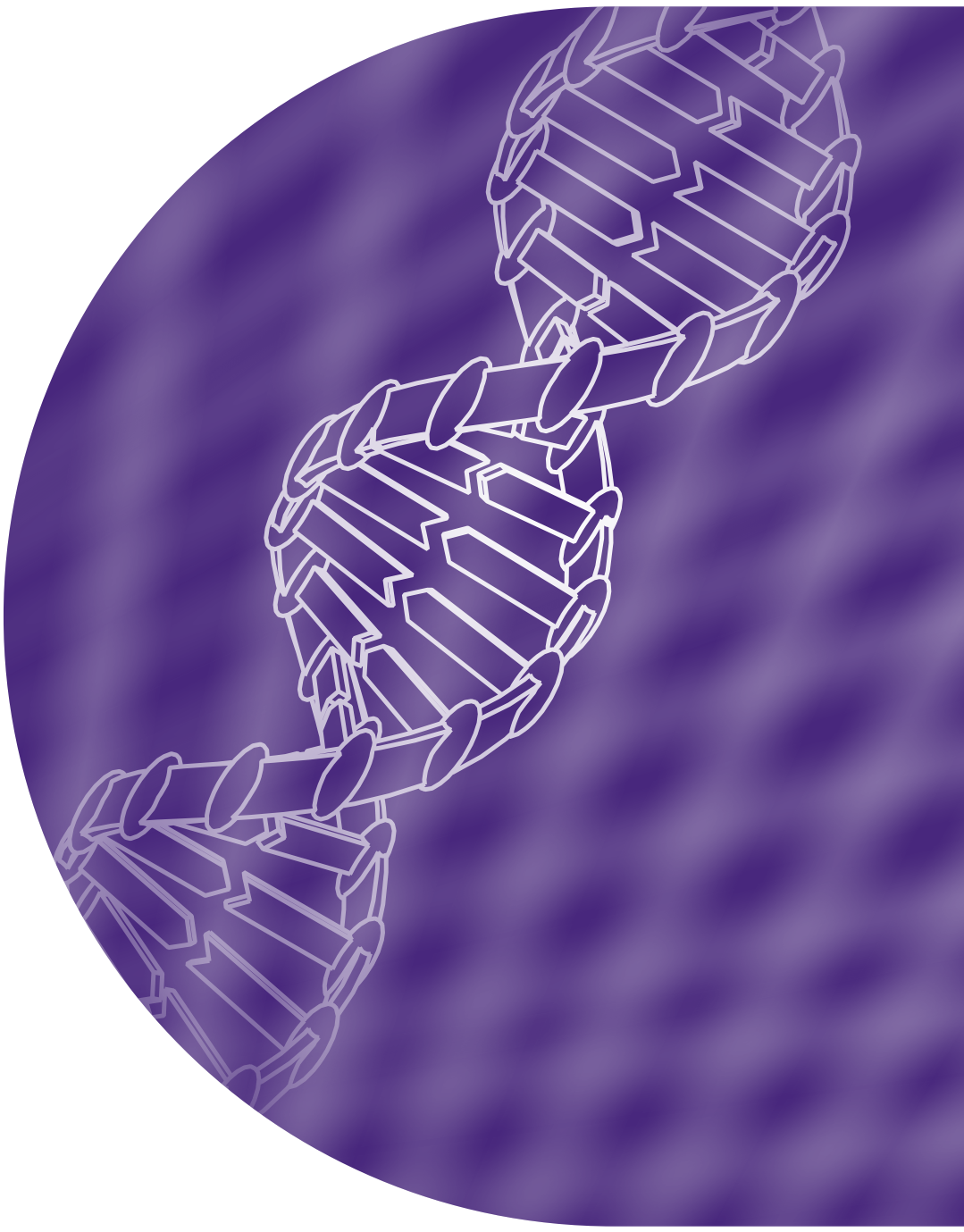
HANIFA
GENETICS



غربالگری ناقلین بیماری‌ها

1. 3 Methylcrotonyl-Coa Carboxylase 1 Deficiency

3-methylcrotonyl-CoA carboxylase deficiency (also known as 3-MCC deficiency) is an inherited disorder in which the body is unable to process certain proteins properly. People with this disorder have a shortage of an enzyme that helps break down proteins containing a particular building block (amino acid) called leucine.Infants with 3-MCC deficiency appear normal at birth but usually develop signs and symptoms in infancy or early childhood. The characteristic features of this condition, which can range from mild to life-threatening, include feeding difficulties, recurrent episodes of vomiting and diarrhea, excessive tiredness (lethargy), and weak muscle tone (hypotonia). If untreated, this disorder can lead to delayed development, seizures, and coma. Many of these complications can be prevented with early detection and lifelong management with a low-protein diet and appropriate supplements.

Gene markers analyzed: 183
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

MCCC1

---

2. 3-Phosphoglycerate Dehydrogenase Deficiency

3-Phosphoglycerate Dehydrogenase Deficiency is an autosomal recessive disorder that affects the brain and nervous system. Signs and symptoms usually begin infancy and include small head size (microcephaly), developmental delays, growth delay, intellectual disability, and seizures. The brain develops abnormally and over time there is loss of brain tissue. Affected infants may not achieve developmental milestones such as speech or sitting up without assistance. In rare cases symptoms do not begin until childhood or adulthood. Currently there is no cure for this condition; however, amino acid therapy may reduce seizures and other symptoms if treatment is started early in life.

Gene markers analyzed: 80
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

PHGDH

---

3. Abetalipoproteinaemia

Abetalipoproteinemia is a rare autosomal recessive disorder that prevents the body from completely absorbing certain dietary fats and the essential vitamins A, D, E, and K. Signs and symptoms of Abetalipoproteinemia usually begin in infancy but may first appear later in childhood, or rarely, not until adulthood. Symptoms include poor weight gain and diarrhea along with abnormally shaped red blood cells (acanthocytosis). Affected children often have problems with balance, coordination, and walking due to problems with nerve function and muscle weakness. Anemia and a type of vision loss called Retinitis Pigmentosa may also occur. Treatment to attempt to slow down the progression of symptoms includes supplementation with the fat-soluble vitamins and other supplements along with special low-fat medical diet.

