Anna L Gloyn

List of Publications by Year
in descending order

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161 papers	21,178 citations	14655 66 h-index	10734 138 g-index
212 all docs	212 docs citations	212 times ranked	22268 citing authors

#	Article	IF	Citations
1	Multi-ancestry genetic study of type 2 diabetes highlights the power of diverse populations for discovery and translation. Nature Genetics, 2022, 54, 560-572.	21.4	250
2	Monogenic diabetes: a gateway to precision medicine in diabetes. Journal of Clinical Investigation, 2021, 131, .	8.2	77
3	The trans-ancestral genomic architecture of glycemic traits. Nature Genetics, 2021, 53, 840-860.	21.4	341
4	There is more than one way to reach type 2 diabetes. Nature Metabolism, 2021, 3, 894-895.	11.9	2
5	Genetics of Type 2 Diabetes: Opportunities for Precision Medicine. Journal of the American College of Cardiology, 2021, 78, 496-512.	2.8	12
6	100 YEARS OF INSULIN: A brief history of diabetes genetics: insights for pancreatic beta-cell development and function. Journal of Endocrinology, 2021, 250, R23-R35.	2.6	7
7	TIGER: The gene expression regulatory variation landscape of human pancreatic islets. Cell Reports, 2021, 37, 109807.	6.4	45
8	Analysis of Differentiation Protocols Defines a Common Pancreatic Progenitor Molecular Signature and Guides Refinement of Endocrine Differentiation. Stem Cell Reports, 2020, 14, 138-153.	4.8	31
9	Response to Comment on Misra et al. Homozygous Hypomorphic HNF1A Alleles Are a Novel Cause of Young-Onset Diabetes and Result in Sulfonylurea-Sensitive Diabetes. Diabetes Care 2020;43:909â€“912. Diabetes Care, 2020, 43, e155-e156.	8.6	0
10	A Multi-omic Integrative Scheme Characterizes Tissues of Action at Loci Associated with Type 2 Diabetes. American Journal of Human Genetics, 2020, 107, 1011-1028.	6.2	23
11	Unsupervised Clustering of Missense Variants in HNF1A Using Multidimensional Functional Data Aids Clinical Interpretation. American Journal of Human Genetics, 2020, 107, 670-682.	6.2	25
12	Identification of type 2 diabetes loci in 433,540 East Asian individuals. Nature, 2020, 582, 240-245.	27.8	282
13	Insights into pancreatic islet cell dysfunction from type 2 diabetes mellitus genetics. Nature Reviews Endocrinology, 2020, 16, 202-212.	9.6	89
14	Editorial Overview: â€œIslet Biology in Type 2 Diabetesâ€• Journal of Molecular Biology, 2020, 432, 1307-1309.	4.2	0
15	From Genetic Association to Molecular Mechanisms for Islet-cell Dysfunction in Type 2 Diabetes. Journal of Molecular Biology, 2020, 432, 1551-1578.	4.2	27
16	Endocrine-Exocrine Signaling Drives Obesity-Associated Pancreatic Ductal Adenocarcinoma. Cell, 2020, 181, 832-847.e18.	28.9	77
17	Genetic variant effects on gene expression in human pancreatic islets and their implications for T2D. Nature Communications, 2020, 11, 4912.	12.8	89
18	Homozygous Hypomorphic HNF1A Alleles Are a Novel Cause of Young-Onset Diabetes and Result in Sulfonylurea-Sensitive Diabetes. Diabetes Care, 2020, 43, 909-912.	8.6	13

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19	Deep learning models predict regulatory variants in pancreatic islets and refine type 2 diabetes association signals. ELife, 2020, 9, .	6.0	28
20	Exocrine or endocrine? A circulating pancreatic elastase that regulates glucose homeostasis. Nature Metabolism, 2019, 1, 853-855.	11.9	4
