Neurology Asia 2023; 28(4) : 1041 – 1052

Clinical exome sequencing reveals an important role for clinical diagnosis of intellectual disability with definition of seven novel variants

[1]Sinem Yalcintepe, [2]Isik Gorker, [2]Leyla Bozatli, [1]Hazal Sezginer Guler, [1]Drenushe Zhuri, [1]Selma Demir, [1]Emine Ikbal Atli, [1]Engin Atli, [1]Damla Eker, [1]Hakan Gurkan

[1]Trakya University Faculty of Medicine, Department of Medical Genetics, Edirne, Turkey;
[2]Trakya University Faculty of Medicine, Department of Child and Adolescent Psychiatry, Edirne, Turkey

Abstract

Intellectual disability can be defined as a significantly below-average general mental function, accompanied by environmental adaptation and behavioural deterioration. Patient files of 87 children with intellectual disability were evaluated in this study. After clinical exclusion criterias, clinical exome sequencing was performed for 25 of 87 intellectual disability cases with a massively parallel targeted sequencing method. Seventeen variants in the genes *MBOAT7, KDM5C, TUBB3, MAN1B1, GFAP, CACNA1A, BCOR, LMNA, LBR, ALS2, ENPP1, UBE3A, TRAPPC9, HSPG2, AFF2, NLGN4,* and *SOX10* were identified in 14 of 25 patients (56%). Seven of the 17 variants (41.1%) were novel in the genes *KDM5C, BCOR, UBE3A, TRAPPC9, AFF2, NLGN4,* and *SOX10*. Seven cases (7/25, 28%) had a definite diagnosis of intellectual disability with their pathogenic variants. The high rate of variant detection (56%) in the current study shows that multiple gene analysis plays an essential role in diagnosing the uncertain etiology of intellectual disability. This study also presents seven novel variants, which are first reported.

Keywords: Intellectual disability, clinical exome sequencing, novel variant

INTRODUCTION

Intellectual disability is a below-average intelligence function that occurs in the developmental period and is found with disorders in adaptive behaviour. The frequency of intellectual disability is 1-3%.[1] In diagnostic evaluation, rational use of the laboratory with medical and developmental history, three-generation pedigree, dysmorphic examination, neurological examination, and neuroimaging are employed. Intellectual disability is difficult to diagnose in most children and requires further studies. A specific diagnosis provides social support and information sharing for the family and the physician.[2] The diagnostic evaluation should include detailed patient history and physical examination, standard karyotyping, fragile X molecular analysis, neurological imaging, and/or array comparative genomic hybridization (aCGH) studies with subtelomeric fluorescent in situ hybridization (FISH). With the aCGH method, an increase of 10% in diagnostic rate in cases with intellectual disability has been reported.[3]

The neuropathology of intellectual disability has a broad spectrum. Neurogenesis, neuronal identity, differentiation, morphogenesis, signal transmission pathways, neurochemical regulation, local energy source deficiencies, metabolic pathways, molecular transport, locally occurring metabolic toxins, gliogenesis, glial identity, differentiation, astrocyte relationships, and systemic and local effects make up this broad spectrum.[2] Intellectual disability may be seen with disorders of some metabolic pathways. The most common deficiencies involve enzymes, transcription regulators, binding proteins, and signal transduction proteins; deficiencies or disorders of these mechanisms are blamed for up to 90% of intellectual disability.[4] Some of these deficiencies include disorders of oxidoreductase, hydrolase, transferase enzymes, transcription regulatory genes, genes encoding transporter and protein binders, signal transduction, and receptor genes, Rho GTPases, which regulate dendritic

Address correspondence to: Sinem Yalcintepe MD, Trakya University Faculty of Medicine, Department of Medical Genetics, Edirne-Turkey. Tel: 00905377168691, e-mail: sinemyalcintepe@gmail.com

Date of Submission: 16 February, 2023; Date of Acceptance: 20 November 2023

https://doi.org/10.54029/2023rfz

growth and branching, and Rab GTPases, which control synaptic vesicle transport.[5] Dendritic movements become prominent in some diseases, such as Angelman syndrome and Fragile X syndrome. Dendrites are formations located on the axonal structure and show continuous development and activation.[6] Dendritic spurs undergo dynamic changes in both early development and learning, and changes in the morphology of these protrusions can be seen after intense synaptic activity. The morphology and density of these protrusions are abnormal in many types of intellectual disability[7], they also differ in certain neurological and neuropsychiatric diseases. In Fragile X syndrome and trisomy 13, they appear elongated and sedimentary. In Down syndrome, they are either completely absent or very few and almost invisible.[8]

Fragile X syndrome is the most common cause of intellectual disability after Down syndrome. It has been reported to be seen in 2-4% of cases of intellectual disability, and it has been observed that the incidence is increased in more severe cases.[9] Fragile X syndrome is less common (1%) in those with borderline intelligence.[9] In various notable studies in the literature, it has been reported that aCGH can determine the etiology in 10-20% of cases with intellectual disability and pervasive developmental delay.[9] Although the aetiological classification of intellectual disability has been specified with different percentages in various studies, unknown causes constitute the largest group.[10]

In the current study, a cohort with intellectual disability was evaluated for single-gene etiology using a clinical exome sequencing panel (6699 genes) after excluding chromosomal abnormalities, microdeletion/duplications, and Fragile X syndrome.

METHODS

This study included cases with unexplained intellectual disability after excluding pathologies for structural and numerical chromosomal abnormalities, microdeletion/duplications, and Fragile X Syndrome. The inclusion criterias were: Age from 5 to 18 years, intelligence quotient (IQ)<70, no history of brain trauma, and no history of infection in the perinatal period. Eighty-seven cases were evaluated firstly in the current study. Considering the inclusion criterias, sixty-two cases with chromosomal abnormalities, microdeletion/duplications, and Fragile X Syndrome were excluded in this study: Thirty-six cases were diagnosed as Trisomy 21, fourteen cases had a structural chromosomal abnormality, two cases had Fragile X diagnosis, ten cases had a microdeletion/duplication. Twenty-five cases out of eighty-seven were included in our study after all exclusion criterias (Figure 1a,b).

All cases were examined in Child and Adolescent Psychiatry and Medical Genetics Departments from January 2017 to February 2021.

Molecular analysis

After obtaining the written informed consent forms, genomic DNA was extracted from each index patient and his or her parents or other family members from peripheral blood using the EZ1 DNA Investigator Kit (Qiagen, Hilden, Germany) according to the standard procedure.

A clinical exome gene panel including 6699 OMIM genes was analyzed on the Illumina NextSeq550 (Illumina Inc., San Diego, CA, USA) system. Libraries were prepared according to the instructions of the manufacturer's instructions. Quality control of the prepared libraries was done with the Qubit dsDNA BR Assay system (Invitrogen, Carlsbad, CA). Fastq generation was performed on the Illumina NextSeq550 (Illumina Inc., San Diego, CA, ABD) system. Libraries covering the target genes were prepared according to the QIAseq Targeted DNA Panel protocol (Qiagen, Hilden, Germany). Following the target enrichment process, libraries were sequenced on the Illumina NextSeq550 (Illumina Inc., San Diego, CA, ABD) system. QCI analysis (Qiagen, Hilden, Germany) was used for Quality control and ordering Variant Call Format files. Variant analysis was performed with Ingenuity software (Qiagen, Hilden, Germany).

ACMG-2015 (American College of Medical Genetics)[11] guidelines were followed for the classification of all the variants, and recommendations of the Human Genome Variation Society[12] were followed to describe the novel variants. In addition, ClinVar[13], HGMD-Professional 2020 database, and literature information were considered for collecting the information about known variants. Variants were classified as "pathogenic," "likely pathogenic," "uncertain significance," "likely benign," and "benign" according to the ACMG guidelines.

Sanger sequencing was performed for the index patients and parents to validate the segregation of the variants using a 3500 Genetic Analyzer (Applied Biosystems, USA) capillary electrophoresis system.

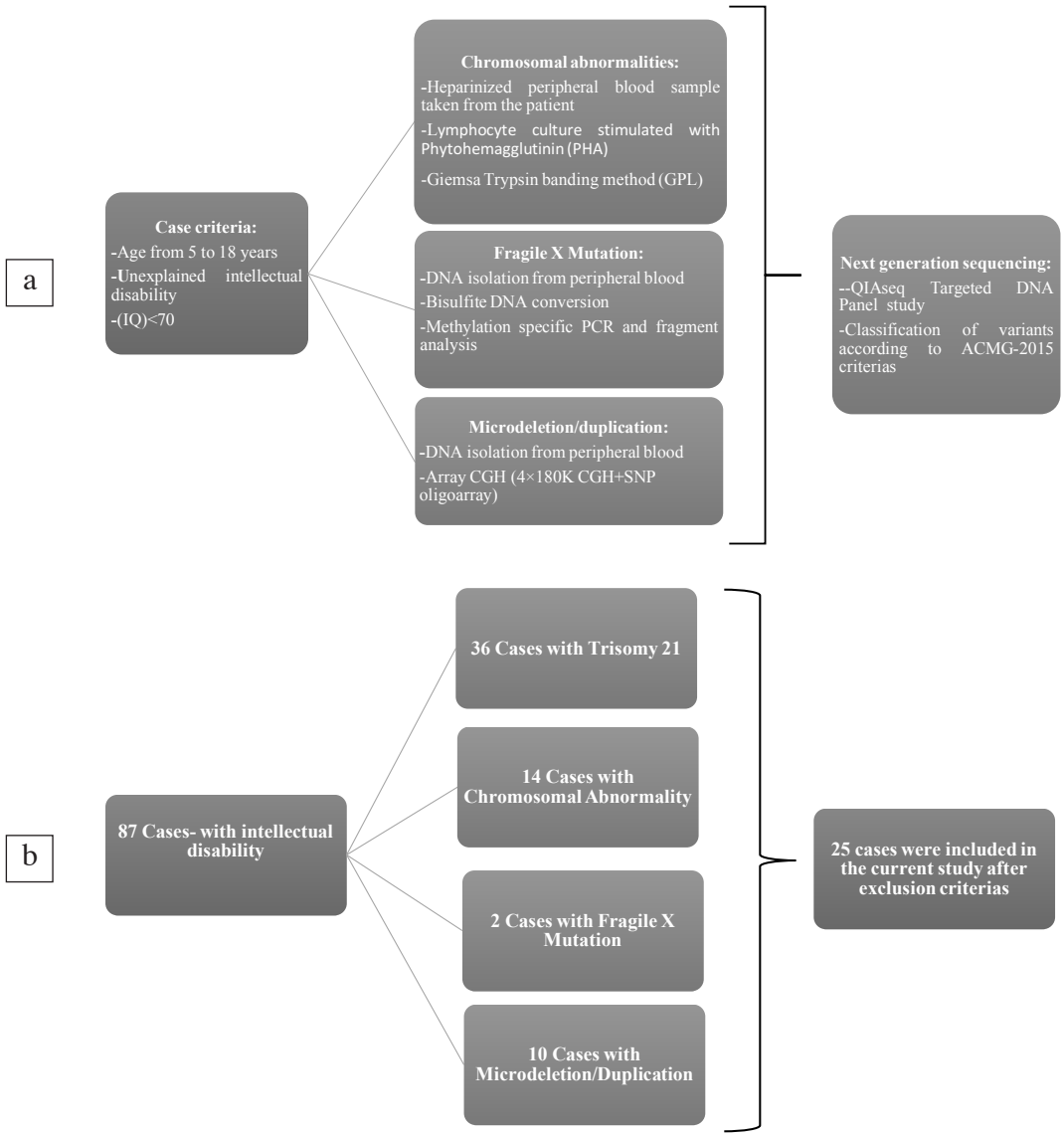


Figure 1. The exclusion chart of our study showing
a. Methods used in the current study for each exclusion step
b. Case numbers excluded from the study for each crtieria

This study is approved by the Ethical Committee of Trakya University Faculty of Medicine with the number 2021/407 and performed in consonance with the principles of the Declaration of Helsinki.

RESULTS

Twenty-five cases who met the inclusion criteria with intellectual disability were evaluated with clinical exome sequencing in the current study. The mean age of these twenty-five cases was 7.2. Clinical exome sequencing showed fourteen of twenty-five cases (56%) had a clinically related variant with intellectual disability (Tables 1,2). Seventeen different variants were identified including seven novel variants. Seven cases (7/25, 28%) had a definite diagnosis of intellectual disability with their pathogenic variants (cases 1,2,3,4,5,6, and 10). The consanguinity marriage rate was not high between the parents, only two of 14 parents were consanguineous. Pedigrees of ten cases were shown in Figures 2 and 3. Four pedigrees could not be able to be included, because parents did not know the exact numbers of their sisters or brothers. The dysmorphic features of our five cases were shown in Figure 4.

Table 1: Pathogenic/likely pathogenic variants detected in the current study and demographic information of the cases

Case/ Gender/ Age	Clinical findings together with ID	Gene	Coordinate (hg19)	Transcript: Nucleotide change, Protein Change	Zygosity/ Segregation	dbSNP	ClinVar Variation Number	HGMD	ACMG Classification	OMIM phenotypes
1/F/5	Epilepsy, speech delay	*MBOAT7*	chr19: 54684654	NM_024298.5:c.680_690del p.(Leu227ProfsTer65)	Homozygous	rs1264222654	-	-	Pathogenic (PVS1, PM2, PP3)	Mental retardation, autosomal recessive 57 (AR)
2/M/17	Short stature	*KDM5C*	chrX: 53246435	NM_004187.5:c.547A>G p.(Asn183Asp)	Hemizygous (maternal)	-	-	-	Likely pathogenic (PS4, PM2, PP2, BP4)	Mental retardation, X-linked, syndromic, Claes-Jensen type (XLR)
3/M/16	Dysmorphic features, epilepsy	*TUBB3*	chr16: 90001472	NM_006086.4:c.613G>A p.(Glu205Lys)	Heterozygous (*de novo*)	rs878853257	30274	CM107130	Pathogenic (PS3, PM2, PM5, PP2, PP3,PP5)	Cortical dysplasia, complex, with other brain malformations 1 (AD), Fibrosis of extraocular muscles, congenital, 3A (AD)
4/M/6	-	*MAN1B1*	chr9: 139996074	NM_016219.5:c.1204C>T p.(Gln402Ter)	Homozygous	rs865997258	-	-	Pathogenic (PVS1, PM2, PP3)	Rafiq syndrome (AR)
5/F/5	Macrocephaly, developmental delay, synophris	*GFAP*	chr17: 42987523	NM_001131019.3:c.1276_1277delACinsGT p.(Thr426Val)	Heterozygous	rs386797323	-	-	Likely pathogenic (PM1, PM2, PP2, PP3)	Alexander disease (AD)
6/M/10	Mild dysmorphic features, epilepsy	*CACNA1A*	chr19: 13320252	NM_001127222.2:c.6400C>T p.(Arg2134Cys)	Heterozygous	rs121908235	68440	CM041265	Likely pathogenic (PM2, PP2, PP3, PP5)	Developmental and epileptic encephalopathy 42 (AD), Episodic ataxia, type 2 (AD), Migraine, familial hemiplegic, 1 (AD), Migraine, familial hemiplegic, 1, with progressive cerebellar ataxia (AD), Spinocerebellar ataxia 6 (AD)
7/F/5	Motor retardation, blue sclera	*LMNA*	chr1: 156106048	NM_005572.4:c.1201C>T p.(Arg401Cys)	Heterozygous	rs61094188	48035	CM023951	Likely pathogenic (PM1, PM2, PP2, PP3, PP5)	Cardiomyopathy, dilated, 1A (AD), Charcot-Marie-Tooth disease, type 2B1 (AR), Emery-Dreifuss muscular dystrophy 2, autosomal dominant (AD), Emery-Dreifuss muscular dystrophy 3, autosomal recessive (AR), Heart-hand syndrome, Slovenian type (AD), Hutchinson-Gilford progeria (AD), Lipodystrophy, familial partial, type 2 (AD), Malouf syndrome (AD), Mandibuloacral dysplasia (AR), Muscular dystrophy, congenital (AD), Restrictive dermopathy, lethal (AR)
8/M/7	Atypic autism, mild dysmorphic features	*LBR*	chr1: 225592154	NM_194442.2:c.1639A>G p.(Asn547Asp)	Heterozygous (de novo)	rs587777171	100900	CM081321	Pathogenic (PM2, PM5, PP3,PP5)	?Reynolds syndrome (AD), Greenberg skeletal dysplasia (AR), Pelger-Huet anomaly (AD)

