Laura R Mccabe

List of Publications by Year in descending order

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LALIDA R MCCARE

#	Article	IF	CITATIONS
1	Longitudinal effects of growth restriction on the murine gut microbiome and metabolome. American Journal of Physiology - Endocrinology and Metabolism, 2022, 323, E159-E170.	3.5	1
2	Loss of interleukinâ€10 exacerbates early Typeâ€1 diabetesâ€induced bone loss. Journal of Cellular Physiology, 2020, 235, 2350-2365.	4.1	12
3	Involvement of the Gut Microbiota and Barrier Function in Glucocorticoidâ€Induced Osteoporosis. Journal of Bone and Mineral Research, 2020, 35, 801-820.	2.8	101
4	Post-antibiotic gut dysbiosis-induced trabecular bone loss is dependent on lymphocytes. Bone, 2020, 134, 115269.	2.9	29
5	Oestrogen-deficiency induces bone loss by modulating CD14+ monocyte and CD4+ T cell DR3 expression and serum TL1A levels. BMC Musculoskeletal Disorders, 2019, 20, 326.	1.9	8
6	Beneficial effects of Lactobacillus reuteri 6475 on bone density in male mice is dependent on lymphocytes. Scientific Reports, 2019, 9, 14708.	3.3	28
7	Probiotic <i>Lactobacillus reuteri</i> Prevents Postantibiotic Bone Loss by Reducing Intestinal Dysbiosis and Preventing Barrier Disruption. Journal of Bone and Mineral Research, 2019, 34, 681-698.	2.8	119
8	Characterizing how probiotic Lactobacillus reuteri 6475 and lactobacillic acid mediate suppression of osteoclast differentiation. Bone Reports, 2019, 11, 100227.	0.4	22
9	Therapeutic Targeting of Gut-Bone Signaling to Treat Osteoporosis. , 2019, , 169-181.		0
10	Novel leptin receptor signaling mutants identify location and sexâ€dependent modulation of bone density, adiposity, and growth. Journal of Cellular Biochemistry, 2019, 120, 4398-4408.	2.6	9
11	Exercise prevents high fat diet-induced bone loss, marrow adiposity and dysbiosis in male mice. Bone, 2019, 118, 20-31.	2.9	69
12	Alterations to the Gut Microbiome Prevent Glucocorticoid induced Osteoporosis. FASEB Journal, 2019, 33, 589.6.	0.5	1
13	2,3,7,8-Tetrachlorodibenzo-p-dioxin dose-dependently increases bone mass and decreases marrow adiposity in juvenile mice. Toxicology and Applied Pharmacology, 2018, 348, 85-98.	2.8	17
14	Advances in Probiotic Regulation of Bone and Mineral Metabolism. Calcified Tissue International, 2018, 102, 480-488.	3.1	61
15	High Molecular Weight Polymer Promotes Bone Health and Prevents Bone Loss Under Salmonella Challenge in Broiler Chickens. Frontiers in Physiology, 2018, 9, 384.	2.8	19
16	Microbiota Reconstitution Does Not Cause Bone Loss in Germ-Free Mice. MSphere, 2018, 3, .	2.9	36
17	G protein-coupled receptor kinase-2-deficient mice are protected from dextran sodium sulfate-induced acute colitis. Physiological Genomics, 2018, 50, 407-415.	2.3	8
18	ERAP1 deficient mice have reduced Type 1 regulatory T cells and develop skeletal and intestinal features of Ankylosing Spondylitis. Scientific Reports, 2018, 8, 12464.	3.3	24