21	Fostering improved human islet research: a European perspective. Diabetologia, 2019, 62, 1514-1516.	6.3	13
22	Loss of ZnT8 function protects against diabetes by enhanced insulin secretion. Nature Genetics, 2019, 51, 1596-1606.	21.4	96
23	Translational genomics and precision medicine: Moving from the lab to the clinic. Science, 2019, 365, 1409-1413.	12.6	133
24	Developing a network view of type 2 diabetes risk pathways through integration of genetic, genomic and functional data. Genome Medicine, 2019, 11, 19.	8.2	33
25	Plasma Fucosylated Glycans and C-Reactive Protein as Biomarkers of HNF1A-MODY in Young Adultâ€“Onset Nonautoimmune Diabetes. Diabetes Care, 2019, 42, 17-26.	8.6	44
26	A CRISPR/Cas9 genome editing pipeline in the EndoC-Î²H1 cell line to study genes implicated in beta cell function. Wellcome Open Research, 2019, 4, 150.	1.8	21
27	A CRISPR/Cas9 genome editing pipeline in the EndoC-Î²H1 cell line to study genes implicated in beta cell function. Wellcome Open Research, 2019, 4, 150.	1.8	16
28	Patterns of differential gene expression in a cellular model of human islet development, and relationship to type 2 diabetes predisposition. Diabetologia, 2018, 61, 1614-1622.	6.3	14
29	Regulatory variants at KLF14 influence type 2 diabetes risk via a female-specific effect on adipocyte size and body composition. Nature Genetics, 2018, 50, 572-580.	21.4	143
30	Precision medicine in the management of type 2 diabetes. Lancet Diabetes and Endocrinology,the, 2018, 6, 891-900.	11.4	115
31	A Partial Loss-of-Function Variant in AKT2 Is Associated With Reduced Insulin-Mediated Glucose Uptake in Multiple Insulin-Sensitive Tissues: A Genotype-Based Callback Positron Emission Tomography Study. Diabetes, 2018, 67, 334-342.	0.6	37
32	Electrophysiological properties of human beta-cell lines EndoC-Î²H1 and -Î²H2 conform with human beta-cells. Scientific Reports, 2018, 8, 16994.	3.3	39
33	Fine-mapping type 2 diabetes loci to single-variant resolution using high-density imputation and islet-specific epigenome maps. Nature Genetics, 2018, 50, 1505-1513.	21.4	1,331
34	Integration of human pancreatic islet genomic data refines regulatory mechanisms at Type 2 Diabetes susceptibility loci. ELife, 2018, 7, .	6.0	103
35	Type 2 diabetes risk alleles in PAM impact insulin release from human pancreatic Î²-cells. Nature Genetics, 2018, 50, 1122-1131.	21.4	59
36	Understanding human fetal pancreas development using subpopulation sorting, RNA sequencing and single-cell profiling. Development (Cambridge), 2018, 145, .	2.5	78

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37	NKX6.1 induced pluripotent stem cell reporter lines for isolation and analysis of functionally relevant neuronal and pancreas populations. Stem Cell Research, 2018, 29, 220-231.	0.7	18
38	Maturity onset diabetes of the young due to HNF1A variants in Croatia. Biochemia Medica, 2018, 28, 020703.	2.7	17
39	Decreased STARD10 Expression Is Associated with Defective Insulin Secretion in Humans and Mice. American Journal of Human Genetics, 2017, 100, 238-256.	6.2	60
40	Human genetics as a model for target validation: finding new therapies for diabetes. Diabetologia, 2017, 60, 960-970.	6.3	19
41	Prioritising Causal Genes at Type 2 Diabetes Risk Loci. Current Diabetes Reports, 2017, 17, 76.	4.2	25
42	Genes Associated with Pancreas Development and Function Maintain Open Chromatin in iPSCs Generated from Human Pancreatic Beta Cells. Stem Cell Reports, 2017, 9, 1395-1405.	4.8	15
