Murat Bastepe

List of Publications by Year
in descending order

95 papers	5,542 citations	76322 40 h-index	79691 73 g-index
132 all docs	132 docs citations	132 times ranked	3777 citing authors

#	Article	IF	Citations
1	DMP1 mutations in autosomal recessive hypophosphatemia implicate a bone matrix protein in the regulation of phosphate homeostasis. Nature Genetics, 2006, 38, 1248-1250.	21.4	487
2	SLC34A3 Mutations in Patients with Hereditary Hypophosphatemic Rickets with Hypercalciuria Predict a Key Role for the Sodium-Phosphate Cotransporter NaPi-IIc in Maintaining Phosphate Homeostasis. American Journal of Human Genetics, 2006, 78, 179-192.	6.2	422
3	Deletion of the NESP55 differentially methylated region causes loss of maternal GNAS imprints and pseudohypoparathyroidism type Ib. Nature Genetics, 2005, 37, 25-27.	21.4	321
4	Autosomal dominant pseudohypoparathyroidism type Ib is associated with a heterozygous microdeletion that likely disrupts a putative imprinting control element of GNAS. Journal of Clinical Investigation, 2003, 112, 1255-1263.	8.2	226
5	Diagnosis and management of pseudohypoparathyroidism and related disorders: first international Consensus Statement. Nature Reviews Endocrinology, 2018, 14, 476-500.	9.6	224
6	Paternal Uniparental Isodisomy of Chromosome 20qâ€”and the Resulting Changes in GNAS1 Methylationâ€”as a Plausible Cause of Pseudohypoparathyroidism. American Journal of Human Genetics, 2001, 68, 1283-1289.	6.2	198
7	A Novel STX16 Deletion in Autosomal Dominant Pseudohypoparathyroidism Type Ib Redefines the Boundaries of a cis-Acting Imprinting Control Element of GNAS. American Journal of Human Genetics, 2005, 76, 804-814.	6.2	185
8	Epigenetic Defects ofGNASin Patients with Pseudohypoparathyroidism and Mild Features of Albrightâ€™s Hereditary Osteodystrophy. Journal of Clinical Endocrinology and Metabolism, 2007, 92, 2370-2373.	3.6	157
9	GNAS Spectrum of Disorders. Current Osteoporosis Reports, 2015, 13, 146-158.	3.6	147
10	<i>GNAS</i> Locus and Pseudohypoparathyroidism. Hormone Research in Paediatrics, 2005, 63, 65-74.	1.8	144
11	Stimulatory G protein directly regulates hypertrophic differentiation of growth plate cartilage in vivo. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 14794-14799.	7.1	141
12	The GNAS Locus and Pseudohypoparathyroidism. Advances in Experimental Medicine and Biology, 2008, 626, 27-40.	1.6	131
13	A novel COL1A1 mutation in infantile cortical hyperostosis (Caffey disease) expands the spectrum of collagen-related disorders. Journal of Clinical Investigation, 2005, 115, 1250-1257.	8.2	129
14	Receptor-Mediated Adenylyl Cyclase Activation Through XLÎ±s, the Extra-Large Variant of the Stimulatory G Protein Î±-Subunit. Molecular Endocrinology, 2002, 16, 1912-1919.	3.7	128
15	Deletion of the NoncodingGNASAntisense Transcript Causes Pseudohypoparathyroidism Type Ib and Biparental Defects ofGNASMethylationin cis. Journal of Clinical Endocrinology and Metabolism, 2010, 95, 3993-4002.	3.6	113
16	Dosage-dependent switch from G protein-coupled to G protein-independent signaling by a GPCR. EMBO Journal, 2007, 26, 53-64.	7.8	103
17	Identification and Functional Characterization of Novel Calcium-Sensing Receptor Mutations in Familial Hypocalciuric Hypercalcemia and Autosomal Dominant Hypocalcemia. Journal of Clinical Endocrinology and Metabolism, 2002, 87, 1309-1318.	3.6	102
