Muscular Dystrophy
Congenital Muscular Dystrophy
Emery-Dreifuss Muscular Dystrophy
Limb Girdle Muscular Dystrophy
Duchenne Muscular Dystrophy
Congenital Myopathy
Distal Myopathy
Myofibrillar Myopathy
Myotonic Syndromes
Congenital Myasthenic Syndrome
Periodic Paralysis
Malignant Hyperthermia

A Phenotype-Based
Next Generation Sequencing Approach
When your patient presents with symptoms of muscle disease, the specific cause is not always clear. Using Next Generation Sequencing (NGS) to uncover a genetic cause of disease may lead to answers that direct the most appropriate treatment and care for your patient.

Athena Diagnostics® now offers NGS evaluations for neuromuscular disease arranged by clinical phenotype, testing only the most relevant genes, which streamlines the diagnostic process and saves time and money.

By combining diagnostic technology, fast turnaround time, and Athena Insight™, Athena Diagnostics offers a uniquely powerful toolset that can make an important difference in the care of your patient. It may also help predict the risk of disease in family members.

The More You Know, The More You Can Do

An accurate and early diagnosis can be essential for both evaluation and treatment because specific interventions are increasingly being recognized.
1. Phenotype-Guided Evaluations
   • Athena Diagnostics Neuromuscular Advanced Evaluations are organized by phenotype, making test selection easy and avoiding costly and unnecessary testing.
   • Evaluations are based on peer-reviewed published literature* to facilitate clinically-meaningful diagnostic and treatment decisions.

2. Fast, Cost-Efficient Testing with Powerful Proprietary Technology
   • Concordance with Sanger sequencing is achieved for excellent sensitivity and specificity.
   • Our hybridization-based enrichment technology allows removal of duplicate fragments, reducing amplification bias, providing excellent allele balance, more confident variant calls, and better sensitivity.
   • Regions with known pseudogenes or other sequence homology are sequenced at no additional charge using Sanger technology if interference with NGS accuracy is detected.
   • Each evaluation is accomplished with one blood draw and a 6-week turnaround.

3. Athena Insight™, In-Depth Variant Interpretation and Reporting
   • Created by scientists and geneticists at Athena Diagnostics, Athena Insight™ incorporates a standardized, evidence-based pathogenicity assessment/scoring process that stratifies variants based on the relative likelihood of pathogenicity.
   • Our team of variant scientists collaborate on evaluations to provide the best possible results and optimal analytic value.
   • Assessments of variants are based on multiple independent types of evidence. This provides the confidence of pathogenicity assessment from accumulated evidence in the medical literature, as well as variant databases.
   • The clinician receives a complete synopsis and interpretation of findings with a determination of the likelihood of variants being benign or pathogenic. Results are presented in clear, concise terms suitable for use during discussions with patients and family members.
   • Over 19,000 pathogenicity assessments on more than 12,000 unique variants have been successfully performed to date, focused on genetic disorders in neurology, endocrinology, and nephrology. This allows a more accurate definition of the region of interest.
   • In many cases, it is beneficial to test other family members to enhance interpretations of variants of unknown significance. Please call an Athena Diagnostics genetic counselor for details.

Comprehensive Services that Go Beyond Results

**Genetic Counseling Services**
Our team of genetic counselors is readily available to provide in-depth information on the nature, inheritance, and implications of genetic test results.
- Genetic counseling can help the physician guide the patient in making informed medical and personal decisions for themselves and their family.

![Image of genetic counselor](image1)

**Pre-Authorization Services**
Pre-authorization for in-network orders eases the workload in your office.

**Care360®**
The power and flexibility of this cloud-based, electronic health record system developed by Quest Diagnostics enables 24/7 access and greater efficiencies in managing lab orders and results.
- Place test orders, analyze results, and gain practice-wide insights quickly and easily.
- Save time and improve access to critical patient information.

New Billing Solutions that Simplify Testing

**Introducing the Athena Alliance Program**
Athena Diagnostics is committed to providing testing that makes a difference in patient care. As a part of that commitment, we are pleased to announce the Athena Alliance Program, a new solution dedicated to simplifying the billing process for you and your patients.