43	Sequence data and association statistics from 12,940 type 2 diabetes cases and controls. Scientific Data, 2017, 4, 170179.	5.3	31
44	The Importance of Context: Uncovering Species- and Tissue-Specific Effects of Genetic Risk Variants for Type 2 Diabetes. Frontiers in Endocrinology, 2016, 7, 112.	3.5	3
45	The genetic architecture of type 2 diabetes. Nature, 2016, 536, 41-47.	27.8	952
46	GCKR: How Genetic Variation Across the Allelic Spectrum Influences Protein Function and Metabolic Traits in Humans. , 2016, , 317-336.		1
47	Systematic Functional Characterization of Candidate Causal Genes for Type 2 Diabetes Risk Variants. Diabetes, 2016, 65, 3805-3811.	0.6	79
48	Insights into islet development and biology through characterization of a human iPSC-derived endocrine pancreas model. Islets, 2016, 8, 83-95.	1.8	21
49	Insights into metabolic disease from studying genetics in isolated populations: stories from Greece to Greenland. Diabetologia, 2016, 59, 938-941.	6.3	8
50	Loss-of-Function Mutations in the Cell-Cycle Control Gene CDKN2A Impact on Glucose Homeostasis in Humans. Diabetes, 2016, 65, 527-533.	0.6	38
51	Genome-edited human stem cell-derived beta cells: a powerful tool for drilling down on type 2 diabetes GWAS biology. F1000Research, 2016, 5, 1711.	1.6	10
52	Transcript Expression Data from Human Islets Links Regulatory Signals from Genome-Wide Association Studies for Type 2 Diabetes and Glycemic Traits to Their Downstream Effectors. PLoS Genetics, 2015, 11, e1005694.	3.5	178
53	Human islet function following 20Âyears of cryogenic biobanking. Diabetologia, 2015, 58, 1503-1512.	6.3	39
54	Recognition and Management of Individuals With Hyperglycemia Because of a Heterozygous Glucokinase Mutation. Diabetes Care, 2015, 38, 1383-1392.	8.6	217

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55	Identification and Functional Characterization of G6PC2 Coding Variants Influencing Glycemic Traits Define an Effector Transcript at the G6PC2-ABCB11 Locus. PLoS Genetics, 2015, 11, e1004876.	3.5	95
56	Glucokinase regulatory protein. Current Opinion in Lipidology, 2015, 26, 88-95.	2.7	94
57	Genetic fine mapping and genomic annotation defines causal mechanisms at type 2 diabetes susceptibility loci. Nature Genetics, 2015, 47, 1415-1425.	21.4	365
58	Isocitrate-to-SENP1 signaling amplifies insulin secretion and rescues dysfunctional Î² cells. Journal of Clinical Investigation, 2015, 125, 3847-3860.	8.2	148
59	When is it MODY? Challenges in the Interpretation of Sequence Variants in MODY Genes. Review of Diabetic Studies, 2015, 12, 330-348.	1.3	21
60	A Panel of Diverse Assays to Interrogate the Interaction between Glucokinase and Glucokinase Regulatory Protein, Two Vital Proteins in Human Disease. PLoS ONE, 2014, 9, e89335.	2.5	4
61	Phenotypic severity of homozygous GCK mutations causing neonatal or childhood-onset diabetes is primarily mediated through effects on protein stability. Human Molecular Genetics, 2014, 23, 6432-6440.	2.9	41
62	Inheritance of rare functional GCKR variants and their contribution to triglyceride levels in families. Human Molecular Genetics, 2014, 23, 5570-5578.	2.9	21
63	Pancreatic islet enhancer clusters enriched in type 2 diabetes risk-associated variants. Nature Genetics, 2014, 46, 136-143.	21.4	475
