Supplementary Material

Supplementary Table 1. Implicated genes and mechanisms of cardiac diseases and/or SCD

Gene	Gene name	Chr	Associations	Proposed mechanisms	Identified SNPs
Coronary artery disease					
ANGPTL4	Angiopoietin-like 4	19	CAD	Inhibits LPL activity[1]	-
APOA5	Apolipoprotein A-V	11	CAD	Regulates plasma TG levels;[2] increases LPL activity[1]	-
APOC3	Apolipoprotein C-III	11	CAD	Regulates plasma TG levels;[2] inhibits LPL activity[1]	-
LDLR	LDL receptor	19	CAD, FH	LDL clearance; associated with FH[2]	Variants associated with CAD: Khera *et al* (USA[3])
LPA	Lipoprotein (A)	6	CAD	Increased lipoprotein(a) levels[2]	-
LPL	Lipoprotein lipase	8	CAD	Lipolysis of TG-rich lipoproteins[2]	-
PCSK9	Proprotein convertase subtilisin/kexin type 9	1	CAD	Regulates LDL receptor recycling[2]	-
Sudden cardiac death in coronary artery disease					
ACYP2	Acylphosphatase 2	2	CAD	Unknown[4]	• rs1559040 (USA[4])
AP1G2	Adaptor related protein complex 1 gamma 2 subunit	14	CAD	Unknown[4]	• rs2281680 (USA[4]) • rs11624056 (USA[4])
BAZ2B	Bromodomain adjacent to zinc finger 2B	2	CAD	Unknown; Involved in chromatin remodelling and gene transcription[5]	• rs4665058 (Europe[6])
CASQ2	Calsequestrin 2	1	CAD, CPVT	increased release of calcium from sarcoplasmic reticulum[7,8]	• rs17500488 (USA[7]) • rs3010396 (USA[7]) • rs7366407 (USA[7])
CXADR	Coxsackie virus and adenovirus receptor	21	CAD	Progressive atrioventricular block;[9] reduced membrane excitability and electrical uncoupling during myocardial ischemia[10]	• rs2824292 (Netherlands[11])
DEGS2	Delta 4-desaturase, sphingolipid 2	14	CAD	Unknown[12]	• rs7157599 (USA[4]) • rs17718586 (USA[4]) • rs597503 (USA[4])
ESR1	Oestrogen receptor alpha	6	CAD	Regulates multiple genes after activation by oestrogen, increases risk of CAD[4]	• **rs2982694** (USA[4], China[13]) • **rs12429889** (USA[4], China[13]) • rs10765792 (USA[4]) • rs7307780 (USA[4])
GPD1L	Glycerol-3-phosphate dehydrogenase 1-like	3	CAD, BrS	Reduced trafficking of cardiac sodium channel to cell surface; reduces inward sodium current; associated with BrS[7,8]	• rs9862154 (USA[7])
KCTD1	Potassium channel tetramerisation domain containing 1	18	CAD	Unknown[12]	• **rs16942421** (USA[4], China[13])
GPC5	Glypican 5	13	CAD	Unknown[12]	• rs3864180 (USA[12])
GRIA1	Glutamate ionotropic receptor AMPA type subunit 1	5	CAD	Unknown[12]	• rs12189362 (USA[4])
NOS1AP *	Nitric oxide synthase 1 adaptor protein	1	CAD, LQTS, CPVT, HCM	Prolongs QT interval; possible role for nitric oxide synthase pathway in myocardial function, but exact mechanism unknown.[7,14]	• rs12084280 (USA[7]) – CAD • rs10918859 (USA[7]) – CAD • **rs10494366** (Germany[15], Australia/New Zealand[16]) – QT • rs12567209 (USA[17]) – QT • **rs12143842** (Europe[18], New Zealand[19],

