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Diversity and evolution of the developmental transcriptome in flowering plants

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The development of higher plants requires an exquisite spatio-temporal regulation of their transcriptomes. Although organ and cell type-specific analyses of global gene expression patterns in the model plant *Arabidopsis thaliana* were performed a decade ago, the dynamic nature of the non-coding transcriptome is poorly understood. Using strand-specific total RNA-Seq, we have investigated the expression patterns of natural antisense transcripts (NATs), long-intergenic RNAs (linc RNAs) and other non-coding transcripts, as well as the tissue-specific usage of isoforms throughout *Arabidopsis* development. The evidence for an *in vivo* function has only been shown for a small fraction of long non-coding RNAs and splice variants in plants to date, we use a comparative approach to predict functional importance. By dissecting the developmental transcriptomes of seven plant species, we will be able to identify the conserved elements and provide insight into the evolutionary history of long non-coding RNA expression and differential exon usage patterns in flowering plants.

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Transcriptomic profiling of personal cell lines as drug response biomarker discovery tool

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Genome-wide pharmacogenomic studies for targeted therapies offer the advantage of hypothesis-free search for tentative drug response biomarkers (efficacy and safety). However, they require very large patient cohorts. My talk will present an alternative research approach as prelude to clinical studies in larger cohorts: Genome-wide transcriptomic profiling of a panel of human lymphoblastoid cell lines (LCLs) representing unrelated healthy donors. These cells are personal cell lines that can be obtained from many bio-banks, including our National Laboratory for the Genetics of Israeli Populations (NLGIP) at Tel Aviv. Our approach offers simple and inexpensive discovery of tentative drug response biomarkers and new potential drug targets. I will present our studies on SSRI response biomarkers for precision medicine of major depressive disorder (MDD). We found that lower expression of *CHL1* (close homologue of L1), coding for a neuronal cell-adhesion protein implicated in thalamocortical circuitry, is predictive for higher sensitivity to SSRI drugs. This, as well as preliminary findings from clinical studies (to be presented), support the role of *CHL1* expression as biomarker for SSRI sensitivity in MDD. These studies also implicate *ITGB3* (integrin beta-3), coding for another cell adhesion protein, in the mode of action of SSRI drugs. Further discussed examples will include transcriptomic profiling of LCLs for discovering lithium response biomarkers for precision medicine in bipolar disorder, and for early detection biomarkers for Alzheimer's disease.

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