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Interpretation of Genomic Sequencing Results in Healthy and Ill Newborns: Results from the BabySeq Project

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Genomic sequencing provides many opportunities in newborn clinical care, but the challenges of interpreting and reporting newborn genomic sequencing (nGS) results need to be addressed for its broader and effective application. The BabySeq Project is a pilot randomized clinical trial that explores the medical, behavioral, and economic impacts of nGS in well newborns and those admitted to a neonatal intensive care unit (NICU). Here we present childhood-onset and actionable adult-onset disease risk, carrier status, and pharmacogenomics findings from nGS of 159 newborns in the BabySeq Project. nGS revealed a risk of childhood-onset disease in 15/159 (9.4%) newborns; none of the disease risks were anticipated based on the infants' known clinical or family histories. nGS also revealed actionable adult-onset disease risk in 3/85 (3.5%) newborns whose parents consented to receive this information. Carrier status for recessive diseases and pharmacogenomics variants were reported in 88% and 5% of newborns, respectively. Additional indication-based analyses were performed in 29/32 (91%) NICU newborns and 6/127 (5%) healthy newborns who later had presentations that prompted a diagnostic analysis. No variants that sufficiently explained the reason for the indications were identified; however, suspicious but uncertain results were reported in five newborns. Testing parental samples contributed to the interpretation and reporting of results in 13/159 (8%) newborns. Our results suggest that nGS can effectively detect risk and carrier status for a wide range of disorders that are not detectable by current newborn screening assays or predicted based on the infant's known clinical or family history, and the interpretation of results can substantially benefit from parental testing.

Introduction

Recent advances in genomic sequencing (GS) technologies have raised the possibility of its routine implementation in newborn care.[1,2] Newborn GS (nGS) provides many potential opportunities in the clinical management of a newborn. First, it might identify risk for a broad range of disorders in babies who are asymptomatic at birth and thereby expand the spectrum of conditions for which screening is possible. This would avoid constraints, such as the availability of a biochemical screening method, or confounding factors, such as the baby's gestational age at birth, transfusion status, age at sample collection, or metabolic and feeding states. Second, nGS could reduce the diagnostic odyssey for ill newborns by allowing for the timely application of appropriate treatments. The success of nGS in providing a rapid diagnosis for critically ill newborns suspected of having single-gene disorders has been demonstrated in recent studies.[3,4] Third, pharmacogenomics (PGx) information from nGS has the potential to inform the selection and dosing of drugs for optimal treatment strategies in childhood and throughout life. Finally, nGS can reveal carrier-status information that could help in future reproductive planning at a time when families are having children. In addition to its utility in the newborn period, nGS can provide a genomic dataset that can be reanalyzed throughout the individual's life whenever new indications arise. All of these potential benefits can be achieved with a single test, which also provides the opportunity to inexpensively and conveniently re-interrogate the sequence over time as needed when new healthcare issues arise.

Despite its anticipated benefits, some of the major challenges in the use of nGS are the analysis, interpretation, and appropriate reporting of healthcare-related information from genomic data in a timely manner. Variant interpretation, as well as the prediction of likelihood, severity, and timing of a phenotype from a specific variant, are especially difficult in the newborn population because of the absence or obscurity of a phenotype for many disorders at birth. Yet estimating the penetrance and age-of-onset of variants is particularly critical for newborns because of concerns about returning low-risk or adult-onset findings. Predicting the inheritance pattern of

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variants might not always be straightforward either because both dominant and recessive variants have been reported for many genes. Data derived from studies addressing the technical and interpretive aspects of nGS are needed if researchers are to develop best practices for its responsible and effective implementation. Newborn Sequencing in Genomic Medicine and Public Health (NSIGHT) is an NIH-funded consortium of four research programs designed to address some of these questions.[5] Within NSIGHT, the BabySeq Project is a pilot randomized clinical trial that explores the application of nGS in healthy and ill newborns without selecting for those suspected to have a genetic disorder, and it assesses the medical, behavioral, and economic impacts of nGS.[6] Here we present the analysis and reporting of nGS results in 159 newborns enrolled in the BabySeq Project; the report includes (1) risk and carrier status for childhood-onset disease, (2) risk for medically actionable adult-onset disease, (3) selected PGx findings relevant to medications used in pediatrics, and (4) variants related to a specific indication that either was present at birth or arose during the course of the study.

Subjects and Methods

The BabySeq Project Study Design

A description of the BabySeq Project, the enrollment process, and the demographic characteristics of the participants has been published elsewhere.[6,7] In brief, two cohorts of newborns and their parents were enrolled in the BabySeq Project: (1) healthy newborns from the well-baby nursery at Brigham and Women's Hospital (BWH) and (2) ill newborns from the neonatal and pediatric intensive care units (NICUs) at Boston Children's Hospital (BCH), BWH, and Massachusetts General Hospital (MGH). Enrollees from the NICUs were not preselected on the basis of having a suspected genetic disorder. Three-generation pedigrees were obtained for each family during the consent and enrollment sessions with a genetic counselor. Half of the newborns in each cohort were randomized to receive standard care, including state-mandated newborn screening, plus genetic counseling based on their family histories; the others received nGS in addition to standard care and genetic counseling based on their family histories. The nGS group consisted of 56% white, 23% multi-racial, 2.5% Hispanic or Latino, 1.3% black or African American, 1.3% Asian, and 1.3% Native Hawaiian, other Pacific Islander, or other, although 16% did not specify their ethnicity. The nGS reports of those who were randomized to receive sequencing were disclosed during genetic counseling and were entered into the newborn's medical record, as well as delivered directly to the newborn's clinicians. The impacts of nGS on the infant's clinical care, parent and clinician behaviors, and economic outcomes were evaluated via deep phenotyping of a subset of infants, as well as implementation of baseline, 3-month, and 10-month post-disclosure surveys in parents and baseline, post-disclosure, and end-of-study surveys in clinicians. The impact on clinicians' behavior was ascertained via baseline, post-disclosure, and end-of-study surveys. This study was approved by the BCH and Partners Healthcare institutional review boards. When the identity of both biological parents was known, both provided informed consent for themselves as well as their infant; if applicable, consent was obtained from any non-biological legal guardians.

nGS Analysis

Whole-exome sequencing (WES) was performed at the CLIA-accredited Clinical Research Sequencing Platform of the Broad Institute, and Sanger confirmation was performed at the CLIA-accredited Partners Healthcare Laboratory for Molecular Medicine as previously described.[8] Variants were assessed and classified as described.[9,10] All nGS results were returned in a Newborn Genomic Sequencing Report (NGSR), which included an indication-based analysis (IBA) for any additional diagnostic assessment related to a clinical indication. The first page of the NGSRs summarized the analysis approach and the results in order to concisely communicate key findings from the nGS; subsequent pages provided more detailed information about each reported variant; such information included gene coverage, interpretation of the variant's clinical significance, a summary of related disease(s), and associated reproductive risks. The criteria used for return of results were as previously described.[6] In brief, three groups of results were returned in the NGSR: (1) monogenic disease risks (MDR, defined as pathogenic or likely pathogenic [P/LP] variants in genes associated with dominantly inherited diseases or as bi-allelic P/LP variants in genes associated with recessively inherited diseases) that present or are manageable during childhood (i.e., the earliest reported onset is before the age of 18); (2) carrier status for any gene meeting the MDR reporting criteria; and (3) PGx-associated genes, which were captured by our WES method, that are related to atypical reactions to medications used in the pediatric population. Later in the study, information about risk for a limited number of actionable adult-onset conditions (for which screening, treatment, and preventative actions that would significantly reduce morbidity and mortality are available during adulthood) was also offered for return, and this information was included in the NGSRs for newborns whose parents consented to receive this information for their infant. The actionable adult-onset disease-associated genes included five additional genes (*BRCA1* [MIM: 113705], *BRCA2* [MIM: 600185], *MLH1* [MIM: 120436], *MSH2* [MIM: 609309], and *MSH6* [MIM: 600678]), which are all found on ACMG 59, a list of clinically important genes that are recommended for reporting incidental findings by the American College of Medical Genetics and Genomics,[11] and are associated with hereditary breast and ovarian cancer or Lynch syndrome. It should be noted that *PMS2* (MIM: 600259) was excluded from the analysis because the majority of its pathogenic variation could not be reliably assessed by standard WES. The remaining 53 genes on the ACMG 59 list were already being returned on the basis of our baseline criteria for returning childhood-onset and childhood-actionable conditions. For the NICU cohort and the newborns who were enrolled from the well-baby nursery and who later had an indication revealed through record review or clinical follow-up by referring study physicians, an IBA was performed so that all variants in genes with potential relevance to the presenting phenotype could be assessed. Only P/LP variants were returned in the NGSRs; however, all variants with evidence supporting a contribution to the infant's indication, including variants of uncertain significance (VUSs) in genes related to the existing phenotype and genes with moderate or limited evidence of causing the specific indication, were returned in IBAs. This allowed for studies such as segregation analysis or further clinical evaluation that could help clarify their clinical significance. When needed, parental samples were also collected and tested so that the clinical

significance could be clarified and/or the familial risk of variants detected in the newborn could be described. All reported variants were confirmed by Sanger sequencing. Because of the limitations of next-generation sequencing and standard Sanger sequencing in analyzing the *CYP21A2* (MIM: 613815) gene, the identified c.844G>T (p.Val282Leu) and c.1447C>T (p.Pro483Ser) variants were confirmed to occur on the authentic gene via a long-range PCR assay at an outside reference laboratory.

Results

127 healthy newborns from the well-newborn nursery and 32 ill newborns who were in a NICU and were enrolled in the BabySeq Project were randomized to receive nGS as previously described.[8]

Monogenic Disease Risk

We interpreted nGS results in 159 subjects to identify P/LP variants in (1) genes with at least strong evidence of causing highly penetrant (>80% penetrance based on cases reported in the literature) disorders that present or are clinically manageable during childhood and (2) genes with moderate evidence and/or penetrance associated with conditions for which intervention during childhood might prevent a devastating outcome later in life.[8] Variants that conferred disease risk, met these criteria, and were unrelated to any known existing phenotype in the newborn were identified in 15 of 159 (9.4%) newborns; 10 of the newborns were healthy and were enrolled from the well-baby nursery (Table 1), and the 5 remaining newborns were from the NICUs. In 3 of 85 (3.5%) newborns whose parents consented to receive information about actionable adult-onset disease risk, pathogenic variants conferring risk for hereditary breast and ovarian cancer or Lynch syndrome were identified (Table 1).

Five genes identified as conferring a risk of childhood-onset disease were reported to have high (>80%) penetrance in the literature. Three of these were associated with autosomal-dominant (AD) conditions. *KCNQ4* (MIM: 603537) is associated with non-syndromic hearing loss (MIM: 600101) that typically has a post-lingual presentation within the second decade of life.[12] *GLMN* (MIM: 601749) is associated with glomuvenous malformations (MIM: 138000), which are vascular lesions with a cobblestone appearance and are painful on palpation.[13] A NICU newborn with an anteriorly displaced and imperforate anus had a *de novo,* likely pathogenic variant in *ANKRD11* (MIM: 611192), which has been previously associated with KBG syndrome (MIM: 148050), a disorder characterized by macrodontia, distinctive craniofacial features, skeletal anomalies, and developmental delay.[14] Although one patient with KBG syndrome has been recently reported as having an anteriorly displaced anus,[15] because anorectal malformations were not part of the known phenotypic spectrum of KBG syndrome at the time of our analysis, this variant was initially returned as an incidental finding unrelated to the newborn's clinical indication; it was later considered to be diagnostically relevant. Two other newborns were found to have bi-allelic variants in genes associated with autosomal-recessive (AR) conditions: biotinidase deficiency (BTD [MIM: 253260]) and congenital adrenal hyperplasia due to 21-hydroxylase deficiency (CAH [MIM: 201910]).

Three of the above disease-risk findings were related to conditions that are tested for by standard newborn screening (NBS) and were identified in newborns who had passed their NBS. As a result of the postlingual onset of *KCNQ4*-related hearing loss, the effects of a likely pathogenic variant in this gene are not expected to be detected by audiological screening at birth. In another newborn enrolled from the well-baby nursery,[16] nGS identified compound heterozygosity for two *BTD* variants; one was classified as pathogenic (GenBank: NM_000060.4; c.1612C>T [p.Arg538Cys]) and the other (c.44+1G>A (p.?)) as a VUS during the initial assessment on the basis of the existence of transcripts without the relevant exon 1 that might abrogate the effects of predicted splicing disruption. To clarify the clinical significance of the c.44+1G>A variant, we further investigated the baby's NBS results and discovered borderline NBS results for BTD; a subsequent diagnostic measure of enzyme levels also confirmed partial BTD.[16] As a result, we classified the c.44+1G>A variant as likely pathogenic, and the newborn was placed on biotin supplementation. Two variants in *CYP21A2* were identified in a female baby with severe chronic lung disease. These two variants have previously been reported in *trans* with each other in several individuals with nonclassic CAH,[17–22] suggesting that compound heterozygosity for these variants is associated with the nonclassic form of the disease. Females with nonclassic CAH present postnatally with hyperandrogenism signs, such as hirsutism, menstrual irregularities, and infertility, all features that would not be expressed in infancy.[23]

Eleven variants were found in genes that were previously reported to have moderate (20%–80%) penetrance, and these were disclosed in our study because knowledge about disease risk could allow early interventions during childhood to reduce morbidity and mortality. These variants included six that were associated with dilated (MIM: 604145, 611407) or hypertrophic cardiomyopathies (MIM: 115197) in *TTN* (MIM: 188840) (four newborns), *VCL* (MIM: 193065), and *MYBPC3* (MIM: 600958). There were also one variant each in *ELN* (MIM: 130160), *CD46* (MIM: 120920), *SLC7A9* (MIM: 604144), and *G6PD* (MIM: 305900), variants in which are associated with supravalvular aortic stenosis (SVAS [MIM: 185500]), atypical hemolytic-uremic syndrome (aHUS [MIM: 612922]), type I cystinuria (MIM: 220100), and G6PD deficiency (MIM: 300908), respectively. It should be noted that the rate of *TTN* variants was quite high, raising concern of false positives because of the challenges in interpreting predicted loss-of-function (LOF) variants in *TTN*.[24] However, we followed the current best practice for ensuring the LOF variants were located in exons that are not

Table 1. Findings Pertaining to Monogenic-Disease Risk

Phenotype at Enrollment	Sex	Ethnicity or Race	Gene (Transcript)	Variant(s), (Classification)	Zygosity	Disease	Inh	Parent of Origin	Penetrance[a]
Well-Baby Cohort									
healthy	m	white	*BRCA2*[b] (GenBank: NM_000059.3)	c.8297delC (p.Thr2766Asnfs*11), (P)	het	hereditary breast and ovarian cancer	AD	mat	high
healthy	f	white	*BTD* (GenBank: NM_000060.2)	c.[44+1G>A;1612C>T] (p.[?;Arg538Cys]), (LP;P)	comp het	biotinidase deficiency	AR	mat & pat	high
healthy	f	unspecified	*CD46* (GenBank: NM_002389.4)	c.286+2T>G (p.?), (LP)	het	atypical hemolytic-uremic syndrome	AD	mat	moderate
healthy	m	white	*ELN* (GenBank: NM_000501.3)	c.1957G>T (p.Gly653*), (P)	het	supravalvular aortic stenosis	AD	pat	moderate
healthy	f	unspecified	*KCNQ4* (GenBank: NM_004700.3)	c.1671_1672insACGAC (p.Val558Thrfs*3), (LP)	het	non-syndromic hearing loss	AD	pat	high
healthy	m	white	*MYBPC3* (GenBank: NM_000256.3)	c.1624G>C (p.Glu542Gln), (P)	het	hypertrophic cardiomyopathy	AD	mat	moderate
healthy	m	white	*TTN* (GenBank: NM_133378.4)	c.34894_34895insG (p.Met11632Serfs*8), (LP)	het	dilated cardiomyopathy	AD	mat	moderate
healthy	f	multi-racial	*TTN* (GenBank: NM_133432.3)	c.12344delC (p.Pro4115Glnfs*14), (LP)	het	dilated cardiomyopathy	AD	mat	moderate
healthy	m	unspecified	*TTN* (GenBank: NM_133378.4)	c.54172C>T (p.Arg18058*), (LP)	het	dilated cardiomyopathy	AD	pat	moderate
healthy	f	white	*TTN* (GenBank: NM_133378.4)	c.64276_64282delinsTA (p.Ala21426*), (P)	het	dilated cardiomyopathy	AD	pat	moderate
healthy	f	white	*VCL* (GenBank: NM_014000.2)	c.1713delA (p.Ala573Hisfs*8), (LP)	het	dilated cardiomyopathy	AD	mat	moderate
NICU Cohort									
anteriorly displaced and imperforate anus	f	white	*ANKRD11* (GenBank: NM_001256182.1)	c.2409_2412del (p.Glu805Argfs*57), (LP)	het	KBG syndrome	AD	*de novo*	high
hypoplastic left heart	m	white	*BRCA2*[b] (GenBank: NM_000059.3)	c.3545_3546del (p.Phe1182*), (P)	het	hereditary breast and ovarian cancer	AD	mat	high
congenital severe chronic lung disease	f	unspecified	*CYP21A2* (GenBank: NM_000500.7)	c.[844G>T;1447C>T] (p.[Val282Leu;Pro483Ser]), (P;P)	comp het[c]	congenital adrenal hyperplasia due to 21-hydroxylase deficiency	AR	unk[c] & pat	high

(*Continued on next page*)

Table 1. ***Continued***

Phenotype at Enrollment	Sex	Ethnicity or Race	Gene (Transcript)	Variant(s), (Classification)	Zygosity	Disease	Inh	Parent of Origin	Penetrance[a]
aortic coarctation	m	native Hawaiian or other Pacific Islander	*G6PD* (GenBank: NM_000402.3)	c.961G>A (p.Val321Met), (LP)	hem	glucose-6-phosphate dehydrogenase deficiency	XLR	mat	moderate
tetralogy of Fallot, pulmonic stenosis, and cryptorchidism	m	white	*GLMN* (GenBank: NM_053274.2)	c.554_558delinsG (p.Lys185Serfs*19), (LP)	het	glomuvenous malformations	AD	pat	high
respiratory distress (surfactant deficiency) and hypoglycemia	f	multi-racial	*MSH2*[b] (GenBank: NM_000251.2)	c.1637_1638insA (p.Asn547Glufs*4), (P)	het	Lynch syndrome	AD	mat	high
neonatal pneumonia and meconium aspiration	m	white	*SLC7A9* (GenBank: NM_014270.4)	c.614dupA (p.Asn206Glufs*3), (P)	het	cystinuria	AD	mat	moderate

Abbreviations are as follows: m = male; f = female; AD = autosomal-dominant; AR = autosomal-recessive; XLR = X-linked recessive; P = pathogenic; LP = likely pathogenic; het = heterozygous; hom = homozygous; hem = hemizygous; comp het = compound-heterozygous; mat = maternal; and pat = paternal, unk = unknown.[a]Estimated penetrance for the gene was defined on the basis of curated literature for reported individuals with pathogenic variants in the gene. It was classified as "high" if ≥80% of individuals were symptomatic, "moderate" if 20%–80% of individuals were symptomatic, and "low" if <20% of individuals were symptomatic, as described.[6]

[b]Actionable adult-onset finding; please see text for explanation.

[c]Although the p.Pro483Ser variant was confirmed to be paternally inherited, the p.Val282Leu variant could not be confirmed in either parent via standard Sanger sequencing. On the basis of multiple occurences in the literature of the two variants on separate chromosomes, these variants were reported to confer disease risk in the newborn in this study. The need to confirm their phase in the newborn by parental testing using targeted *CYP21A2* long-range PCR assay was explained in the newborn's nGS report.

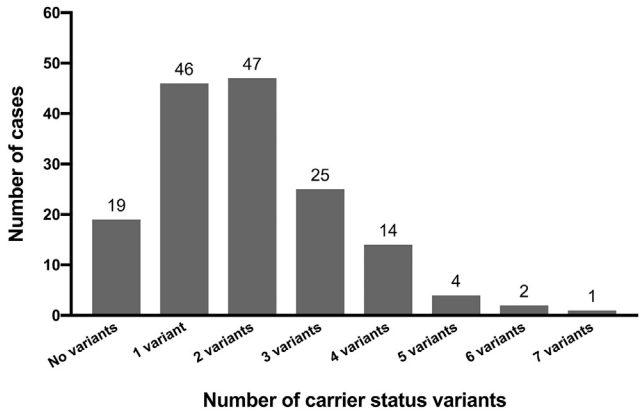

Figure 1. Number of Carrier-Status Variants Reported per Newborn

Number of carrier-status variants reported per newborn is displayed. The numbers above the bars represent the number of newborns who had the specified number of carrier-status variants reported.

alternatively spliced in cardiac tissue.[25] Furthermore, two of the *TTN* variants have been previously reported in patients with dilated cardiomyopathy (see Table S1 for detailed variant interpretations).

For analysis of the risk of adult-onset disease, variants in five genes associated with conditions that are medically actionable during adulthood, have at least moderate penetrance, and are amenable to testing by WES were also assessed: *BRCA1, BRCA2, MLH1, MSH2*, and *MSH6*. Two infants had pathogenic *BRCA2* variants associated with increased risk for breast, ovarian, prostate, and pancreatic cancers,[26] and one infant had a *MSH2* variant pathogenic for Lynch syndrome (MIM: 120435), characterized by elevated risk for colorectal, endometrial, gastric, ovarian, and other cancers.[27]

None of the infants who were found to be at risk for childhood-onset disease were known to be affected with these conditions at the times of enrollment or interpretation of the nGS results. 14 newborns were heterozygous for AD-condition-associated variants (in *BRCA2, CD46, ELN, GLMN, KCNQ4, MSH2, MYBPC3, SLC7A9, TTN*, and *VCL*) inherited from parents who reported themselves as healthy during pre-enrollment genetic counseling. Therefore, we classified variants in the absence of relevant clinical or family histories in the subjects, and we assessed the available evidence from the literature to determine whether the absence of a phenotype in the newborn period and/or absence of family history of disease would exclude a pathogenic role for these variants. Two genes, *KCNQ4* and *GLMN,* are reported to have high penetrance; however, the age of onset and severity of *KCNQ4*-related hearing loss might be variable, and glomuvenous malformations might appear later in life and express only as single small lesions, which could be missed without focused clinical examination.[12,13] The other genes are known to have moderate or age-dependent penetrance and variable expressivity. Therefore, the identification of variants in these genes in reportedly healthy newborns and parents did not exclude a pathogenic role for these variants.

Carrier Status

At least one variant conferring carrier status for a recessive childhood-onset disorder[8] was identified in 140 of 159 (88%) newborns in the nGS group, and the median was two variants per newborn (Table S2). The number of carrier-status variants ranged from one variant each in 46 newborns to seven variants in a single newborn (Figure 1). Of the 310 variants reported for carrier status, 225 (73%) were identified only once in our cohort. The most common genes and variants returned for carrier status are listed in Table 2. Eleven genes and five specific variants were reported in more than three newborns each (Table 2). The most frequently identified variant was the c.1330G>C (p.Asp444His) variant in *BTD*. This pathogenic variant was detected in 15 newborns, including one homozygote. The p.Asp444His variant is predicted to cause a ~25% reduction in biotinidase activity, such that heterozygotes have 75% of normal activity and homozygotes have 50% of normal activity; the latter is similar to that in heterozygotes for a severe variant. This variant has been reported to cause partial BTD when in *trans* with a severe *BTD* variant[28,29] and therefore was reported for carrier status in both heterozygotes and homozygotes. The genes that were most frequently reported for carrier status in our study are known to have high carrier frequency in the general population;[30–33] the exception was *RBM8A,* which is not commonly tested for in routine carrier screening. The *RBM8A* (MIM: 605313) c.−21G>A (p.?) variant has only been reported in *trans* with variants that are expected to cause complete loss of *RBM8A* function in individuals with thrombocytopenia and absent radius (TAR [MIM: 274000]) syndrome;[34,35] Such variants are extremely rare in the general population (gnomAD, see Web Resources). Therefore, although the c.-21G>A variant is common in the population (detected in ~2.8% of European chromosomes in gnomAD), the reproductive risk is low for carriers of this variant.

Although it is unlikely that carrier-status variants would impact phenotypic expression, certain variants associated with AR disorders have been associated with symptoms in the heterozygous state in rare cases of so-called "manifesting heterozygotes." Out of the 140 newborns who were identified as carriers, six (4.3%) were heterozygous for variants that have been previously associated with mild presentations in carriers (Table S2). These include two female infants with *G6PD* variants and one female infant with an *F8* (MIM: 300841) variant associated with X-linked recessive hemophilia A (MIM: 306700); these variants might lead to mild phenotypes in females with skewed X inactivation, although carrier females for these disorders are typically not affected.[36–38] Two newborns had pathogenic *DUOX2* (MIM: 606759) variants causative for congenital hypothyroidism (MIM: 607200)

Table 2. Common Variants Reported for Carrier Status in the BabySeq Project

Gene (Transcript)	Disease	Number of Newborn Carriers	Classification	Variant
Genes Reported for Carrier Status in More Than Three Newborns				
BTD	biotinidase deficiency	15[a]	–	–
RBM8A	thrombocytopenia with absent radius (TAR) syndrome	11	–	–
GJB2	GJB2-related nonsyndromic hearing loss	10	–	–
CFTR	cystic fibrosis	6	–	–
MUTYH	MUTYH-related attenuated familial adenomatous polyposis	6	–	–
ABCA4	Stargardt disease	5	–	–
DHCR7	Smith-Lemli-Opitz	5	–	–
TYR	oculocutaneous albinism type 1	5	–	–
ACADM	medium-chain acyl-CoA dehydrogenase (MCAD) deficiency	4	–	–
NPC1	Niemann-Pick disease type C	4	–	–
SI	congenital sucrase-isomaltase deficiency	4	–	–
Variants Reported for Carrier Status in More Than Two Newborns				
BTD (GenBank: NM_000060.2)	biotinidase deficiency	15[a]	pathogenic	c.1330G>C (p.Asp444His)
RBM8A (GenBank: NM_005105.4)	thrombocytopenia with absent radius (TAR) syndrome	8	likely pathogenic	c.−21G>A (p.?)
CFTR (GenBank: NM_000492.3)	cystic fibrosis	4	pathogenic	c.1521_1523delCTT (p.Phe508del)
GJB2 (GenBank: NM_004004.5)	GJB2-related nonsyndromic hearing loss	4	pathogenic	c.35delG (p.Gly12Valfs*2)
MUTYH (GenBank: NM_001128425.1)	MUTYH-related attenuated familial adenomatous polyposis	4	pathogenic	c.1187G>A (p.Gly396Asp)
CNGB3 (GenBank: NM_019098.4)	achromatopsia	3	pathogenic	c.1148delC (p.Thr383Ilefs*13)
DHCR7 (GenBank: NM_001360.2)	Smith-Lemli-Opitz	3	likely pathogenic	c.724C>T (p.Arg242Cys)
GJB2 (GenBank: GNM_004004.5)	GJB2-related nonsyndromic hearing loss	3	pathogenic	c.101T>C (p.Met34Thr)
RBM8A (GenBank: NM_005105.4)	thrombocytopenia with absent radius (TAR) syndrome	3	likely pathogenic	c.67+32G>C (p.?)

[a]Includes one subject homozygous for the p.Asp444His variant and one subject who had the c.511G>A (p.Ala171Thr) variant in *cis* with this variant.

in a bi-allelic state; these variants might lead to mild transient hypothyroidism in manifesting carriers, although this was reportedly not detected in NBS of the two carrier newborns identified in our study.[39,40] One newborn carried an *MYBPC3* c.3628−41_3628−17del variant that has been associated with increased risk for milder and late-onset cardiomyopathy in heterozygotes,[41] but it is also known to have severe effects in the homozygous state and therefore was classified as LP for early-onset AR cardiomyopathy. The late onset and low-penetrance risk in the heterozygous state was noted in the evidence summary. When variants of this type were reported in the carrier-status section of the report, information about the rare possibility of manifesting symptoms and the limited understanding of their penetrance and expressivity in carriers because of the absence of large numbers of phenotyped carriers and functional studies was included in the evidence description.

For genes that are associated with both AD and AR disorders, individuals with monoallelic pathogenic variants might be at risk for one disease while being a carrier for another disease. Three newborns had *TTN* variants that

were classified as P/LP for AD cardiomyopathy,[42] and these variants were also likely pathogenic for AR centronuclear myopathy.[43] Therefore, they were described as conferring both risk for cardiomyopathy and carrier status for centronuclear myopathy, and they were reported in both sections of the NGSR. In contrast, the more mildly manifesting carrier variants were only reported in the carrier section.

Although the prior probability of identifying bi-allelic pathogenic variants that confer disease risk in a well newborn is extremely low, it should be kept in mind that a second, pathogenic variant in genes where a monoallelic variant is identified cannot be ruled out by GS, particularly when the associated phenotypes are expected to present later in life. First, only SNVs and small insertions and deletions are reliably detected, and GS might miss other types of pathogenic variation, such as copy-number events, larger indels, or repeat variation. GS also has limited utility for genes that have high homology with pseudogenes or other regions and therefore require other targeted assays for reliable testing. Second, many genes might have incomplete coverage in GS. Among the 310 variants reported for carrier status in our study, 168 (54%) resided in genes with 100% coverage of the target exonic and splice (+/−1,2) regions, whereas 46% had reduced coverage ranging from 59.6% to 99.9% (average 92.7%) (Table S2). Additionally, the clinical significance of many variants remains uncertain. 8 of 140 (6%) newborns with carrier-status variants also had a VUS in one of the reported carrier genes (*PCNT* [MIM: 605925], *MMACHC* [MIM: 609831], *G6PD, MUTYH* [MIM: 604933], *SLC22A5* [MIM: 603377], *TTN, DYNC2H1* [MIM: 603297], and *USH2A* [MIM: 608400)] data not shown). In a healthy adult carrier of a highly penetrant recessive disease variant, a second variant detected in *trans* with a pathogenic variant in the same gene is considered to be benign if the individual does not have any symptoms of the associated disease. However, because the features associated with many recessive disorders are not apparent at birth, interpreting the clinical significance of second variants identified in the carrier genes is more challenging in newborns. In our project, we are continuing to explore whether any of the carrier-status findings might have revealed disease risk due to missing a second pathogenic variant in the gene. However, the frequency of pathogenic variants in these genes, as it is for most monogenic recessive disease genes, is low; therefore, the probability that they have a second pathogenic variant remains very low.

Indication-Based Analyses

At the time of enrollment, an indication-based analysis (IBA) was requested for 29 newborns in the NICU cohort for the following presentations: congenital heart defects (CHDs) (11 newborns), multiple congenital anomalies (11 newborns), severe lung disease, encephalopathy, laryngomalacia, congenital anemia, hemivertebrae, esophageal atresia, and anorectal malformation (one newborn each, Table 3). For three newborns with clinical diagnoses of prematurity (two newborns) and neonatal pneumonia due to meconium aspiration (one newborn), the study physicians did not request an IBA.

For the IBAs, genes that have been associated with the newborns' reported clinical features, (including those with limited evidence for disease association and/or low penetrance), were identified, and all variants in these genes were reviewed to assess their clinical significance, as well as relevance to the newborns' indications. The number of genes specifically interrogated in the IBAs ranged from one (*TBX6* [MIM: 602427], for an IBA of hemivertebrae) to 758 (for an IBA of liver disease); the median was 106 genes per analysis.

WES did not identify any variants that unequivocally explained the indications in newborns from the NICU cohort. Inconclusive results, including VUSs in genes that might be related to the indication or monoallelic variants in genes associated with AR conditions, were identified in 5 of 29 (17%) infants (Table 3). Three newborns with CHDs, one of whom also had cryptorchidism, were heterozygous for VUSs in genes associated with AD CHDs. These variants were inherited from parents who did not report having CHDs; however, because of the incomplete penetrance of the phenotypes associated with these genes, the clinical significance of the variants remained uncertain. One newborn with a possible diagnosis of VACTERL (vertebral defects, anal atresia, cardiac defects, tracheo-esophageal fistula, renal anomalies, and limb abnormalities) association and hydrocephalus was heterozygous for a VUS in *FANCE* (MIM: 613976), a gene associated with AR Fanconi anemia (MIM: 600901), which shares some features with VACTERL. Finally, a newborn with encephalopathy was heterozygous for a pathogenic variant in *GLDC* (MIM: 238300), a gene that can cause AR glycine encephalopathy (MIM: 605899). No second variant was identified in *FANCE* or *GLDC* in these newborns, reducing the likelihood that variants identified in these genes were relevant for their phenotypes, although copy-number variants could not be detected by our test. An infant who had an anteriorly displaced anus had a likely pathogenic *de novo ANKRD11* variant identified in the NGSR analysis; this variant, was reported in the Monogenic Disease Risk section of the report rather than in the Indication-Based Analysis results, as described above. The *ANKRD11* gene was not known to be associated with the reported phenotype of the infant at the time of analysis and therefore was not included in the 45 genes that were thought to be potentially related to anorectal malformations and were targeted in the IBA. However, this variant was later considered to be relevant on the basis of the recent association of the gene with this feature.