![Image of billing solution](image2)

**Personalized Patient Services**
- Support from point of order, to specimen collection, to determination of insurance coverage, submission, and appeals
- Regionally structured to provide local expertise
- Decision support algorithms to order the most useful testing for your patients

**Financial Assistance**
- Income-based financial assistance is available to patients in all 50 U.S. states regardless of insurance status.

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Phenotype-Based Next Generation Sequencing for Neuromuscular Disease
## Neuromuscular Genes Tested

<table>
<thead>
<tr>
<th>Test</th>
<th>Genes Tested</th>
</tr>
</thead>
<tbody>
<tr>
<td>5501</td>
<td>Muscular Dystrophy</td>
</tr>
<tr>
<td>5502</td>
<td>Congenital Muscular Dystrophy</td>
</tr>
<tr>
<td>5503</td>
<td>Congenital Myopathy</td>
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<tr>
<td>5504</td>
<td>Distal Myopathy</td>
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<tr>
<td>5505</td>
<td>Myofibrillar Myopathy</td>
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<tr>
<td>5506</td>
<td>Myotonic Syndromes</td>
</tr>
<tr>
<td>5507</td>
<td>Periodic Paralysis</td>
</tr>
<tr>
<td>5508</td>
<td>Malignant Hyperthermia</td>
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<tr>
<td>5509</td>
<td>Emery-Dreifuss Muscular Dystrophy</td>
</tr>
<tr>
<td>5510</td>
<td>Malignant Myoathesic Syndrome</td>
</tr>
<tr>
<td>5511</td>
<td>Limb Girdle Muscular Dystrophy</td>
</tr>
</tbody>
</table>

**NOTE:** *Test available in mid-2015  † Repeat Expansion Analysis  †† Sequencing and Duplication/Deletion*
Athena Diagnostics Phenotype-Based Testing for the Causes of Neuromuscular Disease

Test selection is intuitive - conveniently arranged by clinically relevant groups of neuromuscular phenotypes. This approach is especially useful in diagnosing cases with broad phenotypes and non-specific clinical findings.

<table>
<thead>
<tr>
<th>PHENOTYPIC CHARACTERIZATION</th>
<th>MUSCULAR DYSTROPHIES (MD)</th>
<th>CONGENITAL MUSCULAR DYSTROPHIES (CMD)</th>
<th>EMERY-DREIFFUS MUSCULAR DYSTROPHY</th>
<th>LIMB GIRDLLE MUSCULAR DYSTROPHY</th>
<th>CONGENITAL MYOPATHIES</th>
<th>DISTAL MYOPATHIES</th>
</tr>
</thead>
<tbody>
<tr>
<td>• Characterized by wasting of skeletal muscle and progressive muscle weakness; can lead to premature death</td>
<td>• Visible at birth or early infancy</td>
<td>• Age of onset, severity, and progression of muscle and cardiac involvement vary inter- and intrafamilially</td>
<td>• Weakness and wasting restricted to the limb musculature, proximal greater than distal</td>
<td>• Skeletal muscle weakness of variable severity</td>
<td>• Distal muscles of upper or lower limbs are selectively or disproportionately affected</td>
<td></td>
</tr>
<tr>
<td>• Severity, age of onset, rate of progression, predominant distribution pattern of muscle weakness, mode of inheritance, complications and prognosis vary greatly among the different forms1</td>
<td>• Congenital hypotonia</td>
<td>• Clinically variable from early onset; severe presentation in childhood to late onset with slow progression in adulthood</td>
<td>• Most individuals show relative sparing of heart and bulbar muscles, although exceptions occur depending on genetic subtype</td>
<td>• Relatively stable muscle weakness from birth or soon after is common</td>
<td>• AR or AD inheritance</td>
<td></td>
</tr>
<tr>
<td>• Delayed motor development and contractures</td>
<td>• Progressive muscle weakness</td>
<td>• AD, AR, X-linked inheritance</td>
<td>• Delayed motor milestones</td>
<td>• Different forms vary in phenotype regarding age of onset and pattern of muscle involvement23</td>
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<td></td>
</tr>
<tr>
<td>• Dysrophic features on muscle biopsy</td>
<td>• Possible CK elevation</td>
<td>• Symptoms generally appear as clinical triad: 1. Joint contractures during the first two decades</td>
<td>• Onset, progression, and distribution of weakness and wasting vary considerably among individuals and genetic subtypes</td>
<td>• AD or AR inheritance27</td>
<td>• Congenital hypotonia</td>
<td></td>
</tr>
<tr>
<td>• Respiratory and cardiovascular complications are common</td>
<td>• Abnormalities of eye, skin and brain</td>
<td>• 2. Followed by muscle weakness and wasting</td>
<td>• Hypoactive tendon deep reflexes</td>
<td>• Hypoactive tendon deep reflexes</td>
<td>• Reduced muscle bulk</td>
<td></td>
</tr>
<tr>
<td>• Abnormalities of eye, skin and brain</td>
<td>• Seizures are frequently associated, especially in those with brain malformations14</td>
<td>• 3. Cardiac involvement usually occurs after the second decade15</td>
<td>• Commonly-long narrow face and high arched palate</td>
<td>• Presentations vary from severe neonatal forms with fetal akinesia to mild adult-onset forms50</td>
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<tr>
<td>• Seizures are frequently associated, especially in those with brain malformations14</td>
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</tbody>
</table>