Gene markers analyzed: 87
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

MTTP

4. Achromatopsia

Achromatopsia, CNGB3-Related is an autosomal recessive condition that causes partial or complete loss of color vision. Most people with this condition have complete Achromatopsia, and can only see in shades of black, white, and gray. Other vision problems seen with Achromatopsia, CNGB3-Related include light sensitivity, reduced sharpness of vision, involuntary shaking movements of the eye (nystagmus), farsightedness or, less commonly, nearsightedness. It is common for light sensitivity and nystagmus to appear within the first few weeks or months of life, although this may improve slightly over time. Achromatopsia, CNGB3-Related is not the same as colorblindness, a condition where color can be seen but it is difficult to distinguish between certain colors. Currently there is no cure for this condition. Although rare, some individuals will have incomplete Achromatopsia with the ability to perceive some color. Also rare is another form of the disorder called Progressive Cone Dystrophy where loss of color doesn't begin until childhood or teenage years.

Gene markers analyzed: 190
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

CNGB3

15. Aspartylglucosaminuria

Aspartylglycosaminuria is an autosomal recessive disorder in which the body is unable to breakdown certain types of proteins, called glycoasparagines, in the cells, leading to a toxic buildup in the body. People with Aspartylglycosaminuria typically have normal development in infancy but develop symptoms within the first few years of life. These symptoms can include speech delays, coarse facial features, recurrent respiratory infections, eye abnormalities, spine deformity, behavior problems, and intellectual disability. Symptoms worsen with age, including the loss of most learned speech by adulthood and declining intellectual abilities. Adults with Aspartylglycosaminuria may develop seizures, fragile bones, loose joints and skin, or movement problems. Lifespan is reduced with this condition with most people with Aspartylglycosaminuria living to their thirties or forties. Currently there is no cure for this condition and treatment is based on symptoms.