9/F/16	Motor retardation, cerebral atrophy	*ALS2*	chr2: 202626247	NM_020919.4:c.470G>A p.(Cys157Tyr)	Heterozygous	rs121908138	4415	CM061636	Likely pathogenic (PM2, PP2, PP3, PP5)	Amyotrophic lateral sclerosis 2, juvenile (AR), Primary lateral sclerosis, juvenile (AR), Spastic paralysis, infantile onset ascending (AR)
		ENPP1	chr6: 132206103	NM_006208.3:c.2344C>T p.(Arg782Ter)	Heterozygous	-	-	CM062607	Pathogenic (PVS1, PM2, PP3)	{Diabetes mellitus, non-insulin-dependent, susceptibility to} (AD), {Obesity, susceptibility to} (AD, AR), Arterial calcification, generalized, of infancy, 1 (AR), Cole disease (AD), Hypophosphatemic rickets, autosomal recessive, 2 (AR)
10/M/5	-	*UBE3A*	chr15: 25616056	NM_000462.5:c.1274dupT p.(Asp426GlyfsTer6)	Heterozygous	-	-	-	Pathogenic (PVS1, PM2, PP3)	Angelman syndrome (AD)
14/M/15	Hearing loss, dystophia cantorum, motor retardation	*SOX10*	chr22: 38374075	NM_006941.4:c.496A>G p.(Lys166Glu)	Heterozygous	-	-	-	Likely pathogenic (PM1, PM2, PP2, PP3)	PCWH syndrome (AD), Waardenburg syndrome, type 2E, with or without neurologic involvement (AD), Waardenburg syndrome, type 4C (AD)

ID: Intellectual disability, dbSNP: The Single Nucleotide Polymorphism Database, HGMD: The Human Gene Mutation Database, ACMG: American College of Medical Genetics, *OMIM: Online Mendelian Inheritance in Man, F: Female, M: Male, chr: Chromosome, AR: Autosomal recessive, XLR: X Linked recessive, AD: Autosomal dominant, XLD: X Linked dominant*

Table 2: Variants of unknown clinical significance detected in the current study and demographic information of the cases

Case/ Gender/ Age	Clinical findings together with ID	Gene	Coordinate (hg19)	Transcript: Nucleotide change, Protein Change	Zygosity/ Segregation	dbSNP	ClinVar Variation Number	HGMD	ACMG Classification	OMIM phenotypes
7/F/5	Motor retardation, blue sclera	*BCOR*	chrX: 39914668	NM_001123385.2:c.4694C>T p.(Thr1565Ile)	Heterozygou	-	-	-	VUS (PM2,PP2,PP3)	Microphthalmia, syndromic 2 (XLD)
10/M/5	-	*TRAPPC9*	chr8: 140743401	NM_031466.8:c.3350A>C p.(His1117Pro)	Heterozygous	-	-	-	VUS (PM2,PP2, BP4)	Mental retardation, autosomal recessive 13 (AR)
11/M/5	Motor retardation	*HSPG2*	chr1: 22159838	NM_005529.7:c.11018T>C p.(Phe3673Ser)	Homozygous	rs147707402	587472	-	VUS (PM2,PP2,PP3)	Dyssegmental dysplasia, Silverman-Handmaker type (AR), Schwartz-Jampel syndrome, type 1 (AR)
12/M/10	-	*AFF2*	chrX: 148049184	NM_002025.4:c.3229C>A p.(Gln1077Lys)	Hemizygous (Maternal)	-	-	-	VUS (PM2,PP2,PP3)	Mental retardation, X-linked, FRAXE type (XLR)
13/M/7	Growth retardation Speech delay	*NLGN4*	chrX: 5821410	NM_020742.3:c.1309C>T p.(Arg437Trp)	Hemizygous (Maternal)	-	-	-	VUS (PM2, PP2, PP3)	{Asperger syndrome susceptibility, X-linked 2} (XL), {Autism susceptibility, X-linked 2} (IC, Mu, XL), Mental retardation, X-linked (IC, Mu, XL)

ID: Intellectual disability, dbSNP: The Single Nucleotide Polymorphism Database, HGMD: The Human Gene Mutation Database, ACMG: American College of Medical Genetics, *OMIM: Online Mendelian Inheritance in Man, F: Female, M: Male, chr: Chromosome, AR: Autosomal recessive, XLR: X Linked recessive, AD: Autosomal dominant, XLD: X Linked dominant*

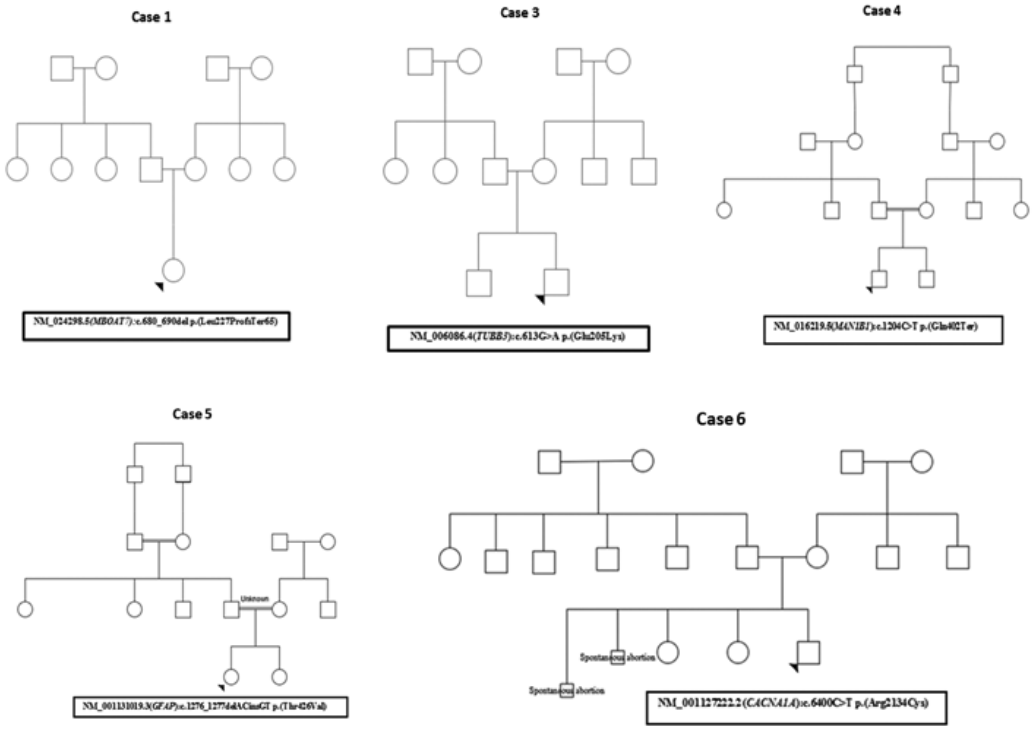


Figure 2. The pedigrees of the cases 1,3,4,5, and 6 in the current study

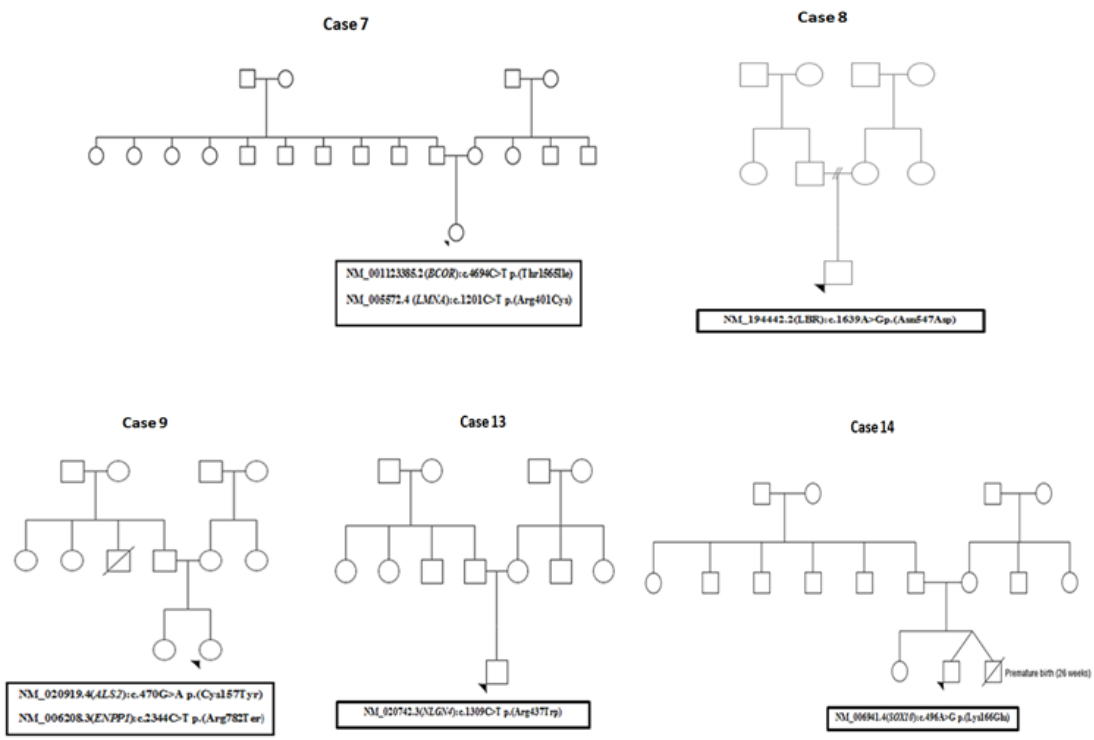


Figure 3. The pedigrees of the cases 7,8,9,13, and 14 in the current study

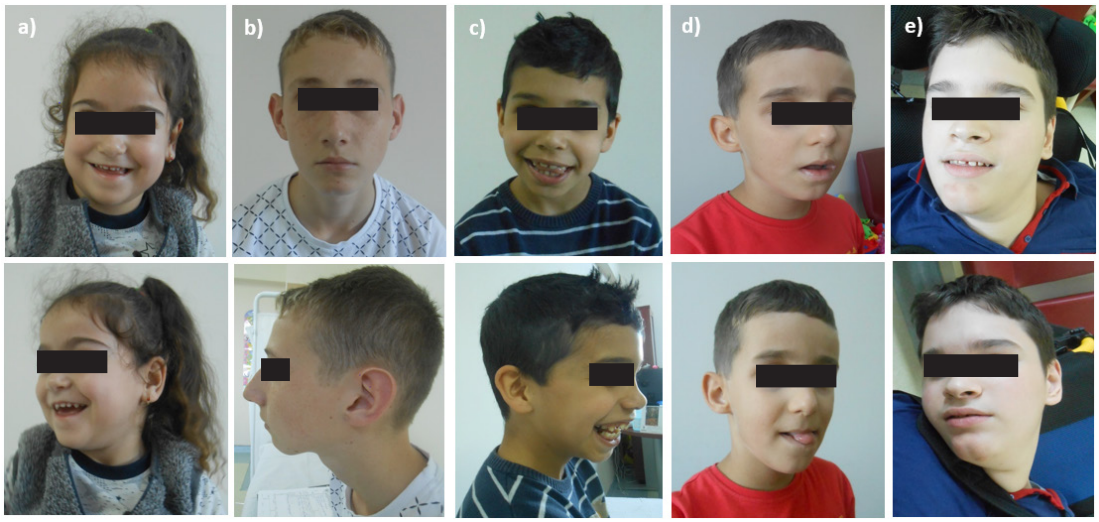


Figure 4. The clinical dysmorphic features of
a. Case 1 with broad forehead, mild synophris, depressed nasal root, smooth philtrum, thin upper lip
b. Case 3 with long face, mild narrow temporal region, big ears, retrognathia
c. Case 6 with low hairline, depressed nasal root, smooth philtrum, thin upper lip, crowded teeth
d. Case 8 with broad forehead, downslanting palpebral fissures, open mouth, low set ears
e. Case 14 with mild hypertelorism, broad nose tip, thin upper lip, short neck

X linked variants

NM_004187.5:c.547A>G p.(Asn183Asp) hemizygous likely pathogenic novel variant in the *KDM5C* gene was detected in case 2, his 11 years old brother had both intellectual disability and short stature, and the same hemizygous variant. Segregation analysis showed that the asymptomatic mother was heterozygous.

NM_001123385.2:c.4694C>T p.(Thr1565Ile) heterozygous novel variant in the *BCOR* gene was classified as VUS in case 7, who has motor retardation and blue sclera. Parents were non-consanguineous.

NM_002025.4:c.3229C>A p.(Gln1077Lys) hemizygous novel variant in the *AFF2* gene was detected in case 12 and his brother. His sister and mother were heterozygous for the same variant. However, this variant was classified as VUS. It is related to the patient's clinical findings defining the intellectual disability.

NM_020742.3:c.1309C>T p.(Arg437Trp) in the *NLGN4* gene was a maternally inherited hemizygous novel VUS variant detected in case 13 who has a speech delay, growth retardation, and absence of seizures. His mouth was open with tongue out, and he had flat feet.

Dominantly inherited variants

NM_006086.4:c.613G>A p.(Glu205Lys) heterozygous *de novo* pathogenic variant in the *TUBB3* gene was in case 3 with microcephaly, short philtrum, micrognathia, arachnodactyly, pectus excavatum, broad toe, and epilepsy.

NM_001131019.3:c.1276_1277delACinsGT p.(Thr426Val) heterozygous pathogenic variant in the *GFAP* gene was detected in case 5 with intellectual disability, macrocephaly, developmental delay, and synophris.

NM_001127222.2:c.6400C>T p.(Arg2134Cys) heterozygous likely pathogenic variant in the *CACNA1A* gene was detected in case 6 with mild dysmorphic features (long philtrum, micrognathia, and macroglossia) and epilepsy.

NM_005572.4:c.1201C>T p.(Arg401Cys) heterozygous likely pathogenic variant in the *LMNA* gene in case 7 who has motor retardation and blue sclera.

NM_194442.2:c.1639A>G p.(Asn547Asp) heterozygous *de novo* pathogenic variant in the *LBR* gene was detected in case 8, who also shows atypic autism, long philtrum, and dysplastic ears.

NM_006208.3:c.2344C>T p.(Arg782Ter) heterozygous pathogenic variant in the *ENPP1*

gene was detected in case 9 who has motor retardation together with cerebral atrophy with intellectual disability.

NM_000462.5:c.1274dupT p.(Asp426GlyfsTer6) heterozygous novel pathogenic variant in the *UBE3A* gene was detected in case 10.

NM_006941.4:c.496A>G p.(Lys166Glu) heterozygous novel likely pathogenic variant in the *SOX10* gene was detected in case 14 with motor retardation, hearing loss, dystopia cantorum, and strabismus.

Recessively inherited variants

NM_024298.5:c.680_690del p.(Leu227ProfsTer65) homozygous pathogenic variant in the *MBOAT7* gene was detected in case 1, who has epilepsy and speech delay. There was no consanguinity between the parents.

NM_016219.5:c.1204C>T p.(Gln402Ter) homozygous pathogenic variant in the *MAN1B1* gene was detected in case 4, whose parents had first-degree cousin marriage.

NM_020919.4:c.470G>A p.(Cys157Tyr) heterozygous likely pathogenic variant in the *ALS2* gene was detected in case 9, who has motor retardation and cerebral atrophy.

NM_031466.8:c.3350A>C p.(His1117Pro) heterozygous novel VUS in the *TRAPPC9* gene was detected in case 10, who was diagnosed as Angelman Syndrome with the *UBE3A* pathogenic variant.

NM_005529.7:c.11018T>C p.(Phe3673Ser) homozygous VUS in the *HSPG2* gene was detected in case 11, who has motor retardation and intellectual disability.

DISCUSSION

The genetic approach of a case with an intellectual disability needs pre-, peri-, and postnatal clinical history; determination of risk factors, noting the pedigree of at least three generations and the parents' socioeconomic status; determination of minor anomalies; and physical measurements, neurological examination, and complete physical examination. The relationship between intellectual disability and congenital malformations is well defined, and detailed dysmorphic examination has a fundamental importance in determining the etiology of intellectual disability. In many studies investigating the causes of intellectual disability, dysmorphic examination and syndrome identification by an experienced clinician is the primary diagnostic method. The clinical history and dysmorphic examination provide the clinician with information about which tests are necessary. In the first examination, genetic testing for intellectual disability etiology includes chromosome analysis, microdeletion/duplication analysis (aCGH method), and Fragile X mutation analysis. The incidence of chromosomal anomalies in cases with intellectual disability varies between 3% and 34%, and it is seen to be around 15% in studies conducted in various centers.[14] After these routine tests, a detailed molecular analysis should be planned for intellectual disability cases. Proof of diagnosis by molecular genetic testing is essential for atypical cases that suggest a diagnosis but do not have typical findings. Atypical presentations of known clinical syndromes are increasing; therefore, molecular studies come to the fore and are useful in clarifying events.

Clinical exome sequencing, which includes 6699 genes, showed 17 intellectual disability-related variants in 14 of 25 (56%) cases in the current study. This is the first report presenting seven novel variants in the *KDM5C, BCOR, UBE3A, TRAPPC9, AFF2, NLGN4,* and *SOX10* genes. Four novel variants and 12 pathogenic/likely pathogenic variants were reported in a study presenting whole-genome sequencing results of 45 intellectual disability cases.[15] Another study analyzing 454 genes in 112 intellectual disability/developmental delay cases reported that 14 variants, 13 of these 14 variants, were novel.[16] A similar study from Turkey reported a molecular diagnosis in 29 patients out of 59 with non-syndromic intellectual disability.[17] With a causative variant detection rate of 56% and with seven novel variants, our study presents valuable data for the literature.

