

Table S1. A list of the most common hereditary HTG types and associated genetic variants [21].

Condition	Prevalence	Inheritance	Genetic basis	Pathophysiology	Clinical features
Familial chylomicronemia syndrome	1 in 1 million	Autosomal recessive	Biallelic mutations in <i>LPL</i> , <i>APOC-II</i> , <i>APOA-V</i> , <i>LMF1</i> , <i>GPIHBP1</i> or <i>GPD1</i>	Defective LPL mediated clearance of chylomicrons	Serum triglycerides generally >1000 mg/dL with triglyceride to total cholesterol ratio around 10:1; recurrent pancreatitis from childhood, eruptive xanthoma, lipemia retinalis, hepatosplenomegaly
Familial hypertriglyceridemia	5-10%	No clear mendelian pattern	Polygenic with environmental influence	Increased production of triglyceride-rich VLDL particles	Serum triglycerides in 200-1000 mg/dL range with normal total cholesterol and apolipoprotein B concentrations; generally not associated with increased risk of ASCVD or pancreatitis in absence of other risk factors
Familial combined hyperlipidemia	1-2%	No clear mendelian pattern	Polygenic with environmental influence	Increased production of apolipoprotein B and associated lipoproteins	Elevated serum triglycerides, total cholesterol, or both, with elevated apolipoprotein B in patients and first degree relatives; high risk of ASCVD
Familial (type 3) dysbetalipoproteinemia	1 in 10 000	Usually autosomal recessive; rarely autosomal dominant	<i>APOE2/E2</i> genotype (AR) or rare <i>APOE2</i> mutations (AD)	Defective apolipoprotein E mediated clearance of VLDL and chylomicron remnants	Near equivalent elevations in serum total cholesterol and triglycerides (usually 300-500 mg/dL); palmar and tuberos xanthomas; secondary factors often present
Inherited lipodystrophy syndromes:					
Congenital generalized lipodystrophy	1 in 10 million	Autosomal recessive	Biallelic mutations in <i>AGPAT2</i> , <i>BSCL2</i> , <i>CAV1</i> or <i>PTRF</i>	Defective adipocyte development and differentiation leading to loss of subcutaneous fat	Generalized loss of body fat from birth with features of extreme insulin resistance
Familial partial lipodystrophy	1 in 1 million	Autosomal dominant; rarely autosomal recessive	Mutations in <i>LMNA</i> , <i>PPARG</i> , <i>PLIN1</i> , <i>CIDEA</i> , <i>LIPE</i> , <i>AKT2</i> or <i>ADRA2A</i>	and tendency for hepatic steatosis and VLDL overproduction	Variable loss of subcutaneous fat from extremities and trunk starting in peripubertal period; features of insulin resistance

AD=autosomal dominant; ADRA2A=adrenoceptor α 2a; AGPAT2=1-acylglycerol-3-phosphate O-acyltransferase 2; AKT2=v-akt murine thymoma viral oncogene homolog 2; APOA5=apolipoprotein A5; APOC2=apolipoprotein C2; APOE2=apolipoprotein E2; AR=autosomal recessive; ASCVD=atherosclerotic cardiovascular disease; BSCL2=Berardinelli-Seip congenital lipodystrophy 2; CAV1=caveolin 1; CIDEA=cell death-inducing DFFA-like effector c; GPIHBP1=glycosylphosphatidylinositol-anchored high density lipoprotein binding protein-1; GPD1=glycerol-3-phosphate dehydrogenase 1; LIPE=hormone sensitive lipase; LMF=lipase maturation factor; LMNA=lamin A/C; LPL=lipoprotein lipase; PLIN1=perilipin; PPARG=peroxisome

proliferator-activated receptor gamma; PTRF=polymerase I and transcript release factor; VLDL=very low density lipoprotein

Table S2. List of 60 genes in our custom panel responsible for the development of primary dyslipidemias.

<i>ABCA1</i>	NM_005502	<i>APOE</i>	NM_000041	<i>HNF1A</i>	NM_000545	<i>NPC1</i>	NM_000271
<i>ABCG1</i>	NM_207174	<i>APOH</i>	NM_000042	<i>LCAT</i>	NM_000229	<i>NPC1L1</i>	NM_013389
<i>ABCG5</i>	NM_022436	<i>BSCL2</i>	NM_032667	<i>LDLR</i>	NM_000527	<i>NPC2</i>	NM_006432
<i>ABCG8</i>	NM_022437	<i>CAV1</i>	NM_001753	<i>LDLRA P1</i>	NM_015627	<i>PCSK9</i>	NM_174936
<i>AGPAT2</i>	NM_006412	<i>CAV2</i>	NM_001233	<i>LIPA</i>	NM_000235	<i>PLIN1</i>	NM_002666
<i>ALMS1</i>	NM_015120	<i>CAVIN1</i>	NM_012232	<i>LIPC</i>	NM_000236	<i>PLTP</i>	NM_006227
<i>ANGPTL3</i>	NM_014495	<i>CETP</i>	NM_000078	<i>LIPE</i>	NM_005357	<i>PPARA</i>	NM_005036
<i>APOA1</i>	NM_000039	<i>CH25H</i>	NM_003956	<i>LIPG</i>	NM_006033	<i>PPARG</i>	NM_015869
<i>APOA2</i>	NM_001643	<i>CIDEA</i>	NM_022094	<i>LMF1</i>	NM_022773	<i>PPP1R17</i>	NM_006658
<i>APOA4</i>	NM_000482	<i>COQ2</i>	NM_015697	<i>LMNA</i>	NM_170707	<i>PYGM</i>	NM_005609
<i>APOA5</i>	NM_052968	<i>CPT2</i>	NM_000098	<i>LMNB2</i>	NM_032737	<i>SAR1B</i>	NM_001033503
<i>APOB</i>	NM_000384	<i>CREB3L3</i>	NM_032607	<i>LPA</i>	NM_005577	<i>SCARB1</i>	NM_005505
<i>APOC1</i>	NM_001645	<i>GCK</i>	NM_000162	<i>LPL</i>	NM_000237	<i>SLCO1B1</i>	NM_006446
<i>APOC2</i>	NM_000483	<i>GPD1</i>	NM_005276	<i>MTTP</i>	NM_000253	<i>SLCO1B3</i>	NM_019844
<i>APOC3</i>	NM_000040	<i>GPIIB P1</i>	NM_178172	<i>MYLIP</i>	NM_013262	<i>STAP1</i>	NM_012108