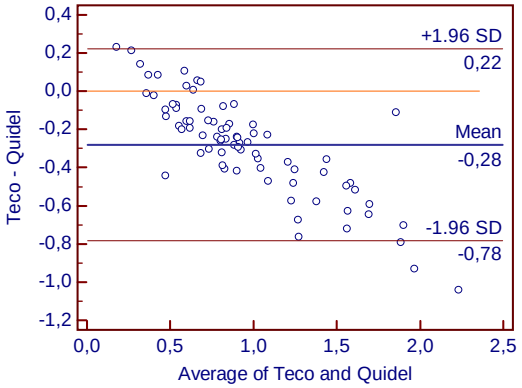
EVALUATION OF SCLEROSTIN LEVELS IN PATIENTS SUFFERING FROM CHRONIC KIDNEY DISEASES

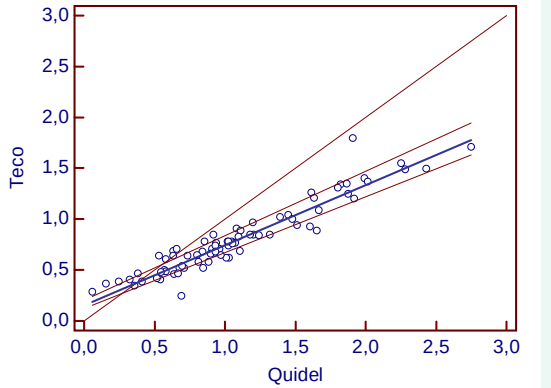
E CAVALIER (1), A CARLISI (1), N FERRANTE, O ROUSSELLE (1),JP CHAPELLE, P DELANAYE (2)
Departments of (1) Clinical Chemistry, and (2) Nephrology Dialysis and Transplantation,
University of Liège, CHU Sart-Tilman, Belgium

Background: Sclerostin (Sost) is a new promising bone marker. Indeed, it inhibits bone formation by regulating osteoblast function and promoting osteoblast apoptosis. Sclerostin also antagonises osteoblast diffentiation by blocking the Wnt-pathway. Finally, sclerostin is produced by the osteocytes and could be a new therapeutical target for vascular calcification. However, little is known about sclerostin in patients suffering from chronic kidney diseases (CKD). In this study, we aimed to correlate sclerostin levels with different bone markers in stage 3 and 4 CKD patients.

Methods: We used the remnant serum of 77 stage 3 and 4 non renal-transplanted patients (64.4±13.5 yo; 46 female) to determine sclerostin levels with 2 different Elisas:
-Quidel Corporation, Santa Clara, CA
-Biomedica, Vienna, Austria).
N-terminal propetide of type 1 collagen (P1NP), C-terminal telopeptides of type1 collagen (CTX) and intact PTH (iPTH) levels were determined with the IDS iSYS (IDS, Boldon, UK). Creatinine (enzymatic, IDMS traceable), calcium and phosphorus levels were determined on Roche Modular (Roche, Mannheim, Germany) and the estimation of glomerular filtration rate (eGFR) was achieved with the MDRD formula.

Results: No correlation was observed between sclerostin levels and the different bone markers or iPTH. Sclerostin levels were not influenced by the glomerular filtration rate. The 5th and 95th percentiles observed with respectively the Quidel and the Biomedica assays were 0.38 – 1.50 and 0.34 – 2.17 pg/mL, respectively.





$$y = 0{,}59\,x + 0{,}15$$

Discussion: In this study, we showed for the first time that sclerostin levels were not influenced by renal deficiency. We also showed, that, contrary to other studied populations, like patients presenting disorders of the parathyroid gland (Costa AG, JCEM 2011) or type 2 diabetes (Garcia-Martin A, JCEM 2012), sclerostin levels were not associated with bone formation or resorption markers.

Conclusions: As it is not influenced by the GFR, sclerostin could be a new marker of choice for the exploration of CKD-Mineral bone disease and vascular calcifications. The lack of correlation with the traditional bone markers in CKD patients is intriguing and, if confirmed in larger studies, opens a new field of research (the osteocyte function) in these patients. Finally, the Quidel and Biomedica assays give similar results, even if a standardisation is needed.