Seven novel variants of 14 (50%) were found in the *KDM5C, BCOR, UBE3A, TRAPPC9, AFF2, NLGN4,* and *SOX10* genes in seven different cases in the current study. The high rate of novel variants determined is remarkable. A study reported a case with a novel *KDM5C* variant (c.3874_3875del) with intellectual disability and severe short stature.[18] A study presenting 19 females carrying 10 novel heterozygous variants, including five probands with *de novo* variants, reported that all affected individuals presented with learning disabilities or intellectual disability; four also had a language impairment mainly affecting expression,

and four heterozygous females were asymptomatic which is similar to case 2 in the current study.[19] A novel variant of uncertain significance (VUS) was detected in case 7 in the *BCOR* gene in our study. Novel pathogenic variants were reported in the *BCOR* gene with congenital cataract[20] and acute myeloid leukemia.[21] *BCOR* gene pathogenic variants are related to syndromic microphthalmia and craniosynostosis.[22] Case 7 had an *LMNA* pathogenic variant with the *BCOR* VUS in the current study. *UBE3A* pathogenic and *TRAPPC9* novel VUS were detected in case 10 in our study. A novel *UBE3A* sequence variant was previously identified in eight related individuals with neurodevelopmental delay, but the phenotypes did not match the clinical criteria for Angelman syndrome in the literature.[23] It is also reported that functional analysis of the reported variant revealed no significant deficits in the UBE3A protein.[23] In our study, case 10 had only intellectual disability. The *TRAPPC9* gene is related to autosomal recessive mental retardation (OMIM #613192), and case 10 had a heterozygous *TRAPPC9* variant, but a functional study could determine the effect for case 10. An *AFF2* novel variant was detected in case 12, a male case inherited maternally, in the current study. It was reported that a heterozygous deletion at Xq27.3q28 including *FMR1, AFF2,* and *IDS* causing intellectual disability and characteristic facial features is infrequent in females, with only 10 patients having been reported.[24] Case 12, in the current study, had a VUS in the *AFF2* gene inherited maternally and due to this gene had been related to intellectual disability. The *NLGN4* novel variant was detected in case 13, in which intellectual disability, growth retardation, and speech delay were seen. *NLGN4* has been reported as a possible implication in autism[25] and has been reported to be mutated in many patients with autism and other neurodevelopmental disorders.[26] With molecular analysis results, case 13 was referred to the child psychiatry department to be evaluated for autism. Case 14, who had intellectual disability, hearing loss, dystopia canthorum, and motor retardation, was diagnosed with Waardenburg syndrome with a novel likely pathogenic *SOX10* gene variant. Novel variants on the *SOX10* gene were reported with clinical heterogeneity[27] however, clinical findings may not be typical.

Ten previously reported variants were also detected in our study. An *MBOAT7* homozygous pathogenic frameshift variant was detected in case 1, with intellectual disability, epilepsy, and speech delay. An *MBOAT7* frameshift variant[28] in-frame deletion variants[29], and loss of function variants[30] have been reported with intellectual disability, seizures, and autistic features previously. Case 3 in the current study had a *TUBB3* pathogenic variant with intellectual disability, epilepsy, and dysmorphic features. *TUBB3* is related to the malformation of cortical development[31,32], neuronal migration disorders[33], and infantile nystagmus.[34] In our study, case 3 had dysmorphic features (microcephaly, short philtrum, micrognathia, arachnodactyly, pectus excavatum, and broad toe) different from those described in the literature. A *MAN1B1* homozygous pathogenic variant was detected in case 4, with isolated intellectual disability. The *MAN1B1* gene is related to congenital disorders of glycosylation, which present with intellectual disability and are thought to be caused by deficient glycosylation of proteins and lipids.[35,36] Case 4 showed a nonsense mutation, (NM_016219.5:c.1204C>T p.[Gln402Ter]), in the current study; while a case had a homozygous nonsense mutation, (NM_016219.3: c.1418G>A [p.Trp473*]), segregated with intellectual disability and additional dysmorphic features in a study.[37] Case 5, who had an intellectual disability, developmental delay, and macrocephaly, was diagnosed with Alexander's disease with a *GFAP* gene mutation. A study evaluating 135 patients diagnosed with Alexander's disease reported that the frequent findings were seizure, macrocephaly, developmental delay, and spasticity.[38] A *CACNA1A* pathogenic variant was detected in case 6, who had an intellectual disability, epilepsy, and dysmorphic features. A study investigating the etiology of undiagnosed neurodevelopmental disorders reported that *CACNA1A* is a promising gene for neurodevelopmental disorders or epileptic encephalopathy.[39] An *LMNA* variant was detected in case 7, with intellectual disability, motor retardation, and blue sclera in our study. A homozygous *LMNA* variant was reported in a family with lipodystrophy.[40] A *de novo* 1q22q23.1 interstitial microdeletion including the *LMNA* gene was reported in a case with short stature, microcephaly, hypoplastic corpus callosum, cleft palate, minor facial anomalies, congenital heart defect, camptodactyly of the fourth to fifth fingers, and intellectual disability.[41] An *LBR* gene *de novo* pathogenic variant was detected in case 8, who had an intellectual disability, atypical autism, and mild dysmorphic features in the current study. *LBR* is associated with Pelger-Huet anomaly, Greenberg dysplasia, and Reynolds syndrome.[42]

To our knowledge, no study has been reported on the relationship between the *LBR* gene and autism. Skeletal dysplasia and dysmorphic features have been reported with the *LBR* gene in two cases reporting a different phenotypic spectrum of LBR-associated disorders.[43] *ALS2* and *ENPP1* heterozygous variants were detected in case 9, with intellectual disability, motor retardation, and cerebral atrophy. The *ALS2* gene is recessively inherited, and both *ALS2* and *ENPP1* gene phenotypes were not related to the phenotype of case 9. *ALS2* gene mutations have been reported for juvenile-onset amyotrophic lateral sclerosis and related motor neuron diseases.[44] Due to case 9 being sixteen years old and having cerebral atrophy, *ALS2* may be reported as a candidate gene for cerebral atrophy. An *HSPG2* homozygous VUS was detected in case 11, with intellectual disability and motor retardation in the current study. A case with profound intellectual disability, infantile-onset seizures, chronic respiratory failure, facial dysmorphisms, skeletal abnormalities, and an atrial septal defect was reported with maternally inherited uniparental disomy of chromosome 16 including the *WWOX* gene explains the seizures and intellectual disability.[45] Another pathogenic variant in the *HSPG2* gene was reported as being responsible or the patient's skeletal abnormalities including 1p36 deletion causing developmental delay, intellectual disability, and seizures, which includes the *HSPG2* gene in the region responsible for skeletal dysplasia and congenital heart defects.[46]

The etiology of intellectual disability is heterogeneous and variable. With the development of new diagnostic methods, our knowledge of this subject increases day by day. In a significant proportion of intellectual disability cases , the etiology is unknown. We aimed to focus on these idiopathic intellectual disability cases in this study and to highlight that multiple gene analysis is beneficial to determine the etiology of intellectual disability.

In conclusion, in the current study, 14 cases of 25 (56%) were defined with a variant related to intellectual disability. Seven of fourteen (50%) cases had a definite diagnosis of intellectual disability with the pathogenic variants in *MBOAT7, KDM5C, TUBB3, MAN1B1, GFAP, CACNA1A,* and *UBE3A* genes. Seven novel variants were determined in the *KDM5C, BCOR, UBE3A, TRAPPC9, AFF2, NLGN4,* and *SOX10* genes. With a 56% variant detection rate, our study showed that multiple gene analysis for intellectual disability is crucial.

The limitations of this study are: the segregation analysis of all participants could not be applied due to the study was retrospectively planned. The functional studies were not performed on novel variants, they are classified according to the bioinformatical tools.

ACKNOWLEDGEMENTS

The authors would like to thank all the patients and family members who participated in this study.

DISCLOSURE

Data availability: The data supporting this study's findings are available from the corresponding author upon reasonable request.

Financial support: None.

Conflict of interest: None

REFERENCES

1. Lee K, Cascella M, Marwaha R. Intellectual disability. StatPearls 2022; Treasure Island (FL). Napoli, Italy: Bookshelf ID: NBK547654.
2. AlMutiri R, Malta M, Shevell MI, Srour M. Evaluation of individuals with non-syndromic global developmental delay and intellectual disability. *Children (Basel)* 2023; 10(3):414. doi:10.3390/children10030414.
3. Ko MH, Chen HJ. Genome-wide sequencing modalities for children with unexplained global developmental delay and intellectual disabilities-A narrative review. *Children (Basel)* 2023; 10(3):501. doi: 10.3390/children10030501.
4. Ford TJL, Jeon BT, Lee H, Kim WY. Dendritic spine and synapse pathology in chromatin modifier-associated autism spectrum disorders and intellectual disability. *Front Mol Neurosci* 2023; 15:1048713. doi: 10.3389/fnmol.2022.1048713.
5. Fell CW, Nagy V. Cellular models and high-throughput screening for genetic causality of intellectual disability. *Trends Mol Med* 2021; 27:220-30. doi: 10.1016/j.molmed.2020.12.003.
6. Batool S, Raza H, Zaidi J, Riaz S, Hasan S, Syed NI. Synapse formation: from cellular and molecular mechanisms to neurodevelopmental and neurodegenerative disorders. *J Neurophysiol* 2019; 121(4):1381-97. doi: 10.1152/jn.00833.2018.
7. Dobrigna M, Poëa-Guyon S, Rousseau V, Vincent A, Toutain A, Barnier JV. The molecular basis of p21-activated kinase-associated neurodevelopmental disorders: From genotype to phenotype. *Front Neurosci* 2023; 17:1123784. doi: 10.3389/fnins.2023.1123784.
8. Ishihara K. Genes associated with disturbed cerebral neurogenesis in the embryonic brain of mouse models of Down syndrome. *Genes (Basel)* 2021; 12(10):1598. doi: 10.3390/genes12101598.

9. Strnadova I, Nevin SM, Scully JL, Palmer EE. The opinions and experiences of people with intellectual disability regarding genetic testing and genetic medicine: A systematic review. *Genet Med* 2022;24(3):535-48. doi: 10.1016/j.gim.2021.11.013.
10. Rustom H, Hassan Eltorki Y, Adil Shah Khoodoruth M, *et al*. Genetic etiology of adult intellectual disability (ID) of unknown cause in Qatar: a retrospective study. *Qatar Med J* 2022; 2022(1):26. doi: 10.5339/qmj.2022.26.
11. Richards S, Aziz N, Bale S, *et al*. Standards and guidelines for the interpretation of sequence variants: a joint consensus recommendation of the American College of Medical Genetics and Genomics and the Association for Molecular Pathology. *Genet Med* 2015; 17: 405-24. doi: 10.1038/gim.2015.30.
12. Den Dunnen JT, Dalgleish R, Maglott DR, *et al*. HGVS recommendations for the description of sequence variants: 2016 update. *Hum Mutat* 2016; 37: 564-9. doi: 10.1002/humu.22981.
13. Landrum MJ, Lee JM, Benson M, *et al*. ClinVar: improving access to variant interpretations and supporting evidence. *Nucleic Acids Res* 2018; 46: D1062-D1067. doi: 10.1093/nar/gkx1153.
14. Maia N, Nabais Sa MJ, Melo-Pires M, De Brouwer APM, Jorge P. Intellectual disability genomics: current state, pitfalls and future challenges. *BMC Genomics* 2021; 22:909. doi: 10.1186/s12864-021-08227-4.
15. Abe-Hatano C, Iida A, Kosugi S, *et al.* Whole genome sequencing of 45 Japanese patients with intellectual disability. *Am J Med Genet A* 2021; 185:1468-80. doi: 10.1002/ajmg.a.62138.
16. Yan H, Shi Z, Wu Y, *et al*. Targeted next generation sequencing in 112 Chinese patients with intellectual disability/developmental delay: novel mutations and candidate gene. *BMC Med Genet* 2019; 20:80. doi: 10.1186/s12881-019-0794-y.
17. Taskiran EZ, Karaosmanoglu B, Kosukcu C, *et al*. Diagnostic yield of whole-exome sequencing in non-syndromic intellectual disability. *J Intellect Disabil Res* 2021; 65:577-88. doi: 10.1111/jir.12835.
18. Kawano-Matsuda F, Maeda T, Kaname T, Yanagi K, Ihara K. X-linked mental retardation and severe short stature with a novel mutation of the KDM5C gene. *Clin Pediatr Endocrinol* 2021; 30:61-4. doi: 10.1297/cpe.30.61.
19. Carmignac V, Nambot S, Lehalle D, *et al*. Further delineation of the female phenotype with KDM5C disease-causing variants: 19 new individuals and review of the literature. *Clin Genet* 2020; 98:43-55. doi: 10.1111/cge.13755.
20. Rechsteiner D, Issler L, Koller S, *et al*. Genetic analysis in a Swiss cohort of bilateral congenital cataract. *JAMA Ophthalmol* 2021; 139:691-700. doi: 10.1001/jamaophthalmol.2021.0385.
21. Eckardt JN, Stasik S, Kramer M, *et al*. Loss-of-function mutations of BCOR are an independent marker of adverse outcomes in intensively treated patients with acute myeloid leukemia. *Cancers (Basel)* 2021; 13(9):2095. doi: 10.3390/cancers13092095.
22. Cinnirella G, Taylor RL, Coco C, *et al*. Craniosynostosis-microphthalmia syndrome belongs to the spectrum of BCOR-related disorders. *Clin Genet* 2020; 98:413-5. doi: 10.1111/cge.13808.
23. Geerts-Haages A, Bossuyt SNV, Den Besten I, *et al*. A novel UBE3A sequence variant identified in eight related individuals with neurodevelopmental delay, results in a phenotype which does not match the clinical criteria of Angelman syndrome. *Mol Genet Genomic Med* 2020; 8: e1481. doi: 10.1002/mgg3.1481.
24. Katoh K, Aiba K, Fukushi D, *et al*. Clinical and molecular genetic characterization of two female patients harboring the Xq27.3q28 deletion with different ratios of X chromosome inactivation. *Hum Mutat* 2020; 41: 1447-60. doi: 10.1002/humu.24058.
25. Talebizadeh Z, Lam DY, Theodoro MF, Bittel DC, Lushington GH, Butler MG. Novel splice isoforms for NLGN3 and NLGN4 with possible implications in autism. *J Med Genet* 2006; 43:e21. doi: 10.1136/jmg.2005.036897.
26. Cast TP, Boesch DJ, Smyth K, Shaw AE, Ghebrial M, Chanda S. An autism associated mutation impairs neuroligin-4 glycosylation and enhances excitatory synaptic transmission in human neurons. *J Neurosci* 2021; 41:392-407. doi: 10.1523/JNEUROSCI.0404-20.2020.
27. Moldenaes MF, Rendtorff ND, Hindbaek LS, Torring PM, Nilssen O, Tranebjaerg L. Clinical manifestations and novel pathogenic variants in SOX10 in eight Danish probands with Waardenburg syndrome. *Eur J Med Genet* 2021; 64:104265. doi: 10.1016/j.ejmg.2021.104265.
28. Khan S, Rawlins LE, Harlalka GV, *et al.* Homozygous variants in the HEXB and MBOAT7 genes underlie neurological diseases in consanguineous families. *BMC Med Genet* 2019; 20:199. doi: 10.1186/s12881-019-0907-7.
29. Sun L, Khan A, Zhang H, *et al*. Phenotypic characterization of intellectual disability caused by MBOAT7 mutation in two consanguineous Pakistani families. *Front Pediatr* 2020; 8:585053. doi: 10.3389/fped.2020.585053.
30. Heidari E, Caddeo A, Zarabadi K, *et al.* Identification of novel loss of function variants in MBOAT7 resulting in intellectual disability. *Genomics* 2020; 112:4072-7. doi 10.1016/j.ygeno.2020.07.008.
31. Accogli A, Severino M, Riva A, *et al*. Targeted re-sequencing in malformations of cortical development: genotype-phenotype correlations. *Seizure* 2020; 80:145-52. doi: 10.1016/j.seizure.2020.05.023.
32. Lee S, Kim SH, Kim B, *et al*. Clinical implementation of targeted gene sequencing for malformation of cortical development. *Pediatr Neurol* 2020; 103:27-34. doi: 10.1016/j.pediatrneurol.2019.07.010.
33. Liu JS. Molecular genetics of neuronal migration disorders. *Curr Neurol Neurosci Rep* 2011; 11:171-8. doi: 10.1007/s11910-010-0176-5.
34. Jin S, Park SE, Won D, Lee ST, Han SH, Han J. TUBB3 M323V syndrome presents with infantile nystagmus. *Genes (Basel)* 2021; 12(4):575. doi: 10.3390/genes12040575.
35. Sakhi S, Cholet S, Wehbi S, *et al*. MAN1B1-CDG: Three new individuals and associated biochemical profiles. *Mol Genet Metab Rep* 2021; 28:100775. doi: 10.1016/j.ymgmr.2021.100775.

