Supplemental Online Content

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This supplemental material has been provided by the authors to give readers additional information about their work.

eMethods. Supplementary Methods

Study Design and Oversight

Between 1995 and 2015, CVPath Institute was referred a total of 5,262 hearts from cases of unexpected sudden death from the Office of the Chief Medical Examiner of the State of Maryland (OCME-MD). (Unexpected sudden death cases in the state of Maryland are routinely referred to our Institute for consultation at the discretion of the medical examiner.) At OCME-MD, a complete and comprehensive autopsy and toxicologic analysis is performed in all referred decedents up to 50 years old and in cases over 50 years old without evidence of possible drug/alcohol abuse. For every case, the heart was systematically evaluated with detailed histopathological analysis performed by an experienced cardiac pathologist at CVPath Institute. To examine race-based differences, only African American or White individuals ≥18 years old were analyzed in the current study (n=4,270). The racial origin of subjects was identified from the OCME-MD report through inquiry of family members. The racial makeup of the study is similar to that of the State of Maryland - White: 50%, African American: 31%, Hispanic: 10%, Asian 6%, and others: 3%. Subjects with a clear non-cardiac cause of death as well as cases with lack of detailed information were excluded from the study (n=950). Subjects with SCD due to sudden coronary death (n=1,875; i.e. acute coronary syndrome, ≥75% stenosis in any major epicardial coronary artery, or previous bypass or stent placement) and other known causes of sudden cardiac death (n=762; i.e. cardiomyopathy (e.g. hypertrophic, dilated or arrhythmogenic right ventricular

cardiomyopathies, etc.), significant valvular disease, congenital heart disease, myo/pericarditis, or infective endocarditis) were excluded from the study. See Figure 1A for study flowchart. The protocol for the study was approved by the Institutional Review Board of the CVPath Institute (Study ID: RP0027).

Assessment of Hearts to rule out any known cause of death.

The hearts were weighed after blood clots had been removed from the cavities and the heart ventricles were sliced parallel to the posterior atrioventricular junction to determine the presence or absence of any necrosis or fibrosis. Pulmonary emboli and any right ventricular abnormalities were assessed to rule out any attributable cause of death. The base of the heart was opened in the direction of blood flow and all valves were determined to be normal. The epicardial coronary arteries were sectioned at three to four intervals to rule out any significant atherosclerosis (>75% cross-sectional area stenosis). The base of the heart at the level of the tip of the mitral valve was used to measure the left ventricle (LV) free wall , ventricular septal thickness, and right ventricular wall thickness followed by left ventricular cavity diameter excluding the pectinate and papillary muscles. In total six sections of myocardium (anterior, posterior, and lateral LV, ventricular septum, anterior and posterior wall of the right ventricle) were routinely taken transversely, embedded in paraffin, and stained with hematoxylin and eosin (H&E) stain for histologic evaluation. Histologic examination of all six parts of myocardium (anterior, posterior, and lateral left ventricle, ventricular septum, anterior and posterior wall of the right ventricle) was

performed to rule out any infiltrative process or any myofiber disarray of the myocardium, intramyocardial small vessel disease of interstitial or focal fibrosis. Presence of any cardiomyopathic process was ruled out by gross and histologic examination. Wall thickness measurements from subjects with normal hearts were consistent with previous autopsy studies of "normal hearts" [1].

Of note, Maron et al. demonstrated that ventricular septal and free wall thicknesses were thicker in autopsy hearts when the thickness was compared with diastolic thickness measured by echocardiogram that was performed prior to death [2]. Therefore, wall thickness criteria used in clinical echocardiography is not used as a diagnostic criterion for cardiomyopathies during autopsy examination. Instead, increased septal/free wall ratio ($\geq$1.3) and/or abnormal histopathologic findings such as myofiber disarray, myocyte hypertrophy, fibrosis (scarring), and thickening of intramural coronary arteries are routinely used [3-8].

Definition of Normal Hearts

"Normal" heart was defined as an individual dying of natural causes with no evidence of heart disease following a complete gross and histopathological evaluation. Assessment of the myocardial sections ruled out the presence of any cardiomyopathic process. In addition, microscopic examination also helped to rule out severe myocyte hypertrophy (defined as cardiac myocyte thickness greater than 25 μm [9]), and severe fibrosis in the myocardium (area of fibrosis >3% of the section) (Figure 1). Suspected unexplained-SCD was defined as symptoms commencing within one hour of death (with or without witnessed

arrest) or death occurring within 24 hours after the victim was last seen alive in their normal state of health, and in whom a clear cause of death (CoD) could not be established after a complete and comprehensive autopsy examination (including cardiac examination). All cases of SCD with normal autopsy were adjudicated to be unexplained by three cardiologists (ST, CCH, AVF) and a cardiac pathologist (RV) from the final autopsy report from OCME-MD and cardiac autopsy report from CVPath Institute. Adjudications were completed before the genetic results were made available.

Postmortem criteria for diagnosis

Sudden coronary death was defined as subjects with acute coronary syndrome (acute thrombus in epicardial coronary artery due to plaque rupture, plaque erosion, or calcified nodule), old myocardial infarction, and at least one epicardial coronary artery with >75% cross-sectional area lumen narrowing by atherosclerotic plaque.

Explained-SCD was defined as subjects with autopsy findings consistent with evidence of cardiomyopathy [hypertrophic cardiomyopathy (HCM), dilated cardiomyopathy (DCM), arrhythmogenic right ventricular cardiomyopathy (ARVC)], congenital heart disease, myocarditis, infective endocarditis, and valvular disease (i.e. mitral valve prolapse, aortic valve stenosis, etc.). Diagnostic criteria of HCM was increased septal/free wall ratio ($\geq$1.3) and/or abnormal histopathologic findings such as myofiber disarray, myocyte hypertrophy, fibrosis (scarring), and thickening of intramural coronary arteries [3-8].

DNA Collection, Next-generation Sequencing and Data Analysis

Genomic DNA was extracted from either fresh frozen tissue or formalin-fixed paraffin-embedded (FFPE) tissue blocks using DNeasy Tissue Kit and QIAamp DNA FFPE Tissue Kit (Qiagen), respectively. DNA samples were analyzed on TapeStation (Agilent) for quality check, and only the DNA samples met the quality requirements for TruSeq Custom Amplicon Low Input by TruSeq FFPE DNA Library Prep QC Kit (Illumina) and Primary Electrical Disorders (PED) MASTR Plus by MASTR QC plex (Muiltiplicom, Agilent) were used for library preparation. TruSeq Custom Amplicon Low Input was used to construct the libraries using custom designed cardiomyopathy gene panel for gene coding regions (Illumina, San Diego, CA). Libraries were multiplexed and sequenced on HiSeq 550 Rapid Run mode for > 100X coverage (Illumina). Fastq sequencing data were analyzed using TruSeq Amplicon workflow Version 3.0.0 on BaseSpace platform (Illumina), including Isas (analysis software) Version 1.1.7.9.271 + TSAv3, SAMtools Version 1.2, Isas Smith –Waterman-Gotoh (Aligner) Version 6.2.1.25+ develop, Pisces Variant Caller Version 5.2.1.22, and Illumina Annotation Engine 1.5.3.82 against human genome GRCh37/hg19. Alignment was performed using the banded Smith-Waterman algorithm in the targeted gene regions. Primary Electrical Disorders (PED) MASTR Plus was used to construct the libraries for the arrhythmia gene panel in gene coding regions (Muiltiplicom, Agilent, Belgium). The libraries were multiplexed and sequenced on HiSeq 550 Rapid Run mode for > 100X coverage (Illumina). Fastq sequencing data were analyzed using Sentieon Workflow pipeline on BaseSpace

platform (Illumina), which uses the same mathematics used in the Broad Institute's BWA-GATK HaplotypeCaller Best Practice Workflow pipelines. The genomic VCF files and BED files from both cardiomyopathy and arrhythmia panels were used for genomic variants callings analyses on SVS software Version 8.0 (Golden Helix, Bozeman, MT) and Integrative Genomics Viewer Version 2.4 (IGV) (Broad Institute).