Gene markers analyzed: 108
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

AGA

16. Ataxia-Telangiectasia Syndrome

Ataxia-Telangiectasia is an autosomal recessive disorder that affects many parts of the body. Signs and symptoms usually begin in the first year of life and include problems with movement and coordination (ataxia), slurred speech, and abnormal eye movements. The ataxia worsens over time and affected children usually require a wheelchair by the teen years. Groups of enlarged blood vessels called telangiectases develop on the skin and eyes. People with Ataxia-Telangiectasia have a weakened immune system, may have frequent sinus and lung infections, are at increased risk to develop cancer, especially leukemia and lymphoma, and are sensitive to the effects of radiation including X-rays. Lifespan is often shortened in this disorder. In some cases, affected individuals have been treated with stem cell transplantation from cord blood or bone marrow. Couples at risk of having an affected child may consider cord blood banking, as siblings have a higher chance of being a match for stem cell transplantation than a non-related individual. Female carriers of Ataxia-Telangiectasia are at increased risk for developing breast cancer. Male and female carriers of Ataxia-Telangiectasia may be sensitive to the effects of radiation and may be at higher risk for developing other types of cancer as well. Carriers also may have a higher risk for heart disease.

Gene markers analyzed: 4172
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: 1*

* Heterozygous variant c.2119T>C (p.Ser707Pro) in Chromosome 11 and location 11q22.3 is a single nucleotide variant with Clinical Significance: Conflicting interpretations of pathogenicity. This variant is predicted to be harmful by 0 mutation effect prediction tools.

Genes analyzed:

ATM

17. Autosomal Recessive Osteopetrosis 1

Osteopetrosis, Infantile Malignant, TCIRG1-Related is a severe autosomal recessive type of Osteopetrosis, a group of disorders that cause bones to become overly dense and fracture easily. Symptoms are usually seen by early infancy and include multiple bone fractures and dense skull bones which often harm nerves in the head and face. The nerve damage may result in loss of vision, hearing, and facial movement. Bones are easily fractured, even with minor falls or stress. Children with Osteopetrosis, Infantile Malignant, TCIRG1-Related may also have reduced bone marrow function which can cause severe anemia and repeated infections. Slow growth, short stature, and enlarged spleen and liver are also common. Some children also have brain abnormalities, seizures and intellectual disability, although this is less common. In some cases, affected individuals have been treated with stem cell transplantation from cord blood or bone marrow. Couples at risk of having an affected child may consider cord blood banking, as siblings have a higher chance of being a match for stem cell transplantation than a non-related individual.

Gene markers analyzed: 144
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

TCIRG1

18. Autosomal Recessive Polycystic Kidney Disease

Polycystic Kidney Disease, Autosomal Recessive (ARPKD) is an autosomal recessive disorder that affects the kidneys and other organs, including the liver. Affected children are typically born with enlarged kidneys with multiple fluid-filled sacs called cysts. The kidneys do not work properly causing serious health problems. The fetal kidney problems begin in pregnancy and often affect fetal lung development. Lung development is affected by low fluid levels in the pregnancy (oligohydramnios) resulting from the kidney disease. Children born with Polycystic Kidney Disease, Autosomal Recessive often have very serious lung disease that may lead to death. Liver disease (congenital hepatic fibrosis) happens in about 45% of infants and children with Polycystic Kidney Disease, Autosomal Recessive. The disorder often leads to death in early infancy; however, some children have less severe symptoms and can survive with medical treatments. In very rare cases, symptoms do not start until adolescence or early adulthood.