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19	Targeting the Intestine to Treat Osteoporosis. FASEB Journal, 2018, 32, .	0.5	ο
20	CCL3 and MMP-9 are induced by TL1A during death receptor 3 (TNFRSF25)-dependent osteoclast function and systemic bone loss. Bone, 2017, 97, 94-104.	2.9	28
21	Quick and inexpensive paraffin-embedding method for dynamic bone formation analyses. Scientific Reports, 2017, 7, 42505.	3.3	25
22	Estrogen Deficiency Exacerbates Type 1 Diabetes–Induced Bone TNF-α Expression and Osteoporosis in Female Mice. Endocrinology, 2017, 158, 2086-2101.	2.8	39
23	Temporal and regional intestinal changes in permeability, tight junction, and cytokine gene expression following ovariectomy-induced estrogen deficiency. Physiological Reports, 2017, 5, e13263.	1.7	43
24	G-protein-coupled receptor kinase-2 is a critical regulator of TNFα signaling in colon epithelial cells. Biochemical Journal, 2017, 474, 2301-2313.	3.7	10
25	Intestinal Microbiota and Bone Health: The Role of Prebiotics, Probiotics, and Diet. Molecular and Integrative Toxicology, 2017, , 417-443.	0.5	8
26	Role of G protein-coupled receptor kinase-6 in <i>Escherichia coli</i> lung infection model in mice. Physiological Genomics, 2017, 49, 682-689.	2.3	3
27	Recent Advances in Intestinal Stem Cells. Current Molecular Biology Reports, 2017, 3, 143-148.	1.6	6
28	Probiotics in Gut-Bone Signaling. Advances in Experimental Medicine and Biology, 2017, 1033, 225-247.	1.6	47
29	Immunology of Gut-Bone Signaling. Advances in Experimental Medicine and Biology, 2017, 1033, 59-94.	1.6	19
30	Epithelial Barrier Function in Gut-Bone Signaling. Advances in Experimental Medicine and Biology, 2017, 1033, 151-183.	1.6	36
31	G Protein-Coupled Receptor Kinases in the Inflammatory Response and Signaling. Advances in Immunology, 2017, 136, 227-277.	2.2	29
32	The Potential of Probiotics as a Therapy for Osteoporosis. Microbiology Spectrum, 2017, 5, .	3.0	112
33	Cytokine and hormonal regulation of bone marrow immune cell Wnt10b expression. PLoS ONE, 2017, 12, e0181979.	2.5	7
34	Interleukinâ€10 in Type 1 Diabetesâ€induced bone loss in mice. FASEB Journal, 2017, 31, 694.10.	0.5	0
35	Intestinal inflammation without weight loss decreases bone density and growth. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2016, 311, R1149-R1157.	1.8	31
36	Lactobacillus reuteri 6475 Increases Bone Density in Intact Females Only under an Inflammatory Setting. PLoS ONE, 2016, 11, e0153180.	2.5	81

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37	Bone Marrow Stem Cells and Bone Turnover in Diabetic Disease. , 2016, , 147-179.		0
38	Bisphosphonate treatment of type I diabetic mice prevents early bone loss but accentuates suppression of bone formation. Journal of Cellular Physiology, 2015, 230, 1944-1953.	4.1	26
39	Prebiotic and Probiotic Regulation of Bone Health: Role of the Intestine and its Microbiome. Current Osteoporosis Reports, 2015, 13, 363-371.	3.6	169
40	Loss of Bone and Wnt10b Expression in Male Type 1 Diabetic Mice Is Blocked by the Probiotic Lactobacillus reuteri. Endocrinology, 2015, 156, 3169-3182.	2.8	113
41	Probiotic <i>L. reuteri</i> Treatment Prevents Bone Loss in a Menopausal Ovariectomized Mouse Model. Journal of Cellular Physiology, 2014, 229, 1822-1830.	4.1	374
42	Surface microcracks signal osteoblasts to regulate alignment and bone formation. Materials Science and Engineering C, 2014, 44, 191-200.	7.3	8
43	β-Arrestin-1 Deficiency Protects Mice from Experimental Colitis. American Journal of Pathology, 2013, 182, 1114-1123.	3.8	34
44	Canonical Nlrp3 Inflammasome Links Systemic Low-Grade Inflammation to Functional Decline in Aging. Cell Metabolism, 2013, 18, 519-532.	16.2	494
45	Probiotic use decreases intestinal inflammation and increases bone density in healthy male but not female mice. Journal of Cellular Physiology, 2013, 228, 1793-1798.	4.1	217
46	Both spontaneous <i>Ins</i> 2 ^{+/â^'} and streptozotocinâ€induced type I diabetes cause bone loss in young mice. Journal of Cellular Physiology, 2013, 228, 689-695.	4.1	31
47	The effects of damage accumulation on the tensile strength and toughness of compact bovine bone. Journal of Biomechanics, 2013, 46, 964-972.	2.1	8
48	Report of the CCFA Pediatric Bone, Growth and Muscle Health Workshop, New York City, November 11–12, 2011, With Updates. Inflammatory Bowel Diseases, 2013, 19, 2919-2926.	1.9	18
49	Colitis-induced Bone Loss is Gender Dependent and Associated with Increased Inflammation. Inflammatory Bowel Diseases, 2013, 19, 1586-1597.	1.9	53
50	Effects of the Diabetic Microenvironment on Esp expression. FASEB Journal, 2013, 27, 1183.11.	0.5	0
51	Probiotic use decreases intestinal inflammation and increases bone density in healthy male but not female mice. FASEB Journal, 2013, 27, 951.4.	0.5	0
52	Enhanced production of early lineages of monocytic and granulocytic cells in mice with colitis. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 16594-16599.	7.1	25
53	Human bone marrow adiposity is linked with serum lipid levels not T1-diabetes. Journal of Diabetes and Its Complications, 2012, 26, 1-9.	2.3	69
54	Amelioration of type I diabetesâ€induced osteoporosis by parathyroid hormone is associated with improved osteoblast survival. Journal of Cellular Physiology, 2012, 227, 1326-1334.	4.1	73