Variant Filtering and Curation

For curating new variants in the *MYH7* gene, we followed the rules and criteria set by the ClinGen expert panel [10]. For curating variants in other genes, we used the following criteria: PP1 (segregation): recommendations by Jarvik and Browning [11]. PP2 (missense variants are a common mechanism of disease): gnomAD missense constraint Z-score >3.09. PP3: REVEL score ≥ 0.7; if the REVEL score is between 0.6 and 0.7, and more than 8 out of 11 computational predictors support deleterious effect, it is also considered to be met. PM2_Supporting: 0.00002 (0.002%) minor allele frequency in gnomAD. PM3 (for variants in genes with potential ressesive phenotype): ClinGen Sequence Variant Interpretation Recommendation for in trans Criterion (PM3) - Version 1.0 (https://clinicalgenome.org/site/assets/files/3717/svi_proposal_for_pm3_criterion_-_version_1.pdf). PM5: if the missense change at the same codon as a likely pathogenic variant, PM5 is downgraded to PM5_Supporting. PM6 and PS2 (*de novo*): evidence evaluated based on the point system recommended by ClinGen Sequence Variant Interpretation Recommendation for de novo Criteria

(PS2/PM6) version 1.0 (https://clinicalgenome.org/site/assets/files/3461/svi_proposal_for_de_novo_criteria_v1_0.pdf). PS4: See supplemental table below. PVS1: followed recommendations of the ClinGen SVI [12]. Since family investigations were not performed in this study, only public data was used for assigning the significance. Variants interpreted as pathogenic and likely pathogenic were selected for further analysis (see Supplemental Table 3 for ACMG criteria).

The prevalence of the variant in affected individuals is significantly increased compared with the prevalence in controls

GnomAD Allele Count excludes Finish and Ashkenazi Jewish. The total number of probands include CVPath Registry data, and published literature where patient died of SCD, or which showed symptoms of arrhythmia or cardiomyopathy.

gnomAD Allele Count	Probands		
	PS4 Supp	PS4 Mod	PS4 Strong
0	1	2-3	4+
1	2	3-4	5+
2	3	4-5	6+
3	4	5-6	7+
4	5	6-7	8+

Variant validationand Allele Frequency

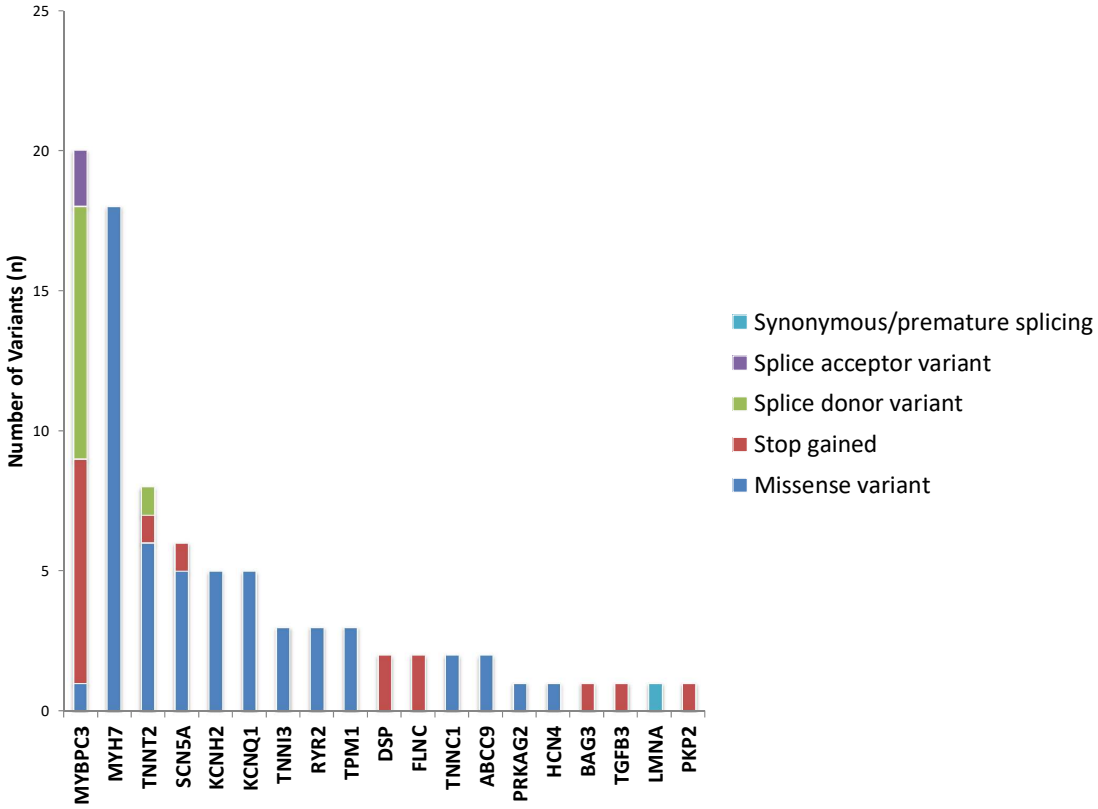
All P/LP variants with a read depth <15 were validated using Taqman genotyping assays (Assay IDs: C_319910867_10, C_319910867_10, C_319237470_10, C_319902197_10, C_321850666_10, C_319906518_10, C_326756379_10,

C_380520918_10, C__86250392_10, C_330741028_10, C_162861639_10, C_345611191_10, C_319954324_10, C_317492721_10, C_332370884_10, C_332290735_10, C_332379549_10, Hs00357608_m1, C_357804660_10, Hs00357608_m1, C_319910870_10, ANH6GP2, ANMF4VW, Thermo Fisher, Waltham, MA). Two additional P/LP variants were also validated in six samples using Taqman genotyping assays to confirm sequencing accuracy (Assay IDs: Hs00740082_CE, and Hs00794484_CE, Thermo Fisher, Waltham, MA). After variant classification, the MAF of benign variants, variants of unknown significance (VUS), and P/LP variants was compared with that of the general population using allele frequency data from TOPMED, ExAC, GnomAD, 1000G, and UK10K.

Detailed evaluation of myocardial fibrosis

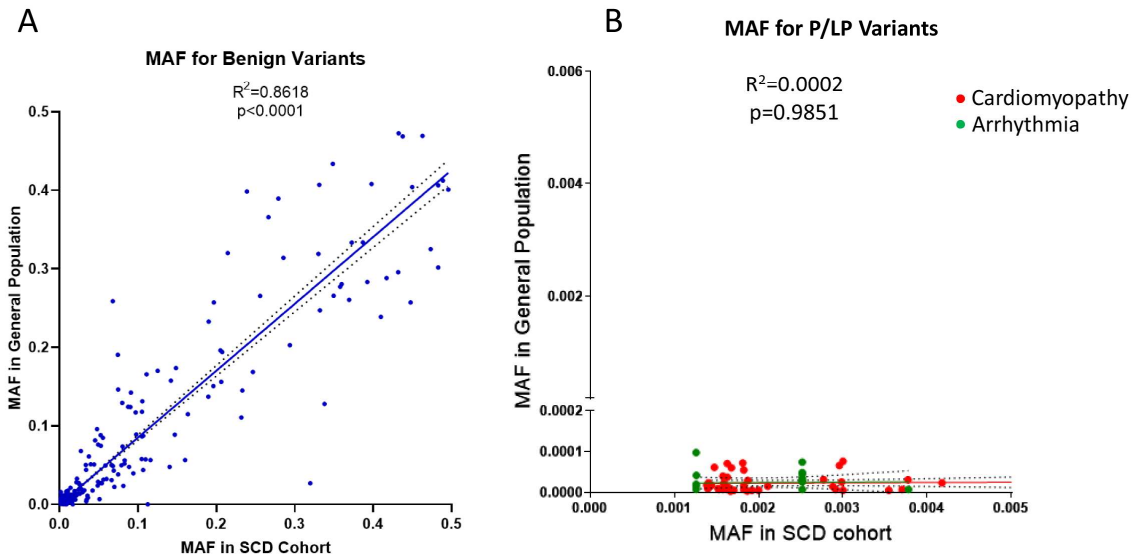
Four sections of anterior, posterior, and lateral left ventricle, ventricular septum from 21 most recent consecutive subjects with P/LP HCM gene variants and 22 most recent consecutive subjects without P/LP gene variants (median age; 36 vs. 34, respectively, p=0.8) were evaluated for evidence of myocardial fibrosis. Whole areas with myocardial fibrosis (which stains blue with Masson Trichrome stain) were measured using ZEN software (Zeiss, Oberkochen, Germany). As areas with perivascular fibrosis around the intramyocardial arteries vary widely between myocardial sections, perivascular fibrosis in $\geq$0.2mm intramyocardial arteries were separately measured and subtracted from areas with myocardial fibrosis, and percent area myocardial fibrosis was calculated.