64	The pancreatic Î² cell: recent insights from human genetics. Trends in Endocrinology and Metabolism, 2014, 25, 425-434.	7.1	29
65	Argonaute2 Mediates Compensatory Expansion of the Pancreatic Î² Cell. Cell Metabolism, 2014, 19, 122-134.	16.2	139
66	Analysis of the co-operative interaction between the allosterically regulated proteins GK and GKRP using tryptophan fluorescence. Biochemical Journal, 2014, 459, 551-564.	3.7	19
67	Reclassification of Diabetes Etiology in a Family With Multiple Diabetes Phenotypes. Journal of Clinical Endocrinology and Metabolism, 2014, 99, E1067-E1071.	3.6	5
68	Translating Genetic Association Signals for Diabetes and Metabolic Traits into Molecular Mechanisms for Disease. Frontiers in Diabetes, 2014, , 133-145.	0.4	0
69	Translating Advances in Our Understanding of the Genetics of Diabetes into the Clinic. Frontiers in Diabetes, 2014, , 173-186.	0.4	0
70	Bridging the Gap Between Genetic Associations and Molecular Mechanisms for Type 2 Diabetes. Current Diabetes Reports, 2013, 13, 778-785.	4.2	10
71	Role of KATP Channels in Glucose-Regulated Glucagon Secretion and Impaired Counterregulation in Type 2 Diabetes. Cell Metabolism, 2013, 18, 871-882.	16.2	179
72	TCF7L2 and Diabetes: A Tale of Two Tissues, and of Two Species. Cell Metabolism, 2013, 17, 157-159.	16.2	21

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73	Apolipoprotein M can discriminate <scp>HNF</scp>1Aâ€<scp>MODY</scp> from Type 1 diabetes. Diabetic Medicine, 2013, 30, 246-250.	2.3	27
74	Small molecular glucokinase activators: has another new antiâ€diabetic therapeutic lost favour?. British Journal of Pharmacology, 2013, 168, 335-338.	5.4	25
75	Mutations in HNF1A Result in Marked Alterations of Plasma Glycan Profile. Diabetes, 2013, 62, 1329-1337.	0.6	97
76	Insights Into the Molecular Mechanism for Type 2 Diabetes Susceptibility at the KCNQ1 Locus From Temporal Changes in Imprinting Status in Human Islets. Diabetes, 2013, 62, 987-992.	0.6	112
77	The miRNA Profile of Human Pancreatic Islets and Beta-Cells and Relationship to Type 2 Diabetes Pathogenesis. PLoS ONE, 2013, 8, e55272.	2.5	178
78	SSTR2 is the functionally dominant somatostatin receptor in human pancreatic Î²- and Î±-cells. American Journal of Physiology - Endocrinology and Metabolism, 2012, 303, E1107-E1116.	3.5	119
79	Novel Loci for Adiponectin Levels and Their Influence on Type 2 Diabetes and Metabolic Traits: A Multi-Ethnic Meta-Analysis of 45,891 Individuals. PLoS Genetics, 2012, 8, e1002607.	3.5	419
80	Insights Into the Pathogenicity of Rare Missense GCK Variants From the Identification and Functional Characterization of Compound Heterozygous and Double Mutations Inherited in Cis. Diabetes Care, 2012, 35, 1482-1484.	8.6	15
81	Reduced Insulin Exocytosis in Human Pancreatic Î²-Cells With Gene Variants Linked to Type 2 Diabetes. Diabetes, 2012, 61, 1726-1733.	0.6	204
82	PTEN Mutations as a Cause of Constitutive Insulin Sensitivity and Obesity. New England Journal of Medicine, 2012, 367, 1002-1011.	27.0	193
83	Metabolic Profiling in Maturity-Onset Diabetes of the Young (MODY) and Young Onset Type 2 Diabetes Fails to Detect Robust Urinary Biomarkers. PLoS ONE, 2012, 7, e40962.	2.5	16
84	Human Î² Cell Transcriptome Analysis Uncovers lncRNAs That Are Tissue-Specific, Dynamically Regulated, and Abnormally Expressed in Type 2 Diabetes. Cell Metabolism, 2012, 16, 435-448.	16.2	410
