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Osteocalcin regulates murine and human fertility through a pancreas-bone-testis axis

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Corrigendum

Original citation: J Clin Invest. 2013;123(6):2421–2433. doi:10.1172/JCI65952. Citation for this corrigendum: J Clin Invest. 2014;124(12):5522. doi:10.1172/JCI79293. In the original article, the substitution mutation in GPRC6A at F464Y was erroneously described as located in one of the transmembrane domains of GPRC6A; however, the F464Y mutation is located in the long N-terminal region of GPRC6A (1-594AA). This error affected portions of the text in the Abstract, Introduction, Results, and Discussion. The corrected sentences appear below. Abstract (page 2421): To determine the importance of osteocalcin in humans, we analyzed a cohort of patients with primary testicular failure and identified 2 individuals harboring the same heterozygous missense variant of GPRC6A, which prevented the receptor from localizing to the cell membrane. Introduction (page 2421): In trying to expand the biological relevance of osteocalcin from mouse to human, we identified in 2 patients with peripheral testicular failure the same amino acid substitution affecting a highly conserved residue in the long N-terminal domain (1-594AA) of GPRC6A. Results (page 2429): We sequenced all exons of Osteocalcin and GPRC6A, the receptor mediating osteocalcin reproductive function in Leydig cells (2), in these patients. Two patients in this cohort harbored a T→A transversion in exon 4 (g.117121904A/T), resulting in an amino acid substitution in the long N-terminal domain of GPRC6A (F464Y) (Figure 7, A and B, and Supplemental Figure 6A). Discussion […]

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Discussion (page 2431):
This missense mutation affected a highly conserved residue, occurred in the N-terminal domain region of the molecule, and prevented its localization to the cell membrane, therefore resulting in a loss of function of GPRC6A.

The authors regret the error.