Additionally, 6 of the 127 newborns (5%) in the well-baby cohort had indications that prompted an IBA during the course of our study. For two of them, an IBA was requested at the time of enrollment on the basis of the

Table 3. Results of Indication-Based Analyses

Sex	Ethnicity/Race	Indication	Day IBA Ordered[a] (DOL)	Number of Genes Analyzed	Result	Gene (Transcript)	Variant(s) (Classification) (Zygosity)	Disease (Inheritance)	Penetrance
Well-Baby Cohort									
f	white	bilateral hip dysplasia	47	52	neg	–	–	–	–
f	white	atrial septal defect (PDA)	4	94	neg	–	–	–	–
m	multi-racial	hyperbilirubinemia (DOL 4–6)	90	103	neg	–	–	–	–
f	white	ventricular septal defect	7	97	neg	–	–	–	–
f	multi-racial	cavernous malformation	400	102	neg	–	–	–	–
f	white	liver disease	212	758	neg	–	–	–	–
NICU Cohort									
f	white	hypoplastic left heart	–	93	VUS	*NKX2-5* (GenBank: NM_004387)	c.111G>A p.Leu37Leu (VUS) (het)	congenital heart disease (AD)	unknown
m	white	multiple congenital anomalies with possible diagnosis of VACTERL w/ hydrocephalus	–	148	VUS	*FANCE* (GenBank: NM_021922)	c.1331T>C p.Leu444Pro (VUS) (het)	Fanconi anemia (AR)	high
m	native Hawaiian or other Pacific Islander	aortic coarctation	–	93	VUS	*NOTCH1* (GenBank: NM_017617)	c.4880G>A p.Arg1627His (VUS) (het)	congenital heart disease (AD)	unknown
m	white	tetralogy of Fallot, pulmonic stenosis, and cryptorchidism	–	356	VUS	*NOTCH1* (GenBank; NM_017617)	c.4168C>A p.Pro1390Thr (VUS) (het)	congenital heart disease (AD)	unknown
m	white	encephalopathy	–	459	inc[b]	*GLDC* (GenBank: NM_000170)	c.128delA p.Asp43Alafs* 48 (P) (het)	glycine encephalopathy (AR)	high
f	white	multiple congenital anomalies including TOF, pulmonary stenosis, TET spells, duodenal atresia, anteriorly displaced anus, and failure to thrive	–	142	neg	–	–	–	–
f	white	Pierre Robin sequence (micrognathia, cleft palate, glossoptosis), hooded eyes, tubular nose	–	266	neg	–	–	–	–
f	white	hemivertebrae	–	1	neg	–	–	–	–
m	white	double outlet right ventricle, atrioventricular canal defect, recurrent respiratory infections, laryngomalacia, enterocolitis, hypocal–cemia, short stature	–	0	neg	–	–	–	–
m	white	hypoplastic left heart	–	94	neg	–	–	–	–

(*Continued on next page*)

Table 3. ***Continued***

Sex	Ethnicity/Race	Indication	Day IBA Ordered[a] (DOL)	Number of Genes Analyzed	Result	Gene (Transcript)	Variant(s) (Classification) (Zygosity)	Disease (Inheritance)	Penetrance
f	white	tetralogy of Fallot with absent pulmonary valve	–	94	neg	–	–	–	–
f	white	anteriorly displaced anus (anorectal malformations)	–	45	neg	–	–	–	–
m	white	hypoplastic left heart	–	93	neg	–	–	–	–
f	white	dextrotransposition of the great arteries	–	93	neg	–	–	–	–
f	white	tricuspid atresia and ventricular septal defect	–	93	neg	–	–	–	–
f	multi-racial	respiratory distress (surfactant deficiency) and hypoglycemia	–	169	neg	–	–	–	–
m	white	hypoplastic left heart	–	93	neg	–	–	–	–
m	white	transposition of great arteries	–	106	neg	–	–	–	–
f	white	interstitial lung disease and facial dysmorphia	–	366	neg	–	–	–	–
m	white	liver disease, thrombocytopenia/anemia, hyperbilirubinemia, and hypoglycemia	–	224	neg	–	–	–	–
f	unspecified	congenital severe chronic lung disease	–	387	neg	–	–	–	–
f	white	aortic coarctation and ventricular septal defect	–	93	neg	–	–	–	–
f	unspecified	flat facial profile, preauricular pits, macroglossia, and hemangioma	–	109	neg	–	–	–	–
m	white	encephalopathy and hemangioma	–	247	neg	–	–	–	–
m	multi-racial	single ventricle, double inlet left ventricle with normally related great arteries	–	186	neg	–	–	–	–
m	white	laryngomalacia	–	79	neg	–	–	–	–
f	white	hypoglycemia and large for gestational age	–	110	neg	–	–	–	–
f	white	congenital anemia	–	400	neg	–	–	–	–
f	white	esophageal atresia with tracheoesophageal fistula	–	251	neg	–	–	–	–

Abbreviations are as follows: m = male; f = female; DOL = day of life; neg = negative; VUS = variant of uncertain significance; inc = inconclusive; P = pathogenic; het = heterozygous; inh = inheritance; AD = autosomal dominant; and AR = autosomal recessive. [a]Day IBA ordered in the well-baby cohort.[b]Single pathogenic variant associated with AR disease.

Table 4. Reported Pharmacogenomic Variants

Gene (Transcript)	Variant	Drug	Dosing Information	Number of Newborns
DPYD (GenBank: NM_000110.3)	c.1905+1G>A (p.?)	fluoropyrimidines	decreased dose requirement	2
DPYD (GenBank: NM_000110.3)	c.2846A>T (p.Asp949Val)	fluoropyrimidines	decreased dose requirement	2
TPMT (GenBank: NM_000367.4)	c.460G>A (p.Ala154Thr)	thiopurines	decreased dose requirement	3
G6PD[a] (GenBank: NM_000402.3)	c.961G>A (p.Val321Met)	certain antimalarials such as primaquine; antibiotics such as quinolones and sulfonamides, and methylene blue. (See reference 68 and G6PD Deficiency Favism Association in Web Resources)	contraindicated	1

[a]Reported in Monogenic Disease Risk section of NGSR.

discovery of a patent ductus arteriosus and a ventricular septal defect. For the other four infants, an IBA was requested after enrollment for the following indications: bilateral hip dysplasia, hyperbilirubinemia (diagnosed at day of life [DOL] 4), abnormal liver function, and seizure resulting from bleeding of a cavernous malformation; the IBAs were requested at DOL 47, 108 (following nGS results disclosure), 212, and 400, respectively. No variants with a potential relationship to their indication were identified in these infants.

PGx Variants

Return of PGx results was limited to genes with substantial evidence of association with atypical responses to drugs that might be used in the pediatric population (Table 4). Variants identified in three genes were determined to be in this category: *DPYD* (MIM: 612779), *TPMT* (MIM: 187680), and *G6PD*. PGx variants identified in these genes were returned in 8 of 159 (5%) newborns who received nGS. Four newborns had *DPYD* variants associated with increased risk for toxicity from the use of fluoropyrimidines and therefore with a decreased dose requirement for these medications. Three newborns had *TPMT* variants associated with higher risk of life-threatening myelosuppression when treated with standard doses of thiopurines and therefore had a decreased dose requirement for thiopurines. The *G6PD* variant was associated with G6PD deficiency and was identified in a hemizygous male infant. It was returned in the disease risk section of NGSR because the disease could be triggered by factors other than medications, as well. The PGx association for this variant was described in the variant summary.

Parental Sample Testing to Help Interpret nGS Results

Interpreting nGS results has unique challenges because of the absence of a phenotype in newborns for non-congenital diseases. To help interpret and communicate nGS findings, we sometimes tested parental samples to establish phase, assess for *de novo* occurrence, and otherwise clarify the significance of variants and/or explain familial risk to the parents. During the course of the project, 37 variants identified in NGSR analysis of 28 newborns were tested in parental samples. 16 P/LP variants conferring disease risk (AD and X-linked) and four carrier-status variants for which adult carriers could present symptoms were tested for in parents so that the associated disease risk could be better interpreted and communicated to the families. For 17 other variants, parental testing results contributed to the decisions made for whether and how to report variants by helping us to determine the variants' clinical significance, phase, and/or mode of inheritance. Seven P/LP variants in genes that have been associated with both AD and AR modes of inheritance (in *BEST1* [MIM: 607854], *COL6A2* [MIM: 120240], *GLRA1* [MIM: 138491], *MYH7* [MIM: 160760], *RNASEH2B* [MIM: 610326], *TECTA* [MIM: 602574], and *VWF* [MIM: 613160]) were tested so that their inheritance pattern could be determined. These variants either were novel truncating variants or had been reported in both heterozygous and homozygous or compound-heterozygous affected individuals in the literature, and their identification in healthy parents was considered to be evidence supporting a recessive mode of inheritance, favoring the decision to report these for carrier status. Seven P/LP variants in AR *BTD* and *CYP21A2* were tested for phasing, and their allelic states were reported accordingly. Three VUSs identified in NGSR analyses were tested so that their clinical significance could be clarified, and their identification in a healthy parent was considered evidence in support of a benign role. Variants in *EXT2* (MIM: 608210) and *RB1* (MIM: 614041), associated with highly penetrant AD disorders, and in a female newborn, a paternally inherited *BRWD3* (MIM: 300553) varaint (associated with an X-linked recessive disorder), were classified as VUSs on the basis of their identification in healthy parents and other lines of evidence, and they were excluded from the NGSRs. Three VUSs identified in IBAs (described above) were also tested in parents; however, because they were in genes associated with moderate penetrance and/or variable expressivity, their identification in reportedly healthy parents did not alter their classification. Overall,

parental testing contributed to determining whether or how a variant was reported in 13 of 159 (8%) of the newborns who received nGS and helped with interpretation and communication of nGS results in a total of 28 of 159 (18%) of the newborns.

Discussion

Because of its potential to target a wide range of disorders for screening and diagnostic purposes with a single test, nGS can be a powerful tool for improving the future healthcare of infants. However, the application of newborn sequencing poses several challenges, including how to interpret variants associated with conditions that might not be apparent in the infant at the time of testing and the potential costs and psychosocial impacts.[44] Our data from 159 newborns sequenced in the BabySeq Project help illustrate the range of situations that might arise from nGS. They also highlight factors that need to be considered for the interpretation and reporting of nGS results, including the age of onset, penetrance, and inheritance patterns of identified variants, and their relevance to the clinical and family histories of the newborns at the time of analysis.

Our study had several limitations. First, we had a small cohort size, which was particularly limited for the NICU group. Second, the fact that participants were randomized to either receive or not receive nGS in our study might have discouraged parents of ill newborns who could receive diagnostic nGS clinically or as part of another non-randomized study. This might have created a self-selection for parents whose newborn was less likely to receive GS in other settings on the basis of their phenotype and therefore might have created an enrichment of phenotypes that were less likely to benefit from nGS. Additionally, our proband-only sequencing approach using phenotype-driven gene filtering had limited ability to detect *de novo* variants in genes that were recently described or had limited association with the infant's indication at the time of our analysis.

The prior probability of a genetic disorder is assumed to be low in healthy newborns. However, nGS identified risk for childhood-onset diseases in 9.4% and risk for actionable adult-onset diseases in 3.5% of the newborns sequenced in the BabySeq Project. Eleven newborns had variants that were expected to have moderate penetrance or variable expressivity on the basis of previous reports in the literature, but these variants were considered as medically actionable during childhood. These include seven newborns who were discovered to have risk for cardiomyopathies or SVAS, for which increased surveillance by regular echocardiograms and EKGs might allow timely interventions that would significantly reduce the risk for heart failure and sudden cardiac death.[45–47] Knowledge about risk for these conditions could also allow informed clinical and lifestyle choices (such as participation in sports or the use of stimulant medications) to further reduce the risk for devastating events.[48,49] Other conditions identified in our cohort, such as aHUS, G6PD deficiency, and cystinuria could also benefit from avoidance of precipitating factors.[50–52] Because many of the conditions for which we have detected risk might have incomplete penetrance, later onset, and/or uncertain immediate medical actionability, it is possible that identifying their risk later in life rather than during the newborn period might also be beneficial for health outcomes. This might also avoid the possibility of negative psychosocial implications or increased medical interventions and healthcare costs when detected within the first days of life. On the other hand, in the absence of a significant family history, a genomic screening approach might be the only setting where an individual's risk gets identified before any symptoms arise. The results of our study and other studies on the use of GS in newborns and other populations will help develop best practices for the optimal timing and application of such a screen in an individual's life.

None of the disease risk findings were predicted on the basis of known clinical and family histories of the newborns at the time of testing. Our results prompted follow-up studies to search for evidence of disease and/or family history that was not appreciated during enrollment. After the disclosure of the nGS results, the parents of three infants expressed that they had a family history of the disease for which their newborn was identified to be at risk (a grandparent of a newborn with *TTN* variant had dilated cardiomyopathy, a grandparent of a newborn with *KCNQ4* variant had hearing loss, and a parent of a newborn with *BRCA2* variant had family history of breast cancer). Clinical follow-up with the infants and their parents harboring the disease-risk variants is ongoing so that clinicians can assess whether there are any symptoms of disease. Because many of the genes we detected are known to have incomplete penetrance or might present later in life with variable expressivity, the absence of a phenotype or family history in the parents did not exclude a pathogenic role for the variants, although it was informative to predict the likelihood of disease in the newborns who had these variants.

Interestingly, P/LP variants in genes related to cardiomyopathies and SVAS were found in 7 of 159 (4%) newborns, a rate that is higher than the known prevalence of these conditions in the general populationand which emphasizes the incomplete and age-dependent penetrance of these conditions. The *ELN* and *MYBPC3* variants were classified as pathogenic on the basis of a truncating effect or segregation in multiple families, respectively (Table S1). Truncating *VCL* variants, such as the one identified in our study, are currently considered likely pathogenic for DCM on the basis of their identification and segregation in affected families (LMM internal data, Table S1), although additional studies are needed to clarify this gene's penetrance. Four newborns had truncating *TTN* variants that were classified as P/LP for DCM. Two of these variants have previously been reported in multiple DCM patients

and/or have been found to segregate with disease in affected family members (Table S1), providing further support for their pathogenicity. *TTN* truncating variants are prevalent in the general population,[24,53] which makes it challenging to interpret their clinical significance. It has been demonstrated that truncating variants in control individuals were more likely to affect minor *TTN* isoforms and occur in alternatively spliced exons, whereas those in constitutively expressed exons are enriched in DCM patients as compared to controls.[24,53–56] Two truncating *TTN* variants identified in our study (p.Pro4115Glnfs*14 and p.Met11632Serfs*8) have not been previously reported in individuals with DCM, and although they are located in the I-band, where alternative splicing occurs frequently, the exons they are located in have been demonstrated to be not alternatively spliced in cardiac tissue.[24] These variants were classified as LP for DCM on the basis of the current best practice of classifying as LP the truncating *TTN* variants located in exons that are not alternatively spliced.[25] The penetrance of *TTN* truncating variants has been demonstrated to be ~60% in a study of family members of affected individuals,[56] although it is possible that the penetrance might vary depending on the location of these variants. Analyses in larger cohorts are needed to clarify the penetrance of truncating *TTN* variants located in various regions of this gene.

Our results suggest that nGS might also expand the detectable phenotypic spectrum of disorders that are targeted by current NBS, although the identification of these conditions at birth might or might not provide additional benefit. In three newborns who passed NBS, nGS identified risk for NBS-targeted conditions (hearing loss, BTD and CAH). Postlingual hearing loss due to *KCNQ4* variants is not expected to be detected by audiological screening at birth. However, recognizing early stages of hearing loss in children is challenging and can often delay diagnosis and interventions. Information about this risk could allow additional vigilance and screening to provide timely interventions and reduce its impact on the child's development and social skills, particularly if the onset of hearing loss is during childhood. Detecting risk for later-onset hearing loss in presymptomatic individuals might have less significance for the individual's health and quality of life. Partial BTD identified on the basis of the nGS results might be missed in NBS, as it was in our subject, although it might be clinically significant, particularly at times of stress. Although many individuals with partial BTD might not experience any symptoms throughout their lifetime and detecting partial BTD in the newborn period might not be critical, symptoms can effectively be prevented with a simple and inexpensive treatment of biotin supplementation, as was prescribed in this case.[16] Finally, NBS rarely detects nonclassic CAH.[57] Identifying individuals at risk for nonclassic CAH might be beneficial for facilitating early diagnosis and therapies, if needed, although many individuals with this condition might not need treatment. Therefore, our results serve as a reminder that negative NBS results do not rule out pathogenic variants in genes associated with NBS conditions, and they suggest that nGS might identify individuals with milder or later-onset phenotypes of NBS conditions, whose detection might not be as critical in the newborn period.

Currently, there is ongoing debate about whether adult-onset disease risk should be returned to children and whether nondisclosure of particularly actionable adult-onset disease risk might do more harm to the children and families.[58–60] In our study, families were offered the option to receive information regarding risk for medically actionable adult-onset conditions in their infant. Three of 85 (3.5%) infants whose parents consented to receive this information harbored pathogenic variants associated with adult-onset conditions for which early knowledge leading to increased surveillance and preventative treatments might be lifesaving.[26,27] These variants were also identified in the mothers of the three children, and early interventions based on this knowledge might also have lifesaving consequences for the child's parent, which undoubtedly could impact the child's quality of life. The risks and benefits of returning adult-onset disease risk to children will continue to be discussed on the basis of the results of studies that address this question.[5]

Recent studies using GS in adult cohorts reported a rate of 3%–5.6% for secondary findings in the ACMG59[11] genes or in other small groups of actionable genes determined by the authors.[61–64] Our reporting criteria were much broader and included a higher number of genes to be returned for disease risk findings (>900 genes met criteria for reporting in our initial curation efforts).[8] In our study, four newborns had a disease risk variant in one of the ACMG59 genes: three of those were in adult-onset disease genes (*BRCA2* and *MSH2*) and one (*MYBPC3*) was considered a childhood-onset disease gene. This corresponds to a combined rate of 4.1% ([3/85] + [1/159]), a rate that is similar to the rate of incidental findings in ACMG59 genes reported in adult cohorts.[61–63]

nGS also allows detection of carrier status for a wide range of disorders that are not included in currently available expanded carrier screening panels. We identified at least one carrier-status variant in 88% of the newborns and up to seven variants per subject. The majority (73%) of these variants were identified only once in our cohort, suggesting that returning only common pathogenic variants with high frequency in the general population would miss the majority of carriers for childhood-onset diseases. In addition, each of these novel variants required considerable manual curation to determine their clinical significance, implying that returning carrier status in nGS would significantly increase the amount of work done by clinical laboratories; this increase might impact the turnaround time and cost for reporting nGS results. Carrier-status information is mostly relevant for future reproductive planning for the infant and the parents because genetic testing in the newborn nearly always reveals variants that are also carried by one of the parents. An estimate of a couple's reproductive risk can

be provided on the basis of assumptions that (1) the parents are not related and (2) the probability for the other parent to be a carrier for the same gene is equal to the gene's known or estimated carrier frequency in their ethnic subpopulation. However, to determine the actual reproductive risk, couples might want to pursue carrier testing for both the identified variants and subsequently the full gene in the non-carrier parent. Effectively determining carrier status in the parents would require targeted gene tests in the case of many genes that have been identified for carrier status in our cohort; such tests might have varying availability in clinical laboratories and therefore pose a challenge for future reproductive planning. Our study continues to review what portion of our participants are pursuing such targeted carrier testing on the basis of our nGS results and the outcomes of these tests to assess the utility and impact of returning carrier-status information in nGS.

nGS has recently been shown to have a high clinical yield in critically ill newborns who had been admitted to a NICU and were suspected of having a genetic disorder.[3,4] In contrast, we observed a lower rate of positive findings in our NICU cohort. Several reasons might account for this difference. First, the NICU patients in our study were not pre-selected for a suspected genetic disorder but were chosen with minimal exclusion criteria to represent a more general population. Additionally, our randomized study design might have led to some self-selection of families whose newborns had a lower likelihood of benefitting from nGS. Because our participants had a 50% probability of receiving nGS, it is possible that parents of newborns who could receive diagnostic GS as part of their standard care or in a non-randomized nGS study might have had less interest in enrolling in the BabySeq Project. In our study, 22 of 29 ill newborns (76%) had nonsyndromic congenital heart defects or multiple congenital anomalies. Monogenic diseases usually have a small contribution to these conditions, and they might also frequently be explained by chromosome abnormalities and structural alterations that are not reliably detected by WES.[65–67] Although our study design might have led to self-selection of NICU newborns who were less likely to have a monogenic disease etiology, our results suggest that the diagnostic yield of nGS might depend on the phenotype of the subjects. Finally, we performed proband-only GS and used a phenotype-driven gene-filtering approach focusing on genes with known association with the infant's features, a method that is limited to detecting *de novo* and other variants in genes that had recent or limited association with the disease of interest at the time of our analysis. The aim of our study was to explore the use of singleton nGS in newborn care, although trio sequencing is known to have a higher clinical yield for a wide range of indications.[3,68] Our identification of a likely pathogenic *de novo* variant in *ANKRD11* in the NGSR analysis as opposed to the IBA highlights the limitations of performing phenotype-driven gene and/or variant assessments and of proband-only sequencing. This variant was detected as a novel LOF variant in a disease gene and was initially interpreted as an incidental finding; therefore, it was reported under the Monogenic Disease Risk section rather than IBA results and was later thought to be diagnostically relevant in light of recent studies. Although all variants in genes known to be relevant for the indication were analyzed in our IBAs regardless of their reporting status and predicted impact on the protein, because *ANKRD11* was not known to be associated with anorectal malformations at the time of our analysis, it was not included in the phenotype-driven gene list for the IBA of this infant. In the absence of their known association with the infant's phenotype, VUSs in this gene would not have been captured or returned in our study, and a clinically relevant variant could be missed.

In addition to discovering short-term disease risk and diagnosing existing but clinically unsuspected disease, nGS allows genomic information that can be specifically interrogated for new indications and inform personalized medicine applications to be accessible throughout an individual's lifetime. During the course of our project, 5% of infants enrolled from the well-baby nursery developed an indication that prompted an IBA. Because our subjects are currently between 8 months and 3 years old, it is likely that additional participants will eventually develop indications that benefit from genomic IBAs. Although GS might not be the most appropriate test for all presentations, an IBA on already available GS data will be a rapid, first-tier approach when gene sequencing is indicated and can be supplemented or followed by additional tests. An IBA for a new presentation in an individual who has already been sequenced will allow for the review of genes that are recently associated with the disease and for the full assessment of all variants in relevant disease genes. Other uses of nGS data in a newborn's future life might include analyses for a wide range of adult-onset disease risk, polygenic risk estimates for complex traits, and PGx for drugs used in the adult population.

Having access to the known clinical and family histories of our subjects, as well as to samples from their parents, was invaluable for the interpretation of nGS results in our study. Parental sample testing, when available, is frequently performed in diagnostic GS; however, its utility for interpreting nGS findings in a screening setting has not, to our knowledge, been previously addressed. Results of parental testing in light of provided family histories helped determine whether and how a variant was reported for 8% of our participants; all of these variants were part of the NGSR analyses directed to screening purposes. Although obtaining detailed clinical and family history information might be challenging in a population-wide application of nGS, the collection of parental samples and informing laboratories of existing or newly diagnosed conditions in the newborn and family members should be performed to help better interpret nGS results.

In summary, we present our nGS findings from 159 newborns sequenced in the BabySeq Project. Although detecting disease risk for many actionable early-onset conditions would be beneficial in improving health outcomes,

potential healthcare costs and psychosocial impacts need to be considered in the development of best practices for nGS. Our study continues to explore the medical, behavioral, and economic impacts of our nGS reports on the basis of medical observations and post-disclosure surveys in parents and clinicians. As these newborn cohorts age, future analyses of economic and healthcare utilization patterns in our nGS and control cohorts will allow for the eventual assessment and quantification of both costs and benefits of GS in the newborn setting. The results from our study, as well as future efforts to prospectively analyze the long-term implications of nGS in larger cohorts, will help inform the effective and responsible application of nGS in wider medical practice.

Accession Numbers

The data/analyses reported in this paper have been deposited in the NBSTRN LPDR under accession identifier nbs000002.v1.p1.

Supplemental Data

Supplemental Data include two tables and can be found with this article online at https://doi.org/10.1016/j.ajhg.2018.11.016.

Consortia

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Declaration of interests

Dr. Green receives compensation for consultation from AIA, Helix, Ohana, Prudential, and Veritas, and is co-founder, advisor, and equity holder in Genome Medical, Inc. The remaining authors declare no competing interests.

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Web Resources

OMIM, http://www.omim.org/
G6PD Deficiency Favism Association, https://www.g6pd.org
gnomAD, http://gnomad.broadinstitute.org
NBSTRN LPDR, https://nbstrn.org/research-tools/longitudinal-pediatric-data-resource

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Supplementary Materials and Methods

Sequencing

Exome sequencing was performed on peripheral blood-derived genomic DNA at the Clinical Research Sequencing Platform of The Broad Institute of Harvard and MIT. DNA was hybridized to the Illumina Content Exome (ICE) probe set and next generation sequencing was performed on the Illumina HiSeq platform. Exomes were sequenced to at least 100X mean coverage and a minimum of 90% of bases were sequenced to at least 20X coverage. Paired-end reads were aligned to the NCBI reference sequence (GRCh37) using BWA and variant calls were made using GATK. Variants were subsequently filtered to identify: (1) variants classified as disease causing mutations in public databases (pathogenic or likely pathogenic in ClinVar or disease-causing mutations (DM) in the Human Gene Mutation Database (HGMD)) that had a minor allele frequency ≤3% in European American or African American chromosomes from the NHBLI Exome Sequencing Project (ESP); (2) novel or reported nonsense, frameshift, and +/- 1,2 splice site variants that have a minor allele frequency <1% in European American or African American chromosomes from the NHBLI ESP in over 4,000 genes that have been implicated in disease.

Supplementary Table S2: Example genes in Category B

	Gene	Disease	Reason the gene is included in Category B
Genes that have moderate evidence or penetrance for which noninvasive interventions during childhood may prevent a devastating outcome	*MYBPC3*	Hypertrophic cardiomyopathy	*MYBPC3* has definitive evidence for a causal role in hypertrophic cardiomyopathy, which may present during childhood [1-8]. This gene, like many other genes associated with inherited cardiomyopathies, has moderate penetrance such that an individual with a pathogenic *MYBPC3* variant may have ~50% risk for cardiomyopathy (penetrance may vary depending on the variant). Although the penetrance is only moderate, knowing this risk at birth may allow routine surveillance by echocardiography and noninvasive interventions when needed, which would provide tremendous benefit to reduce the risk for sudden cardiac death.
	SDHAF2	Hereditary paraganglioma	*SDHAF2* has been associated with hereditary paraganglioma that may present during childhood, but currently there is only a moderate level of evidence for this gene's role in disease [9-13]. Although the gene-disease association has not been fully established, because screening for cancer may prevent a devastating outcome, the benefits of having this information outweigh the disease risk uncertainty.
Genes that typically present in adulthood for which noninvasive intervention during childhood may	*CP*	Aceruloplasminemia	Pathogenic variants in *CP*, encoding ceroluplasmin, cause aceruloplasminemia. Symptoms result from iron accumulation in brain and viscera and is characterized by diabetes mellitus (DM), retinal degeneration anemia, and neurologic disturbances [14-17]. Anemia is often the

significantly improve the outcome			presenting symptom, but iron supplementation should be avoided. Annual glucose tolerance tests starting at 15 years of age are recommended for surveillance of DM onset [18]. Although the symptoms typically become apparent in adulthood, knowing disease risk may allow noninvasive interventions (avoidance of iron supplements to treat anemia and early surveillance for DM), which could significantly improve the clinical outcomes.

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Supplementary Table S3: Variants in genes that met NGSR criteria in the first 15 BabySeq cases

Variants that were returned in the first 15 BabySeq NGSRs					
Gene	**Disease**	**Variant**		**Zygosity**	**Classification**
		cDNA	**Amino acid**		
ABCA3	Surfactant metabolism dysfunction, pulmonary	c.875A>T	p.Glu292Val	Het	Likely pathogenic
ABCA4	Stargardt disease	c.2588G>C	p.Gly863Ala	Het	Pathogenic
ADAR	Aicardi-Goutieres syndrome	c.577C>G	p.Pro193Ala	Het	Likely pathogenic
BTD	Biotinidase deficiency	c.1330G>C	p.Asp444His	Het	Pathogenic
CFTR	Cystic fibrosis	c.1865G>A	p.Gly622Asp	Het	Likely pathogenic
CNGB3	Achromatopsia	c.1148del	p.Thr383IlefsX13	Het	Pathogenic
DHCR7	Smith-Lemli-Opitz syndrome	c.724C>T	p.Arg242Cys	Het	Likely pathogenic
DHCR7	Smith-Lemli-Opitz syndrome	c.964-1G>C	p.?	Het	Pathogenic
DOK7	Congenital myasthenia syndrome	c.1124_1127dup	p.Ala378SerfsX30	Het	Pathogenic
GYS2	Glycogen storage disease type 0	c.736C>T	p.Arg246X	Het	Likely pathogenic
IQCB1	Senior-Loken syndrome	c.1363C>T	p.Arg455X	Het	Pathogenic
MUTYH	MUTYH-associated polyposis	c.925-2A>G	p.?	Het	Likely pathogenic
PCCB	Propionic acidemia	c.337C>T	p.Arg113X	Het	Pathogenic
PCNT	Microcephalic osteodysplastic	c.7126C>T	p.Gln2376X	Het	Pathogenic

	primordial dwarfism type 2				
PKHD1	Polycystic kidney and hepatic disease	c.3766del	p.Gln1256Arg	Het	Pathogenic
RBM8A	Thrombocytopaenia-absent radius syndrome	c.67+32G>C	p.?	Het	Pathogenic
SLC26A4	Pendred syndrome	c.1003T>C	p.Phe335Leu	Het	Likely pathogenic
USH1G	Usher syndrome type 1	c.1373A>T	p.Asp458Val	Het	Likely pathogenic

Variants that did not meet criteria to be returned in NGSR

Gene	**Disease**	**Variant**		**Zygosity**	**Classification**
		cDNA	**Amino acid**		
ABCA4	Stargardt disease	c.6320G>A	p.Arg2107His	Het	Uncertain significance
ABCB11	Intrahepatic cholestasis, familial progressive 2	c.1435-13_1435-8del	p.?	Het	Uncertain significance
ABCC6	Pseudoxanthoma elasticum	c.4375C>T	p.Arg1459Cys	Het	Uncertain significance
ABCC6	Pseudoxanthoma elasticum, autosomal recessive	c.1171A>G	p.Arg391Gly	Het	Uncertain significance
ABCD1	Adrenoleukodystrophy	c.1816T>C	p.Ser606Pro	Het	Likely benign
ABCD1	Adrenoleukodystrophy	c.1823G>A	p.Gly608Asp	Het	Likely benign
ABCG5	Hypercholesterolaemia	c.80G>C	p.Gly27Ala	Het	Likely benign
ACSF3	Malonic & methylmalonic aciduria, combined	c.728C>T	p.Pro243Leu	Het	Uncertain significance
AGL	Glycogen storage disease 3	c.1481G>A	p.Arg494His	Het	Uncertain significance

AGXT	Hyperoxaluria	c.26C>A	p.Thr9Asn	Het	Benign
AIP	Pituitary adenoma	c.911G>A	p.Arg304Gln	Het	Uncertain significance
AIRE	APECED	c.652+14C>T	p.?	Het	Benign
ALOX12B	Ichthyosis, congenital, autosomal recessive	c.379C>T	p.Pro127Ser	Het	Benign
APOB	Apolipoprotein B deficiency	c.10520G>C	p.Arg3507Pro	Het	Uncertain significance
AR	Androgen insensitivity	c.173A>T	p.Gln58Leu	Het	Uncertain significance
ATM	Ataxia telengiectasia	c.2362A>C	p.Ser788Arg	Het	Likely benign
ATP1A2	Hemiplegic migraine	c.25T>A	p.Tyr9Asn	Het	Benign
ATP7B	Wilson disease	c.3355A>G	p.Ile1119Val	Het	Uncertain significance
ATP8B1	Intrahepatic cholestasis of pregnancy	c.607A>G	p.Lys203Glu	Het	Uncertain significance
BBS1	Bardet-Biedl syndrome	c.616T>G	p.Leu206Val	Het	Uncertain significance
BTD	Biotinidase deficiency	c.133G>A	p.Gly45Arg	Het	Benign
CACNA1F	Night blindness, congenital stationary, incomplete	c.1903G>A	p.Val635Ile	Het	Likely benign
CCDC40	Hemiplegic migraine	c.3040_3068del	p.Arg1014Glnfs*70	Het	Likely benign
CFTR	Cystic fibrosis	c.3705T>G	p.Ser1235Arg	Het	Likely benign
CFTR	Cystic fibrosis	c.1523T>G	p.Phe508Cys	Het	Uncertain significance
CFTR	Cystic fibrosis	c.1210-11T>G	p.?	Het	Likely

					pathogenic[§]
CLN6	Neuronal ceroid lipofuscinosis, late infantile	c.34G>A	p.Ala12Thr	Het	Benign
CLN6	Neuronal ceroid lipofuscinosis, late infantile	c.755G>A	p.Arg252His	Het	Uncertain significance
COL7A1	Epidermolysis bullosa dystrophica	c.6654C>G	p.=	Het	Benign
CREBBP	Rubinstein-Taybi syndrome	c.5933A>G	p.Asn1978Ser	Het	Benign
CYBB	Chronic granulomatous disease	c.142-12C>T	p.?	Hom	Likely benign
CYP11B1	Adrenal hyperplasia, congenital, due to 11-beta-hydroxylase deficiency	c.1120C>A	p.=	Het	Benign
CYP21A2	Adrenal hyperplasia	c.797C>T	p.Ala266Val	Het	Likely benign
DCLRE1C	Immunodeficiency, severe combined	c.1693dup	p.Arg565Lysfs*17	Het	Uncertain significance
DCLRE1C	Immunodeficiency, severe combined	c.97G>A	p.Gly33Arg	Het	Likely benign
DSP	Arrhythmogenic right ventricular dysplasia/ cardiomyopathy	c.4372C>G	p.Arg1458Gly	Het	Uncertain significance
ELANE	Neutropaenia, congenital	c.770C>T	p.Pro257Leu	Het	Benign
FAM161A	Retinal dystrophy	c.1133T>G	p.Leu378Arg	Het	Likely benign

FANCA	Fanconi anemia	c.3427C>G	p.Leu1143Val	Het	Uncertain significance
FOXC2	Lymphoedema, primary	c.1331A>G	p.Gln444Arg	Het	Uncertain significance
GALNS	Mucopolysaccharidosis IVa	c.499T>G	p.Phe167Val	Het	Uncertain significance
GBA	Gaucher disease	c.1223C>T	p.Thr408Met	Het	Likely benign
GCK	Diabetes mellitus	c.1016+18G>A	p.?	Het	Likely benign
GFPT1	Congenital myasthenic syndrome, limb-girdle	c.*22C>A	p.?	Het	Uncertain significance
JAK3	Immunodeficiency, severe combined	c.452C>G	p.Pro151Arg	Het	Benign
JAK3	Immunodeficiency, severe combined	c.2164G>A	p.Val722Ile	Het	Benign
KIT	Piebaldism	c.67+4G>A	p.?	Het	Benign
MKKS	McKusick-Kaufman syndrome	c.724G>T	p.Ala242Ser	Het	Uncertain significance
MKKS	McKusick-Kaufman syndrome	c.724G>T	p.Ala242Ser	Het	Uncertain significance
MSH2	Colorectal cancer, non-polyposis	c.1168C>T	p.Leu390Phe	Het	Likely benign
MSH6	Colorectal cancer	c.1526T>C	p.Val509Ala	Het	Likely benign
MYBPC3	Cardiomyopathy, hypertrophic	c.713G>A	p.Arg238His	Het	Uncertain significance
MYH2	Inclusion body myositis	c.2414T>C	p.Val805Ala	Het	Uncertain significance
NCF2	Chronic granulomatous disease	c.1256A>T	p.Asn419Ile	Het	Benign

