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Aldosterone-regulated miR-466-669 family controls cellular remodeling and cell cycle networks in the distal nephron

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Objectives : MicroRNAs (miRNA) are small non-coding RNAs that function as post-transcriptional regulators of gene expression through interactions with target mRNAs. Aldosterone regulates extracellular fluid volume and electrolyte balance in the aldosterone-sensitive distal nephron (ASDN). Excess aldosterone, as observed in primary aldosteronism (PA), promotes hypertension, kidney injury, renal tubular cell proliferation, and tissue remodeling. However, the regulatory networks linking aldosterone-responsive miRNAs to downstream gene expression remain poorly understood. Here, we investigated aldosterone-regulated miRNAs and their target genes to identify miRNA-mRNA regulatory networks.

Methods : Aldosterone-responsive miRNAs were identified by microarray analysis of kidney cortical tissues from aldosterone-infused mice (3 days), and miRNA genomic clusters were analyzed. Mouse cortical collecting duct (mpkCCD) cells were treated with aldosterone for 3 days, followed by RNA sequencing to identify differentially expressed genes (DEGs). miRNA-mRNA interactions were then analyzed by integrating datasets using experimentally validated miRNA-mRNA interaction data from the ENCORI/starBase database.

Results : Among 63 differentially expressed miRNAs ($p < 0.05$), many belonging to the miR-466-669 family were altered in response to aldosterone. miRNAs in Cluster 1 (miR-467c-3p, miR-669k-3p, miR-466e-3p, miR-467b-3p) were significantly decreased, whereas miRNAs in Cluster 2 (miR-467e-5p, miR-466a-3p, miR-466l-3p) were increased. In silico target prediction (microT-CDS, microT > 0.9 , $p < 0.05$) revealed strong enrichment in cell state transition processes, including the Wnt signaling pathway. KEGG and GO analysis of DEGs in aldosterone-treated mpkCCD cells showed strong enrichment of PI3K-Akt and MAPK pathways, with notable downregulation of cell cycle-related genes. Integration of miRNA and mRNA datasets showed coordinated regulation of the miR-466-699 family, with inverse expression patterns between miRNAs and target genes, supporting miRNA-mediated repression of downstream genes.

Conclusions : These findings suggest that aldosterone regulates miRNA-associated gene networks linked to cellular remodeling and cell cycle control. Further studies are needed to elucidate the functional roles of these miRNAs and target genes in aldosterone-induced renal responses.