Lee S Weinstein

List of Publications by Year
in descending order

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Version: 2024-02-01

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	InÂvivo metabolic effects after acute activation of skeletal muscle Gs signaling. Molecular Metabolism, 2022, 55, 101415.	3.0	5
2	Clenbuterol exerts antidiabetic activity through metabolic reprogramming of skeletal muscle cells. Nature Communications, 2022, 13, 22.	5.8	15
3	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
4	Stimulatory G-Protein Î± Subunit Modulates Endothelial Cell Permeability Through Regulation of Plasmalemma Vesicle-Associated Protein. Frontiers in Pharmacology, 2022, 13, .	1.6	1
5	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
6	G-proteins | Gs Family of Heterotrimeric G Proteins. , 2021, , 456-461.		0
7	Mechanochemical control of epidermal stem cell divisions by B-plexins. Nature Communications, 2021, 12, 1308.	5.8	24
8	GsÎ±-dependent signaling is required for postnatal establishment of a functional Î²-cell mass. Molecular Metabolism, 2021, 53, 101264.	3.0	6
9	Parathyroid Hormone Resistance and Autoantibodies to the PTH1 Receptor. New England Journal of Medicine, 2021, 385, 1974-1980.	13.9	4
10	Diseases resulting from defects in the G protein GsÎ±. , 2020, , 1431-1461.		0
11	Single-Cell RNA Profiling Reveals Adipocyte to Macrophage Signaling Sufficient to Enhance Thermogenesis. Cell Reports, 2020, 32, 107998.	2.9	60
12	Control of Adipocyte Thermogenesis and Lipogenesis through Î²3-Adrenergic and Thyroid Hormone Signal Integration. Cell Reports, 2020, 31, 107598.	2.9	37
13	Disturbed flowâ€“induced Gs-mediated signaling protects against endothelial inflammation and atherosclerosis. JCI Insight, 2020, 5, .	2.3	16
14	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
15	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
16	Shear stressâ€“induced endothelial adrenomedullin signaling regulates vascular tone and blood pressure. Journal of Clinical Investigation, 2019, 129, 2775-2791.	3.9	129
17	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
18	GsÎ±, Pseudohypoparathyroidism, Fibrous Dysplasia, and McCuneâ€“Albright Syndrome. , 2018, , 637-653.		0

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19	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
20	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
21	Heterotrimeric G Stimulatory Protein Î± Subunit Is Required forÂIntestinal Smooth Muscle Contraction in Mice. Gastroenterology, 2017, 152, 1114-1125.e5.	0.6	12
22	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
23	Oriented clonal cell dynamics enables accurate growth and shaping of vertebrate cartilage. ELife, 2017, 6, .	2.8	46
24	G-Protein Î±-Subunit GsÎ± Is Required for Craniofacial Morphogenesis. PLoS ONE, 2016, 11, e0147535.	1.1	8
25	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
26	Reoperative Surgery in Patients with Multiple Endocrine Neoplasia Type 1 Associated Primary Hyperparathyroidism. Annals of Surgical Oncology, 2016, 23, 701-707.	0.7	22
27	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
28	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
29	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
30	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
31	GsÎ± deficiency in the dorsomedial hypothalamus underlies obesity associated with GsÎ± mutations. Journal of Clinical Investigation, 2016, 127, 500-510.	3.9	40
32	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
33	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
34	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
35	Gq/11Î± and GsÎ± mediate distinct physiological responses to central melanocortins. Journal of Clinical Investigation, 2015, 126, 40-49.	3.9	74
36	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

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37	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
38	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
39	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
40	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
41	Agonist-Independent GPCR Activity Regulates Anterior-Posterior Targeting of Olfactory Sensory Neurons. Cell, 2013, 154, 1314-1325.	13.5	126
42	Activation of Hedgehog signaling by loss of GNAS causes heterotopic ossification. Nature Medicine, 2013, 19, 1505-1512.	15.2	187
43	Myelopoiesis is regulated by osteocytes through GsÎ±-dependent signaling. Blood, 2013, 121, 930-939.	0.6	146
44	Reduced Insulin Sensitivity in Adults With Pseudohypoparathyroidism Type 1a. Journal of Clinical Endocrinology and Metabolism, 2013, 98, E1796-E1801.	1.8	40
45	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
46	GsÎ±, Pseudohypoparathyroidism, Fibrous Dysplasia, and McCuneâ€"Albright Syndrome. , 2013, , 425-440.		3
47	Sleeping Parathyroid Tumor: Rapid Hyperfunction after Removal of the Dominant Tumor. Journal of Clinical Endocrinology and Metabolism, 2012, 97, 1834-1841.	1.8	13
48	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
49	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
50	Preoperative Localizing Studies for Initial Parathyroidectomy in MEN1 Syndrome: Is There Any Benefit?. World Journal of Surgery, 2012, 36, 1368-1374.	0.8	32
51	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
52	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
53	Effects of deficiency of the G protein GsÎ± on energy and glucose homeostasis. European Journal of Pharmacology, 2011, 660, 119-124.	1.7	18
54	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