NOG	Fibrodysplasia ossificans progressiva	c.275G>A	p.Gly92Glu	Het	Uncertain significance
NOTCH3	CADASIL	c.509A>G	p.His170Arg	Het	Likely benign
NPHP3	Nephronophthisis	c.154G>A	p.Ala52Thr	Het	Likely benign
NPHP4	Nephronophthisis	c.3131G>A	p.Arg1044His	Het	Uncertain significance
NPHP4	Nephronophthisis	c.3329C>T	p.Ala1110Val	Het	Uncertain significance
NPHS1	Minimal change nephrotic syndrome	c.881C>T	p.Thr294Ile	Het	Benign
OBSL1	3-M syndrome	c.4951G>T	p.Glu1651*	Het	Uncertain significance
PFKM	Glycogen storage disease 7	c.2300G>A	p.Arg767His	Het	Benign
PHYH	Refsum disease	c.734G>A	p.Arg245Gln	Het	Benign
PKD1	Polycystic kidney disease 1	c.11537+3_11537+5dup	p.?	Het	Benign
PKHD1	Polycystic kidney disease	c.9215C>T	p.Ala3072Val	Het	Likely benign
PKHD1	Polycystic kidney disease	c.8606C>A	p.Thr2869Lys	Het	Likely benign
PKHD1	Polycystic kidney disease	c.3407A>G	p.Tyr1136Cys	Het	Benign
PMM2	Congenital disorder of glycosylation 1a	c.590A>C	p.Glu197Ala	Het	Benign
PNKP	Epileptic encephalopathy	c.58C>T	p.Pro20Ser	Het	Benign
PROKR2	Hypogonadotropic	c.151G>A	p.Ala51Thr	Het	Benign

	hypogonadism				
PTPN11	Noonan syndrome	c.556C>T	p.Arg186Trp	Het	Uncertain significance
RET	Hirschsprung disease	c.833C>A	p.Thr278Asn	Het	Likely benign
SCN5A	Long QT syndrome	c.1844G>A	p.Gly615Glu	Het	Uncertain significance
SFTPB	Surfactant protein B deficiency	c.439G>A	p.Gly147Ser	Het	Uncertain significance
SLC12A3	Gitelman syndrome	c.965C>T	p.Ala322Val	Het	Uncertain significance
SLC12A3	Gitelman syndrome	c.2884-6G>A	p.?	Het	Likely benign
SLC3A1	Cystinuria	c.1035G>A	p.=	Het	Likely benign
SLC3A1	Cystinuria	c.797T>C	p.Phe266Ser	Het	Likely benign
SLC4A1	Spherocytosis	c.539G>A	p.Arg180His	Het	Uncertain significance
SLC5A2	Renal glucosuria	c.1961A>G	p.Asn654Ser	Het	Likely benign
STX11	Haemophagocytic lymphohistiocytosis	c.616G>A	p.Glu206Lys	Het	Uncertain significance
STXBP2	Hemophagocytic lymphohistiocytosis type 5	c.1034C>T	p.Thr345Met	Het	Likely benign
STXBP2	Hemophagocytic lymphohistiocytosis	c.795-4C>T	p.?	Het	Benign
TERT	Aplastic anaemia	c.1323_1325del	p.Glu441del	Het	Uncertain significance
TGM1	Ichthyosis, lamellar	c.125C>A	p.Ser42Tyr	Het	Likely benign
TSHR	Hypothyroidism	c.100G>A	p.Glu34Lys	Het	Uncertain significance

TTC21B	Meckel-Gruber-like syndrome	c.3004C>G	p.Leu1002Val	Het	Uncertain significance
TWIST1	Synostotic frontal plagiocephaly	c.259_276del	p.Ala87_Gly92del	Het	Uncertain significance
TWIST1	Synostotic frontal plagiocephaly	c.259_276del	p.Ala87_Gly92del	Het	Likely benign
UGT1A1	Gilbert syndrome	c.686C>A	p.Pro229Gln	Het	Likely benign
UMOD	Nephropathy	c.-14del	p.?	Het	Likely benign
VWF	Von Willebrand disease 1	c.3797C>T	p.Pro1266Leu	Het	Uncertain significance

Het, heterozygous; Hom, homozygous

[§]This variant was the 5T variant in intron 8 of *CFTR* gene and was in *cis* with 12TG repeats. The individual did not have the p.Arg117His variant. It was not reported in NGSR, because the phenotype in the majority of reported individuals who had this variant alone in *trans* with a CFTR pathogenic variant (infertility) did not meet NGSR criteria

Supplementary Table 1: Gene-disease association reference list

Gene	Curated disease	Evidence for gene-disease association
AAAS	Achalasia-addisonianism-alacrimia syndrome	Definitive
AARS	Charcot-Marie-Tooth disease	Strong
AARS2	Leukoencephalopathy, and ovarian failure in females	Moderate
ABAT	GABA-transaminase deficiency	Moderate
ABCA12	Ichthyosis, congenital, autosomal recessive	Definitive
ABCA3	Surfactant metabolism dysfunction, pulmonary, 3	Definitive
ABCA4	Stargardt disease	Definitive
ABCB11	Cholestasis, progressive familial intrahepatic 2	Definitive
ABCB4	Cholestasis, progressive familial intrahepatic 3	Definitive
ABCB7	Sideroblastic anaemia and ataxia	Moderate
ABCC2	Dubin-Johnson syndrome	Definitive
ABCC6	Pseudoxanthoma elasticum	Definitive
ABCC8	Hyperinsulinemic hypoglycemia, familial	Definitive
ABCC9	Atrial fibrillation, familial	Limited
ABCC9	Cardiomyopathy, dilated	Moderate
ABCC9	Hypertrichotic osteochondrodysplasia	Strong
ABCD1	Adrenoleukodystrophy	Definitive
ABCD4	Methylmalonic aciduria and homocystinuria, cblJ type	Moderate
ABCG5	Sitosterolemia	Definitive
ACAD8	Isobutyryl-CoA dehydrogenase deficiency	Definitive
ACAD9	ACAD9 deficiency	Strong
ACADL	Sudden infant death	Limited
ACADM	Medium chain acyl CoA dehydrogenase deficiency	Definitive
ACADS	Acyl-CoA dehydrogenase, short-chain, deficiency of	Definitive
ACADSB	2-Methylbutyryl-CoA dehydrogenase deficiency	Strong
ACADVL	VLCAD deficiency	Definitive
ACAT1	Alpha-methylacetoacetic aciduria	Definitive
ACBD5	Thrombocytopaenia	Limited
ACE	Renal tubular dysgenesis	Strong
ACO2	Cerebellar-retinal degeneration, infantile	Moderate
ACOX1	Peroxisomal acyl-CoA oxidase deficiency	Strong
ACSF3	Combined malonic and methylmalonic aciduria	Strong
ACTA1	Nemaline myopathy	Definitive
ACTA1	Congenital myopathy with fiber type disproportion	Moderate
ACTA2	Aortic aneurysm, familial thoracic	Definitive
ACTB	Baraitser-Winter syndrome	Strong

ACTB	Neutrophil dysfunction and recurrent infection	Limited
ACTC1	Atrial septal defect	Limited
ACTC1	Cardiomyopathy, dilated	Moderate
ACTC1	Cardiomyopathy, familial hypertrophic	Strong
ACTC1	Left ventricular noncompaction	Limited
ACTG1	Deafness, autosomal dominant	Strong
ACTG1	Baraitser-Winter syndrome	Strong
ACTG2	Megacystis-microcolon-intestinal hypoperistalsis syndrome	Strong
ACTN1	Macrothrombocytopenia	Strong
ACTN2	Cardiomyopathy, dilated	Limited
ACTN2	Cardiomyopathy, familial hypertrophic	Moderate
ACTN4	Glomerulosclerosis, focal segmental, 1	Definitive
ACVR1	Fibrodysplasia ossificans progressiva	Definitive
ACVR2B	Left-right axis malformation	Limited
ACVRL1	Telangiectasia, hereditary hemorrhagic, type 2	Definitive
ADA	Severe combined immunodeficiency due to ADA deficiency	Definitive
ADAM17	Neonatal inflammatory skin and bowel disease	Limited
ADAMTS13	Thrombotic thrombocytopenic purpura, familial	Definitive
ADAMTS2	Ehlers-Danlos syndrome VIIc	Moderate
ADAMTSL2	Geleophysic dysplasia 1	Strong
ADAR	Aicardi-Goutieres syndrome	Strong
ADAR	Dyschromatosis symmetrica hereditaria	Definitive
ADK	Hypermethioninemia due to adenosine kinase deficiency	Strong
AGA	Aspartylglucosaminuria	Strong
AGL	Glycogen storage disease IIIa	Definitive
AGPS	Rhizomelic chondrodysplasia punctata, type 3	Moderate
AGRN	Myasthenia, limb-girdle, familial	Strong
AGT	Renal tubular dysgenesis	Moderate
AGTR1	Renal tubular dysgenesis	Moderate
AGXT	Hyperoxaluria, primary, type 1	Definitive
AHI1	Joubert syndrome-3	Definitive
AHSP	Thalassaemia	Limited
AIFM1	Cowchock syndrome	Strong
AIP	Pituitary adenoma	Definitive
AIRE	Autoimmune polyendocrinopathy syndrome , type I, with or without reversible metaphyseal dysplasia	Definitive
AK1	Hemolytic anemia due to adenylate kinase deficiency	Moderate
AKAP9	Long QT syndrome	Limited
AKR1D1	Bile acid synthesis defect, congenital, 2	Strong
AKT2	Severe insulin resistance and diabetes mellitus	Limited

AKT3	Megalencephaly-polymicrogyria-polydactyly-hydrocephalus syndrome	Moderate
ALAS2	Anemia, sideroblastic, X-linked	Definitive
ALB	Analbuminemia	Strong
ALDH18A1	Cutis laxa, autosomal recessive, type IIIA	Strong
ALDH1A2	Tetralogy of Fallot	Limited
ALDH3A2	Sjogren-Larsson syndrome	Definitive
ALDH4A1	Hyperprolinemia, type II	Moderate
ALDH5A1	Succinic semialdehyde dehydrogenase deficiency	Definitive
ALDOA	Aldolase A deficiency	Moderate
ALDOB	Fructose intolerance	Definitive
ALG1	Congenital disorder of glycosylation, type Ik	Strong
ALG11	Congenital disorder of glycosylation type 1P	Moderate
ALG12	Congenital disorder of glycosylation, type Ig	Strong
ALG2	Congenital disorder of glycosylation, type Ii	Limited
ALG3	Congenital disorder of glycosylation, type Id	Strong
ALG6	Congenital disorder of glycosylation, type Ic	Strong
ALG8	Congenital disorder of glycosylation, type Ih	Strong
ALG9	Congenital disorder of glycosylation, type Il	Moderate
ALMS1	Alstrom syndrome	Definitive
ALOX12B	Ichthyosis, congenital, autosomal recessive	Strong
ALOXE3	Ichthyosis, congenital, autosomal recessive	Strong
ALPL	Hypophosphatasia	Definitive
ALS2	Amyotrophic lateral sclerosis	Definitive
ALX4	Parietal foramina 2	Strong
AMACR	Bile acid synthesis defect, congenital, 4	Moderate
AMACR	Alpha-methylacyl-CoA racemase deficiency	Moderate
AMELX	Amelogenesis imperfecta	Definitive
AMN	Megaloblastic anemia-1, Norwegian type	Strong
AMPD1	Adenosine monophosphate deaminase deficiency	Moderate
AMT	Hyperglycinaemia, non-ketotic	Strong
ANK1	Spherocytosis	Definitive
ANK2	Long QT syndrome	Definitive
ANKH	Craniometaphyseal dysplasia	Definitive
ANKRD1	Cardiomyopathy, dilated	Moderate
ANKRD1	Cardiomyopathy, hypertrophic	Limited
ANKRD26	Thrombocytopenia 2	Strong
ANO10	Spinocerebellar ataxia, autosomal recessive 10	Strong
ANO5	Muscular dystrophy, limb-girdle, type 2L	Strong
ANO5	Gnathodiaphyseal dysplasia	Moderate

ANTXR2	Hyaline fibromatosis syndrome	Definitive
AP1S3	Pustular psoriasis	Limited
AP3B1	Hermansky-Pudlak syndrome 2	Strong
AP4M1	Spastic paraplegia 50, autosomal recessive	Moderate
APC	Adenomatous polyposis coli	Definitive
APC	Adenomatous polyposis coli, attenuated	Definitive
APOB	Apolipoprotein B deficiency	Definitive
APOE	Sea-blue histiocyte disease	Limited
APP	Alzheimer disease 1, familial	Definitive
APRT	Adenine phosphoribosyltransferase deficiency	Definitive
APTX	Ataxia, early-onset, with oculomotor apraxia and hypoalbuminemia	Definitive
AR	Androgen insensitivity	Definitive
AR	Spinal and bulbar muscular atrophy of Kennedy	Definitive
ARFGEF2	Periventricular heterotopia with microcephaly	Strong
ARG1	Arginase deficiency	Definitive
ARHGAP31	Syndromic cutis aplasia & limb anomalies	Limited
ARHGEF9	Hyperekplexia and epilepsy	Moderate
ARID1A	Coffin-Siris syndrome	Moderate
ARID1B	Coffin-Siris syndrome	Strong
ARL13B	Joubert syndrome	Limited
ARMC4	Primary ciliary dyskinesia	Strong
ARSA	Metachromatic leukodystrophy	Definitive
ARSB	Mucopolysaccharidosis type VI (Maroteaux-Lamy)	Definitive
ARSE	Chondrodysplasia punctata, X-linked recessive	Strong
ARX	Lissencephaly, X-linked 2	Definitive
ASCL1	Congenital central hypoventilation	Limited
ASL	Argininosuccinic aciduria	Definitive
ASNS	Microcephaly, intellectual disability, cerebral atrophy & intractable seizures	Moderate
ASPA	Canavan disease	Definitive
ASS1	Citrullinemia	Definitive
ATIC	AICA-Ribosiduria	Limited
ATM	Ataxia-telangiectasia	Definitive
ATN1	Dentatorubral-pallidoluysian atrophy 1	Moderate
ATP1A2	Hemiplegic migraine	Strong
ATP1A3	Rapid-onset dystonia-parkinsonism	Strong
ATP2A1	Brody myopathy	Strong
ATP6AP2	X-linked recessive intellectual deficit - epilepsy	Moderate
ATP6V0A2	Cutis laxa, autosomal recessive, type IIA	Strong

ATP6V1B1	Renal tubular acidosis & hearing loss	Strong
ATP7A	Menkes syndrome	Definitive
ATP7A	Occipital horn syndrome	Definitive
ATP7A	Spinal muscular atrophy, distal, X-linked 3	Limited
ATP7B	Wilson disease	Definitive
ATP8B1	Cholestasis, progressive familial intrahepatic 1	Definitive
ATR	Seckel syndrome	Moderate
ATRX	Alpha-thalassemia/mental retardation syndrome	Definitive
AUH	3-methylglutaconic aciduria, type I	Strong
AVPR2	Diabetes insipidus, nephrogenic	Definitive
AXL	Hypogonadotropic hypogonadism	Limited
B3GALTL	Peters-Plus syndrome	Strong
B3GAT3	Multiple joint dislocations, short stature, craniofacial dysmorphism, and congenital heart defects	Moderate
B4GALT1	CDG syndrome type IId	Limited
B9D2	Meckel syndrome	Limited
BAAT	Bile acid amidation defect	Strong
BAG3	Cardiomyopathy, dilated	Strong
BAG3	Myopathy, myofibrillar	Moderate
BANF1	Progeroid syndrome	Moderate
BARD1	Tetralogy of Fallot	Limited
BBS1	Bardet-Biedl syndrome	Definitive
BBS10	Bardet-Biedl syndrome	Definitive
BBS12	Bardet-Biedl syndrome	Definitive
BBS2	Bardet-Biedl syndrome	Definitive
BBS4	Bardet-Biedl syndrome	Definitive
BBS5	Bardet-Biedl syndrome	Strong
BBS7	Bardet-Biedl syndrome	Strong
BBS9	Bardet-Biedl syndrome	Strong
BCKDHA	Maple syrup urine disease	Definitive
BCKDHB	Maple syrup urine disease	Definitive
BCL9	Congenital heart disease	Limited
BCS1L	Complex 3 deficiency	Definitive
BDNF	Central hypoventilation syndrome	Limited
BICD2	Congenital spinal muscular atrophy	Strong
BIN1	Myopathy, centronuclear, autosomal recessive	Strong
BLM	Bloom syndrome	Definitive
BLOC1S3	Hermansky-Pudlak syndrome 8	Limited
BLOC1S6	Hermansky-pudlak syndrome 9	Limited
BMPR1A	Juvenile polyposis syndrome	Definitive

BMPR1A	Tetralogy of Fallot	Limited
BMPR2	Pulmonary hypertension, familial primary	Definitive
BNC2	Total anomalous pulmonary venous return	Limited
BPGM	Erythrocytosis due to bisphosphoglycerate mutase deficiency	Limited
BRAF	Cardiofaciocutaneous syndrome	Definitive
BRAF	LEOPARD syndrome	Moderate
BRCA1	Breast-ovarian cancer, familial, 1	Definitive
BRCA2	Breast-ovarian cancer, familial, 2	Definitive
BRCA2	Fanconi anemia, complementation group D1	Definitive
BSCL2	Silver spastic paraplegia syndrome	Strong
BSCL2	Berardinelli-Seip lipodystrophy	Definitive
BSND	Bartter syndrome with sensorineural deafness	Strong
BTD	Biotinidase deficiency	Definitive
BTK	Agammaglobulinemia, X-linked 1	Definitive
BVES	Congenital heart disease	Limited
C10ORF2	Spinocerebellar ataxia infantile-onset	Strong
C15ORF41	Congenital dyserythropoietic anemia type I	Limited
C3	Haemolytic uraemic syndrome	Definitive
CA2	Osteopetrosis, autosomal recessive 3, with renal tubular acidosis	Definitive
CACNA1A	Episodic ataxia, type 2	Definitive
CACNA1C	Brugada syndrome	Moderate
CACNA1D	Sinoatrial node dysfunction and deafness	Limited
CACNA1F	Night blindness, congenital stationary (complete), 1A, X-linked	Definitive
CACNA1S	Malignant hyperthermia	Limited
CACNA2D1	Brugada syndrome	Limited
CACNB2	Brugada syndrome	Limited
CAPN3	Muscular dystrophy, limb-girdle, type 2A	Definitive
CARS2	Epileptic encephalopathy	Limited
CASK	Mental retardation and microcephaly with pontine and cerebellar hypoplasia	Strong
CASP10	Autoimmune lymphoproliferative syndrome II	Moderate
CASQ2	Ventricular tachycardia, catecholaminergic polymorphic	Strong
CAV3	Cardiomyopathy, familial hypertrophic	Limited
CAV3	Long QT syndrome-9	Limited
CAV3	Muscular dystrophy, limb-girdle, type IC,	Definitive
CAV3	Caveolinopathy	Definitive
CAV3	Rippling muscle disease	Moderate

CBL	Noonan syndrome-like disorder with or without juvenile meylomonocytic leukemia	Strong
CBS	Homocystinuria, B6-responsive and nonresponsive types	Definitive
CC2D2A	Joubert syndrome	Strong
CCDC103	Primary ciliary dyskinesia	Moderate
CCDC39	Primary ciliary dyskinesia	Strong
CCDC40	Primary ciliary dyskinesia	Strong
CCDC50	Hearing loss	Limited
CCDC78	Congenital myopathy with prominent internal nuclei and atypical cores	Limited
CCDC88C	Hydrocephalus	Moderate
CD2AP	Glomerulosclerosis, focal segmental, 3	Moderate
CD36	Platelet glycoprotein IV deficiency	Strong
CD40LG	Immunodeficiency, X-linked, with hyper-IgM	Definitive
CD46	Haemolytic uraemic syndrome	Definitive
CD96	C syndrome	Moderate
CDAN1	Anemia, congenital dyserythropoietic, type I	Definitive
CDAN3	Congenital dyserythropoietic anemia type 3	Limited
CDH1	Orofacial clefts	Limited
CDH1	Gastric cancer	Definitive
CDH23	Deafness, autosomal recessive	Definitive
CDH23	Usher syndrome, type 1D	Definitive
CDK5RAP2	Microcephaly 3, primary, autosomal recessive	Moderate
CDKL5	Epileptic encephalopathy, early infantile, 2	Definitive
CDKN1C	Beckwith-Wiedemann syndrome	Definitive
CDKN2A	Melanoma	Definitive
CDON	Holoprosencephaly	Moderate
CDSN	Hypotrichosis	Strong
CDT1	Meier-Gorlin syndrome	Moderate
CEACAM16	Hearing loss, autosomal dominant	Moderate
CENPJ	Primary microcephaly	Moderate
CEP152	Seckel syndrome	Strong
CEP290	Joubert syndrome	Strong
CEP41	Joubert syndrome	Moderate
CFB	Haemolytic uraemic syndrome	Moderate
CFC1	Congenital heart defects	Strong
CFD	Complement factor D deficiency	Limited
CFH	Haemolytic uraemic syndrome	Definitive
CFHR1	Haemolytic uraemic syndrome	Moderate
CFHR3	Haemolytic uraemic syndrome	Limited

CFHR4	Hemolytic-uremic syndrome, atypical, susceptibility to	Limited
CFHR5	Haemolytic uraemic syndrome	Moderate
CFI	Haemolytic uraemic syndrome	Strong
CFL2	Nemaline myopathy	Strong
CFP	Properdin deficiency, X-linked	Strong
CFTR	Cystic fibrosis	Definitive
CHAT	Congenital myasthenic syndrome	Strong
CHD2	Developmental delay, intellectual disability, epilepsy	Strong
CHD7	CHARGE syndrome	Definitive
CHEK2	Breast cancer, susceptibility to	Strong
CHKB	Muscular dystrophy, congenital, megaconial type	Strong
CHM	Choroideremia	Definitive
CHRM2	Cardiomyopathy, dilated	Limited
CHRNA1	Congenital myasthenic syndrome	Strong
CHRNA2	Epilepsy	Moderate
CHRNB1	Congenital myasthenic syndrome	Moderate
CHRND	Congenital myasthenic syndrome	Strong
CHRNE	Congenital myasthenic syndrome	Definitive
CHRNG	Pterygium syndrome	Strong
CHST3	Larsen syndrome	Moderate
CHSY1	Temtamy preaxial brachydactyly syndrome	Moderate
CIRH1A	North American Indian childhood cirrhosis	Moderate
CISD2	Wolfram syndrome	Moderate
CITED2	Congenital heart defects	Limited
CLCN1	Myotonia congenita	Definitive
CLCN5	Dent disease	Definitive
CLCN7	Osteopetrosis	Definitive
CLDN1	Ichthyosis, leukocyte vacuoles, alopecia, and sclerosing cholangitis	Moderate
CLDN14	Hearing loss, non-syndromic, autosomal recessive	Strong
CLDN19	Hypomagnesemia 5, renal, with ocular involvement	Strong
CLMP	Congenital short-bowel syndrome	Moderate
CLN3	Ceroid lipofuscinosis, neuronal, 3	Definitive
CLN5	Ceroid lipofuscinosis, neuronal, 5	Definitive
CLN6	Ceroid lipofuscinosis, neuronal, 6	Definitive
CLN8	Ceroid lipofuscinosis, neuronal, 8	Strong
CLPP	Perrault syndrome	Moderate
CLRN1	Usher syndrome, type 3A	Strong
CNGB3	Achromatopsia-3	Definitive
CNTNAP2	Autism spectrum disorder	Moderate

COCH	Deafness, non-syndromic, autosomal dominant	Strong
COG4	Congenital disorder of glycosylation, type IIj	Limited
COG5	Congenital disorder of glycosylation, type IIi	Moderate
COG7	Congenital disorder of glycosylation, type IIe	Moderate
COL11A1	Stickler syndrome	Definitive
COL11A2	Otospondylomegaepiphyseal dysplasia	Definitive
COL17A1	Epidermolysis bullosa, junctional, non-Herlitz type	Definitive
COL1A1	Osteogenesis imperfecta, type I	Definitive
COL1A1	Caffey disease	Strong
COL1A2	Osteogenesis imperfecta, type II	Definitive
COL2A1	Stickler syndrome	Definitive
COL3A1	Ehlers-Danlos syndrome, type IV	Definitive
COL4A3	Alport syndrome	Definitive
COL4A4	Alport syndrome	Definitive
COL4A5	Alport syndrome	Definitive
COL5A1	Ehlers-Danlos syndrome, type I	Definitive
COL5A2	Ehlers-Danlos syndrome	Strong
COL6A1	Ullrich congenital muscular dystrophy	Definitive
COL6A2	Ullrich congenital muscular dystrophy	Definitive
COL6A3	Ullrich congenital muscular dystrophy	Definitive
COL7A1	Epidermolysis bullosa dystrophica	Definitive
COL9A1	Stickler syndrome	Moderate
COL9A2	Stickler syndrome	Limited
COLQ	Congenital myasthenic syndrome	Definitive
COQ2	Coenzyme Q10 deficiency, primary, 1	Limited
COQ6	Nephrotic syndrome with sensorineural deafness	Moderate
COX4I2	Exocrine pancreatic insufficiency, dyserythropoietic anemia, and calvarial hyperostosis	Limited
CP	Aceruloplasminaemia	Definitive
CPOX	Coproporphyria	Definitive
CPS1	Carbamoylphosphate synthetase I deficiency	Definitive
CPT1A	Carnitine palmitoyltransferase I deficiency	Definitive
CPT2	Carnitine palmitoyltransferase 2 deficiency	Definitive
CPZ	Autism	Limited
CR2	Hypogammaglobulinaemia	Limited
CREBBP	Rubinstein-Taybi syndrome	Definitive
CRELD1	Cardiac atrioventricular septal defect	Moderate
CRLF1	Crisponi syndrome	Definitive
CRTAP	Osteogenesis imperfecta, type VII	Strong
CRYAB	Cardiomyopathy, dilated	Moderate

CRYAB	Myofibrillar myopathy	Strong
CSF1R	Leukoencephalopathy, diffuse hereditary, with spheroids	Definitive
CSF2RA	Pulmonary alveolar proteinosis	Strong
CSF2RB	Pulmonary alveolar proteinosis	Limited
CSRP3	Cardiomyopathy, dilated, 1M	Limited
CSRP3	Cardiomyopathy, familial hypertrophic, 12	Moderate
CSTA	Exfoliative ichthyosis	Moderate
CSTB	Epilepsy, progressive myoclonic 1A	Strong
CTC1	Coats plus syndrome	Strong
CTDP1	Congenital cataracts - facial dysmorphism - neuropathy	Moderate
CTF1	Cardiomyopathy, dilated	Limited
CTNS	Cystinosis	Definitive
CTSD	Ceroid lipofuscinosis, neuronal, 10	Strong
CTSK	Pycnodysostosis	Definitive
CUBN	Megaloblastic anemia-1, Finnish type	Strong
CUL7	3-M syndrome	Definitive
CYBA	Chronic granulomatous disease	Definitive
CYBB	Chronic granulomatous disease	Definitive
CYCS	Thrombocytopenia 4	Limited
CYP11A1	Adrenal insufficiency, congenital, with 46XY sex reversal, partial or complete	Strong
CYP11B1	Adrenal hyperplasia, congenital, due to 11-beta-hydroxylase deficiency	Definitive
CYP21A2	Adrenal hyperplasia, congenital, due to 21-hydroxylase deficiency	Definitive
CYP27A1	Cerebrotendinous xanthomatosis	Definitive
CYP27B1	Vitamin D-dependent rickets, type I	Definitive
CYP4F22	Ichthyosis, congenital, autosomal recessive	Strong
CYP7A1	Hypercholesterolemia due to cholesterol 7alpha-hydroxylase deficiency	Limited
CYP7B1	Cholestasis, severe	Limited
D2HGDH	D-2-hydroxyglutaric aciduria	Strong
DAG1	Muscular dystrophy-dystroglycanopathy (limb-girdle), type C, 9	Moderate
DAPK3	Congenital heart disease	Limited
DBH	Dopamine beta-hydroxylase deficiency	Moderate
DBT	Maple syrup urine disease	Definitive
DCLRE1C	Severe combined immunodeficiency, Athabascan type	Definitive
DCTN1	Amyotrophic lateral sclerosis	Moderate
DCX	Lissencephaly, X-linked	Definitive

DCX	Lennox-Gastaut syndrome	Limited
DDB2	Xeroderma pigmentosum	Strong
DDC	Aromatic L-amino acid decarboxylase deficiency	Strong
DDHD1	Spastic paraplegia	Moderate
DDOST	Congenital disorder of glycosylation, type Ir	Limited
DDR2	Spondylometaepiphyseal dysplasia, short limb-hand type	Moderate
DECR1	2,4-Dienoyl-CoA reductase deficiency	Limited
DES	Cardiomyopathy, dilated	Strong
DES	Myopathy, myofibrillar	Definitive
DFNA5	Hearing loss	Strong
DFNB31	Hearing loss	Moderate
DFNB59	Hearing loss	Strong
DGKE	Haemolytic uraemic syndrome, atypical	Strong
DGUOK	Mitochondrial DNA depletion syndrome	Definitive
DHCR24	Desmosterolosis	Moderate
DHCR7	Smith-Lemli-Opitz syndrome	Definitive
DIABLO	Deafness, autosomal dominant	Limited
DIAPH1	Hearing loss	Moderate
DKC1	Dyskeratosis congenita	Definitive
DLC1	Congenital heart disease	Limited
DLD	Maple syrup urine disease, type III	Strong
DLL3	Spondylocostal dysostosis, autosomal recessive, 1	Strong
DMD	Duchenne muscular dystrophy	Definitive
DMD	Becker muscular dystrophy	Definitive
DMD	Cardiomyopathy, dilated	Definitive
DMP1	Hypophosphatemic rickets, AR	Strong
DMPK	Myotonic dystrophy 1	Definitive
DNAAF1	Primary ciliary dyskinesia	Strong
DNAAF2	Primary ciliary dyskinesia	Moderate
DNAAF3	Primary ciliary dyskinesia	Limited
DNAH11	Primary ciliary dyskinesia	Definitive
DNAH5	Primary ciliary dyskinesia	Definitive
DNAI1	Primary ciliary dyskinesia	Strong
DNAI2	Primary ciliary dyskinesia	Moderate
DNAJB6	Muscular dystrophy, limb girdle	Strong
DNAJC19	3-methylglutaconic aciduria, type V	Limited
DNAJC5	Neuronal ceroid lipofuscinosis, adult-onset	Strong
DNAL1	Primary ciliary dyskinesia	Limited
DNM2	Myopathy, centronuclear	Definitive
DNM2	Charcot-Marie-Tooth disease, axonal, type 2M	Definitive

DNMT3B	Immunodeficiency-centromeric instability-facial anomalies syndrome 1	Strong
DOCK8	Hyper-IgE syndrome	Definitive
DOK7	Congenital myasthenic syndrome	Definitive
DOLK	Congenital disorder of glycosylation, type Im	Moderate
DPAGT1	Congenital disorder of glycosylation, type Ij	Strong
DPM1	Congenital disorder of glycosylation, type Ie	Moderate
DPP6	Ventricular fibrillation, paroxysmal familial, 2	Limited
DPYD	Dihydropyrimidine dehydrogenase deficiency	Definitive
DSC2	Arrhythmogenic right ventricular cardiomyopathy	Definitive
DSG2	Arrhythmogenic right ventricular cardiomyopathy	Definitive
DSP	Arrhythmogenic right ventricular dysplasia/cardiomyopathy	Definitive
DSP	Epidermolysis bullosa, lethal acantholytic	Definitive
DTHD1	Leber congenital amaurosis with myopathy	Limited
DTNA	Left ventricular noncompaction 1	Limited
DTNBP1	Hermansky-Pudlak syndrome 7	Limited
DUOX2	Thyroid dyshormonogenesis	Definitive
DUOXA2	Thyroid dyshormonogenesis	Moderate
DYSF	Muscular dystrophy, limb-girdle, type 2B	Definitive
DYSF	Miyoshi muscular dystrophy 1	Definitive
ECE1	Hirschsprung disease	Limited
EDA	Ectodermal dysplasia, hypohidrotic	Definitive
EDAR	Ectodermal dysplasia, hypohidrotic	Definitive
EDARADD	Ectodermal dysplasia, hypohidrotic	Strong
EDN3	Hirschsprung disease	Moderate
EDN3	Waardenburg syndrome	Moderate
EDNRB	Hirschsprung disease	Strong
EDNRB	Waardenburg syndrome	Strong
EFEMP2	Cutis laxa, autosomal recessive, type IB	Moderate
EFHC1	Myoclonic epilepsy	Strong
EFTUD2	Mandibulofacial dysostosis with microcephaly	Strong
EGR2	Charcot-Marie-Tooth disease	Strong
EIF2AK3	Wolcott-Rallison syndrome	Definitive
EIF2B1	Leukoencephalopathy with vanishing white matter	Moderate
ELANE	Neutropenia, congenital	Definitive
ELN	Supravalvar aortic stenosis	Definitive
EMD	Muscular dystrophy, Emery-Dreifuss	Definitive
ENG	Telangiectasia, hereditary hemorrhagic, type 1	Definitive
ENPP1	Arterial calcification, generalized, of infancy, 1	Strong
EPB42	Spherocytosis	Moderate