					International[20], Australia/New Zealand[16]) – QT • rs1202945 (Europe[18]) – QT • rs16857031 (Europe[18]) – QT • rs16847548 (New Zealand[19]) – QT
ZNF385B	Zinc finger protein 385B	2	CAD	Unknown[12]	• rs16866933 (USA[4]) • rs4621553 (USA[4])
Channelopathies					
CALM1	Calmodulin 1	14	CPVT	Disrupts calcium binding and causes calcium overload; abnormal interaction with *RYR2* calmodulin-binding-domain peptide[8]	-
CALM2	Calmodulin 2	2	CPVT	Disrupts calcium binding; reduces calcium-binding affinity[8]	-
CCR7	C-C chemokine receptor type 7	17	CPVT	Regulates expression of cardiac calcium channel, *RYR2* and *NOS1AP;* increases L-type calcium current density[21]	• rs78960694 (China[21]) • rs2229095 (China[21])
HEY2	Hairy/enhancer-of-split related with YRPW motif protein 2	6	BrS	Transcriptional regulator of cardiac electrical activity;[22] exact mechanisms unclear[23]	• rs9388451 (International[22], Japan[23]) – BrS
KCNE1	Potassium voltage-gated channel subfamily E regulatory subunit 1	21	LQTS	Decreases outward potassium current prolongs action potential, leading to prolonged QT interval[24]	Associated with QT interval: • **rs1805128** (Europe[18], North America and Europe[25] – *associated with Torsades de Pointe*) Associated with SCD: Khera *et al* (USA[3])
KCNH2	Potassium voltage-gated channel subfamily H member 2	7	LQTS	Decreases outward potassium current prolongs action potential, leading to prolonged QT interval[24]	Associated with QT interval: • rs2968863 (Europe[26]) • rs4725982 (Europe[18]) Associated with SCD: • Khera *et al* (USA[3]) • Guo *et al.* (USA[27])
KCNQ1	Potassium voltage-gated channel subfamily Q member 1	11	LQTS	Decreases outward potassium current prolongs action potential, leading to prolonged QT interval[24]	Associated with QT interval: • rs12296050 (Europe[26]) • rs2074238 (Europe[18]) • rs10798 (New Zealand[19]) • rs8234 (New Zealand[19]) • rs179405 (International[20]) Associated with SCD: • Khera *et al* (USA[3]) • Guo *et al.* (USA[27])
KCNJ2	Potassium inwardly rectifying channel subfamily J member 2	17	CPVT	Loss of potassium inward rectifier channel (Kir2) function results in membrane potential instability, promoting or failing to inhibit triggered activity[28]	• rs17779747 (Europe[26])
KLF12	Kruppel like factor 12	13	LQTS	Unknown[13,20,29]	• rs12429889 (China[13]) • rs728926 (Europe[29]) • rs17061696 (International[20])
RYR2	Ryanodine receptor 2	1	CPVT	Increases release of calcium from sarcoplasmic reticulum, triggering delayed afterdepolarisations[30]	Associated with SCD: • Guo *et al.* (USA[27]) • Weizman *et al.* (France[31])
SCN5A	Sodium voltage-gated channel alpha subunit 5	3	LQTS, BrS, DCM	• LQTS: increased inward sodium current prolongs action potential, leading to prolonged QT interval[24]	• rs11129795 (Europe[26]) – QT • rs12053903 (Europe[18]) – QT • rs11708996 (International[22]) – BrS

