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IN BRIEF

CHRONIC KIDNEY DISEASE

Changes in filtration markers associated with mortality

Kidney disease progression indicated by changes in filtration markers is associated with poor outcomes, according to new data. Among 9,716 ARIC study participants, a >30% decrease in estimated glomerular filtration rate determined using levels of creatinine, cystatin C or $\beta 2$ microglobulin, or a mean decline of >30% across all three markers, was associated with increased risks of cardiovascular disease and death during a median follow-up of 14 years.

Original article Rebholz, C. M. et al. Change in multiple filtration markers and subsequent risk of cardiovascular disease and mortality. Clin. J. Am. Soc. Nephrol. doi:10.2215/CJN.10101014

ACUTE KIDNEY INJURY

Renoprotective effects of CDK4/6 pathway inhibitors

Inhibitors of the cyclin-dependent kinase (CDK) 4/6 pathway could protect against drug-induced acute kidney injury (AKI), say researchers. They report that this pathway is activated together with renal cell-cycle entry before the development of AKI in mouse models of cisplatin nephrotoxicity. The CDK4/6 pathway inhibitors palbociclib and ribociclib ameliorated cisplatin-induced AKI and improved survival in mice. As well as preventing cell-cycle progression, palbociclib and ribociclib inhibited the function of organic cation transporter 2, which has a role in the uptake of cisplatin by renal cells.

Original article Pabla, N. et al. Mitigation of acute kidney injury by cell-cycle inhibitors that suppress both CDK4/6 and OCT2 functions. *Proc. Natl Acad. Sci.* doi:10.1073/pnas.1424313112

POLYCYSTIC KIDNEY DISEASE

Sec63 and Xbp1 regulate cyst formation

In mice, loss of the HSP40 co-chaperone Sec63 results in decreased expression of polycystin-1 and the formation of liver and kidney cysts. Now, using mouse models, researchers have shown that Sec63 exists in a complex with polycystin-1 and regulates the Ire1 α -Xbp1 unfolded protein response, which protects against cyst formation. Concomitant inactivation of Sec63 and Xbp1 suppressed maturation of polycystin-1 and exacerbated polycystic kidney disease, whereas overexpression of Xbp1 had a protective effect in the setting of impaired polycystin-1 biogenesis.

 $\label{eq:continuous} \textbf{Original article} \ \ \text{Fedeles, S.V.} \ \ \textit{et al. Sec63} \ \ \text{and } \ \textit{Xbp1} \ \ \text{regulate IRE1} \alpha \ \ \text{activity and polycystic disease severity.} \ \textit{J. Clin. Invest. doi:} \ 10.1172/JCI78863$

HYPERTENSION

Management of hypertensive disorders of late pregnancy

New data from a randomized controlled trial suggest that a strategy of expectant monitoring aimed at prolonging pregnancy until 37 weeks of gestation might be preferable to immediate delivery in women with nonsevere hypertensive disorders of late pregnancy. In this study, which included 703 women, the composite incidence of adverse maternal outcomes (including thromboembolic disease and eclampsia) did not differ significantly between the expectant monitoring and immediate delivery groups (3.1% versus 1.1%), whereas the incidence of neonatal respiratory distress syndrome was significantly higher in the immediate delivery group (5.7% versus 1.7%).

Original article Broekhuijsen, K. et al. Immediate delivery versus expectant monitoring for hypertensive disorders of pregnancy between 34 and 37 weeks of gestation (HYPITAT-II): an open-label, randomised controlled trial. *Lancet* doi:10.1016/S0140-6736(14)61998-X