Predicting Patient Response to Standard-of-care Therapies in Glomerular Diseases

ISN INTERNATIONAL SOCIETY OF NEPHROLOGY

Methods

Kidney biopsies
NEPTUNE study

RNAeq libraries
Glom (n=325), Tub (n=360)

Clinical phenotypes
(steroid and CNI exposure)

WGCN and FE analysis to identify modules associated with CR during CNI

Results

Identified modules

Nucleoside metabolism
Proximal tubule, adaptive PT, DTL

Purple
141 genes

Angiogenesis and vascular biology
Endothelial and glomerular cells

Dark red
32 genes
Lower in CNI resp.

Fatty acid oxidation and metabolic processes
Proximal tubule

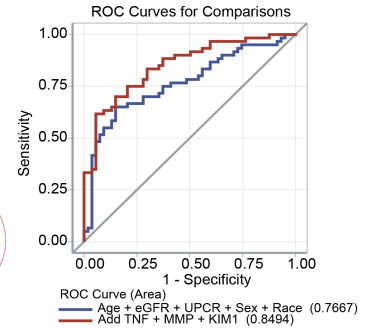
Red
747 genes

Higher in CNI resp

Turquoise
4726 genes
Lower in CNI resp.

Inflammation and cytokine signaling. Immune cells

Identified biomarkers



Glom: Glomeruli, Tub: Tubule, PT: proximal tubule, WGCN: Weighted gene co-expression network, FE: functional enrichment, CR: complete remission, CNI: calcineurin inhibitor, CNI resp: CNI responsive patients

Conclusion An integrated analysis identified non-invasive surrogates that predicted CNI response in patients with glomerular diseases. Non-invasive surrogates are being prioritized for validation in larger cohorts.

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