18	Similar clinical and laboratory findings in patients with symptomatic autosomal dominant and sporadic pseudohypoparathyroidism type Ib despite different epigenetic changes at the GNAS locus. Clinical Endocrinology, 2007, 67, 822-831.	2.4	98

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19	GsÎ± enhances commitment of mesenchymal progenitors to the osteoblast lineage but restrains osteoblast differentiation in mice. Journal of Clinical Investigation, 2011, 121, 3492-3504.	8.2	91
20	Identification and Characterization of Two Parathyroid Hormone-Like Molecules in Zebrafish. Endocrinology, 2004, 145, 1634-1639.	2.8	83
21	Inherited hypophosphatemic disorders in children and the evolving mechanisms of phosphate regulation. Reviews in Endocrine and Metabolic Disorders, 2008, 9, 171-180.	5.7	78
22	Mechanisms of Ligand Binding to the Parathyroid Hormone (PTH)/PTH-Related Protein Receptor: Selectivity of a Modified PTH(1â€“15) Radioligand for GÎ±S-Coupled Receptor Conformations. Molecular Endocrinology, 2006, 20, 931-943.	3.7	73
23	Molecular Diagnosis and Clinical Characterization of Pseudohypoparathyroidism Type-Ib in a Patient With Mild Albrightâ€™s Hereditary Osteodystrophy-Like Features, Epileptic Seizures, and Defective Renal Handling of Uric Acid. American Journal of the Medical Sciences, 2008, 336, 84-90.	1.1	73
24	Paternal uniparental isodisomy of the entire chromosome 20 as a molecular cause of pseudohypoparathyroidism type Ib (PHP-Ib). Bone, 2011, 48, 659-662.	2.9	67
25	Postnatal Establishment of Allelic GÎ±s Silencing as a Plausible Explanation for Delayed Onset of Parathyroid Hormone Resistance Owing to Heterozygous GÎ±s Disruption. Journal of Bone and Mineral Research, 2014, 29, 749-760.	2.8	64
26	Functional characterization of GNAS mutations found in patients with pseudohypoparathyroidism type Ic defines a new subgroup of pseudohypoparathyroidism affecting selectively GsÎ±-receptor interaction. Human Mutation, 2011, 32, 653-660.	2.5	62
27	The <i>GNAS</i> Complex Locus and Human Diseases Associated with Loss-of-Function Mutations or Epimutations within This Imprinted Gene. Hormone Research in Paediatrics, 2013, 80, 229-241.	1.8	60
28	The Î²-blocker Nebivolol Is a GRK/Î²-arrestin Biased Agonist. PLoS ONE, 2013, 8, e71980.	2.5	58
29	CodingGNASMutations Leading to Hormone Resistance Impairin VitroAgonist- and Cholera Toxin-Induced Adenosine Cyclic 3â€²,5â€²-Monophosphate Formation Mediated by Human XLÎ±s. Endocrinology, 2006, 147, 2253-2262.	2.8	56
30	Targeted deletion of the Nesp55 DMR defines another Gnas imprinting control region and provides a mouse model of autosomal dominant PHP-Ib. Proceedings of the National Academy of Sciences of the United States of America, 2010, 107, 9275-9280.	7.1	55
31	Identification of a novel dentin matrix protein-1 (DMP-1) mutation and dental anomalies in a kindred with autosomal recessive hypophosphatemia. Bone, 2010, 46, 402-409.	2.9	55
32	A rapid microarray based whole genome analysis for detection of uniparental disomy. Human Mutation, 2005, 26, 153-159.	2.5	53
33	Long-term clinical outcome and carrier phenotype in autosomal recessive hypophosphatemia caused by a novel DMP1 mutation. Journal of Bone and Mineral Research, 2010, 25, 2165-2174.	2.8	53