Gene markers analyzed: 910
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

PKHD1

53. Cystic Fibrosis

Cystic Fibrosis is an autosomal recessive disorder that affects many different areas of the body including the lungs, digestive system, and fertility. Cystic Fibrosis does not affect intelligence. Signs and symptoms of Cystic Fibrosis start in early childhood and include delayed growth caused by problems in digestion and repeated lung infections that lead to permanent lung damage. Children and adults with Cystic Fibrosis usually have frequent hospitalizations because of lung infections. Over time, complications of Cystic Fibrosis can lead to lung transplants and early death. There are treatments for Cystic Fibrosis that can lessen the severity of the symptoms; however, there is currently no cure.

Gene markers analyzed: 1251
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

CFTR

54. Cystinosis

Cystinosis is an autosomal recessive disorder that causes the amino acid cysteine, one of the building blocks of protein, to build up in cells of the body. The excess cysteine forms crystals which can damage tissues and organs in the body. Damage to the kidneys and eyes occurs most often, but damage to the muscles, thyroid, pancreas, and testes may also occur. There are three forms of Cystinosis that have symptoms which range from mild to severe. The most severe form, called Nephropathic Cystinosis, starts shortly after birth. Symptoms include poor growth and a kidney disorder that leads to loss of minerals and nutrients in the urine. Cysteine crystals also build up in the eyes, causing sensitivity to light, eye pain, and vision loss. Symptoms also include loss of muscle mass, difficulty swallowing, diabetes, thyroid and nervous system problems. The childhood-onset form starts later but shows the same type of symptoms. There is also a milder form that causes eye problems but usually does not cause kidney damage. Medical treatment can lessen or delay some of symptoms of Cystinosis. Without treatment, children with Cystinosis may develop kidney failure by age 10 and need a kidney transplant.

Gene markers analyzed: 232
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

CTNS

55. Deafness, Autosomal Recessive 77

Deafness, Autosomal Recessive 77 is an autosomal recessive disorder that affects hearing. Affected individuals usually develop hearing loss beginning in childhood. The hearing loss worsens with age. This condition does not cause other health problems.

Gene markers analyzed: 211
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

LOXHD1

56. Deficiency of Alpha-Mannosidase

Alpha-Mannosidosis is an autosomal recessive disorder that causes toxic buildup of certain types of sugars, called oligosaccharides, in the body. There are mild and severe forms of Alpha-Mannosidosis with signs and symptoms typically beginning in infancy or later in childhood. In rare cases, symptoms may not begin until adulthood. Many parts of the body are affected leading to distinctive facial features, intellectual disability, developmental delays, bone abnormalities, movement problems, muscle weakness, joint problems, frequent infections, psychiatric problems, and hearing loss. The condition worsens with time. People with Alpha-Mannosidosis often require a wheelchair. Death may occur in childhood; however life-span may be near normal in individuals with a milder form of the condition. In some cases, affected individuals have been treated with stem cell transplantation from cord blood or bone marrow. Couples at risk of having an affected child may consider cord blood banking, as siblings have a higher chance of being a match for stem cell transplantation than a non-related individual.

Gene markers analyzed: 238
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

MAN2B1

101. Hypophosphatasia

Hypophosphatasia, ALPL-Related is a disorder inherited in either an autosomal recessive or autosomal dominant pattern that causes weakened bones and teeth. Symptoms vary from person to person and may start in infancy or not until later in childhood or adulthood. Infants with the severe form of this disorder have short bowed limbs, an abnormally shaped chest, and soft skull bones. Other symptoms may include feeding difficulties, growth delays, breathing problems, too much calcium in the blood, vomiting, and kidney disease, which can sometimes be life-threatening. In some cases symptoms do not start until later childhood or early adulthood, are often less severe, and can include early loss of baby teeth and then adult teeth, bowed legs, repeated bone fractures, softening of the bones (osteomalacia), short stature, and enlarged painful joints. Some people with a milder form of this condition typically only have abnormalities of the teeth, excess cavities, and early loss of teeth with no other symptoms.