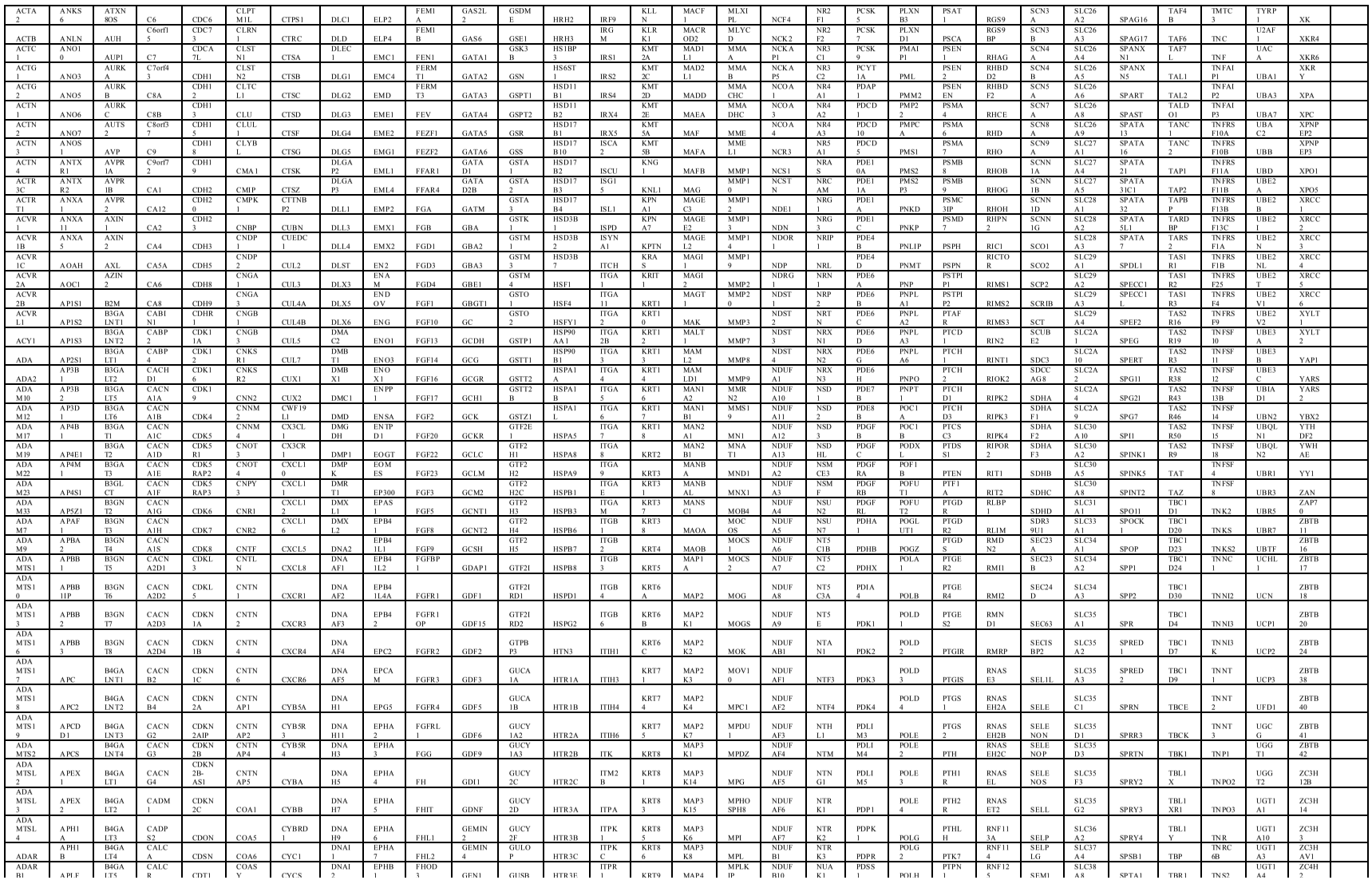
36. Van Scherpenzeel M, Timal S, Rymen D, *et al*. Diagnostic serum glycosylation profile in patients with intellectual disability as a result of MAN1B1 deficiency. *Brain* 2014; 137:1030-8. doi: 10.1093/brain/awu019.
37. Rafiq MA, Kuss AW, Puettmann L, *et al*. Mutations in the alpha 1,2-mannosidase gene, MAN1B1, cause autosomal-recessive intellectual disability. *Am J Hum Genet* 2011; 89:176-82. doi: 10.1016/j.ajhg.2011.06.006.
38. Heshmatzad K, Haghi Panah M, Tavasoli AR, Ashrafi MR, Mahdieh N, Rabbani B. GFAP variants leading to infantile Alexander disease: Phenotype and genotype analysis of 135 cases and report of a de novo variant. *Clin Neurol Neurosurg* 2021; 207:106754. doi: 10.1016/j.clineuro.2021.106754.
39. Yamamoto T, Imaizumi T, Yamamoto-Shimojima K, *et al*. Genomic backgrounds of Japanese patients with undiagnosed neurodevelopmental disorders. *Brain Dev* 2019; 41:776-82. doi: 10.1016/j.braindev.2019.05.007.
40. Patni N, Hatab S, Xing C, Zhou Z, Quittner C, Garg A. A novel autosomal recessive lipodystrophy syndrome due to homozygous LMNA variant. *J Med Genet* 2020; 57:422-6. doi: 10.1136/jmedgenet-2019-106395.
41. Aleksiuniene B, Preiksaitiene E, Morkuniene A, Ambrozaityte L, Utkus A. A de novo 1q22q23.1 interstitial microdeletion in a girl with intellectual disability and multiple congenital anomalies including congenital heart defect. *Cytogenet Genome Res* 2018; 154:6-11. doi: 10.1159/000486947.
42. Giorgio E, Sirchia F, Bosco M, *et al*. A novel case of Greenberg dysplasia and genotype-phenotype correlation analysis for LBR pathogenic variants: An instructive example of one gene-multiple phenotypes. *Am J Med Genet A* 2019; 179:306-11. doi: 10.1002/ajmg.a.61000.
43. Thompson E, Abdalla E, Superti-Furga A, *et al*. Lamin B receptor-related disorder is associated with a spectrum of skeletal dysplasia phenotypes. *Bone* 2019; 120:354-63. doi: 10.1016/j.bone.2018.11.006.
44. Kim J, Kim S, Nahm M, *et al.* ALS2 regulates endosomal trafficking, postsynaptic development, and neuronal survival. *J Cell Biol* 2021; 220(5): e202007112. doi: 10.1083/jcb.202007112.
45. Davids M, Markello T, Wolfe LA, *et al.* Early infantile-onset epileptic encephalopathy 28 due to a homozygous microdeletion involving the WWOX gene in a region of uniparental disomy. *Hum Mutat* 2019; 40:42-7. doi: 10.1002/humu.23675.
46. Jordan VK, Zaveri HP, Scott DA. 1p36 deletion syndrome: an update. *Appl Clin Genet* 2015; 8:189-200. doi: 10.2147/TACG.S65698.

Supplementary

The genes included in the clinical exome sequencing panel in our study

A2M	AKR7A3	ASB1	BMPR1A	CCHCR1	CHPF	CRADD	DCLRE1C	DYNC1I1	FABP7	FSHR	GNRH1	HIF1A	IL12RB2	KCNV2	LIPT1	MESP1	MYB	NKX3-1	OXA1L	PIGZ	PRAM1	RAP1B	rs37535	SIGLEC16	SMARCE1	STOX1	THBD	TRPC5	VHL	ZNHIT6
A4GALT	AKT1	ASB10	BMPR1B	CCK	CHPF2	CRAT	DCN	DYNC2H1	FADD	FST	GNRHR	HIF1AN	IL13	KCTD1	LITAF	MESP2	MYBBP1A	NKX3-2	OXCT1	PIK3CG	PRAMEF2	RAP1GDS1	rs4411641	SIGMAR1	SMC1A	STRA6	THBS1	TRPC6	VIL1	ZNRF1
A4GNT	AKT2	ASCC1	BMPR2	CCKAR	CHRD	CRB1	DCP1B	DYRK1A	FADS2	FSTL1	GNS	HGD2A	IL15	KCTD13	LLGL1	MEST	MYBL2	NLGN1	OXTR	PIK3C3	PRB1	RAPGEF1	rs6074704	SIK3	SMC1B	STRADA	THBS2	TRPM1	VIM	ZP1
AAAS	AKT3	ASCC3	BMS1	CCKBR	CHRDL1	CRB2	DCPS	DYRK1B	FAH	FSTL4	GOLGA3	HINT1	IL16	KCTD3	LMAN1	MET	MYBPC1	NLGN2	P2RX1	PIK3CA	PRB3	RAPGEF4	rs6512586	SIL1	SMC3	STRC	THBS4	TRPM2	VIP	ZPBP
AADAC	ALAD	ASCL1	BNC2	CCL11	CHRFAM7A	CRBN	DCTD	DYSF	FAM111A	FSTL5	GOLGA5	HIP1	IL17A	KCTD7	LMAN2L	METTL14	MYBPC3	NLGN3	P2RX2	PIK3CB	PRB4	RAPSN	rs6666954	SIM1	SMC5	STS	THOC2	TRPM3	VIPAS39	ZPBP2
AADACL2	ALAS2	ASH1L	BOC	CCL13	CHRM1	CREB1	DCTN1	E2F1	FAM111B	FTCD	GON4L	HIST1H2AE	IL17F	KDM1A	LMBR1	METTL23	MYC	NLGN4X	P2RX4	PIK3CD	PRCC	RARA	rs6841061	SIM2	SMC6	STT3A	THOC6	TRPM4	VIPR2	ZPLD1
AAGAB	ALB	ASIC3	BOLA1	CCL17	CHRM2	CREB3	DCX	E2F4	FAM120A	FTH1	GOPC	HIST1H4B	IL17RA	KDM3A	LMBR1	MFAP4	MYCBP2	NLGN4Y	P2RX5	PIK3CG	PRCD	RARB	rs8045964	SIPA1	SMCHD1	STT3B	THPO	TRPM6	VKORC1	ZSCAN29
AANAT	ALCAM	ASIP	BOLA2	CCL2	CHRM3	CREB3L1	DCXR	E2F5	FAM120AOS	FTHL17	GORAB	HIST2H4A	IL17RB	KDM4C	LMF1	MFAP5	MYCL	NLRC4	P2RX7	PIK3R1	PRCP	RARS	rs9388856	SIPA1L1	SMG1	STUB1	THRA	TRPM7	VLDLR	ZSWIM6
AARS	ALDH16A1	ASL	BOLA2B	CCL22	CHRNA1	CREB3L3	DDAH1	EARS2	FAM120B	FTL	GOSR2	HIST3H3	IL17RD	KDM5A	LMNA	MFF	MYCN	NLRP1	P2RY1	PIK3R2	PRDM1	RARS2	RSC1A1	SIRT1	SMG6	STX11	THRB	TRPS1	VMA21	ZXDA
AARS2	ALDH18A1	ASMT	BOLA3	CCL26	CHRNA2	CREBBP	DDAH2	EBAG9	FAM126A	FTO	GOT1	HIVEP2	IL17REL	KDM5B	LMNB1	MFGE8	MYCT1	NLRP10	P2RY11	PIK3R4	PRDM16	RASA1	RSPH1	SIRT3	SMIM1	STX16	THSD7A	TRPV1	VNN1	
AASS	ALDH1A1	ASMTL	BPGM	CCL3	CHRNA3	CRELD1	DDB1	EBF4	FAM136A	FTSJ1	GP1BA	HK1	IL18	KDM5C	LMNB2	MFHAS1	MYD88	NLRP11	P2RY12	PIK3R5	PRDM2	RASA2	RSPH4A	SIRT5	SMIM3	STX1A	THSD7B	TRPV3	VPRE B1	
ABAT	ALDH1A2	ASNS	BPI	CCL3L1	CHRNA4	CRELD2	DDB2	EBP	FAM161A	FUBP1	GP1BB	HK2	IL18R1	KDM5D	LMO1	MFN2	MYF5	NLRP12	P2RY4	PIKFYVE	PRDM5	RASGEF1B	RSPH9	SIX1	SMN1	STX1B	TIA1	TRPV4	VPS13A	
ABCA1	ALDH1A3	ASPA	BPIFA1	CCL4L1	CHRNA5	CRH	DDC	ECE1	ABRAXAS2	FUCA1	GP2	HLA-A	IL18RAP	KDM6A	LMO2	MFRP	MYF6	NLRP13	P3H2	PIM1	PRDM9	RASGRP1	RSPO1	SIX2	SMN2	STXBP1	TICAM1	TRPV5	VPS13B	
ABCA10	ALDH1B1	ASPH	BPIFA3	CCL5	CHRNA7	CRHR1	DDHD1	ECE2	FAM177A1	FURIN	GP6	HLA-B	IL19	KDM6B	LMO4	MFSD2A	MYH1	NLRP14	PABPC4L	PIMREG	PRDX4	RASGRP2	RSPO4	SIX3	SMNDC1	STXBP2	TIMELESS	TRRAP	VPS13B	
ABCA12	ALDH2	ASPM	BPIFB6	CCL7	CHRNA9	CRIM1	DDHD2	ECEL1	FAM189B	FUS	GP9	HLA-C	IL1A	KDR	LMOD3	MFSD4B	MYH13	NLRP2	PABPN1	PIN1	PREPL	RASSF1	RSRC1	SIX5	SMO	STXBP3	TIMM21	TSC1	VPS34	
ABCA13	ALDH3A2	ASPN	BPY2	CCM2	CHRNB1	CRIPT	DDIT3	ECHS1	FAM205A	FUT1	GPAM	HLA-DMB	IL1B	KDSR	LMTK3	MFSD8	MYH14	NLRP3	PACRG	PINK1	PRF1	RASSF5	RTEL1	SIX6	SMOC1	STXBP5	TIMM44	TSC2	VPS37A	
ABCA2	ALDH4A1	ASPRV1	BRAF	CCNA2	CHRNB2	CRISP2	DDOST	ECI1	FAM208A	FUT2	GPANK1	HLA-DOA	IL1R1	KEAP1	LMX1B	MGAT1	MYH15	NLRP4	PACS1	PIP4K2A	PRG4	RAX	RTF1	SKI	SMOC2	STXBP5L	TIMM8A	TSEN15	VPS44	
ABCA3	ALDH5A1	ASPSCR1	BRAP	CCND1	CHRNB3	CRK	DDR1	ECM1	FAM220C	FUT3	GPAT2	HLA-DPB1	IL1RAPL1	KEL	LNX2	MGAT2	MYH2	NLRP5	PADI4	PIP5K1B	PRICKLE1	RAX2	RTL4	SKIV2L	SMOX	STYX	TIMP1	TSEN2	VPS53	
ABCA4	ALDH6A1	ASS1	BRAT1	CCND2	CHRNB4	CRKL	DDR2	ECM2	FAM3C	FUT6	GPBAR1	HLA-DPB2	IL1RL1	KER	loc344967	MGAT3	MYH3	NLRP6	PADI6	PIP5K1C	PRICKLE2	RB1	RTN2	SLA2	SMPD1	SUCLA2	TIMP2	TSEN34	VPS54	
ABCA5	ALDH7A1	ASTN2	BRCA1	CCND3	CHRND	CRLF1	DDRGK1	ECSIT	FAM46C	FUT7	GPC3	HLA-DQA1	IL1RN	KHDC3L	LONP1	MGAT4A	MYH4	NLRP7	PAFAH1B1	PIR	PRIMPOL	RB1CC1	RTN3	SLBP	SMPD3	SUCLG1	TIMP3	TSEN54	VRK1	
ABCA7	ALDOA	ASXL1	BRCA2	CCNE1	CHRNE	CRMP1	DDX10	EDA	FAM47B	FUT8	GPC4	HLA-DQB1	IL2	KHK	LOR	MGAT5B	MYH6	NLRP8	PAFAH1B3	PITPNA	PRKAA2	RBBP8	RTN4R	SLC10A1	SMPX	SUCLG2	TINAG	TSFM	VSIG1	
ABCB1	ALDOB	ASXL3	BRCC3	CCNH	CHRNG	CROCC	DDX11	EDA2R	FAM71A	FUZ	GPC5	HLA-DRA	IL20RA	KIAA0100	LOX	MGAT4C	MYH7	NLRP9	PAH	PITPNM3	PRKACA	RBCK1	RTN4RL1	SLC10A2	SMS	SUCO	TINF2	TSG101	VSIG4	
ABCB11	ALG1	ATAD3A	BRD1	CCNO	CHST14	CRP	DDX20	EDAR	FAM81B	FXN	GPC6	HLA-DRB1	IL20RB	KIAA0232	LOXHD1	MGAT5	MYH7B	NLRX1	PAK3	PITX1	PRKCG	RBFA	RTTN	SLC11A1	SMUG1	SUFU	TIPARP	TSHB	VSX1	
ABCB4	ALG10	ATAD3B	BRD2	CCNQ	CHST3	CRTAP	DDX25	EDARADD	FAM83H	FXYD2	GPD1	HLA-DRB5	IL21	KIAA0319	LOXL1	MGAT5B	MYH8	NMB	PAK5	PITX2	PRKAG2	RBFOX1	RUBCN	SLC11A2	SMYD3	SUGCT	TIPIN	TSHR	VSX2	
ABCB5	ALG10B	ATCAY	BRD4	CCNT2	CHST6	CRTC1	DDX39B	EDN1	FAM8A1	FXYD6	GPD1L	HLA-E	IL21R	KIAA0513	LOXL2	MGEA5	MYH9	NME1	PALB2	PITX3	PRKAG3	RBFOX3	RUNX1	SLC12A1	SNAI2	SULF1	TIRAP	TSHZ1	VTN	
ABCB6	ALG11	ATF1	BRF1	CCPG1	CHST7	CRX	DDX3X	EDN2	FAM91A1	FYB2	GPD2	HLA-G	IL23R	KIAA1109	LPA	MGLL	MYL1	NME5	PALLD	PIWIL3	PRKAR1A	RBL1	RUNX1T1	SLC12A2	SNAP25	SULT1A1	TJP1	TSLP	VWA3B	
ABCB7	ALG12	ATF3	BRIP1	CCR1	CHST8	CRY1	DDX3Y	EDN3	FAM9C	FYCO1	GPHN	HLCS	IL2RA	KIAA1210	LPAR1	MGME1	MYL2	NME7	PAM16	PIWIL4	PRKCA	RBL2	RUNX2	SLC12A3	SNAP29	SULT1A3	TJP2	TSPAN12	VWF	
ABCC1	ALG13	ATF5	BRSK2	CCR2	CHSY1	CRY2	DDX47	EDNRA	FAN1	FZD1	GPI	HLTF	IL2RG	KIAA1217	LPAR6	MGMT	MYL3	NME8	PAMR1	PJVK	PRKCB	RBM10	RUNX3	SLC12A5	SNAPC4	SULT1C2	TK2	TSPAN7	WAC	
ABCC11	ALG14	ATF6	BRWD1	CCR3	CHSY3	CRYAA	DDX5	EDNRB	FANCA	FZD3	GPIHBP1	HLX	IL3	KIAA1549	LPIN1	MGP	MYLPF	NMNAT1	PANK2	PKD1	PRKCD	RBM15	RUVBL1	SLC12A6	SNAPC5	SULT1C3	TLE6	TSPAN7	WAS	
ABCC12	ALG2	ATG16L1	BRWD3	CCR5	CHUK	CRYAB	DDX53	EDRF1	FANCB	FZD4	GPNMB	HMBS	IL31RA	KIAA1586	LPIN2	MGST2	MYLK2	NMT2	PAPD7	PKD1L1	PRKCG	RBM20	RXFP2	SLC12A7	SNCA	SULT1E1	TLK1	TSPEAR	WASF3	
ABCC2	ALG3	ATG7	BSCL2	CCR6	CIAO1	CRYBA1	DDX58	EEF1A2	FANCC	FZD6	GPR1	HMCN1	IL34	KIF11	LPIN3	MGST3	MYLK2	NMU	PAPOLA	PKD2	PRKCH	RBM28	RXRA	SLC13A2	SNCAIP	SULT2A1	TLL1	TSPO	WAS HC4	
ABCC3	ALG5	ATIC	BSG	CCR7	CIB2	CRYBA2	DDX59	EEF1B2	FANCD2	FZD9	GPR101	HMG20B	IL36RN	KIF14	LPL	MIA3	MYO15A	NNMT	PAPSS2	PKHD1	PRKCSH	RBM43	RXRG	SLC13A5	SNCB	SULT2B1	TLR1	TSPYL1	WAS HC5	
ABCC4	ALG6	ATL1	BSND	CCRL2	CIC	CRYBA4	DEAF1	EEF2E	FANCE	FZR1	GPR12	HMGA1	IL4	KIF17	LPP	MIAT	MYO1 8B	NNT	PARD3B	PKHD IL1	PRKD1	RBM8A	RYK	SLC14A1	SNIP1	SULT4A1	TLR10	TSR2	WDFY3	
ABCC6	ALG8	ATL3	BST1	CCSER1	CIDEA	CRYBB1	DEC1	EEF2K	FANCF	G6PC	GPR132	HMGA2	IL4R	KIF18A	LRAT	MIB1	MYO1A	NOBOX	PARD6A	PKLR	PRKD3	RBMXL2	RYR1	SLC14A2	SNORD115-1	SUMF1	TLR2	TSSC4	WDFY4	
ABCC8	ALG9	ATM	BTAF1	CCT5	CIDEC	CRYBB2	DECR1	EFCAB13	FANCG	G6PC2	GPR137B	HMGB1	IL5	KIF1A	LRBA	MICAL1	MYO1C	NOD1	PARD6G	PKM	PRKDC	RBMY1AJ	RYR2	SLC15A1	SNORD116-1	SUMO1	TLR3	TSSK2	WDPCP	
ABCC9	ALK	ATN1	BTBD9	CCT7	CIITA	CRYBB3	DEF6	EFCAB5	FANCI	G6PC3	GPR139	HMGB3	IL6	KIF1B	LRCH1	MICAL3	MYO1D	NOD2	PARK7	PKN3	PRKG1	RBP3	RYR3	SLC16A1	SNORD116-10	SUMO4	TLR4	TSSK3	WDR11	
ABCD1	ALKBH2	ATOH7	BTC	CD109	CILP	CRYGA	DEFB1	EFEMP1	FANCL	G6PD	GPR143	HMGCL	IL6R	KIF1BP	LRFN5	MICALCL	MYO1E	NODAL	PARL	PKNOX1	PRKG2	RBP4	S100A14	SLC16A12	SNORD50A	SUN1	TLR5	TSSK4	WDR13	
ABCD3	ALKBH3	ATP10A	BTD	CD14	CISD2	CRYGB	DEFB124	EFEMP2	FANCM	GAA	GPR152	HMGCR	IL6ST	KIF1C	LRGUK	MICB	MYO1F	NOG	PARN	PKP1	PRKN	RBPJ	S100B	SLC16A2	SNRK	SUN2	TLR6	TST	WDR19	
ABCD4	ALMS1	ATP10D	BTK	CD151	CISH	CRYGC	DEFB126	EFHC1	FAR1	GAB2	GPR179	HMGCS2	IL7	KIF21A	LRIG2	MICU1	MYO3A	NOL3	PARP1	PKP2	PRKRA	RBX1	S1PR1	SLC16A3	SNRNP200	SUOX	TLR7	TTBK2	WDR27	
ABCG1	ALOX12	ATP11B	BTLA	CD177	CITED2	CRYGD	DEFB4A	EFHC2	FARS2	GABARAPL1	GPR33	HMMR	IL7R	KIF22	LRIT3	MID1	MYO5A	NOP10	PARP2	PLA2G10	PRL	RC3H1	S1PR3	SLC16A7	SNRPB	SUPT16H	TLR8	TTC19	WDR31	
ABCG2	ALOX12B	ATP13A2	BTN1A1	CD19	CIZ1	CRYGE	DEK	EFNA5	FAS	GABBR1	GPR37	HMOX1	IL9	KIF23	LRMDA	MID2	MYO5B	NOP56	PARP3	PLA2G2A	PRLH	RCAN1	SAA1	SLC17A1	SNRPE	SUPT3H	TLR9	TTC21B	WDR34	
ABCG5	ALOX15	ATP13A4	BTN2A1	CD1A	CKAP2L	CRYGS	DENND1A	EFNB1	FASLG	GABBR2	GPR55	HMOX2	IL9R	KIF27	LRP1	MIF	MYO5C	NOS1	PARP4	PLA2G2D	PRLHR	RCBTB1	SAA2	SLC17A3	SNRPN	SUPT5H	TLX1	TTC7	WDR35	

