UNIVERSITY OF DELAWARE

CENTER FOR BIOINFORMATICS & COMPUTATIONAL BIOLOGY

CBCB SEMINAR
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BIOINFORMATICS SEMINAR

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MS Student **UNIVERSITY OF DELAWARE**

INVESTIGATION OF SMALL MICRORNAS IN *TDRD7-/-* MAMMALIAN LENS

Clouding of the eye lens, termed cataract, is the leading cause of blindness worldwide. Mutations in Tdrd7 (Tudor domain containing protein 7) causes congenital cataract in human and mouse. Tdrd7 protein is an RNA granule component that is involved in several aspects of post-transcriptional regulation. It is predicted that Tdrd7 protein binds to other proteins through its Tudor domains and binds to RNA through its OST-HTH/LOTUS domains. MicroRNAs (miRNAs), a class of small noncoding RNAs, are also known to regulate gene expression post-transcriptionally and have been implicated in the pathogenesis of cataract. However, the impact of Tdrd7 deficiency on miRNAs in the lens is unexplored. In this study, we used Tdrd7-targeted germline knockout (Tdrd7-/-) mouse, that exhibit 100% penetrant cataracts similar to those in humans, as a model to investigate the impact of Tdrd7 on miRNAs. We performed RNA-seq to analyze small RNA expression in postnatal day 15 Tdrd7-/- mouse lens. Differential expression analysis identified significantly misregulated miRNAs in Tdrd7-/- mouse lens prior to the detection of overt cataract suggesting their potential involvement in gene expression control. Analysis of these differentially expressed miRNAs in the context of genome-wide mRNA expression profiling data and proteome data on Tdrd7-/- mouse lenses will inform on the miRNA regulatory network underlying lens defects resulting from Tdrd7 deficiency. In sum, this work identifies miRNAs downstream of Tdrd7, providing new evidence that supports a role for post-transcriptional regulatory factors in lens development and early onset cataractogenesis.

XIHAN QIN

PhD Student, Department of CIS **UNIVERSITY OF DELAWARE**

iTEXTMINE FRAMEWORK FOR ALZHEIMER'S AND OTHER NEURODEGENERATIVE DISEASES

Alzheimer's Disease (AD) is a progressive neurodegenerative disorder characterized typically by memory problems, and different levels of impairment of cognitive skills, among other symptoms. It is one of the leading causes of death in the US. Its underlying molecular mechanism is not yet fully understood, and there is no effective treatment available. With the aim of helping researchers in the field increase their pace of discovery of molecular mechanisms and therapeutics, we are adapting iTextMine to capture information from the literature related to neurodegenerative diseases. iTextMine is an integrative text mining framework to retrieve and extract relations relevant to proteins/genes with direct links to the evidence. iTextMine power relies on the integration of text mining results from diverse dockerized tools for a publication and across the bibliome (Medline). For this work, we are interested in capturing relations between protein/gene, variants, disease and drug response. There are two existing tools that seem relevant for these tasks: DiMeX, extracts gene mutation to disease associations, and eGARD extracts associations between genomic anomalies, disease and drug responses. Both tools have been successfully tailored and implemented in cancer-centric projects. We are now exploring if these text mining tools can be successfully integrated in iTextMine to extract information about neurodegenerative diseases. We are currently in the process of collecting relevant examples for the relations to be captured and disease terms. The overall introduction to AD, iTextmine framework and its integrated tools, challenges for mining AD and future work will be talked in the seminar.

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