Overview

Useful For

Providing a comprehensive genetic evaluation for patients with a personal or family history suggestive of congenital neutropenia, cyclic neutropenia, or other primary immunodeficiency disorder (PIDD) presenting with significant neutropenia

Establishing a diagnosis and, in some cases, allowing for appropriate management and surveillance for disease features based on the gene involved

Identifying variants within genes known to be associated with PIDD characterized by significant neutropenia allowing for predictive testing of at-risk family members

Genetics Test Information

This test includes next-generation sequencing and supplemental Sanger sequencing to test for variants in the AP3B1(HP2), CSF3R, CXCR4, ELANE(ELA2), G6PC3, GATA2, GFI1, HAX1, LAMTOR2(MAPBPIP), RAC2, SBDS, SLC37A4, TAZ, USB1(C16ORF57), VPS13B(COH1), VPS45, WAS, and WIPF1 genes.

Identification of a pathogenic variant may assist with prognosis, clinical management, familial screening, and genetic counseling.

Reflex Tests

<table>
<thead>
<tr>
<th>Test ID</th>
<th>Reporting Name</th>
<th>Available Separately</th>
<th>Always Performed</th>
</tr>
</thead>
<tbody>
<tr>
<td>FIBR</td>
<td>Fibroblast Culture</td>
<td>Yes</td>
<td>No</td>
</tr>
<tr>
<td>CRYOB</td>
<td>Cryopreserve for Biochem Studies</td>
<td>No</td>
<td>No</td>
</tr>
</tbody>
</table>

Testing Algorithm

For skin biopsy or cultured fibroblast specimens, fibroblast culture and cryopreservation testing will be performed at an additional charge. If viable cells are not obtained, the client will be notified.

Special Instructions

- [Informed Consent for Genetic Testing](#)
- [Blood Spot Collection Card-Spanish Instructions](#)
- [Primary Immunodeficiencies Patient Information](#)
- [Blood Spot Collection Card-Chinese Instructions](#)
- [Informed Consent for Genetic Testing (Spanish)](#)

Method Name

Custom Sequence Capture and Targeted Next-Generation Sequencing followed by Polymerase Chain Reaction (PCR) and Supplemental Sanger Sequencing

NY State Available

Yes
Test Definition: SCNGP
Congenital Neutropenia PID Panel

Specimen

Specimen Type
Varies

Advisory Information
Targeted testing for familial variants (also called site-specific or known mutation testing) is available for the genes on this panel. See:

-KVAR1 / Known Variant Analysis-1 Variant, Varies
-KVAR2 / Known Variant Analysis-2 Variants, Varies
-KVAR3 / Known Variant Analysis-3+ Variants, Varies

Call 800-533-1710 to confirm the appropriate test for targeted testing.

Necessary Information
1. Primary Immunodeficiencies Patient Information (T791) is required. See Special Instructions.

Note: Testing may proceed without the Patient Information, however, it aids in providing a more thorough interpretation. Ordering physicians are strongly encouraged to fill out the form.

2. Include physician name and phone number with specimen.

Specimen Required
Submit only 1 of the following specimens:

Preferred:

Specimen Type: Whole blood
Container/Tube: Lavender top (EDTA)
Specimen Volume: 5 mL

Collection Instructions:
1. Invert several times to mix blood.
2. Send specimen in original tube.

Additional Information: Please note that for patients with severe neutropenia, DNA yield may be insufficient for testing. Consider sending additional volume or an alternate specimen type.

Specimen Stability Information: Ambient (preferred)4 days/Refrigerated 14 days
Specimen Type: Blood spot
Supplies: Card-Blood Spot Collection Filter Paper (T493)

Container/Tube:

Preferred: Collection card (Whatman Protein Saver 903 Paper)

Acceptable: Whatman FTA Classic paper, Ahlstrom 226 filter paper, or Blood Spot Collection Card

Specimen Volume: 5 blood spots on collection card

Collection Instructions:

1. An alternative blood collection option for a patient <1 year of age is finger stick.
2. Let blood dry on the filter paper at ambient temperature in a horizontal position for 3 hours.
3. Do not expose specimen to heat or direct sunlight.
4. Do not stack wet specimens.
5. Keep specimen dry.