34	A Form of Jansenâ€™s Metaphyseal Chondrodysplasia with Limited Metabolic and Skeletal Abnormalities Is Caused by a Novel Activating Parathyroid Hormone (PTH)/PTH-Related Peptide Receptor Mutation. Journal of Clinical Endocrinology and Metabolism, 2004, 89, 3595-3600.	3.6	50
35	Phenotypic and Molecular Genetic Aspects of Pseudohypoparathyroidism Type Ib in a Greek Kindred: Evidence for Enhanced Uric Acid Excretion Due to Parathyroid Hormone Resistance. Journal of Clinical Endocrinology and Metabolism, 2004, 89, 5942-5947.	3.6	50
36	The GNAS Locus: Quintessential Complex Gene Encoding Gsα, XLαs, and other Imprinted Transcripts. Current Genomics, 2007, 8, 398-414.	1.6	50

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37	PSEUDOHYPOPARATHYROIDISM. Endocrinology and Metabolism Clinics of North America, 2000, 29, 569-589.	3.2	49
38	Molecular Diagnosis of Pseudohypoparathyroidism Type Ib in a Family With Presumed Paroxysmal Dyskinesia. Pediatrics, 2005, 115, e242-e244.	2.1	48
39	Potent constitutive cyclic AMP-generating activity of XLÎ±s implicates this imprinted GNAS product in the pathogenesis of McCuneâ€“Albright Syndrome and fibrous dysplasia of bone. Bone, 2011, 48, 312-320.	2.9	44
40	The Long Cytoplasmic Carboxyl Terminus of the Prostaglandin E₂Receptor EP₄Subtype Is Essential for Agonist-Induced Desensitization. Molecular Pharmacology, 1997, 51, 343-349.	2.3	42
41	Recommendations for Diagnosis and Treatment of Pseudohypoparathyroidism and Related Disorders: An Updated Practical Tool for Physicians and Patients. Hormone Research in Paediatrics, 2020, 93, 182-196.	1.8	42
42	Parathyroid hormone signaling via GÎ±s is selectively inhibited by an NH2-terminally truncated GÎ±s: Implications for pseudohypoparathyroidism. Journal of Bone and Mineral Research, 2011, 26, 2473-2485.	2.8	39
43	IntragenicGNASDeletion Involving Exon A/B in Pseudohypoparathyroidism Type 1A Resulting in an Apparent Loss of Exon A/B Methylation: Potential for Misdiagnosis of Pseudohypoparathyroidism Type 1B. Journal of Clinical Endocrinology and Metabolism, 2010, 95, 765-771.	3.6	38
44	GNAS mutations and heterotopic ossification. Bone, 2018, 109, 80-85.	2.9	37
45	Pseudohypoparathyroidism and Mechanisms of Resistance toward Multiple Hormones: Molecular Evidence to Clinical Presentation. Journal of Clinical Endocrinology and Metabolism, 2003, 88, 4055-4058.	3.6	35
46	Exclusion of the GNAS locus in PHP-Ib patients with broad GNAS methylation changes: Evidence for an autosomal recessive form of PHP-Ib?. Journal of Bone and Mineral Research, 2011, 26, 1854-1863.	2.8	34
47	Extralarge XLÎ±s (XXLÎ±s), a Variant of Stimulatory G Protein Î±-Subunit (GsÎ±), Is a Distinct, Membrane-Anchored GNAS Product that Can Mimic GsÎ±. Endocrinology, 2009, 150, 3567-3575.	2.8	32
48	De NovoSTX16 Deletions: An Infrequent Cause of Pseudohypoparathyroidism Type Ib that Should Be Excluded in Sporadic Cases. Journal of Clinical Endocrinology and Metabolism, 2012, 97, E2314-E2319.	3.6	32
49	Lack of Gnas Epigenetic Changes and Pseudohypoparathyroidism Type Ib in Mice with Targeted Disruption of Syntaxin-16. Endocrinology, 2007, 148, 2925-2935.	2.8	30
50	Evidence of hormone resistance in a pseudo-pseudohypoparathyroidism patient with a novel paternal mutation in GNAS. Bone, 2015, 71, 53-57.	2.9	29
51	Secondary ossification center induces and protects growth plate structure. ELife, 2020, 9, .	6.0	29