Gene markers analyzed: 175
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

ALPL

102. Inclusion Body Myopathy 2

Inclusion Body Myopathy 2 is an autosomal recessive disorder that causes progressive muscle weakness in the legs and arms. Signs and symptoms of this disorder usually start in the late teenage years or early twenties but some people do not have problems until their thirties or forties. The first symptom is typically weakness in the muscles of the lower leg. As these muscles slowly weaken, walking becomes more difficult. The ability to walk is usually lost about 20 years after symptoms first appear. Muscle weakness worsens and starts to affect the muscles of the hips, hands, shoulders, neck, and occasionally the face. Intelligence is not affected. A small number of people with the gene mutations that cause this condition never show symptoms. Currently there is no cure for Inclusion Body Myopathy 2 and treatment is based on symptoms.

Gene markers analyzed: 222
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

GNE

103. Infantile Sialic Acid Storage Disease ;Salla Disease

Salla Disease, one form of Sialic Acid Storage Disease, is an autosomal recessive disorder that mainly affects the nervous system. Most infants with Salla Disease appear normal at birth. Then during infancy signs and symptoms begin to appear including slowly progressing loss of skills, poor muscle tone (hypotonia) that changes with time to tight and stiff muscles (spasticity), seizures, developmental delay, intellectual disability, speech problems, coordination problems (ataxia), and slow involuntary movements (athetosis) of the arms and legs. Although symptoms vary from person to person, about two-thirds of people with Salla Disease are not able to walk. Most people with Salla Disease live into adulthood. Currently there is no cure for this condition and treatment is based on the symptoms.

Gene markers analyzed: 107
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

SLC17A5

104. Isovaleryl-Coa Dehydrogenase Deficiency

Isovaleric Acidemia is a type of autosomal recessive condition known as an organic acid disorder. People with Isovaleric Acidemia have problems breaking down an amino acid called leucine from the food they eat. This inability to breakdown proteins that contain leucine causes harmful substances to build up in their blood and urine. The symptoms of Isovaleric Acidemia range from mild to severe. In the severe form signs and symptoms can begin in the first days of life and include poor appetite, lethargy (extreme tiredness), vomiting, a “sweaty feet” smell, and seizures. Early death may occur if the condition is not treated. Children with a milder form of Isovaleric Acidemia may have failure to grow and gain weight at the typical rate and may have delayed development. Most people with Isovaleric Acidemia need lifelong dietary and medical treatment. However, there are rare individuals with Isovaleric Acidemia who never show symptoms.

Gene markers analyzed: 146
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

IVD

117. Lysinuric Protein Intolerance

Lysinuric Protein Intolerance is an autosomal recessive disorder in which certain building blocks of protein (amino acids) cannot be broken down correctly by the body. This leads to a toxic buildup of ammonia in the blood. Symptoms of Lysinuric Protein Intolerance usually first begin in infancy after the baby is weaned off breast milk or formula and starts eating solid food. Symptoms include nausea, vomiting, poor feeding and poor growth, aversion to protein-rich foods, poor muscle tone, brittle bones, enlarged liver and spleen, and lung and kidney problems. Treatment with a medical low-protein diet along with specific supplements and medications can lessen the severity of symptoms but cannot prevent them.

Gene markers analyzed: 127
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

SLC7A7

118. Lysosomal Acid Lipase Deficiency

Lysosomal acid lipase deficiency is a metabolic lipid storage disease. Two rare conditions may result from this deficiency. 1. Wolman disease: The early-onset and most severe form of the disease where lipids accumulate throughout the body, mostly in the liver, within the first weeks of life. Symptoms include an enlarged liver and spleen (hepatosplenomegaly), poor weight gain, a yellowish color of the skin and the whites of the eyes (jaundice), vomiting, diarrhea, fatty stool (steatorrhea), and poor absorption of nutrients from food (malabsorption), as well as calcium deposits in adrenal glands, anemia, liver disease (cirrhosis), and developmental delay. Infants with this form of lysosomal acid lipase deficiency develop failure in multiple organs, and severe malnutrition. 2. Cholesteryl ester storage disease: Less severe and starting later in life. Symptoms may include hepatosplenomegaly, liver disease (cirrhosis), and malabsorption with diarrhea, vomiting, and steatorrhea.