Additional Information:

1. Please note that for patients with severe neutropenia, DNA yield may be insufficient for testing. Consider sending additional volume or an alternate specimen type.
2. For collection instructions in Spanish, see Blood Spot Collection Card-Spanish Instructions (T777) in Special Instructions.
3. For collection instructions in Chinese, see Blood Spot Collection Card-Chinese Instructions (T800) in Special Instructions.

Specimen Stability Information: Ambient (preferred)/Refrigerated

Specimen Type: Peripheral blood mononuclear cells (PBMCs)

Container/Tube: Cell pellet

Collection Instructions: Send as a suspension in freezing medium or cell pellet frozen on dry ice.

Specimen Stability Information: Frozen

Specimen Type: Cultured fibroblasts

Container/Tube: T-75 or T-25 flask

Specimen Volume: 1 Full T-75 or 2 full T-25 flasks

Additional Information: Indicate the tests to be performed on the fibroblast culture cells. A separate culture charge
will be assessed under FIBR / Fibroblast Culture. An additional 4 weeks is required to culture fibroblasts before genetic testing can occur.

**Specimen Stability Information:** Ambient (preferred)/Refrigerated <24 hours

**Specimen Type:** Skin biopsy

**Supplies:** Fibroblast Biopsy Transport Media (T115)

**Container/Tube:** Sterile container with any standard cell culture media (eg, minimal essential media, RPMI 1640). The solution should be supplemented with 1% penicillin and streptomycin. Tubes of culture media can be supplied upon request (Eagle's minimum essential medium with 1% penicillin and streptomycin).

**Specimen Volume:** 4-mm punch

**Additional Information:** A separate culture charge will be assessed under FIBR / Fibroblast Culture. An additional 4 weeks is required to culture fibroblasts before genetic testing can occur.

**Specimen Stability Information:** Refrigerated (preferred)/Ambient

**Specimen Type:** DNA

**Container/Tube:** 2 mL screw top tube

**Specimen Volume:** 100 mcL (microliters)

**Collection Instructions:**

1. The preferred volume is 100 mcL at a concentration of 250 ng/mcL

2. Include concentration and volume on tube.

**Specimen Stability Information:** Frozen (preferred)/Ambient/Refrigerated

**Forms**

1. **Primary Immunodeficiencies Patient Information** (T791) is required. See Special Instructions.

2. **New York Clients-Informed consent is required.** Document on the request form or electronic order that a copy is on file. The following documents are available in Special Instructions:

   - **Informed Consent for Genetic Testing** (T576)
   - **Informed Consent for Genetic Testing-Spanish** (T826)

**Specimen Minimum Volume**

Whole blood: 1 mL

**Reject Due To**

All specimens will be evaluated at Mayo Clinic Laboratories for test suitability.
Severe congenital neutropenia is a primary immunodeficiency disorder (PIDD) that is characterized by severe and recurrent bacterial infections, such as otitis media, bronchitis, pneumonia, osteomyelitis, and cellulitis, typically with the absence of pus at the infected site. Susceptibility to fungal infections may also be observed. Neutropenia may be an isolated finding or may be part of a syndrome. This panel includes genes associated with neutropenia as a major presenting feature; other panels may be more appropriate when neutropenia is identified but not as the main finding.