ABCG 5	ALOX 5AP	ATP1 A1	BTNL 2	CD1E	CKM	CRYL1	DENN D5A	EFR3	FASN	GABR A2	GPR68	HMSD	ILDR 1	KIF2	LRP1B	MIP	MYO6	NOS 2	PARS 2	PLA2 G4A	PRLR	RCHY 1	SACS	SNTA1	SURF	TLX2	TTC5	WDR 5
ABHD 1	ALOX 5AP	ATP1 A1	BTRC	CD200	CLCA 2	CRYM	DENR	DFTU	FASTK	GABR A2	GPRC 5A	HMX1	ILK	KIF3 C	LRP2	MINP	MYO7 A	NOS 2	PASK	PLA2 G4C	PRM1	RD3	SAE1	SNTB1	SUZI 2	TLX3	TTC7	WDR 4
ABHD 12	ALOX E3	ATP1 A2	BUB1	CD207	CLCA 1	CRYZL 1	DIPD C5	EGF	FAT1	GABR A5	GPRIN 3	HMX2	ILVBL	KIF4 A	LRP4	MINP P1	MYO7 B	NOS 3	PAW R	PLA2 G5	PRM2	RDH1	SAG	SNTG1	SV2B	TM4SF 19	TTC8	WDR 45
ABHD 16A	ALPL	ATP1 B	BUB1	CD209	CLCA 2	CSAG1	DES	EGFR	FAT2	GABR A6	GPSM	HNF1A	IMPA 1	KIF5 A	LRP5	MIP	MYO9 B	NOT 1	PAX1	PLA2 G7	PRM3	RDH1 2	SAGE	SNTG2	SVIL 20	TM4SF 19	TTF2	WDR 48
ABL1	ALPP	BUB3	CD22	CLCF1	CSDE1	DESI2	EGLN 1	FAT3	HNF1B	GABR B3	GPX1	IMPA 2	IQCA1	KIF5 B	LRP6	MIPOL1	MYOC	NOT CH2	PAX2	PLA2 G7	PRM4	RDH5	SALL 1	SNTG1	SYCE 2	TMC1	TTI2	WDR 48
ABL2	ALS2	BVES	CD226	CLCN 1	CSF1	DGAT 1	EGR2	FAT4	HNF4A	GABR D	GPT	IMPA D1	IQCB1	KIF5 C	LRP8	MIR10 6B	MYOC D	NOT CH3	PAX3	PLAG 1	PRMT 7	RDH8	SALL 2	SNX1	SYCP 3	TMC3	TTLL 1	WDR 5
ABO	ALX1	ATP2 A1	C1orf 40	CD24	CLCN 2	CSF1R	EGR3 1	FBLIM 1	GABR G1	GPT2	HMMT PA1	IMPD H2	KIF6	LRPPR C	MIR12 4	MYO 1	NOX 3	PAX4	PLAG L1	PRMT 9	RDM1	SALL 4	SNX10	SYK	TMC6	TTLL 11	WDR 60
ABRA XAS1	ALX3	ATP2 A3	C1orf 43	CD244	CLCN 3	CSF2	DGCR 2	EHBP 1	FBLN1	GABR G2	GPX1	HNRN PA2B1	IMPG 1	KIF7	LRR1	MIR12 5A	MYOF 3	PAX5	PLAT	PRND	RDX	SAMD 4A	SNX14	SYN1	TMC8 5	TMED 7	TUBA 1A	WDR 62
ACAC A	ALX4	ATP2 B2	C1orf 57	CD247	CLCN 4	CSF2R A	DGCR 5	EHD2	FBLN2	GABR G3	GPX4	HNRN PA2B1	IMPG 2	KIF9	LRRC 29	MIR12 6	MYOT 0	PAX6	PLAU M1	PRNP 1	RECQ L4	SAMD 9	SNX19	SYN2 1	TTN 1	TMEM 114	TTPA	WDR 72
ACAC A	ALX4	ATP2 B2	C1orf 57	CD27	CLCN 5	CSF2RB 6	DGCR 6	EHD4	FBLN5	GABR R2	GRB1 0	GREM 1	HNRN PDL	IKBIP 2	LRRC 4	MIR14 6	MYOT 0	PAX7	PLAU R	PRO1 0	RECQ L4	SAMD D1	SNX3	SYN3	SYNC 1	TMEM 114	TTPA	WDR 72
ACAD 10	AMBA 1	ATP2 B4	C1orf 66	CD320	CLCN 7	CSF3R NACT1	DGKA D	EHM 2	FBN1	GAD1	GRB10 1	GRHL 1	HNRN PU	INF2	LRRC 6	MIR16 -1	MYPN	PAX8	PLCB 2	PROD H1	REEP1	SART 1	SLC1A 3	SNX5	SYNE 1	TMEM 126A	TTR	WDR 81
ACAD 11	AMD1	ATP2 C1	C1orf 2	CD36	CLCN KA	CSH1	DGKE	EI2FA K3	FBN3	GADD 45A	GRHL 2	HOGA 1	INHA	LRRC DL1	LRRC 69	MIR17 HG	MYT1	PAX1	PLCB	PROK 1	REEP2	SABS	SLC22 A1	SOAT1	SYNE 2	TMEM 127	TUB	WDR 93
ACAD 9	AMEL Y	ATP5 B	C1orf 62	CD38	CLDN 1	CSMD1	DHCR 24	EI2FB 1	FBP1	GADD 45B	GRIA1	HOME Z	INHB	KIF3 A	LRRC 8A	MIR17 HG	MYT1	NFC	PBRM 1	PLCD 4	PROK R1	REL	SART 1	SLC22 A12	SOBP	SYNG AP1	TMEM 138	TUBA 4A	WFS1
ACAD 1	AMER 1	ATP5 C1	C1orf 58	CD3D	CLDN 14	CSNK1 A1L	DHCR 7	EI2FB 1	FBXL4	GAGE8	GRIA1	HOXA 1	INMT	KIF9 EL3	LRRD1 2	MIR18 0	NAA1 0	NFC 1L1	PBX1	PLCD 4	PROK R1	REN 1	SART 3	SLC22 A12	SOCS1	SYNG AP1	TMEM 138	TUBA 4A	WIPF 1
ACAD 1	AMH	ATP5 C1	C1orf 58	CD3E	CLDN 16	CSNK1 A1L	DHDH 7	EI2FB 2	FBXL6	GAK	GRIA2	HOXA 1	INPP 0	KISS 1R	LRRK 1	MIR19 4	NAA1 5	NFIC 1	PBX3	PLCG 1	PROM R2	REN	SASH 1	SLC22 A14	SOCS3	SYNJ 1	TMEM 165	TUBB 1	WISP 1
ACAD 2	AMH	ATP5 G1	C1orf 59	CD3G 19	CLDN 19	CSNK1 E	DHDH 1	EI2FB 1	FBXO1 1	GALE	GRIA2	HOXA 11	INPP 5B	KIT	LRRK M1	MIR19 6A2	NAA1 2	NFIH P1	PC 2	PLCG 1	PROP 1	RERE	SASS6	SLC22 A2	SOD2	SYNP M	TMEM 185A	TUBB 1	WNK 1
ACAD 8	AMN	ATP5 G2	C1QA LT1	CD4	CLDN 23	CSNK1 G1	DHH 5	EI2FB 8	FBXO1 8	GALE3 T3	GRID1	HOXA 13	INPP 5D	KITL G	LRSA M1	MIR20 6	NAGA	NFIH P3	PCBD 1	PLCZ 1	PROS 1	REST	SATI	SLC22 A25	SOD3	SYNP O	TMEM 187	TUBB 2A	WNK 3
ACAD VL	AMPD 3	ATP5 G3	C1QA LT1C1	CD40	CLDN 6A	CSNK2 A2	DHO 5	EI2FD 5	FBXO2 T4	GALE3 T4	GRID2	HOXA 3	INPP 5K	KIZ	LRTO MT	MIR24	NAGL 1	NAGP S1	NFIH P4	PLD2	PROX 1	RET	SATB 2	SLC23 A1	SOGA3 R	SYNP R	TMEM 189	TUBB 2B	WNK 4
ACAN 2	AMPD 3	ATP5J	C1orf 27	C1QB	CLEC1 A3	CSNK2 A3	DHRS 4L1	EI2FS 8	FBXO2 4	GALC	GRIK1	HOXA 5	INPP 5J	KL	LTA	MIR27 A	NAGPA	NFIH S1	PCCA	PLD3	PROX 2	RETN 1	SATL 1	SLC22 A4	SOHLH 1	SYP	TMEM 190	TUBB 3	WNT 3
ACAT 1	AMPD 3	ATP5J	C1QA	CD44	CLEC2 D1	CSPP1	DHTK D1	EI2FH 1	FBXO3 1	GALE	GRIK2	HOXA 4	INS	KLB	LTBP1	MIR28 61	NAGS S2	NFIH P1	PCCB	PLEC	PROZ	RETR EG1	SBDS	SLC22 A5	SORBS 1	SYT1	TMEM 216	TUBB 4A	WNT 10A
ACAT 2	AMT	ATP5 O	C1QB	CD46	CLEC3 B	CSRP3	DHX1 6	EIF4A 3	FBXO3 8	GALK1	GRIK3	HOXB 1	INSI G1	KLC1	LTBP2	MIR30 C1	NAIP	NFIL3	PCDH 11X	PLEK HA1	PRPF1 9	REV1	SBF1	SLC22 A6	SORCS 1	SYT1	TMEM 231	TUBG 1	WNT 10B
ACBD 7	ANAP C1	ATP6 C1QC	C1QC	CD5	CLEC4 C3	CSF3	DHX3 6	EIF4E	FBXW 7	GALN T1	GRIK4	HOXB 6	INSI G2	KLF1	LTBP3	MIR34 B	NALC N2	NAMPT	PCDH 15	PLEK HA4	PRPF3 1	RFC1	SBF2	SLC22 A7	SORD	SYT1	TMEM 232	TUBG 1	WNT 3
ACBD 6	ANG	ATP6 V0A1	C1QC	CD55	CLEC4 E	CST6	DHX3 7	EIF4E 1I	FBXW 11	GALN T11	GRIN1	HOXB 6	INSL 3	KLF1 0	LTBP4	MIR49 9A	NAMPT	NIPP A	PCDH 18	PLEK HG5	PRPF3 1	RFC1	SBK3 A1	SLC23 A1	SORL1	SYT2	TMEM 240	TUBG CP5	WNT 3
ACCS	ANGP T1	ATP6 V0A2	C1R	CD58	CLEC4 M	CST9	DHX3 8	EIF4G 1	FBXW 4	GALN T12	GRIN2 A	HOXB 8	INSL 6	KLF1 1	LTBR	MIR51 2	NAPR Si	NIPP B	PCDH 19	PLEK HM1	PRPF3 9	RFC2	SBNO 1	SLC24 A1	SORT1	SYTL 3	TMEM 80B	TUBG CP6	WNT 4
ACE	ANGP T13	ATP6 V0A4	C1S	CD59	CLEC7 A	CSTA	DIAB 2	EIF4H 1	FBXW 7	GALN T13	GRIN3 A	HOXC 8	INSR	KLF5	LTC4S	MIR89 0	NAIP5	NIPP B	PCDH 1	PLG	PRPF4	RFC3	SCSD	SCAM 1	SOS1	SYTL 5	TMEM 81	TULP 1	WNT 5B
ACHE	ANGP TL3	ATP6 V0C	C2	CD72	CLEC1C 1	CSTB	DIAP H1	EIF4H 2	FCAR	GALN T14	GRIN3 A	HOXD 10	INTU	KLF6	LTF	MIB89 0	NAT1	PCDH A1	PLIN1	PRPF6	RFC4	SCAM 1	SLC24 A4	SOS2	SZT2	SLC24	TULP 1	WNT 7A
ACKR 3	ANGP TL4	ATP6 V1B1	C2 1orf 2	CD74	CLEC2	CSTF2	DIAP H2	ELAV E	FCER1 A	GALN T17	GRIP1	HOXD 13	INVS	KLF7	LTK	MIB93 2B	NAT2	PCDH A10	PLIN4	PRPF8	RFC5	SCAP	SLC25 A5	SOS1	T	SLC24 47	TULP 3	WNT 7A
ACLY	ANGP TL6	ATP6 V1C1	C2 1orf 91	CD79 A	CLIP1	CT45A2	DICE 1	ELAV L4	FCER2 T2	GALN T2	GRK3	HP 1	IQCB	KLF8	LUM	MIB96 L	NAT8	NFS1	PCDH 9	PLK1 A3	PRPH	RFT1	SCAR B1	SOX10	TAAR 1	TMEM 67	TULP 3	WNT 9B
ACMS	ANK1	ATP7 B	C2CD3	CD80	CLIP2	CTC1	DIO1	ELF4	FCGR1 A	GALN T3	GRK4	HPCA	IQCE	KLH L10	MIB1	NAV1	NFIT N	PCDH B16	PLN	PRPS1	RFX2	SCAR B2	SLC25 A13	SOX17	TAAR 6	TMEM 70	TUSC 3	WRA P53
ACO2	ANK2	ATP8 A2	C2CD5	CD81	CLK1	CTCF	DIP2	ELK1	FCGR2	GALN T5	GRK5	HPCA L4	IQGA P1	KLH L15	LUZP4	MITF	NAV2	NPY	PCDH 1R	PLOD 2	PRRC	RFX3	SCAR F2	SLC25 A19	SOX18	TAAR 9	TMEM 8A	TWIS T1	WRN
ACOX 1	ANK3	ATP8 B1	C2orf4 2	CD82	CLK2	CTDP1	DIP2 A	ELK3 B	FCGR2 T6	GALN T6	GRM1	HPD	IQGA P3	KLH L3	LY6E	MKKS	NBEA	NPY1R	PCDH GB4	PLOD 2A	PRUNE	RFX5	SCG2	SLC25 A19	SOX2	TAB2	TMEM 9	TP53	WT1
ACP1	ANKH	ATPA F2	C2orf7 1	CD86	CLMP	CTF1	DIP2B	ELM O2	FCGR2 C	GALN T7	GRM3	HPGD	IQGA P3	KLH L40	LY96	MKL1	NBEA 2R	NPY Q	PCGF 2	PLOD 3	PRRT 2	RFX6	SCG3	SLC25 A20	SOX3	TAC3	TMEM 92	TWN K	WT1 AS
ACP2	ANKR D28	ATR 1	C2orf8 0	CD9A	CLN3	CTGF	DIP2C O1D3	ELN A	FCGR3 T8	GALN T9	GRM5	HPRT1	IQSE C2	KLH L41	LY11	MKL2 L2	NBEA L2	NCK1	PLP1	PLS1 1	PRX	RFX8	SCG5	SLC25 A22	SOX4	TACC 3	TMEM 98	TXLN B	WWC 1
ACP5	ANKR D2	ATRI P	C3	CD96	CLN5	CTH 2	DIRC 1	ELN B	FCGR3 T9	GALN T10	GRM6	HPS1	IRAK 1	KLH L42	LYN	NBN 2	NCK2	PLP2	PRKG	PRSS1	RFXA	SCGB 1A1	SLC25 A25	SOX5	TACO 1	TMEM 99	TXN2	WWC 2
ACP6	ANKR D1	ATRN	C3AR1	CD9A	CLN6	CTHRC 1	DISC1 2	ELOA	FCGR3 T	GALN TL5	GRM7	HPS3	IRAK 4	KLH L7	LYRM 4	MKS1	NBPF1	NR0 B1	PCLO 1	PLS3 2	PRX 2	RGL1	SCGB 1D2	SLC25 A3	SOX6	TACR 1	TMIE	TXNL P	WWO X
ACR	ANKR D11	ATRN L1	C3orf3 5	CD98	CLN8	CTLA4	DISC1 2	ELOA 1	FCN1	GALN TL6	GRM8	HPS4	IRF1	KLK3	LYRM 7	M1G1	NCAL D	NR0 B2	PCM1	PLS3	PRSS3	RGL1	SCGB 3A2	SLC25 A38	SOX7	TACR 3	TMLH 4A	TXNL 4A	WWO X
ACSF3	ANKR D28	ATRX 1	C4A	CD9A	CLNK 1	CTNNA 1	DISP1	ELOA 2	FCN2	GALP	GRN	HPS5	IRF1	KLK1	LYST	MLF1	NCAM 1	NR1 H3	PCNA	PLTP	PRSS8	RGM A	SCN1 A	SCN5 A	SOX8	TACR 1	TMPO	TXNR D2	WWT 1
ACSL 4	ANKR D28	ATRX 1	C4B 3A	CD9A	CLOC K	CTNNA 2	DKC1	ELOA 3D	FCN3	GALT	GRPR	HPS6	IRF2	KLK10 2	LYZ	MLH1	NCAN H4	NR1I	PCNT	PLTP	PRSS8	RGM A	SCN1 0A	SCN9 A	SOX9	TACR 2A	TMPR SS11A	TYK2	XBP1
ACSL 5	ANKR D44	ATXN 10	C4BP A	CD2 5C	CLP1	CTNNA 3	DKK1	FCRL3	GAMT	GRXC R1	HPS1 2	IRF4	KLK11	LZTFL1 1	MLH3	NCAN H4	NR1I P	PCNT	PLXN A1	PRTG	RGR	SCN1 1A	SCN5 A45	SP100	TAF1	TMPR SS15	TYM P	XDH
ACSL 2B	ANKR D52	ATXN 3	C4orf1 9	CD2	CLPB	CTNNB 1	DKK2	ELOC	FCRL6	GAN	HR	IRF5	KLK3	LZTR1	MLLT 11	NCAP G2	NR1I P1	PCOL CE	PLXN A3	PRX	RGS2	SCN1 B	SCN1 SB4	SP110	TAF1 C	TMPR SS4	TYR	XIAP
ACSM 3	ANKS 1A	ATXN 3	C5	CD2	CLPP	CTNND 1	DKK3	L4	FDFT1	GAP43	GSC	HRAS	IRF6	KLK4	LZTS1	MLLT 3	NCAP G	NR2 E1	PCP4	PCSK	PLXN A4	PRY	RGS5	SCN1 B	SCN5 A5	SP7	TAF1 L	TMPR SS5	TYRO	XIRP 1
ACTA 1	ANKS 3L	ATXN 7	C5AR2 2BP8	CD2AP	CLPS 2	CTNND 2	DKK4	L5	FDXR	GARS	HRC	IRF7	KLK7	MACC 1	NCF1	MLPH	NCF2	NR2 F1	PCSK 1	PLXN A4	PRY2	RGS6	SCN2 A	SCN5 A10	SP8	SPAG11	TAF2	TMPR SS6	TYRO BP	XIST
ACTA 3	ANKS 7	ATXN 2	C5AR1	CD4	CLPTM 1	CTNS	DLAT	ELP1	FFCH	GAS1	GSDM B	HRG	IRF8	KLK8 B11	MACC 1	MLPH	NCF2	NR2 F2	PCSK 2	PLXN B1	PSAP	RGS7	SCN2 B	SCN5 A10	SPAG11	TAF2	TMPR SS6	TYRO BP	XIST



