Lee S Weinstein

List of Publications by Year
in descending order

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133 papers	10,443 citations	34016 52 h-index	33814 99 g-index
136 all docs	136 docs citations	136 times ranked	8634 citing authors

#	Article	IF	Citations
1	Activating Mutations of the Stimulatory G Protein in the McCuneâ€“Albright Syndrome. New England Journal of Medicine, 1991, 325, 1688-1695.	13.9	1,804
2	Endocrine Manifestations of Stimulatory G Protein Î±-Subunit Mutations and the Role of Genomic Imprinting. Endocrine Reviews, 2001, 22, 675-705.	8.9	390
3	Severe endocrine and nonendocrine manifestations of the McCune-Albright syndrome associated with activating mutations of stimulatory G protein Gs. Journal of Pediatrics, 1993, 123, 509-518.	0.9	316
4	Receptor-Effector Coupling by G Proteins: Implications for Normal and Abnormal Signal Transduction. Endocrine Reviews, 1992, 13, 536-565.	8.9	308
5	Minireview: GNAS: Normal and Abnormal Functions. Endocrinology, 2004, 145, 5459-5464.	1.4	291
6	A GNAS1 imprinting defect in pseudohypoparathyroidism type IB. Journal of Clinical Investigation, 2000, 106, 1167-1174.	3.9	263
7	Inherited Diseases Involving G Proteins and G Proteinâ€“Coupled Receptors. Annual Review of Medicine, 2004, 55, 27-39.	5.0	228
8	Osteoblastic regulation of B lymphopoiesis is mediated by G _s Î±-dependent signaling pathways. Proceedings of the National Academy of Sciences of the United States of America, 2008, 105, 16976-16981.	3.3	222
9	The Stimulatory G Protein Î±-Subunit GsÎ± Is Imprinted in Human Thyroid Glands: Implications for Thyroid Function in Pseudohypoparathyroidism Types 1A and 1B. Journal of Clinical Endocrinology and Metabolism, 2003, 88, 4336-4341.	1.8	188
10	Activation of Hedgehog signaling by loss of GNAS causes heterotopic ossification. Nature Medicine, 2013, 19, 1505-1512.	15.2	187
11	Thyrotrophin receptor signaling dependence of Braf-induced thyroid tumor initiation in mice. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 1615-1620.	3.3	183
12	Identification of a Methylation Imprint Mark within the Mouse Gnas Locus. Molecular and Cellular Biology, 2000, 20, 5808-5817.	1.1	181
13	Body Mass Index Differences in Pseudohypoparathyroidism Type 1aVersusPseudopseudohypoparathyroidism May Implicate Paternal Imprinting of GÎ±s in the Development of Human Obesity. Journal of Clinical Endocrinology and Metabolism, 2007, 92, 1073-1079.	1.8	181
14	Alternative Gnas gene products have opposite effects on glucose and lipid metabolism. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 7386-7391.	3.3	174
15	Paternal versus maternal transmission of a stimulatory G-protein Î± subunit knockout produces opposite effects on energy metabolism. Journal of Clinical Investigation, 2000, 105, 615-623.	3.9	151
16	Myelopoiesis is regulated by osteocytes through GsÎ±-dependent signaling. Blood, 2013, 121, 930-939.	0.6	146
17	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.	3.3	141
18	Gs_{Î±} Mutations and Imprinting Defects in Human Disease. Annals of the New York Academy of Sciences, 2002, 968, 173-197.	1.8	137

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19	Inactivation of a GÎ±sâ€“PKA tumour suppressor pathway in skin stem cells initiates basal-cell carcinogenesis. Nature Cell Biology, 2015, 17, 793-803.	4.6	134
20	Shear stressâ€“induced endothelial adrenomedullin signaling regulates vascular tone and blood pressure. Journal of Clinical Investigation, 2019, 129, 2775-2791.	3.9	129
21	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
