Combined Cardiac Panel

**Panel Gene List:** ABCC9, ACTC1, ACTN2, AKAP9, ALMS1, ALPK3, ANK2, ANKRD1, BAG3, BRAF, CACNA1C, CACNA2D1, CACNB2, CALM1, CALM2, CALM3, CASQ2, CAV3, CHRM2, CRYAB, CSRP3, DES, DMD, DOLK, DSC2, DSG2, DSP, DTNA, EMD, FHL1, FKRP, FKTN, GATA1, GLA, GPD1L, HCN4, HRAS, ILK, JPH2, JUP, KCND3, KCNE1, KCNE2, KCNE3, KCN14L (KCNE5), KCNH2 (HERG), KCNJ2, KCNJ5, KCNJ8, KCNQ1, KRAS, LAMA4, LAMP2, LDB3, LMNA, MAP2K1, MAP2K2, MIB1, MTND1, MTND5, MTND6, MTTD, MTG, MTTH, MTV, MTTK, MTTI, MTTL1, MTTL2, MTM1, MTM2, MTS1, MTS2, MURC, MYBPC3, MYH6, MYH7, MYL2, MYL3, MYLK2, MYOZ2, NEBL, NEXN, NXX2-5, NRAS, PDLIM3, PKP2, PLN, PRDM16, PRKAG2, PTPN11, RAF1, RANGRF, RBM20, RIT1, RYR2, SCN10A, SCN1B, SCN2B, SCN3B, SCN4B, SCN5A, SGCD, SNTA1, SOS1, TAZ, TCAP, TGFB3, TMEM43, TMPO, TNNC1, TNNT1, TNNT2, TPM1, TRDN, TRPM4, TTN, TTR, TXNRD2, VCL

Additional genes from our cardiology test menu may be added to this panel by selecting test code 935C.

**Clinical Features:**

Cardiac arrhythmias occur due to disruption of the heart’s natural rhythm and cardiomyopathy is defined as disease of the heart muscle. In some individuals or families, the clinical picture may be complex or features of both these conditions may be present, in which case a broader approach to genetic testing may be helpful. Both phenotypes are genetically heterogeneous and have many different clinical presentations.

Several risk factors can predispose an individual to develop an arrhythmia, including genetic disorders, trauma, electrolyte imbalance and structural abnormalities of the heart. There are several different genetic arrhythmia disorders. **Arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVC)** affects the cardiac desmosome, which is a protein complex that maintains cell-to-cell connections and provides mechanical attachments between adjacent cells. ARVC is characterized by myocyte death and replacement by fat and fibrous tissue in the right ventricle, predisposing to ventricular tachyarrhythmia and sudden cardiac death.1,2 **Brugada syndrome (BrS)** is caused by abnormal ion channel function and is characterized by ST segment elevation on ECG (leads V1-3) in the absence of structural heart disease. BrS is associated with increased risk for syncope, entricular tachyarrhythmia and sudden cardiac death.3-5 **Catecholaminergic polymorphic ventricular tachycardia (CPVT)** is characterized by cardiac calcium channel dysfunction that is precipitated by stress-induced release of catecholamines.6-8 **Long QT syndrome (LQTS)** is characterized by prolongation of the QT interval on ECG and is associated with increased risk for syncope, ventricular arrhythmia, and sudden cardiac death in individuals with normal heart structure.9-11 Genetic predisposition to a cardiac arrhythmia may occur as an isolated feature or may be part of a multisystemic disorder, such as Jervell and Lange-Nielsen syndrome, Timothy syndrome, Andersen-Tawil syndrome, Naxos disease, Carvajal syndrome and muscular dystrophy.1,12-15
Several risk factors may also contribute towards development of a cardiomyopathy, including genetic disorders, hypertension, ischemic heart disease and infection. There are several different genetic cardiomyopathies. **Hypertrophic cardiomyopathy (HCM)** is characterized by myocardial hypertrophy and myocyte disarray in the absence of other cardiac or systemic causes.\(^{16-18}\) **Dilated cardiomyopathy (DCM)** usually presents with one or more of the following: i) heart failure with symptoms of congestion (edema, orthopnea or paroxysmal dyspnea), ii) reduced cardiac output resulting in fatigue or dyspnea on exertion, arrhythmias and/or conduction system disease and iii) thromboembolic disease or stroke, mainly from left ventricular mural thrombus. However, some individuals with a DCM pathogenic variant may also be asymptomatic.\(^{15,19}\) **Left ventricular non-compaction (LVNC)** is characterized by abnormal trabeculations in the left ventricle, most frequently at the apex, and can share the same clinical presentation as DCM, ranging from asymptomatic disease to progressive deterioration of cardiac function, arrhythmias, thromboembolic events, or sudden cardiac death.\(^{19,20}\) **Noonan syndrome (NS)** is a relatively common multi-system disorder that may include HCM, facial dysmorphism, congenital heart defects, short stature, skeletal malformations, motor delay, learning disabilities, and impaired blood clotting ability.\(^{21}\) Cardiomyopathy can also be a presenting feature of other inherited disorders, such as Danon disease, Fabry disease, mitochondrial myopathy, or muscular dystrophy.\(^{15-19}\)

