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# Omics Data and Machine Learning Combination used in the Therapy of the Majority of Cancer Cells

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Finding drugs that can positively offer treatment of tumors and subsequently providing a prescription of an ideal clinical treatment regimen are regarded as the core activities of precision oncology [1]. In most cases, precision oncology involves the use of effective therapies to identify and target tumor-explicit abnormalities. Nonetheless, there are no biomarkers to provide guidance on how the majority of the first-line chemotherapy medications such as the nonspecific cytotoxic drugs can be applied [2]. Some restrictions that negatively affect the utilization of a drug target's genomic status as a therapeutic indicator in relation to molecularly targeted pharmacotherapies [3]. Notably, the method only benefits a small number of patients. The researchers relied on train classifiers to predict the drugs' effectiveness in cancer cell lines as well as deep learning or machine learning to explore the genome-scale omics data in order to determine any informative features [4].

The technique proposed in the study can correctly make prediction of the effectiveness of the drug despite being nonspecific chemotherapy drugs or molecularly targeted drugs [5]. The methodology can detect subtle cancer cells with an average specificity and sensitivity of 0.8 respectively on a per-drug basis [6]. Additionally, it can determine the effectiveness of the drugs with an average specificity and sensitivity of 0.8 correspondingly on the basis of the per-cell line [7-8]. The study seeks to propose a precision medicine method that is largely data-driven, maximizes therapeutic efficacy, and it is highly generalizable [9]. The framework discussed in the article can considerably benefit a majority of cancer patients since it has the potential of widening the space of precision oncology further than the targeted therapies especially when it accurately explained in clinical settings [10].

Cancer therapeutics can greatly be improved through data-driven methods. The present techniques fail to correctly match the sensitive drug-cancer pairs despite latest extensive pharmacogenomics screening on patient-derived xenografts (PDX) and cancer cell lines proving that nearly every PDX or cancer cell line has sensitivity to a single or multiple non-targeted or targeted drugs [11]. Additionally, there are minimal data-driven models that cover non-specific cytotoxic medications. The genomic markers cannot be regarded as correct indicators when applied in molecularly targeted medications [12]. When one relates or applies the scenario in clinical environment, it means that majority of the patients receive treatment using wrong chemotherapy owing to the absence of prognostic predictors. Alternatively, with regards to molecularly targeted medications, most of the complex cancers fail to host genomic variations in the targeted genes [13]. The medical effect is that there are patients who might greatly benefit on such molecularly targeted drugs, but they are overlooked owing to the imprecision of the genomic markers [14]. The therapeutic significance of the current anticancer drugs for improving treatment results can be exploited and optimized when such groups of patients are correctly identified [15]. Presently, researchers have been able to collect transcriptomic and genomic data as well as drug sensitivity data related to a huge number of PDXs and cancer cell lines using pharmacogenomics experiments [16]. Mostly, such investigations have sought to determine the relationship between drug-sensitivity evaluations (for instance IC50) as well as the omics features [17]. Furthermore, other researchers have used advanced classification models like the SVM and the ridge regression in training prognostic models whereby the genome-scale omics data is relied upon input features [18]. Nonetheless, the computational models have performed dismally. Therefore, drug sensitivity can correctly be predicted using a model-based technique which helps expert to learn the unique features from the omics data as well as deal with overfitting triggered by the challenges that are usually experienced owing to extreme dimensionality of the omics data as well as the comparatively minimal number of training cases which are accessible [19].

The researchers attempted to develop a prognostic model that can successfully be used in both targeted and untargeted therapies (conventional chemotherapy and molecularly targeted drugs) to predict their effectiveness on cancer cell lines [20]. They combined modern machine learning methods such as the support vector machines with the genomescale omics data [21]. The findings reveal that data-driven methods can considerably

outdo the present rule-based methods which use drug targets' genomic status as the main therapeutic indicators [22]. The findings support the need for additional studies on the degree by which the introduced techniques can enhance prediction of how patient tumors are sensitive to the presently accessible cancer medication [23]. The investigations proves that omics data have details that can be considered as valuable and convenient with regards to predicting a sensitivity of cancer drugs [24]. Metabolomics, proteomic, transcriptomic, and genomic data will play an active role with regards to advancing data-driven precision medicine as the expenses related to collection of omics data considerably reduces while biotechnology continues to advance and assist in collection of molecular phenotypes [25]. Computer-based decision support system can be used to provide the available cancer treatment such as the cytotoxic chemotherapies, immunotherapy, and molecularly targeted drugs [26]. It is evident that precision oncology can improve therapeutic efficacy in cancer treatment.

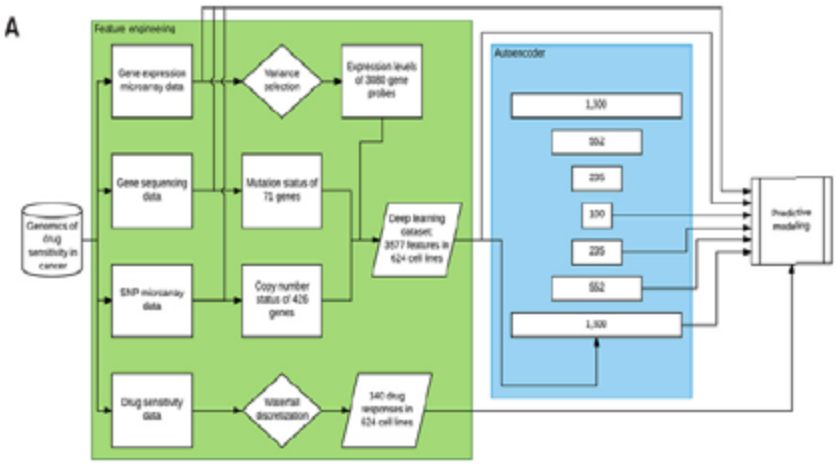


Figure 1: Workflow for the optimization and training of the drug-sensitivity prediction models (Source: Michael Q. Ding et al. (2018) Precision Oncology beyond Targeted Therapy: Combining Omics Data with Machine Learning Matches the Majority of Cancer Cells to Effective Therapeutics. Molecular Cancer Research 16: 269-278).

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