Some forms of MD include:
• Duchenne muscular dystrophy (DMD)
• Becker muscular dystrophy (BMD)
• Emery-Dreifuss muscular dystrophy (EDMD)
• Limb-girdle muscular dystrophy (LGMD)
• Facioscapulohumeral muscular dystrophy (FSMD)
• Congenital muscular dystrophy (CMD)
• Distal muscular dystrophy (DM)

The most common CMD subtypes, grouped by protein function in which causative mutations occur, include:
- LAMA2-related
- Collagen VI-deficient
- Dystroglycanopathy-related
- SEPN1-related
- LMNA-related.67

Subtypes within these groups include: LAMA2-related muscular dystrophy, Ullrich congenital muscular dystrophy (UCMD), Bethlem CMD, muscle-eye-brain (MEB) disease, Fukuyama CMD (FCMD), and rigid spine CMD.128

LGMD consists of many genetically distinct subtypes, including:
- LGMD2L
- LGMD2A (Calpainopathy)
- LGMD2R
- LGMD2B (Dyserinopathy)
- LGMD2I (MDDGC5)
- LGMD2G
- LGMD2H
- LGMD2D (alpha-sarcoglycanopathy)
- LGMD2E (beta-sarcoglycanopathy)
- LGMD2F (delta-sarcoglycanopathy)
- LGMD2C (gamma-sarcoglycanopathy)
- LGMD2J
- LGMD2K (MDDGC1)
- LGMD2N (MDDGC2)
- LGMD2O (MDDGC3)
- LGMD2Q
- LGMD2P (MDDGC9)
- LGMD2S
- LGMD2M (MDDGC4)

Many forms of congenital myopathy have been identified. The general categories include:
- Congenital myopathies with cores
- Congenital myopathies with protein aggregates
- Myosin storage myopathy
- Congenital myopathies with central nuclei
- Congenital myopathies with abnormal fiber ratios or sizes510

Major forms of Distal Myopathy include:
- Welander distal myopathy
- Tibial muscular dystrophy
- Distal myotilinopathy
- ZASPopathy
- Vocal cord and pharyngeal distal myopathy
- Alpha-B crystallin mutated distal myopathy
- Desminopathy
- Distal ABD-filaminopathy
- Laing distal myopathy
- KLHL9 mutated distal myopathy
- Distal nebulin myopathy
- Miyoshi myopathy
- Distal Arracinopathy
- Distal myopathy with rimmed vacuoles516