**Inheritance Pattern/Genetics:** Autosomal Dominant, Autosomal Recessive, X-linked, or Mitochondrial

**Test Methods:**

Using genomic DNA extracted from the submitted specimen, the coding regions and splice junctions of the 120 genes (Only exons 1-44 for **CACNA1C**, only the **KCNQ1**-binding domains including Ser1570 residue for **AKAP9**, excluding exon 6 of the **PKP2** gene and the following genomic regions of the **TTN** gene: chr2:179527692-179527782,179523898-179523982, 179523731-179523815) are enriched using a proprietary targeted capture system developed by GeneDx for next-generation sequencing with CNV calling (NGS-CNV). The enriched targets are simultaneously sequenced with paired-end reads on an Illumina platform. Bi-directional sequence reads are assembled and aligned to reference sequences based on NCBI RefSeq transcripts and human genome build GRCh37/UCSC hg19. After gene specific filtering, data are analyzed to identify sequence variants and most deletions and duplications involving coding exons; however, technical limitations and inherent sequence properties effectively reduce this resolution for some genes. Alternative sequencing or copy number detection methods are used to analyze or confirm regions with inadequate sequence or copy number data by NGS. Reportable variants include pathogenic variants, likely pathogenic
variants and variants of uncertain significance. Likely benign and benign variants, if present, are not routinely reported but are available upon request.

