Lithium (Li) is one of the most efficacious treatments for Bipolar Disorder (BD). Recent research on the genetics of BD has implicated genes affiliated with cell signaling and ion channels (CACNA1C, ANK3, and DGKH). Lithium is known to interact with and inhibit GSK3β and IMPases to modulate the wnt and phosphoinositol signaling pathways. However, much remains unknown with regards to the downstream gene expression changes affected by the regulation of these pathways.

**Methods**

We cultured Lymphoblastoid Cell Lines (LCL's) from the whole blood of 10 subjects diagnosed with BD. These cells were divided into 2 groups (one group bathed in Lithium at clinically relevant concentrations and the other group bathed in saline) and evaluated for gene expression changes over 16 days. On days 4, 8, and 16 respectively, we investigated for changes in genome-wide expression profiles. These data outline the lithium induction of hundreds of genes (FDR < 5%) from Bipolar lymphoblastoid cell lines. This provides further evidence in support of a genetic hypothesis in the explanation of lithium's mechanism of action.

EASE functional analysis of the 144 genes (nodes) in the C8orf33 network. Shown are the GO terms resulting from the EASE analysis.

**Conclusions**

These data outline the lithium induction of hundreds of genes (FDR < 5%) from Bipolar lymphoblastoid cell lines. This provides further evidence in support of a genetic hypothesis in the explanation of lithium’s mechanism of action.

MIMI has been useful in identifying the gene interaction networks involved with those genes whose expression was induced by lithium exposure.