HIGH DOSES OF CALCITRIOL LEAD TO ALTERATIONS IN OSTEOGENESIS AND BONE DISEASE

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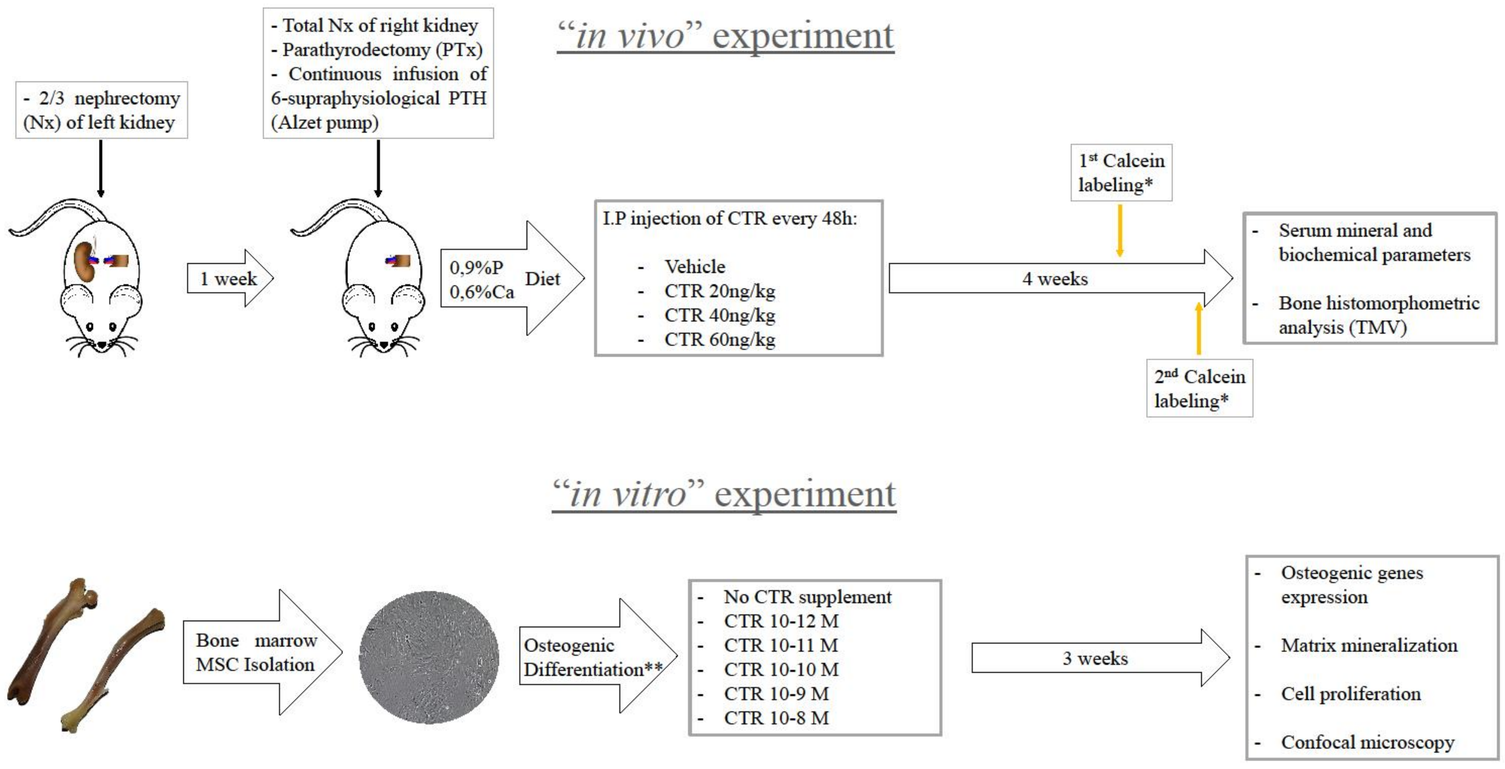
INTRODUCTION

In chronic kidney disease (CKD) patients, active vitamin D levels (CTR) are reduced due to the impairment of the renal 1-α-hydroxylase, leading to low serum calcium and high PTH[1]. In order to normalize mineral parameters, CTR supplementation is widely prescribed in CKD patient[2,3]. In the other hand, bone is mainly affected by mineral abnormalities, and CKD patients commonly develop bone disorders and fractures[4]. In this study, we investigated the dose-dependent impact of CTR supplementation on bone in a rat model of CKD and an in vivo model of osteoblasts differentiation from mesenchymal stem cells.