EPCAM	Lynch syndrome	Strong
EPHX1	Hypercholanemia, familial	Limited
EPM2A	Epilepsy, progressive myoclonic 2A (Lafora)	Definitive
ERBB3	Lethal congenital contractural syndrome 2	Limited
ERCC1	Xeroderma pigmentosum	Limited
ERCC2	Xeroderma pigmentosum	Definitive
ERCC3	Xeroderma pigmentosum	Moderate
ERCC4	Xeroderma pigmentosum	Moderate
ERCC5	Xeroderma pigmentosum	Definitive
ERCC6	Cockayne syndrome	Definitive
ERCC8	Cockayne syndrome	Definitive
ESCO2	Roberts syndrome	Strong
ESPN	Hearing loss	Moderate
ESRRB	Hearing loss	Strong
ETFA	Glutaric acidemia IIA	Strong
ETFB	Glutaric acidemia IIB	Strong
ETFDH	Glutaric acidemia IIC	Strong
ETHE1	Ethylmalonic encephalopathy	Strong
EVC	Ellis-van Creveld syndrome	Definitive
EVC2	Ellis-van Creveld syndrome	Definitive
EXT1	Exostoses, multiple, type 1	Definitive
EXT2	Exostoses, multiple, type 2	Definitive
EYA1	Branchiootorenal syndrome	Definitive
EYA4	Deafness, autosomal dominant	Strong
EZH2	Weaver syndrome 2	Strong
F11	Factor XI deficiency	Definitive
F2	Prothrombin deficiency	Definitive
F5	Risk for deep vein thrombosis	Definitive
F8	Hemophilia A	Definitive
F9	Hemophilia B	Definitive
FAAH2	Autism spectrum disorder	Limited
FAH	Tyrosinemia, type I	Definitive
FAM111B	Hereditary fibrosing poikiloderma with tendon contracture, myopathy, and pulmonary fibrosis	Moderate
FAM126A	Hypomyelination and congenital cataract	Strong
FAM134B	Neuropathy, hereditary sensory and autonomic, type IIB	Moderate
FAM161A	Retinal dystrophy	Strong
FAM20C	Osteosclerotic bone dysplasia	Strong
FAM58A	Syndactyly - telecanthus - anogenital and renal malformations	Strong

FANCA	Fanconi anaemia	Definitive
FANCB	Fanconi anaemia	Strong
FANCC	Fanconi anaemia	Definitive
FANCD2	Fanconi anaemia	Strong
FANCE	Fanconi anaemia	Moderate
FANCF	Fanconi anaemia	Moderate
FANCG	Fanconi anaemia	Definitive
FANCI	Fanconi anaemia	Strong
FANCL	Fanconi anaemia	Moderate
FANCM	Fanconi anaemia	Limited
FBLN5	Age-related macular degeneration	Moderate
FBLN5	Cutis laxa	Strong
FBN1	Marfan's syndrome	Definitive
FBN1	Weill-Marchesani syndrome 2, dominant	Moderate
FBN1	Shprintzen-Goldberg syndrome	Limited
FBN2	Contractural arachnodactyly	Strong
FGA	Afibrinogenaemia	Definitive
FGB	Afibrinogenaemia	Definitive
FGD1	Aarskog-Scott syndrome	Definitive
FGD4	Charcot-Marie-Tooth disease	Strong
FGF3	Deafness, congenital with inner ear agenesis, microtia, and microdontia	Strong
FGFR1	Kallmann syndrome	Definitive
FGFR2	Pfeiffer syndrome	Definitive
FGFR2	Apert syndrome	Definitive
FGFR2	Crouzon syndrome	Definitive
FGFR2	Beare-Stevenson cutis gyrata syndrome	Strong
FGFR2	Jackson-Weiss syndrome	Strong
FGFR3	Achondroplasia	Definitive
FGFR3	Hypochondroplasia	Definitive
FGFR3	Crouzon syndrome with acanthosis nigricans	Definitive
FGFR3	Thanatophoric dysplasia type 1	Definitive
FGFR3	Muenke syndrome	Definitive
FGFR3	CATSHL syndrome	Limited
FGFR3	LADD syndrome	Limited
FGG	Afibrinogenaemia	Strong
FH	Leiomyomatosis and renal cell cancer	Definitive
FH	Fumarase deficiency	Definitive
FHL1	Myofibrillar myopathy	Limited
FHL1	Emery-Dreifuss muscular dystrophy	Strong

FHL2	Cardiomyopathy, hypertrophic	Limited
FKBPL	Infertility	Limited
FKRP	Muscular dystrophy, limb girdle 2I	Definitive
FKRP	Muscle-eye-brain disease	Definitive
FKTN	Muscular dystrophy, Fukuyama	Definitive
FKTN	Congenital muscular dystrophy-dystroglycanopathy with brain and eye anomalies	Definitive
FLCN	Birt-Hogg-Dube syndrome	Definitive
FLG	Ichthyosis vulgaris	Strong
FLNA	Otopalatodigital spectrum disorder	Definitive
FLNC	Myofibrillar myopathy	Moderate
FMO3	Trimethylaminuria	Definitive
FOXC1	Axenfeld-Rieger syndrome	Definitive
FOXC2	Lymphoedema, primary	Strong
FOXE1	Bamforth-Lazarus syndrome	Moderate
FOXF1	Alveolar capillary dysplasia with misalignment of pulmonary veins	Definitive
FOXF2	Disorders of sex development with cleft palate	Limited
FOXH1	Congenital heart defects	Limited
FOXN1	Congenital alopecia with T-cell immunodeficiency	Moderate
FOXP3	IPEX syndrome	Definitive
FRAS1	Fraser syndrome	Strong
FREM1	Manitoba oculotrichoanal syndrome	Strong
FREM2	Fraser syndrome	Moderate
FSCN2	Retinitis pigmentosa	Limited
FTCD	Glutamate formiminotransferase deficiency	Limited
FTL	Neuroferritinopathy	Strong
FUCA1	Fucosidosis	Strong
FXN	Friedreich ataxia	Definitive
G6PC	Glycogen storage disease Ia	Definitive
G6PC3	Neutropaenia, congenital	Strong
G6PD	Glucose-6-phosphate dehydrogenase deficiency	Definitive
GAA	Glycogen storage disease II	Definitive
GABRA1	Epilepsy, idiopathic generalised	Strong
GABRG2	Epilepsy, childhood absence with febrile seizure	Definitive
GALC	Krabbe disease	Definitive
GALK1	Galactokinase deficiency with cataracts	Definitive
GALNS	Mucopolysaccharidosis IVA	Definitive
GALT	Galactosaemia	Definitive
GAN	Giant axonal neuropathy	Definitive

GATA1	Porphyria, congenital erythropoietic	Limited
GATA1	Dyserythropoietic anemia with thrombocytopenia	Strong
GATA4	Congenital heart defects	Definitive
GATA5	Familial atrial fibrillation	Moderate
GATA6	Atrial fibrillation	Limited
GATAD1	Cardiomyopathy, dilated, 2B	Limited
GBA	Gaucher disease 1	Definitive
GBE1	Glycogen storage disease IV	Definitive
GBE1	Polyglucosan body disease, adult form	Definitive
GCDH	Glutaricaciduria, type I	Definitive
GCH1	Dystonia, dopa-responsive	Definitive
GCK	Hyperinsulinemic hypoglycemia, familial	Definitive
GCLC	Hemolytic anemia due to gamma-glutamylcysteine synthetase deficiency	Limited
GCSH	Glycine encephalopathy	Limited
GDAP1	Charcot-Marie-Tooth disease	Definitive
GDF1	Congenital heart defects	Limited
GDNF	Hirschsprung disease	Moderate
GDNF	Central hypoventilation syndrome	Limited
GFAP	Alexander disease	Definitive
GFER	Myopathy, mitochondrial progressive, with congenital cataract, hearing loss, and developmental delay	Limited
GFM1	Combined oxidative phosphorylation deficiency 1	Strong
GFPT1	Congenital myasthenic syndrome, limb-girdle	Strong
GIPC3	Hearing loss	Strong
GJA1	Oculodentodigital dysplasia	Definitive
GJA5	Atrial fibrillation	Strong
GJB1	Charcot-Marie-Tooth neuropathy	Definitive
GJB2	Deafness	Definitive
GJB2	Deafness and palmoplantar keratoderma	Definitive
GJC2	Pelizaeus-Merzbacher-like disease	Strong
GLA	Fabry disease	Definitive
GLB1	Gangliosidosis GM1	Definitive
GLDC	Glycine encephalopathy	Definitive
GLE1	Lethal arthrogryposis with anterior horn cell disease	Moderate
GLI2	Holoprosencephaly-9	Moderate
GLI3	Greig cephalopolysyndactyly syndrome	Definitive
GLIS3	Diabetes mellitus, neonatal, with congenital hypothyroidism	Moderate

GLRA1	Hyperekplexia, hereditary 1, autosomal dominant or recessive	Strong
GLRB	Hyperekplexia 2, autosomal recessive	Moderate
GLUD1	Hyperinsulinism	Strong
GLUL	Congenital brain dysgenesis due to glutamine synthetase deficiency	Moderate
GMPPA	Congenital disorder of glycosylation	Moderate
GNAS	Pseudohypoparathyroidism	Definitive
GNAS	Pseudopseudohypoparathyroidism	Definitive
GNE	Inclusion body myopathy	Definitive
GNPTAB	Mucolipidosis II	Definitive
GNPTG	Mucolipidosis III gamma	Strong
GNS	Mucopolysaccharidosis IIId	Strong
GPC3	Simpson-Golabi-Behmel syndrome	Definitive
GPC4	Simpson-Golabi-Behmel syndrome	Limited
GPC6	Omodysplasia	Moderate
GPD1L	Brugada syndrome	Moderate
GPHN	Hyperekplexia	Limited
GPR143	Ocular albinism, type I	Definitive
GPR56	Polymicrogyria, bilateral frontoparietal	Definitive
GPR98	Usher syndrome	Definitive
GPSM2	Chudley-McCullough syndrome	Strong
GPX1	Hemolytic anemia due to glutathione peroxidase deficiency	Limited
GRHL2	Hearing loss	Limited
GRHPR	Hyperoxaluria, primary, type II	Strong
GRIN2A	Epilepsy with neurodevelopmental defects	Strong
GRXCR1	Deafness, autosomal recessive	Moderate
GSS	Glutathione synthetase deficiency	Definitive
GTF2H5	Trichothiodystrophy	Moderate
GUCY2C	Meconium ileus	Moderate
GUSB	Mucopolysaccharidosis VII	Definitive
GYG1	Glycogen storage disease XV	Moderate
GYS2	Glycogen storage disease 0	Definitive
H19	Beckwith-Wiedemann Syndrome	Definitive
HADH	3-hydroxyacyl-CoA dehydrogenase deficiency	Moderate
HADH	Hyperinsulinemic hypoglycemia, familial, 4	Strong
HADHA	Mitochondrial trifunctional protein deficiency	Definitive
HADHB	Mitochondrial trifunctional protein deficiency	Definitive
HAMP	Haemochromatosis	Strong
HARS	Usher syndrome type 3B	Limited

HARS2	Perrault syndrome	Limited
HAS2	Congenital heart disease	Limited
HBA1	Thalassaemia alpha	Definitive
HBA2	Thalassemia, alpha	Definitive
HBB	Beta-thalassemia	Definitive
HCCS	Microphthalmia	Moderate
HCN4	Brugada syndrome	Limited
HDAC8	Cornelia de Lange syndrome-like features, ocular hypertelorism & large fontanelle	Strong
HEATR2	Primary ciliary dyskinesia	Limited
HERC2	Autism spectrum disorder	Limited
HESX1	Pituitary hypoplasia	Moderate
HEXA	Tay-Sachs disease	Definitive
HEXB	Sandhoff disease, infantile, juvenile, and adult forms	Definitive
HFE	Hemochromatosis	Definitive
HFE2	Haemochromatosis	Strong
HGD	Alkaptonuria	Definitive
HGF	Deafness, autosomal recessive	Limited
HGSNAT	Mucopolysaccharidosis IIIC	Definitive
HIBCH	Neurodegeneration, progressive infantile	Moderate
HINT1	Axonal neuropathy with neuromyotonia	Strong
HK1	Hemolytic anemia due to hexokinase deficiency	Moderate
HLCS	Holocarboxylase synthetase deficiency	Strong
HMBS	Porphyria, acute intermittent	Definitive
HMGCL	3-hydroxy-3-methylglutaric aciduria	Definitive
HNF1B	Renal cysts and diabetes syndrome	Strong
HNF4A	Hypoglycaemia, hyperinsulinaemic	Definitive
HOMEZ	Congenital heart disease	Limited
HOXA1	Athabaskan brainstem dysgenesis syndrome	Limited
HPD	Tyrosinemia, type III	Moderate
HPRT1	Lesch-Nyhan syndrome 1	Definitive
HPS1	Hermansky-Pudlak syndrome 1	Definitive
HPS3	Hermansky-Pudlak syndrome 3	Strong
HPS4	Hermansky-Pudlak syndrome 4	Definitive
HPS5	Hermansky-Pudlak syndrome 5	Strong
HPS6	Hermansky-Pudlak syndrome 6	Moderate
HRAS	Costello syndrome	Definitive
HSD17B10	17-beta-hydroxysteroid dehydrogenase X deficiency	Strong
HSD17B3	Pseudohermaphroditism, male, with gynecomastia	Definitive
HSD17B4	D-bifunctional protein deficiency	Strong

HSD3B7	3 beta-hydroxysteroid dehydrogenase deficiency	Strong
HSPB8	Charcot-Marie-Tooth disease, axonal, type 2L	Strong
HSPG2	Schwartz-Jampel syndrome	Strong
HTRA1	CARASIL syndrome	Strong
HYDIN	Primary ciliary dyskinesia	Moderate
HYLS1	Hydrolethalus syndrome	Limited
IDS	Mucopolysaccharidosis II	Definitive
IDUA	Mucopolysaccharidosis Ih	Definitive
IFT122	Cranioectodermal dysplasia	Moderate
IFT43	Cranioectodermal dysplasia	Limited
IFT80	Asphyxiating thoracic dystrophy 2	Moderate
IGBP1	Agenesis of the corpus callosum - intellectual deficit - coloboma - micrognathia	Limited
IGF1	Insulin-like growth factor deficiency	Moderate
IGHMBP2	Spinal muscular atrophy with respiratory distress	Strong
IGSF1	Central hypothyroidism and testicular enlargement	Strong
IKBKAP	Dysautonomia, familial	Definitive
IKBKG	Incontinentia pigmenti 1	Definitive
IL10RA	Inflammatory bowel disease	Strong
IL10RB	Inflammatory bowel disease	Moderate
IL2RG	Severe combined immunodeficiency, X-linked	Definitive
ILDR1	Deafness, autosomal recessive	Strong
ILK	Cardiomyopathy, dilated	Limited
INSR	Leprechaunism	Definitive
INVS	Nephronophthisis 2	Strong
IQCB1	Senior-Loken syndrome 5	Strong
IRF6	Popliteal pterygium syndrome	Definitive
IRF6	van der Woude syndrome	Definitive
IRS1	Diabetes mellitus, noninsulin dependent	Limited
ISCU	Myopathy with defiency of succinate dehydrogenase	Moderate
ISL1	Diabetes, type 2	Limited
ISPD	Muscular dystrophy-dystroglycanopathy (congenital with brain and eye anomalies), type A, 7	Strong
ITGA3	Interstitial lung disease, nephrotic syndrome, and epidermolysis bullosa, congenital	Moderate
ITGA6	Epidermolysis bullosa, junctional, with pyloric stenosis	Moderate
ITGA7	Congenital muscular dystrophy with integrin deficiency	Limited
ITGB4	Epidermolysis bullosa, junctional, with pyloric atresia	Definitive
IVD	Isovaleric acidemia	Definitive
IYD	Thyroid dyshormonogenesis	Limited

JAG1	Alagille syndrome	Definitive
JAK3	SCID, autosomal recessive, T-negative/B-positive type	Strong
JPH2	Cardiomyopathy, hypertrophic	Limited
JUP	Arrhythmogenic right ventricular dysplasia 12	Strong
JUP	Naxos disease	Strong
KANSL1	Koolen-De Vries syndrome	Strong
KARS	Charcot-Marie-Tooth disease, recessive intermediate	Moderate
KARS	Hearing loss	Limited
KAT6B	Genitopatellar syndrome	Strong
KBTBD13	Nemaline myopathy	Strong
KCNA1	Episodic ataxia type 1	Definitive
KCNA5	Atrial fibrillation	Strong
KCND3	Brugada syndrome	Limited
KCNE1	Long QT syndrome-5	Definitive
KCNE1	Jervell and Lange-Nielsen syndrome	Definitive
KCNE1L	Atrial fibrillation	Limited
KCNE2	Long QT syndrome-6	Strong
KCNE3	Brugada syndrome	Limited
KCNH2	Long QT syndrome-2	Definitive
KCNJ1	Bartter syndrome	Strong
KCNJ11	Hyperinsulinemic hypoglycemia, familial	Definitive
KCNJ18	Hypokalaemic periodic paralysis	Moderate
KCNJ2	Andersen cardiodysrhythmic periodic paralysis	Definitive
KCNJ5	Long QT syndrome	Limited
KCNJ8	Sudden infant death syndrom	Limited
KCNQ1	Long QT syndrome-1	Definitive
KCNQ1	Jervell and Lange-Nielsen syndrome	Strong
KCNQ1OT1	Beckwith-Wiedemann syndrome	Moderate
KCNQ2	Epilepsy, benign neonatal	Definitive
KCNQ3	Epilepsy, benign neonatal	Definitive
KCNQ4	Deafness, autosomal dominant	Strong
KCTD7	Epilepsy, progressive myoclonic	Strong
KDM5B	Congenital heart disease	Limited
KDM6A	Kabuki syndrome 2	Strong
KIAA1279	Goldberg-Shprintzen megacolon syndrome	Moderate
KIF1B	Charcot-Marie-Tooth disease	Limited
KIF21A	Fibrosis of extraocular muscles, congenital	Strong
KIF22	Spondyloepimetaphyseal dysplasia with joint laxity, type 2	Moderate
KIT	Piebaldism	Definitive
KLF1	Anemia, dyserythropoietic congenital, type IV	Moderate

KLHL40	Nemaline myopathy	Strong
KLHL41	Nemaline myopathy	Strong
KMT2D	Kabuki syndrome 1	Definitive
KPTN	Macrocephaly, neurodevelopmental delay, and seizures	Moderate
KRAS	Noonan syndrome	Definitive
KRT14	Epidermolysis bullosa simplex	Definitive
KRT16	Pachyonychia congenita	Strong
KRT17	Pachyonychia congenita	Strong
KRT18	Cirrhosis, cryptogenic	Moderate
KRT5	Epidermolysis bullosa simplex	Definitive
KRT6A	Pachyonychia congenita	Strong
KRT6B	Pachyonychia congenita	Moderate
KRT8	Cirrhosis, cryptogenic	Moderate
L1CAM	X-linked hydrocephalus syndrome	Definitive
LAMA2	Muscular dystrophy, congenital merosin-deficient	Definitive
LAMA3	Epidermolysis bullosa, junctional	Definitive
LAMA4	Cardiomyopathy, dilated	Limited
LAMB2	Pierson syndrome	Definitive
LAMB3	Epidermolysis bullosa, junctional	Definitive
LAMC2	Epidermolysis bullosa, junctional	Definitive
LAMP2	Danon disease	Definitive
LARGE	Walker-Warburg syndrome	Strong
LARS	Infantile liver failure syndrome	Moderate
LARS2	Perrault syndrome	Limited
LBR	Pelger-Huet anomaly	Strong
LBR	Reynolds syndrome	Limited
LDB3	Myofibrillar myopathy	Strong
LDLR	Hypercholesterolemia	Definitive
LEPR	Obesity, morbid, due to leptin receptor deficiency	Strong
LGI1	Epilepsy, familial temporal lobe, 1	Definitive
LHB	Hypogonadism	Moderate
LHFPL5	Deafness, autosomal recessive	Strong
LHX3	Pituitary hormone deficiency, combined	Strong
LIFR	Stuve-Wiedemann syndrome	Strong
LIG4	Severe combined immunodeficiency with sensitivity to ionizing radiation	Strong
LIPA	Wolman syndrome	Strong
LITAF	Charcot-Marie-Tooth disease	Strong
LMBRD1	Methylmalonic aciduria and homocystinuria	Strong
LMNA	Emery-Dreifuss muscular dystrophy 2	Definitive

LMNA	Dilated cardiomyopathy	Definitive
LMNA	Charcot-Marie-Tooth disease	Strong
LMNB2	Lipodystrophy, partial	Limited
LMOD3	Nemaline myopathy	Strong
LMX1B	Nail patella syndrome	Definitive
LOXHD1	Deafness, autosomal recessive	Strong
LPIN2	Majeed syndrome	Moderate
LPP	Tetralogy of Fallot	Limited
LRP2	Donnai-Barrow syndrome	Strong
LRP4	Cenani-Lenz syndactyly syndrome	Strong
LRP5	Osteoporosis-pseudoglioma syndrome	Definitive
LRP5	Osteopetrosis, autosomal dominant	Definitive
LRPPRC	Leigh syndrome	Strong
LRRC6	Primary ciliary dyskinesia	Strong
LRRK2	Parkinson disease	Strong
LRSAM1	Charcot-Marie-Tooth disease	Strong
LRTOMT	Deafness, autosomal recessive	Strong
LTBP4	Cutis laxa, autosomal recessive, type IC	Strong
LUM	Amyotrophic lateral sclerosis	Limited
LYST	Chediak-Higashi syndrome	Definitive
LYZ	Amyloidosis, systemic	Moderate
MAFB	Multicentric carpotarsal osteolysis syndrome	Strong
MAGI2	Infantile spasms	Strong
MAN2B1	Mannosidosis, alpha	Strong
MAP2K1	Cardiofaciocutaneous syndrome	Definitive
MAP2K2	Cardiofaciocutaneous syndrome	Definitive
MAPK10	Epileptic encephalopathy	Limited
MAPT	Dementia, frontotemporal, with or without parkinsonism	Strong
MARVELD2	Deafness, autosomal recessive	Strong
MAT1A	Methionine adenosyltransferase deficiency	Strong
MATN4	Multiple anomalies	Limited
MBTPS2	Ichthyosis follicularis, alopecia & photophobia	Strong
MCCC1	3-Methylcrotonyl-CoA carboxylase 1 deficiency	Definitive
MCCC2	3-Methylcrotonyl-CoA carboxylase 2 deficiency	Definitive
MCEE	Methylmalonyl-CoA epimerase deficiency	Moderate
MCFD2	Factor V and Factor VIII deficiency, combined	Strong
MCOLN1	Mucolipidosis IV	Definitive
MCPH1	Microcephaly 1, primary, autosomal recessive	Strong
MECP2	Rett syndrome	Definitive
MED12	Intellectual disability	Strong

MED13L	Transposition of great arteries	Limited
MED20	Congenital heart disease	Limited
MED25	Charcot-Marie-Tooth disease	Limited
MEFV	Mediterranean fever, familial	Definitive
MEGF10	Myopathy, areflexia, respiratory distress, and dysphagia, early-onset	Strong
MEN1	Multiple endocrine neoplasia I	Definitive
MESP2	Spondylocostal dysostosis, autosomal recessive 2	Moderate
MFN2	Charcot-Marie-Tooth disease	Definitive
MFSD8	Ceroid lipofuscinosis, neuronal	Strong
MGAT2	CDG syndrome type IIa	Moderate
MGP	Keutel syndrome	Strong
MIB1	Left ventricular noncompaction	Limited
MIR96	Hearing loss	Limited
MITF	Waardenburg syndrome	Strong
MKKS	Bardet-Biedl syndrome	Strong
MKS1	Meckel syndrome	Strong
MLC1	Megalencephalic leukoencephalopathy	Definitive
MLH1	Lynch syndrome	Definitive
MLPH	Griscelli syndrome type 3	Limited
MLYCD	Malonyl-CoA decarboxylase deficiency	Strong
MMAA	Methylmalonic aciduria, vitamin B12-responsive	Definitive
MMAB	Methylmalonic aciduria, vitamin B12-responsive, due to defect in synthesis of adenosylcobalamin, cblB complementation type	Definitive
MMACHC	Methylmalonic aciduria and homocystinuria, cblC type	Definitive
MMADHC	Methylmalonic aciduria and homocystinuria, cblD type	Strong
MOCS1	Molybdenum cofactor deficiency	Strong
MOCS2	Molybdenum cofactor deficiency	Strong
MOGS	Glucosidase 1 deficiency	Limited
MPDU1	Congenital disorder of glycosylation, type If	Moderate
MPI	Congenital disorder of glycosylation 1b	Strong
MPL	Amegakaryocytic thrombocytopaenia, congenital	Strong
MPV17	Mitochondrial DNA depletion syndrome, hepatic	Strong
MPZ	Charcot-Marie-Tooth disease	Definitive
MRPS16	Mitochondrial respiratory chain disorder	Limited
MRPS22	Mitochondrial respiratory chain disorder	Moderate
MSH2	Lynch syndrome	Definitive
MSH6	Lynch syndrome	Definitive
MSRB3	Deafness, autosomal recessive	Limited

MSX2	Parietal foramina 1	Strong
MT-ND1	Leber hereditary optic neuropathy	Definitive
MT-ND4	Leber hereditary optic neuropathy	Definitive
MT-ND6	Leber hereditary optic neuropathy	Definitive
MTHFR	Homocystinuria due to MTHFR deficiency	Definitive
MTM1	Myotubular myopathy, X-linked	Definitive
MTO1	Hypertrophic cardiomyopathy & lactic acidosis	Limited
MTR	Methylmalonic aciduria and homocystinuria	Strong
MTRR	Methylmalonic aciduria and homocystinuria	Strong
MTTP	Abetalipoproteinaemia	Strong
MUC5B	Pulmonary fibrosis, idiopathic	Limited
MURC	Cardiomyopathy, dilated	Limited
MUSK	Congenital myasthenic syndrome	Strong
MUT	Methylmalonic aciduria, mut(0) type	Definitive
MUTYH	MUTYH-associated polyposis	Definitive
MVK	Hyperimmunoglobulin D and periodic fever syndrome	Strong
MYBPC1	Distal arthrogryposis type I	Moderate
MYBPC3	Cardiomyopathy, familial hypertrophic	Definitive
MYBPC3	Cardiomyopathy, dilated	Conflicting
MYCN	Feingold syndrome	Strong
MYH11	Aortic aneurysm, familial thoracic 4	Strong
MYH14	Deafness, autosomal dominant	Strong
MYH2	Proximal myopathy and ophthalmoplegia	Strong
MYH3	Arthrogryposis, distal	Definitive
MYH6	Atrial septal defect	Limited
MYH6	Cardiomyopathy, dilated	Limited
MYH6	Cardiomyopathy, familial hypertrophic	Limited
MYH7	Cardiomyopathy, familial hypertrophic	Definitive
MYH7	Cardiomyopathy, dilated	Strong
MYH7	Laing distal myopathy	Definitive
MYH7	Left ventricular noncompaction	Strong
MYH7	Myopathy, myosin storage	Strong
MYH7	Scapuloperoneal syndrome, myopathic type	Limited
MYH7	Congenital fiber type disproportion	Moderate
MYH9	Macrothrombocytopenia and progressive sensorineural deafness	Strong
MYL2	Cardiomyopathy, familial hypertrophic, 10	Strong
MYL3	Cardiomyopathy, familial hypertrophic, 8	Strong
MYLK	Aortic aneurysm, familial thoracic 7	Moderate
MYLK2	Cardiomyopathy, hypertrophic	Limited

MYO15A	Sensorineural hearing loss	Definitive
MYO1C	Sensorineural hearing loss	Limited
MYO1E	Focal segmental glomerulosclerosis	Moderate
MYO1F	Sensorineural hearing loss	Limited
MYO3A	Sensorineural hearing loss	Strong
MYO5A	Griscelli syndrome	Moderate
MYO6	Deafness	Strong
MYO7A	Usher syndrome	Definitive
MYOM1	Cardiomyopathy, hypertrophic	Limited
MYOT	Myofibrillar myopathy	Strong
MYOZ2	Cardiomyopathy, hypertrophic	Limited
MYPN	Cardiomyopathy, dilated	Limited
MYPN	Cardiomyopathy, hypertrophic	Limited
NAA10	N-terminal acetyltransferase deficiency	Moderate
NAA15	Congenital heart disease	Limited
NAGA	N-acetylgalactosaminidase alpha deficiency	Strong
NAGLU	Sanfilippo syndrome type B	Definitive
NAGS	N-acetylglutamate synthetase deficiency	Strong
NBN	Nijmegen breakage syndrome	Strong
NCF1	Chronic granulomatous disease	Definitive
NCF2	Chronic granulomatous disease	Definitive
NCF4	Chronic granulomatous disease	Limited
NDP	Norrie disease	Definitive
NEB	Nemaline myopathy	Definitive
NEBL	Cardiomyopathy, dilated	Moderate
NEDD4L	Epilepsy, photosensitive generalised	Limited
NEFL	Charcot-Marie-Tooth disease	Strong
NEK1	Short rib-polydactyly syndorme, type II	Moderate
NEK8	Nephronophthisis	Moderate
NEU1	Sialidosis	Strong
NEUROG3	Diarrhea 4, malabsorptive, congenital	Moderate
NEXN	Cardiomyopathy, dilated	Limited
NEXN	Cardiomyopathy, familial hypertrophic	Limited
NF1	Neurofibromatosis, type 1	Definitive
NF2	Neurofibromatosis 2	Definitive
NFATC1	Congenital heart disease	Limited
NGLY1	Developmental delay, multifocal epilepsy & abnormal liver function	Strong
NHEJ1	Severe combined immunodeficiency with microcephaly, growth retardation, and sensitivity to ionizing radiation	Strong

NHLRC1	Myoclonic epilepsy of Lafora	Strong
NHP2	Dyskeratosis congenita	Limited
NIN	Seckel syndrome	Limited
NIPAL4	Ichthyosis, autosomal recessive	Strong
NIPBL	Cornelia de Lange syndrome	Definitive
NKX2-1	Choreoathetosis, hypothyroidism, and neonatal respiratory distress	Strong
NKX2-5	Congenital heart disease	Definitive
NKX3-2	Spondylo-megaepiphyseal-metaphyseal dysplasia	Moderate
NLGN3	Autism	Strong
NLGN4X	Autism	Strong
NLRP7	Hydatidiform mole	Definitive
NME8	Ciliary dyskinesia, primary	Limited
NOG	Symphalangism, proximal, 1A	Strong
NOP10	Dyskeratosis congenita	Moderate
NOTCH1	Aortic valve disease	Moderate
NOTCH2	Hajdu-Cheney syndrome	Strong
NOTCH3	Cerebral arteriopathy with subcortical infarcts and leukoencephalopathy	Definitive
NPC1	Niemann-Pick disease type C1	Definitive
NPC2	Niemann-Pick disease type C2	Strong
NPHP1	Nephronophthisis	Definitive
NPHP3	Nephronophthisis	Definitive
NPHP4	Nephronophthisis	Definitive
NPHS1	Congenital nephrotic syndrome, Finnish type	Definitive
NPPA	Atrial fibrillation	Limited
NR0B1	Congenital adrenal hypoplasia	Definitive
NR1H4	Cholestasis, infantile	Limited
NRG1	Hirschsprung disease	Limited
NRXN1	Autism	Strong
NSD1	Sotos syndrome	Definitive
NSDHL	CHILD syndrome	Strong
NSDHL	CK syndrome	Moderate
NTRK1	Congenital insensitivity to pain with anhidrosis	Definitive
NTRK1	Medullary thyroid carcinoma, familial	Limited
NUB1	Congenital heart disease	Limited
NUP155	Atrial fibrillation	Limited
NUP62	Striatonigral degeneration, infantile	Moderate
OBSL1	3-M syndrome	Strong
OCA2	Albinism, oculocutaneous	Definitive

OCRL	Lowe oculocerebrorenal syndrome	Definitive
OFD1	Oral-facial-digital syndrome	Definitive
OPA1	Optic atrophy 1	Definitive
OPA3	3-methylglutaconic aciduria, type III	Strong
OPA3	Optic atrophy 3 with cataract	Strong
ORC1	Meier-Gorlin syndrome	Strong
ORC4	Meier-Gorlin syndrome	Moderate
ORC6	Meier-Gorlin syndrome	Moderate
OSMR	Amyloidosis, primary cutaneous	Strong
OSTM1	Osteopetrosis	Strong
OTC	Ornithine transcarbamylase deficiency	Definitive
OTOA	Deafness, autosomal recessive	Strong
OTOF	Deafness, autosomal recessive	Definitive
OTOG	Deafness, autosomal recessive	Moderate
OTOGL	Deafness, autosomal recessive	Strong
OTUD4	Hypogonadotropic hypogonadism, ataxia & dementia	Limited
P2RX2	Hearing loss	Moderate
PABPN1	Oculopharyngeal muscular dystrophy	Strong
PAH	Phenylketonuria	Definitive
PAK3	Mental retardation syndrome, X-linked	Strong
PALB2	Breast cancer	Definitive
PANK2	Neurodegeneration with brain iron accumulation 1	Definitive
PAX3	Waardenburg syndrome	Definitive
PAX6	Aniridia	Definitive
PAX8	Hypothyroidism, congenital, due to thyroid dysgenesis or hypoplasia	Strong
PC	Pyruvate carboxylase deficiency	Definitive
PCCA	Propionicacidemia	Definitive
PCCB	Propionicacidemia	Definitive
PCDH15	Usher syndrome	Strong
PCNT	Microcephalic osteodysplastic primordial dwarfism type 2	Definitive
PCSK9	Hypercholesterolemia	Strong
PDE11A	Adrenocortical hyperplasia	Moderate
PDE4D	Acrodysostosis 2, with or without hormone resistance	Strong
PDHA1	Pyruvate dehydrogenase deficiency	Definitive
PDHX	Pyruvate dehydrogenase complex deficiency	Strong
PDLIM3	Cardiomyopathy, dilated	Limited
PDP1	Pyruvate dehydrogenase phosphatase deficiency	Limited
PDSS1	Deafness - encephaloneuropathy - obesity - valvulopathy Neonatal	Limited