				• BrS: impairs trafficking of channel to cardiomyocyte membrane or alters channel biophysical properties, leading to decreased inward sodium current[32]	Associated with SCD: • Guo *et al.* (USA[27]) • Khera *et al.* (USA[3]) • Weizman *et al.* (France[31])
SCN10A	Sodium voltage-gated channel alpha subunit 10	3	BrS	Modifies sodium channel function; loss of function in sodium channel current[8]	• rs10428132 (International[22], Japan[23])
TRDN	Triadin	6	CPVT	Diastolic leak of calcium and overload of calcium in myocytes[8]	-
ZSCAN20	Zinc finger and SCAN domain containing 20	1	BrS	Unknown[33]	• rs2336244 (Japan and Europe[33])
Cardiomyopathies					
BAG3	Bcl-2-associated athanogene 3	10	DCM	Disrupted myofibril structure, relocated BAG3 protein in sarcomeric Z-disc and impaired contractile function[34]	• rs2234962 (Europe[35]) – DCM • rs61869036 (Europe[36]) – DCM • rs72840788 (United Kingdom[37]) – HCM
LMNA	Lamin A/C	1	DCM, ARVC	Unknown; May lead to changes in cellular signalling, nuclear fragility and abnormal interactions between lamins and other nuclear protein[38]	• p.R471H (Denmark[39]) – DCM • p.R644C (Denmark[39]) – ARVC • rs505058 (Iran[40]) – DCM
PKP2	Plakophilin 2	12	ACM	Disrupted desmosomes and gap junctions; reduced gap junction protein connexin 43, leading to impaired electrical coupling and conduction[41]	Associated with SCD: • p.R79X (Denmark[39]) • Khera *et al.* (USA[3]) • Guo *et al.* (USA[27]) • Weizman *et al.* (France[31])
SLC6A6	Solute carrier family 6 member 6	3	DCM	Depleted taurine levels; impaired myocardial function[36]	• Garnier *et al.* (Europe[36]) – DCM • rs13061705 (United Kingdom[37])
TMEM43	Transmembrane protein 43	3	DCM, ACM	Unknown[32]	• Garnier *et al.* (Europe[36]) – DCM
TNNT2	Cardiac troponin T	1	HCM	Increases myofilament calcium sensitivity and decreases calcium transients arising from increased cytosolic calcium buffering[42]	• Guo *et al.* (United States[27])
TTN	Titin	2	DCM	Disrupts assembly process of sarcomere, mechanical properties of cardiomyocytes, and prevents cardiac contractile function (for truncating mutations)[43]	• Khera et al. (United States[3])

ACM, arrhythmogenic cardiomyopathy; BrS. Brugada syndrome; CAD, coronary artery disease; Chr, chromosome; CPVT, catecholaminergic polymorphic ventricular tachycardia; DCM, dilated cardiomyopathy; FH, familial hypercholesterolaemia; HCM, hypertrophic cardiomyopathy; LDL, low-density lipoprotein; LDL-C, low-density lipoprotein cholesterol; LPL, lipoprotein lipase; LQTS, long QT syndrome; SNP, single nucleotide polymorphism; TG, triglyceride; USA, United States of America

Supplementary Table 2. Common genetic variants associated with cardiac disorders and/or risk of SCD