22	Agonist-Independent GPCR Activity Regulates Anterior-Posterior Targeting of Olfactory Sensory Neurons. Cell, 2013, 154, 1314-1325.	13.5	126
23	Increased glucose tolerance and reduced adiposity in the absence of fasting hypoglycemia in mice with liver-specific GsÎ± deficiency. Journal of Clinical Investigation, 2005, 115, 3217-3227.	3.9	125
24	Central Nervous System Imprinting of the G Protein GsÎ± and Its Role in Metabolic Regulation. Cell Metabolism, 2009, 9, 548-555.	7.2	118
25	Distinct patterns of abnormal GNAS imprinting in familial and sporadic pseudohypoparathyroidism type IB. Human Molecular Genetics, 2005, 14, 95-102.	1.4	117
26	The G protein Î± subunit GÎ±s is a tumor suppressor in Sonic hedgehogâ^'driven medulloblastoma. Nature Medicine, 2014, 20, 1035-1042.	15.2	110
27	GsÎ± Mutations in Fibrous Dysplasia and McCune-Albright Syndrome. Journal of Bone and Mineral Research, 2006, 21, P120-P124.	3.1	102
28	Identification of the control region for tissue-specific imprinting of the stimulatory G protein Â-subunit. Proceedings of the National Academy of Sciences of the United States of America, 2005, 102, 5513-5518.	3.3	97
29	A heterozygous 4-bp deletion mutation in the GsÎ± gene (GNAS1) in a patient with albright hereditary osteodystrophy. Genomics, 1992, 13, 1319-1321.	1.3	96
30	Chondrocyte-Specific Knockout of the G Protein GsÎ± Leads to Epiphyseal and Growth Plate Abnormalities and Ectopic Chondrocyte Formation. Journal of Bone and Mineral Research, 2004, 20, 663-671.	3.1	95
31	Fibroblast Growth Factor-23 Is Regulated by 1Î±,25-Dihydroxyvitamin D. Journal of Bone and Mineral Research, 2005, 20, 1944-1950.	3.1	92
32	Wnt/Î²-catenin signaling is differentially regulated by GÎ± proteins and contributes to fibrous dysplasia. Proceedings of the National Academy of Sciences of the United States of America, 2011, 108, 20101-20106.	3.3	92
33	Results of initial operation for hyperparathyroidism in patients with multiple endocrine neoplasia type 1. Surgery, 2003, 134, 858-864.	1.0	91
34	GsÎ± enhances commitment of mesenchymal progenitors to the osteoblast lineage but restrains osteoblast differentiation in mice. Journal of Clinical Investigation, 2011, 121, 3492-3504.	3.9	91
35	Genetic diseases associated with heterotrimeric G proteins. Trends in Pharmacological Sciences, 2006, 27, 260-266.	4.0	90
36	The Alternative Stimulatory G Protein Î±-Subunit XLÎ±s Is a Critical Regulator of Energy and Glucose Metabolism and Sympathetic Nerve Activity in Adult Mice*. Journal of Biological Chemistry, 2006, 281, 18989-18999.	1.6	90

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37	Deficiency of the G-protein Î±-Subunit GsÎ± in Osteoblasts Leads to Differential Effects on Trabecular and Cortical Bone. Journal of Biological Chemistry, 2005, 280, 21369-21375.	1.6	88
38	The Parathyroid/Pituitary Variant of Multiple Endocrine Neoplasia Type 1 Usually Has Causes Other thanp27Kip1Mutations. Journal of Clinical Endocrinology and Metabolism, 2007, 92, 1948-1951.	1.8	84
39	Regulation of renin in mice with Cre recombinase-mediated deletion of G protein GsÎ± in juxtaglomerular cells. American Journal of Physiology - Renal Physiology, 2007, 292, F27-F37.	1.3	83
40	Increased Insulin Sensitivity in PaternalGnasKnockout Mice Is Associated with Increased Lipid Clearance. Endocrinology, 2004, 145, 4094-4102.	1.4	79
41	Genetic mapping of the Gs-Î± subunit gene (GNAS1) to the distal long arm of chromosome 20 using a polymorphism detected by denaturing gradient gel electrophoresis. Genomics, 1991, 9, 782-783.	1.3	74
42	Gq/11Î± and GsÎ± mediate distinct physiological responses to central melanocortins. Journal of Clinical Investigation, 2015, 126, 40-49.	3.9	74