eFigure 1. Types of P/LP Variants in Arrhythmia and Cardiomyopathy Genes in Individuals With Unexplained SCD



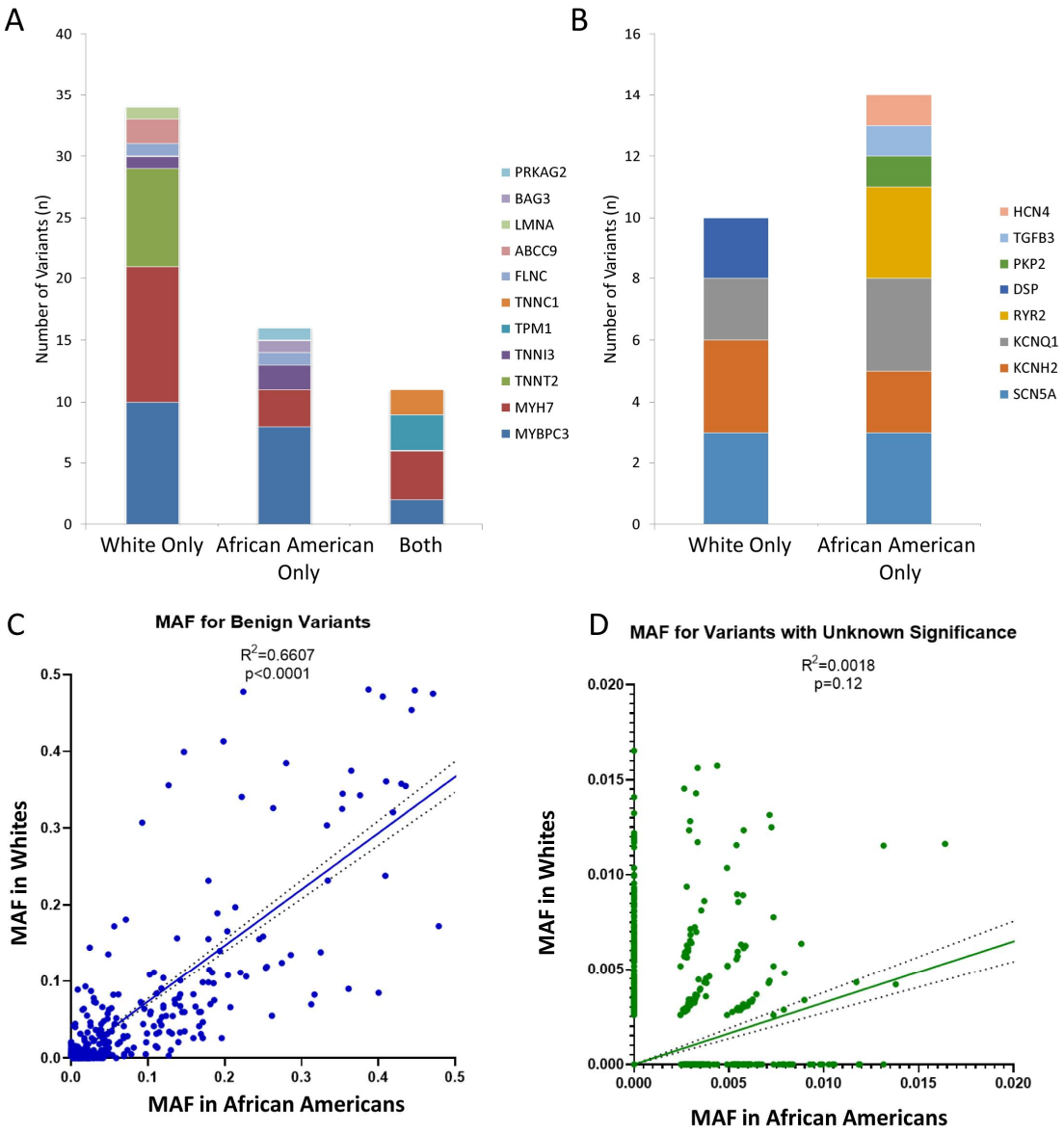
The numbers of each type of P/LP variants in each gene were categorized as missense variant, stop gained variant, synonymous/premature splicing variant, as well as splicing variant, including splicing donor and acceptor variants.

eFigure 2. Comparison of Allele Frequency in Cardiomyopathy and Arrhythmia Gene Variants in Individuals With Unexplained SCD vs the General Population



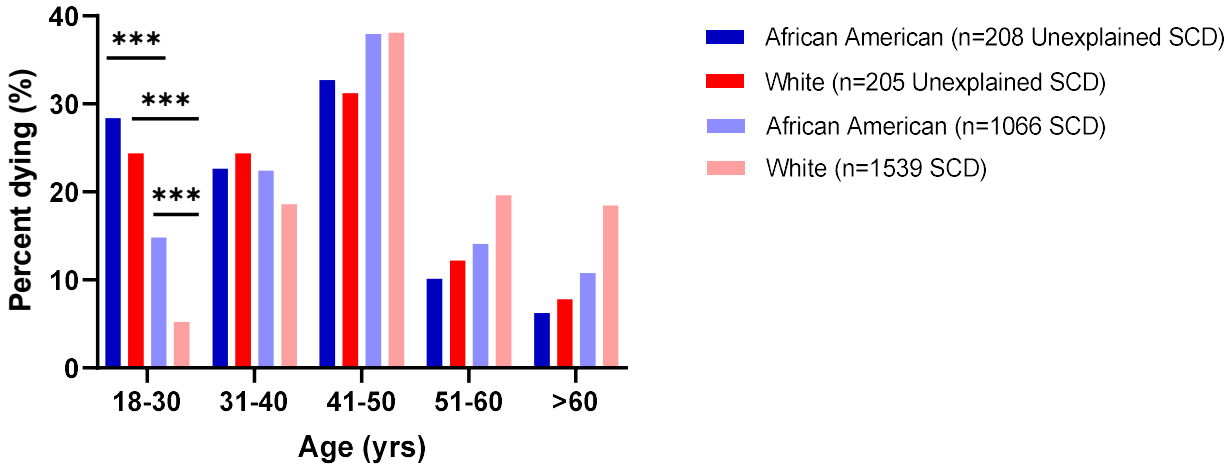
Comparison of minor allele frequency (MAF) for benign (A) and P/LP (B) variants between unexplained SCD cohort and the general population using Spearman's correlation. Note the MAF for benign variants was linear in nature (A) whereas the MAF for P/LP was much more common in our unexplained-SCD cohort versus the general population (B).

eFigure 3. Racial Differences in P/LP, Benign and Variants of Unknown Significance (VUS) in Arrhythmia and Cardiomyopathy Genes in Unexplained SCD



The distribution of numbers of P/LP variants per gene in African Americans only, Whites only, or in both, in cardiomyopathy genes (A) and arrythmia genes (B). **C**. The correlation of MAF for benign variants between African Americans and Whites was similar in subjects with explained SCD. **D**. The correlation of MAF in subjects with unexplained SCD for VUS was skewed towards African Americans.

eFigure 4. Racial Differences in Survival SCD Between African American and White Individuals



Age range at death for unexplained SCD (darker color) and autopsy evidenced SCD (lighter color) for both African Americans (blue) and Whites (red). *** p<0.001 by Fisher's Exact Test.

