than in the sensitive group when cisplatin-resistant cells were compared to cisplatin-sensitive cells. This shows that chemotherapy can change TME by modifying tumour DNA directly. The expression pattern of MGs was next investigated using the bulk RNA-seq data of GSE42127. 176 NSCLC samples were split into two groups using unsupervised consensus clustering based on the expression of MGs: C1 and C2. The algorithm is a widely used method of determining tumour purity, and numerous studies have shown its accuracy.

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