PDSS2	Leigh syndrome with nephropathy and COQ10 deficiency	Limited
PEX1	Zellweger syndrome	Definitive
PEX10	Zellweger syndrome	Strong
PEX11B	Peroxisome biogenesis disorder	Limited
PEX12	Zellweger syndrome	Strong
PEX13	Zellweger syndrome	Strong
PEX14	Zellweger syndrome	Moderate
PEX16	Zellweger syndrome	Moderate
PEX19	Zellweger syndrome	Moderate
PEX2	Zellweger syndrome	Strong
PEX26	Zellweger syndrome	Strong
PEX3	Zellweger syndrome	Strong
PEX5	Zellweger syndrome	Strong
PEX6	Zellweger syndrome	Definitive
PEX7	Refsum disease	Strong
PEX7	Rhizomelic chondrodysplasia punctata	Definitive
PFKM	Glycogen storage disease 7	Definitive
PHF6	Borjeson-Forssman-Lehmann syndrome	Strong
PHKA1	Phosphorylase kinase deficiency	Moderate
PHKA2	Phosphorylase kinase deficiency	Strong
PHKB	Phosphorylase kinase deficiency	Strong
PHKG2	Phosphorylase kinase deficiency	Strong
PHOX2A	Fibrosis of extraocular muscles, congenital	Moderate
PHOX2B	Central hypoventilation syndrome	Definitive
PHYH	Refsum disease	Strong
PIEZO2	Arthrogryposis, distal, type 5	Strong
PIGA	Epileptic encephalopathy, early-onset	Moderate
PINK1	Parkinson disease 6, early onset	Definitive
PITX2B	Congenital heart disease	Limited
PITX2C	Atrial fibrillation	Limited
PKD1	Polycystic kidney disease	Definitive
PKD2	Polycystic kidney disease	Definitive
PKHD1	Polycystic kidney and hepatic disease	Definitive
PKLR	Pyruvate kinase deficiency	Definitive
PKP2	Arrhythmogenic right ventricular dysplasia 9	Definitive
PLA2G6	Infantile neuroaxonal dystrophy 1	Strong
PLCE1	Nephrotic syndrome	Strong
PLEC	Epidermolysis bullosa simplex	Strong
PLEC	Muscular dystrophy	Strong
PLG	Plasminogen deficiency	Strong

PLN	Cardiomyopathy, dilated	Moderate
PLN	Cardiomyopathy, familial hypertrophic	Limited
PLOD1	Ehlers-Danlos syndrome, kyphoscoliotic type	Strong
PLOD2	Bruck syndrome	Moderate
PLP1	Pelizaeus-Merzbacher disease	Definitive
PLP1	Spastic paraplegia 2, X-linked	Strong
PMM2	Congenital disorder of glycosylation, type Ia	Definitive
PMP22	Charcot-Marie-Tooth disease	Definitive
PMS2	Lynch syndrome	Strong
PNKD	Paroxysmal nonkinesiogenic dyskinesia	Definitive
PNKP	Microcephaly - seizures - developmental delay	Strong
PNPLA1	Ichthyosis, autosomal recessive congenital	Moderate
PNPO	Epileptic encephalopathy, neonatal	Strong
PODXL	Focal and segmental glomerulosclerosis	Limited
POLG	POLG-Related Ataxia Neuropathy Spectrum Disorders	Definitive
POLH	Xeroderma pigmentosum	Strong
POMC	Proopiomelanocortin deficiency	Moderate
POMGNT1	Muscular dystrophy-dystroglycanopathy (congenital with brain and eye anomalies)	Strong
POMGNT1	Muscular dystrophy-dystroglycanopathy (limb-girdle), type C, 3	Strong
POMT1	Muscular dystrophy-dystroglycanopathy (limb-girdle), type C, 1	Strong
POMT1	Walker-Warburg syndrome	Definitive
POMT2	Muscular dystrophy-dystroglycanopathy (limb-girdle), type C, 2	Strong
POR	Disordered steroidogenesis with and without Antley-Bixler syndrome	Definitive
PORCN	Focal dermal hypoplasia	Definitive
POU1F1	Pituitary hormone deficiency	Strong
POU3F4	Deafness, X-linked	Strong
POU4F3	Deafness, autosomal dominant	Strong
PPOX	Porphyria variegata	Definitive
PPT1	Neuronal ceroid lipofuscinosis	Definitive
PQBP1	Mental retardation	Strong
PRDM16	Left ventricular noncompaction	Limited
PREPL	Hypotonia - cystinuria syndrome	Moderate
PRF1	Hemophagocytic lymphohistiocytosis, familial, 2	Definitive
PRICKLE1	Epilepsy, progressive myoclonic 1B	Limited
PRKAG2	Cardiomyopathy, hypertrophic	Strong

PRKAG2	Wolff-Parkinson-White syndrome	Strong
PRKAG2	Glycogen storage disease of heart, lethal congenital	Limited
PRKAR1A	Carney complex	Definitive
PRKCSH	Polycystic liver disease	Strong
PROC	Thrombophilia due to protein C deficiency	Definitive
PRODH	Hyperprolinemia, type I	Moderate
PROKR2	Hypogonadotropic hypogonadism	Strong
PROP1	Pituitary hormone deficiency, combined, 2	Strong
PROS1	Protein S deficiency	Definitive
PRPS1	Arts syndrome	Moderate
PRPS1	Charcot-Marie-Tooth disease	Limited
PRRX1	Agnathia-otocephaly complex	Moderate
PRX	Charcot-Marie-Tooth disease	Strong
PSAP	Metachromatic leukodystrophy	Strong
PSAT1	Phosphoserine aminotransferase deficiency	Moderate
PSEN1	Alzheimer disease, type 3	Definitive
PSEN2	Alzheimer disease, type 4	Strong
PTCH1	Nevoid basal cell carcinoma syndrome	Definitive
PTEN	Cowden disease	Definitive
PTEN	Bannayan-Riley-Ruvalcaba syndrome	Definitive
PTH1R	Metaphyseal chondrodysplasia	Strong
PTPN11	Noonan syndrome	Definitive
PTRF	Lipodystrophy, congenital generalized, type 4	Strong
PTS	Hyperphenylalaninemia, BH4-deficient, A	Strong
PVRL1	Cleft lip / palate	Limited
PYGL	Glycogen storage disease VI	Strong
QDPR	Dihydropteridine reductase deficiency	Strong
RAB10	Congenital heart disease	Limited
RAB23	Carpenter syndrome	Strong
RAB27A	Griscelli syndrome	Strong
RAB3GAP1	Warburg micro syndrome	Strong
RAB3GAP2	Warburg micro syndrome	Moderate
RAB7A	Charcot-Marie-Tooth disease	Strong
RAD51B	Breast and/or ovarian cancer	Limited
RAF1	Noonan syndrome	Definitive
RAG1	Omenn syndrome	Definitive
RAG2	Omenn syndrome	Strong
RAI1	Smith-Magenis syndrome	Definitive
RAI1	Potocki-Lupski syndrome	Strong
RANGRF	Brugada syndrome	Limited

RAPSN	Congenital myasthenic syndrome	Strong
RASA1	Capillary malformation-arteriovenous malformation	Strong
RB1	Retinoblastoma	Definitive
RBM20	Cardiomyopathy, dilated, 1DD	Strong
RBM8A	Thrombocytopaenia-absent radius syndrome	Strong
RDX	Deafness, autosomal recessive	Moderate
RECQL4	Baller-Gerold syndrome	Strong
RECQL4	Rapadilino syndrome	Strong
RECQL4	Rothmund-Thomson syndrome	Definitive
RELN	Lissencephaly syndrome	Moderate
REN	Renal tubular dysgenesis	Strong
RET	Multiple endocrine neoplasia IIA	Definitive
RET	Multiple endocrine neoplasia IIB	Definitive
RFX6	Diabetes, neonatal, with intestinal atresia	Limited
RHAG	Rh-deficiency syndrome	Moderate
RMRP	Cartilage-hair hypoplasia	Strong
RNASEH2A	Aicardi-Goutieres syndrome	Strong
RNASEH2B	Aicardi-Goutieres syndrome	Strong
RNASEH2C	Aicardi-Goutieres syndrome	Strong
ROR2	Robinow syndrome	Strong
ROR2	Brachydactyly, type B1	Strong
RPGR	Retinitis pigmentosa	Definitive
RPGRIP1L	Meckel syndrome	Strong
RPGRIP1L	Joubert syndrome	Strong
RPL11	Diamond-Blackfan anemia	Strong
RPL35A	Diamond-Blackfan anemia	Moderate
RPL5	Diamond-Blackfan anemia	Strong
RPS10	Diamond-Blackfan anemia	Moderate
RPS15	Diamond-Blackfan anemia	Strong
RPS17	Diamond-Blackfan anemia	Strong
RPS19	Diamond-Blackfan anemia	Strong
RPS24	Diamond-Blackfan anemia	Strong
RPS26	Diamond-Blackfan anemia	Strong
RPS6KA3	Coffin-Lowry syndrome	Definitive
RPS7	Diamond-Blackfan anemia	Moderate
RRM2B	Mitochondrial DNA depletion syndrome	Strong
RS1	Retinoschisis, X linked	Strong
RSPH4A	Ciliary dyskinesia, primary	Strong
RSPH9	Ciliary dyskinesia, primary	Strong
RUNX2	Cleidocranial dysostosis	Definitive

RYR1	Central core disease	Strong
RYR1	Centronuclear myopathy	Definitive
RYR1	Malignant hyperthermia	Definitive
RYR1	Congenital fiber type disproportion	Moderate
RYR1	Multiminicore disease	Strong
RYR2	Arrhythmogenic right ventricular dysplasia 2	Definitive
RYR2	Ventricular tachycardia, catecholaminergic polymorphic	Definitive
SACS	Spastic ataxia Charlevoix-Saguenay type	Strong
SALL1	Townes-Brocks syndrome	Definitive
SAMHD1	Aicardi-Goutieres syndrome	Strong
SBDS	Shwachman-Bodian-Diamond syndrome	Strong
SC5D	Lathosterolosis	Moderate
SCN11A	Episodic pain syndrome	Strong
SCN1A	Dravet syndrome	Definitive
SCN1B	Brugada syndrome	Limited
SCN2B	Atrial fibrillation	Limited
SCN3B	Brugada syndrome	Limited
SCN4A	Hyperkalemic periodic paralysis, type 2	Strong
SCN4A	Hypokalemic periodic paralysis, type 2	Strong
SCN4B	Long QT syndrome	Limited
SCN5A	Long QT syndrome	Definitive
SCN5A	Brugada syndrome	Definitive
SCNN1A	Pseudohypoaldosteronism	Definitive
SCNN1B	Pseudohypoaldosteronism	Definitive
SCNN1B	Liddle syndrome	Definitive
SCNN1G	Pseudohypoaldosteronism	Moderate
SCO1	Hepatic failure, early onset, and neurologic disorder	Moderate
SCO2	Cardioencephalomyopathy, fatal infantile, due to cytochrome c oxidase deficiency	Strong
SCP2	Leukoencephalopathy - dystonia - motor neuropathy	Limited
SDHAF2	Hereditary Paraganglioma-Pheochromocytoma Syndromes	Moderate
SDHB	Hereditary Paraganglioma-Pheochromocytoma Syndromes	Definitive
SDHC	Hereditary Paraganglioma-Pheochromocytoma Syndromes	Strong
SDHD	Hereditary Paraganglioma-Pheochromocytoma Syndromes	Definitive
SEC63	Polycystic liver disease	Strong
SEMA3A	Kallmann syndrome 1	Moderate
SEPN1	Muscular dystrophy, rigid spine	Strong
SEPN1	Myopathy, congenital, with fiber-type disproportion	Strong
SEPT9	Amyotrophy, hereditary neuralgic	Strong
SERPINA1	Antitrypsin alpha 1 deficiency	Definitive

SERPINB6	Deafness, autosomal recessive	Limited
SERPINC1	Thrombophilia due to antithrombin III deficiency	Definitive
SERPIND1	Heparin cofactor 2 deficiency	Limited
SETBP1	Schinzel-Giedion syndrome	Strong
SETX	Ataxia-ocular apraxia 2	Strong
SFTPA2	Pulmonary fibrosis, idiopathic	Limited
SFTPB	Surfactant metabolism dysfunction, pulmonary	Definitive
SFTPC	Interstitial lung disease	Definitive
SGCA	Muscular dystrophy, limb-girdle, type 2D	Definitive
SGCB	Muscular dystrophy, limb-girdle, type 2E	Definitive
SGCD	Cardiomyopathy, dilated	Limited
SGCD	Muscular dystrophy, limb-girdle, type 2F	Definitive
SGCG	Muscular dystrophy, limb-girdle, type 2C	Strong
SGSH	Mucopolysaccharidisis type IIIA (Sanfilippo A)	Strong
SH2D1A	Lymphoproliferative syndrome	Definitive
SH3BP2	Cherubism	Strong
SH3TC2	Charcot-Marie-Tooth disease	Strong
SHANK3	Phelan-McDermid syndrome	Strong
SHH	Holoprosencephaly-3	Definitive
SHOC2	Noonan-like syndrome with loose anagen hair	Moderate
SIL1	Marinesco-Sjogren syndrome	Strong
SIX1	Branchiootorenal syndrome	Strong
SIX2	Renal hypodysplasia	Moderate
SIX3	Holoprosencephaly-2	Definitive
SIX5	Branchiootorenal syndrome	Moderate
SKI	Shprintzen-Goldberg syndrome	Strong
SLC11A2	Anemia, hypochromic microcytic	Moderate
SLC12A1	Bartter syndrome	Definitive
SLC12A3	Gitelman syndrome	Definitive
SLC12A5	Febrile seizures	Moderate
SLC12A6	Agenesis of the corpus callosum with peripheral neuropathy	Strong
SLC16A1	Monocarboxylate transporter 1 deficiency	Moderate
SLC16A12	Cataract, juvenile with microcornea and renal glucosuria	Limited
SLC16A2	Allan-Herndon-Dudley syndrome	Strong
SLC17A5	Sialic acid storage disorder, infantile	Strong
SLC19A2	Thiamine-responsive megaloblastic anemia syndrome	Strong
SLC19A3	Basal ganglia disease, biotin-responsive	Strong
SLC22A5	Carnitine deficiency, systemic primary	Definitive
SLC25A12	Hypomyelination, global cerebral	Limited

SLC25A13	Citrullinemia	Definitive
SLC25A15	Hyperornithinemia-hyperammonemia-homocitrullinemia syndrome	Strong
SLC25A20	Carnitine-acylcarnitine translocase deficiency	Strong
SLC25A22	Early myoclonic encephalopathy	Moderate
SLC25A38	Anemia, sideroblastic, pyridoxine-refractory, autosomal recessive	Definitive
SLC25A4	Progressive external ophthalmoplegia	Definitive
SLC26A2	Achondrogenesis 1B	Strong
SLC26A3	Chloride diarrhea, congenital, Finnish type	Definitive
SLC26A4	Pendred syndrome	Definitive
SLC27A4	Ichthyosis prematurity syndrome	Strong
SLC27A5	Bile acid amidation defect	Limited
SLC2A1	GLUT1 deficiency syndrome 1	Definitive
SLC2A10	Arterial tortuosity syndrome	Definitive
SLC33A1	Congenital cataracts, hearing loss and low serum copper and ceruloplasmin	Moderate
SLC33A1	Spastic paraplegia, autosomal dominant	Limited
SLC34A2	Pulmonary alveolar microlithiasis	Strong
SLC34A3	Hypophosphatemic rickets with hypercalciuria	Definitive
SLC35A1	CDG syndrome type IIf	Limited
SLC35A2	Early-onset epileptic encephalopathy	Moderate
SLC35C1	Congenital disorder of glycosylation 2c	Moderate
SLC35D1	Schneckenbecken dysplasia	Strong
SLC37A4	Glycogen storage disease Ib	Definitive
SLC39A4	Acrodermatitis enteropathica	Definitive
SLC3A1	Cystinuria	Definitive
SLC41A1	Parkinson disease, idiopathic	Limited
SLC45A2	Oculocutaneous albinism, type IV	Definitive
SLC46A1	Folate malabsorption, hereditary	Strong
SLC4A1	Spherocytosis	Strong
SLC4A10	Epilepsy & mental retardation	Moderate
SLC4A11	Corneal endothelial dystrophy	Definitive
SLC4A4	Renal tubular acidosis, proximal, with ocular abnormalities	Moderate
SLC5A2	Renal glucosuria	Definitive
SLC5A5	Thyroid dyshormonogenesis 1	Strong
SLC6A19	Hartnup disorder	Definitive
SLC6A2	Orthostatic intolerance	Limited
SLC6A5	Hyperekplexia 3	Strong
SLC6A8	Creatine deficiency syndrome, X-linked	Definitive

SLC7A7	Lysinuric protein intolerance	Definitive
SLC7A9	Cystinuria	Definitive
SLC9A3R1	Nephrolithiasis/osteoporosis, hypophosphatemic, 2	Limited
SLC9A6	Christianson syndrome	Strong
SLCO1B1	Hyperbilirubinemia, Rotor type, digenic	Moderate
SLCO1B3	Hyperbilirubinemia, Rotor type, digenic	Moderate
SLCO2A1	Hypertrophic osteoarthropathy, primary, autosomal recessive 2	Strong
SMAD1	Pulmonary arterial hypertension	Limited
SMAD3	Loeys-Dietz syndrome	Definitive
SMAD4	Juvenile polyposis syndrome	Definitive
SMAD6	Cardiovascular malformation, congenital	Limited
SMAD9	Pulmonary arterial hypertension	Limited
SMARCAL1	Schimke immunoosseous dysplasia	Definitive
SMC1A	Cornelia de Lange syndrome	Definitive
SMN1	Spinal muscular atrophy	Definitive
SMO	Medulloblastoma	Limited
SMPD1	Niemann-Pick disease, type A	Definitive
SMPD1	Niemann-Pick disease, type B	Definitive
SMPX	Deafness, X-linked	Strong
SNAP29	Cerebral dysgenesis, neuropathy, ichthyosis, and palmoplantar keratoderma syndrome	Moderate
SNTA1	Long QT syndrome	Moderate
SOD1	Amyotrophic lateral sclerosis	Definitive
SOX10	Shah-Waardenburg syndrome	Definitive
SOX18	Hypotrichosis-lymphedema-telangiectasia syndrome	Moderate
SOX9	Campomelic dysplasia	Definitive
SP110	Hepatic venoocclusive disease with immunodeficiency	Strong
SP7	Osteogenesis imperfecta, type XII	Limited
SPEG	Centronuclear myopathy with dilated cardiomyopathy	Moderate
SPINK5	Netherton syndrome; Netherton syndrome 1	Strong
SPR	Sepiapterin reductase deficiency	Strong
SPRED1	Legius syndrome	Definitive
SPTA1	Elliptocytosis	Strong
SPTB	Spherocytosis	Definitive
SPTLC1	Neuropathy, hereditary sensory and autonomic, type IA	Strong
SPTLC2	Neuropathy, hereditary sensory and autonomic, type IC	Moderate
SRCAP	Floating-Harbor syndrome	Strong
ST14	Ichthyosis hypotrichosis syndrome	Moderate
ST3GAL5	Amish infantile epilepsy syndrome	Moderate

STAC3	Myopathy, Native American	Moderate
STAR	Congenital lipoid adrenal hyperplasia,	Strong
STAT3	Hyper-IgE recurrent infection syndrome	Definitive
STK11	Peutz-Jeghers syndrome	Definitive
STRA6	Microphthalmia, syndromic	Strong
STRC	Deafness, autosomal recessive	Strong
STS	Ichthyosis, X-linked	Definitive
STX11	Hemophagocytic lymphohistiocytosis, familial, 4	Strong
STXBP1	Epileptic encephalopathy, early infantile	Strong
STXBP2	Hemophagocytic lymphohistiocytosis	Strong
SUCLA2	Mitochondrial DNA depletion syndrome 5 (encephalomyopathic with methylmalonic aciduria)	Strong
SUCLG1	Mitochondrial DNA depletion syndrome 9 (encephalomyopathic type with methylmalonic aciduria)	Strong
SUOX	Sulphite oxidase deficiency	Strong
SURF1	Leigh syndrome, due to COX deficiency	Definitive
SYNE4	Hearing loss	Limited
SYT14	Spinocerebellar ataxia, autosomal recessive 11	Limited
TAB2	Congenital heart disease, nonsyndromic	Limited
TARDBP	Amyotrophic lateral sclerosis type 10	Strong
TAT	Tyrosinemia, type II	Definitive
TAZ	Barth syndrome	Definitive
TBC1D24	Deafness, onychodystrophy, osteodystrophy, mental retardation, and seizures syndrome	Strong
TBCE	Hypoparathyroidism retardation dysmorphism syndrome	Moderate
TBX1	DiGeorge syndrome	Strong
TBX20	Congenital heart disease	Moderate
TBX5	Holt-Oram syndrome	Definitive
TCAP	Cardiomyopathy, dilated	Moderate
TCAP	Muscular dystrophy, limb-girdle, type 2G	Strong
TCIRG1	Osteopetrosis, infantile malignant	Definitive
TCOF1	Treacher Collins syndrome 1	Definitive
TCTN1	Joubert syndrome	Moderate
TCTN3	Joubert syndrome	Moderate
TECTA	Deafness	Definitive
TERC	Dyskeratosis congenita	Strong
TERT	Dyskeratosis congenita	Strong
TFAP2A	Branchiooculofacial syndrome	Strong
TFAP2B	Char syndrome	Strong
TFG	Hereditary motor and sensory neuropathy	Strong

TFR2	Hemochromatosis type 3	Strong
TG	Thyroid dyshormonogenesis 3	Definitive
TGFB1	Camurati-Engelmann disease	Strong
TGFB3	Arrhythmogenic right ventricular dysplasia	Limited
TGFBR1	Loeys-Dietz syndrome	Definitive
TGFBR2	Loeys-Dietz syndrome	Definitive
TGFBR3	Premature ovarian failure	Limited
TGIF1	Holoprosencephaly-4	Strong
TGM1	Ichthyosis, congenital, autosomal recessive	Strong
TGM5	Peeling skin syndrome, acral type	Strong
TH	Tyrosine hydroxylase deficiency	Strong
THBD	Haemolytic uraemic syndrome	Moderate
THBS1	Pulmonary hypertension	Limited
THRA	Hypothyroidism, congenital, nongoitrous, 6	Strong
THRB	Thyroid hormone resistance	Definitive
TIMM8A	Mohr-Tranebjaerg syndrome	Strong
TINF2	Dyskeratosis congenita	Strong
TJP2	Hypercholanemia, familial	Moderate
TK2	Mitochondrial DNA depletion syndrome	Strong
TMC1	Deafness	Strong
TMC8	Epidermodysplasia verruciformi	Limited
TMEM216	Joubert syndrome	Moderate
TMEM216	Meckel syndrome	Moderate
TMEM237	Joubert syndrome	Moderate
TMEM43	Arrhythmogenic right ventricular dysplasia 5	Strong
TMEM67	Joubert syndrome	Strong
TMEM67	Meckel syndrome	Strong
TMIE	Deafness, autosomal recessive	Strong
TMPO	Cardiomyopathy, dilated	Conflicting
TMPRSS3	Deafness, autosomal recessive	Strong
TNFRSF11B	Paget disease	Strong
TNFSF11	Osteopetrosis, autosomal recessive 2	Strong
TNNC1	Cardiomyopathy, dilated	Moderate
TNNI2	Distal arthrogryposis syndrome 2b	Definitive
TNNI3	Cardiomyopathy, dilated	Moderate
TNNI3	Familial hypertrophic cardiomyopathy	Strong
TNNT1	Nemaline myopathy, Amish type	Strong
TNNT2	Cardiomyopathy, dilated	Strong
TNNT2	Familial hypertrophic cardiomyopathy	Definitive
TNNT3	Arthyrgryposis, distal	Strong

TNXB	Ehlers-Danlos syndrome due to tenascin X deficiency	Moderate
TP53	Li-Fraumeni syndrome	Definitive
TPM1	Cardiomyopathy, hypertrophic	Strong
TPM2	Nemaline myopathy	Strong
TPM2	Arthrogryposis multiplex congenita, distal	Strong
TPM3	Nemaline myopathy	Strong
TPM3	Congenital fiber-type disproportion myopathy	Strong
TPO	Thyroid dyshormonogenesis 2A	Strong
TPP1	Neuronal ceroid lipofuscinosis	Definitive
TPRN	Deafness, autosomal recessive	Moderate
TRAPPC2	Spondyloepiphyseal dysplasia tarda	Definitive
TRDN	Catecholaminergic polymorphic ventricular tachycardia	Limited
TREX1	Aicardi-Goutieres syndrome 1	Strong
TRH	Thyrotropin-releasing hormone deficiency	Limited
TRHR	Thyrotropin-releasing hormone resistance, generalized	Limited
TRIM32	Muscular dystrophy, limb-girdle, type 2H	Strong
TRIM37	Mulibrey nanism syndrome	Strong
TRIOBP	Deafness, autosomal recessive	Strong
TRIP11	Achondrogenesis type 1A	Moderate
TRMU	Liver failure, transient infantile	Strong
TRPM2	ALS and Parkinson's disease	Limited
TRPM4	Cardiac conduction disease	Strong
TSC1	Tuberous sclerosis 1	Definitive
TSC2	Tuberous sclerosis 2	Definitive
TSEN54	Pontocerebellar hypoplasia type 4	Strong
TSFM	Combined oxidative phosphorylation deficiency	Moderate
TSHB	Hypothryoidism, congenital, nongoitrous 4	Strong
TSHR	Hypothyroidism	Definitive
TSPEAR	Sensorineural deafness	Limited
TSPYL1	Sudden infant death with dysgenesis of the testes syndrome	Limited
TTC21B	Bardet-Biedl syndrome	Moderate
TTC37	Trichohepatoenteric syndrome	Strong
TTC7A	Immunodeficiency, combined, with intestinal atresias	Strong
TTN	Centronuclear myopathy	Strong
TTN	Cardiomyopathy, dilated	Definitive
TTPA	Ataxia with isolated vitamin E deficiency	Strong
TTR	Amyloidosis, hereditary, transthyretin-related	Definitive
TUBA8	Polymicrogyria with optic nerve hypoplasia	Limited
TWIST1	Saethre-Chotzen syndrome	Definitive

TYMP	Mitochondrial DNA depletion syndrome	Definitive
TYR	Albinism, oculocutaneous 1	Definitive
UBA1	Spinal muscular atrophy, X-linked infantile	Moderate
UBR1	Johanson-Blizzard syndrome	Strong
UCP2	Hyperinsulinism	Limited
UGT1A1	Crigler-Najjar syndrome	Definitive
UGT1A4	Crigler-Najjar syndrome	Limited
UGT1A5	UDP glucuronosyltransferase deficiency	Limited
UMOD	Nephropathy	Definitive
UNC13D	Hemophagocytic lymphohistiocytosis, familial, 3	Strong
UQCRB	Mitochondrial complex III deficiency	Limited
UQCRQ	Mitochondrial complex III deficiency	Moderate
UROD	Porphyria, hepatoerythropoietic	Strong
UROS	Porphyria, congenital erythropoietic	Strong
USH1C	Usher syndrome 1	Strong
USH1G	Usher syndrome 1	Strong
USH2A	Usher syndrome 2	Definitive
VAMP1	Spastic ataxia	Limited
VCAN	Wagner syndrome	Strong
VCL	Cardiomyopathy, dilated	Strong
VCP	Inclusion body myopathy with early-onset paget disease and frontotemporal dementia	Strong
VDR	Vitamin D-dependent rickets	Definitive
VHL	von Hippel-Lindau syndrome	Definitive
VIPAS39	Arthrogryposis, renal dysfunction and cholestasis	Strong
VLDLR	Cerebellar hypoplasia and mental retardation with or without quadrupedal locomotion 1	Strong
VPS13A	Choreoacanthocytosis	Definitive
VPS13B	Cohen syndrome	Strong
VPS33B	Arthrogryposis renal dysfunction cholestasis syndrome	Strong
VPS53	Progressive cerebello-cerebral atrophy	Limited
VSX1	Keratoconus	Moderate
VWF	von Willebrand disease	Definitive
WAS	Wiskott-Aldrich syndrome	Definitive
WDR19	Nephronophthisis	Moderate
WDR35	Cranioectodermal dysplasia	Moderate
WDR36	Glaucoma	Limited
WDR62	Microcephaly 2, primary, autosomal recessive, with or without cortical malformations	Strong
WFS1	Wolfram syndrome	Definitive

WNK1	Neuropathy, hereditary sensory and autonomic, type I	Limited
WNT10A	Ectodermal dysplasia	Strong
WNT3	Tetra-amelia, autosomal recessive	Limited
WNT5A	Robinow syndrome	Moderate
WNT7A	Ulna and fibula absence of with severe limb deficiency	Moderate
WRAP53	Dyskeratosis congenita	Moderate
WRN	Werner syndrome	Definitive
WT1	Wilms tumor, type 1	Definitive
WT1	Denys-Drash syndrome	Definitive
WT1	Frasier syndrome	Definitive
XPA	Xeroderma pigmentosum	Definitive
XPC	Xeroderma pigmentosum	Strong
YARS2	Myopathy, lactic acidosis, and sideroblastic anemia	Moderate
ZAP70	ZAP70-related severe combined immunodeficiency	Strong
ZEB2	Mowat-Wilson syndrome	Strong
ZFPM2	Tetralogy of Fallot	Limited
ZIC2	Holoprosencephaly-5	Strong
ZIC3	Heterotaxy	Strong
ZMPSTE24	Restrictive dermopathy	Strong
ZNF252P	Hypothyroidism	Limited
ZNF469	Brittle cornea syndrome	Strong
ZNF674	Mental retardation	Limited

AR, autosomal recessive; AD, autosomal dominant; XLR, X-linked recessive;XLR, X-linked do
DERM, dermatologic disease; GLY, hypoglycinemia; HL, hearing loss; HYPOTO, hypotonia; IE

Typical inheritance	Penetrance	Age of onset <18 Yrs	BabySeq Category	Meets NGSR citeria?
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	MODERATE (B)	No	B	Yes
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	C	No
AR	LOW (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes

AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (B)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	MODERATE (B)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No

AD	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	LOW (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	No	B	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No

AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	No	C	No
AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
XLR	HIGH (B)	Yes	C	No
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	MODERATE (A)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	B	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes

AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	No	C	No
AD	MODERATE (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	B	Yes
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No

AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	LOW (A)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	B	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	MODERATE (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	MODERATE (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No

UNKNOWN	UNKNOWN	Yes	C	No
AD	MODERATE (B)	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	LOW (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
COMPLEX	MODERATE (B)	Yes	C	No

AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	No	B	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	LOW (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	No	B	Yes

AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	No	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (B)	Yes	B	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
UNKNOWN	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	LOW (B)	No	C	No
XLR	HIGH (A)	Yes	A	Yes

XLR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
XLR	MODERATE (A)	Yes	B	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	No	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	No	C	No
AR	MODERATE (A)	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	No	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No

AD	MODERATE (A)	No	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	LOW (A)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	MODERATE (B)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes

AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	No	C	No
AR	HIGH (A)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

XLR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	No	C	No
AD	UNKNOWN	No	C	No
AR	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	LOW (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	MODERATE (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No

AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
IMP	HIGH (A)	Yes	A	Yes
IMP	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
XLR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	MODERATE (B)	Yes	B	Yes
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	No	C	No
AR	HIGH (A)	Yes	A	Yes
IMP	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	LOW (A)	Yes	C	No
AR	UNKNOWN	Yes	C	No

AR	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	MODERATE (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	LOW (A)	Yes	C	No
AR	LOW (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	LOW (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	MODERATE (A)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
XLR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No

AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	No	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	No	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	No	B	Yes
AD	UNKNOWN	No	C	No
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	LOW (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
IMP	HIGH (B)	Yes	C	No
AD	MODERATE (A)	Yes	C	No
AD	MODERATE (A)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No

AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	LOW (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	LOW (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes

AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	C	No
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	MODERATE (A)	Yes	B	Yes
AR	MODERATE (A)	Yes	B	Yes
AR	MODERATE (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes

AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	MODERATE (A)	No	C	No
AD	MODERATE (A)	No	C	No
AR	UNKNOWN	Yes	C	No

AD	HIGH (A)	Yes	A	Yes
MITOCHOND	MODERATE (A)	Yes	C	No
MITOCHOND	MODERATE (A)	Yes	C	No
MITOCHOND	MODERATE (A)	Yes	C	No
AR	MODERATE (A)	Yes	B	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AD	UNKNOWN	No	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	No	B	Yes
AD	MODERATE (B)	Yes	B	Yes
UNKNOWN	UNKNOWN	Yes	C	No

AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	No	C	No
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
XLD	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	No	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	No	C	No
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (B)	Yes	C	No
COMPLEX	MODERATE (A)	Yes	C	No
COMPLEX	MODERATE (A)	Yes	C	No
AR	HIGH (A)	No	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	LOW (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
COMPLEX	MODERATE (A)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

XLR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR-DIGENIC	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	LOW (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	UNKNOWN	No	C	No
AR	UNKNOWN	Yes	C	No

AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
XLR	LOW (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

AD	MODERATE (B)	No	B	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	LOW (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	MODERATE (A)	No	B	Yes

AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	MODERATE (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
XLR	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	No	C	No
AD	HIGH (A)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No

AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes

AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	No	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	MODERATE (B)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	LOW (A)	Yes	C	No

AR	UNKNOWN	Yes	C	No
AD	MODERATE (A)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No

AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
XLD	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	LOW (A)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
XLD	HIGH (A)	Yes	A	Yes
AR-DIGENIC	HIGH (B)	Yes	C	No
AR-DIGENIC	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (B)	Yes	B	Yes
AD	HIGH (A)	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No

AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	No	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	No	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	MODERATE (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes

AR	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	MODERATE (A)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	C	No
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	No	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (B)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes

AR	HIGH (B)	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
XLR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
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AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	No	C	No
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	No	B	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes

AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
XLR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	No	C	No
AD	MODERATE (B)	No	C	No
AD	MODERATE (A)	Yes	B	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AD	UNKNOWN	No	C	No
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes

AR	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AR	UNKNOWN	Yes	C	No
AD	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	MODERATE (A)	Yes	B	Yes
AD	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AR	HIGH (B)	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
AD	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AD	HIGH (A)	Yes	A	Yes
XLR	HIGH (A)	Yes	A	Yes
AR	HIGH (A)	Yes	A	Yes
AD	UNKNOWN	Yes	C	No
AR	HIGH (A)	Yes	A	Yes
XLR	UNKNOWN	Yes	C	No

minant; yrs; years; BabySeq gene panels: AN_TH, anemia-thrombocy
M, inborn errors of metabolism; REN, renal disorder; RESP, respirato