Supplemental Tables

eTable 1. List of Genes in the Cardiomyopathy Panel and Arrhythmia Panel

No	Genes	**Cardiac Conditions** (ClinGen Gene-Disease Validity Classification and Gene Curation Coalition by December 23, 2020)	Panel
1	*ABCC9*	DCM(*limited*), Cantú syndrome(*definitive*)	Cardiomyopathy Panel
2	*ACTC1*	HCM(*definitive*), DCM(*moderate*). LVNC, RCM	Cardiomyopathy Panel
3	*ACTN2*	HCM(*moderate*), DCM(*moderate*), LVNC(*moderate*)	Cardiomyopathy Panel
4	*ANKRD1*	HCM(*limited*), DCM(*limited*)	Cardiomyopathy Panel
5	*BAG3*	HCM, DCM(*definitive*), Myofibrillar myopathy(*definitive*), RCM	Cardiomyopathy Panel
6	*CAV3*	HCM(*limited*), DCM, LQTS	Cardiomyopathy Panel
7	*CSRP3*	HCM(*strong/moderate*), DCM(*limited*)	Cardiomyopathy Panel
8	*DES*	DCM(*definitive*), Myofibrillar myopathy(*definitive*), ARVD/C(*moderate*)	Cardiomyopathy Panel
9	*FHL1*	X-linked myopathy(*definitive*), DCM	Cardiomyopathy Panel
10	*FLNC*	HCM(*strong*), DCM(*definitive*), Myofibrillar myopathy(*definitive*), ARVD/C, RCM	Cardiomyopathy Panel
11	*LAMP2*	Danon disease (HCM, DCM) (*definitive, X-Linked*)	Cardiomyopathy Panel
12	*LMNA*	DCM(*definitive*), ARVD/C(*limited*)	Cardiomyopathy Panel
13	*LDB3*	HCM, DCM(*limited*), LVNC, ARVD/C(*disputed*)	Cardiomyopathy Panel
14	*MYBPC3*	HCM(*definitive*), DCM(*limited*), ARVD/C(*limited*), LVNC, RCM	Cardiomyopathy Panel
15	*MYH6*	HCM(*limited*), DCM(*limited*)	Cardiomyopathy Panel
16	*MYH7*	HCM(*definitive*), DCM(*definitive*),ARVD/C(*limited*), RCM	Cardiomyopathy Panel
17	*MYL2*	HCM(*definitive/strong*), DCM(*limited*)	Cardiomyopathy Panel
18	*MYL3*	HCM(*definitive/strong*), ARVD/C(*limited*), DCM(*disputed*), RCM	Cardiomyopathy Panel
19	*MYPN*	Nemaline myopathy(*definitive*), HCM(*limited*), DCM(*limited*)	Cardiomyopathy Panel
20	*NEXN*	HCM(*limited*), DCM(*moderate*)	Cardiomyopathy Panel
21	*PDLIM3*	HCM(*limited*), DCM(*disputed*)	Cardiomyopathy Panel
22	*PLN*	HCM(*definitive/strong*), DCM, ARVD/C	Cardiomyopathy Panel
23	*PRKAG2*	HCM(*definitive/strong*), Wolff-Parkinson-White syndrome	Cardiomyopathy Panel
24	*TCAP*	HCM(*limited*), DCM(*limited*)	Cardiomyopathy Panel
25	*TNNC1*	HCM(*moderate*), DCM(*definitive*)	Cardiomyopathy Panel
26	*TNNI3*	HCM(*definitive/strong*), DCM(*moderate*), RCM	Cardiomyopathy Panel
27	*TNNT2*	HCM(*definitive/strong*), DCM(*definitive*), LVNC, RCM	Cardiomyopathy Panel
28	*TPM1*	HCM(*definitive/strong*), DCM(*moderate*), RCM	Cardiomyopathy Panel

No	Genes	Cardiac Conditions (ClinGen Gene-Disease Validity Classification by 2020)	Panel
29	*TTR*	ATTR Amyloidosis (HCM)(*definitive*)	Cardiomyopathy Panel
30	*VCL*	HCM(*limited*), DCM(*moderate*), LVNC	Cardiomyopathy Panel
31	*AKAP9*	LQTS(*limited*)	Arrhythmia Panel
32	*ANK2*	LQTS, CPVT, BrS(*disputed*)	Arrhythmia Panel
33	*CACNA1C*	Timothy syndrome(*definitive*), LQTS(*moderate*), BrS(*disputed*)	Arrhythmia Panel
34	*CACNA2D1*	BrS(*disputed*)	Arrhythmia Panel
35	*CACNB2*	BrS(*disputed*)	Arrhythmia Panel
36	*CAV3*	LQTS, HCM(*limited*), DCM	Arrhythmia Panel
37	*CTNNA3*	ARVD/C(*limited*)	Arrhythmia Panel
38	*DES*	DCM(*definitive*), Myofibrillar myopathy(*definitive*), ARVD/C(*moderate*)	Arrhythmia Panel
39	*DSC2*	ARVD/C(*definitive*), DCM	Arrhythmia Panel
40	*DSG2*	ARVD/C(*definitive*), DCM(*limited*)	Arrhythmia Panel
41	*DSP*	ARVD/C(*definitive*), DCM(*strong*)	Arrhythmia Panel
42	*GPD1L*	BrS(disputed)	Arrhythmia Panel
43	*HCN4*	BrS(*disputed*), Familial thoracic aortic aneurysm and aortic dissection(*limited*), LVNC	Arrhythmia Panel
44	*JUP*	ARVD/C(*definitive*)	Arrhythmia Panel
45	*KCND3*	BrS(*disputed*)	Arrhythmia Panel
46	*KCNE1*	LQTS, Jervell and Lange-Nielsen syndrome(*strong/moderate*)	Arrhythmia Panel
47	*KCNE2*	LQTS	Arrhythmia Panel
48	*KCNE3*	BrS(*disputed*)	Arrhythmia Panel
49	*KCNE5*	BrS(*disputed*)	Arrhythmia Panel
50	*KCNH2*	LQTS(*strong*)	Arrhythmia Panel
51	*KCNJ5*	LQTS	Arrhythmia Panel
52	*KCNJ2*	SQTS	Arrhythmia Panel
53	*KCNJ8*	BrS(*disputed*)	Arrhythmia Panel
54	*KCNQ1*	LQTS(*strong*), SQTS, Jervell and Lange-Nielsen syndrome(*definitive/strong*)	Arrhythmia Panel
55	*NKX2-5*	Atrial heart septal defect(*definitive*), tetralogy of Fallot(*definitive*), Ventricular fibrillation, DCM(*limited*)	Arrhythmia Panel
56	*NOS1AP*	Ventricular fibrillation	Arrhythmia Panel
57	*PKP2*	ARVD/C(*definitive*), BrS(*disputed*)	Arrhythmia Panel
58	*RANGRF*	BrS(*disputed*)	Arrhythmia Panel
59	*RYR2*	ARVD/C(*refuted*), CPVT(*definitive*), LQTS, HCM(*limited*)	Arrhythmia Panel
60	*SCN1B*	BrS(*definitive*/*disputed*), epilepsy with febrile seizures (*definitive*)	Arrhythmia Panel
61	*SCN2B*	BrS(*disputed*)	Arrhythmia Panel
62	*SCN3B*	BrS(*disputed*)	Arrhythmia Panel