Pathogenic variants in \textit{ELANE}, which encodes neutrophil elastase, can result in severe congenital neutropenia type 1 (SCN1) or cyclic neutropenia. SCN1 often presents immediately with omphalitis, while diarrhea, pneumonia, and deep abscesses affecting the liver, lungs, or subcutaneous tissues are noted within the first year. Patients are at risk for development of myelodysplastic syndrome or acute myelogenous leukemia, presumably due to acquired mutations in \textit{CSF3R} (which may also be identified in the presence of congenital neutropenia due to variants in genes other than \textit{ELANE}, see below). Biallelic mutations in \textit{CSF3R} have also been recently reported to be associated with severe congenital neutropenia. Cyclic neutropenia typically presents in the first year of life with 3-week-long oscillations in cell counts along with intervals of fever, oral ulcerations, and ulcerations; between intervals, patients are generally healthy. Unlike SCN1, cyclic neutropenia is not associated with risk of malignancy. Both SCN1 and cyclic neutropenia are inherited in an autosomal dominant pattern from an affected parent, although de novo variants have been identified. Studies have demonstrated pathogenic variants in \textit{ELANE} in nearly 100% of cases with well-documented classical cyclic neutropenia, while in some cases with atypical presentations (ie, oscillations that are not 3 weeks) a variant in \textit{ELANE} is not identified. \textit{ELANE} variants are identified in 38% to 80% of cases of congenital neutropenia, depending on the criteria used to identify patients. Although there is some overlap, generally, variants at the active site of neutrophil elastase result in cyclic neutropenia, while variants that prevent normal folding or packaging of the enzyme cause congenital neutropenia.

In addition to variants in \textit{ELANE}, severe congenital neutropenia, where the predominant finding is neutropenia, can be inherited as a result of pathogenic variants in other genes. Dominant variants in \textit{GFI1} (encoding growth factor independent 1) result in severe congenital neutropenia type 2 (SCN2). Pathogenic variants in \textit{G6PC3} (encoding glucose-6-phosphate 3), which are inherited in an autosomal recessive manner, can result in a phenotypic spectrum from isolated/nonsyndromic severe congenital neutropenia to classic \textit{G6PC3} deficiency (severe neutropenia along with cardiovascular and urogenital abnormalities) to severe \textit{G6PC3} deficiency (also known as Dursun syndrome, which includes features of classic \textit{G6PC3} deficiency along with severe lymphopenia, primary pulmonary hypertension, thymic hypoplasia, among other features). Kostmann disease or severe congenital neutropenia type 3 (SCN3) is due to recessive inheritance of pathogenic variants in \textit{HAX1} (which encodes HCLS1-associated protein X-1) and may result in seizures and developmental delay in addition to neutropenia. Along with neutropenia, variants in \textit{VPS45} inherited in an autosomal recessive manner (also known as severe congenital neutropenia type 5 [SNC5]) are associated with neutrophil dysfunction, bone marrow fibrosis, and nephromegaly due to renal extramedullary hematopoiesis. While loss-of-function variants in \textit{WAS}, which is located on the X chromosome, cause Wiskott-Aldrich syndrome (characterized by thrombocytopenia, eczema, and recurrent infections), gain-of-function variants affecting the autoinhibitory structure of the protein, have been associated with congenital neutropenia, along with
variable lymphopenia, decreased lymphocyte proliferation, and impaired phagocyte activity. Pathogenic variants in WIPF1 can present with similar findings to Wiskott-Aldrich syndrome.

Severe neutropenia may also be present as part of a multisystem disorder. Barth syndrome, due to pathogenic variants in TAZ, which is located on the X-chromosome, is characterized by neutropenia, cardio- and skeletal myopathy, growth delay, and distinctive facial features. Biallelic variants in C16orf75 manifest as poikilodermia with neutropenia; the neutropenia may be cyclical. In Cohen syndrome, an autosomal recessive disorder due to variants in COH1 (also known as VPS13B), neutropenia is accompanied by hyponatremia, developmental delays, microcephaly, failure to thrive in infancy, truncal obesity in adolescent years, ophthalmologic findings, joint hypermobility, a cheerful disposition, and characteristic facial features. Glycogen storage disease type I (GSDI), caused by biallelic pathogenic variants in either G6PC or SLC37A4, when untreated can result in chronic neutropenia and impaired neutrophil and monocyte function, as well as the characteristic findings that include accumulation of glycogen and fat in the liver and kidneys. Pathogenic variants in LAMTOR2/MAPBP1 have been shown to result in neutropenia, decreased cytotoxic activity of CD8+ T-cells, short stature, and hypopigmented skin. Persistent or intermittent neutropenia is often a presenting feature of Shwachman-Diamond syndrome (SDS), which is also characterized by exocrine pancreatic dysfunction (with malabsorption, malnutrition, and growth failure), bone abnormalities, and hematologic abnormalities (single- or multilineage cytopenias along with predisposition to myelodysplastic syndrome and acute myelogenous leukemia). SDS is an autosomal recessive disorder due to pathogenic variants in SBDS. Warts, hypogammaglobulinemia, immunodeficiency, and myelokathexis (WHIM) syndrome is characterized by neutropenia in addition to hypogammaglobulinemia, and susceptibility to human papillomavirus. It is due to autosomal dominant pathogenic variants in CXCR4. Although most forms of Hermansky-Pudlak syndrome do not include significant neutropenia, type 2 caused by variants in AP3B1 can be associated with persistent neutropenia and increased infections in addition to the typical findings of tyrosinase-positive oculocutaneous albinism, platelet storage pool deficiency, pulmonary fibrosis, and granulomatous colitis. Few patients with RAC2 pathogenic variants have been identified, but neutrophil dysfunction appears to be a feature, though CD11b expression and specific granule release appear to be preserved. Both individuals with dominant and individuals with recessive inheritance have been identified, with and without additional associated phenotypic findings.

