Correction

Correction: Glucocorticoid impairs cell-cell communication by autophagy-mediated degradation of connexin 43 in osteocytes

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This article has been corrected: Due to a software malfunction while cropping the western blot image into the PDF, the previous image for β -action (in IGF-1+Dex group) and for Akt T³⁰⁸-P (in Dex group) in Figure 6A is incorrect. The corrected Figure 6A is shown below. The original image for western blot is also attached. The authors declare that these corrections do not change the results or conclusions of this paper.

Original western blot image	A Dex IGF-1+Dex
Dex IGF+Dex Ctrl 6 12 18 24 Ctrl 6 12 18 24 (hrs)	Ctrl 6 12 18 24 6 12 18 24 (hrs) Akt 75kDa Akt 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa 75kDa
Original western blot image IGF+Dex Dex Ctrl 6 12 18 24 Ctrl 6 12 18 24 (hrs)	p70S6K1 75kDa LC3-I LC3-I LC3-I Cx43 57kDa β-actin 57kDa 37kDa 37kDa 37kDa

Figure 6: Akt-mTORC1 signaling pathway is involved in Dex-induced autophagy and Cx43 degradation. (A) Dex inhibition of Akt and p70S6K phosphorylation, LC3 lipidation and Cx43 degradation were attenuated by the potent Akt activator, IGF-1. TCPs from MLO-Y4 cells treated with 10-6M Dex alone or in combination with 100nM IGF-1 for indicated times were immunoblotted with antibodies against p-Akt T308, total Akt, p-p70S6K1 T389, total p70S6K1, LC3 I/II, and Cx43. β-actin served as loading and normalization control.

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