52	Identification of a region of the Câ€terminal domain involved in shortâ€term desensitization of the prostaglandin EP₄ receptor. British Journal of Pharmacology, 1999, 126, 365-371.	5.4	28
53	Osteosclerosis in two brothers with autosomal dominant pseudohypoparathyroidism type 1b: bone histomorphometric analysis. European Journal of Endocrinology, 2011, 164, 295-301.	3.7	28
54	Heterotrimeric G proteins in the control of parathyroid hormone actions. Journal of Molecular Endocrinology, 2017, 58, R203-R224.	2.5	28

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55	Transgenic Overexpression of the Extra-Large GsÎ± Variant XLÎ±s Enhances GsÎ±-Mediated Responses in the Mouse Renal Proximal Tubule in Vivo. Endocrinology, 2011, 152, 1222-1233.	2.8	27
56	Extra-long GÎ±s Variant XLÎ±s Protein Escapes Activation-induced Subcellular Redistribution and Is Able to Provide Sustained Signaling. Journal of Biological Chemistry, 2011, 286, 38558-38569.	3.4	26
57	Different Mutations Within or Upstream of the GNAS Locus Cause Distinct Forms of Pseudohypoparathyroidism. Journal of Pediatric Endocrinology and Metabolism, 2006, 19, 641-6.	0.9	25
58	Genetics and Epigenetics of Parathyroid Hormone Resistance. Endocrine Development, 2013, 24, 11-24.	1.3	25
59	The G protein Î± subunit variant XLÎ± _s promotes inositol 1,4,5-trisphosphate signaling and mediates the renal actions of parathyroid hormone in vivo. Science Signaling, 2015, 8, ra84.	3.6	23
60	Loss of XLÎ±s (extra-large Î±s) imprinting results in early postnatal hypoglycemia and lethality in a mouse model of pseudohypoparathyroidism Ib. Proceedings of the National Academy of Sciences of the United States of America, 2012, 109, 6638-6643.	7.1	19
61	Autosomal-Dominant Pseudohypoparathyroidism Type Ib is Caused by Different Microdeletions Within or Upstream of the GNAS Locus. Annals of the New York Academy of Sciences, 2006, 1068, 250-255.	3.8	18
62	D2 dopamine receptor-induced sensitization of adenylyl cyclase type 1 is GÎ±s independent. Neuropharmacology, 2006, 50, 576-584.	4.1	16
63	A G proteinâ€“coupled, IP3/protein kinase C pathway controlling the synthesis of phosphaturic hormone FGF23. JCI Insight, 2019, 4, .	5.0	16
64	Characterization of the molecular mechanisms of the coupling between intracellular loops of prostacyclin receptor with the C-terminal domain of the GÎ±s protein in human coronary artery smooth muscle cells. Archives of Biochemistry and Biophysics, 2006, 454, 80-88.	3.0	12
65	NHERF1Mutations and Responsiveness of Renal Parathyroid Hormone. New England Journal of Medicine, 2008, 359, 2615-2617.	27.0	11
66	Huntington Disease: Molecular Diagnostics Approach. Current Protocols in Human Genetics, 2015, 87, 9.26.1-9.26.23.	3.5	11
67	Ablation of the Stimulatory G Protein Î±-Subunit in Renal Proximal Tubules Leads to Parathyroid Hormone-Resistance With Increased Renal Cyp24a1 mRNA Abundance and Reduced Serum 1,25-Dihydroxyvitamin D. Endocrinology, 2016, 157, 497-507.	2.8	10
68	Large G protein Î±-subunit XLÎ±s limits clathrin-mediated endocytosis and regulates tissue iron levels in vivo. Proceedings of the National Academy of Sciences of the United States of America, 2017, 114, E9559-E9568.	7.1	9
69	Pseudohypoparathyroidism, GsÎ±, and the GNAS locus. BoneKEy Osteovision, 2005, 2, 20-32.	0.6	6
70	Constitutive stimulatory G protein activity in limb mesenchyme impairs bone growth. Bone, 2018, 110, 230-237.	2.9	6