GATA-binding protein (GATA2) deficiency demonstrates a wide spectrum of clinical presentations, including neutropenia. Most variants appear to arise de novo (spontaneously) and are then transmitted in an autosomal dominant manner. If the clinical phenotype strongly suggests GATA2 deficiency, this gene is available as a stand-alone test (see GATA2 / GATA-Binding Protein 2 (GATA2), Full Gene, Next-Generation Sequencing, Varies). This panel does not evaluate for somatic (acquired) ASXL1 mutations associated with GATA2 deficiency.

Table 1. Genes included in the Congenital Neutropenia/Neutrophil-Related PID Gene Panel

<table>
<thead>
<tr>
<th>GENE SYMBOL (ALIAS)</th>
<th>PROTEIN</th>
<th>OMIM</th>
<th>INCIDENCE</th>
<th>INHERITANCE</th>
<th>PHENOTYPE DISORDER</th>
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<tbody>
<tr>
<td>AP3B1</td>
<td>AP-3 complex subunit beta-1 isoform 1</td>
<td>603401</td>
<td>Rare</td>
<td>AR</td>
<td>Hermansky-Pudlak syndrome 2</td>
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<tr>
<td>CSF3R</td>
<td>Granulocyte colony-stimulating factor receptor isoform a precursor</td>
<td>138971</td>
<td>AR, acquired</td>
<td>Severe congenital neutropenia</td>
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<tr>
<td>CXCR4</td>
<td>C-X-C chemokine receptor type 4 isoform b</td>
<td>162643</td>
<td>AD</td>
<td>Myelokathexis, isolated, WHIM syndrome (AD)</td>
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<tr>
<td>Gene</td>
<td>Description</td>
<td>Reference</td>
<td>Mode of Inheritance</td>
<td>Clinical Presentation</td>
<td></td>
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<tr>
<td>------</td>
<td>-------------</td>
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<td>----------------------</td>
<td></td>
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<tr>
<td>ELANE</td>
<td>Neutrophil elastase preproprotein</td>
<td>130130</td>
<td>AD</td>
<td>Severe congenital neutropenia (SCN), cyclic neutropenia</td>
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<tr>
<td>G6PC3</td>
<td>Glucose-6-phosphatase 3</td>
<td>611045</td>
<td>AR</td>
<td>Dursun syndrome, severe congenital neutropenia (SCN) 4</td>
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<tr>
<td>GATA2</td>
<td>Endothelial transcription factor GATA-2 isoform 1</td>
<td>137295</td>
<td>AD</td>
<td>Immunodeficiency 21, Emberger syndrome, susceptibility to acute myeloid leukemia and myelodysplastic syndrome</td>
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<tr>
<td>GFI1</td>
<td>Zinc finger protein Gfi-1</td>
<td>600871</td>
<td>AD</td>
<td>Severe congenital neutropenia (SCN) 2(AD), nonimmune chronic idiopathic neutropenia of adults</td>
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<tr>
<td>HAX1</td>
<td>HCLS1-associate d protein X-1 isoform a</td>
<td>605998</td>
<td>AR</td>
<td>Severe congenital neutropenia (SCN) 3</td>
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<tr>
<td>LAMTOR2 (MAPBPIP)</td>
<td>Ragulator complex protein LAMTOR2 isoform 1</td>
<td>610389</td>
<td>AR</td>
<td>Immunodeficiency due to defect in MAPB-interacting protein</td>
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<td>RAC2</td>
<td>ras-Related C3 botulinum toxin substrate 2</td>
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<td>AD/AR</td>
<td>Neutrophil functional defects</td>
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<td>SBDS</td>
<td>Ribosome maturation protein SBDS</td>
<td>607444</td>
<td>AR</td>
<td>Shwachman-Diamond syndrome, susceptibility to aplastic anemia</td>
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<td>SLC37A4</td>
<td>Ddipeptidyl peptidase 1 isoform a preproprotein</td>
<td>602671</td>
<td>AR</td>
<td>Glycogen storage disease Ib and 1c</td>
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## Test Definition: SCNGP