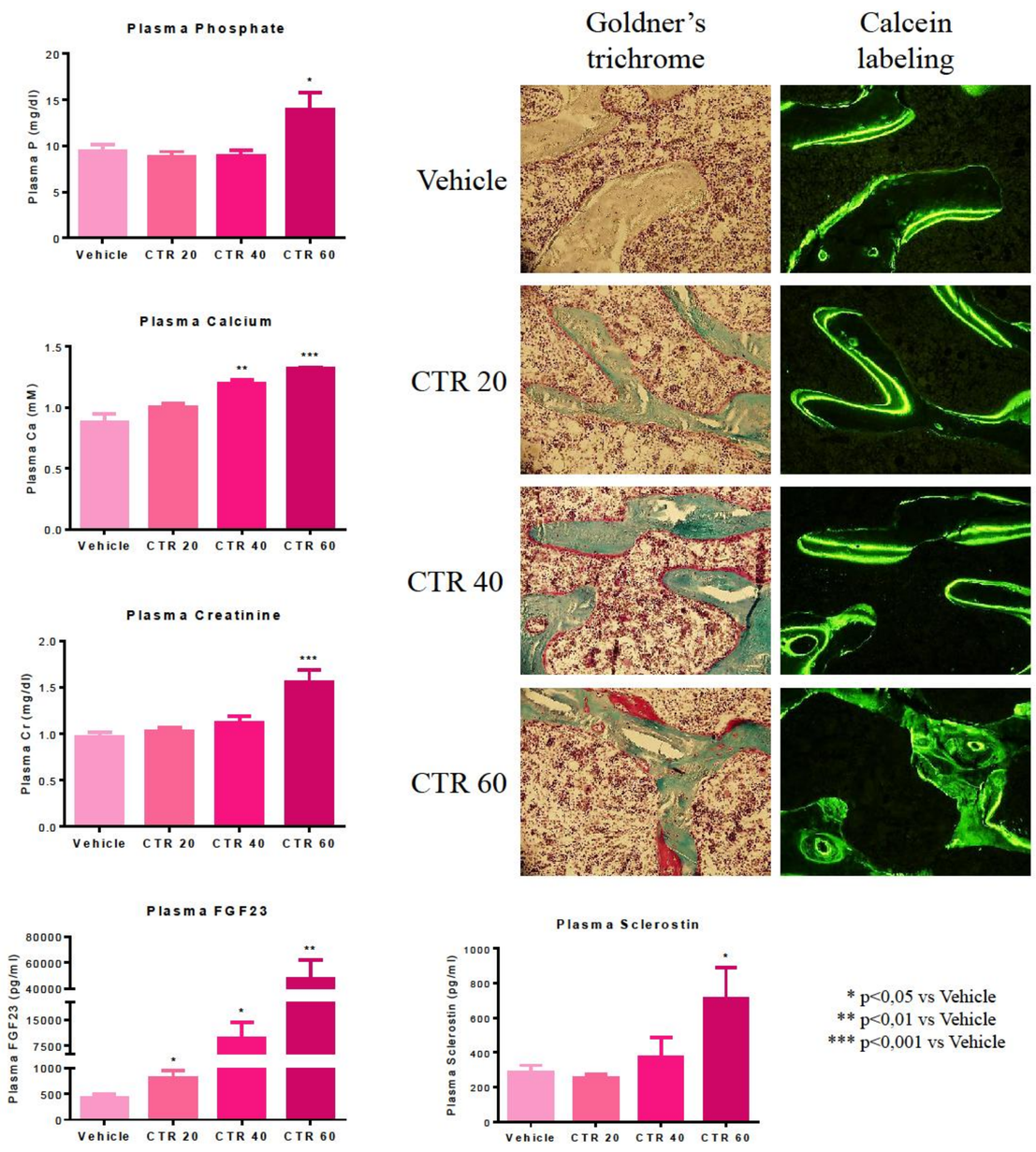
METHODS



* Mineralization was examined by calcein staining. 25 mg/kg of calcein was administered at days 9 and 2 before sacrifice.
** Osteogenic stimuli was based on Dexamethasone 1μM, ascorbic acid 0,2 mM and β-Glycerol-phosphate 10mM)