85	A tale of two glucose transporters: how GLUT2 re-emerged as a contender for glucose transport into the human beta cell. Diabetologia, 2012, 55, 2312-2315.	6.3	24
86	A Genome-Wide Association Search for Type 2 Diabetes Genes in African Americans. PLoS ONE, 2012, 7, e29202.	2.5	197
87	Identification and Functional Characterisation of Novel Glucokinase Mutations Causing Maturity-Onset Diabetes of the Young in Slovakia. PLoS ONE, 2012, 7, e34541.	2.5	22
88	Cellular characterisation of the GCKR P446L variant associated with type 2 diabetes risk. Diabetologia, 2012, 55, 114-122.	6.3	97
89	Correlation of rare coding variants in the gene encoding human glucokinase regulatory protein with phenotypic, cellular, and kinetic outcomes. Journal of Clinical Investigation, 2012, 122, 205-217.	8.2	41
90	High-Sensitivity CRP Discriminates HNF1A-MODY From Other Subtypes of Diabetes. Diabetes Care, 2011, 34, 1860-1862.	8.6	90

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91	Genome-Wide Association Identifies Nine Common Variants Associated With Fasting Proinsulin Levels and Provides New Insights Into the Pathophysiology of Type 2 Diabetes. Diabetes, 2011, 60, 2624-2634.	0.6	335
92	GLUT2 (SLC2A2) is not the principal glucose transporter in human pancreatic beta cells: Implications for understanding genetic association signals at this locus. Molecular Genetics and Metabolism, 2011, 104, 648-653.	1.1	142
93	Genome-wide association studies of Type 2 diabetes: are these ready to make an impact in the clinic?. Diabetes Management, 2011, 1, 379-387.	0.5	0
94	Comprehensive Human Adipose Tissue mRNA and MicroRNA Endogenous Control Selection for Quantitative Realâ€Timeâ€PCR Normalization. Obesity, 2011, 19, 888-892.	3.0	108
95	The previously reported T342P GCK missense variant is not a pathogenic mutation causing MODY. Diabetologia, 2011, 54, 2202-2205.	6.3	15
96	A large multi-centre European study validates high-sensitivity C-reactive protein (hsCRP) as a clinical biomarker for the diagnosis of diabetes subtypes. Diabetologia, 2011, 54, 2801-2810.	6.3	105
97	Discovery of a Novel Site Regulating Glucokinase Activity following Characterization of a New Mutation Causing Hyperinsulinemic Hypoglycemia in Humans. Journal of Biological Chemistry, 2011, 286, 19118-19126.	3.4	21
98	Genetically Programmed Defects in Î²-Cell Function. , 2011, , 299-326.		1
99	Global microRNA expression profiles in insulin target tissues in a spontaneous rat model of type 2 diabetes. Diabetologia, 2010, 53, 1099-1109.	6.3	261
100	From Genetic Association to Molecular Mechanism. Current Diabetes Reports, 2010, 10, 452-466.	4.2	27
101	Twelve type 2 diabetes susceptibility loci identified through large-scale association analysis. Nature Genetics, 2010, 42, 579-589.	21.4	1,631
102	Variation across the allele frequency spectrum. Nature Genetics, 2010, 42, 648-650.	21.4	23
103	Evaluation of Serum 1,5 Anhydroglucitol Levels as a Clinical Test to Differentiate Subtypes of Diabetes. Diabetes Care, 2010, 33, 252-257.	8.6	37
104	Naturally Occurring Glucokinase Mutations Are Associated with Defects in Posttranslational S-Nitrosylation. Molecular Endocrinology, 2010, 24, 171-177.	3.7	24
105	New genetic loci implicated in fasting glucose homeostasis and their impact on type 2 diabetes risk. Nature Genetics, 2010, 42, 105-116.	21.4	1,982