Year published	Author	Study type	Country	Population size	Gene (chromosome or locus), SNP(s)	Findings
Coronary artery disease and/or risk of SCD						
2010	Bezzina et al.[11] Arrhythmia Genetics in the Netherlands Study (AGNES)	GWAS	Netherlands	• 515 individuals with acute MI and VF • 457 individuals with acute MI only	Near *CXADR* (21q21), rs2824292	This SNP was an independent risk factor for VF in first acute MI.
2010	Arking et al.[12]	GWAS	USA	• 424 individuals with SCA and CAD • 226 controls with CAD only	*GPC5* (chr 13), rs3864180	This SNP conferred protection against SCA among individuals with CAD.
2011	Aouizerat et al.[4]	GWAS	USA	• 89 individuals with CAD and SCA due to VT/VF • 517 healthy controls	• *ACYP2* (2p16.2), rs1559040 • *ZNF385B:* 2q31.2 (rs16866933), 5q22.2 (rs4621553) • *GRIA1* (5q33.2), rs12189362 • *ESR1*: (6q25.1), rs2982694; (11q21), rs10765792; (12q21.2), rs7307780; (13q22.1), rs12429889 • *AP1G2:* (14q11.2), rs2281680; (14q31.3), rs11624056 • *DEGS2*: (14q32.2), rs7157599; (17q24.3), rs17718586; (18p11.31), rs597503 • *KCTD1* (18q11.2), rs16942421	These 14 SNPs were associated with increased arrhythmic SCA risk in the setting of CAD.
2011	Bugert et al.[44]	Case-control study	Germany	• 90 individuals with acute MI and VF • 167 individuals with acute MI without VF	-	There was no significant association of rs2824292 locus and risk of VF during acute MI.
2011	Westaway et al.[7]	Candidate-gene approach	USA	• 134 individuals with SCD and CAD • 147 controls with CAD	• *CASQ2* region (chr 1), rs17500488, rs3010396 and rs7366407 • *GPD1L* (chr 3), rs9862154 • *NOS1AP* (chr 1), rs12084280 and rs10918859	These 6 SNPs were associated with increased risk of SCD in CAD patients.
2021	Zhang et al.[13]	GWAS	China	198 Han Chinese individuals: • 70 individuals with CAD • 53 individuals with SCD • 75 controls	Associated with SCD: • *KLF12, RNY1P5* (chr 13), rs12429889 • *ARL5B* (chr 10), rs10829156 • *KCTD1* (chr 18), rs16942421 • *EFCAB1, SNAI2* (chr 8), rs12155623 Associated with SCD from CAD: • *MIR1973, TRMT112P1* (chr 4), rs2389202 • *ESR1* (chr 6), rs2982694 • *ACVR1, UPP2* (chr 2), rs10183640 • *SCML2P1, LAMA1* (chr 18), rs597503 • *KCTD1* (chr 18), rs16942421 • *EFCAB1, SNAI2* (chr 8), rs12155623	Identified four SNPS associated with SCD and six SNPS associated with SCD from CAD. No effect for rs4665058 (previously identified in Arking *et al.*[33]), suggesting that genetic variants may have different effects in different ethnic populations.
Channelopathies and/or risk of SCD						

2006	Arking et al.[15]	GWAS	Germany	200 individuals at the extremes of population-based QT interval distribution	*NOS1AP* (rs10494366)	*NOS1AP was* significantly associated with QT interval variation (explaining up to 1.5% of QT interval variation). *NOS1AP* modulates cardiac repolarisation.
2009	Kao et al.[17]	Population-based prospective cohort studies	USA	19,295 black and white individuals	*NOS1AP* (rs12567209)	Sequence variations in NOS1AP were associated with baseline QT interval and increased risk of SCD in white adults.
2009	Pfeufer et al.[26] QTSCD Consortium	GWAS of five population-based cohorts	Europe	15,842 individuals	• *NOS1AP* (chr 1), rs12143842 • *KCNQ1* (chr 11), rs12296050 • *KCNH2* (chr 7), rs2968863 • *SCN5A* (chr 3), rs11129795 • *KCNJ2* (chr 17), rs17779747 • *ATP1B1* (chr 1), rs10919071 • *PLN* (chr 6), rs11970286 • *RNF207* (chr 1), rs846111 • *LITAF* (chr 16), rs8049607 • *NDRG4* (chr 16), rs7188697	These 10 SNPs modulated QT interval duration.
2009	Newton-Cheh et al.[18] QTGEN Consortium	Meta-analysis of three GWAS	Europe	13,685 individuals (from Framingham Heart Study, Rotterdam Study, Cardiovascular Health)	Previously identified loci: • *NOS1AP* (chromosome 1q), rs12143842, rs12029454, rs16857031 • *KCNQ1* (chromosome 11p), rs2074238 • *KCNE1* (chromosome 21q), rs1805128 • *KCNH2* (chromosome 7q), rs4725982 • *SCN5A* (chromosome 3p), rs12053903 Newly identified loci: • Near *NDRG4* and *GINS3, falls in intron CNOT1* (16q21), rs37062 • *c6orf204-PLN* region (6q22), rs11756438 • Near *RNF207* (1p36), rs846111 • Near *LITAF* (16p13), rs8049607 • Near *LIG3* and *RFFL* (17q12), rs2074518	These SNPs explained a substantial proportion of QT interval variation (5.4% Framingham, 6.5% Rotterdam, 2.3% Cardiovascular Health Study).
2011	Arking et al.[6]	Meta-analysis of five GWAS	Europe	• 1283 SCD cases • Around 20,000 controls	*BAZ2B* (2q24.2), rs4665058	Risk allele increases risk of SCD by 1.92-fold per allele. However, no effect for rs4665058 was seen in AGNES (only involved first MI patients).
2011	Kääb et al.[25]	Candidate-gene approach	North America and Europe (European descent)	176 individuals with drug-induced long-QT syndrome	*KCNE1* D85N (chr 21), rs1805128	Common variant in KCNE1 D85N (rs1805128) increased risk of torsades de pointes in association with QT-prolonging drugs
2013	Bezzina et al.[22]	GWAS	International	• 312 individuals with BrS • 1115 controls	• *HEY2* (chr 6), rs9388451 • *SCN5A* (chr 3), rs1170899 • *SCN10A* (chr 3), rs10428132	These SNPs were associated with BrS. Disease risk increased with increasing number of risk alleles, with an estimated odds ratio of 21.5 with more than four risk alleles (versus less than two)

