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IN BRIEF

HYPERTENSION

Role of p67^{phox} in salt-sensitive hypertension

A study has shown that a genetic variance in the promoter region of the NAD(P)H oxidase subunit *p67^{phox}* is associated with higher promoter activity, which could contribute to enhanced salt sensitivity. Levels of *p67^{phox}* were upregulated in an animal model of salt-sensitive hypertension, leading to an increase in NAD(P)H oxidase activity. By contrast, disruption of *p67^{phox}* led to a reduction in oxidative stress, salt sensitivity and renal injury. p67^{phox} may therefore represent a therapeutic target for salt-sensitive hypertension.

Original article Feng, D. *et al.* Increased expression of NAD(P)H oxidase subunit p67^{phox} in the renal medulla contributes to excess oxidative stress and salt-sensitive hypertension. *Cell Metab.* **15**, 201–208 (2012)

TRANSPLANTATION

Non-HLA antibodies predict development of CAI

A humoral response to mismatched HLA antigens is an important contributor to chronic allograft injury (CAI). However, a panel of non-HLA antibodies that correlate with CAI have now been identified and validated in renal transplant recipients. The researchers say that measuring pretransplant serum levels of antibodies to CXCL9, CXCL11, IFN-γ and glial-derived neurotrophic factor could be used to stratify risk of developing CAI.

Original article Sigdel, T. K. *et al.* Non-HLA antibodies to immunogenic epitopes predict the evolution of chronic renal allograft injury. *J. Am. Soc. Nephrol.* doi:10.1681/ASN.2011060596

BASIC RESEARCH

Ischemic conditioning provides myocardial protection in a rodent model of uremia

Patients with chronic kidney disease who experience acute myocardial infarction might benefit from ischemic conditioning strategies, suggest researchers. Byrne *et al.* found that animals with uremia sustained larger myocardial infarctions than nonuremic animals, but conditioning strategies (ischemic preconditioning, remote ischemic preconditioning and postconditioning) significantly reduced myocardial infarct size. Components of the RISK and SAFE pathways were implicated in mediating this cardioprotection.

Original article Byrne, C. J. *et al.* Ischemic conditioning protects the uremic heart in a rodent model of myocardial infarction. *Circulation* doi:10.1161/CIRCULATIONAHA.111.055392

TRANSPLANTATION

Protective effects of erythropoietin in transplant patients

A study has demonstrated that erythropoietin prevents chronic allograft injury via mechanisms beyond anemia correction. In contrast to erythropoietin, blood transfusions did not prevent chronic allograft injury in rats despite normalization of hemoglobin levels. The researchers suggest that the effects of erythropoietin might be mediated by the upregulation of angiogenic and antiapoptotic factors.

Original article Cassis, P. *et al.* Erythropoietin, but not the correction of anemia alone, protects from chronic kidney allograft injury. *Kidney Int.* doi:10.1038/ki.2011.473