Reason for BabySeq category	Pediatric disease gene in Bell 2011	Key references used in curation (PubMed ID)
Strong evidence for highly penetrant childhood-onset disease	Y	12730363, 11159947
Strong evidence for highly penetrant childhood-onset disease		22009580, 22206013
Moderate evidence for gene's role in disease		24808023
Moderate evidence for gene's role in disease		20052547, 9746906,
Strong evidence for highly penetrant childhood-onset disease	Y	19434086, 10712205
Strong evidence for highly penetrant childhood-onset disease		16641205, 16728712
Strong evidence for highly penetrant childhood-onset disease		9054934, 10958763, 9
Strong evidence for highly penetrant childhood-onset disease	Y	9806540, 15300568,
Strong evidence for highly penetrant childhood-onset disease		9419367, 17726488,
Moderate evidence for gene's role in disease		10196363, 11050011
Moderate penetrance, Not actionable in childhood		8235715, 9425227
Strong evidence for highly penetrant childhood-onset disease		10835643 10811882
Strong evidence for highly penetrant childhood-onset disease		14715863, 23275527
Limited evidence for gene's role in disease		17245405, 24439875
Moderate evidence, Actionable in childhood		15034580, 24503780
Strong evidence for highly penetrant childhood-onset disease		22610116, 24352916
Strong evidence for highly penetrant childhood-onset disease	Y	7904210, 11992258 ,
Moderate evidence for gene's role in disease		22922874, 23141461
Strong evidence for highly penetrant childhood-onset disease		11378826, 11099417
Strong evidence for highly penetrant childhood-onset disease		17304052, 12359132
Strong evidence for highly penetrant childhood-onset disease	Y	21057504, 22499348
Limited evidence for gene's role in disease	Y	24591516
Strong evidence for highly penetrant childhood-onset disease	Y	11349232, 22542437
Moderate penetrance, Not actionable in childhood	Y	16926354, 9499414,
Low penetrance	Y	20547083, 15615815
Strong evidence for highly penetrant childhood-onset disease	Y	7479827, 10077518,
Strong evidence for highly penetrant childhood-onset disease		17236799, 15877211
Limited evidence for gene's role in disease		20626622
Strong evidence for highly penetrant childhood-onset disease	Y	2359105, 22095942,
Moderate evidence for gene's role in disease		25351951, 22405087
Strong evidence for highly penetrant childhood-onset disease	Y	8040306, 18536048,
Strong evidence for highly penetrant childhood-onset disease		21841779, 21785126
Strong evidence for highly penetrant childhood-onset disease		10508519, 12601110
Moderate evidence for gene's role in disease		17387733, 15468086
Moderate penetrance, Actionable in childhood		17994018, 19409525,
Strong evidence for highly penetrant childhood-onset disease		23756437, 23649928

Limited evidence for gene's role in disease		10411937
Limited evidence for gene's role in disease		17611253, 17947298
Moderate evidence, Actionable in childhood		9563954, 22464770,
Moderate penetrance, Actionable in childhood		17611253
Limited evidence for gene's role in disease		17611253, 25201647
Strong evidence for highly penetrant childhood-onset disease		13680526, 19477959
Strong evidence for highly penetrant childhood-onset disease		22366783
Strong evidence for highly penetrant childhood-onset disease		24676022, 24337657
Strong evidence for highly penetrant childhood-onset disease		23434115, 25949529
Limited evidence for gene's role in disease		14567970
Moderate evidence, Actionable in childhood		17097056, 20022194
Strong evidence for highly penetrant childhood-onset disease		10700177, 12444222
Strong evidence for highly penetrant childhood-onset disease		19085907, 18203193
Limited evidence for gene's role in disease		9916847, 21864452
Strong evidence for highly penetrant childhood-onset disease		18312453, 17786384
Strong evidence for highly penetrant childhood-onset disease	Y	9758612, 11807006,
Limited evidence for gene's role in disease		22010916, 25804906
Strong evidence for highly penetrant childhood-onset disease	Y	14563640, 12576319
Moderate evidence for gene's role in disease	Y	10417273, 15373769,
Strong evidence for highly penetrant childhood-onset disease	Y	2090119, 18677313,
Strong evidence for highly penetrant childhood-onset disease		23001123, 25604658
Strong evidence for highly penetrant childhood-onset disease		15955093, 24950769
Strong evidence for highly penetrant childhood-onset disease		21963049, 26642971
Strong evidence for highly penetrant childhood-onset disease	Y	11174635, 1904874,
Strong evidence for highly penetrant childhood-onset disease	Y	11977176, 10982190
Moderate evidence for gene's role in disease	Y	21990100, 24849933
Strong evidence for highly penetrant childhood-onset disease		21990100, 24951643
Moderate evidence for gene's role in disease	Y	2359105, 22095942,
Moderate evidence for gene's role in disease	Y	2359105, 22095942,
Strong evidence for highly penetrant childhood-onset disease	Y	1703535, 10453743,
Strong evidence for highly penetrant childhood-onset disease	Y	16453322, 15322546
Limited evidence for gene's role in disease		20371604
Strong evidence for highly penetrant childhood-onset disease		23217327, 20362274
Moderate evidence, Actionable in childhood		17244780, 23371967
Strong evidence for highly penetrant childhood-onset disease	Y	10677297, 9398839,
Moderate evidence for gene's role in disease		12649162, 10233365
Limited evidence for gene's role in disease		18093912, 23174487
Strong evidence for highly penetrant childhood-onset disease		3198770, 8301429, 1
Limited evidence for gene's role in disease		15166380, 21979934

Moderate evidence for gene's role in disease		22729224, 23745724
Strong evidence for highly penetrant childhood-onset disease		7592563, 24323989,
Strong evidence for highly penetrant childhood-onset disease		9266687, 20025859,
Strong evidence for highly penetrant childhood-onset disease		21739576, 22411858
Limited evidence for gene's role in disease		19886994
Strong evidence for highly penetrant childhood-onset disease	Y	7485163, 2666627, 1
Moderate evidence for gene's role in disease		9700195, 2624476
Strong evidence for highly penetrant childhood-onset disease	Y	14635103, 9266358,
Moderate evidence for gene's role in disease		14615364, 2825199,
Strong evidence for highly penetrant childhood-onset disease	Y	8071980, 738900, 33
Strong evidence for highly penetrant childhood-onset disease	Y	14973782, 20679665
Moderate evidence for gene's role in disease		20080937, 22213132
Strong evidence for highly penetrant childhood-onset disease		15639192, 12093361
Limited evidence for gene's role in disease		12684507
Strong evidence for highly penetrant childhood-onset disease		10581255, 16053906
Strong evidence for highly penetrant childhood-onset disease	Y	10359825, 9710431,
Strong evidence for highly penetrant childhood-onset disease		1523502, 19648040,
Moderate evidence for gene's role in disease		15148656, 15945070
Strong evidence for highly penetrant childhood-onset disease	Y	17594715, 11941370
Strong evidence for highly penetrant childhood-onset disease		11773004, 11398099
Strong evidence for highly penetrant childhood-onset disease		19434086, 10712205
Strong evidence for highly penetrant childhood-onset disease	Y	3174660, 8954059, 9
Strong evidence for highly penetrant childhood-onset disease	Y	2328408, 12601111,
Strong evidence for highly penetrant childhood-onset disease		16319823, 11137991
Moderate evidence for gene's role in disease	Y	10594127, 12512044
Moderate evidence for gene's role in disease		10655068, 21576695
Strong evidence for highly penetrant childhood-onset disease		1483698, 7599636, 1
Strong evidence for highly penetrant childhood-onset disease		12590260, 22929189
Moderate evidence for gene's role in disease	Y	11331279, 1631143,
Strong evidence for highly penetrant childhood-onset disease	Y	6336599, 3297708, 8
Strong evidence for highly penetrant childhood-onset disease		7883994, 2961992, 1
Moderate penetrance, Actionable in childhood		16253912, 17242276
Strong evidence for highly penetrant childhood-onset disease		19449425, 20358596
Moderate evidence, Actionable in childhood		19525294, 19608030
Limited evidence for gene's role in disease		19608031
Strong evidence for highly penetrant childhood-onset disease		21211618, 21467542
Strong evidence for highly penetrant childhood-onset disease		21092923, 25089919,
Strong evidence for highly penetrant childhood-onset disease		20096397, 23606453
Moderate evidence for gene's role in disease		15124103, 23843187

Strong evidence for highly penetrant childhood-onset disease	Y	12214284, 14508707
Limited evidence for gene's role in disease		24791904
Strong evidence for highly penetrant childhood-onset disease		23403622, 16551969
Moderate evidence for gene's role in disease		21937992, 24700674
Strong evidence for highly penetrant childhood-onset disease		17938238, 1651174, 1
Strong evidence for highly penetrant childhood-onset disease		20105204, 8064829, 9
Strong evidence for highly penetrant childhood-onset disease		15805152, 8468533, 1
Limited evidence for gene's role in disease		11095479, 16094309
Adult-onset, Not actionable in childhood		1671712, 2111584, 14
Moderate penetrance, Not actionable in childhood		11532677, 20150536
Strong evidence for highly penetrant childhood-onset disease	Y	11176953, 11022012
Strong evidence for highly penetrant childhood-onset disease	Y	12838569, 10995865,
Adult-onset, Not actionable in childhood		2062380, 25449081,
Strong evidence for highly penetrant childhood-onset disease		14647276, 19384555
Strong evidence for highly penetrant childhood-onset disease		7649538, 6422160, 2
Limited evidence for gene's role in disease		21565291
Moderate evidence for gene's role in disease		21633362, 17893116
Moderate evidence for gene's role in disease		22426308, 23929686
Strong evidence for highly penetrant childhood-onset disease		22426308, 23929686
Limited evidence for gene's role in disease		18674751, 24168557
Strong evidence for highly penetrant childhood-onset disease		23849778, 24203976
Strong evidence for highly penetrant childhood-onset disease	Y	2574462, 2906225, 1
Strong evidence for highly penetrant childhood-onset disease	Y	1550123, 15324318,
Moderate penetrance, Not actionable in childhood	Y	12567415, 23470839
Strong evidence for highly penetrant childhood-onset disease	Y	12379852, 18462864
Limited evidence for gene's role in disease		14532329, 18173746
Strong evidence for highly penetrant childhood-onset disease	Y	12384776, 1594374,
Moderate evidence for gene's role in disease		24139043, 25227173
Strong evidence for highly penetrant childhood-onset disease	Y	8252036, 16437572,
Strong evidence for highly penetrant childhood-onset disease	Y	11941481, 2358466,
Limited evidence for gene's role in disease	Y	15114530
Strong evidence for highly penetrant childhood-onset disease	Y	9915942, 12497634,
Moderate evidence for gene's role in disease		7824105, 8929958, 2
Strong evidence for highly penetrant childhood-onset disease		18513263, 18644608,
Moderate penetrance, Not actionable in childhood		24523486, 17282997
Strong evidence for highly penetrant childhood-onset disease		10914677, 8841193,
Moderate evidence for gene's role in disease		15746149, 1976568
Strong evidence for highly penetrant childhood-onset disease	Y	22773132, 10406678

Strong evidence for highly penetrant childhood-onset disease		9916796, 12414817,
Strong evidence for highly penetrant childhood-onset disease	Y	17717039, 15981243
Strong evidence for highly penetrant childhood-onset disease		8914740, 7887410, 1
Limited evidence for gene's role in disease		19153371, 20170900
Strong evidence for highly penetrant childhood-onset disease	Y	8298639, 10441329,
Strong evidence for highly penetrant childhood-onset disease	Y	15239083, 9500542,
Moderate evidence for gene's role in disease	Y	8358044, 12640452,
Strong evidence for highly penetrant childhood-onset disease	Y	9326931, 18409179,
Strong evidence for highly penetrant childhood-onset disease	Y	112434311, 7130438
Strong evidence for highly penetrant childhood-onset disease		10820168, 8104196,
Limited evidence for gene's role in disease		24476074
Strong evidence for highly penetrant childhood-onset disease		18798333, 16909395
Moderate evidence for gene's role in disease		21763480, 23664117
Limited evidence for gene's role in disease	Y	11901181
Limited evidence for gene's role in disease		21763481
Strong evidence for highly penetrant childhood-onset disease		12704386
Moderate penetrance, Actionable in childhood		21898660, 21353195
Moderate evidence for gene's role in disease		19085932, 21361913
Moderate evidence for gene's role in disease		21549337, 21932319
Limited evidence for gene's role in disease		22912587
Strong evidence for highly penetrant childhood-onset disease		12118255, 12524598
Strong evidence for highly penetrant childhood-onset disease		16823392, 16582908
Strong evidence for highly penetrant childhood-onset disease		17160889, 20827784
Strong evidence for highly penetrant childhood-onset disease		11285252, 11567139
Strong evidence for highly penetrant childhood-onset disease		11381270, 12016587
Strong evidence for highly penetrant childhood-onset disease		15137946, 18203199
Strong evidence for highly penetrant childhood-onset disease		12567324, 9402160,
Strong evidence for highly penetrant childhood-onset disease		16380913, 20177705
Strong evidence for highly penetrant childhood-onset disease	Y	2703538, 9609836, 9
Strong evidence for highly penetrant childhood-onset disease	Y	2022752, 14517957,
Limited evidence for gene's role in disease		23665959
Strong evidence for highly penetrant childhood-onset disease	Y	11528392, 12215968,
Limited evidence for gene's role in disease		11840487
Strong evidence for highly penetrant childhood-onset disease		24482476, 23664116
Strong evidence for highly penetrant childhood-onset disease		20927630, 21129173
Strong evidence for highly penetrant childhood-onset disease	Y	7585968, 17407155,
Limited evidence for gene's role in disease		16385460, 22709368
Limited evidence for gene's role in disease		22461475, 21665000
Strong evidence for highly penetrant childhood-onset disease		11536076, 11381269

Limited evidence for gene's role in disease		24127225
Moderate penetrance, Actionable in childhood	Y	10973254, 6703480,
Limited evidence for gene's role in disease		21368915
Limited evidence for gene's role in disease		1421379, 25015942
Strong evidence for highly penetrant childhood-onset disease		16474404, 19206169
Moderate evidence for gene's role in disease		19206169
Adult-onset, Not actionable in childhood		7894491, 7894492, 7
Adult-onset, Not actionable in childhood		8524414, 11257103,
Strong evidence for highly penetrant childhood-onset disease		16115142, 16825431
Moderate penetrance, Not actionable in childhood		23553728, 15358725
Strong evidence for highly penetrant childhood-onset disease		12362029, 11479539
Strong evidence for highly penetrant childhood-onset disease		11687798, 16328537
Strong evidence for highly penetrant childhood-onset disease	Y	7550325, 2502673, 1
Strong evidence for highly penetrant childhood-onset disease	Y	16159644, 9445504,
Limited evidence for gene's role in disease		2340379
Strong evidence for highly penetrant childhood-onset disease	Y	21681116, 8133312,
Limited evidence for gene's role in disease		23716552
Moderate penetrance, Not actionable in childhood		20595690, 16621965
Strong evidence for highly penetrant childhood-onset disease	Y	9143915, 1301935, 1
Strong evidence for highly penetrant childhood-onset disease		19633872, 10371528
Moderate evidence, Actionable in childhood		17224476, 12676817
Limited evidence for gene's role in disease		21131953
Strong evidence for highly penetrant childhood-onset disease		10900517, 15761389
Limited evidence for gene's role in disease		9199552, 20861472,
Limited evidence for gene's role in disease		20817017, 25527503
Limited evidence for gene's role in disease		17224476, 12676817
Strong evidence for highly penetrant childhood-onset disease		7720071, 7670461, 1
Limited evidence for gene's role in disease		25787132, 25361775
Strong evidence for highly penetrant childhood-onset disease		19165920, 21735175
Moderate evidence for gene's role in disease		10412980, 16446975
Strong evidence for highly penetrant childhood-onset disease		21618644, 12386154
Limited evidence for gene's role in disease		14672715
Limited evidence for gene's role in disease		17060380, 23631430
Strong evidence for highly penetrant childhood-onset disease		9537420, 9536092, 1
Strong evidence for highly penetrant childhood-onset disease		15580566, 18930476
Moderate evidence for gene's role in disease		11431690, 12666119

Strong evidence for highly penetrant childhood-onset disease		20619386, 24493670
Strong evidence for highly penetrant childhood-onset disease	Y	10364517, 8554066,
Strong evidence for highly penetrant childhood-onset disease		18950740, 22241855
Moderate evidence for gene's role in disease		2258122, 22581229,
Strong evidence for highly penetrant childhood-onset disease		21131972, 22693285
Strong evidence for highly penetrant childhood-onset disease		22693285, 23255504
Limited evidence for gene's role in disease		17503326, 24875298
Limited evidence for gene's role in disease		22818856, 25635128
Moderate evidence for gene's role in disease		23042809, 23042809
Moderate evidence for gene's role in disease		17713465, 18443213
Low penetrance, non-disease trait, clinical relevance of CD36 deficiency is unce		11352982, 10890433
Strong evidence for highly penetrant childhood-onset disease	Y	8097258, 8550833, 7
Moderate penetrance, Not actionable in childhood		16621965, 17089378
Moderate evidence for gene's role in disease	Y	3981579, 17847009
Strong evidence for highly penetrant childhood-onset disease		16141353, 16098079
Limited evidence for gene's role in disease		7711721
Limited evidence for gene's role in disease		23197654, 26123647
Moderate penetrance, Actionable in childhood		10477433, 20373070,
Strong evidence for highly penetrant childhood-onset disease	Y	1785063, 11090341, 1
Strong evidence for highly penetrant childhood-onset disease		21940737, 21569298
Moderate evidence for gene's role in disease		15793586, 17764569
Strong evidence for highly penetrant childhood-onset disease	Y	22872100, 19793311
Strong evidence for highly penetrant childhood-onset disease		9341892, 20503313,
Moderate penetrance, Actionable in childhood		9425228, 21801156,
Moderate evidence for gene's role in disease		21802063, 26728615
Strong evidence for highly penetrant childhood-onset disease		12754508, 16307662
Moderate evidence for gene's role in disease		21358632, 21358631
Moderate evidence for gene's role in disease		21368133, 26648831
Moderate evidence for gene's role in disease		15793586, 16900296
Strong evidence for highly penetrant childhood-onset disease		21131973, 26436113
Strong evidence for highly penetrant childhood-onset disease	Y	17617513, 16682973
Moderate evidence for gene's role in disease		22246503
Moderate evidence for gene's role in disease		19584399, 20513133
Strong evidence for highly penetrant childhood-onset disease		18538293, 19853937
Limited evidence for gene's role in disease		16527897, 11457876
Moderate penetrance, Not actionable in childhood		12960213, 20513133
Moderate evidence for gene's role in disease		19745068, 19861685
Limited evidence for gene's role in disease		22626820, 16621965

Limited evidence for gene's role in disease		15562282, 10622723
Moderate evidence for gene's role in disease		20513133, 17000000
Moderate penetrance, Not actionable in childhood		16621965, 17599974
Strong evidence for highly penetrant childhood-onset disease		17160903, 22343409
Strong evidence for highly penetrant childhood-onset disease	Y	3141111, 10909851, 8
Strong evidence for highly penetrant childhood-onset disease	Y	10103316, 15528020
Strong evidence for highly penetrant childhood-onset disease		11172068, 3010100,
Strong evidence for highly penetrant childhood-onset disease		24834135, 24932903
Strong evidence for highly penetrant childhood-onset disease		2596527, 16207732,
Adult-onset, Not actionable in childhood		11479295, 12454775,
Strong evidence for highly penetrant childhood-onset disease		21665002, 23692895
Strong evidence for highly penetrant childhood-onset disease	Y	5146581, 17698759, 2
Limited evidence for gene's role in disease		18451336, 23743182
Strong evidence for highly penetrant childhood-onset disease	Y	15367858, 18707767
Moderate evidence for gene's role in disease		16826524, 21703448
Moderate evidence for gene's role in disease		8651643, 8872460, 2
Strong evidence for highly penetrant childhood-onset disease	Y	18398509, 16916845
Strong evidence for highly penetrant childhood-onset disease		7531341, 7538206, 8
Strong evidence for highly penetrant childhood-onset disease	Y	16826520, 22167768
Moderate evidence for gene's role in disease		18513679, 2253933,
Moderate evidence for gene's role in disease		21129728, 21129727
Moderate evidence for gene's role in disease		12417987, 11045837,
Moderate evidence for gene's role in disease		17846994, 10739754
Limited evidence for gene's role in disease		16287139, 24848765
Moderate penetrance, Not actionable in childhood		11840191,17932099,
Strong evidence for highly penetrant childhood-onset disease		8559248, 1372109, 1
Moderate penetrance, Not actionable in childhood		19953639, 16234969
Moderate evidence for gene's role in disease	Y	12164927, 15521008
Strong evidence for highly penetrant childhood-onset disease		11163249, 22246673
Strong evidence for highly penetrant childhood-onset disease	Y	17033971, 7947033,
Moderate evidence for gene's role in disease		22155368
Strong evidence for highly penetrant childhood-onset disease	Y	9311735, 21990111,
Strong evidence for highly penetrant childhood-onset disease	Y	9662406, 10477428,
Strong evidence for highly penetrant childhood-onset disease	Y	15996215, 11727201
Strong evidence for highly penetrant childhood-onset disease	Y	10508524, 15024724
Moderate evidence for gene's role in disease		23541340, 25956234
Strong evidence for highly penetrant childhood-onset disease	Y	7711740, 11524702
Strong evidence for highly penetrant childhood-onset disease	Y	10958649, 10466422,
Moderate penetrance, Not actionable in childhood		18179893, 20808228

Strong evidence for highly penetrant childhood-onset disease		9806553, 9931344, 2
Limited evidence for gene's role in disease		21185756, 19494034
Moderate evidence for gene's role in disease		23430875, 23228021
Moderate evidence for gene's role in disease		15107842, 17356545
Strong evidence for highly penetrant childhood-onset disease		110573014, 1528616
Strong evidence for highly penetrant childhood-onset disease	Y	7859284, 10677296,
Strong evidence for highly penetrant childhood-onset disease	Y	18374450, 2135794(
Strong evidence for highly penetrant childhood-onset disease		458828, 7023758, 94
Moderate penetrance, Not actionable in childhood		15864348, 15864348
Strong evidence for highly penetrant childhood-onset disease	Y	458828, 7023758, 21
Strong evidence for highly penetrant childhood-onset disease	Y	16752401, 17721977
Strong evidence for highly penetrant childhood-onset disease		8884076, 9399899, 1
Strong evidence for highly penetrant childhood-onset disease	Y	15954103, 12028435
Strong evidence for highly penetrant childhood-onset disease	Y	24052634, 24854265
Strong evidence for highly penetrant childhood-onset disease		2904407, 1635357, 1
Strong evidence for highly penetrant childhood-onset disease		22696272, 8673139,
Strong evidence for highly penetrant childhood-onset disease		9425231, 22696272 ,
Strong evidence for highly penetrant childhood-onset disease		15955946, 16278855
Strong evidence for highly penetrant childhood-onset disease		8782832, 20302629,
Strong evidence for highly penetrant childhood-onset disease		10399756, 11992252
Strong evidence for highly penetrant childhood-onset disease	Y	10827412, 9892921,
Limited evidence for gene's role in disease		21421862, 16909383
Limited evidence for gene's role in disease		21671392
Strong evidence for highly penetrant childhood-onset disease		3010100, 11865139,
Limited evidence for gene's role in disease	Y	16400613, 17855635
Moderate evidence for gene's role in disease		21540551, 24140869
Limited evidence for gene's role in disease		19268275
Adult-onset, Actionable in childhood		7708681, 7539672, 7
Moderate penetrance, Not actionable in childhood		7757079, 9843038, 7
Strong evidence for highly penetrant childhood-onset disease	Y	19793055, 17310273
Strong evidence for highly penetrant childhood-onset disease	Y	7014807, 9691089, 1
Strong evidence for highly penetrant childhood-onset disease	Y	12673791, 16996287
Limited evidence for gene's role in disease		22495306, 24859339
Limited evidence for gene's role in disease		26325596, 2632559
Strong evidence for highly penetrant childhood-onset disease		12749047, 20717166
Moderate evidence for gene's role in disease		21080147, 12632326
Strong evidence for highly penetrant childhood-onset disease	Y	17436252, 19012339,
Strong evidence for highly penetrant childhood-onset disease	Y	12110406, 17055431
Moderate evidence, Actionable in childhood		16793013, 16483541

Strong evidence for highly penetrant childhood-onset disease		14681890, 9731540,
Adult-onset, Not actionable in childhood		22197934, 23038421
Strong evidence for highly penetrant childhood-onset disease		18955570, 20622029
Limited evidence for gene's role in disease		21205713, 21075760
Limited evidence for gene's role in disease		18505755, 12507422
Moderate evidence, Actionable in childhood		18505755, 12642359
Moderate evidence for gene's role in disease		21944047, 23534700
Strong evidence for highly penetrant childhood-onset disease	Y	8596935, 20548044,
Strong evidence for highly penetrant childhood-onset disease		18005359, 9886310,
Moderate evidence for gene's role in disease		10439962
Limited evidence for gene's role in disease		11058912
Strong evidence for highly penetrant childhood-onset disease	Y	9537412, 9792862, 1
Strong evidence for highly penetrant childhood-onset disease	Y	16670177, 16685649
Strong evidence for highly penetrant childhood-onset disease	Y	10074491 , 17397052
Strong evidence for highly penetrant childhood-onset disease		22929189, 10080186
Strong evidence for highly penetrant childhood-onset disease		21364696, 21396581
Strong evidence for highly penetrant childhood-onset disease		10910929, 1415254,
Strong evidence for highly penetrant childhood-onset disease		1710153, 8634410, 1
Limited evidence for gene's role in disease		18345000, 24326104
Strong evidence for highly penetrant childhood-onset disease	Y	22968487, 19116240
Strong evidence for highly penetrant childhood-onset disease		2022736, 8506298, 2
Strong evidence for highly penetrant childhood-onset disease	Y	9556656, 9521938, 7
Strong evidence for highly penetrant childhood-onset disease	Y	7315872, 2019602, 7
Strong evidence for highly penetrant childhood-onset disease	Y	17488797, 9837822,
Strong evidence for highly penetrant childhood-onset disease		16436457, 10712205
Limited evidence for gene's role in disease		12093894
Limited evidence for gene's role in disease		9802883, 18367963
Strong evidence for highly penetrant childhood-onset disease	Y	20020533, 16037974
Moderate evidence for gene's role in disease		21388311, 24361964
Limited evidence for gene's role in disease		23665959
Moderate evidence for gene's role in disease		15060114, 11857564
Strong evidence for highly penetrant childhood-onset disease		8037208, 13813934,
Strong evidence for highly penetrant childhood-onset disease	Y	1916741, 10456341,
Adult-onset, Not actionable in childhood		15326253, 25109764,
Strong evidence for highly penetrant childhood-onset disease	Y	19342486, 18685874

Limited evidence for gene's role in disease	Y	20726879
Strong evidence for highly penetrant childhood-onset disease	Y	20346687, 11338401
Strong evidence for highly penetrant childhood-onset disease	Y	17240182, 15079002
Moderate evidence for gene's role in disease		24989667, 23176821
Limited evidence for gene's role in disease		22305527
Moderate evidence for gene's role in disease		19110212, 20223752
Limited evidence for gene's role in disease		2332510, 19578400
Moderate penetrance, Actionable in childhood		17325244, 10430757
Strong evidence for highly penetrant childhood-onset disease		14711882, 9697706,
Strong evidence for highly penetrant childhood-onset disease		9771715, 17868390,
Moderate evidence for gene's role in disease		24194196, 15841483
Strong evidence for highly penetrant childhood-onset disease		17373699, 17718865
Moderate penetrance, Not actionable in childhood		23542698, 25135762
Strong evidence for highly penetrant childhood-onset disease	Y	12205643, 15883261
Moderate evidence for gene's role in disease		11519011, 21671375
Strong evidence for highly penetrant childhood-onset disease	Y	15954111, 9653161,
Limited evidence for gene's role in disease		21722859, 26969326
Moderate evidence for gene's role in disease		9360932, 22938506,
Moderate penetrance, Actionable in childhood	Y	18005359, 9886310,
Limited evidence for gene's role in disease		24587289, 24476948
Strong evidence for highly penetrant childhood-onset disease	Y	8037208, 13813934,
Strong evidence for highly penetrant childhood-onset disease	Y	12746394, 12791036
Strong evidence for highly penetrant childhood-onset disease	Y	19945913, 19937601
Strong evidence for highly penetrant childhood-onset disease		3384440, 19937601,
Moderate penetrance, Actionable in childhood		9170393, 12565910,
Strong evidence for highly penetrant childhood-onset disease	Y	17033625, 22695891
Strong evidence for highly penetrant childhood-onset disease		1310900, 16285929,
Strong evidence for highly penetrant childhood-onset disease		19944405, 19944400
Moderate evidence for gene's role in disease		1905262, 24498942
Limited evidence for gene's role in disease		22387996
Strong evidence for highly penetrant childhood-onset disease		22184204, 24450482
Strong evidence for highly penetrant childhood-onset disease		11788826, 16627867
Strong evidence for highly penetrant childhood-onset disease		11713099, 16858015
Moderate evidence for gene's role in disease		18950741, 23261302
Strong evidence for highly penetrant childhood-onset disease		21376592, 2236678
Limited evidence for gene's role in disease	Y	22797137
Adult-onset, Not actionable in childhood		21820099, 22073189
Limited evidence for gene's role in disease		21496787
Strong evidence for highly penetrant childhood-onset disease		22396310, 17932957
Strong evidence for highly penetrant childhood-onset disease		15731758, 19502294

Strong evidence for highly penetrant childhood-onset disease	Y	11102980 , 21559330
Strong evidence for highly penetrant childhood-onset disease		20004785, 20622910
Strong evidence for highly penetrant childhood-onset disease		18626973, 16917026
Moderate evidence for gene's role in disease	Y	23890587, 22242004
Strong evidence for highly penetrant childhood-onset disease	Y	22742743, 23249953
Moderate evidence for gene's role in disease	Y	10642597, 10642602
Limited evidence for gene's role in disease		19285295
Moderate penetrance, Not actionable in childhood	Y	19296131, 10071185
Moderate penetrance, Actionable in childhood		20031616, 17033975
Moderate penetrance, Actionable in childhood		17505751, 1650573,
Moderate penetrance, Actionable in childhood	Y	12373648, 21606390
Strong evidence for highly penetrant childhood-onset disease		16175511, 20302578
Limited evidence for gene's role in disease		23105016
Limited evidence for gene's role in disease		18506004, 11238270
Limited evidence for gene's role in disease		12923531, 23364359
Strong evidence for highly penetrant childhood-onset disease		16134168, 15863666
Moderate evidence for gene's role in disease		21367925, 18042646
Strong evidence for highly penetrant childhood-onset disease		8012357, 16087766,
Strong evidence for highly penetrant childhood-onset disease		8808603, 3942856, 1
Limited evidence for gene's role in disease		9915973
Strong evidence for highly penetrant childhood-onset disease	Y	9683615, 18510547,
Strong evidence for highly penetrant childhood-onset disease		16329325, 18231121
Strong evidence for highly penetrant childhood-onset disease		17354266, 11780064
Moderate evidence for gene's role in disease	Y	20009762, 9587491,
Moderate evidence for gene's role in disease		20127975, 8630502,
Moderate penetrance, Not actionable in childhood	Y	17009072 , 20009762
Moderate penetrance, Not actionable in childhood		19764031, 20127975
Moderate evidence for gene's role in disease	Y	17937443, 16685658
Strong evidence for highly penetrant childhood-onset disease		15258581, 17159113
Strong evidence for highly penetrant childhood-onset disease		24470203, 22305528
Strong evidence for highly penetrant childhood-onset disease	Y	10502832, 9537424,
Strong evidence for highly penetrant childhood-onset disease	Y	19837917, 15220213
Moderate evidence for gene's role in disease		11835386, 18263758
Strong evidence for highly penetrant childhood-onset disease		10581030, 14962902
Strong evidence for highly penetrant childhood-onset disease		8096434, 11175284,
Strong evidence for highly penetrant childhood-onset disease		7894480, 10377322,
Strong evidence for highly penetrant childhood-onset disease		16752392, 7802026,
Strong evidence for highly penetrant childhood-onset disease	Y	12881724, 15605415
Moderate evidence for gene's role in disease		1558976, 8547605, 8

Adult-onset, Not actionable in childhood		17539898, 15872200
Limited evidence for gene's role in disease		12878321
Strong evidence for highly penetrant childhood-onset disease	Y	12019207, 15781812
Limited evidence for gene's role in disease	Y	17701904, 15378541
Limited evidence for gene's role in disease		21612988, 23623389
Strong evidence for highly penetrant childhood-onset disease	Y	1372108, 1757099, 9
Moderate evidence for gene's role in disease	Y	16947863, 26884178
Moderate evidence for gene's role in disease	Y	9580660, 8797827, 2
Strong evidence for highly penetrant childhood-onset disease	Y	12060391, 7951246,
Strong evidence for highly penetrant childhood-onset disease	Y	641373, 16865293, 1
Strong evidence for highly penetrant childhood-onset disease	Y	641373, 16865293, 1
Strong evidence for highly penetrant childhood-onset disease	Y	19574259, 15821733
Moderate evidence for gene's role in disease		15286153, 15930085
Strong evidence for highly penetrant childhood-onset disease		21802533, 22951369
Strong evidence for highly penetrant childhood-onset disease	Y	1882842, 1430199, 1
Strong evidence for highly penetrant childhood-onset disease	Y	7912128, 12815589,
Strong evidence for highly penetrant childhood-onset disease	Y	17584774, 21347544
Strong evidence for highly penetrant childhood-onset disease	Y	20978941, 14732903
Strong evidence for highly penetrant childhood-onset disease		10700184, 23220543
Strong evidence for highly penetrant childhood-onset disease		23220543, 19810119
Strong evidence for highly penetrant childhood-onset disease		77726168, 8317501,
Strong evidence for highly penetrant childhood-onset disease		9463333, 15586175,
Strong evidence for highly penetrant childhood-onset disease		19206155, 12404110
Strong evidence for highly penetrant childhood-onset disease		11159937, 17568404
Strong evidence for highly penetrant childhood-onset disease		22177091, 22190405
Strong evidence for highly penetrant childhood-onset disease	Y	3369441, 10606881,
Strong evidence for highly penetrant childhood-onset disease		1334372, 1421398, 1
Low penetrance	Y	8164741, 8979136, 1
Strong evidence for highly penetrant childhood-onset disease		15741993, 11857744
Strong evidence for highly penetrant childhood-onset disease		9556658, 5420360, 7
Limited evidence for gene's role in disease		23352160
Strong evidence for highly penetrant childhood-onset disease	Y	11209059, 15759101
Moderate evidence for gene's role in disease		24268661, 26471370
Strong evidence for highly penetrant childhood-onset disease	Y	17683097, 21911699
Moderate evidence for gene's role in disease		19838196, 24327336
Strong evidence for highly penetrant childhood-onset disease		20705279, 20705278,
Strong evidence for highly penetrant childhood-onset disease	Y	17924334, 19250384
Strong evidence for highly penetrant childhood-onset disease		18297069, 20848651