2013	Earle et al.[19]	Population-based cohort study	New Zealand	273 individuals with LQTS	• *NOS1AP* (rs12143842 and rs16847548) • *KCNQ1* (rs10798 and rs8234)	SNPs at *NOS1AP* were predictors for SCD in LQTS patients, but not predictors for QTc length.
2014	Arking et al.[29] QT Interval–International GWAS Consortium (QT-IGC)	Meta-analysis of GWAS	Europe	76,061 individuals	22 newly identified loci: • *TCEA3* (1p36), rs2298632 • *SP3* (2q31.1), rs938291 • *TTN-CCDC141* (2q31.2), rs7561149 • *SPATS2L* (2q33), rs295140 • *C3ORF75* (3p21), rs17784882 • *SLC4A4* (4q13), rs2363719 • *SMARCAD1* (4q22), rs3857067 • *GFRA3* (5q31), rs10040989 • *GMPR* (6p22), rs7765828 • *CAV1* (7q31), rs9920 • *NCOA2* (8q13), rs16936870 • *LAPTM4B* (8q22.1), rs11779860 • *AZIN1* (8q22.3), rs1961102 • *GBF1* (10q24), rs2485376 • *FEN1-FADS2* (11q12), rs174583 • *ATP2A2* (12q24), rs3026445 • *KLF12* (13q22), rs728926 • ANKRD9 (14q32), rs2273905 • *USP50-TRPM7* (15q21), rs3105593 • *CREBBP* (16p13.3), rs1296720 • *MKL2* (16p13.12), rs246185 • *PRKCA* (17q24), rs9892651	These 35 SNPs (including 22 newly identified loci) were associated with QT interval and collectively explained 8-10% of QT interval variation.
2016	Nakano et al.[23]	Population-based cohort study	Japan	• 95 individuals with BrS 1,978 controls	• *SCN10A* (rs10428132) • *HEY2* (rs9388451)	These SNPs were associated with BrS. No relationship between previously reported SCN5A (rs11708996). HEY2 C allele was more common in BrS patients without VF than in those with VF.
2020	Lahrouchi et al.[20]	GWAS, followed by transethnic meta-analysis (European or Japanese ancestry)	International : Europe, New Zealand, North America, Japan	1,781 multi-ethnic, unrelated individuals with LQTS: • 1,344 individuals from 23 referral centres (Europe, New Zealand, North America) 437 patients from Japan	• *NOS1AP* (chr 1), rs12143842 • *KCNQ1* (chr 11), rs179405 • *KLF12* (chr 13), rs17061696	These SNPs were associated with LQTS. Around 50% of the variance in QT interval susceptibility was due to common genetic variants.
2022	Fang et al.[21]	GWAS	Chinese	• 246 individuals with idiopathic VT 648 controls without VT	• *PKN* (chromosome 1p22.3, rs2390325 – 21.19% in cases, 15.12% in controls) • *CSMD1* (chromosome 8p23.2, rs270065 – 33.63% in cases, 40.25% in controls) • *NELL1* (chromosome 11p15.1, rs68126098 – 40.98% in cases, 32.07% in controls) • *CCR7* (chromosome 17q21.2, rs78960694 – 5.02% in cases and 1.84% in controls; rs2229095 – 3.25% in cases, 1.63% in controls)	These SNPs were associated with idiopathic VT.