Gene markers analyzed: 128
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

LIPA

119. Maple Syrup Urine Disease Type 1A

Maple Syrup Urine Disease, Type 1A is an autosomal recessive disorder in which the body is unable to break down certain building blocks of protein from food. Signs and symptoms usually begin in infancy and include poor feeding, vomiting, lack of energy, failure to grow at the normal rate, and developmental delay. Maple Syrup Urine Disease gets its name from the maple syrup odor of the urine in babies with the disease. Symptoms may worsen after going a long time without food or with illness and can be life-threatening. Lifelong dietary treatment is needed. If untreated, Maple Syrup Urine Disease, Type 1A can lead to intellectual disability, seizures, coma, and sometimes death. Even with treatment affected children continue to have symptoms of the disorder. Some children have a milder form of Maple Syrup Urine Disease, Type 1A with fewer symptoms.

Gene markers analyzed: 30
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

BCKDHA

120. Meckel-Gruber Syndrome

Meckel-Gruber syndrome is a rare and lethal autosomal recessive disorder characterized by occipital encephalocele, postaxial polydactyly and bilateral dysplastic cystic kidneys. It can be associated with many other conditions. Antenatal ultrasound examination establishes the diagnosis by identifying at least two of the major features described. We describe a female baby who had the typical triad of Meckel-Gruber syndrome and died shortly after birth.

Gene markers analyzed: 131
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

RPGRIP1L

139. Nephronophthisis 1

Nephronophthisis is a disorder that affects the kidneys. It is characterized by inflammation and scarring (fibrosis) that impairs kidney function. These abnormalities lead to increased urine production (polyuria), excessive thirst (polydipsia), general weakness, and extreme tiredness (fatigue). In addition, affected individuals develop fluid-filled cysts in the kidneys, usually in an area known as the corticomedullary region. Another feature of nephronophthisis is a shortage of red blood cells, a condition known as anemia.

Gene markers analyzed: 112
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

NPHP1

140. Nephrotic Syndrome, Idiopathic, Steroid-Resistant

Steroid-Resistant Nephrotic Syndrome is an autosomal recessive disorder that causes abnormal kidney function. People with this condition have large amounts of protein in their urine, low amounts of albumin (a protein in the plasma of the blood), high levels of fat in the blood, and excess fluid in body tissues (edema). Symptoms vary from person to person but usually start in childhood. The kidney problems worsen over time, often leading to kidney failure in the teenage years or early adulthood. Once kidney failure occurs, dialysis and then kidney transplantation are needed. Currently there is no cure for this condition and treatment is based on symptoms.

Gene markers analyzed: 107
Gene markers present in your genome data: 1
Potential pathogenic variants found in your genome data: None

Genes analyzed:

NPHS2

141. Neuronal Ceroid Lipofuscinosis 2

Neuronal Ceroid Lipofuscinosis, TPP1-Related (also known as CLN2 Disease, Late-Infantile Neuronal Ceroid Lipofuscinosis, or Juvenile Batten Disease) is autosomal recessive. It is one of a group of inherited disorders that affect the nervous system as well as other parts of the body. Signs and symptoms of Neuronal Ceroid Lipofuscinosis, TPP1-Related typically begin in early childhood with epileptic seizures. Over time, affected children have vision loss and intellectual decline, develop a movement disorder, and lose developmental and motor skills. Symptoms worsen with time and lifespan is shortened with death usually occurring by adolescence or early adulthood. Currently there is no cure or specific treatment for this disorder.

