

TREATMENT WITH PTH 1-84 INFLUENCES GLUCOSE **METABOLISM TROUGH OSTEOCALCIN**



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INTRODUCTION

In the recent years it has been demonstrated bone turnover may influence fat and glucose homeostasis mainly trough the osteoblast-specific protein osteocalcin (OC), in its undercarboxylated form (uOC).







To evaluate the effect of bone anabolic treatment with 1-84 PTH on glucose metabolism.



• We enrolled in the study 43 women affected by postmenopausal osteoporosis; the patients were randomly assigned to treatment with: - 1-84 PTH 100 µg plus calcium 1200 mg and vitamin D 800 UI daily (21, iPTH)

- calcium 1200 mg and vitamin D 800 UI daily (22)

• Glucose and bone metabolism were evaluated at basal and after 3, 6, 12 and 18 months of treatment.

RESULTS

iPTH increases bone formation The administration of intermittent PTH significantly increases markers of bone turnover, and in particular it increases OC and uOC at each time point.

iPTH lowers plasma glucose In patients treated with PTH there is a significant decrease in fasting plasma glucose (FPG), without effect on insulin secretion, resistance and β -cell functions measured by OGTT derived parameters and by serum amylin. iPTH does not influence fat mass and adipokines production.

To evaluate if the increase in uOC and OC mediates the effect of treatment on FPG we

Calcium and vitamin D $\overline{}$ Calcium and vitamin D + iPTH 10 p<0.001 p<0.028 **100** 8 (Jm/gu) 20n 90 (lp/bu) 80 FPG

adopted a mediation analyses model.

Our analyses suggest that the effect of PTH on FPG is partially mediated by its effect on OC (61.2%).

Regression coefficients for FPG vs OC and treatment		
	Coefficient	95% CI
Treatment θ_1	0.034	-0.208, 0.277
Log (OC) θ_2	0.037	-0.053, 0.127
Interaction θ_3	-0.063	-0.175, 0.049
Constant θ_0	4.347	4.234, 4.461
Regression co	pefficients for OC vs	treatment
	Coefficient	95% CI
Treatment β_1	2.019	1.492, 2.546
Constant β_2	1.073	0.701, 1.446
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In this study we show that PTH treatment influences fasting plasma glucose levels partially through its anabolic effect on bone turnover and particularly by the increase in OC, otherwise we cannot find an effect of treatment mediated by uOC, this could be due to the lower effect of PTH treatment on uOC as compared to OC.

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