Figure. S1. Effect of GlcN on hOCs and hOBs viability. (A) hMCs were isolated from peripheral blood and used as a source of osteoclast progenitors. The ability of hMCs to differentiate into hOCs in presence of MCSF (25 ng/ml) and RANKL (30 ng/ml) for 14 days was evaluated by measuring TRAP activity, as well as cathepsin K and NFATc1 expression (scale bars, 50 μm). The effect of different GlcN concentrations (10, 100 and 200 μg/ml) on the viability of hOCs was also evaluated via an MTT assay. Data are presented as the percentage of surviving cells (mean ± SD, n=3). The viability of control cells was normalized to 100%. (B) hMCs from patients with OA and healthy donors were cultured in osteoclast differentiation media with different doses of GlcN (100 and 200 μg/ml) for 14 days. Osteoclasts were detected by TRAP staining and counting cell numbers (cells with >3 nuclei were considered osteoclasts), and quantified per area (scale bars, 50 μm). Data are presented as the mean ± SD (healthy donors, n=3; patients with OA, n=7). (C) hOBs were obtained from vertebral lamina. The expression of OPN, COL1a1 and RUNX2 osteogenic markers was evaluated via immunocytochemistry in hOBs at P2 passage of culture (scale bars, 20 μm). The effect of different GlcN concentrations (10, 100 and 200 μg/ml) on the viability of hOBs was also evaluated via an MTT assay. Data are presented as the percentage of surviving cells (mean ± SD, n=3). The viability of control cells was normalized to 100%. COL1a1, collagen type 1α; CTR, control; GlcN, glucosamine; hMC, human primary monocyte; hOB, human primary osteoblast; hOC, human primary osteoclast; NFATc1, nuclear factor of activated T-cells, cytoplasmic 1; OA, osteoarthritis; OPN, osteopontin; RUNX2, runt-related transcription factor 2; TRAP, tartrate-resistant acid phosphatase.