71	Recessive versus imprinted disorder: consanguinity can impede establishing the diagnosis of autosomal dominant pseudohypoparathyroidism type Ib. European Journal of Endocrinology, 2010, 163, 489-493.	3.7	5
72	A novel deletion involving GNAS exon 1 causes PHP1A and further refines the region required for normal methylation at exon A/B. Bone, 2017, 103, 281-286.	2.9	5

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73	Parathyroid Disorders. , 2003, , 485-508.		5
74	Pseudohypoparathyroidism, Albright's Hereditary Osteodystrophy, and Progressive Osseous Heteroplasia. , 2010, , 1223-1235.		5
75	Severe brachydactyly and short stature resulting from a novel pathogenic TRPS1 variant within the GATA DNA-binding domain. Bone, 2019, 123, 153-158.	2.9	4
76	Extra-Large GÎ± Protein (XLÎ±s) Deficiency Causes Severe Adenine-Induced Renal Injury with Massive FGF23 Elevation. Endocrinology, 2020, 161, .	2.8	4
77	Maternal GNAS Contributes to the Extra-Large G Protein Î±-Subunit (XLÎ±s) Expression in a Cell Type-Specific Manner. Frontiers in Genetics, 2021, 12, 680537.	2.3	4
78	A Novel T55A Variant of G_sÎ±Associated with Impaired cAMP Production, Bone Fragility, and Osteolysis. Case Reports in Endocrinology, 2016, 2016, 1-6.	0.4	3
79	Sporadic Pseudohypoparathyroidism Type 1B in Monozygotic Twins: Insights Into the Pathogenesis of Methylation Defects. Journal of Clinical Endocrinology and Metabolism, 2022, 107, e947-e954.	3.6	3
80	A naturally occurring membrane-anchored GÎ±s variant, XLÎ±s, activates phospholipase CÎ²4. Journal of Biological Chemistry, 2022, 298, 102134.	3.4	3
81	Haplotype frequencies and linkage disequilibrium analysis of four frequent polymorphisms at the PTH/PTH-related peptide receptor gene locus. Molecular and Cellular Probes, 2004, 18, 353-357.	2.1	2
82	A Gain-of-Function CASR Mutation Causing Hypocalcemia in a Recessive Manner. Journal of Clinical Endocrinology and Metabolism, 2018, 103, 3514-3515.	3.6	2
83	Parathyroid Disorders. , 2012, , 557-588.		1
84	Pseudohypoparathyroidism, Albrightâ€™s Hereditary Osteodystrophy, and Progressive Osseous Heteroplasia. , 2016, , 1147-1159.e6.		1
85	GNAS Complex Locus. , 2018, , 2173-2185.		1
86	Parathyroid Disorders. , 2013, , 1-34.		0
87	Sporadic Pseudohypoparathyroidism 1B in Monozygotic Twins: Insights Into the Pathogenesis of Methylation Defects. Journal of the Endocrine Society, 2021, 5, A224-A224.	0.2	0
88	XLÎ± s is a novel activator of PLCÎ²4. FASEB Journal, 2021, 35, .	0.5	0
89	G Proteinâ€Coupled Receptor Kinase 4Î³ (GRK4Î³) Interacts with the Alpha Helical Domain of GÎ± s. FASEB Journal, 2009, 23, .	0.5	0
90	The interaction of G proteinâ€coupled receptor kinase 4Î³ with GÎ± s is required for inhibition of the Î² 2 â€AR. FASEB Journal, 2010, 24, 585.3.	0.5	0

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91	GNAS Complex Locus. , 2016, , 1-13.		0
92	MON-LB087 Synthesis of Osteocyte-Derived Phosphaturic Hormone FGF23 via IP3/PKC Signaling Downstream of the Extra-Large Gî± Subunit (XLî±s). Journal of the Endocrine Society, 2019, 3, .	0.2	0
93	Kinetic changes in Ga cycling can increase cAMP accumulation while decreasing G proteinâ€coupled receptor kinaseâ€mediated receptor desensitization. FASEB Journal, 2019, 33, 502.7.	0.5	0
94	Kinetic control of signaling: role of RGS proteins and GRKs. FASEB Journal, 2020, 34, 1-1.	0.5	0
95	XLî±s is a novel activator of PLCî²4. FASEB Journal, 2020, 34, 1-1.	0.5	0