ADCY1	APLNR	B4GALT6	CALCRL	CDX1	COCH	CYFIP1	DNAJA4	EPHB2	FIG4	GFAP	GYG1	HTR5A	ITPR2	KRTAP1-1	MAP4K5	MPO	NDUFB11	NUB1	PDSS2	POLI	PTPN11	RNF128	SEMA3A	SLC39A12	SPTAN1	TBX1	TNS3	UGT1A5	ZCCHC12	
ADCY10	APOA1	B4GALT7	CALHM1	CDX2	COG1	CYLD	DNAJB2	EPHB4	FIGLA	GFER	GYG2	HTR6	ITPR3	KRTAP4-8	MAP6	MPP3	NDUFB3	NUBPL	PDX1	POLK	PTPN12	RNF135	SEMA3D	SLC39A13	SPTB	TBX10	TNXB	UGT1A6	ZCCHC13	
ADCY3	APOA2	B4GAT1	CALM1	CDY1	COG2	CYP11A1	DNAJB6	EPHB6	FIGN	GFI1	GYPA	HTR7	ITSN1	KRTAP9-3	MAP7D1	MPP4	NDUFB4	NUCB2	PDXK	POLL	PTPN13	RNF139	SEMA3E	SLC39A4	SPTBN1	TBX15	TOMM40	UGT1A7	ZCCHC8	
ADCY5	APOA4	B9D1	CALM2	CDY2A	COG3	CYP11B1	DNAJC13	EPHX1	FIP1L1	GFI1B	GYPB	HTRA1	ITSN2	KYNU	MAP7D3	MPP6	NDUFB6	NUDC	PDYN	POLM	PTPN14	RNF168	SEMA4A	SLC39A5	SPTBN2	TBX18	TOP1	UGT1A8	ZDHHC15	
ADCY6	APOA5	B9D2	CALM3	CDYL	COG4	CYP11B2	DNAJC19	EPHX2	FKBP10	GFM1	GYPC	HTRA2	IVD	L1CAM	MAPK1	MPP7	NDUFB7	NUDT1	PDZD7	POLN	PTPN2	RNF170	SEMA4C	SLC3A1	SPTBN5	TBX19	TOP1MT	UGT1A9	ZDHHC17	
ADCY9	APOB	BAALC	CALR	CEACAM16	COG5	CYP17A1	DNAJC3	EPM2A	FKBP14	GFM2	GYPE	HTT	IYD	L2HGDH	MAPK10	MPST	NDUFB8	NUDT15	PEAR1	POLQ	PTPN21	RNF2	SEMA4D	SLC40A1	SPTLC1	TBX2	TOP2A	UGT2A1	ZDHHC2	
ADCYAP1	APOBEC1	BAAT	CALR3	CEBPA	COG6	CYP19A1	DNAJC30	EPO	FKBP1A	GFPT1	GYS1	HUS1	JAG1	L3MBTL1	MAPK14	MPV17	NDUFB9	NUDT6	PECAM1	POLR1C	PTPN22	RNF20	SEMA4G	SLC41A1	SPTLC2	TBX20	TOP3A	UGT2A3	ZDHHC8	
ADD1	APOBEC3B	BABAM1	CAMK2G	CEBPE	COG7	CYP1A1	DNAJC5	EPOR	FKBP4	GFPT2	GYS2	HUS1B	JAG2	LACTB	MAPK3	MPZ	NDUFC1	NUDT7	PECR	POLR1D	PTPN23	RNF212	SEMA5A	SLC44A2	SQSTM1	TBX21	TOP3B	UGT2B10	ZDHHC9	
ADD2	APOBEC3G	BACE1	CAMK4	CECR2	COG8	CYP1A2	DNAJC6	EPS8	FKBP5	GFRA1	GZMB	HUWE1	JAGN1	LAMA1	MAPK8IP1	MR1	NDUFC2	NUMA1	PEMT	POLR2A	PTPN6	RNF213	SEMA6D	SLC45A2	SRC	TBX22	TOPBP1	UGT2B15	ZEB1	
ADGRA2	APOBEC3H	BACH1	CAMKK1	CEL	COL10A1	CYP1B1	DNAL1	EPX	FKBP6	GFRA2	H19	HVCN1	JAK1	LAMA2	MAPRE2	MRAP	NDUFS1	NUMBL	PENK	POLR2B	PTPRB	RNF216	SEMA7A	SLC46A1	SRCAP	TBX3	TOPORS	UGT2B17	ZEB2	
ADGRA3	APOC1	BAG3	CAMKK2	CELF4	COL11A1	CYP21A2	DNAL4	ERAP1	FKBP8	GGCX	H2AFX	HYAL1	JAK2	LAMA3	MAPT	MRAP2	NDUFS2	NUP107	PEPD	POLR2C	PTPRC	RNF219	SEMG1	SLC47A1	SRD5A2	TBX4	TOR1A	UGT2B28	ZFAT	
ADGRG1	APOC2	BAG6	CAMKMT	CELF6	COL11A2	CYP24A1	DNASE1	ERAP2	FKBPL	GGH	H2BFWT	HYDIN	JAK3	LAMA4	MARCH3	MRC1	NDUFS3	NUP155	PER1	POLR2D	PTPRCAP	RNF32	SELENOF	SLC47A2	SRD5A3	TBX5	TOR1AIP1	UGT2B4	ZFHX3	
ADGRG4	APOC3	BANF1	CAMP	CELSR1	COL12A1	CYP26A1	DNASE1L3	ERBB2	FKRP	GGT5	H3F3A	HYLS1	JAM3	LAMA5	MARK1	MRE11A	NDUFS4	NUP214	PER2	POLR2E	PTPRD	RNF38	SEPSECS	SLC4A1	SREBF1	TBX6	TOX3	UGT2B7	ZFHX4	
ADGRV1	APOC4	BANK1	CAMSAP2	CELSR2	COL15A1	CYP26B1	DNASE2	ERBB3	FKTN	GH1	H6PD	HYMAI	JARID2	LAMB1	MARS	MREG	NDUFS5	NUP62	PER3	POLR2F	PTPRF	RNF39	SEPT12	SLC4A10	SREBF2	TBXA2R	TP53	UGT8	ZFP36	
ADH1A	APOD	BAP1	CAMTA1	CEMIP	COL17A1	CYP26C1	DND1	ERBB4	FLCN	GH2	HABP2	IAPP	JCAD	LAMB2	MARS2	MRI1	NDUFS6	NXF3	PET100	POLR2G	PTPRJ	RNF6	SEPT14	SLC4A11	SREK1	TBXAS1	TP53AIP1	UHRF1BP1	ZFP36L1	
ADH1B	APOE	BARD1	CAMTA2	CENPE	COL18A1	CYP27A1	DNM1	ERC1	FLG	GHR	HACD1	IARS2	JMJD1C	LAMB3	MARVELD2	MRPL12	NDUFS7	NXF5	PEX1	POLR2H	PTPRK	RNF8	SEPT5	SLC4A3	SRGAP2	TCAP	TP53BP1	UIMC1	ZFP36L2	
ADH1C	APOH	BARX2	CANT1	CENPJ	COL1A1	CYP27B1	DNM1L	ERCC1	FLI1	GHRH	HACE1	IBA57	JMJD7-PLA2G4B	LAMB4	MASP1	MRPL3	NDUFS8	NXN	PEX10	POLR2I	PTPRN2	RNLS	SEPT7	SLC4A4	SRGAP3	TCF12	TP53I3	ULK2	ZFP57	
ADH4	APOL1	BAX	CAPN1	CENPS	COL1A2	CYP2A13	DNM2	ERCC2	FLNA	GHRHR	HADH	IBSP	JPH2	LAMC1	MASP2	MRPL36	NDUFV1	NXNL1	PEX11B	POLR2J	PTPRO	RNMT	SEPT9	SLC4A5	SRI	TCF21	TP53RK	ULK4	ZFP69	
ADH5	APOL2	BAZ1B	CAPN10	CENPX	COL21A1	CYP2A6	DNMT1	ERCC3	FLNB	GHRL	HADHA	ICAM1	JPH3	LAMC2	MAST4	MRPL40	NDUFV2	NYX	PEX12	POLR2J3	PTPRQ	RNU4ATAC	SERAC1	SLC4A7	SRP72	TCF3	TP63	UMOD	ZFP90	
ADH7	APOL3	BBIP1	CAPN12	CEP120	COL25A1	CYP2B6	DNMT3A	ERCC4	FLNC	GHSR	HADHB	ICAM4	JRK	LAMC3	MASTL	MRPL44	NDUFV3	OAS1	PEX13	POLR2K	PTPRT	ROBO1	SERHL2	SLC52A1	SRPX	TCF4	TP73	UMPS	ZFPL1	
ADIPOQ	APOL4	BBS1	CAPN13	CEP126	COL27A1	CYP2C18	DNMT3B	ERCC5	FLOT1	GIF	HAGH	ICAM5	JUN	LAMP2	MAT1A	MRPL48	NEB	OAS2	PEX14	POLR2L	PTPRZ1	ROBO2	SERPINA1	SLC52A2	SRPX2	TCF7	TPCN2	UNC119	ZFPM2	
ADIPOR1	APOL6	BBS10	CAPN3	CEP135	COL2A1	CYP2C19	DNMT3L	ERCC6	FLRT1	GIGYF2	HAL	ICK	JUNB	LAMTOR2	MAT2A	MRPS16	NEBL	OAS3	PEX16	POLR2M	PTRH2	ROBO3	SERPINA10	SLC52A3	SRR	TCF7L1	TPH1	UNC13B	ZFR	
ADK	APOM	BBS12	CAPN5	CEP152	COL3A1	CYP2C8	DNTT	ERCC6L2	FLRT3	GIMAP8	HAMP	ICOS	JUP	LAPTM4B	MATN3	MRPS2	NECAP1	OAT	PEX19	POLR3A	PTS	ROCK1	SERPINA3	SLC5A1	SRSF2	TCF7L2	TPH2	UNC13C	ZFY	
ADM	APOPT1	BBS2	CAPRIN1	CEP164	COL4A1	CYP2C9	DOC2A	ERCC8	FLT1	GIP	HAND1	ID3	KALRN	LARGE1	MATN4	MRPS22	NECTIN1	OAZ1	PEX2	POLR3B	PUF60	ROCK2	SERPINA6	SLC5A11	SRY	TCIRG1	TPI1	UNC13D	ZFYVE26	
ADNP	APP	BBS4	CARD11	CEP170B	COL4A2	CYP2D6	DOCK3	ERF	FLT3	GIPC3	HAND2	ID4	KANK1	LARGE2	MATR3	MRRF	NECTIN3	OBSCN	PEX26	POLRMT	PURA	ROGDI	SERPINA7	SLC5A2	SS18	TCL1A	TPK1	UNC45B	ZFYVE27	
ADORA1	APRT	BBS5	CARD14	CEP19	COL4A3	CYP2D7	DOCK4	ERG	FLT4	GIPR	HAP1	IDE	KANK2	LARP7	MAVS	MS4A1	NECTIN4	OBSL1	PEX3	POMC	PUS1	ROM1	SERPINB11	SLC5A5	SS18L1	TCL1B	TPM1	UNC5C	ZHX3	
ADORA2A	APTX	BBS7	CARD8	CEP250	COL4A3BP	CYP2E1	DOCK6	ERLIN1	FLVCR1	GIT1	HAPLN1	IDH1	KANK4	LARS	MAX	MS4A10	NEDD4	OCA2	PEX5	POMGNT1	PVR	ROPN1L	SERPINB3	SLC5A7	SSBP1	TCN1	TPM2	UNC5CL	ZIC1	
ADORA3	AQP1	BBS9	CARD9	CEP290	COL4A4	CYP2F1	DOCK7	ERLIN2	FLVCR2	GJA1	HARS	IDH2	KANSL1	LARS2	MBD1	MS4A12	NEDD4L	OCLN	PEX6	POMGNT2	PVT1	ROR2	SERPINB4	SLC6A1	SSH1	TCN2	TPM3	UNC80	ZIC2	
ADRA1A	AQP2	BCAM	CARS2	CEP41	COL4A5	CYP2G1P	DOCK8	ERMAP	FMN1	GJA3	HARS2	IDH3B	KANSL2	LAS1L	MBD3	MS4A2	NEDD9	OCRL	PEX7	POMK	PWRN1	RORA	SERPINB5	SLC6A11	SSPN	TCOF1	TPMT	UNC93A	ZIC3	
ADRA2A	AQP3	BCAP31	CARTPT	CEP57	COL4A6	CYP2J2	DOCK9	ERMARD	FMN2	GJA4	HAS1	IDO1	KARS	LAT2	MBD4	MS4A3	NEFH	ODAPH	PFAS	POMP	PWWP2A	RORC	SERPINB6	SLC6A12	SSR4	TCP1	TPO	UNC93B1	ZIC4	
ADRA2B	AQP4	BCAS2	CASC16	CEP63	COL5A1	CYP2R1	DOK1	ERRFI1	FMO1	GJA5	HAS2	IDS	KAT2A	LBH	MBD5	MS4A6A	NEFL	ODC1	PFKFB1	POMT1	PXDN	ROS1	SERPINB7	SLC6A13	SST	TCTE1	TPP1	UNG	ZIC5	
ADRA2C	AQP5	BCAT1	CASC4	CEP68	COL5A2	CYP2U1	DOK2	ESAM	FMO2	GJA8	HAVCR1	IDUA	KAT6A	LBP	MBD6	MS4A6E	NEFM	OFD1	PFKL	POMT2	PYCR1	RP1	SERPINC1	SLC6A14	SSTR2	TCTE3	TPP2	UNKL	ZKSCAN5	
ADRB1	AQP7	BCAT2	CASK	CEP83	COL6A1	CYP2W1	DOK5	ESCO2	FMO3	GJB1	HAX1	IER3IP1	KAT6B	LBR	MBL2	MSH2	NEGR1	OGDH	PFKM	PON1	PYCR2	RP1L1	SERPIND1	SLC6A17	SSTR5	TCTN1	TPPP	UPB1	ZMPSTE24	
ADRB2	AR	BCHE	CASP1	CEP85L	COL6A2	CYP3A4	DOK7	ESPL1	FMO4	GJB2	HBA1	IFI30	KATNAL2	LCA5	MBNL1	MSH3	NEIL1	OGG1	PFN1	PON2	PYCR3	RP2	SERPINE1	SLC6A18	SSX1	TCTN2	TPRN	UPF2	ZMYM2	
ADRB3	ARAF	BCKDHA	CASP10	CEP89	COL6A3	CYP3A43	DOLK	ESPN	FMO5	GJB3	HBA2	IFI44L	KATNB1	LCAT	MBOAT7	MSH4	NEIL2	OGT	PGAM1	PON3	PYGL	RP9	SERPINF1	SLC6A19	SSX2	TCTN3	TPSB2	UPF3B	ZMYM3	
ADSL	AREG	BCKDHB	CASP12	CER1	COL6A4P2	CYP3A5	DOLPP1	ESR1	FMO6P	GJB4	HBB	IFIH1	KBTBD13	LCE3B	MBTPS2	MSH5	NEIL3	OLFM2	PGAM2	POP1	PYGM	RPA1	SERPINF2	SLC6A2	SSX7	TDG	TPTE	UPK3A	ZMYM6	
ADTRP	AREL1	BCKDK	CASP2	CERKL	COL6A5	CYP3A7-CYP3A51P	DOT1L	ESR2	FMOD	GJB6	HBD	IFITM3	KCNA1	LCE3C	MC1R	MSH6	NEK1	OLIG2	PGAM5	POR	PYGO1	RPA2	SERPING1	SLC6A20	ST14	TDGF1	TRA2B	UQCRB	ZMYND10	
AEBP1	ARF4	BCL10	CASP3	CERS3	COL6A6	CYP3A7	DPAGT1	ESRRB	FMR1	GJC2	HBE1	IFITM5	KCNA2	LCE5A	MC2R	MSMB	NEK2	OLR1	PGAP1	PORCN	PYY	RPA3	SERPINH1	SLC6A3	ST3GAL1	TDO2	TRAC	UQCRC1	ZMYND11	
AFF2	ARFGEF2	BCL11A	CASP5	CERS6	COL7A1	CYP46A1	DPCD	ESRRG	FN1	GJC3	HBEGF	IFNA1	KCNA3	LCK	MC3R	MSMO1	NEK8	OMG	PGAP2	POSTN	PZP	RPA4	SERPINI1	SLC6A4	ST3GAL2	TDP1	TRADD	UQCRC2	ZMYND15	
AFF3	ARFRP1	BCL2	CASP8	CES1	COL8A2	CYP4A11	DPH1	ESS2	FN3K	GJD2	HBG1	IFNA10	KCNA4	LCN10	MC4R	MSR1	NELFA	OOEP	PGAP3	POT1	QARS	RPE65	SERPINI2	SLC6A5	ST3GAL3	TDP2	TRAF3	UQCRFS1	ZNF141	
AFF4	ARG1	BCL2A1	CASP9	CES2	COL9A1	CYP4A22	DPM1	ETFA	FNBP1	GK	HBG2	IFNA17	KCNA5	LCT	MCC	MSRA	NELL1	OPA1	PGBD1	POU1F1	QDPR	RPGR	SERTAD1	SLC6A6	ST3GAL4	TDRD7	TRAF3IP1	UQCRH	ZNF157	
AFG3L2	ARHGAP18	BCL2L1	CASQ1	CETN2	COL9A2	CYP4B1	DPM2	ETFB	FNBP4	GLA	HBM	IFNA2	KCNA6	LDB1	MCCC1	MSRB3	NEO1	OPA3	PGBD3	POU2F1	QKI	RPGRIP1	SESN2	SLC6A8	ST3GAL5	TEAD1	TRAF3IP2	UQCRQ	ZNF175	
AFP	ARHGAP24	BCL2L11	CASQ2	CETP	COL9A3	CYP4F12	DPM3	ETFDH	FOLH1	GLB1	HBQ1	IFNAR1	KCNAB1	LDB3	MCCC2	MST1	NEU1	OPCML	PGBD4	POU3F3	RAB10	RPGRIP1L	SETBP1	SLC6A9	ST3GAL6	TEC	TRAF6	UROC1	ZNF202	
AGA	ARHGAP26	BCL2L2	CASR	CFAP53	COLEC11	CYP4F2	DPP10	ETHE1	FOLR1	GLCCI1	HBS1L	IFNAR2	KCNAB2	LDHA	MCEE	MST1R	NEU2	OPHN1	PGC	POU3F4	RAB11FIP5	RPH3AL	SETD2	SLC7A1	ST5	TECPR2	TRAF7	UROD	ZNF213	