Strong evidence for highly penetrant childhood-onset disease		15790592, 15522956
Strong evidence for highly penetrant childhood-onset disease	Y	15502827, 23613520
Strong evidence for highly penetrant childhood-onset disease		8128956, 16429406,
Strong evidence for highly penetrant childhood-onset disease		17436244, 11239453
Moderate evidence for gene's role in disease		11001585, 17924555
Moderate evidence for gene's role in disease		10615118, 16084127
Strong evidence for highly penetrant childhood-onset disease		12552564, 11093276
Strong evidence for highly penetrant childhood-onset disease		17452773, 17460694
Moderate evidence for gene's role in disease		23613520, 25754594
Limited evidence for gene's role in disease		16116422, 21681190
Adult-onset, moderate evidence, moderate penetrance	Y	15269314, 20007835
Strong evidence for highly penetrant childhood-onset disease		19401719, 12189163
Strong evidence for highly penetrant childhood-onset disease		22461464, 17568394
Moderate evidence for gene's role in disease		12525539, 17718856
Limited evidence for gene's role in disease		8563763, 16333834,
Strong evidence for highly penetrant childhood-onset disease		10797416, 11754102
Strong evidence for highly penetrant childhood-onset disease	Y	10891444, 11354637
Strong evidence for highly penetrant childhood-onset disease	Y	10666208, 12393540
Strong evidence for highly penetrant childhood-onset disease		20082460, 11093277
Strong evidence for highly penetrant childhood-onset disease	Y	175649172, 1756495
Strong evidence for highly penetrant childhood-onset disease		18701883, 21480479
Strong evidence for highly penetrant childhood-onset disease		7874169, 12627230,
Strong evidence for highly penetrant childhood-onset disease	Y	7719345, 9475591, 9
Strong evidence for highly penetrant childhood-onset disease		7719344, 9700203, 1
Strong evidence for highly penetrant childhood-onset disease		10712195, 20635358
Strong evidence for highly penetrant childhood-onset disease		8696350, 19610084,
Strong evidence for highly penetrant childhood-onset disease		7874170, 7806229, 8
Strong evidence for highly penetrant childhood-onset disease		16140722, 20081435
Strong evidence for highly penetrant childhood-onset disease		7670477, 9450868, 1
Strong evidence for highly penetrant childhood-onset disease		11426459, 8880573,
Strong evidence for highly penetrant childhood-onset disease		2596513, 8589699, 9
Strong evidence for highly penetrant childhood-onset disease		9042914, 9279753, 1
Limited evidence for gene's role in disease		17033969, 27139183
Limited evidence for gene's role in disease		16501574, 1415342
Strong evidence for highly penetrant childhood-onset disease	Y	10891444, 11354637
Adult-onset, Not actionable in childhood		16597677, 20618355
Strong evidence for highly penetrant childhood-onset disease	Y	9635293, 11865300,
Limited evidence for gene's role in disease		18274675, 22094483
Strong evidence for highly penetrant childhood-onset disease		19716112, 19687455

Limited evidence for gene's role in disease		25358972
Limited evidence for gene's role in disease		20210997
Strong evidence for highly penetrant childhood-onset disease	Y	14523375, 1266624,
Strong evidence for highly penetrant childhood-onset disease		15121789, 11320179
Strong evidence for highly penetrant childhood-onset disease	Y	17878207, 10545611
Strong evidence for highly penetrant childhood-onset disease		14627679, 10545611
Strong evidence for highly penetrant childhood-onset disease		18505456, 15657874
Moderate penetrance, Not actionable in childhood		16444271, 19037238
Strong evidence for highly penetrant childhood-onset disease	Y	1733165, 16596676
Adult-onset, moderate evidence		19050726, 14711882
Non-disease phenotype		9398858, 9536088, 1
Strong evidence for highly penetrant childhood-onset disease		17197537, 12210347
Strong evidence for highly penetrant childhood-onset disease		19760751, 12114478,
Moderate evidence for gene's role in disease		12165566, 16882747
Strong evidence for highly penetrant childhood-onset disease		19500772, 23505205
Limited evidence for gene's role in disease		19276632
Limited evidence for gene's role in disease		18538293, 19933292
Moderate evidence for gene's role in disease	Y	10206641, 8911612,
Strong evidence for highly penetrant childhood-onset disease	Y	11137993, 14671208
Strong evidence for highly penetrant childhood-onset disease	Y	17163535, 12766769
Moderate penetrance, Not actionable in childhood		21507892, 19732862
Moderate evidence for gene's role in disease	Y	18203166, 16894541
Limited evidence for gene's role in disease		24265693
Limited evidence for gene's role in disease		12815595
Strong evidence for highly penetrant childhood-onset disease		19176363, 7669675,
Strong evidence for highly penetrant childhood-onset disease	Y	2012122, 7095811, 1
Strong evidence for highly penetrant childhood-onset disease		7397485, 2899844, 1
Strong evidence for highly penetrant childhood-onset disease		10447271, 10612834
Strong evidence for highly penetrant childhood-onset disease	Y	19118303, 20799326,
Strong evidence for highly penetrant childhood-onset disease	Y	7949118, 9427729, 2
Strong evidence for highly penetrant childhood-onset disease	Y	15985590, 2403755,
Moderate penetrance, Not actionable in childhood		11992121, 21703448
Moderate penetrance, Not actionable in childhood		11748509, 11326274
Strong evidence for highly penetrant childhood-onset disease	Y	8940268, 9272171, 7
Strong evidence for highly penetrant childhood-onset disease		12705493, 10521295
Strong evidence for highly penetrant childhood-onset disease		9298823, 15241807,
Strong evidence for highly penetrant childhood-onset disease	Y	6262213, 10408771,
Strong evidence for highly penetrant childhood-onset disease		11062483, 12655563

Limited evidence for gene's role in disease	Y	17148589, 25251786
Strong evidence for highly penetrant childhood-onset disease		11809723, 10700180
Strong evidence for highly penetrant childhood-onset disease		12845333, 15863664
Moderate evidence, Actionable in childhood		23295592, 23175127
Limited evidence for gene's role in disease		22750565, 22257684
Limited evidence for gene's role in disease		21965549
Strong evidence for highly penetrant childhood-onset disease	Y	18338393, 2495719,
Strong evidence for highly penetrant childhood-onset disease	Y	15452297, 15019703
Adult-onset, Not actionable in childhood, nontreatable disease, but allelic disod		8494336, 9851430, 1
Strong evidence for highly penetrant childhood-onset disease	Y	8139602, 8552212, 1
Moderate penetrance, Actionable in childhood		9778264, 11346370,
Strong evidence for highly penetrant childhood-onset disease		21454522, 12941786
Limited evidence for gene's role in disease		10515893, 10733484
Limited evidence for gene's role in disease	Y	11450847, 1671321,
Strong evidence for highly penetrant childhood-onset disease		11166163, 15805163
Limited evidence for gene's role in disease		17924340
Moderate evidence for gene's role in disease		8896569, 8968758, 8
Limited evidence for gene's role in disease		9497256, 8896569
Strong evidence for highly penetrant childhood-onset disease		11138011, 15732097
Limited evidence for gene's role in disease		19409522, 25269795
Strong evidence for highly penetrant childhood-onset disease	Y	15537906, 16632485
Strong evidence for highly penetrant childhood-onset disease		21310273, 23794683
Strong evidence for highly penetrant childhood-onset disease		21326233, 21660509
Strong evidence for highly penetrant childhood-onset disease	Y	12457340, 19338053
Moderate penetrance, Actionable in childhood		16790700, 20650941
Strong evidence for highly penetrant childhood-onset disease		1674715, 17353473,
Strong evidence for highly penetrant childhood-onset disease	Y	15994881, 21776002
Strong evidence for highly penetrant childhood-onset disease		8736341, 12372058,
Strong evidence for highly penetrant childhood-onset disease	Y	15192806, 22669416
Strong evidence for highly penetrant childhood-onset disease	Y	17371887, 12519371
Strong evidence for highly penetrant childhood-onset disease	Y	131309, 10841810, 1
Strong evidence for highly penetrant childhood-onset disease	Y	6336599, 3297708, 1
Moderate evidence for gene's role in disease	Y	18204449, 24961629
Moderate evidence for gene's role in disease		21940735, 17096318
Strong evidence for highly penetrant childhood-onset disease	Y	12794692, 18000979
Moderate evidence for gene's role in disease		16715098, 21139041

Strong evidence for highly penetrant childhood-onset disease		16832093, 1425575,
Moderate evidence for gene's role in disease		11929858, 23182654
Strong evidence for highly penetrant childhood-onset disease		10871207, 9571255,
Moderate evidence for gene's role in disease		16267323, 21353613
Moderate evidence for gene's role in disease		24035193
Strong evidence for highly penetrant childhood-onset disease		23281139, 23884777
Strong evidence for highly penetrant childhood-onset disease		23281139, 23884777
Strong evidence for highly penetrant childhood-onset disease	Y	11528398, 12473780
Strong evidence for highly penetrant childhood-onset disease	Y	16630736, 16200072
Strong evidence for highly penetrant childhood-onset disease		10712439, 19370764
Strong evidence for highly penetrant childhood-onset disease	Y	12573255, 17998446
Strong evidence for highly penetrant childhood-onset disease		10814714, 16010674
Limited evidence for gene's role in disease		21082656, 9787072,
Moderate evidence for gene's role in disease		19481194
Moderate evidence, Actionable in childhood		17967972, 12676817
Limited evidence for gene's role in disease		12684523
Strong evidence for highly penetrant childhood-onset disease		17516023, 1355560,
Strong evidence for highly penetrant childhood-onset disease		15044805, 16240336
Strong evidence for highly penetrant childhood-onset disease	Y	19357117, 9624053,
Strong evidence for highly penetrant childhood-onset disease		22578326, 22987632
Limited evidence for gene's role in disease		7829093
Limited evidence for gene's role in disease		12393799, 23813623
Strong evidence for highly penetrant childhood-onset disease	Y	2040928, 10484776,
Moderate penetrance, Not actionable in childhood		20384727, 23933819
Moderate evidence for gene's role in disease		20137778, 25802247
Strong evidence for highly penetrant childhood-onset disease	Y	11445798, 15717202
Moderate evidence for gene's role in disease	Y	15220921, 23562818
Moderate evidence for gene's role in disease		22521417, 25370039
Strong evidence for highly penetrant childhood-onset disease	Y	9099834, 8644704, 1
Adult-onset, moderate evidence		25272951, 20357282
Strong evidence for highly penetrant childhood-onset disease		9691087, 12072888,
Strong evidence for highly penetrant childhood-onset disease		15314640, 24154661
Moderate evidence for gene's role in disease	Y	16176262, 1835339,
Strong evidence for highly penetrant childhood-onset disease		21252247, 11489939
Strong evidence for highly penetrant childhood-onset disease	Y	10518286, 9003853,
Strong evidence for highly penetrant childhood-onset disease	Y	8651282, 12754706,
Low penetrance	Y	12915468, 15198949
Limited evidence for gene's role in disease		22279524

Limited evidence for gene's role in disease		21464306
Limited evidence for gene's role in disease		24558368
Strong evidence for highly penetrant childhood-onset disease	Y	21381239, 17129226
Strong evidence for highly penetrant childhood-onset disease		21381239, 20854116
Strong evidence for highly penetrant childhood-onset disease	Y	11283697, 20233970
Moderate evidence for gene's role in disease		16059943, 17033964
Limited evidence for gene's role in disease		12676817, 11901046
Strong evidence for highly penetrant childhood-onset disease		22885700, 24403048
Limited evidence for gene's role in disease		23040496, 24307375
Limited evidence for gene's role in disease		24927284, 23065719,
Moderate evidence for gene's role in disease	Y	14561704, 11748154,
Strong evidence for highly penetrant childhood-onset disease	Y	3156697, 8747922, 1
Strong evidence for highly penetrant childhood-onset disease	Y	2525553, 2973515, 2
Low penetrance	Y	10401000, 9341868, 1
Low penetrance	Y	14647275, 14982873,
Strong evidence for highly penetrant childhood-onset disease	Y	9154114, 10482952, 2
Limited evidence for gene's role in disease		19576567
Strong evidence for highly penetrant childhood-onset disease	Y	19479962, 16960811
Moderate evidence for gene's role in disease	Y	17160907, 25591832,
Strong evidence for highly penetrant childhood-onset disease		22961002, 24105373
Moderate evidence for gene's role in disease		12393545, 7655856,
Strong evidence for highly penetrant childhood-onset disease		16134170, 11585745
Low penetrance		12372055, 2789372,
Strong evidence for highly penetrant childhood-onset disease	Y	3128690, 3063529, 8
Moderate penetrance, Not actionable in childhood		15930087, 22706971
Moderate penetrance, Not actionable in childhood		20164212, 23348805
Limited evidence for gene's role in disease		23574532
Limited evidence for gene's role in disease		16155570
Moderate evidence for gene's role in disease		9343288, 7774914, 1
Strong evidence for highly penetrant childhood-onset disease	Y	1266847, 2071157, 1
Strong evidence for highly penetrant childhood-onset disease		10768343, 9497254,
Strong evidence for highly penetrant childhood-onset disease		11455388, 11590544
Strong evidence for highly penetrant childhood-onset disease		12664304, 11836498
Strong evidence for highly penetrant childhood-onset disease		12548288, 15296495
Moderate evidence for gene's role in disease		19843503, 12548288
Strong evidence for highly penetrant childhood-onset disease		8882404, 16170316,
Strong evidence for highly penetrant childhood-onset disease	Y	12696021, 11102558
Strong evidence for highly penetrant childhood-onset disease		8075637, 8550739, 9
Strong evidence for highly penetrant childhood-onset disease	Y	16385454, 11165012

Strong evidence for highly penetrant childhood-onset disease		12679481, 17645593
Strong evidence for highly penetrant childhood-onset disease		15122253, 15565283
Strong evidence for highly penetrant childhood-onset disease	Y	16927315, 11279527
Strong evidence for highly penetrant childhood-onset disease		11889251, 19387015,
Moderate evidence for gene's role in disease		24450482, 23022101
Limited evidence for gene's role in disease	Y	15843405, 18648327
Strong evidence for highly penetrant childhood-onset disease		1303211, 8281149, 8
Strong evidence for highly penetrant childhood-onset disease	Y	7550242, 1301941, 2
Moderate evidence for gene's role in disease		20493458, 23826986
Limited evidence for gene's role in disease		21378380, 26489029
Moderate evidence for gene's role in disease		17468754, 23339108
Limited evidence for gene's role in disease	Y	14556245
Moderate evidence for gene's role in disease	Y	15769976, 20668042,
Strong evidence for highly penetrant childhood-onset disease	Y	14681881, 17431882
Strong evidence for highly penetrant childhood-onset disease		23143598, 23966245
Strong evidence for highly penetrant childhood-onset disease	Y	11179008, 22975760
Strong evidence for highly penetrant childhood-onset disease	Y	11590134,10756353,
Strong evidence for highly penetrant childhood-onset disease		19890111, 25373860
Moderate evidence for gene's role in disease		25373860, 24216686
Strong evidence for highly penetrant childhood-onset disease	Y	15032591, 7668284, 8
Strong evidence for highly penetrant childhood-onset disease		21255762, 24768815
Limited evidence for gene's role in disease		17646580, 21252143
Strong evidence for highly penetrant childhood-onset disease	Y	2569023, 8105179, 8
Strong evidence for highly penetrant childhood-onset disease	Y	12872123, 9792867,
Strong evidence for highly penetrant childhood-onset disease	Y	15723066, 21866095
Moderate penetrance, Not actionable in childhood		19449419, 12219090
Strong evidence for highly penetrant childhood-onset disease		19449419, 11920830
Limited evidence for gene's role in disease		8104271, 10206679, 1
Moderate evidence for gene's role in disease		18304497, 19567699
Limited evidence for gene's role in disease		10969846
Strong evidence for highly penetrant childhood-onset disease		22522421, 22522420
Moderate evidence for gene's role in disease		22512483, 23114595
Moderate evidence for gene's role in disease	Y	18348258, 9185503,
Limited evidence for gene's role in disease		9590299, 23800289
Strong evidence for highly penetrant childhood-onset disease	Y	18348258, 9185503,
Strong evidence for highly penetrant childhood-onset disease	Y	2063866, 15486829,
Limited evidence for gene's role in disease		18434651, 18765512

Strong evidence for highly penetrant childhood-onset disease		16575836, 11180599
Strong evidence for highly penetrant childhood-onset disease	Y	9354668, 11668610,
Limited evidence for gene's role in disease		17509612, 17476457
Moderate penetrance, Actionable in childhood		17924338, 25087486
Strong evidence for highly penetrant childhood-onset disease		2945574, 11874502,
Strong evidence for highly penetrant childhood-onset disease		22544363, 22544367
Moderate evidence for gene's role in disease		20920668, 25476837
Limited evidence for gene's role in disease		23768514
Strong evidence for highly penetrant childhood-onset disease		22265014, 22265017
Strong evidence for highly penetrant childhood-onset disease		12805120, 21109227
Strong evidence for highly penetrant childhood-onset disease		19307729, 9600245 ,
Moderate penetrance, actionable in childhood		16772329, 19343045
Limited evidence for gene's role in disease		21349352, 22840528
Moderate evidence, actionable in childhood		14760488, 19716085
Strong evidence for highly penetrant childhood-onset disease		14228001, 9020846,
Limited evidence for gene's role in disease		18313602
Moderate penetrance, actionable in childhood		14760488, 16922724
Limited evidence for gene's role in disease		12676817, 11901046
Moderate penetrance, actionable in childhood		14760488, 9753711,
Strong evidence for highly penetrant childhood-onset disease	Y	9002665, 10611379,
Strong evidence for highly penetrant childhood-onset disease		16357843, 15448107
Moderate evidence for gene's role in disease		20074522, 21665951
Strong evidence for highly penetrant childhood-onset disease		16217063, 16419128
Limited evidence for gene's role in disease		20560207, 24574546
Limited evidence for gene's role in disease		21215473, 23465283
Moderate penetrance, actionable in childhood	Y	9753711, 15051636,
Strong evidence for highly penetrant childhood-onset disease		14228001, 9020846,
Moderate evidence for gene's role in disease		15372379, 21920939
Moderate penetrance, Not actionable in childhood		9425895, 14534157,
Moderate penetrance, Not actionable in childhood		9425900, 14534157,
Strong evidence for highly penetrant childhood-onset disease		10369879, 16596322
Strong evidence for highly penetrant childhood-onset disease	Y	22693283, 22638565
Limited evidence for gene's role in disease		23665959
Strong evidence for highly penetrant childhood-onset disease		23913813, 23076834
Moderate evidence for gene's role in disease		15883926, 23427148
Limited evidence for gene's role in disease		11389829, 25802885
Strong evidence for highly penetrant childhood-onset disease		18332320, 15827546
Moderate evidence for gene's role in disease		22152677, 22152678
Strong evidence for highly penetrant childhood-onset disease		7529964, 15737214,
Moderate evidence for gene's role in disease		21055716, 24443441

Strong evidence for highly penetrant childhood-onset disease		23746549
Strong evidence for highly penetrant childhood-onset disease		24268659
Strong evidence for highly penetrant childhood-onset disease		23913813, 20711175
Moderate evidence for gene's role in disease		24239382, 25847626
Strong evidence for highly penetrant childhood-onset disease		1705663, 19396835,
Strong evidence for highly penetrant childhood-onset disease		3372762, 17039244,
Strong evidence for highly penetrant childhood-onset disease		7539673, 11886499,
Strong evidence for highly penetrant childhood-onset disease		9008238, 9767294, 7
Moderate evidence for gene's role in disease	Y	12724528, 9011570,
Strong evidence for highly penetrant childhood-onset disease		3372762, 21375516,
Strong evidence for highly penetrant childhood-onset disease		6829608, 7545493, 1
Moderate evidence for gene's role in disease		9618173, 24354895,
Moderate evidence for gene's role in disease	Y	16143128, 22419260
Strong evidence for highly penetrant childhood-onset disease	Y	1303258, 7562969, 1
Strong evidence for highly penetrant childhood-onset disease	Y	16216942, 7550355,
Strong evidence for highly penetrant childhood-onset disease	Y	11810295, 16473856
Limited evidence for gene's role in disease		17646580, 26406308
Strong evidence for highly penetrant childhood-onset disease	Y	16097004, 18672223,
Strong evidence for highly penetrant childhood-onset disease	Y	8824879, 7698759, 9
Strong evidence for highly penetrant childhood-onset disease	Y	8012393, 10660342,
Strong evidence for highly penetrant childhood-onset disease		8504498, 17899313,
Strong evidence for highly penetrant childhood-onset disease	Y	17878207, 17436019
Moderate evidence for gene's role in disease		22607940, 25476837
Limited evidence for gene's role in disease		23541342, 26657938
Strong evidence for highly penetrant childhood-onset disease	Y	12118250, 23824842
Limited evidence for gene's role in disease		20522425
Adult-onset, not actionable in childhood		15668942, 21676617
Strong evidence for highly penetrant childhood-onset disease		3924410, 11810272,
Strong evidence for highly penetrant childhood-onset disease		17229951, 9537324,
Moderate penetrance, Not actionable in childhood		15079011, 17562837
Moderate evidence for gene's role in disease		1727547, 15602022,
Strong evidence for highly penetrant childhood-onset disease		16459341, 16752389
Strong evidence for highly penetrant childhood-onset disease	Y	22238406, 17327381
Strong evidence for highly penetrant childhood-onset disease		14740318, 20447141
Strong evidence for highly penetrant childhood-onset disease		16357942, 26172957
Strong evidence for highly penetrant childhood-onset disease		8146180, 10562460,
Strong evidence for highly penetrant childhood-onset disease		8264707, 19705173,
Strong evidence for highly penetrant childhood-onset disease		19136951, 21303734
Strong evidence for highly penetrant childhood-onset disease	Y	10739764, 10939567

Moderate penetrance, Actionable in childhood		10580077, 12920062
Strong evidence for highly penetrant childhood-onset disease		15965218, 11799477
Limited evidence for gene's role in disease		16826530, 22768673
Strong evidence for highly penetrant childhood-onset disease		25250574
Strong evidence for highly penetrant childhood-onset disease		9323941, 9618165, 1
Strong evidence for highly penetrant childhood-onset disease		19732867, 21465660
Moderate evidence for gene's role in disease		15994876, 17330256
Limited evidence for gene's role in disease		20949626
Strong evidence for highly penetrant childhood-onset disease	Y	17632512, 18553518
Strong evidence for highly penetrant childhood-onset disease		20381006, 24924585
Strong evidence for highly penetrant childhood-onset disease	Y	11719191, 16252235
Strong evidence for highly penetrant childhood-onset disease		12579474, 12054167
Strong evidence for highly penetrant childhood-onset disease	Y	21266382, 12529507
Strong evidence for highly penetrant childhood-onset disease		23891469, 23122589
Adult-onset, Not actionable in childhood		17914064, 22170881,
Strong evidence for highly penetrant childhood-onset disease		24894446, 22781092
Strong evidence for highly penetrant childhood-onset disease		18953341, 22903915
Strong evidence for highly penetrant childhood-onset disease		22829427, 19836010
Limited evidence for gene's role in disease		21220648
Strong evidence for highly penetrant childhood-onset disease	Y	8751864, 13465231,
Adult-onset, Not actionable in childhood		8464497, 17269695,
Strong evidence for highly penetrant childhood-onset disease		22387013, 23956186
Strong evidence for highly penetrant childhood-onset disease		18565486
Strong evidence for highly penetrant childhood-onset disease	Y	22161967, 9915946,
Strong evidence for highly penetrant childhood-onset disease		16439621, 17366577
Strong evidence for highly penetrant childhood-onset disease		17366577, 19156172
Limited evidence for gene's role in disease	Y	16249883, 23329067
Adult-onset, Not actionable in childhood		11708988, 19884572,
Strong evidence for highly penetrant childhood-onset disease		17186462, 23967202
Moderate penetrance, Not actionable in childhood		7573050, 7560086, 1
Limited evidence for gene's role in disease		25558065
Strong evidence for highly penetrant childhood-onset disease		19361614, 23316014
Moderate penetrance, Actionable in childhood		11406611, 9187484,
Moderate penetrance, Actionable in childhood	Y	11181649, 11406611
Moderate evidence for gene's role in disease		16752391, 16697227
Strong evidence for highly penetrant childhood-onset disease		12717434, 18391077
Strong evidence for highly penetrant childhood-onset disease	Y	10973263, 11317355
Strong evidence for highly penetrant childhood-onset disease		16211557, 20978018
Strong evidence for highly penetrant childhood-onset disease	Y	10508514, 12655490
Strong evidence for highly penetrant childhood-onset disease	Y	17369503, 17036352

Limited evidence for gene's role in disease		14638541
Limited evidence for gene's role in disease		23665959
Limited evidence for gene's role in disease		19290556, 26257172
Strong evidence for highly penetrant childhood-onset disease	Y	7726228, 19863562,
Strong evidence for highly penetrant childhood-onset disease		22101682, 22371254
Strong evidence for highly penetrant childhood-onset disease		9215690, 9215689, 1
Moderate evidence for gene's role in disease		15122512, 18485326
Strong evidence for highly penetrant childhood-onset disease		18458227, 9409358,
Strong evidence for highly penetrant childhood-onset disease	Y	17564970, 21990111
Moderate evidence for gene's role in disease	Y	8808595, 11228641,
Strong evidence for highly penetrant childhood-onset disease		9916809, 15810001,
Limited evidence for gene's role in disease		23314057
Limited evidence for gene's role in disease		19363479, 22038834
Strong evidence for highly penetrant childhood-onset disease		8589691 , 9279758,
Strong evidence for highly penetrant childhood-onset disease		10973251, 11179009
Strong evidence for highly penetrant childhood-onset disease	Y	6486167, 17377820,
Strong evidence for highly penetrant childhood-onset disease	Y	11254442, 11935341
Adult-onset, Not actionable in childhood		17539898, 15872200,
Limited evidence for gene's role in disease		12897212, 21883982
Strong evidence for highly penetrant childhood-onset disease		10417274, 12955715
Strong evidence for highly penetrant childhood-onset disease		12438653, 15523652
Strong evidence for highly penetrant childhood-onset disease	Y	12471062, 16410054
Strong evidence for highly penetrant childhood-onset disease	Y	16311595, 20631720
Strong evidence for highly penetrant childhood-onset disease		18385497, 19058814
Strong evidence for highly penetrant childhood-onset disease	Y	9731530, 9921896, 1
Strong evidence for highly penetrant childhood-onset disease	Y	12754701, 21031595
Limited evidence for gene's role in disease	Y	10788335, 26805780
Moderate evidence for gene's role in disease		11733564, 11733556
Strong evidence for highly penetrant childhood-onset disease	Y	10980531, 15771971
Strong evidence for highly penetrant childhood-onset disease	Y	11133753, 16470591
Strong evidence for highly penetrant childhood-onset disease	Y	18695062, 16582910
Strong evidence for highly penetrant childhood-onset disease	Y	8264707, 19705173,
Limited evidence for gene's role in disease	Y	15505824
Moderate evidence for gene's role in disease	Y	17873122, 21189481
Adult-onset, Not actionable in childhood		8261515, 17539898, 1
Adult-onset, Not actionable in childhood		9354786, 9307272, 1
Limited evidence for gene's role in disease		21185009, 24949729

Strong evidence for highly penetrant childhood-onset disease		16222674, 10767351
Moderate penetrance, Not actionable in childhood		1928099, 1732158, 1
Moderate penetrance, Not actionable in childhood		1937476, 8457609, 1
Moderate penetrance, Not actionable in childhood		1634041, 9012411, 1
Moderate penetrance, Actionable in childhood	Y	1866027, 10679944,
Strong evidence for highly penetrant childhood-onset disease	Y	10502779, 8544184,
Limited evidence for gene's role in disease		23929671 , 22608499
Strong evidence for highly penetrant childhood-onset disease		12068375, 8968736 ,
Strong evidence for highly penetrant childhood-onset disease		12555939, 10484769
Strong evidence for highly penetrant childhood-onset disease	Y	10679949, 8533758,
Limited evidence for gene's role in disease		21506741
Limited evidence for gene's role in disease		21642240, 23299917
Strong evidence for highly penetrant childhood-onset disease		15496425, 20371544
Strong evidence for highly penetrant childhood-onset disease	Y	1970180, 6132336, 2
Strong evidence for highly penetrant childhood-onset disease (Al	Y	15690400, 12853198,
Strong evidence for highly penetrant childhood-onset disease	Y	10369261, 11313768
Moderate evidence for gene's role in disease		20045868, 23657818
Moderate penetrance, Actionable in childhood		7493025, 7493026, 1
Disputed evidence for gene's role in disease		20215591, 23281406
Strong evidence for highly penetrant childhood-onset disease		15821734, 18470948
Moderate penetrance, Actionable in childhood		16444274, 15998682
Strong evidence for highly penetrant childhood-onset disease		15015131, 16222661
Strong evidence for highly penetrant childhood-onset disease		20418530, 23388406
Strong evidence for highly penetrant childhood-onset disease		16642020, 23401156
Limited evidence for gene's role in disease		15735645, 20656787
Limited evidence for gene's role in disease		15998695, 20215591
Limited evidence for gene's role in disease		11815426, 26656175
Moderate penetrance, Actionable in childhood		12351586, 23598715
Moderate penetrance, Actionable in childhood		23274168, 21127202
Strong evidence for highly penetrant childhood-onset disease		16103042, 12975303
Moderate penetrance, Actionable in childhood		23794396, 18506004
Strong evidence for highly penetrant childhood-onset disease		15699387, 16684601
Limited evidence for gene's role in disease		17336526
Moderate evidence for gene's role in disease		20733148, 21288719
Strong evidence for highly penetrant childhood-onset disease		11776386, 25077172
Moderate penetrance, Actionable in childhood		9535554, 24111713,
Moderate penetrance, Actionable in childhood		16267253, 23283745
Moderate evidence, Actionable in childhood		21055718, 25907466
Limited evidence for gene's role in disease		11733062, 24082139

Strong evidence for highly penetrant childhood-onset disease		17546645, 17851452
Limited evidence for gene's role in disease		25342930, 19027848
Moderate evidence for gene's role in disease		21756023, 23595123
Limited evidence for gene's role in disease		19027848
Strong evidence for highly penetrant childhood-onset disease		23967202, 23990876
Moderate evidence for gene's role in disease	Y	9207796, 12897212,
Strong evidence for highly penetrant childhood-onset disease		12687499, 23767834
Strong evidence for highly penetrant childhood-onset disease	Y	11391666, 16679490
Limited evidence for gene's role in disease		21256114, 26656175
Adult-onset, Not actionable in childhood		14711882, 15111675
Limited evidence for gene's role in disease		17347475, 22987565
Limited evidence for gene's role in disease		22286171, 18006477
Limited evidence for gene's role in disease		22286171, 18006477
Moderate evidence for gene's role in disease		21700266, 25489052
Limited evidence for gene's role in disease		23665959
Strong evidence for highly penetrant childhood-onset disease	Y	8782044, 2243144, 1
Strong evidence for highly penetrant childhood-onset disease		9950362, 10094189,
Strong evidence for highly penetrant childhood-onset disease	Y	12754705, 12594532
Strong evidence for highly penetrant childhood-onset disease	Y	9590181, 9590180, 1
Strong evidence for highly penetrant childhood-onset disease		8634410, 16972229,
Strong evidence for highly penetrant childhood-onset disease		20167518, 10598813
Limited evidence for gene's role in disease		19692703
Strong evidence for highly penetrant childhood-onset disease	Y	10484772, 8807344,
Strong evidence for highly penetrant childhood-onset disease	Y	12899878, 15336686
Moderate evidence for gene's role in disease		20951326, 23632046
Limited evidence for gene's role in disease		17331106
Strong evidence for highly penetrant childhood-onset disease		8264707, 19705173,
Moderate evidence for gene's role in disease		21211617, 22795106
Moderate evidence for gene's role in disease		18199800, 26862157
Strong evidence for highly penetrant childhood-onset disease	Y	11063730, 10767332
Moderate evidence for gene's role in disease	Y	16855267, 21378176,
Limited evidence for gene's role in disease		19881492, 24503780
Limited evidence for gene's role in disease		20970104, 26265630
Strong evidence for highly penetrant childhood-onset disease		2491776, 4633999, 2
Strong evidence for highly penetrant childhood-onset disease		8081368, 12011146,
Limited evidence for gene's role in disease		23226213, 25205790
Strong evidence for highly penetrant childhood-onset disease		22581936, 24651605
Strong evidence for highly penetrant childhood-onset disease		16439204, 20597108