**Test Sensitivity:**
The technical sensitivity of sequencing is estimated to be >99% at detecting single nucleotide events. It will not reliably detect deletions greater than 20 base pairs, insertions or rearrangements greater than 10 base pairs, or low-level mosaicism. The copy number assessment methods used with this test cannot reliably detect copy number variants of less than 500 base pairs or mosaicism and cannot identify balanced chromosome aberrations. Assessment of exon-level copy number events is dependent on the inherent sequence properties of the targeted regions, including shared homology and exon size.
<table>
<thead>
<tr>
<th>Gene</th>
<th>Protein</th>
<th>Inheritance</th>
<th>Disease Association(s)</th>
</tr>
</thead>
<tbody>
<tr>
<td>EMD</td>
<td>EMERIN</td>
<td>XL</td>
<td>EMD</td>
</tr>
<tr>
<td>FHL1</td>
<td>FOUR-AND-A-HALF LIM DOMAINS 1</td>
<td>XL</td>
<td>HCM, LVH, EMD, skeletal muscle, muscle hypertrophy, Myofibrillar myopathy</td>
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<td>FUKUTIN RELATED PROTEIN</td>
<td>AR</td>
<td>DCM, muscular dystrophy</td>
</tr>
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<td>FUKUTIN</td>
<td>AR</td>
<td>DCM, LGMD, Fukuyama Congenital Muscular Dystrophy</td>
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<td>GATA ZINC FINGER DOMAIN-CONTAINING PROTEIN 1</td>
<td>AR</td>
<td>DCM</td>
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<td>GALACTOSIDASE, ALPHA</td>
<td>XL</td>
<td>Fabry disease</td>
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<td>GLYCEROL-3-PHOSPHATE DEHYDROGENASE 1-LIKE</td>
<td>AD</td>
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<td>BrS, SSS</td>
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<td>V-HA-RAS HARVEY RAT SARCOMA VIRAL ONCOGENE HOMOLOG</td>
<td>AD</td>
<td>Costello syndrome</td>
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<td>ILK</td>
<td>INTEGRIN-LINKED KINASE</td>
<td>AD</td>
<td>DCM</td>
</tr>
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<td>JUNCTOCHILIN 2</td>
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<td>HCM</td>
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<td>Andersen-Tawil syndrome, SQTS</td>
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<td>Noonan/CFC/Costello</td>
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<td>LAMININ, ALPHA-4</td>
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<td>XL</td>
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<td>LIM DOMAIN-BINDING 3</td>
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<td>Noonan/CFC/Costello</td>
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<td>HCM</td>
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<td>MYOSIN LIGHT CHAIN KINASE 2</td>
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</tr>
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<td>MYOZ2</td>
<td>MYOZENIN 2</td>
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<tr>
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<td>Noonan/CFC/Costello</td>
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<td>PDLIM3</td>
<td>PDZ AND LIM DOMAIN PROTEIN 3</td>
<td>AD</td>
<td>HCM, DCM</td>
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<tr>
<td>PKP2</td>
<td>PLAKOPHILIN 2</td>
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<td>ARVC, DCM, BrS</td>
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<tr>
<td>PLN</td>
<td>PHOSPHOLAMAN</td>
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<td>DCM, HCM, ARVC</td>
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<td>HCM, Wolff-Parkinson-White syndrome</td>
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<td>Noonan/CFC/Costello</td>
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<tr>
<td>RAF1</td>
<td>V-RAF-1 MURINE LEUKEMIA VIRAL ONCOGENE HOMOLOG 1</td>
<td>AD</td>
<td>Noonan/CFC/Costello</td>
</tr>
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<td>RNA-BINDING MOTIF PROTEIN 20</td>
<td>AD</td>
<td>DCM</td>
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<td>RIT1</td>
<td>RAS-LIKE WITHOUT CAAX 1</td>
<td>AD</td>
<td>Noonan</td>
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<td>RANGRF</td>
<td>RAN GUANINE NUCLEOTIDE RELEASE FACTOR</td>
<td>AD</td>
<td>BrS</td>
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<td>BrS</td>
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<td>SCN1B</td>
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<td>LQTS</td>
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<td>AD, AR</td>
<td>BrS, DCM, Heart block, LQTS, SSS, SIDS</td>
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<td>SGCDC</td>
<td>SARCOCYLAN, DELTA</td>
<td>AD, AR</td>
<td>DCM, LGMD</td>
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<tr>
<td>SNTA1</td>
<td>ALPHA SYNTROPHIN</td>
<td>AD</td>
<td>LQTS</td>
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<tr>
<td>SOS1</td>
<td>SON OF SEVENLESS, DROSOPHILA, HOMOLOG 1</td>
<td>AD</td>
<td>Noonan/CFC/Costello</td>
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<tr>
<td>TAZ</td>
<td>TAFAZZIN</td>
<td>AD</td>
<td>DCM, LVNC, Barth syndrome</td>
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<td>TCAP</td>
<td>TITIN-CAP (TELETHONIN)</td>
<td>AD, AR</td>
<td>HCM, DCM, LGMD</td>
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<td>TEMEM43</td>
<td>TRANSMEMBRANE PROTEIN 43</td>
<td>AD</td>
<td>ARVC, EMD</td>
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<td>TMPO</td>
<td>THYMOPOIETIN</td>
<td>AD</td>
<td>DCM</td>
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<td>Gene (cont.)</td>
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<td>Inheritance</td>
<td>Disease Association(s)</td>
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<td>AD</td>
<td>DCM, HCM</td>
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<td>TROPONIN I, CARDIAC</td>
<td>AD, AR</td>
<td>DCM, HCM, RCM</td>
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<td>DCM, HCM, RCM, LVNC</td>
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<td>DCM, HCM</td>
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<td>TRIADIN</td>
<td>AR</td>
<td>CPVT, LQTS</td>
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<td>TRPM4</td>
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<td>AD</td>
<td>HB, BrS</td>
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<td>TTN</td>
<td>TITIN</td>
<td>AD</td>
<td>DCM, ARVC, TTN-related myopathies and muscular dystrophies</td>
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<td>TRANSTHYRETIN</td>
<td>AR</td>
<td>TTR-related amyloidosis</td>
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<td>DCM</td>
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<td>HCM, DCM, LVNC</td>
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References: