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Foreword

Secondary hyperparathyroidism (SHPT) develops in chronic kidney disease (CKD) as a consequence of disturbances in mineral metabolism and vitamin D synthesis [1]. Alterations in signalling via the parathyroid gland calcium-sensing receptor (CaR) are known to play a central role in the development of SHPT [2,3], making the CaR a therapeutic target in the management of this condition. Type II calcimimetics act as allosteric modulators of the CaR, increasing its sensitivity to calcium and inhibiting parathyroid hormone (PTH) secretion [4,5]. Consequently, the efficacy of calcimimetics in treating SHPT and improving outcomes for CKD patients has attracted a great deal of attention within the medical community.

A panel of experts presented data at the symposium 'Calcimimetics: Changing the Treatment Paradigm of SHPT,' in Barcelona, Spain, to share preclinical and clinical findings on the pathophysiology and treatment of SHPT, and new directions in disease management. This supplement comprises seven papers developed from the presentations given at that meeting. In the first of these articles, Dr Cannata-Andía and I describe the burden that SHPT represents for CKD patients and the need for new and more effective treatments. The second article, by Drs Riccardi and Martin, explores the pathogenesis of SHPT, with a special emphasis on the central role of the CaR in the development of the disease. It also discusses the results of preclinical studies that have shown the potential of calcimimetic agents to retard the progression of SHPT. In another article, Drs Frazão and Rodriguez examine recent data suggesting that treatment with calcimimetics may stabilize progression of SHPT.

Although calcimimetic treatment is relatively new and not yet included in international clinical guidelines, a variety of different treatment regimens incorporating calcimimetics have been explored in clinical trials. In their article, Drs Bushinsky and Messa describe the potential of calcimimetics combined with low-dose vitamin D treatment to improve treatment of SHPT. Dr de Francisco and I extend these concepts in our article, which explores whether improved control of SHPT might be achieved by a new treatment paradigm using calcimimetics with other therapies, including calcium-based and calcium-free phosphate binders and vitamin D. Building on the studies that have demonstrated improved control of serum PTH and biochemistry during calcimimetic treatment, the article by Drs Cunningham, Floege, London, Rodriguez and Shanahan presents findings from recent preclinical and clinical studies, showing that using calcimimetics to control SHPT may also result in improved outcomes for CKD patients.

The calcimimetic cinacalcet is currently approved for the treatment of SHPT in dialysis patients, but there has been interest in its use in other patient populations. In the final article of this supplement, Drs Chonchol and Wüthrich discuss recent studies that have explored the potential use of calcimimetic treatment in two diverse groups: stage 3 and 4 CKD patients, and renal transplant recipients.

It is our sincere hope that this supplement will prove to be a valuable educational resource for the nephrology community. Furthermore, we hope that the information presented will stimulate debate on novel treatments for SHPT, ultimately leading to improved outcomes for CKD patients.

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