DEEP LEARNING APPROACHES FOR PREDICTING AND TARGETING DRUG RESISTANCE IN CANCER CHEMOTHERAPY

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ABSTRACT

Drug resistance phases have remained a major roadblock in cancer chemotherapy leading to relapse and metastasis despite advances in precision medicine and immunotherapy. Resistance arises through varied and complex pathways such as drug efflux preferred DNA repair mechanisms evasion of apoptosis and inactivation of drugs. With data such as multi-omics, histopathology and clinical data at one's disposal machine learning and deep learning methods are useful in analyzing and predicting resistance. The study considers models such as CNNs, RNNs, autoencoders and ensemble methods for their roles in chemoresistance. The paper proposes a hybrid deep learning framework which incorporates pharmacogenomic and multi-omics features for better personalized treatment strategies.

Keywords: *Cancer Drug Resistance, Chemotherapy Resistance, Drug Response Prediction Artificial Intelligence, Machine Learning, Deep Learning*

1. INTRODUCTION

Cancer stands as one of the foremost reasons for deaths worldwide for instance approximately 10 million deaths took place in 2020 said-by World Health Organization2021. It is a vast gathering of more than 200 diseases linked through different genes or epigenetic alterations affecting orderly cell division. These changes are a series of disorders in cellular mechanisms e.g. proliferative signaling, evading growth-suppressive signals, activating invasion and metastasis and resisting cell death (Hanahan & Weinberg, 2011) [1]. The extrinsic risk factors interact with a person's intrinsic genetic susceptibility to a cancer e.g. smoking, microbial exposure, diet and radiation (Zhou et al., 2022) [2]. Nevertheless, tumor drug resistance and tumor heterogeneity persisted amid earlier diagnosis through liquid biopsy, immunotherapy and precision oncology-based treatment (Zhao et al., 2025) [3]. Generally chemotherapeutic agents target cells in the active phases of cell division i.e. DNA synthesis (S phase) and mitosis (M phase). A few interfere with microtubule function (taxanes and vinca alkaloids) others trigger apoptosis or inhibit replication (Amjad et al., 2025) [4]. Hematologic cancer may respond well but solid tumors are hard to treat because of toxicity and resistance (Wang et al., 2024) [5]. Combination treatments on the basis of principles like the fraction kill hypothesis and the Goldie-Coldman model select the agents based on different mechanism of action and toxicity to