COMPUTATIONAL BIOLOGY WEBINAR @ IMSc

INTEGRATED SYSTEMS APPROACHES TO PREDICT METABOLIC VULNERABILITIES OF CHEMO-RESISTANT GLIOBLASTOMA CELLS

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The field of cancer research is caught in a data deluge by the advent of inexpensive genome-scale high throughput technologies. The complexity of a living system justifies the need for data acquisition at all levels of cell hierarchy from DNA to tissue and organ level delineation. However, just listing candidate genes (from genomic/exome data), metabolite profiles or gene expression signatures (from transcriptomic data) are not enough to understand a complex, multi-hit, multifactorial emergent disease like cancer. Although there are many methods that exist to analyze individual data types, no method exists to put heterogenous data-types into a platform or mathematical model and integrate it, let alone predict outcomes and cell behavior. Glioblastoma, the most severe form of brain cancer is even more complex due to its inherent heterogeneity, as the only drug used to treat it is being rendered less useful due to chemo resistance.

To understand the difference between cells of glioblastoma that are resistant or susceptible to temozolomide a population of cells from the model cell line U87MG have been isolated and characterized extensively using whole exome sequencing, growth-resistance-metabolic profiling and metabolite respiration phenotyping to understand the intrinsic changes in its molecular components and higher order phenotypes. This talk will discuss these results in the context of a constraints-based tissue specific flux balance model of human metabolism. Such models not only explain the heterogeneity of cells and predict differences in the drug response but can predict alternate targetable cell vulnerabilities. Such scalable work flows could fill a critical need for predictive models for tumor growth and proliferation in personalized medicine.

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