43	Reoperation for hyperparathyroidism in multiple endocrine neoplasia type 1. Surgery, 2001, 130, 991-998.	1.0	73
44	Results of heterotopic parathyroid autotransplantation: A 13-year experience. Surgery, 1999, 126, 1042-1048.	1.0	70
45	Studies of the regulation and function of the GsÎ± gene Gnas using gene targeting technology. , 2007, 115, 271-291.		70
46	Haematopoietic stem cells depend on GÎ±s-mediated signalling to engraft bone marrow. Nature, 2009, 459, 103-107.	13.7	69
47	Multiple Endocrine Neoplasia Type 1 Variant with Frequent Prolactinoma and Rare Gastrinoma. Journal of Clinical Endocrinology and Metabolism, 2004, 89, 3776-3784.	1.8	66
48	Î² cell-specific deficiency of the stimulatory G protein Î±-subunit G _s Î± leads to reduced Î² cell mass and insulin-deficient diabetes. Proceedings of the National Academy of Sciences of the United States of America, 2007, 104, 19601-19606.	3.3	64
49	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.	3.1	64
50	A Novel Mutation in the Switch 3 Region of GsÎ± in a Patient with Albright Hereditary Osteodystrophy Impairs GDP Binding and Receptor Activation. Journal of Biological Chemistry, 1998, 273, 23976-23983.	1.6	61
51	A deletion hot-spot in exon 7 of the G8Î± gene (GNAS1) in patients with Aibright hereditary osteodystrophy. Human Molecular Genetics, 1995, 4, 2001-2002.	1.4	60
52	Single-Cell RNA Profiling Reveals Adipocyte to Macrophage Signaling Sufficient to Enhance Thermogenesis. Cell Reports, 2020, 32, 107998.	2.9	60
53	Reoperation for parathyroid adenoma: A contemporary experience. Surgery, 2009, 146, 1144-1155.	1.0	57
54	Divergent requirement for GÎ±s and cAMP in the differentiation and inflammatory profile of distinct mouse Th subsets. Journal of Clinical Investigation, 2012, 122, 963-973.	3.9	57

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55	HRPT2,a Marker of Parathyroid Cancer. New England Journal of Medicine, 2003, 349, 1691-1692.	13.9	54
56	Increased Insulin Sensitivity in GsÎ± Knockout Mice. Journal of Biological Chemistry, 2001, 276, 19994-19998.	1.6	53
57	G protein mutations in human disease. Clinical Biochemistry, 1993, 26, 333-338.	0.8	49
58	G_sÎ± deficiency in skeletal muscle leads to reduced muscle mass, fiber-type switching, and glucose intolerance without insulin resistance or deficiency. American Journal of Physiology - Cell Physiology, 2009, 296, C930-C940.	2.1	49
59	Identification of Two Novel Deletion Mutations within the GsÎ± Gene (GNAS1) in Albright Hereditary Osteodystrophy1. Journal of Clinical Endocrinology and Metabolism, 1999, 84, 3254-3259.	1.8	48
60	GsÎ± Deficiency in the Paraventricular Nucleus of the Hypothalamus Partially Contributes to Obesity Associated with GsÎ± Mutations. Endocrinology, 2012, 153, 4256-4265.	1.4	48
61	Stimulation of Renin Secretion by Angiotensin II Blockade is GsÎ±-Dependent. Journal of the American Society of Nephrology: JASN, 2010, 21, 986-992.	3.0	47
62	Oriented clonal cell dynamics enables accurate growth and shaping of vertebrate cartilage. ELife, 2017, 6, .	2.8	46
63	A Novel Mutation Adjacent to the Switch III Domain of G_{sÎ±}in a Patient with Pseudohypoparathyroidism. Molecular Endocrinology, 1997, 11, 1718-1727.	3.7	45
64	Development of vascular renin expression in the kidney critically depends on the cyclic AMP pathway. American Journal of Physiology - Renal Physiology, 2009, 296, F1006-F1012.	1.3	44
65	Development and Treatment of Tertiary Hyperparathyroidism in Patients with Pseudohypoparathyroidism Type 1B. Journal of Clinical Endocrinology and Metabolism, 2012, 97, 3025-3030.	1.8	42