Next Generation Sequencing Testing for Distal Neuromuscular Disorders

**NMD PHENOTYPE**

- Becker muscular dystrophy include:
- Duchenne muscular dystrophy (DMD)
- Dystrophic features on muscle biopsy
- Respiratory and cardiovascular complications are common
- Seizures are frequently associated, especially in those with brain malformations14

**33 genes tested**

**23 genes tested**

**5 genes tested**

**21 genes tested**

**20 genes tested**

**17 genes tested**

**5501, Muscular Dystrophy Advanced Evaluation** 33 genes tested

**5502, Congenital Muscular Dystrophy Advanced Sequencing Evaluation** 23 genes tested

**5508, Emery-Dreifuss Muscular Dystrophy Advanced Sequencing Evaluation** 6 genes tested

**5509, Limb Girdle Muscular Dystrophy Advanced Evaluation** 23 genes tested

**5510, Congenital Myopathy Advanced Sequencing Evaluation** 21 genes tested

**5511, Distal Myopathy Advanced Sequencing Evaluation** 17 genes tested
The organization of Athena Diagnostics Evaluations is based on evidence from publications using NGS as a diagnostic tool for neuromuscular disorders and relevant variant databases.

### MYOFIBRILLAR MYOPATHIES
- Slowly progressive weakness in distal and/or proximal muscles of individuals
- Distal muscle weakness is more pronounced and common (80%) than proximal (25%)
- Sensory symptoms, muscle stiffness, aching, or cramps may occur
- Peripheral neuropathy present in 20% of cases
- Overt cardiomyopathy present in 15-30%6

### MYOTONIC SYNDROMES
- Myotonic dystrophy type 1 (DM1) and type 2 (DM2) are progressive disorders, associated with cardiac defects, endocrine abnormalities, neurological dysfunctions and cataracts
  - DM1, the most common form of adult onset MD, characterized by marked variability between and within family members. The most severe form is congenital, showing developmental delay, severe muscle weakness, hypotonia.
  - DM2 patients may have milder clinical presentation than DM1, and in mildest form, DM2 can be difficult to recognize.10,11
- Non-dystrophic myotonic disorders are a heterogeneous group of skeletal muscle disorders.
  - Myotonia Congenita (MC) is the most common skeletal muscle channelopathy. There are two forms: autosomal dominant (Thomsen), and autosomal recessive (Becker). Both show a remarkable degree of phenotypic and genotypic variability, and are more severe in men than in women. Variable age of onset.8
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### CONGENITAL MYASTHENIC SYNDROMES
- Spectrum of muscle disease with X-linked inheritance can present as:
  - Mild-muscle cramps with myoglobinuria and quadriiceps myopathy
  - Severe-progressive muscle diseases classified as Duchenne (DMD)/Becker (BMD) when skeletal muscle is primarily affected and as DMD-associated dilated cardiomyopathy (DCM) when heart is primarily affected
  - DMD usually presents in early childhood with delayed milestones, including delays in sitting and standing independently; rapidly progressive
  - BMD is characterized by later-onset skeletal muscle weakness20
  - More severe respiratory involvement in the mildest form of DMD

### DYSTROPHINOPATHIES
- Periodic paralyses are classified into groups:
  - Hyperkalemic PP (hyperPP) is characterized by attacks of flaccid limb paralysis with weakness associated with abnormally elevated levels in blood potassium concentrations. Three clinically distinct manifestations: (1) without myotonia, (2) with clinical or electromyographic (EMG) myotonia, or (3) with paramyotonia congenital (PMC).
  - Hypokalemic PP (hypoPP) is generally characterized by reversible attacks of muscle weakness associated with an abnormal decrease in blood potassium concentrations. Recovery occurs when serum potassium normalizes.20
  - Andersen-Tawil syndrome (ATS) is characterized by a unique phenotype consisting of periodic paralysis, cardiac arrhythmias, and skeletal dysmorphic abnormalities. Paralysis may occur with either hyperkalemia or hypokalemia that exacerbate the cardiac arrhythmia with sudden cardiac arrest in 10% of patients.23