No	Genes	Cardiac Conditions (ClinGen Gene-Disease Validity Classification by 2020)	Panel
63	*SCN4B*	LQTS(*limited*)	Arrhythmia Panel
64	*SCN5A*	ARVD/C(*limited*), BrS(*definitive*), LQTS, DCM(*definitive*)	Arrhythmia Panel
65	*SLMAP*	BrS(*disputed*)	Arrhythmia Panel
66	*SNTA1*	LQTS(*disputed*)	Arrhythmia Panel
67	*TGFB3*	ARVD/C(*limited*), Loeys-Dietz syndrome(*definitive/strong*), Familial thoracic aortic aneurysm and aortic dissection(*limited*)	Arrhythmia Panel
68	*TMEM43*	ARVD/C(*definitive*)	Arrhythmia Panel
69	*TRDN*	LQTS(*definitive/strong*)	Arrhythmia Panel

CAV3 and DES genes are present in both Hypertrophic Cardiomyopathy; HCM. Dilated Cardiomyopathy; DCM. Restrictive Cardiomyopathy; RCM. Long QT Syndrome; LQTS. Brugada Syndrome; BrS. Catecholaminergic polymorphic ventricular tachycardia; CPVT. Left ventricular non-compaction syndrome; LVNC. Arrhythmogenic Right Ventricular Dysplasia/Cardiomyopathy; ARVD/C.

eTable 2. Patient Characteristics of Participants With Unexplained SCD (A) and HCM (B)

A. The patient characteristics in the total of 413 subjects with unexplained sudden cardiac death (SCD)

Patient Characteristics	Unexplained SCD n=413	
Age	41 (29-48)	
Sex (Female), n (%)	154 (37%)	
Race (African American), n (%)	208 (50%)	
Body / Heart Dimensions	Women n=154	Men n=259
Body height, cm	168 (163-173)	178 (173-183)
Body Weight, kg	79 (63-98)	83 (71-97)
Body Mass Index, kg/m^2	28.7 (23.3-34.9)	26.8 (22.6-30.0)
Body Surface Area, m^2	1.79 (1.60-1.99)	1.89 (1.73-2.05)
Heart Weight, g	350 (300-420)	420 (380-460)
LV diameter, mm	35 (30-40)	35 (30-40)
LV free wall thickness, mm	13 (11-14)	14 (12-15)
Septum thickness, mm	14 (12-15)	15 (14-16)
RV thickness, mm	4 (4-5)	5 (4-5)
Septum / Free wall ratio	1.1 (1.0-1.2)	1.1 (1.0-1.2)

B. The patient characteristics in the total of 49 subjects with hypertrophic cardiomyopathy

Patient Characteristics	HCM n=49		p value (Comparing to A.)	
Age	42 (30-50)		0.8	
Sex (Female), n (%)	6 (12%)		0.0004	
Race (African American), n (%)	28 (57%)		0.5	
Body / Heart Dimensions	Women n=6	Men n=43	P (women)	P (men)
Body height, cm	164 (159-171)	179 (174-185)	0.5	0.3
Body Weight, kg	86 (65-108)	93 (83-114)	0.5	0.0004
Body Mass Index, kg/m^2	31.5 (26.2-37.6)	30.2 (26.5-35.8)	0.4	0.0001
Body Surface Area, m^2	1.93 (1.70-2.14)	2.11 (1.97-2.37)	0.3	<0.0001
Heart Weight, g	653 (475-838)	600 (510-750)	0.0003	<0.0001
LV diameter, mm	33 (29-51)	38 (35-45)	0.7	0.001
LV free wall thickness, mm	17 (14-23)	17 (15-19)	0.0001	<0.0001
Septum thickness, mm	25 (19-35)	20 (19-25)	<0.0001	<0.0001
RV thickness, mm	6 (5-10)	6 (5-7)	0.0004	<0.0001
Septum / Free wall ratio	1.4 (1.1-1.9)	1.3 (1.1-1.5)	0.03	<0.0001

eTable 3. Participants With Pathogenic or Likely Pathogenic Variants

Case ID	Variant ID	Gene	cDNA variation	Amino acid changes	rs number	Age	Race	Gender	Circumstances of death	ACMG	ACMG Criteria Evidence	PMID
57	6	*MYBPC3*	c.505+1G>A		rs730880620	44	White	Female	witnessed collapse	P	PVS1, PM2_supporting, PS4_supporting	
67	6	*MYBPC3*	c.505+1G>A		rs730880620	41	White	Female	found dead at home	P	PVS1, PM2_supporting, PS4_supporting	
25	7	*MYBPC3*	c.1927+2T>C		rs869025467	46	White	Male	found dead at home	P	PVS1, PM2_supporting, PS1_supporting	
34	7	*MYBPC3*	c.1927+2T>C		rs869025467	20	Black	Male	witnessed collapse	P	PVS1, PM2_supporting, PS1_supporting	
70	12	*MYBPC3*	c.2905C>T	p.Q969*	rs397515992	40	White	Male	found dead at home	P	PS4, PM2_supporting, PVS1, PP1_strong	20359594, 21302287, 9541104
64	23	*MYBPC3*	c.3811C>T	p.R1271*	rs397516042	42	Black	Male	found dead at home	LP	PVS1_strong, PM2_supporting, PS4_supporting	18533079, 19574547, 23396983
42	24	*MYBPC3*	c.3627+1G>A		rs397516031	43	Black	Female	found dead at home	P	PVS1, PP1_strong, PS4, PM2_supporting	25351510, 19574547, 11499718

68	25	*MYBPC3*	c.3331-1G>A		rs727504305	37	Black	Female	witnessed collapse	P	PVS1, PM2_supporting, PS4_supporting	
47	26	*MYBPC3*	c.3293G>A	p.W1098*	rs397516013	50	White	Male	found dead outside	P	PM2_supporting, PS4, PVS1	20624503, 25031304, 23233322
22	27	*MYBPC3*	c.3253G>T	p.E1085*	rs397516010	28	Black	Female	found dead outside	P	PVS1, PM2_supporting, PS4_supporting	28492532, 27532257, 25611685
45	28	*MYBPC3*	c.3190+1G>A		rs111683277	41	Black	Female	found dead at home	P	PVS1, PS4_moderate, PM2_supporting	27532257, 25611685, 25132132
6	29	*MYBPC3*	c.2992C>T	p.Q998*	rs11570112	54	Black	Male	found dead in bed	P	PM2_supporting, PS4_supporting, PVS1	20624503
71	30	*MYBPC3*	c.1458G>A	p.W486*	rs1057517920	52	White	Female	found dead at home	P	PVS1, PM2_supporting, PS4_supporting	
40	31	*MYBPC3*	c.1351+1G>A		rs727503204	23	White	Male	witnessed collapse after exercise	P	PVS1, PS4_moderate, PM2_supporting, PP1	21750094, 19574547
66	32	*MYBPC3*	c.1273C>T	p.Q425*	rs397515895	36	White	Male	found dead at home	P	PVS1, PM2_supporting, PS4_supporting	11815426, 18803133
51	33	*MYBPC3*	c.1223+1G>A		rs730880639	18	White	Female	witnessed collapse	P	PVS1, PM2_supporting,	29497013

