Microarray analysis of gene expression in FGS/Kist mouse kidney tissue

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Background

The etiology and pathogenesis of focal segmental glomerulosclerosis (FSGS) are still not understood, despite a few recent achievements in molecular pathogenesis. The FGS/Kist strain of mice is a new animal model for FSGS, manifesting spontaneous high proteinuria and glomerulosclerosis. We attempted to obtain the putative gene profiles related to the progression of kidney disease in this FSGS model, using oligonucleotide microarray technology.

Methods

We extracted RNA from renal cortex samples of 3 FGS/kist mice (disease) and 3 RFM/kist mice (control), and produced labeled cRNA for hybridization to Applied Biosystems 1700 Chemiluminescent Microarray analyzer.

Results

Among 13,261 genes filtered by S/N and flags<100, total 112 genes, each with a $P<0.05$, using quantile normalization and at least 1.5-fold change, were differentially expressed in kidney tissue from FGS/kist mice when compared with the control sample. The expression of 62 genes, including those involved in lipid metabolism and DNA metabolism and replication, was increased and the expression of 50 genes, including that associated with B-cell and antibody-mediated immunity, was decreased significantly.

Conclusions

Oligonucleotide DNA microarray analysis of kidney specimens identified a gene expression fingerprint for FGS mice, providing an overview on the biological process in FSGS. Further studies to clarify the role of the identified genes in the molecular pathogenesis of FSGS are required.