Gene markers analyzed: 242
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

TPP1

142. Neuronal Ceroid Lipofuscinosis 3

Neuronal ceroid lipofuscinosis 3 is a condition associated with mutation(s) in the CLN3 gene, encoding battenin. The condition is one of a group of genetically heterogeneous neurodegenerative disorders, characterized by accumulation of intracellular lipopigments

Gene markers analyzed: 223
Gene markers present in your genome data: 2
Potential pathogenic variants found in your genome data: None

Genes analyzed:

CLN3

182. Usher Syndrome Type 2A

Usher Syndrome, Type 2A is autosomal recessive. It is one of a group of inherited disorders that cause progressive hearing and vision loss. In most cases of Usher Syndrome, Type 2A, moderate to severe hearing loss is present at birth and affects higher frequencies more than lower frequencies. Speech involves lower frequencies, so speech and understanding language is often possible for children with this condition, although hearing aids and speech therapy are often needed. Retinitis Pigmentosa is an eye condition that occurs in individuals with Usher Syndrome, Type 2A and leads to damage to the retina, causing progressive loss of eyesight. Retinitis Pigmentosa with vision loss usually starts in the teenage years. Usher Syndrome, Type 2A does not affect intelligence or life span. Some individuals with Usher Syndrome, Type 2A have Retinitis Pigmentosa only and do not have hearing loss. Currently there is no cure for this condition.

Gene markers analyzed: 655
Gene markers present in your genome data: 3
Potential pathogenic variants found in your genome data: None

Genes analyzed:

USH2A

183. Usher Syndrome Type 3A

Usher Syndrome, Type 3 is autosomal recessive. It is one of a group of inherited disorders that cause progressive hearing and vision loss. People with Usher Syndrome, Type 3 usually start losing their hearing in late childhood or the early teenage years and typically have profound deafness by adulthood. Balance may also be affected, causing problems with walking and coordination. Retinitis Pigmentosa is an eye condition that occurs in most individuals with Usher Syndrome, Type 3 and leads to damage to the retina, causing progressive loss of eyesight starting in childhood or the teenage years. Usher Syndrome, Type 3 does not affect intelligence or life span. The symptoms of Usher Syndrome, Type 3 vary from person to person and some people have less severe hearing loss. Other people may have hearing loss only and do not develop Retinitis Pigmentosa. Currently there is no cure for this Usher Syndrome, Type 3.

Gene markers analyzed: 71
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

CLRN1

184. Walker-Warburg Congenital Muscular Dystrophy

A rare autosomal recessive inherited muscular dystrophy. It presents with generalized hypotonia, muscle weakness, mental retardation, developmental delays, and brain and eye abnormalities.

Gene markers analyzed: 141
Gene markers present in your genome data: NOT DETECTED
Potential pathogenic variants found in your genome data: NOT DETECTED

Genes analyzed:

FKTN

185. Wilson Disease

Wilson Disease is an autosomal recessive disorder that causes copper from the diet to build up in certain parts of the body, especially the liver, eyes, and brain. Signs and symptoms of Wilson Disease usually begin in the teenage years and in rare cases not until adulthood. Symptoms include liver disease, nervous system and psychiatric problems, and specific eye findings called Kayser-Fleischer rings (green/brown colored areas of excess copper on the surface of the eyes that do not interfere with vision). Other symptoms may include problems with coordination, movement, and behavior. Wilson Disease is commonly treated through chelation therapy to remove the excess stored copper from the body. This treatment helps to slow, and in some cases stop, the progression of the disease and improve symptoms. With treatment, people with Wilson Disease can have a normal lifespan.

Gene markers analyzed: 483
Gene markers present in your genome data: 7
Potential pathogenic variants found in your genome data: None

Genes analyzed:

ATP7B

مرکز ژنتیک حنیفا

نشانی: تهران، بزرگراه مدرس به سمت شمال
خیابان الهـیه شمالی، خیابان گلـنار، پـلاک ۵۲
ساختمان گالریا رزیدنس، طبقه ۱، واحد غربی

(۰۲۱) ۹۱۰۰۶۰۰۰
(۰۹۹۸) ۱۱۱۶۰۰۰
hanifagenetics
www.hanifa.ir