106	Assessment of High-Sensitivity C-Reactive Protein Levels as Diagnostic Discriminator of Maturity-Onset Diabetes of the Young Due to HNF1A Mutations. Diabetes Care, 2010, 33, 1919-1924.	8.6	103
107	Coexpression of the Type 2 Diabetes Susceptibility Gene Variants KCNJ11 E23K and ABCC8 S1369A Alter the ATP and Sulfonylurea Sensitivities of the ATP-Sensitive K+ Channel. Diabetes, 2009, 58, 2419-2424.	0.6	104
108	Severe Insulin Resistance and Intrauterine Growth Deficiency Associated With Haploinsufficiency forINSRandCHN2. Diabetes, 2009, 58, 2954-2961.	0.6	23

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109	The P446L variant in GCKR associated with fasting plasma glucose and triglyceride levels exerts its effect through increased glucokinase activity in liver. Human Molecular Genetics, 2009, 18, 4081-4088.	2.9	328
110	Identification of a Novel Î²-Cell Glucokinase (GCK) Promoter Mutation (â^'71G>C) That ModulatesGCKGene Expression Through Loss of Allele-Specific Sp1 Binding Causing Mild Fasting Hyperglycemia in Humans. Diabetes, 2009, 58, 1929-1935.	0.6	34
111	Update of mutations in the genes encoding the pancreatic beta-cell K_{ATP}channel subunits Kir6.2 (KCNJ11) and sulfonylurea receptor 1 (ABCC8) in diabetes mellitus and hyperinsulinism. Human Mutation, 2009, 30, 170-180.	2.5	209
112	Update on mutations in glucokinase (GCK), which cause maturity-onset diabetes of the young, permanent neonatal diabetes, and hyperinsulinemic hypoglycemia. Human Mutation, 2009, 30, 1512-1526.	2.5	403
113	Prevalence of GCK mutations in individuals screened for fasting hyperglycaemia. Diabetologia, 2009, 52, 172-174.	6.3	26
114	Mutations in the third gene shown to alter fasting glucose levels in the population (G6PC2) are not a common cause of monogenic forms of pancreatic Bâ€cell dysfunction. Diabetic Medicine, 2009, 26, 113-114.	2.3	2
115	RD Lawrence Lecture 2009 Old genes, new tricks: learning about blood glucose regulation from naturally occurring genetic variation in humans. Diabetic Medicine, 2009, 26, 1083-1089.	2.3	3
116	Type 2 Diabetes Susceptibility Gene TCF7L2 and Its Role in Î²-Cell Function. Diabetes, 2009, 58, 800-802.	0.6	58
117	Low Frequency Variants in the Exons Only Encoding Isoform A of HNF1A Do Not Contribute to Susceptibility to Type 2 Diabetes. PLoS ONE, 2009, 4, e6615.	2.5	5
118	Species-Specific Differences in the Expression of the HNF1A, HNF1B and HNF4A Genes. PLoS ONE, 2009, 4, e7855.	2.5	67
119	Prevalence and clinical characteristics of maternally inherited diabetes and deafness caused by the mt3243AÂ>ÂG mutation in young adult diabetic subjects in Sri Lanka. Diabetic Medicine, 2008, 25, 370-374.	2.3	11
120	Genetics: how the UKPDS contributed to determining the genetic landscape of Type 2 diabetes. Diabetic Medicine, 2008, 25, 35-40.	2.3	8
121	Permanent Neonatal Diabetes Mellitus Caused by a Novel Homozygous (T168A) Glucokinase (GCK) Mutation: Initial Response to Oral Sulphonylurea Therapy. Journal of Pediatrics, 2008, 153, 122-126.	1.8	46
122	Gene duplications resulting in over expression of glucokinase are not a common cause of hypoglycaemia of infancy in humans. Molecular Genetics and Metabolism, 2008, 94, 268-269.	1.1	3
123	Activating glucokinase (GCK) mutations as a cause of medically responsive congenital hyperinsulinism: prevalence in children and characterisation of a novel GCK mutation.. European Journal of Endocrinology, 2008, 159, 27-34.	3.7	97