2023	Makita et al.[33]	Meta-analysis of GWAS	Japan and Europe	• Japanese population: 940 individuals with BrS and 1,634 controls • European population: 3,760 individuals with BrS and 11,635 controls	• Previously reported SNPs in *SCN5A/SCN10A, HEY2* • Novel SNPs: *ZSCAN20* (rs2336244), *USP34, CAMK2D, TPPP, NREP, MYH7B*	Identified 17 association signals including 6 novel loci associated with BrS in both Japanese and European populations (exact SNPs not reported for all genes).
Cardiomyopathies and/or risk of SCD						
2011	Villard et al.[35]	GWAS	Europe	• 1,109 individuals with DCM • 1,108 controls	• ZBTB17 (chr 1p36.13), rs10927875 *BAG3* (chr 10), rs2234962	These SNPs were associated with sporadic DCM. Rare mutations in *BAG3* contributed to monogenic DCM.
2015	Early et al.[16]	Registry-based study	Australia and New Zealand	Individuals with HCM: • 208 individuals from Australia • 88 individuals from New Zealand	*NOS1AP* (chr 1), rs10494366 and rs12143842	These SNPs were associated with prolonged QTc, but not with risk of SCD in HCM patients.
2021	Garnier et al.[36]	GWAS	Europe	• 2,719 individuals with sporadic DCM • 4,440 controls	Two newly identified loci: • Distal to *LSM3* (3p25.1), rs62232870 • Topologically associating domain that encompasses six genes: CHCHD4, TMEM43, XPC, LSM3, SLC6A6, and GRIP2 (22q11.23), rs7284877 Two previously identified loci: • *BAG3* (chr 10), rs61869036 • *HSPB7* (chr 1), rs10927886	These SNPs were associated with DCM.
2021	Harper et al.[37]	GWAS	United Kingdom	Two GWAS of unrelated patients with HCM: • 2,541 unselected cases vs. 40,283 controls • 239 sarcomere-negative cases vs. 7,203 controls	• *HSPB7* (chr 1), rs1048302 • *SLC6A6* (chr 3), rs13061705 • *CDKN1A* (chr 6), rs3176326 • *PLN* (chr 6), rs12212795 • *BAG3* (chr 10), rs72840788 • *TBX3* (chr 12), rs7301677 • *ADPRHL1* (chr 13), rs41306688 • *ALPK3* (chr 15), rs8033459 • *SPPL2* (chr 17), rs28768976 • *PRKCA* (chr 17), rs7210446 • *FHOD3* (chr 18), rs4799426 • *FHOD3* (chr 18), rs118060942 • *MMP11* (chr 22), rs2070458	These 13 SNPs were associated with HCM.
2023	Erfanian et al.[40]	Case-control study	Iran	• 70 DCM patients • 70 healthy controls with normal LVEF (age- and sex-matched)	*LMNA* (chr 1), rs505058	This SNP was associated with familial DCM in the Iranian population.

BrS, Brugada syndrome; CAD, coronary artery disease; DCM, dilated cardiomyopathy; HCM, hypertrophic cardiomyopathy; GWAS, genome-wide association study; LQTS, long QT syndrome; LVEF, left ventricular ejection fraction; MI, myocardial infarction; SCA, sudden cardiac arrest; SCD, sudden cardiac death; SNP, single nucleotide polymorphism; USA, United States of America; VF, ventricular fibrillation; VT, ventricular tachycardia

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