### PERIODIC PARALYSIS
- Muscle weakness (i.e. ocular, bulbar, limb muscles, but no cardiac or smooth muscle movement)
- Motor developmental delay
- Exercise intolerance
- Apneas
- Risk to infection
- Earlier onset cases typically display:
  - Difficulty feeding
  - Weak cry and sucking
  - Choking spells
  - Specific clinical course and severity are often highly variable
  - Subsets can be AD or AR23,24,25

### MALIGNANT HYPERThERMIA
- In most cases, first signs (tachycardia and tachypnea) may appear when patient is given anesthesia, during anesthetization or early post-op
- Affected patient can present with metabolic acidosis, hypercapnia, tachycardia, hyperthermia, muscle rigidity, hyperkalemia with risk for cardiac arrhythmia or cardiac arrest, muscle breakdown with CK increase, and myoglobinuria with risk for renal failure
- AD inheritance22
- Clinical manifestation may depend on genetic predisposition, dose of trigger agents, or duration of exposure. Almost all individuals with MH appear normal without any pathologic signs; it is impossible to diagnose susceptibility without either the exposure to triggering agents or by specific diagnostic testing.26

**Table: Myopathies and Myotonic Syndromes**

<table>
<thead>
<tr>
<th>MYOFIBRILLAR MYOPATHIES</th>
<th>MYOTONIC SYNDROMES</th>
<th>CONGENITAL MYASTHENIC SYNDROMES</th>
<th>DYSTROPHINOPATHIES</th>
<th>PERIODIC PARALYSIS</th>
<th>MALIGNANT HYPERThERMIA</th>
</tr>
</thead>
<tbody>
<tr>
<td>Slowly progressive weak</td>
<td>Myotonic dystrophy</td>
<td>Spectrum of muscle disease</td>
<td>Episodes of muscle</td>
<td>Hyperkalemic PP</td>
<td>1. In most cases, first</td>
</tr>
<tr>
<td>in distal and/or proxim</td>
<td>type 1 (DM1) and</td>
<td>with X-linked inheritance</td>
<td>weakness associated</td>
<td>(hyperPP)</td>
<td>signs (tachycardia and</td>
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<td>al muscles of</td>
<td>type 2 (DM2) are</td>
<td>can present as:</td>
<td>with changes in</td>
<td>Hypokalemic PP</td>
<td>tachypnea) may appear</td>
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<tr>
<td>individuals</td>
<td>progressive</td>
<td>Mild-muscle cramps</td>
<td>serum potassium</td>
<td>(hypoPP)</td>
<td>when patient is given</td>
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<td></td>
<td>disorders</td>
<td>with myoglobinuria</td>
<td>concentration</td>
<td>Andersen-Tawil</td>
<td>anesthesia, during</td>
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<td></td>
<td>associated with</td>
<td>and quadriiceps myopathy</td>
<td>Hyperkalemic PP</td>
<td>syndrome (ATS)</td>
<td>anesthetization or early</td>
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<td></td>
<td>cardiac defects,</td>
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<td>(3) with paramyotonia</td>
<td>characterized by</td>
<td>post-op.</td>
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<td></td>
<td>endocrine</td>
<td>Severe-progressive</td>
<td>congenital (PMC).</td>
<td>a unique phenotype</td>
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<td>abnormalities,</td>
<td>muscle diseases</td>
<td>Attacks typically</td>
<td>consisting of</td>
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<td>neurological</td>
<td>classified as Duchenne</td>
<td>begin in the first</td>
<td>periodic paralysis,</td>
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<td></td>
<td>dysfunctions and</td>
<td>(DMD)/Becker (BMD) when</td>
<td>decade of life,</td>
<td>cardiac arrhythmia,</td>
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<td></td>
<td>cataracts</td>
<td>skeletal muscle</td>
<td>increase in</td>
<td>muscle breakdown,</td>
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<td>is primarily affected</td>
<td>frequency and severity</td>
<td>CK increase,</td>
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<td>and as DMD-associated</td>
<td>over time.</td>
<td>myoglobinuria</td>
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<td>dilated cardiomyopathy (DCM)</td>
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<td>with risk for</td>
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<td>when heart is primarily</td>
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<td>renal failure</td>
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<td>affected</td>
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<td>AD inheritance22</td>
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<td>DMD usually presents</td>
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<td>Clinical manifesta-</td>
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<td>progressive</td>
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<td>sure. Almost all</td>
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<td>BMD is characterized</td>
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<td>individuals with</td>
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<td>by later-onset skeletal</td>
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<td>MH appear normal</td>
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<td>muscle weakness</td>
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<td>without any patho-</td>
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<td>with normal</td>
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<td>logic signs; it is</td>
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<td>without (i.e. ocular, bulbar,</td>
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<td>limb muscles, but no</td>
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<td>movement)</td>
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<td>to triggering agents</td>
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</tbody>
</table>
|                        |                   |                                 |                   | or by specific dia-
|                        |                   |                                 |                   | gonstic testing.26   |

