

**Invitation to M.Tech. Thesis Defense of Prashant Sharma (MT21227):** **July 06, 2023 (Thursday):** **10:30 AM – 11:30 AM IST**

In Partial Fulfillment of the Requirements for the Degree of

**M.Tech. CB**

**Prashant Sharma (MT21227)**

Will defend his thesis

**Title: “Machine Learning aided Cancer drug response prediction using chemo-genomic data”**

IIIT-D Faculty and Students are invited

**Date: July 06, 2023 (Thursday)
Time:** **10:30 AM – 11:30 AM IST**

**Meeting Link:** **https://meet.google.com/cqw-kdae-foj**

**Examiner:**

**Internal:   Gaurav Ahuja**

**~~External~~/Internal: Tarini Shankar Ghosh**

**Advisor: Debarka Sengupta**

**Co-Advisor NA**

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**Abstract**

The intricate nature of tumor heterogeneity and the personalized responses of cancer patients to drug treatments present significant challenges in the field of oncology. In this thesis, I aim to address such challenges by developing a predictive modeling approach to forecast drug responses in cancer cell lines using a combination of drug and somatic mutations based features. A comprehensive dataset for every drug-cell-line pair was constructed by concatenating drug descriptors derived from SMILES representations with mutation features obtained through the application of the Personalized PageRank (PPR) algorithm on a Protein-Protein Interaction Network (PPIN). This approach provides a deeper insight into the propagation of gene deleteriousness within the PPIN. The objective of this study is to provide a modeling approach to predict the drug responses for these drug-cell-line pairs. Through rigorous model selection and hyperparameter tuning, the most effective model for the prediction task was selected. The final model - Precily 2.0 exhibited promising performance during evaluation and generated reliable results. Precily 2.0 achieved a Pearson Correlation Coefficient of 0.83, which is better than the other proposed models with a similar methodology. The study highlights the potential of incorporating drug descriptors and network-based propagation of mutation as predictive features for determining drug responses in cancer cell lines. This model would empower oncologists to make informed treatment decisions for individual patients and thereby contribute to the advancement of precision oncology.