124	Monogenic Î²-cell dysfunction in children: clinical phenotypes, genetic etiology and mutational pathways. Pediatric Health, 2008, 2, 517-532.	0.3	9
125	Glucokinase (GCK) and other susceptibility genes for Î²-cell dysfunction: the candidate approach. Biochemical Society Transactions, 2008, 36, 306-311.	3.4	10
126	Mutations in ATP-Sensitive K+ Channel Genes Cause Transient Neonatal Diabetes and Permanent Diabetes in Childhood or Adulthood. Diabetes, 2007, 56, 1930-1937.	0.6	320

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127	Origin of de Novo KCNJ11 Mutations and Risk of Neonatal Diabetes for Subsequent Siblings. Journal of Clinical Endocrinology and Metabolism, 2007, 92, 1773-1777.	3.6	52
128	Cell Biology Assessment of Glucokinase Mutations V62M and G72R in Pancreatic Â-Cells: Evidence for Cellular Instability of Catalytic Activity. Diabetes, 2007, 56, 1773-1782.	0.6	22
129	Monogenic disorders of the pancreatic Î²-cell: personalizing treatment for rare forms of diabetes and hypoglycemia. Personalized Medicine, 2007, 4, 247-259.	1.5	3
130	Relationship between E23K (an established type II diabetes-susceptibility variant within KCNJ11), polycystic ovary syndrome and androgen levels. European Journal of Human Genetics, 2007, 15, 679-684.	2.8	17
131	Heterogeneity in disease severity in a family with a novel G68V GCK activating mutation causing persistent hyperinsulinaemic hypoglycaemia of infancy. Diabetic Medicine, 2007, 24, 1393-1399.	2.3	58
132	Mutations in HHEX are not a common cause of monogenic forms of beta cell dysfunction. Diabetologia, 2007, 50, 2019-2022.	6.3	5
133	Asian MODY: are we missing an important diagnosis?. Diabetic Medicine, 2006, 23, 1257-1260.	2.3	22
134	KCNJ11 activating mutations are associated with developmental delay, epilepsy and neonatal diabetes syndrome and other neurological features. European Journal of Human Genetics, 2006, 14, 824-830.	2.8	134
135	Mutations in KCNJ11, which encodes Kir6.2, are a common cause of diabetes diagnosed in the first 6Âmonths of life, with the phenotype determined by genotype. Diabetologia, 2006, 49, 1190-1197.	6.3	221
136	Mutations in the genes encoding the pancreatic beta-cell K_{ATP}channel subunits Kir6.2 (KCNJ11) and SUR1 (ABCC8) in diabetes mellitus and hyperinsulinism. Human Mutation, 2006, 27, 220-231.	2.5	105
137	Assessment of the Role of Common Genetic Variation in the Transient Neonatal Diabetes Mellitus (TNDM) Region in Type 2 Diabetes: A Comparative Genomic and Tagging Single Nucleotide Polymorphism Approach. Diabetes, 2006, 55, 2272-2276.	0.6	13
138	Defining the genetic aetiology of monogenic diabetes can improve treatment. Expert Opinion on Pharmacotherapy, 2006, 7, 1759-1767.	1.8	22
139	A gating mutation at the internal mouth of the Kir6.2 pore is associated with DEND syndrome. EMBO Reports, 2005, 6, 470-475.	4.5	99
140	KCNJ11activating mutations in Italian patients with permanent neonatal diabetes. Human Mutation, 2005, 25, 22-27.	2.5	131
141	Relapsing diabetes can result from moderately activating mutations in KCNJ11. Human Molecular Genetics, 2005, 14, 925-934.	2.9	184
142	Insights into the Structure and Regulation of Glucokinase from a Novel Mutation (V62M), Which Causes Maturity-onset Diabetes of the Young. Journal of Biological Chemistry, 2005, 280, 14105-14113.	3.4	87