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55	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
56	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
57	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
58	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
59	Pancreas-specific GsÎ± deficiency has divergent effects on pancreatic Î±- and Î²-cell proliferation. Journal of Endocrinology, 2010, 206, 261-269.	1.2	24
60	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
61	Renal Failure in Mice with Gs-alpha Deletion in Juxtaglomerular Cells. American Journal of Nephrology, 2010, 32, 83-94.	1.4	20
62	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
63	GNASHaploinsufficiency Leads to Subcutaneous Tumor Formation With Collagen and Elastin Deposition and Calcification. Endocrine Research, 2009, 34, 1-9.	0.6	8
64	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
65	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
66	Reoperation for parathyroid adenoma: A contemporary experience. Surgery, 2009, 146, 1144-1155.	1.0	57
67	Haematopoietic stem cells depend on GÎ±s-mediated signalling to engraft bone marrow. Nature, 2009, 459, 103-107.	13.7	69
68	Central Nervous System Imprinting of the G Protein GsÎ± and Its Role in Metabolic Regulation. Cell Metabolism, 2009, 9, 548-555.	7.2	118
69	Fluorosis. , 2009, , 665-665.		0
70	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
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	Diseases Resulting from Defects in the G Protein GsÎ±. , 2008, , 1453-1477.		2
74	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
75	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
76	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
77	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
78	Studies of the regulation and function of the GsÎ± gene Gnas using gene targeting technology. , 2007, 115, 271-291.		70
79	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
80	GsÎ± Mutations in Fibrous Dysplasia and McCune-Albright Syndrome. Journal of Bone and Mineral Research, 2006, 21, P120-P124.	3.1	102
81	Genetic diseases associated with heterotrimeric G proteins. Trends in Pharmacological Sciences, 2006, 27, 260-266.	4.0	90
82	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
83	Fibroblast Growth Factor-23 Is Regulated by 1Î±,25-Dihydroxyvitamin D. Journal of Bone and Mineral Research, 2005, 20, 1944-1950.	3.1	92
84	Imprinting at theGNASlocus and endocrine disease. , 2005, , .		0
85	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
86	Distinct patterns of abnormal GNAS imprinting in familial and sporadic pseudohypoparathyroidism type IB. Human Molecular Genetics, 2005, 14, 95-102.	1.4	117
87	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
88	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
89	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
90	Increased Insulin Sensitivity in PaternalGnasKnockout Mice Is Associated with Increased Lipid Clearance. Endocrinology, 2004, 145, 4094-4102.	1.4	79

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91	Minireview: GNAS: Normal and Abnormal Functions. Endocrinology, 2004, 145, 5459-5464.	1.4	291
92	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
93	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
94	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
95	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
96	Inherited Diseases Involving G Proteins and G Proteinâ€“Coupled Receptors. Annual Review of Medicine, 2004, 55, 27-39.	5.0	228
97	Persistent Primary Hyperparathyroidism Caused by Adenomas Identified in Pharyngeal or Adjacent Structures. World Journal of Surgery, 2003, 27, 675-679.	0.8	38
98	Results of initial operation for hyperparathyroidism in patients with multiple endocrine neoplasia type 1. Surgery, 2003, 134, 858-864.	1.0	91
99	HRPT2,a Marker of Parathyroid Cancer. New England Journal of Medicine, 2003, 349, 1691-1692.	13.9	54
100	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
101	Analysis of Genomic Imprinting of GsÎ± Gene. Methods in Enzymology, 2002, 344, 369-383.	0.4	6
102	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
103	Gs_{Î±} Mutations and Imprinting Defects in Human Disease. Annals of the New York Academy of Sciences, 2002, 968, 173-197.	1.8	137
104	Other Skeletal Diseases Resulting from G Protein Defects. , 2002, , 1165-XLII.		0
105	The role of tissue-specific imprinting as a source of phenotypic heterogeneity in human disease. Biological Psychiatry, 2001, 50, 927-931.	0.7	27
106	Reoperation for hyperparathyroidism in multiple endocrine neoplasia type 1. Surgery, 2001, 130, 991-998.	1.0	73
107	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
108	Increased Insulin Sensitivity in GsÎ± Knockout Mice. Journal of Biological Chemistry, 2001, 276, 19994-19998.	1.6	53

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109	Endocrine Manifestations of Stimulatory G Protein Î±-Subunit Mutations and the Role of Genomic Imprinting. Endocrine Reviews, 2001, 22, 675-705.	8.9	390
110	Signal Transduction of PTH and PTHrP. , 2001, , 117-126.		0
111	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
112	Identification of a Methylation Imprint Mark within the Mouse Gnas Locus. Molecular and Cellular Biology, 2000, 20, 5808-5817.	1.1	181
113	Fibrous Dysplasia and the McCune-Albright Syndrome. , 2000, , 163-177.		1
114	A GNAS1 imprinting defect in pseudohypoparathyroidism type IB. Journal of Clinical Investigation, 2000, 106, 1167-1174.	3.9	263
115	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
116	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
117	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
118	Results of heterotopic parathyroid autotransplantation: A 13-year experience. Surgery, 1999, 126, 1042-1048.	1.0	70
119	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
120	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
121	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
122	Albright Hereditary Osteodystrophy, Pseudohypoparathyroidism, and Gs Deficiency. , 1998, , 23-56.		38
123	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
124	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
125	[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
126	G protein mutations in human disease. Clinical Biochemistry, 1993, 26, 333-338.	0.8	49

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127	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
128	Receptor-Effector Coupling by G Proteins: Implications for Normal and Abnormal Signal Transduction. Endocrine Reviews, 1992, 13, 536-565.	8.9	308
129	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
130	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
131	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
132	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
133	Characterization of the Promoter of the Human Gi2Î±-Subunit Gene. Molecular Endocrinology, 1990, 4, 958-964.	3.7	15