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55	Understanding the Skeletal Pathology of Type 1 and 2 Diabetes Mellitus. Critical Reviews in Eukaryotic Gene Expression, 2011, 21, 187-206.	0.9	67
56	The bone marrow microenvironment contributes to type I diabetes induced osteoblast death. Journal of Cellular Physiology, 2011, 226, 477-483.	4.1	67
57	Caspaseâ€2 deficiency protects mice from diabetesâ€induced marrow adiposity. Journal of Cellular Biochemistry, 2011, 112, 2403-2411.	2.6	9
58	Low Dose Aspirin Therapy Decreases Blood Glucose Levels but Does not Prevent Type I Diabetes-induced Bone Loss. Cellular Physiology and Biochemistry, 2011, 28, 923-932.	1.6	17
59	CCAAT/enhancer binding protein β-deficiency enhances type 1 diabetic bone phenotype by increasing marrow adiposity and bone resorption. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2011, 300, R1250-R1260.	1.8	29
60	Bone and glucose metabolism: A two-way street. Archives of Biochemistry and Biophysics, 2010, 503, 2-10.	3.0	93
61	Inflammatory bowel disease causes reversible suppression of osteoblast and chondrocyte function in mice. American Journal of Physiology - Renal Physiology, 2009, 296, G1020-G1029.	3.4	55
62	Leptin treatment prevents type I diabetic marrow adiposity but not bone loss in mice. Journal of Cellular Physiology, 2009, 218, 376-384.	4.1	55
63	Bone inflammation and altered gene expression with type I diabetes early onset. Journal of Cellular Physiology, 2009, 218, 575-583.	4.1	62
64	Streptozotocin, Type I Diabetes Severity and Bone. Biological Procedures Online, 2009, 11, 296-315.	2.9	100
65	Switching fat from the periphery to bone marrow: why in Type I diabetes?. Expert Review of Endocrinology and Metabolism, 2009, 4, 203-207.	2.4	7
66	Muscle Atrophy and Increased Atroginâ€1 Occur in Fast―but not Slowâ€Muscle Early in Acute Streptozotocinâ€Induced Diabetes. FASEB Journal, 2008, 22, 959.7.	0.5	0
67	Bone Loss and Increased Bone Adiposity in Spontaneous and Pharmacologically Induced Diabetic Mice. Endocrinology, 2007, 148, 198-205.	2.8	225
68	Prolyl-hydroxylase inhibition and HIF activation in osteoblasts promotes an adipocytic phenotype. Journal of Cellular Biochemistry, 2007, 100, 762-772.	2.6	36
69	Understanding the pathology and mechanisms of type I diabetic bone loss. Journal of Cellular Biochemistry, 2007, 102, 1343-1357.	2.6	209
70	Type I diabetic bone phenotype is location but not gender dependent. Histochemistry and Cell Biology, 2007, 128, 125-133.	1.7	54
71	MC3T3-E1 osteoblast attachment and proliferation on porous hydroxyapatite scaffolds fabricated with nanophase powder. International Journal of Nanomedicine, 2006, 1, 189-194.	6.7	67
72	Regulation of Osteoblast Gene Expression and Phenotype by Polylactide-fatty Acid Surfaces. Molecular Biology Reports, 2006, 33, 1-12.	2.3	3