GBL	ARHG AP29		BCL3	CAST	CFAP 57	COLQ	CYP4F2 2		DPP3	ETNP PL	FOS	GLDC	HBZ	IFNB1	KCN B1	LDH B	MCF2	MSTN	NEUR OD1	OPL AH	PGD	POU4 F3	RAB1 8	RPIA	SETD 5	SLC7A 10	ST6GA L1	TECR	TRAK 1	UROS	ZNF2 24		
GBL	ARHG AP31		BCL6	CAT	CFB	COM MD1	CYP4F3		DPP4	ETS1	FOXA 1	GLE1	HCAR 1	IFNG	KCN B2	LDLR	MCF2 L2	MSX1	NEUR OG3	OPN 1LW	PGK1	POU5 F1	RAB2 3	RPL10	SETD B1	SLC7A 11	ST6GA L2	TECT A	TRAK 2	USB1	ZNF2 4		
GER	ARHG AP45		BCL7 A	CATS PER1	CFC1	COMP	CYP4V 2		DPP6	ETV4	FOXA 2	GLI1	HCCS	IFNGR 1	KCN C1	LDLR AD4	MCFD 2	MSX2	NEXM IF	OPN 1MW	PGM1	POU5 F1B	RAB2 5	RPL11	SETD B2	SLC7A 14	ST6GA LNAC1	TEK	TRAP1	USF1	ZNF2 92		
GGF	ARHG AP6		BCL9	CATS PER2	CFD	COMT	CYP7A 1	DPY19L2	ETV6	FOXA 3	GLI2	HCFC 1	IFNGR 2	KCN C2	LDLR AP1	MCHR 1	MT1A	NEXN	OPN 1SW	PGM3	POU6 F2	RAB2 7A	RPL15	SETX	SLC7A 2	ST6GA LNAC2	TEKT 2	TRAPP C10	USH1 C	ZNF3 11		
GK	ARHG AP9		BCL A F1	CATS PER3	CFH	COPS2	CYP7B1	DPYD	EVC	FOXC1	GLI3	HCK	IFNL3	KCN C3	LECT 2	MCL1	MT2A	NF1	OPN 4	PGR	PPAR A	RAB2 7B	RPL21	SEZ6	SLC7A 5	ST6GA LNAC3	TELO 2	TRAPP C11	USH1 G	ZNF3 35		
GL	ARHG DIA		BCO1	CATS PER4	CFHR 1	COQ2	CYR61	DPYS	EVC2	FOXC2	GLIS2	HCLS 1	IFNL4	KCN D2	LEF1	MCM3 AP	MTA1	NF2	OPR D1	PGR MC1	PPAR D	RAB2 8	RPL24	SEZ6L	SLC7A 7	ST6GA LNAC4	TEN M1	TRAPP C2	USH2 A	ZNF3 3A		
GM	ARHG EF10		BCOR	CAV1	CFHR 2	COQ4	CYS1	DPYS L2	EVI5	FOXD 1	GLIS3	HCN1	IFRD1	KCN D3	LEFT Y2	MCM4	MTA2	NFAT 5	OPR K1	PHAC TR1	PPAR G	RAB2 9	RPL26	SEZ6L 2	SLC7A 9	ST6GA LNAC5	TEN M2	TRAPP C9	USP1	ZNF3 47		
GO1	ARHG EF11		BCOR L1	CAV3	CFHR 3	COQ5	CYSLT R1	DRC1	EWS R1	FOXD 3	GLMN	HCN2	IFT122	KCN E1	LEM D3	MCM5	MTA3	NFAT C1	OPR L1	PHB	PPAR GC1A	RAB2 A	RPL27	SF3B1	SLC8A 1	ST6GA LNAC6	TEN M3	TRB	USP1 5	ZNF3 50		
GO2	ARHG EF12		BCR	CAV1 N1	CFHR 4	COQ6	CYSLT R2	DRD1	EXO1	FOXD 4	GLO1	HCN3	IFT140	KCN E2	LEP	MCM6	MTAP	NFAT C2	OPR M1	PHB2	PPAR GC1B	RAB3 3B	RPL35 A	SF3B4	SLC9A 1	ST7	TEN M4	TRBV 9	USP2 4	ZNF3 65		
GPA 2	ARHG EF4		BCS1 L	CAV1 N4	CFHR 5	COQ8 A	D2HGD H	DRD2	EXO5	FOXE1	GLP1R	HCN4	IFT172	KCN E3	LEPR	MCM8	MTCH 2	NFAT C3	OPT C	PHC1	PPAT	RAB3 4	RPL36	SFPQ	SLC9A 3	ST8SIA 1	TEP1	TRD	USP2 6	ZNF3 85B		
GPS	ARHG EF6		BDKR B2	CBFA 2T2	CF1	COQ8 B	DAAM1	DRD3	EXOC 4	FOXE3	GLRA1	HCP5	IFT27	KCN E4	P3H1	MCM9	MTF1	NFAT C4	OPT N	PHEX	PP1A	RAB3 9B	RPL38	SFRP1	SLC9A 3R1	ST8SIA 2	TERC	TRDN	USP2 7X	ZNF3 95		
GRN	ARHG EF7		BDNF	CBFA 2T3	CFL1	COQ9	DAB1	DRD4	EXOC 6B	FOXF1	GLRA4	HCRT	IFT43	KCN E5	LET M1	MCOL N1	MTF MT	NFE2L 1	OR10 V1	PHF1 1	PP1B	RAB3 A	RPL5	SFTP A1	SLC9A 6	ST8SIA 3	TERT	TREH	USP3 07	ZNF4 07		
GRP	ARHG EF9		BDP1	CBFB	CFL2	CORI N	DAB2	DRD5	EXOC 8	FOXF2	GLRB	HCRT R1	IFT80	KCN H1	LFNG	MCPH 1	MTHF D1	NFE2L 2	OR10 X1	PHF2	PPIG	RAB3 GAP1	RPL6	SFTP A2	SLC9A 9	ST8SIA 4	TET1	TREM 2	USP3 0	ZNF4 08		
GT	ARID 1A		BEAN 1	CBL	CFLA R	CORO 1A	DACH1	DROS HA	EXOS C3	FOXG 1	GLRX5	HCRT R2	IFT88	KCN H2	LGA LS13	MCTP 2	MTHF D1L	NF1A	OR13 G1	PHF2 1A	PPM1 B	RAB3 GAP2	RPN2	SFTP B	SLCO1 A2	ST8SIA 5	TET2	TRERF 1	USP3 4	ZNF4 1		
GTP P1	ARID 1B		BEND 2	CBLB	CFP	COX1 0	DAD1	DRP2	EXOS C5	FOXH 1	GLS	HDAC 1	IGBP1	KCN H3	LGA LS2	MCUB	MTHF R	NF1B	OR13 H1	PHF3	PPM1 D	RAB4 0AL	RPS10	SFTP C	SLCO1 B1	ST8SIA 6	TEX1 3B	TREX1	USP4 4	ZNF4 19		
GTR	ARID 2		BEST 1	CBR1	CFTR	COX1 4	DAG1	DSC2	EXOS C8	FOXI1	GLTP	HDAC 4	IGF1	KCN H5	LGA LS3	MDC1	MTHF S	NFIX	OR1 B1	PHF6	PPM1 G	RAB7 A	RPS14	SFTP D	SLCO1 B3	STAC3	TEX1 4	TREX2	USP4 6	ZNF4 20		
GTR	ARID 4A		BEX4	CBR3	CGA	COX1 5	DAO	DSC3	EXPH 5	FOXJ1	GLUD1	HDAC 6	IGF1R	KCNI P4	LG11	MDGA 2	MTM1	NFKB ID1	OR1J 1	PHF8	PPM1 K	RABG GTA	RPS15	SFXN 4	SLCO1 C1	STAG1	TEX2 8	TRH	USP5 3	ZNF4 23		
GXT	ARID 4B		BFSP1	CBS	CGB3	COX2 0	DAOA	DSCAM	EXT1	FOXK 1	GLUD2	HDAC 8	IGF2	KCNJ 1	LG14	MDH1	MTM R12	NFKB 2	OR2J 3	PHGDH	PBLA 6	RPS17	SGCA	SLCO2 A1	STAG2	TF	TRHR	USP7 4	ZNF4 33			
GXT	ARID 5A		BFSP2	CBX2	CGNL 1	COX4I 1	DAPK1	DSCR 8	EXT2	FOXL1	GLUL	HDAC 9	IGF2B P2	KCNJ 10	LGR4	MDH2	MTM R14	NFKBI A	OR2 M2	PHIP	PPP1R 12B	RAC1	RPS19	SGCB	SLCO2 B1	STAG3	TFAM	TRIB1	USP7	ZNF4 4		
HCY	ARIH 1		BGN	CBX4	CHAF 1B	COX4I 2	DAPK3	DSE	EXTL 1	FOXL2	GLYC TK	HDC	IGF2R	KCNJ 11	LGR5	MDK	MTM R2	NFKBI L1	OR4 M2	PHKA 1	PPP1R 15B	RAC2	RPS24	SGCD	SLCO5 A1	STAMB P	TFAP 2A	TRIB2	USP8	ZNF4 5		
HDC	ARL1 1		BHLH A9	CBY1	CHA MP1	COX5 A	DAPP1	DSG1	EXTL 2	FOXM 1	GM2A	HDGF L2	IGFAL S	KCNJ 12	LHB	MDM2	MTM R8	NFKBI Z	OR51 G1	PHKA 2	PPP1R 17	RAD1	RPS26	SGCE	SLFN5	STAR	TFAP 2B	TRIB3	USP9 X	ZNF4 51		
HI1	ARL1 3B		CC2D1 A	CHAT	COX5 B	DARS	DSG2	EXTL 3	FOXN 1	GMDS	HDHD 5	IGFBP 1	KCNJ 13	LHCGR	MDM4	MTM R9	NFS1	OR52 H1	PHKB	PPP1R 1A	RAD1 7	RPS27	SGCG	SLIT1	STARD 6	TFB1 M	TRIL	USP9 Y	ZNF4 69		
HNA 2	ARL1 4EP		BHM T	CC2D2 A	CHCH D10	COX6 A1	DARS2	DSG3	EYA1	FOXO 1	GMIP	HDLB P	IGFBP 3	KCNJ 15	LHFP L5	MDN1	MTM R1A	NFU1	OR52 N4	PHKG 2	PPP1R 3A	RAD1 8	RPS27 A	SGK1	SLIT3	STARD 9	TFCP 2	TRIM1 7	UST	ZNF4 80		
HR	ARL2 BP		BICC1	CCBE 1	CHD1	COX6 B1	DAXX	DSG4	EYA4	FOXP1	GMPP A	HDX	IGFBP 5	KCNJ 2	LHX1	ME2	MTNR 1B	NGEF	OR5 AC2	PHLD A2	PPP1R 3C	RAD2 1	RPS28	SGO1	SLITR K1	STAT1	TFDP 1	TRIM2	UTF1	ZNF5 07		
HRR	ARL3		BICD 1	CCDC 103	CHD1 L	COX6 C	DAZ1	DSP	EYS	FOXP2	GMPP B	HELQ	IGFBP 7	KCNJ 3	LHX3	MECOM	MTO1	NGF	OR5 H6	PHLP P2	PPP1R 8	RAD2 1L1	RPS29	SGSH	SLITR K5	STAT2	TFE3	TRIM2 1	UTP1 4C	ZNF5 13		
HSG	ARL6		BICD 2	CCDC 107	CHD2	COX7 A1	DAZ2	DSPP	EZH2	FOXP3	GMPS	HEPA CAM	IGHA1	KCNJ 5	LHX4	MECP 2	MTOR	NGFR	OR7 D4	PHOX 2A	PPP2R 1A	RAD2 3A	RPS3	SGSM 3	SLITR K6	STAT3	TFF1	TRIM2 2	UTP4	ZNF5 17		
HSP	ARL6I P1		BICR A	CCDC 114	CHD3	COX7 A2	DAZ3	DST	F10	FOXP4	GNA11	HEPA CAM2	IGHG2	KCNJ 6	LHX8	MED1 2	MTPA P	NGLY 1	OR8 H1	PHOX 2B	PPP2R 1B	RAD2 3B	RPS4 Y2	SH2B 1	SLK	STAT4	TFG	TRIM2 4	UTP6	ZNF5 26		
ICD	ARL6I P5		BIN1	CCDC 127	CHD4	COX7 B	DAZ4	DSTYK	F11	FOXRED1	GNA13	HEPH	IGHM	KCNJ 8	LIAS	MED1 3	MTR	NHEJ1	OR8 K3	PHYH	PPP2R 2A	RAD5 0	RPS5	SH2B 3	SLMA P	STAT5 B	TFPI	TRIM2 8	UTRN	ZNF5 82		
IF1	ARL6I P6		BIRC5	CCDC 14	CHD6	COX7 B2	DAZL	DTHD 1	F12	FPGS	GNA14	HERC 1	IGHM BP2	KCNJ 9	LIF	MED1 3L	MTRR	NHLR C1	ORA I1	PHYKPL	PPP2R 2B	RAD5 1	RPS6 KA3	SH2D 1A	SLTM	STAT6	TFR2	TRIM3 2	UTS2	ZNF5 92		
IFM1	ARM C4		BIVM	CCDC 148	CHD7	COX7 C	DBF4B	DTNA	F13A 1	FPR1	GNAI2	HERC 2	IGKV1 -5	KCNK17	LIFR	MED1 7	MTSS 1	NHP2	ORC 1	PI3	PPP2R 2C	RAD5 1B	RPS6 KB1	SH3B P2	SLUR P1	STBD1	TFRC	TRIM3 3	UVSS	ZNF5 99		
IMP1	ARM C5		BLK	CCDC 151	CHD8	CP	DBH	DTNB	F13B	FPR2	GNAI3	HES1	IGLL1	KCNK18	LIG1	MED2 0	MTSS 1L	NHS	ORC 4	PI4K A	PPP2R 5B	RAD5 1C	RPS6 KL1	SH3G L1	SLX1 A	STEAP3	TG	TRIM3 7	VAM P1	ZNF6 27		
IP	ARMS 2		BLM	CCDC 170	CHDH	CPA1	DBI	DTNBP1	F2	FRA10 AC1	GNAL	HES6	IGSF1	KCNK3	LIG3	MED2 3	MTIP	NICN1	ORC 6	PIAS1	PPP2R 5C	RAD5 1D	RPS7	SH3P XD2B	SLX1B	STEAP4	TGDS	TRIM5	VAM P7	ZNF6 38		
IPL1	ARNT		BLM H	CCDC 18	CHEK 1	CPA4	DBP	DUO X1	F2R	FRAS1	GNAO 1	HES7	IGSF3	KCNK6	LIG4	MED2 5	MTUS 1	NID1	ORM 1	PIBF1	PPP2R 5D	RAD5 2	RPSA	SH3T C2	SLX4	STH	TGFB 1	TRIM6 3	VAN GL1	ZNF6 44		
IRE	ARNT 2		BLNK	CCDC 198	CHEK 2	CPA6	DBT	DUO X2	F2RL 1	FREM 1	GNAQ	HESX 1	IGSF8	KCNK9	LILR A3	MEF2 A	MUC1	NIN	ORM DL3	PICA LM	PPP3C A	RAD5 4B	RPTOR	SHAN K1	SMAD 1	STIL	TGFB 2	TRIO	VAN GL2	ZNF6 68		
K1	ARNT L		BLOC 1S3	CCDC 22	CHFR	CPB2	DCAF1 1	DUO XA2	F3	FREM 2	GNAS	HEXA	IHH	KCNMA1	LIM2	MEF2 C	MUC1 3	NINJ1	OSBPL2	PICK1	PPP3R 1	RAD5 4L	RRAS	SHAN K2	SMAD 2	STIM1	TGFB 3	TRIOB P	VAPB	ZNF6 74		
K2	ARPC 3		BLOC 1S5	CCDC 28B	CHGA	CPE	DCAF1 3	DUSP 15	F5	FREM 3	GNAS-AS1	HEXB	IKBIP	KCNMB1	LIMK 1	MEFV	MUC2	NIPA1	OSGIN1	PID1	PPP4C	RAD5 4L2	RRAS 2	SHAN K3	SMAD 3	STIM2	TGFB 1	TRIP1 1	VARS	ZNF6 83		
K7	ARSA		BLOC 1S6	CCDC 33	CHGB	CPLX2	DCAF1 7	DUSP 22	F7	FRG1	GNAT1	HEY1	IKBKB	KCNMB3	LIMS 2	MEGF 10	MUC3 A	NIPA2	OSMR	PIEZ O1	PPP4R 1	RAD9 A	RREB 1	SHAR PIN	SMAD 4	STK10	TGFB R1	TRIP1 11	VAV3	ZNF7 11		
K8	ARSB		BLVR A	CCDC 39	CHI3L 1	CPN1	DCAF4	DUSP 23	F8	FRK	GNAT2	HEY2	IKBKE	KCNMB4	LIN28A	MEGF 11	MUC4	NIPAL 4	OSR1	PIEZ O2	PPP4R 2	RAD9 B	RRH	SHBG	SMAD 5	STK11	TGFB R2	TRIP1 3	VAX1	ZNF7 13		
KAP	ARSE		BMI1	CCDC 40	CHI3L 2	CPOX	DCAF8	DUSP 3	F9	FRMD 5	GNB1L	HFE	IKBKG	KCNN2	LIN28B	MEGF 8	MUC5 B	NIPBL	OST M1	PIF1	PPP4R 3A	RAET 1L	RRM1	SHH	SMAD 6	STK11I P	TGFB R3	TRIT1	VAX2	ZNF7 50		
KAP 0	ARSF		BMP1	CCDC 50	CHIA	CPQ	DCAKD	DUSP 6	FA2H	FRMD 6	GNB3	HFE2	IKZF1	KCNN3	LIN7 A	MEIS1	MUC6	NIPSNAP1	OTC	PIGA	PPP4R 3B	RAF1	RRM2	SHMT 1	SMAD 7	STK19	TGFB RAP1	TRMT 1	VCA M1	ZNF7 5D		
KAP 3	ARSH		BMP1 0	CCDC 6	CHIC2	CPS1	DCBLD 1	DUT	FAAH	FRMD 7	GNB4	HFM1	IKZF3	KCNQ1	LING O2	MEIS2	MUC7	NIPSNAP3A	OTOA	PIGL	PPP4R 4	RAG1	RRM2B	SHOC 2	SMAD 9	STK3	TGIF1	TRMT 10A	VCA N	ZNF7 7		
KAP	ARSI		BMP1 5	CCDC 62	CHIT1	CPT1A	DCBLD 2	DUX4	FAAH 2	FRMPD4	GNB5	HGD	IL10	KCNQ2	LINS 1	MELK	MUS81	NISCH	OTOF	PIGM	PPP6C	RAG2	RRP1B	SHOX	SMARCA1	STK32 A	TGM1	TRMT 44	VCL	ZNF7 78		
KAP	ART4		BMP2	CCDC 65	CHKB	CPT1B	DCC	DUX4 L3	FAAP 100	FRRS1 L	GNE	HGF	IL10R A	KCNQ3	LIPA	MELT F	MUSK	NKAIN2	OTOG	PIGN	PPP6R 1	RAI1	RS1	SHOX 2	SMARCA2	STK33	TGM2	TRMU	VCP	ZNF7 80B		
KR1 1	ARV1		BMP2 K	CCDC 66	CHL1	CPT1C	DCDC2	DVL1	FAAP 24	FRY	GNG2	HGSNAT	IL10R B	KCNQ4	LIPC	MEN1	MUT	NKAIN3	OTOGL	PIGO	PPP6R 2	RALGAPA1	rs1074 8087	SHROOM3	SMARCA4	STK35	TGM4	TRNT1	A	ZNF7 83		
KR1 2	ARVCF		BMP3	CCDC 78	CHM	CPT2	DCHS1	DVL2	FABP 1	FRZB	GNG7	HHEX	IL11	KCNQ5	LIPE	MEOX 1	MUT YH	NKX2-1	OTOR	PIGQ	PPP6R 3	RALGDS	rs1113 0795	SHROOM4	SMARCAD1	STK36	TGM5	TROAP	VCY	ZNF7 9		
KR1 3	ARX		BMP4	CCDC 8	CHMP 1A	CPXCR1	DCK	DXO	FABP 2	FSBP	GNMT	HHIP	IL11RA	KCNS1	LIPG	MEP1B	MVK	NKX2-2	OTUD4	PIGR	PPT1	RANBP2	rs1268 2834	SI	SMARCAL1	STK39	TGM6	TROVE2	VDAC1	ZNF8 0		