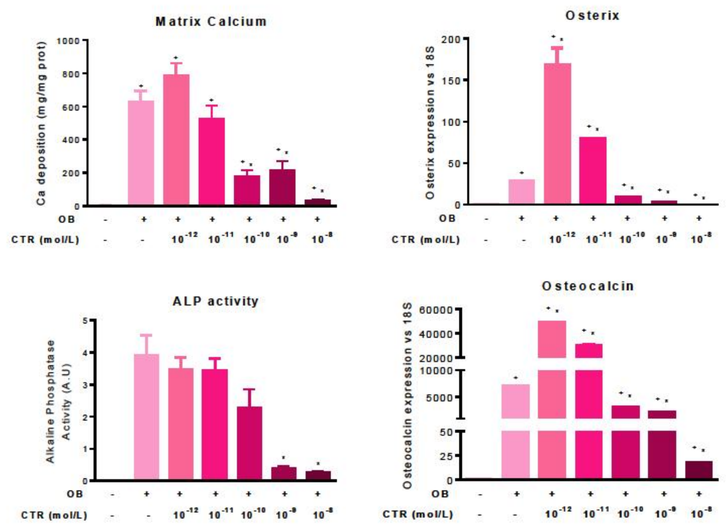
RESULTS

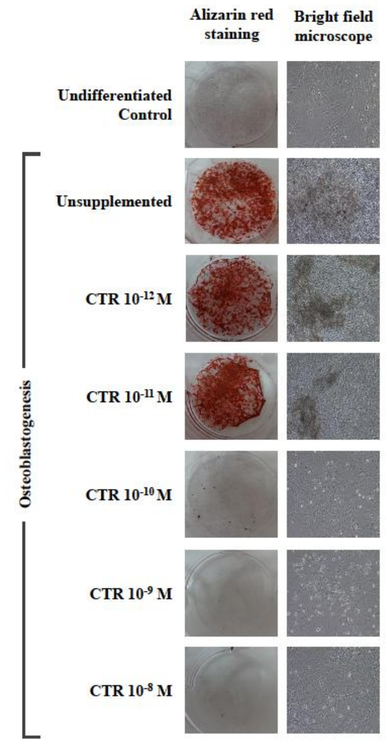
"in vivo experiment"

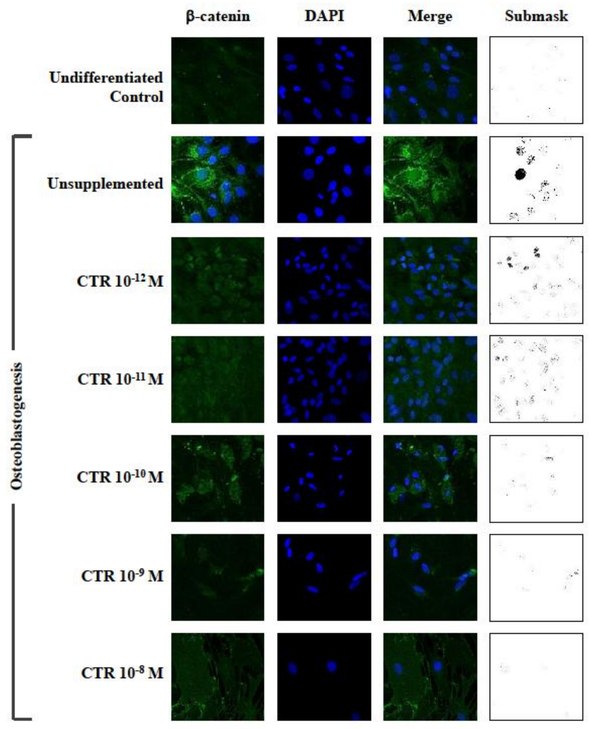


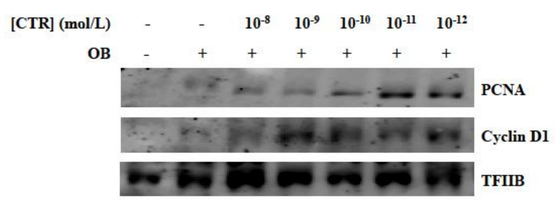
* p<0,05 vs Vehicle
** p<0,01 vs Vehicle
*** p<0,001 vs Vehicle

"in vitro experiment"









Magnifications: Bright field microscope images 200x; Confocal microscopy images 400x.

+ p<0,05 vs undifferentiated control
* p<0,01 vs OB

CONCLUSIONS

High dose of Calcitriol, independently of its bone effect mediated by a reduction in PTH levels, impairs mineralization and decreases osteogenesis which directly favors the development of adynamic bone disease.

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Affiliations:

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