66	Tissue-specific imprinting of the G protein GsÂ is associated with tissue-specific differences in histone methylation. Human Molecular Genetics, 2004, 13, 819-828.	1.4	41
67	G-protein stimulatory subunit alpha and Gq/11Î± G-proteins are both required to maintain quiescent stem-like chondrocytes. Nature Communications, 2014, 5, 3673.	5.8	41
68	Reduced Insulin Sensitivity in Adults With Pseudohypoparathyroidism Type 1a. Journal of Clinical Endocrinology and Metabolism, 2013, 98, E1796-E1801.	1.8	40
69	GsÎ± deficiency in the dorsomedial hypothalamus underlies obesity associated with GsÎ± mutations. Journal of Clinical Investigation, 2016, 127, 500-510.	3.9	40
70	The Role of Genomic Imprinting of GsÎ± in the Pathogenesis of Albright Hereditary Osteodystrophy. Trends in Endocrinology and Metabolism, 1999, 10, 81-85.	3.1	39
71	Removal of the N-terminal Extension of Cardiac Troponin I as a Functional Compensation for Impaired Myocardial Î²-Adrenergic Signaling. Journal of Biological Chemistry, 2008, 283, 33384-33393.	1.6	39
72	Severe Obesity and Insulin Resistance due to Deletion of the Maternal GsÎ± Allele Is Reversed by Paternal Deletion of the GsÎ± Imprint Control Region. Endocrinology, 2008, 149, 2443-2450.	1.4	39

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73	Limited Parathyroidectomy in Multiple Endocrine Neoplasia Type 1-Associated Primary Hyperparathyroidism: A Setup for Failure. Annals of Surgical Oncology, 2016, 23, 416-423.	0.7	39
74	Persistent Primary Hyperparathyroidism Caused by Adenomas Identified in Pharyngeal or Adjacent Structures. World Journal of Surgery, 2003, 27, 675-679.	0.8	38
75	GsÎ± Deficiency in Adipose Tissue Leads to a Lean Phenotype with Divergent Effects on Cold Tolerance and Diet-Induced Thermogenesis. Cell Metabolism, 2010, 11, 320-330.	7.2	38
76	Albright Hereditary Osteodystrophy, Pseudohypoparathyroidism, and Gs Deficiency. , 1998, , 23-56.		38
77	Variable imprinting of the heterotrimeric G protein G_sÎ±-subunit within different segments of the nephron. American Journal of Physiology - Renal Physiology, 2000, 278, F507-F514.	1.3	37
78	Control of Adipocyte Thermogenesis and Lipogenesis through Î²3-Adrenergic and Thyroid Hormone Signal Integration. Cell Reports, 2020, 31, 107598.	2.9	37
79	Loss of GsÎ± in the Postnatal Skeleton Leads to Low Bone Mass and a Blunted Response to Anabolic Parathyroid Hormone Therapy. Journal of Biological Chemistry, 2016, 291, 1631-1642.	1.6	36
80	The Stimulatory G Protein Î±-Subunit Gene: Mutations and Imprinting Lead to Complex Phenotypes. Journal of Clinical Endocrinology and Metabolism, 2001, 86, 4622-4626.	1.8	35
81	Loss of GsÎ± Early in the Osteoblast Lineage Favors Adipogenic Differentiation of Mesenchymal Progenitors and Committed Osteoblast Precursors. Journal of Bone and Mineral Research, 2014, 29, 2414-2426.	3.1	33
82	G _s Î± deficiency in adipose tissue improves glucose metabolism and insulin sensitivity without an effect on body weight. Proceedings of the National Academy of Sciences of the United States of America, 2016, 113, 446-451.	3.3	33
83	The stimulatory G protein GsÎ± is required in melanocortin 4 receptorâ€“expressing cells for normal energy balance, thermogenesis, and glucose metabolism. Journal of Biological Chemistry, 2018, 293, 10993-11005.	1.6	33
84	Preoperative Localizing Studies for Initial Parathyroidectomy in MEN1 Syndrome: Is There Any Benefit?. World Journal of Surgery, 2012, 36, 1368-1374.	0.8	32
85	The role of tissue-specific imprinting as a source of phenotypic heterogeneity in human disease. Biological Psychiatry, 2001, 50, 927-931.	0.7	27