AKR1C4	ASSM T	BMP5	CCDC 85C	CHMP 2B	CPZ	DCLK1	DYM	FABP 3	FSCB	GNPA T2	HBA 1/HBA 2	IL12A	KCNS3	LIPH	MEPE	MX1	NKX2-3	OTX2	PIGT	PQBP 1	RANBP2	rs1395 5974	SLAE	SMARCA1	STK4	TH	TRPA1	VDR	ZNF84
AKR1D1	ASAH1	BMP7	CCDC 88C	CHMP 4B	CR1	DCLRE 1A	DYNAP	FABP 4	FSCN2	GNPTAB	HBB	IL12B	KCNT1	LIPI	MERTK	MX11	NKX2-5	OVCH2	PIGV	PRAF 2	RANGRF	rs2383 136	SIGLEC12	SMARCC1	STMN1	THAD A	TRPC3	VIGFA	ZNF8 1
AKR7A2	ASAH2	BMPR	CCDC 90B	CHN1	CR2	DCLRE 1B	DYNC1I1	FABP 6	FSHB	GNPTG	HBD	IL12R B1	KCNV1	LIPN	MESD	MXRA5	NKX2-6	OVGP1	PIGW	PRAG 1	RAP1A	rs2988 039	SIGLEC14	SMARCC2	STOM	THAP 1	TRPC4	VIGFC	ZNF8 13