OFFICIAL ABSTRACT and CERTIFICATION

An Omics Approach to Identify Model-agnostic Disease-driving Nodes in AKI: Implications for Drug Development

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Acute Kidney Injury (AKI) is the abrupt loss in kidney function caused by either injury or impairment. To classify the severity of cases of AKI, Acute Tubular Necrosis (ATN) scoring is utilized. Clinical treatments of AKI are often punctuated with failures. Lack of robust translational success can at least in part be explained by the fact that model systems may not fully recapitulate human AKI. It is therefore important to develop strategies that are governed by the same pathway in both human and animal models to properly treat AKI. This study examines the expression of Rho-associated protein kinase (ROCK) in AKI in mice induced by mercury chloride, folic acid and domoic acid and correlates expression with ATN scoring.
Prepared hematoxylin-eosin-stained kidney slides were obtained from mice treated with mercury chloride, folic acid or domoic acid and examined by microscopy. Tissues were categorized by the size of their urinary casts and scores were designed to mirror ATN scoring. Analysis of ROCK2 expression by RT-PCR showed that there was significantly increased in expression in all models when compared to the control (Sham). The highest level of ROCK2 expression can be found in the HgCl2 model of AKI. Moreover, renal ROCK2 expression level exhibited a significant and direct correlation with ATN scores. This is the first report of increased renal ROCK2 expression in multiple models of AKI accompanied by ATN. These data suggest that ROCK2 is a model agnostic injury driving node in AKI and potential therapeutic target for treatment.

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