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ISSN: 2684-1614
Journal of Tumour Research & Reports

Perspective

Importance of Cisplatin Resistance towards Lung Cancer Therapy

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DESCRIPTION

Cancer is the leading causes of mortality worldwide. The majority of lung cancer patients have a group of histologic subtypes collectively referred to as Non-Small Cell Lung Cancer (NSCLC). There are various subtypes of cancers, lung adenocarcinoma and lung squamous cell carcinomas are the most common subtypes.

Drug resistance is the greatest obstacle to long-term patient survival. Cancer cells can evade therapy and develop drug resistance *via* various pathways, but many of these pathways remain unpredictable and difficult to characterize. A better understanding of the molecular mechanisms contributing to tumour progression and drug resistance is essential for the development of specific therapeutics for cancer subtypes.

Drug resistance is defined as the genetic ability of cells to survive clinically relevant drug concentrations. Drug resistance can exist before treatment initiation, known as hereditary resistance, or develop in response to treatment, known as acquired resistance. Genetic drug resistance is generally assumed to involve genetic mutations, whereas acquired resistance is generally assumed to result from both genetic and non-genetic/epigenetic alterations.

Anticancer drug resistance is multifactorial and not caused by genetic mechanisms alone. Indeed, accumulating evidence suggests that non-genetic mechanisms such as lineage plasticity (changes in cell identity), epigenetic factors regulating gene expression, and phenotypic plasticity contribute to cancer drug resistance indicates that there is Cancer cells evade drug attack through two phenomena. Drug-resistant survivors remain an important factor in cancer recurrence and the development of drug resistance. Persistence is observed at a lower frequency in tumour cells with reduced growth rates and reduced metabolism, helping them to withstand drug exposure. The genetic makeup of Developmental Therapeutics Program (DTPs) is indistinguishable from the bulk of the tumour population, and withdrawal of the drug revert the resistance they exhibit to a sensitive state. Moreover, this variability ultimately cell fate, whether cells become irreversibly resistant to drug treatment.

Two phenomena that determine whether a particular cancer cell population undergoes non-genetic evolution of drug resistance are epigenetic heterogeneity and epigenetic plasticity. Epigenetic heterogeneity refers to the general variability of the epigenetic landscape across a particular cell population and is influenced by both intracellular and extracellular stimuli. Epigenetic plasticity, on the other hand, is the ability of cells to change their epigenetic state in response to internal or external stimuli. It is easy to understand both epigenetic heterogeneity and also epigenetic plasticity which are not completely independent variables to each other. If different cancer cell types exhibit heterogeneity then epigenetic state of the population shows more plastic effect.

A stable mechanism of non-genetic resistance could lead to the pre-existence of resistant clones in subpopulations. In this case, drug resistance arises simply by Darwinian selection and is entirely dependent on epigenetic heterogeneity.

Recent studies suggest genetic and non-genetic mechanisms of the drug resistance which are not mutually exclusive indeed coexist within certain cancer types by driving drug resistance which ultimately leads to the treatment failure. Genetic/non-genetic duality is believed to be a major contributor to the complexity of drug resistance, as described in this study. Developing drugs that target only genetic mutations is like playing the mole game and never winning. Therefore, a deeper understanding of the relative contributions of genetic and non-genetic mechanisms is important. In particular, critical to achieve desired targets by understanding how and why these non-genetic alterations occur today.

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Received: 01-Mar-2023, Manuscript No. JTRR-23-20760; **Editor assigned:** 06-Mar-2023, Pre QC No. JTRR-23-20760 (PQ); **Reviewed:** 21-Mar-2023, QC No. JTRR-23-20760; **Revised:** 28-Mar-2023, Manuscript No. JTRR-23-20760 (R); **Published:** 05-Apr-2023, DOI: 10.35248/2684-1614.23.8.187

Citation: Claws M (2023) Importance of Cisplatin Resistance towards Lung Cancer Therapy. J Tum Res Reports. 8:187.