											PS1_supporting, PS4_supporting	
65	34	*MYBPC3*	c.1090+1G>A		rs727504269	39	Black	Male	witnessed collapse	P	PVS1, PM2_supporting, PS4_supporting, PS1_supporting	16858239, 26914223
61	35	*MYBPC3*	c.927-2A>G		rs397516082	24	White	Male	found dead at home	P	PVS1, PP1_strong, PM2_supporting, PS4_supporting	9562578, 22574137, 25078086
10	36	*MYBPC3*	c.292G>T	p.E98*	rs868819340	57	Black	Female	witnessed collapse	P	PM2_supporting, PS3, PVS1	19574547
35	71	*MYBPC3*	c.2458C>T	p.R820W	rs775404728	36	White	Male	found dead at home	LP	PP1_moderate, PM2_supporting, PM5, PS4	20378854
36	1	*MYH7*	c.2710C>T	p.R904C	rs727503253	46	White	Female	found dead in bed	LP	PP1_strong, PM2_supporting, PM5, PS4_supporting	20573160, 27532257, 29212898
38	1	*MYH7*	c.2710C>T	p.R904C	rs727503253	49	White	Male	found dead at home	LP	PP1_strong, PM2_supporting, PM5, PS4_supporting	20573160, 27532257, 29212898
75	1	*MYH7*	c.2710C>T	p.R904C	rs727503253	43	White	Male	found dead	LP	PP1_strong, PM2_supporting, PM5, PS4_supporting	20573160, 27532257, 29212898

39	4	*MYH7*	c.1207C>T	p.R403W	rs3218714	36	Black	Female	witnessed collapse	P	PP1_strong, PS4, PM2, PM5, PM1, PP3	1052196, 7662452, 7848420
46	4	*MYH7*	c.1207C>T	p.R403W	rs3218714	30	Black	Female	witnessed collapse	P	PP1_strong, PS4, PM2, PM5, PM1, PP3	1052196, 7662452, 7848420
12	8	*MYH7*	c.596C>T	p.A199V	rs727504283	38	Black	Female	witnessed collapse	LP	PP1_strong, PP3, PM1, PM2_supporting	27532257, 21310275, 23074333
59	8	*MYH7*	c.596C>T	p.A199V	rs727504283	20	White	Male	found dead in bed	LP	PP1_strong, PP3, PM1, PM2_supporting	27532257, 21310275, 23074333
4	9	*MYH7*	c.5740G>A	p.E1914K	rs397516254	38	White	Male	found dead in bed	LP	PM2, PM6, PP3, PS4_supporting	27532257, 24664454
21	9	*MYH7*	c.5740G>A	p.E1914K	rs397516254	43	White	Male	found dead at home	LP	PM2, PM6, PP3, PS4_supporting	27532257, 24664454
17	10	*MYH7*	c.4135G>A	p.A1379T	rs397516202	24	Black	Male	witnessed collapse	LP	PP1_Strong, PP3, PM2_supporting, PS4_moderate	28790153, 21310275, 12707239, 11861413
28	10	*MYH7*	c.4135G>A	p.A1379T	rs397516202	21	White	Female	found dead at home	LP	PP1_Strong, PP3, PM2_supporting, PS4_moderate	28790153, 21310275, 12707239, 11861413
33	11	*MYH7*	c.1816G>A	p.V606M	rs121913627	26	White	Male	found dead at home	P	PS3, PM2_supporting,	9826622, 9172070, 11377367

											PM1, PP3, PP1_strong, PS4	
56	11	*MYH7*	c.1816G>A	p.V606M	rs121913627	20	White	Female	found dead at home	P	PS3, PM2_supporting, PM1, PP3, PP1_strong, PS4	9826622, 9172070, 11377367
59	14	*MYH7*	c.2609G>A	p.R870H	rs36211715	20	White	Male	found dead in bed	P	PP1_strong, PM1, PM2_supporting, PS4	
50	37	*MYH7*	c.2334C>G	p.D778E	rs2069544	55	White	Male	witnessed collapse	LP	PM5, PM2_supporting, PP2, PP3, PS1_supporting, PS4_supporting, PP1	12707239, 12566107, 11748309
68	38	*MYH7*	c.741C>A	p.F247L	14:23900682	37	Black	Female	witnessed collapse	P	PS1, PM2_supporting, PM1, PP3, PS4_moderate	19150014, 22765922
69	39	*MYH7*	c.428G>A	p.R143Q	rs397516209	68	White	Male	found dead at home	LP	PS4, PM2, PP1	
35	48	*MYH7*	c.3133C>T	p.R1045C	rs45611033	36	White	Male	found dead at home	LP	PP3, PM2_supporting, PS4	
14	13	*TNNT2*	c.322G>A	p.E108K	rs869312881	47	White	Male	witnessed collapse	P	PP1, PP3, PM1, PM2, PS3, PS4_moderate	20083571

48	15	*TNNT2*	c.888G>A	p.W296*	rs730881116	25	White	Male	found dead at home	LP	PM1, PM2_supporting, PS4, PVS1_Moderate	9060892, 12707239, 228587948
58	16	*TNNT2*	c.848+1G>A		rs111377893	30	White	Male	found dead outside	P	PM1, PM2_supporting, PS3, PS4_Supporting, PVS1_Moderate	9060892, 21245263
36	17	*TNNT2*	c.457C>T	p.R151W	rs74315379	46	White	Female	found dead in bed	P	PP1_strong, PP3, PM1, PM6, PS3, PS4	14654368, 15623536, 12923187
18	18	*TNNT2*	c.364T>A	p.F122I	rs121964858	48	White	Female	found dead at home	P	PP1_strong, PP3, PM2_supporting, PM1, PS3, PS4	10965086, 10617660, 12409295
60	19	*TNNT2*	c.317G>A	p.R106H	rs397516457	52	White	Female	found dead outside	LP	PP1, PP3, PM2_supporting, PM5, PS4	20800588, 20031602, 20624503, 26507537
69	20	*TNNT2*	c.311G>T	p.R104L	rs121964856	68	White	Male	found dead at home	LP	PP3, PM2_supporting, PM5, PS4_Moderate	11560853
55	21	*TNNT2*	c.280G>A	p.G94R	rs727504255	25	White	Female	found dead at home	LP	PP1, PP3, PM1, PM2_supporting, PS4_Moderate	18809796, 19996403

9	51	*SCN5A*	c.4868G>A	p.R1623Q	rs137854600	78	Black	Female	MVA without any clear CoD	LP	PP3, PM2_supporting, PM5_supporting, PS4	15840476, 19716085, 9506831
20	54	*SCN5A*	c.481G>A	p.E161K	rs199473062	46	White	Male	found dead outside	P	PP1_moderate, PP3, PM2_supporting, PM5_supporting, PM6, PS3	15910881, 20448214
26	55	*SCN5A*	c.4883G>A	p.R1628Q	rs199473623	44	Black	Male	found dead outside	LP	PP3, PM1, PM2_supporting, PS3	22581653, 20129283, 24167619
41	62	*SCN5A*	c.2204C>T	p.A735V	rs137854611	48	White	Male	found dead at home	LP	PP3, PM1, PM2_supporting, PM5_supporting, PS3	22795782, 20129283, 11823453
52	65	*SCN5A*	c.1603C>T	p.R535*	rs1417036453	24	White	Male	found dead at home	P	PM2_supporting, PS3, PS4, PVS1	25757662, 20129283, 15890323
72	69	*SCN5A*	c.4859C>T	p.T1620M	rs199473282	58	Black	Male	MVA without any clear CoD	LP	PP2, PP3, PM1, PM2_supporting, PS4_Supporting	
5	47	*KCNH2*	c.1886A>G	p.N629S	rs199472957	57	White	Male	found dead outside	P	PP2, PP3, PM2_supporting, PM5, PS3, PS4	22581653, 19841300, 19716085, 9544837

