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The Renal Association, UK

T-type calcium channel inhibition: A novel therapy in renal disease

There is a lack of effective therapeutic options for patients with Chronic Kidney Disease (CKD) related to IgA nephropathy and proteinuric nephropathies. Treatment with inhibitors of the Renin Angiotensin System (RAS) is standard but residual risk of progression of CKD remains high. In this context this lecture will explore an innovative strategy for therapy in CKD targeting T-type calcium channels. T-Type Calcium Channels (TTCC) are closely related to the more familiar L-Type Calcium Channels (LTCC). We have extended work on the role of TTCC in smooth muscle proliferation by demonstrating that TTCC have a role in mesangial cell function. TTCC are also expression in the efferent arteriole and TTCC inhibition reduces glomerular capillary pressure. *In vitro* human and rat mesangial cell proliferation is dependent on TTCC and not LTCC. Moreover, in models of glomerular cell proliferation, inhibition of TTCC reduces glomerular damage, reduces cell proliferation and inhibits monocyte infiltration, with improved renal function. In parallel with this work a series of studies in Japan has demonstrated that TTCC inhibition reduces glomerular proteinuria in animal studies and in small clinical studies of diabetic nephropathy. Taken together these studies provide the basis for optimism about TTCC inhibition as a new therapy in renal diseases where mesangial cell proliferation is coupled with significant proteinuria. Such diseases include IgA Nephropathy, Diabetic Nephropathy and lupus nephritis.

Biography

Bruce Hendry is President of the UK Renal Association and Emeritus Professor of Renal Medicine at King's College London. He graduated in medicine from the University of Oxford and received his PhD in Biophysics from the University of Cambridge. His major research interests are in the cell and molecular biology of progressive renal fibrosis and in the design of novel therapeutic approaches. His current work is focused on the study of new approaches to the problem of aberrant renal cell proliferation leading to fibrosis; this work includes the use of antisense and small molecule strategies. Recent work has examined the role of antisense as therapy in renal fibrosis and ADPKD and the targeting of T-type calcium channels in glomerular disease. He has clinical research interests in diabetic nephropathy, HIV and the kidney and in polycystic kidney disease.

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