**Notes:**
- Data on file.
Test Menu for Neuromuscular Disease from Athena Diagnostics

<table>
<thead>
<tr>
<th>Test Code</th>
<th>Test Name</th>
<th>Whole Blood, Lavender Top Tube/ Minimum Volume* (mL)</th>
<th>Turnaround Time</th>
</tr>
</thead>
<tbody>
<tr>
<td>5501</td>
<td>Muscular Dystrophy Advanced Evaluation CPT Codes**: 81404 x4, 81405 x9, 81406 x8, 81408 x2, 81161, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
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<tr>
<td>5502</td>
<td>Congenital Muscular Dystrophy Advanced Sequencing Evaluation CPT Codes: 81404 x2, 81405 x2, 81406 x3, 81407 x3, 81408, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5503</td>
<td>Congenital Myopathy Advanced Sequencing Evaluation CPT Codes: 81404 x2, 81405 x2, 81408 x2, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5504</td>
<td>Distal Myopathy Advanced Sequencing Evaluation CPT Codes: 81404, 81405 x2, 81407, 81408 x2, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5505</td>
<td>Myofibrillar Myopathy Advanced Sequencing Evaluation CPT Codes: 81404, 81405 X2, 81406, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5506</td>
<td>Myotonic Syndromes Advanced Sequencing Evaluation CPT Codes: 81401, 81404, 81406, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5507</td>
<td>Periodic Paralysis Advanced Sequencing Evaluation CPT Codes: 81403, 81404, 81406, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5508</td>
<td>Malignant Hyperthermia Advanced Sequencing Evaluation CPT Codes: 81408, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5518</td>
<td>Emery-Dreifuss Muscular Dystrophy Advanced Sequencing Evaluation CPT Codes: 81404, 81405, 81406, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5519</td>
<td>Limb Girdle Muscular Dystrophy Advanced Evaluation CPT Codes: 81404 x3, 81405 x7, 81406 x7, 81408, 81409, 81479</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5511</td>
<td>Congenital Myasthenic Syndrome Advanced Sequencing Evaluation***</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5530</td>
<td>DMD Evaluation (new code, existing test) CPT Codes: 81161, 81608</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
<tr>
<td>5531</td>
<td>DMD Duplication/Deletion (new code, existing test) CPT Code: 81161</td>
<td>6-8 Adult, 1-2 Pediatric</td>
<td>6 weeks</td>
</tr>
</tbody>
</table>

*We require whole blood in EDTA tubes (lavage top tubes). Adults: 10 mL; Children: 4 mL; Infants: 2 mL. Outside DNA is discouraged; however, high quality extracted DNA that meet our standards can be accepted. The test requires a minimum of 20 ng of DNA at a concentration of 50 ng/µL with a minimum volume of 400 µl. **The CPT codes provided are based on AMA guidelines and are for informational purposes only. CPT coding is the sole responsibility of the billing party. ***Available in mid-2015. Please contact one of our genetic counselors regarding specific acceptance policies and specimen requirements for prenatal testing at 800-394-4493.

Client Services Representatives are available from 8:30am to 6:30pm Eastern Time (U.S.). Customers in the U.S. and Canada please call toll free 800-394-4493 or visit us on our website at AthenaDiagnostics.com.