86	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.	1.4	27
87	Decreased renal Na-K-2Cl cotransporter abundance in mice with heterozygous disruption of the GsÎ± gene. American Journal of Physiology - Renal Physiology, 1999, 277, F235-F244.	1.3	24
88	Pancreas-specific GsÎ± deficiency has divergent effects on pancreatic Î±- and Î²-cell proliferation. Journal of Endocrinology, 2010, 206, 261-269.	1.2	24
89	Mechanochemical control of epidermal stem cell divisions by B-plexins. Nature Communications, 2021, 12, 1308.	5.8	24
90	Utility of Intraoperative Parathyroid Hormone Monitoring in Patients with Multiple Endocrine Neoplasia Type 1â€Associated Primary Hyperparathyroidism Undergoing Initial Parathyroidectomy. World Journal of Surgery, 2013, 37, 1966-1972.	0.8	23

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91	Reoperative Surgery in Patients with Multiple Endocrine Neoplasia Type 1 Associated Primary Hyperparathyroidism. Annals of Surgical Oncology, 2016, 23, 701-707.	0.7	22
92	Probability of Positive Genetic Testing Results in Patients with Family History of Primary Hyperparathyroidism. Journal of the American College of Surgeons, 2018, 226, 933-938.	0.2	21
93	Smooth muscle-specific GsÎ± deletion exaggerates angiotensin II-induced abdominal aortic aneurysm formation in mice in vivo. Journal of Molecular and Cellular Cardiology, 2019, 132, 49-59.	0.9	21
94	Renal Failure in Mice with Gs-alpha Deletion in Juxtaglomerular Cells. American Journal of Nephrology, 2010, 32, 83-94.	1.4	20
95	Skeletal abnormalities and extra-skeletal ossification in mice with restricted GsÎ± deletion caused by a renin promoter-Cre transgene. Cell and Tissue Research, 2007, 330, 487-501.	1.5	18
96	Effects of deficiency of the G protein GsÎ± on energy and glucose homeostasis. European Journal of Pharmacology, 2011, 660, 119-124.	1.7	18
97	GÎ±s regulates asymmetric cell division of cortical progenitors by controlling Numb mediated Notch signaling suppression. Neuroscience Letters, 2015, 597, 97-103.	1.0	16
98	Disturbed flowâ€“induced Gs-mediated signaling protects against endothelial inflammation and atherosclerosis. JCI Insight, 2020, 5, .	2.3	16
99	Characterization of the Promoter of the Human Gi2Î±-Subunit Gene. Molecular Endocrinology, 1990, 4, 958-964.	3.7	15
100	Interference with GsÎ±-Coupled Receptor Signaling in Renin-Producing Cells Leads to Renal Endothelial Damage. Journal of the American Society of Nephrology: JASN, 2017, 28, 3479-3489.	3.0	15
101	Clenbuterol exerts antidiabetic activity through metabolic reprogramming of skeletal muscle cells. Nature Communications, 2022, 13, 22.	5.8	15
102	Mutagenesis of the Conserved Residue Glu259 of GsÎ± Demonstrates the Importance of Interactions between Switches 2 and 3 for Activation. Journal of Biological Chemistry, 1999, 274, 4977-4984.	1.6	14
103	The in vivo regulation of heart rate in the murine sinoatrial node by stimulatory and inhibitory heterotrimeric G proteins. American Journal of Physiology - Regulatory Integrative and Comparative Physiology, 2013, 305, R435-R442.	0.9	14
104	The role of Gâ€protein in matrixâ€mediated motility of highly and poorly invasive melanoma cells. International Journal of Cancer, 1991, 48, 113-120.	2.3	13
105	Improved fatigue resistance in G_sÎ±-deficient and aging mouse skeletal muscles due to adaptive increases in slow fibers. Journal of Applied Physiology, 2011, 111, 834-843.	1.2	13
106	Sleeping Parathyroid Tumor: Rapid Hyperfunction after Removal of the Dominant Tumor. Journal of Clinical Endocrinology and Metabolism, 2012, 97, 1834-1841.	1.8	13
107	GsÎ± Deficiency in the Ventromedial Hypothalamus Enhances Leptin Sensitivity and Improves Glucose Homeostasis in Mice on a High-Fat Diet. Endocrinology, 2016, 157, 600-610.	1.4	13
108	Heterotrimeric G Stimulatory Protein Î± Subunit Is Required forÂIntestinal Smooth Muscle Contraction in Mice. Gastroenterology, 2017, 152, 1114-1125.e5.	0.6	12

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109	Absence of the Glucagon-Like Peptide-1 Receptor Does Not Affect the Metabolic Phenotype of Mice with Liver-Specific GsÎ± Deficiency. Endocrinology, 2011, 152, 3343-3350.	1.4	10
110	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.	1.4	10
111	Partial thyrocyteâ€specific GÎ± _s deficiency leads to rapidâ€onset hypothyroidism, hyperplasia, and papillary thyroid carcinomaâ€like lesions in mice. FASEB Journal, 2018, 32, 6239-6251.	0.2	9
112	GNASHaploinsufficiency Leads to Subcutaneous Tumor Formation With Collagen and Elastin Deposition and Calcification. Endocrine Research, 2009, 34, 1-9.	0.6	8
113	G-Protein Î±-Subunit GsÎ± Is Required for Craniofacial Morphogenesis. PLoS ONE, 2016, 11, e0147535.	1.1	8
114	GsÎ± deficiency in the dorsomedial hypothalamus leads to obesity, hyperphagia, and reduced thermogenesis associated with impaired leptin signaling. Molecular Metabolism, 2019, 25, 142-153.	3.0	8
115	GÎ±s Relays Sphingosine-1-Phosphate Receptor 1 Signaling to Stabilize Vascular Endothelial-Cadherin at Endothelial Junctions to Control Mouse Embryonic Vascular Integrity. Journal of Genetics and Genomics, 2015, 42, 613-624.	1.7	7
116	[24] Detection of mutations and polymorphisms of GsÎ± subunit gene by denaturing gradient Gel electrophoresis. Methods in Enzymology, 1994, 237, 308-320.	0.4	6
117	Analysis of Genomic Imprinting of GsÎ± Gene. Methods in Enzymology, 2002, 344, 369-383.	0.4	6
118	GsÎ±-dependent signaling is required for postnatal establishment of a functional Î²-cell mass. Molecular Metabolism, 2021, 53, 101264.	3.0	6
119	InÂvivo metabolic effects after acute activation of skeletal muscle Gs signaling. Molecular Metabolism, 2022, 55, 101415.	3.0	5
120	Deletion of GÎ±q/11 or GÎ±s Proteins in Gonadotropes Differentially Affects Gonadotropin Production and Secretion in Mice. Endocrinology, 2022, 163, .	1.4	5
121	G_qÎ±/G₁₁Î± deficiency in dorsomedial hypothalamus leads to obesity resulting from decreased energy expenditure and impaired sympathetic nerve activity. American Journal of Physiology - Endocrinology and Metabolism, 2021, 320, E270-E280.	1.8	4
122	Parathyroid Hormone Resistance and Autoantibodies to the PTH1 Receptor. New England Journal of Medicine, 2021, 385, 1974-1980.	13.9	4
123	GsÎ±, Pseudohypoparathyroidism, Fibrous Dysplasia, and McCuneâ€“Albright Syndrome. , 2013, , 425-440.		3
124	Diseases Resulting from Defects in the G Protein GsÎ±. , 2008, , 1453-1477.		2
125	Fibrous Dysplasia and the McCune-Albright Syndrome. , 2000, , 163-177.		1
126	Stimulatory G-Protein Î± Subunit Modulates Endothelial Cell Permeability Through Regulation of Plasmalemma Vesicle-Associated Protein. Frontiers in Pharmacology, 2022, 13, .	1.6	1

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127	Imprinting at theGNASlocus and endocrine disease. , 2005, , .		0
128	Fluorosis. , 2009, , 665-665.		0
129	GsÎ±, Pseudohypoparathyroidism, Fibrous Dysplasia, and McCuneâ€“Albright Syndrome. , 2018, , 637-653.		0
130	Diseases resulting from defects in the G protein GsÎ±. , 2020, , 1431-1461.		0
131	G-proteins | Gs Family of Heterotrimeric G Proteins. , 2021, , 456-461.		0
132	Signal Transduction of PTH and PTHrP. , 2001, , 117-126.		0
133	Other Skeletal Diseases Resulting from G Protein Defects. , 2002, , 1165-XLII.		0