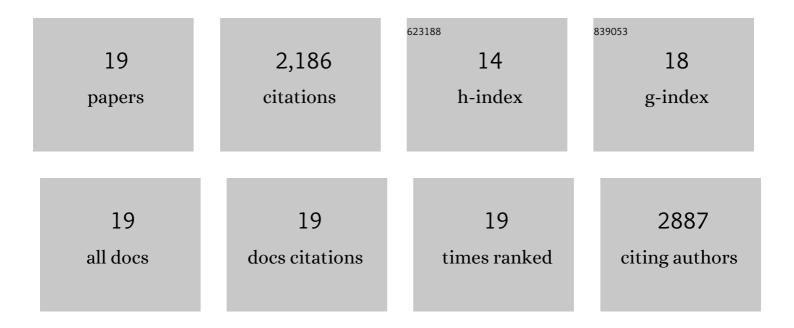
## Melda Onal

List of Publications by Year in descending order

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Μείδα Οναι

#	Article	IF	CITATIONS
1	Matrix-embedded cells control osteoclast formation. Nature Medicine, 2011, 17, 1235-1241.	15.2	1,115
2	Osteocytes, not Osteoblasts or Lining Cells, are the Main Source of the RANKL Required for Osteoclast Formation in Remodeling Bone. PLoS ONE, 2015, 10, e0138189.	1.1	236
3	Receptor Activator of Nuclear Factor κB Ligand (RANKL) Protein Expression by B Lymphocytes Contributes to Ovariectomy-induced Bone Loss. Journal of Biological Chemistry, 2012, 287, 29851-29860.	1.6	202
4	Suppression of Autophagy in Osteocytes Mimics Skeletal Aging. Journal of Biological Chemistry, 2013, 288, 17432-17440.	1.6	165
5	Low bone mass and changes in the osteocyte network in mice lacking autophagy in the osteoblast lineage. Scientific Reports, 2016, 6, 24262.	1.6	83
6	A kidney-specific genetic control module in mice governs endocrine regulation of the cytochrome P450 gene Cyp27b1 essential for vitamin D3 activation. Journal of Biological Chemistry, 2017, 292, 17541-17558.	1.6	74
7	Genomic Determinants of Vitamin D-Regulated Gene Expression. Vitamins and Hormones, 2016, 100, 21-44.	0.7	67
8	A Novel Distal Enhancer Mediates Inflammationâ€, PTHâ€, and Early Onset Murine Kidney Diseaseâ€Induced Expression of the Mouse <i>Fgf23</i> Gene. JBMR Plus, 2018, 2, 31-46.	1.3	52
9	Suppression of autophagy in osteocytes does not modify the adverse effects of glucocorticoids on cortical bone. Bone, 2015, 75, 18-26.	1.4	46
10	Deletion of the Distal <i>Tnfsf11</i> RL-D2 Enhancer That Contributes to PTH-Mediated RANKL Expression in Osteoblast Lineage Cells Results in a High Bone Mass Phenotype in Mice. Journal of Bone and Mineral Research, 2016, 31, 416-429.	3.1	33
11	The RANKL Distal Control Region Is Required for the Increase in RANKL Expression, But Not the Bone Loss, Associated with Hyperparathyroidism or Lactation in Adult Mice. Molecular Endocrinology, 2012, 26, 341-348.	3.7	27
12	Effective CRISPR interference of an endogenous gene via a single transgene in mice. Scientific Reports, 2019, 9, 17312.	1.6	25
13	A Control Region Near the Fibroblast Growth Factor 23 Gene Mediates Response to Phosphate, 1,25(OH)2D3, and LPS In Vivo. Endocrinology, 2019, 160, 2877-2891.	1.4	20
14	A DNA Segment Spanning the Mouse <i>Tnfsf11</i> Transcription Unit and Its Upstream Regulatory Domain Rescues the Pleiotropic Biologic Phenotype of the RANKL Null Mouse. Journal of Bone and Mineral Research, 2015, 30, 855-868.	3.1	18
15	Absence of the Vitamin D Receptor Inhibits Atherosclerotic Plaque Calcification in Female Hypercholesterolemic Mice. Journal of Cellular Biochemistry, 2017, 118, 1050-1064.	1.2	7
16	Loss of chaperone-mediated autophagy is associated with low vertebral cancellous bone mass. Scientific Reports, 2022, 12, 3134.	1.6	6
17	Deletion of a Distal RANKL Gene Enhancer Delays Progression of Atherosclerotic Plaque Calcification in Hypercholesterolemic Mice. Journal of Cellular Biochemistry, 2017, 118, 4240-4253.	1.2	4
18	Deletion of a putative promoter-proximal Tnfsf11 regulatory region in mice does not alter bone mass or Tnfsf11 expression in vivo. PLoS ONE, 2021, 16, e0250974.	1.1	4

#	Article	IF	CITATIONS
19	Genome-Wide Perspectives on Vitamin D Receptor–Mediated Control of Gene Expression in Target Cells. , 2018, , 141-174.		2