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73	Chronic hyperglycemia modulates osteoblast gene expression through osmotic and nonâ€osmotic pathways. Journal of Cellular Biochemistry, 2006, 99, 411-424.	2.6	218
74	Inhibition of PPARÎ ³ prevents type I diabetic bone marrow adiposity but not bone loss. Journal of Cellular Physiology, 2006, 209, 967-976.	4.1	106
75	Normal Bone Density Obtained in the Absence of Insulin Receptor Expression in Bone. Endocrinology, 2006, 147, 5760-5767.	2.8	64
76	Biaxial flexure testing of calcium phosphate bioceramics for use in tissue engineering. Journal of Biomedical Materials Research Part B, 2005, 72A, 115-126.	3.1	29
77	Adsorption of serum fetuin to hydroxylapatite does not contribute to osteoblast phenotype modifications. Journal of Biomedical Materials Research - Part A, 2005, 73A, 39-47.	4.0	14
78	Hepatocyte growth factor (HGF) adsorption kinetics and enhancement of osteoblast differentiation on hydroxyapatite surfaces. Biomaterials, 2005, 26, 2595-2602.	11.4	48
79	Inhibition of cross-bridge formation has no effect on contraction-associated phosphorylation of p38 MAPK in mouse skeletal muscle. American Journal of Physiology - Cell Physiology, 2005, 288, C824-C830.	4.6	25
80	Increased Bone Adiposity and Peroxisomal Proliferator-Activated Receptor-γ2 Expression in Type I Diabetic Mice. Endocrinology, 2005, 146, 3622-3631.	2.8	216
81	Osteoblasts respond to hydroxyapatite surfaces with immediate changes in gene expression. Journal of Biomedical Materials Research Part B, 2004, 71A, 108-117.	3.1	116
82	Hypoxia suppresses runx2 independent of modeled microgravity. Journal of Cellular Physiology, 2004, 200, 169-176.	4.1	57
83	CCAAT/enhancer-binding protein-β has a role in osteoblast proliferation and differentiation. Experimental Cell Research, 2004, 295, 128-137.	2.6	30
84	Simulated microgravity suppresses osteoblast phenotype, Runx2 levels and AP-1 transactivation. Journal of Cellular Biochemistry, 2003, 88, 427-437.	2.6	93
85	Reduced gap junctional intercellular communication and altered biological effects in mouse osteoblast and rat liver oval cell lines transfected with dominant-negative connexin 43. Molecular Carcinogenesis, 2003, 37, 192-201.	2.7	38
86	Adrenomedullin Increases AP-1 Expression in Rat Mesangial Cells via Activation of Protein Kinase-A and p38 MAPK. Cellular Physiology and Biochemistry, 2003, 13, 367-374.	1.6	9
87	p38 and Activating Transcription Factor-2 Involvement in Osteoblast Osmotic Response to Elevated Extracellular Glucose. Journal of Biological Chemistry, 2002, 277, 37212-37218.	3.4	42
88	Extracellular glucose influences osteoblast differentiation and c-jun expression. Journal of Cellular Biochemistry, 2000, 79, 301-310.	2.6	90
89	AP-1 and Vitamin D Receptor (VDR) Signaling Pathways Converge at the Rat Osteocalcin VDR Element: Requirement for the Internal Activating Protein-1 Site for Vitamin D-Mediated Trans-Activation**This work was supported by NIH Grants AR-45689, AR-39588, and DE-12528. The contents are solely the responsibility of the authors and do not necessarily represent the official views of the NIH	2.8	41
90	Endocrinology, 1999, 140, 63-70. AP-1 and Vitamin D Receptor (VDR) Signaling Pathways Converge at the Rat Osteocalcin VDR Element: Requirement for the Internal Activating Protein-1 Site for Vitamin D-Mediated Trans-Activation. Endocrinology, 1999, 140, 63-70.	2.8	18

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91	Runt homology domain proteins in osteoblast differentiation: AML3/CBFA1 is a major component of a bone-specific complex. Journal of Cellular Biochemistry, 1997, 66, 1-8.	2.6	427
92	Bone tissue-specific transcription of the osteocalcin gene: Role of an activator osteoblast-specific complex and suppressor hox proteins that bind the OC box. Journal of Cellular Biochemistry, 1996, 61, 310-324.	2.6	55
93	The Osteocalcin Gene Promoter Provides a Molecular Blueprint for Regulatory Mechanisms Controlling Bone Tissue Formation: Role of Transcription Factors Involved in Development. Connective Tissue Research, 1996, 35, 15-21.	2.3	13
94	Selective Expression of fos- and jun-Related Genes during Osteoblast Proliferation and Differentiation. Experimental Cell Research, 1995, 218, 255-262.	2.6	137
95	Expression of cell growth and bone phenotypic genes during the cell cycle of normal diploid osteoblasts and osteosarcoma cells. Journal of Cellular Biochemistry, 1994, 56, 274-282.	2.6	13
96	TGF? alters growth and differentiation related gene expression in proliferating osteoblasts in vitro, preventing development of the mature bone phenotype. Journal of Cellular Physiology, 1994, 160, 323-335.	4.1	127
97	Transcriptional control of the tissue-specific, developmentally regulated osteocalcin gene requires a binding motif for the Msx family of homeodomain proteins Proceedings of the National Academy of Sciences of the United States of America, 1994, 91, 12887-12891.	7.1	124
98	The Potential of Probiotics as a Therapy for Osteoporosis. , 0, , 213-233.		6