Strong evidence for highly penetrant childhood-onset disease	Y	15781812, 12958597
Limited evidence for gene's role in disease		18005359, 9886310
Limited evidence for gene's role in disease		22933543
Strong evidence for highly penetrant childhood-onset disease		15317751, 17557927
Strong evidence for highly penetrant childhood-onset disease		15146186, 20824775
Strong evidence for highly penetrant childhood-onset disease		18957494, 22825795
Moderate penetrance, Actionable in childhood		17891520, 19073351
Moderate evidence for gene's role in disease		20004766, 22791571
Moderate penetrance, Not actionable in childhood		160880825, 1041729
Moderate penetrance, Not actionable in childhood		160880825, 1041729
Adult-onset, Not actionable in childhood		16462743, 17579354,
Limited evidence for gene's role in disease		17360648
Strong evidence for highly penetrant childhood-onset disease		10080184, 11846737,
Moderate evidence for gene's role in disease		18005359, 9886310
Moderate evidence for gene's role in disease		18593716, 17662764
Strong evidence for highly penetrant childhood-onset disease		21378985, 23389697
Strong evidence for highly penetrant childhood-onset disease		9818928, 9388399, 1
Strong evidence for highly penetrant childhood-onset disease	Y	12555942, 12955717
Strong evidence for highly penetrant childhood-onset disease	Y	11125141, 17470133
Strong evidence for highly penetrant childhood-onset disease	Y	9598719, 9856524, 9
Strong evidence for highly penetrant childhood-onset disease	Y	19177160, 23188109
Strong evidence for highly penetrant childhood-onset disease	Y	15776426, 23559409
Strong evidence for highly penetrant childhood-onset disease	Y	12495287, 11317351
Limited evidence for gene's role in disease		19646991, 18614783
Strong evidence for highly penetrant childhood-onset disease	Y	18762570, 9709929,
Limited evidence for gene's role in disease		21633855, 26888176
Limited evidence for gene's role in disease		22574178, 23400839
Moderate penetrance, Not actionable in childhood		160880825, 1041729
Strong evidence for highly penetrant childhood-onset disease		11896389, 12807965
Strong evidence for highly penetrant childhood-onset disease		12966526, 10710235
Moderate evidence for gene's role in disease		21129721, 25900314
Strong evidence for highly penetrant childhood-onset disease	Y	10982191, 18077166
Limited evidence for gene's role in disease		10443680
Limited evidence for gene's role in disease		23665959
Limited evidence for gene's role in disease		19070573
Moderate evidence for gene's role in disease	Y	16786527
Strong evidence for highly penetrant childhood-onset disease		21364696, 21396581
Strong evidence for highly penetrant childhood-onset disease		7762554, 23504663,

Strong evidence for highly penetrant childhood-onset disease	Y	11146467, 9430698,
Strong evidence for highly penetrant childhood-onset disease	Y	1941964, 17963220,
Strong evidence for highly penetrant childhood-onset disease		9006432, 17306754,
Strong evidence for highly penetrant childhood-onset disease	Y	11668429, 12126933
Moderate penetrance, Not actionable in childhood		18496845, 15342707
Strong evidence for highly penetrant childhood-onset disease		11477602, 21358631
Moderate evidence for gene's role in disease		21358632, 21358631
Moderate evidence for gene's role in disease		21358632, 22333897
Strong evidence for highly penetrant childhood-onset disease		18179886, 20507362,
Strong evidence for highly penetrant childhood-onset disease	Y	12627228, 23685543
Strong evidence for highly penetrant childhood-onset disease	Y	2012137, 9831349, 1
Strong evidence for highly penetrant childhood-onset disease		11972037, 23173898
Strong evidence for highly penetrant childhood-onset disease		12114484, 10192385
Moderate evidence for gene's role in disease		23122587, 24378291
Strong evidence for highly penetrant childhood-onset disease		23122586, 23850727
Limited evidence for gene's role in disease		23656588
Moderate evidence for gene's role in disease		23345450, 24211385
Adult-onset, Not actionable in childhood		15725589, 10719989
Strong evidence for highly penetrant childhood-onset disease	Y	9399896 , 1671852, 7
Strong evidence for highly penetrant childhood-onset disease		10946356, 9731525,
Adult-onset, Not actionable in childhood		17200668, 21618343
Strong evidence for highly penetrant childhood-onset disease		11479594, 22221393
Strong evidence for highly penetrant childhood-onset disease		8799378, 11683776,
Strong evidence for highly penetrant childhood-onset disease	Y	16712695, 10234503
Strong evidence for highly penetrant childhood-onset disease		15547625, 11344199
Strong evidence for highly penetrant childhood-onset disease	Y	9585612, 9585002, 1
Strong evidence for highly penetrant childhood-onset disease		19238581, 17051315
Strong evidence for highly penetrant childhood-onset disease		2249848, 22033733,
Strong evidence for highly penetrant childhood-onset disease	Y	18719945, 21436283
Strong evidence for highly penetrant childhood-onset disease		18174396, 21270239
Moderate penetrance, Actionable in childhood		14727179, 16211558,
Moderate evidence for gene's role in disease, low pentrance		16767104, 20351491,
Strong evidence for highly penetrant childhood-onset disease		22464252, 23033274
Strong evidence for highly penetrant childhood-onset disease	Y	12163191, 9187674,
Strong evidence for highly penetrant childhood-onset disease	Y	16904023, 11935326
Limited evidence for gene's role in disease		17254821
Limited evidence for gene's role in disease	Y	19184109, 15855260
Limited evidence for gene's role in disease	Y	17332895

Limited evidence for gene's role in disease	Y	17186472
Strong evidence for highly penetrant childhood-onset disease		16141001, 9539740,
Strong evidence for highly penetrant childhood-onset disease		21031596, 19127411
Limited evidence for gene's role in disease		22581968, 26233629
Strong evidence for highly penetrant childhood-onset disease		21031596, 14571262
Strong evidence for highly penetrant childhood-onset disease		17041890, 21031596
Moderate evidence for gene's role in disease		21031596, 15146459
Moderate evidence for gene's role in disease		20647552, 20681997
Moderate evidence for gene's role in disease		21031596, 20683989
Strong evidence for highly penetrant childhood-onset disease		14630978, 10652207
Strong evidence for highly penetrant childhood-onset disease		15542397, 21031596
Strong evidence for highly penetrant childhood-onset disease		10958759, 21031596
Strong evidence for highly penetrant childhood-onset disease		18712838, 21031596
Strong evidence for highly penetrant childhood-onset disease		19877282, 10408779
Strong evidence for highly penetrant childhood-onset disease	Y	11781871
Strong evidence for highly penetrant childhood-onset disease		9090381, 9090382, 1
Strong evidence for highly penetrant childhood-onset disease		7513946, 8659544, 8
Strong evidence for highly penetrant childhood-onset disease		12415272, 15994862
Moderate evidence for gene's role in disease		7874115, 22238410,
Strong evidence for highly penetrant childhood-onset disease		21646031, 17689125
Strong evidence for highly penetrant childhood-onset disease		12825073, 9215682,
Strong evidence for highly penetrant childhood-onset disease		9384616, 8896567, 1
Moderate evidence for gene's role in disease		11600883, 14597037
Moderate penetrance, Actionable in childhood		12640453, 16873766
Strong evidence for highly penetrant childhood-onset disease		9326939, 9326940, 1
Strong evidence for highly penetrant childhood-onset disease		23487782, 24726473
Moderate evidence for gene's role in disease		24706016, 22305531
Strong evidence for highly penetrant childhood-onset disease		18524835, 15596610,
Limited evidence for gene's role in disease		23665959
Limited evidence for gene's role in disease		23611745, 24333117
Strong evidence for highly penetrant childhood-onset disease		9005987, 1605247, 1
Strong evidence for highly penetrant childhood-onset disease		8650545, 22508176,
Strong evidence for highly penetrant childhood-onset disease	Y	16133180, 19021613
Strong evidence for highly penetrant childhood-onset disease		8616073, 16704447,
Moderate penetrance, Actionable in childhood		15489853, 24125834
Strong evidence for highly penetrant childhood-onset disease	Y	10227637, 20584031
Strong evidence for highly penetrant childhood-onset disease	Y	20591883, 25349199
Strong evidence for highly penetrant childhood-onset disease	Y	14675180, 23289980
Strong evidence for highly penetrant childhood-onset disease		21109228, 25987458
Strong evidence for highly penetrant childhood-onset disease	Y	16849641, 10233898

Moderate evidence, Actionable in childhood		12610310, 12639993
Limited evidence for gene's role in disease		21167350, 16829191
Strong evidence for highly penetrant childhood-onset disease	Y	10874315, 21699693
Moderate evidence for gene's role in disease		15523624, 22689593
Strong evidence for highly penetrant childhood-onset disease	Y	1384324, 8012387, 7
Strong evidence for highly penetrant childhood-onset disease		3840606, 7522741, 1
Strong evidence for highly penetrant childhood-onset disease	Y	12409504, 10571956
Strong evidence for highly penetrant childhood-onset disease	Y	8264707, 19705173,
Adult-onset, Not actionable in childhood		17539898, 15872200,
Strong evidence for highly penetrant childhood-onset disease		15262732, 19124534
Strong evidence for highly penetrant childhood-onset disease		20118933, 24965255
Moderate evidence for gene's role in disease		22246504, 24344921
Strong evidence for highly penetrant childhood-onset disease	Y	15772097, 18024216
Limited evidence for gene's role in disease		24048372
Strong evidence for highly penetrant childhood-onset disease	Y	14694057, 12565911
Strong evidence for highly penetrant childhood-onset disease		20346687, 11338401
Moderate evidence for gene's role in disease		23649472, 21860632
Strong evidence for highly penetrant childhood-onset disease	Y	17878207, 11709191
Strong evidence for highly penetrant childhood-onset disease	Y	18195152, 22419172
Strong evidence for highly penetrant childhood-onset disease	Y	15792865, 16717220
Strong evidence for highly penetrant childhood-onset disease	Y	16575835, 15637732
Strong evidence for highly penetrant childhood-onset disease	Y	17923109, 17878207
Strong evidence for highly penetrant childhood-onset disease		16467261, 19258400
Strong evidence for highly penetrant childhood-onset disease		17546030, 17546031
Strong evidence for highly penetrant childhood-onset disease	Y	1509263, 15928241,
Strong evidence for highly penetrant childhood-onset disease		7839145, 8872461, 2
Strong evidence for highly penetrant childhood-onset disease		9506947, 18228599,
Low penetrance		10486317, 8673113,
Strong evidence for highly penetrant childhood-onset disease	Y	9664077, 11589012,
Strong evidence for highly penetrant childhood-onset disease	Y	14634649, 20950397,
Limited evidence for gene's role in disease		23768516, 24387995
Moderate evidence for gene's role in disease	Y	16385448, 22796000
Strong evidence for highly penetrant childhood-onset disease		17873118, 14757862
Limited evidence for gene's role in disease		18976727
Moderate penetrance, Actionable in childhood	Y	1182799, 15673802,

Strong evidence for highly penetrant childhood-onset disease		11586982, 11407343
Limited evidence for gene's role in disease		6424667
Strong evidence for highly penetrant childhood-onset disease	Y	18241045, 11115848
Moderate penetrance, Not actionable in childhood		12577059, 20095989
Strong evidence for highly penetrant childhood-onset disease	Y	10669160, 7482420,
Moderate evidence for gene's role in disease		11510941, 17412540
Strong evidence for highly penetrant childhood-onset disease		17054399, 22773735,
Strong evidence for highly penetrant childhood-onset disease	Y	11549703, 9920061,
Strong evidence for highly penetrant childhood-onset disease		8943854, 9241758, 7
Moderate evidence for gene's role in disease	Y	22546954, 17701896
Limited evidence for gene's role in disease		17701900, 24285972
Moderate evidence for gene's role in disease		23444262, 22674740,
Strong evidence for highly penetrant childhood-onset disease	Y	11157804, 24011642
Strong evidence for highly penetrant childhood-onset disease	Y	10196694, 17616409
Moderate evidence for gene's role in disease	Y	17436247, 25152457
Adult-onset, Not actionable in childhood		7596406, 1411576, 7
Adult-onset, Not actionable in childhood		7638621, 7651536 , 1
Strong evidence for highly penetrant childhood-onset disease		8352281, 16301862,
Strong evidence for highly penetrant childhood-onset disease		17526800, 9140396,
Strong evidence for highly penetrant childhood-onset disease		17526800, 12844284
Strong evidence for highly penetrant childhood-onset disease	Y	8703170, 15240651,
Strong evidence for highly penetrant childhood-onset disease		12161596, 16523510
Strong evidence for highly penetrant childhood-onset disease		19726876, 20684003
Strong evidence for highly penetrant childhood-onset disease		8178819, 9222757, 1
Limited evidence for gene's role in disease		17089422, 16674562,
Strong evidence for highly penetrant childhood-onset disease		17705025, 9529348,
Strong evidence for highly penetrant childhood-onset disease		2116088, 9744478, 1
Limited evidence for gene's role in disease		23665959
Strong evidence for highly penetrant childhood-onset disease	Y	20358613, 17503333,
Strong evidence for highly penetrant childhood-onset disease	Y	10835631, 19953648
Strong evidence for highly penetrant childhood-onset disease	Y	15696165, 23420520
Moderate evidence for gene's role in disease	Y	23420520, 20967465
Strong evidence for highly penetrant childhood-onset disease		11389829, 15455439
Limited evidence for gene's role in disease		26261251, 24139550,
Strong evidence for highly penetrant childhood-onset disease		17603482, 17603483
Strong evidence for highly penetrant childhood-onset disease	Y	11313270, 11133745
Strong evidence for highly penetrant childhood-onset disease	Y	25869295, 11313270
Strong evidence for highly penetrant childhood-onset disease		12652298, 15565467
Strong evidence for highly penetrant childhood-onset disease		20188345, 19015223
Limited evidence for gene's role in disease		21447824, 26173111

Strong evidence for highly penetrant childhood-onset disease	Y	14504330, 12730725
Strong evidence for highly penetrant childhood-onset disease		14639529, 18446851
Strong evidence for highly penetrant childhood-onset disease		2594929, 2521957, 8
Moderate penetrance, Actionable in childhood		19712804, 20590677
Strong evidence for highly penetrant childhood-onset disease		22366785, 17236129
Moderate evidence for gene's role in disease		17226784, 19215054
Strong evidence for highly penetrant childhood-onset disease		18716613, 15964893
Strong evidence for highly penetrant childhood-onset disease		1481838, 12952869,
Strong evidence for highly penetrant childhood-onset disease		12835862, 10319867
Moderate evidence for gene's role in disease	Y	17431900, 10973257
Strong evidence for highly penetrant childhood-onset disease	Y	2359105, 22095942,
Strong evidence for highly penetrant childhood-onset disease		19469690, 8632274,
Strong evidence for highly penetrant childhood-onset disease		19469690, 8632274,
Limited evidence for gene's role in disease		20148032, 24411943
Moderate evidence for gene's role in disease		8563755, 9746795, 1
Strong evidence for highly penetrant childhood-onset disease	Y	16838329, 12107819
Strong evidence for highly penetrant childhood-onset disease		17846997, 23592335
Strong evidence for highly penetrant childhood-onset disease		16845400, 17846997
Strong evidence for highly penetrant childhood-onset disease		16845400, 17846997
Strong evidence for highly penetrant childhood-onset disease		10932186, 17256787
Strong evidence for highly penetrant childhood-onset disease		10700182, 19461659
Strong evidence for highly penetrant childhood-onset disease		8673101, 8817343, 1
Strong evidence for highly penetrant childhood-onset disease	Y	17558409, 17960139
Strong evidence for highly penetrant childhood-onset disease		17558407, 23351400
Strong evidence for highly penetrant childhood-onset disease		10541318, 19061985
Moderate evidence for gene's role in disease		16990592, 10590074
Strong evidence for highly penetrant childhood-onset disease		19061985, 20960466
Moderate evidence for gene's role in disease		20116044, 25946618
Strong evidence for highly penetrant childhood-onset disease		20960466, 16990592
Strong evidence for highly penetrant childhood-onset disease		20960466, 16990592
Strong evidence for highly penetrant childhood-onset disease		9988267, 16990592,
Strong evidence for highly penetrant childhood-onset disease		17186470, 19689926
Strong evidence for highly penetrant childhood-onset disease		20960466, 20116044
Strong evidence for highly penetrant childhood-onset disease		16879200, 17100996
Moderate evidence for gene's role in disease		20960466, 23718193
Strong evidence for highly penetrant childhood-onset disease		19667227, 17486094
Strong evidence for highly penetrant childhood-onset disease	Y	9618178, 9326935, 1
Strong evidence for highly penetrant childhood-onset disease		19200523, 23798057
Strong evidence for highly penetrant childhood-onset disease		19200523, 23993197
Strong evidence for highly penetrant childhood-onset disease		16575894, 7747775,

Strong evidence for highly penetrant childhood-onset disease		20583297, 16084090
Strong evidence for highly penetrant childhood-onset disease		21062345, 20839240
Moderate penetrance, Actionable in childhood		12124989, 14732627
Moderate evidence for gene's role in disease		16084090
Strong evidence for highly penetrant childhood-onset disease		12112081, 16380615
Strong evidence for highly penetrant childhood-onset disease		11159936, 25041964
Strong evidence for highly penetrant childhood-onset disease		11157710, 17875969
Strong evidence for highly penetrant childhood-onset disease	Y	20876471, 18398442,
Strong evidence for highly penetrant childhood-onset disease		14755477, 16892410
Strong evidence for highly penetrant childhood-onset disease		19525956, 24300241
Strong evidence for highly penetrant childhood-onset disease	Y	12496757, 21536732
Moderate evidence for gene's role in disease		12189593, 12812989
Strong evidence for highly penetrant childhood-onset disease		24207120, 24036948
Strong evidence for highly penetrant childhood-onset disease		18930999, 24168886
Limited evidence for gene's role in disease		18464934, 12676817
Limited evidence for gene's role in disease		9070470, 19808477,
Limited evidence for gene's role in disease		12676817, 11901046
Strong evidence for highly penetrant childhood-onset disease		1651050, 15596759
Moderate penetrance, Not actionable in childhood		15557532, 10599760
Limited evidence for gene's role in disease		17592081, 20226894
Moderate penetrance, Actionable in childhood		9753711, 15051636,
Moderate penetrance, Actionable in childhood		12417552, 12676817
Strong evidence for highly penetrant childhood-onset disease	Y	16207733, 8589714,
Strong evidence for highly penetrant childhood-onset disease	Y	12107247, 16207733
Moderate penetrance, Not actionable in childhood		8524790, 21956615,
Moderate evidence for gene's role in disease	Y	8640238, 7550319, 1
Moderate evidence for gene's role in disease		19295170, 11013136
Strong evidence for highly penetrant childhood-onset disease	Y	12538779, 12020273
Limited evidence for gene's role in disease		16685654
Moderate evidence for gene's role in disease, Actionable in childhood		19628817, 20071235,
Moderate penetrance, Actionable in childhood		18057081, 15328326,
Moderate penetrance, Actionable in childhood		11062460, 19351833,
Strong evidence for highly penetrant childhood-onset disease		10657297, 17557926
Moderate penetrance, Not actionable in childhood		15133510, 20095989
Moderate evidence for gene's role in disease		22927827, 22416012
Strong evidence for highly penetrant childhood-onset disease	Y	10665485, 9585610,
Strong evidence for highly penetrant childhood-onset disease		16365872
Strong evidence for highly penetrant childhood-onset disease		11523561, 19139049
Low penetrance	Y	18515255, 2227940,

Limited evidence for gene's role in disease		20451170, 25719458
Moderate penetrance, Not actionable in childhood		9031473, 21264449,
Limited evidence for gene's role in disease		8902986, 8562924, 1
Strong evidence for highly penetrant childhood-onset disease		20436468, 25217958
Strong evidence for highly penetrant childhood-onset disease		14770181, 17159128
Limited evidence for gene's role in disease		19100526, 20466729
Strong evidence for highly penetrant childhood-onset disease	Y	10571948, 10712351
Moderate penetrance, Not actionable in childhood	Y	18383112, 19443464
Strong evidence for highly penetrant childhood-onset disease	Y	9032047, 7663524, 8
Strong evidence for highly penetrant childhood-onset disease		7581449, 18285821,
Limited evidence for gene's role in disease		10974018, 24503780
Strong evidence for highly penetrant childhood-onset disease		10069710, 8841194,
Strong evidence for highly penetrant childhood-onset disease		7668303, 11053682,
Strong evidence for highly penetrant childhood-onset disease	Y	11182930, 9401012,
Strong evidence for highly penetrant childhood-onset disease	Y	20926771, 10556288,
Moderate penetrance, Not actionable in childhood		11381256, 10364528
Strong evidence for highly penetrant childhood-onset disease		21291453, 19272779
Strong evidence for highly penetrant childhood-onset disease		17173049, 21779178
Strong evidence for highly penetrant childhood-onset disease		21940735, 8896572,
Moderate evidence for gene's role in disease		19684605, 22528146
Strong evidence for highly penetrant childhood-onset disease	Y	14012309, 17309654
Strong evidence for highly penetrant childhood-onset disease		15141091, 18330911
Moderate evidence for gene's role in disease		18305125, 24429398
Strong evidence for highly penetrant childhood-onset disease		21940735, 18791198
Moderate evidence for gene's role in disease		17357085
Strong evidence for highly penetrant childhood-onset disease		23103230, 15884042
Moderate evidence for gene's role in disease		15459009, 16439678
Strong evidence for highly penetrant childhood-onset disease	Y	9585600, 18391953,
Strong evidence for highly penetrant childhood-onset disease		8528245, 12112667,
Moderate evidence for gene's role in disease		24668262, 24928908
Strong evidence for highly penetrant childhood-onset disease	Y	8292134, 12368912,
Moderate evidence for gene's role in disease		25390740
Limited evidence for gene's role in disease		18304496, 21778275
Strong evidence for highly penetrant childhood-onset disease	Y	23568789, 18398436
Strong evidence for highly penetrant childhood-onset disease	Y	10581036, 10947946
Strong evidence for highly penetrant childhood-onset disease		10391221, 10874303
Strong evidence for highly penetrant childhood-onset disease		20065143, 15871139
Strong evidence for highly penetrant childhood-onset disease		9826541, 10072434,
Limited evidence for gene's role in disease		19641205, 24515575

Strong evidence for highly penetrant childhood-onset disease		8105687, 16059747,
Strong evidence for highly penetrant childhood-onset disease	Y	18978333, 1432421,
Strong evidence for highly penetrant childhood-onset disease		8054358, 15363639,
Moderate evidence for gene's role in disease	Y	15592994, 19780765
Strong evidence for highly penetrant childhood-onset disease		19412178, 21393332
Strong evidence for highly penetrant childhood-onset disease		10926541, 10364542
Strong evidence for highly penetrant childhood-onset disease	Y	7977372, 8528239, 2
Strong evidence for highly penetrant childhood-onset disease		8896562, 11524734,
Strong evidence for highly penetrant childhood-onset disease	Y	11317356, 23918157
Strong evidence for highly penetrant childhood-onset disease		14715877, 19631310
Limited evidence for gene's role in disease		22089923
Strong evidence for highly penetrant childhood-onset disease		9462754, 10980529,
Strong evidence for highly penetrant childhood-onset disease		16550171, 17935213
Moderate evidence for gene's role in disease		22243965
Limited evidence for gene's role in disease		19061983, 25402622
Strong evidence for highly penetrant childhood-onset disease	Y	16960801, 17095743
Strong evidence for highly penetrant childhood-onset disease		16358214, 16358215
Limited evidence for gene's role in disease	Y	23873973, 15576474
Moderate evidence for gene's role in disease		24115232, 23561849
Moderate evidence for gene's role in disease	Y	11326280, 23806237
Strong evidence for highly penetrant childhood-onset disease	Y	19508970, 17952091
Strong evidence for highly penetrant childhood-onset disease	Y	10482962, 9758626,
Strong evidence for highly penetrant childhood-onset disease		12068297, 11254458
Strong evidence for highly penetrant childhood-onset disease	Y	15635077, 8792820,
Limited evidence for gene's role in disease		21812739, 20683486,
Strong evidence for highly penetrant childhood-onset disease		11574907, 23504663
Strong evidence for highly penetrant childhood-onset disease		11804211, 17446347
Strong evidence for highly penetrant childhood-onset disease		8640229, 7530501, 1
Moderate evidence for gene's role in disease		21204806, 18413482
Strong evidence for highly penetrant childhood-onset disease	Y	17220209, 16767101
Moderate evidence for gene's role in disease		10545938, 15930088
Strong evidence for highly penetrant childhood-onset disease		12436245, 18622023
Strong evidence for highly penetrant childhood-onset disease		15863666, 9171822,
Low penetrance		15286788, 18484095
Limited evidence for gene's role in disease		10684912, 23580201
Strong evidence for highly penetrant childhood-onset disease		16751771, 22700964
Strong evidence for highly penetrant childhood-onset disease	Y	21910234, 22281021

Strong evidence for highly penetrant childhood-onset disease		10080183, 18716612
Strong evidence for highly penetrant childhood-onset disease		12820697, 11157794
Limited evidence for gene's role in disease		18784102, 25296721
Strong evidence for highly penetrant childhood-onset disease	Y	21932316, 18342287
Moderate evidence for gene's role in disease		22232210, 25546334
Moderate evidence for gene's role in disease		22232210, 25546334
Strong evidence for highly penetrant childhood-onset disease		22197487, 22553128
Limited evidence for gene's role in disease		21898662
Strong evidence for highly penetrant childhood-onset disease		22167769, 21217753
Strong evidence for highly penetrant childhood-onset disease		9582123, 10764709,
Limited evidence for gene's role in disease		22275001
Limited evidence for gene's role in disease		19211612, 21898662
Strong evidence for highly penetrant childhood-onset disease		11799392, 17089404
Strong evidence for highly penetrant childhood-onset disease		16604071, 19701948
Strong evidence for highly penetrant childhood-onset disease	Y	12815596, 8787675,
Limited evidence for gene's role in disease		24651015
Strong evidence for highly penetrant childhood-onset disease	Y	19405096, 12369017
Strong evidence for highly penetrant childhood-onset disease		2023926, 19405096,
Strong evidence for highly penetrant childhood-onset disease		21549336, 21549342
Moderate evidence for gene's role in disease	Y	15968592, 23231787
Moderate evidence, Actionable in childhood		19684871, 18591664
Adult-onset, Not actionable in childhood		8446170, 9365366, 7
Strong evidence for highly penetrant childhood-onset disease	Y	20127975, 21898658
Moderate evidence for gene's role in disease		12740761, 24167460
Strong evidence for highly penetrant childhood-onset disease		7485151, 8411055, 1
Strong evidence for highly penetrant childhood-onset disease	Y	16648851, 22621957
Limited evidence for gene's role in disease		20579626
Moderate evidence for gene's role in disease		25087613
Strong evidence for highly penetrant childhood-onset disease		7822652, 10835624
Strong evidence for highly penetrant childhood-onset disease		11443547, 22291068
Strong evidence for highly penetrant childhood-onset disease		22753041, 21649642
Strong evidence for highly penetrant childhood-onset disease		1679439, 1878597, 2
Strong evidence for highly penetrant childhood-onset disease		2961992, 1486040, 8
Strong evidence for highly penetrant childhood-onset disease		19651702, 8673084
Moderate evidence for gene's role in disease		20920666, 26392352
Strong evidence for highly penetrant childhood-onset disease		22265015, 23621943
Moderate evidence for gene's role in disease		17273967, 18445049
Moderate evidence for gene's role in disease	Y	15502825, 22990144

Moderate evidence for gene's role in disease		23736855
Strong evidence for highly penetrant childhood-onset disease	Y	8948562, 16968793, 1
Strong evidence for highly penetrant childhood-onset disease		17881745, 17676033
Strong evidence for highly penetrant childhood-onset disease		15121768, 10874301
Strong evidence for highly penetrant childhood-onset disease	Y	17273977, 19309693
Strong evidence for highly penetrant childhood-onset disease		22147502, 23208854
Strong evidence for highly penetrant childhood-onset disease		7546451, 10922387,
Strong evidence for highly penetrant childhood-onset disease		16582076, 20486178
Strong evidence for highly penetrant childhood-onset disease	Y	20887364, 24623842
Strong evidence for highly penetrant childhood-onset disease		19804848, 20798128
Strong evidence for highly penetrant childhood-onset disease		17287286, 15877282
Strong evidence for highly penetrant childhood-onset disease	Y	20693550, 17668387
Strong evidence for highly penetrant childhood-onset disease	Y	12112661, 9428520
Strong evidence for highly penetrant childhood-onset disease		9837813, 9843204, 1
Limited evidence for gene's role in disease		23348741
Limited evidence for gene's role in disease		21835308
Limited evidence for gene's role in disease		20493459, 25516202
Adult-onset, Not actionable in childhood		22539580, 18372902,
Strong evidence for highly penetrant childhood-onset disease		1356171, 1357662, 1
Strong evidence for highly penetrant childhood-onset disease	Y	15098233, 8042670,
Strong evidence for highly penetrant childhood-onset disease		20727515, 24291220
Moderate evidence for gene's role in disease	Y	12389028, 16938882
Strong evidence for highly penetrant childhood-onset disease		11748311, 14585638
Moderate evidence for gene's role in disease		17668378, 18834961
Strong evidence for highly penetrant childhood-onset disease		5904863, 12818525,
Moderate evidence for gene's role in disease		12507402, 9245996,
Strong evidence for highly penetrant childhood-onset disease		10655062, 25724973
Strong evidence for highly penetrant childhood-onset disease	Y	22231430, 11532986
Strong evidence for highly penetrant childhood-onset disease		9042910, 22317976,
Moderate evidence for gene's role in disease		21725307, 22693042
Moderate evidence for gene's role in disease		22883145, 25118024
Strong evidence for highly penetrant childhood-onset disease		10987647, 21520338
Moderate penetrance, Actionable in childhood		18005359, 9886310,
Moderate penetrance, Actionable in childhood		18042801, 17785587
Strong evidence for highly penetrant childhood-onset disease		19764023, 1456287,
Strong evidence for highly penetrant childhood-onset disease		10368122, 10802654
Strong evidence for highly penetrant childhood-onset disease		22883144, 23553329

Moderate penetrance, Not actionable in childhood	Y	10802645, 21524769
Strong evidence for highly penetrant childhood-onset disease		8626865, 16720658,
Moderate penetrance, Not actionable in childhood		11810278, 7917133,
Limited evidence for gene's role in disease		15639475, 24238504
Strong evidence for highly penetrant childhood-onset disease		16928994, 15731757
Strong evidence for highly penetrant childhood-onset disease		15731757, 17470566
Limited evidence for gene's role in disease		16613887, 22036907
Moderate penetrance, Not actionable in childhood		21940735, 10835638
Strong evidence for highly penetrant childhood-onset disease	Y	19434086, 10712205
Strong evidence for highly penetrant childhood-onset disease		22622422, 24628291
Strong evidence for highly penetrant childhood-onset disease	Y	19491146, 18058633
Moderate evidence for gene's role in disease		16621965, 23314101
Limited evidence for gene's role in disease		22198906
Strong evidence for highly penetrant childhood-onset disease		22168587, 24969835
Strong evidence for highly penetrant childhood-onset disease		7838159, 8040303, 9
Strong evidence for highly penetrant childhood-onset disease	Y	15037720, 8841189,
Moderate penetrance, Actionable in childhood		1866989, 18252230,
Moderate evidence for gene's role in disease		12704386, 24614073
Strong evidence for highly penetrant childhood-onset disease	Y	16908738, 15907288
Strong evidence for highly penetrant childhood-onset disease		11850618, 16134132
Limited evidence for gene's role in disease		12426567, 17368633,
Moderate evidence for gene's role in disease		20036350, 20512146
Moderate evidence for gene's role in disease		6486167, 17377820
Moderate evidence for gene's role in disease		22152675, 22693042
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Strong evidence for highly penetrant childhood-onset disease	Y	17160906, 21068128
Strong evidence for highly penetrant childhood-onset disease		6486167, 17377820,
Strong evidence for highly penetrant childhood-onset disease		12145746, 19438934
Disputed evidence for gene's role in disease		16247757
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Strong evidence for highly penetrant childhood-onset disease	Y	12189164, 14672344
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Moderate evidence, Actionable in childhood		15542288, 20215591
Strong evidence for highly penetrant childhood-onset disease		12592607, 16924011
Moderate evidence, Actionable in childhood		19590045, 15607392
Moderate penetrance, Actionable in childhood		9241277, 20624503,
Strong evidence for highly penetrant childhood-onset disease	Y	10952871, 24689076
Moderate penetrance, Actionable in childhood		20083571, 15542288
Moderate penetrance, Actionable in childhood		8205619, 7898523, 1
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Moderate evidence for gene's role in disease		9288108, 12865992,
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Limited evidence for gene's role in disease		3938792, 6432893
Limited evidence for gene's role in disease		9141550, 26735259
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Limited evidence for gene's role in disease		19004782
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Moderate evidence for gene's role in disease	Y	17033963, 22499341
Strong evidence for highly penetrant childhood-onset disease	Y	9589689, 15292359,
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Limited evidence for gene's role in disease		22678063, 26969326
Limited evidence for gene's role in disease	Y	15273283
Moderate evidence for gene's role in disease		21258341, 22773737
Strong evidence for highly penetrant childhood-onset disease		20176027, 21120949
Strong evidence for highly penetrant childhood-onset disease		23830146, 24417819
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Moderate penetrance, Actionable in childhood		22335739, 24119082
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Strong evidence for highly penetrant childhood-onset disease		1626556, 1301926, 9
Limited evidence for gene's role in disease		19896110
Strong evidence for highly penetrant childhood-onset disease		9259286, 9585583,

Strong evidence for highly penetrant childhood-onset disease		12177387, 9924029,
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Strong evidence for highly penetrant childhood-onset disease	Y	16311597, 24599544
Limited evidence for gene's role in disease		19065272, 23275527
Strong evidence for highly penetrant childhood-onset disease		7989595, 19830808,
Limited evidence for gene's role in disease		8280139
Limited evidence for gene's role in disease		16786511
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Strong evidence for highly penetrant childhood-onset disease		16528436, 16278825
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Strong evidence for highly penetrant childhood-onset disease	Y	18655112, 11477603
Strong evidence for highly penetrant childhood-onset disease	Y	15052268, 16896922
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Moderate evidence for gene's role in disease		20817137, 22486404
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Strong evidence for highly penetrant childhood-onset disease		10521293, 21446023

Limited evidence for gene's role in disease		16534117, 15911806
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Limited evidence for gene's role in disease	Y	14872406
Moderate evidence for gene's role in disease		19918918, 24716670
Moderate evidence for gene's role in disease	Y	16826533, 21271649
Moderate evidence for gene's role in disease		18005359, 9886310
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Strong evidence for highly penetrant childhood-onset disease	Y	9354794, 14681828,
Strong evidence for highly penetrant childhood-onset disease	Y	15843403, 16297189
Limited evidence for gene's role in disease		23295299
Strong evidence for highly penetrant childhood-onset disease	Y	21664999, 23680354,
Limited evidence for gene's role in disease		16385466, 23871722

rtopenia; BIL, hyperbilirubinemia; BOW, bowel dysfunction; CHD, congenital heart disease; CM, cardio
ory disorder; SEIZ, seizure; SK, skeletal dysplasia; THROM, thrombophilia; THYR, hypothyroidism.

BabySeq IBA panel	Reviewed
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