7	49	*KCNH2*	c.2464G>A	p.V822M	rs121912506	45	Black	Female	found dead at home	P	PS3, PS4_supporting, PM2_supporting, PM1, PP2, PP3	23303164, 10086971, 8914737
13	52	*KCNH2*	c.2467C>T	p.R823W	rs199473538	58	Black	Male	found dead at home	P	PP2, PP3, PM2_supporting, PS3, PS4	23631430, 21440677, 19716085
30	58	*KCNH2*	c.1847A>G	p.Y616C	rs199472946	46	White	Male	found dead at home	LP	PP2, PP3, PM1, PM2_supporting, PS4_Moderate	22581653, 19716085
53	66	*KCNH2*	c.1898A>G	p.N633S	rs199472961	26	White	Male	witnessed collapse	P	PP1_strong, PP2, PP3, PM2_supporting, PS3, PS4	16842670, 9544837, 22949429, 17088455
19	53	*KCNQ1*	c.686G>A	p.G229D	rs199472708	34	White	Male	witnessed collapse	LP	PP3, PM2_supporting, PS3, PS4_moderate	24096004, 30967788
27	56	*KCNQ1*	c.395G>A	p.R132H	rs199472720	41	White	Male	found dead at home	LP	PP3, PM2_supporting, PM5, PS3	
44	64	*KCNQ1*	c.914G>C	p.W305S	rs120074186	37	Black	Female	witnessed collapse	P	PP3, PM2_supporting, PM3_supporting, PM5_supporting, PS3, PS4	9312006

54	67	*KCNQ1*	c.773A>G	p.H258R	rs199472718	24	Black	Female	witnessed collapse	LP	PP3, PM1, PM2_supporting, PS3	19913547
63	68	*KCNQ1*	c.425G>A	p.G142D	rs120074194	35	Black	Male	found dead at home	P	PP1_moderate, PP3, PM2_supporting, PM5, PS3	
11	3	*RYR2*	c.7159G>A	p.A2387T	rs794728753	36	Black	Male	found dead at home	LP	PP2, PP3, PM2_supporting, PM5, PS4	16188589, 19398665
24	3	*RYR2*	c.7159G>A	p.A2387T	rs794728753	40	Black	Male	found dead at home	LP	PP2, PP3, PM2_supporting, PM5, PS4	16188589, 19398665
37	61	*RYR2*	c.1259G>A	p.R420Q	rs794728721	38	Black	Male	found dead	LP	PP3, PM1, PM2_supporting, PS3	27452199, 28422759, 26153920
15	41	*TNNI3*	c.607G>A	p.G203S	rs267607127	39	White	Female	found dead outside	LP	PM2_supporting, PM1, PP3, PS4_supporting	9241277
54	42	*TNNI3*	c.544G>A	p.E182K	rs397516355	24	Black	Female	witnessed collapse	LP	PM2_supporting, PM1, PS4_moderate, PM6_supporting	22464770, 24503780
63	43	*TNNI3*	c.470C>T	p.A157V	rs397516353	35	Black	Male	found dead at home	P	PM2_supporting, PM1, PP1_strong, PP3, PS4	12707239, 15607392, 16335287, 19645627

16	2	*TPM1*	c.605G>A	p.R202H	rs199476311	42	White	Male	found dead	LP	PP2, PP3, PM2_supporting, PS2, PS4_Supporting	20530761
23	2	*TPM1*	c.605G>A	p.R202H	rs199476311	28	Black	Male	found dead at home	LP	PP2, PP3, PM2_supporting, PS2, PS4_Supporting	20530761
62	2	*TPM1*	c.605G>A	p.R202H	rs199476311	25	Black	Female	witnessed collapse	LP	PP2, PP3, PM2_supporting, PS2, PS4_Supporting	20530761
2	45	*ABCC9*	c.3460C>T	p.R1154W	rs387907208	34	White	Male	undetermined	LP	PP2, PP3, PM2_supporting, PM5_supporting, PS4_strong	22608503, 23307537, 22610116
3	46	*ABCC9*	c.4537G>A	p.A1513T	rs121909304	44	White	Female	found dead in bed	LP	PP2, PP3, PM2_supporting, PS3, PS4_supporting	15034580
29	57	*DSP*	c.3337C>T	p.R1113X	rs746877365	19	White	Female	found dead in bed	P	PP1_strong, PM2_supporting, PS4, PVS1	19095136, 19279339
32	60	*DSP*	c.699G>A	p.W233X	rs397516955	27	White	Female	witnessed collapse	P	PM2_supporting, PS4_supporting, PVS1	16917092

74	40	*FLNC*	c.697C>T	p.Q233*	rs1554397464	44	Black	Male	MVA without any clear CoD	P	PM2_supporting, PVS1, PS4_supporing	27908349
49	70	*FLNC*	c.6976C>T	p.R2326*	rs748416758	28	White	Male	witnessed collapse	P	PM2_supporting, PVS1, PS4_supporting, PP1_supporting	27908349
1	5	*TNNC1*	c.251G>A	p.C84Y	rs267607126	50	Black	Female	found dead in bed	LP	PP3, PM2_supporting, PS3, PS4_moderate	18572189
15	5	*TNNC1*	c.251G>A	p.C84Y	rs267607126	39	White	Female	found dead outside	LP	PP3, PM2_supporting, PS3, PS4_moderate	18572189
73	44	*BAG3*	c.262C>T	p.Q88*	rs1554877001	20	Black	Female	found dead in bed	LP	PM1, PM2_supporting, PVS1_Strong	21353195
8	50	*HCN4*	c.1444G>A	p.G482R	rs794727637	55	Black	Female	found dead in bed	LP	PS1_moderate, PM2_supporting, PM1, PP2, PP3	25145518, 25145517
76	72	*LMNA*	c.768G>A	p.Val256=	rs794728593	44	White	Male	witnessed collapse	LP	PP1_strong, PM2_supporting, PS4_supporting, PM4	28679633
31	59	*PKP2*	c.1138G>T	p.E380X	rs878898365	25	Black	Male	witnessed collapse	P	PM2_supporting, PS4_moderate, PVS1	

23	22	*PRKAG2*	c.1463A>T	p.N488I	rs121908989	28	Black	Male	found dead at home	LP	PP1, PP3, PM1, PM2_supporting, PS3	11827995
43	63	*TGFB3*	c.989G>A	p.W330X	rs1555360222	42	Black	Male	found dead	LP	PM2_supporting, PS4_supporting, PVS1_strong	

MVA- motor vehicle accident; CoD- cause of death; P-Pathogenic; LP-Likely Pathogenic; *- termination or stop

eTable 4. Patient Characteristics and Percentage Area Myocardial Fibrosis in Unexplained Sudden Cardiac Death cases with P/LP in HCM Genes and Detailed Septal Evaluations

Patient characteristics	P/LP in HCM gene (+) N=21	P/LP in HCM gene (-) N=22	P value
Age	39 (25-47)	31 (22-44)	0.2
Race (African American), n (%)	8 (38%)	13 (59%)	0.2
Sex (Female), n (%)	14 (67%)	9 (41%)	0.1
Body height, cm	175 (165-179)	178 (168-184)	0.3
Body Weight, kg	91 (70-114)	86 (81-104)	0.8
Body Mass Index, kg/m^2	29.7 (23.5-36.7)	27.9 (25.9-34.0)	0.9
Body Surface Area, m^2	1.99 (1.70-2.20)	1.91 (1.82-2.07)	0.9
Heart Weight, g	380 (308-456)	429 (358-461)	0.4
LV diameter, mm	35 (28-40)	33 (30-36)	0.7
LV free wall thickness, mm	13 (11-15)	13 (11-15)	0.9
Septum thickness, mm	14 (12-15)	15 (13-16)	0.4
RV thickness, mm	4 (4-5)	5 (4-5)	0.4
Septum / Free wall ratio	1.1 (1.0-1.1)	1.1 (1.1-1.2)	0.6
Results			
Percent area fibrosis in myocardium, (%) (mean value from 4 locations)	1.02 (0.76-1.55)	0.93 (0.70-1.40)	0.5