143	Permanent neonatal diabetes in an Asian infant. Journal of Pediatrics, 2005, 146, 131-133.	1.8	51
144	Permanent Neonatal Diabetes due to Mutations in KCNJ11 Encoding Kir6.2. Diabetes, 2004, 53, 2713-2718.	0.6	350

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145	Activating Mutations in the KCNJ11 Gene Encoding the ATP-Sensitive K+ Channel Subunit Kir6.2 Are Rare in Clinically Defined Type 1 Diabetes Diagnosed Before 2 Years. Diabetes, 2004, 53, 2998-3001.	0.6	51
146	Molecular basis of Kir6.2 mutations associated with neonatal diabetes or neonatal diabetes plus neurological features. Proceedings of the National Academy of Sciences of the United States of America, 2004, 101, 17539-17544.	7.1	223
147	Permanent Neonatal Diabetes due to Paternal Germline Mosaicism for an Activating Mutation of the KCNJ11 Gene Encoding the Kir6.2 Subunit of the Î²-Cell Potassium Adenosine Triphosphate Channel. Journal of Clinical Endocrinology and Metabolism, 2004, 89, 3932-3935.	3.6	87
148	Kir6.2 Mutations Are a Common Cause of Permanent Neonatal Diabetes in a Large Cohort of French Patients. Diabetes, 2004, 53, 2719-2722.	0.6	171
149	Mutations in PTF1A cause pancreatic and cerebellar agenesis. Nature Genetics, 2004, 36, 1301-1305.	21.4	405
150	Activating Mutations in the Gene Encoding the ATP-Sensitive Potassium-Channel Subunit Kir6.2 and Permanent Neonatal Diabetes. New England Journal of Medicine, 2004, 350, 1838-1849.	27.0	1,077
151	Quantitative traits associated with the Type 2 diabetes susceptibility allele in Kir6.2. Diabetologia, 2003, 46, 1021-1023.	6.3	14
152	Glucokinase (GCK) mutations in hyper- and hypoglycemia: Maturity-onset diabetes of the young, permanent neonatal diabetes, and hyperinsulinemia of infancy. Human Mutation, 2003, 22, 353-362.	2.5	249
153	Genetics for Endocrinologists: The Molecular Genetic Basis of Endocrine Disorders. Clinical Endocrinology, 2003, 59, 826-826.	2.4	2
154	Large-Scale Association Studies of Variants in Genes Encoding the Pancreatic Â-Cell KATP Channel Subunits Kir6.2 (KCNJ11) and SUR1 (ABCC8) Confirm That the KCNJ11 E23K Variant Is Associated With Type 2 Diabetes. Diabetes, 2003, 52, 568-572.	0.6	688
155	The search for type 2 diabetes genes. Ageing Research Reviews, 2003, 2, 111-127.	10.9	57
156	Insights Into the Biochemical and Genetic Basis of Glucokinase Activation From Naturally Occurring Hypoglycemia Mutations. Diabetes, 2003, 52, 2433-2440.	0.6	150
157	A Putative Functional Polymorphism in the IGF-I Gene: Association Studies With Type 2 Diabetes, Adult Height, Glucose Tolerance, and Fetal Growth in U.K. Populations. Diabetes, 2002, 51, 2313-2316.	0.6	129
158	Maturity-Onset Diabetes of the Young Caused by a Balanced Translocation Where the 20q12 Break Point Results in Disruption Upstream of the Coding Region of Hepatocyte Nuclear Factor-4Â (HNF4A) Gene. Diabetes, 2002, 51, 2329-2333.	0.6	27
159	The role of the HNF4Î± enhancer in type 2 diabetes. Molecular Genetics and Metabolism, 2002, 76, 148-151.	1.1	15
160	The genetics of type 2 diabetes. Best Practice and Research in Clinical Endocrinology and Metabolism, 2001, 15, 293-308.	4.7	56
161	Association studies of variants in promoter and coding regions of beta-cell ATP-sensitive K-channel genes SUR1 and Kir6.2 with Type 2 diabetes mellitus (UKPDSâ€‚53). Diabetic Medicine, 2001, 18, 206-212.	2.3	150