HCM= hypertrophic cardiomyopathy, P/LP=pathogenic / likely pathogenic, SCD=sudden cardiac death, LV=left ventricle

eTable 5. Patient Characteristics in African American and White Individuals With and Without P/LP Gene Variants

Patient Characteristics	P/LP variant (+) n=76		P/LP variant (-) n=337		p value	
Age	39.0 (28.0-48.0)		41.2 (31.0-48.0)		0.22	
Sex (Female), n (%)	29 (38.2%)		125 (37.1%)		0.52	
Race (African American), n (%)	32 (42.1%)		176 (52.2%)		0.47	
Body / Heart Dimensions	African American n=32	Whites n=44	African American n=176	Whites n=161	P (AA)	P (C)
Body height, cm	170.2 (165.1-177.8)	174.0 (169.5-180.3)	172.7 (165.1-180.3)	175.3 (167.2-180.3)	0.04*	0.94
Body Weight, kg	76.0 (63.5-108.4)	81.0 (72.6-93.9)	82.6 (69.0-95.3)	82.8 (70.8-96.7)	0.54	0.73
Body Mass Index, kg/m^2	26.3 (22.0-33.5)	26.7 (23.0-29.5)	27.3.0 (22.4-31.0)	27.1 (22.6-30.9)	0.78	0.57
Body Surface Area, m^2	1.78 (1.58-2.00)	1.86 (1.70-1.99)	1.83 (1.69-2.03)	1.87 (1.70-2.05)	0.32	0.77
Heart Weight, g	390.0 (320.0-450.0)	395.0 (348.0-452.5)	410.0 (340.0-450.0)	410.0 (350.0-451.3)	0.18	0.20
LV diameter, mm	35.0 (30.0-40.0)	30.0 (20.0-40.0)	35.0 (30.0-40.0)	35.0 (30.0-40.0)	0.25	<0.01**
LV free wall thickness, mm	13.0 (12.0-15.0)	14.0 (12.0-15.0)	14.0 (12.0-15.0)	13.0 (12.0-15.0)	0.70	0.06
Septum thickness, mm	14.0 (12.0-16.0)	14.0 (10.8-15.0)	15.0 (13.0-16.0)	15.0 (13.0-16.0)	<0.01**	<0.01**
RV thickness, mm	4.0 (4.0-5.0)	4.0 (4.0-5.0)	4.0 (4.0-5.0)	5.0 (4.0-5.0)	0.53	0.60
Septum / Free wall ratio	1.08 (1.02-1.18)	1.08 (1.03-1.17)	1.08 (1.00-1.17)	1.08 (1.00-1.15)	0.88	0.11

AA=African American; W= Whites; P/LP=pathogenic / likely pathogenic, HCM= hypertrophic cardiomyopathy, SCD=sudden cardiac death, MAF= minor allele frequency, LV=left ventricle, RV=right ventricle, BW=body weight. Data are shown at median (IQR) unless indicated. T test results were shown as p-vaule, * p<0.05, **p<0.01, ***p<0.001

eTable 6. Variants With Unknown Significance in African American Individuals Only, With Suspicion for a Pathogenic Role in Unexplained SCD

Chr	Position	Gene	Identifier	Ref	Alt	MAF in CVPath Cohort	Carrier	Ontology	HGVS p.	Global MAF	Enrichment Fold
14	23886493	MYH7	rs890401818	G	A	0.0083	4	missense variant	p.Ser1463Leu	0	-
17	41758792	JUP	rs782309611	G	A	0.0060	3	missense variant	p.Arg526Cys	0.0000187	320

SCD=sudden cardiac death, MAF= minor allele frequency

eTable 7. Allele Frequencies of P/LP Variants in Unexplained SCD Cohort and in gnomAD

Gene	Number of P/LP variants	Variants	Study unexplained SCD Cohort		gnomAD Data	
			Whites (n=205)	African Americans (n=208)	Europeans (non-Finnish) (n=129,166)	Africans/African Americans (n=24,966)
MYBPC3	18	c.505+1G>A, c.1927+2T>C, c.2905C>T p.Q969*, c.3811C>T p.R1271*, c.3627+1G>A, c.3331-1G>A, c.3293G>A p.W1098*, c.3253G>T p.E1085*, c.3190+1G>A, c.2992C>T p.Q998*, c.1458G>A p.W486*, c.1351+1G>A, c.1273C>T p.Q425*, c.1223+1G>A, c.1090+1G>A, c.927-2A>G, c.292G>T p.E98*, c.2458C>T p.R820W	0.0536(11)	0.0432(9)	0.00008(10)	0.00016(4)
MYH7	10	c.2710C>T p.R904C, c.1207C>T p.R403W, c.596C>T p.A199V, c.5740G>A p.E1914K, c.4135G>A p.A1379T, c.1816G>A p.V606M, c.2334C>G p.D778E, c.741C>A p.F247L, c.428G>A p.R143Q, c.3133C>T p.R1045C	0.0634(13)	0.0288 (6)	0.00006(8)	0.00040(10)
TNNT2	7	c.322G>A p.E108K, c.888G>A p.W296*, c.848+1G>A , c.457C>T p.R151W, c.364T>A p.F122I, c.317G>A p.R106H, c.311G>T p.R104L, c.280G>A p.G94R	0.0390(8)	0.0(0)	0.000008(1)	0.0(0)
SCN5A	6	c.4868G>A p.R1623Q, c.481G>A p.E161K, c.4883G>A p.R1628Q, c.2204C>T p.A735V, c.1603C>T p.R535*, c.4859C>T p.T1620M	0.0146(3)	0.0144(3)	0.000008(1)	0.0(0)

KCNH2	5	c.1886A>G p.N629S, c.2464G>A p.V822M, c.2467C>T p.R823W, c.1847A>G p.Y616C, c.1898A>G p.N633S	0.0146(3)	0.0096(2)	0.000016(2)	0.00016(4)
KCNQ1	5	c.686G>A p.G229D, c.395G>A p.R132H, c.914G>C p.W305S, c.773A>G p.H258R, c.425G>A p.G142D	0.0098(2)	0.0144(3)	0.0(0)	0.0(0)
TNNI3	3	c.607G>A p.G203S, c.544G>A p.E182K, c.470C>T p.A157V	0.0049(1)	0.0096(2)	0.0(0)	0.0(0)
DSP	2	c.3337C>T p.R1113X, c.699G>A p.W233X	0.0098(2)	0.0(0)	0.000008(1)	0.0(0)
RYR2	2	c.7159G>A p.A2387T, c.1259G>A p.R420Q	0.0(0)	0.0144(3)	0.0(0)	0.0(0)
FLNC	2	c.697C>T p.Q233*, c.6976C>T p.R2326*	0.0049(1)	0.0048(1)	0.0(0)	0.0(0)
ABCC9	2	c.3460C>T p.R1154W, c.4537G>A p.A1513T	0.0098(2)	0.0(0)	0.0(0)	0.0(0)
TPM1	1	c.605G>A p.R202H	0.0049(1)	0.0096(2)	0.0(0)	0.0(0)
BAG3	1	c.262C>T p.Q88*	0.0(0)	0.0048(1)	0.0(0)	0.0(0)
TNNC1	1	c.251G>A p.C84Y	0.0049(1)	0.0048(1)	0.0(0)	0.0(0)
LMNA	1	c.768G>A p.Val256=	0.0049(1)	0.0(0)	0.0(0)	0.0(0)
HCN4	1	c.1444G>A p.G482R	0.0(0)	0.0048(1)	0.0(0)	0.0(0)
TGFB3	1	c.989G>A p.W330X	0.0(0)	0.0048(1)	0.0(0)	0.0(0)
PKP2	1	c.1138G>T p.E380X	0.0(0)	0.0048(1)	0.0(0)	0.0(0)
PRKAG2	1	c.1463A>T p.N488I	0.0(0)	0.0048(1)	0.0(0)	0.0(0)

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