### Congenital Neutropenia PID Panel

<table>
<thead>
<tr>
<th>Gene</th>
<th>Description</th>
<th>Symbol</th>
<th>Reference Value</th>
<th>Inheritance</th>
<th>Disease</th>
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<tr>
<td>TAZ</td>
<td>Tafazzin isoform 1</td>
<td>300394</td>
<td>XL</td>
<td>Barth syndrome</td>
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<tr>
<td>USB1 (C16orf57)</td>
<td>U6 snRNA phosphodiesterase isoform 1</td>
<td>613276</td>
<td>Rare</td>
<td>AR</td>
<td>Poikiloderma with neutropenia, rare</td>
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<tr>
<td>VPS13B (COH1)</td>
<td>Vacuolar protein sorting-associated protein 13B isoform 5</td>
<td>607817</td>
<td>AR</td>
<td>Cohen syndrome</td>
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<td>VPS45</td>
<td>Vacuolar protein sorting-associated protein 45 isoform 1</td>
<td>610035</td>
<td>AR</td>
<td>Severe congenital neutropenia (SCN) 5</td>
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<tr>
<td>WAS (Gain-of-function mutations)</td>
<td>Wiskott-Aldrich syndrome protein</td>
<td>300392</td>
<td>XL (gain of function)</td>
<td>Neutropenia, severe congenital, X-linked, thrombocytopenia, X-linked</td>
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<tr>
<td>WIPF1</td>
<td>WAS/WASL-interacting protein family member 1</td>
<td>602357</td>
<td>In progress</td>
<td>Wiskott-Aldrich syndrome 2</td>
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</table>

**AD**=autosomal dominant, **AR**=autosomal recessive, **XL**=X-linked

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**Reference Values**

An interpretive report will be provided.

**Interpretation**

Evaluation and categorization of variants is performed using the most recent published American College of Medical Genetics and Genomics (ACMG) recommendations as a guideline. Variants are classified based on known, predicted, or possible pathogenicity and reported with interpretive comments detailing their potential or known significance. Multiple in silico evaluation tools may be used to assist in the interpretation of these results. The accuracy of predictions made by in silico evaluation tools is highly dependent upon the data available for a given gene, and predictions made by these tools may change over time. Results from in silico evaluation tools should be interpreted with caution and professional clinical judgment.

**Cautions**

The majority of DNA extracted from whole blood is derived from neutrophils. Therefore, blood specimens collected from patients with severe neutropenia may yield limited quantity of DNA that is insufficient for testing. When ordering this test for patients with severe neutropenia, please consider timing the collection when the patient has a higher neutrophil count, submitting an alternate specimen type, or collecting additional blood volume.

**Clinical Correlations:**
Some individuals who have involvement of one or more of the genes on the panel may have a variant that is not identified by the methods performed (e.g., promoter variants, deep intronic variants). The absence of a variant, therefore, does not eliminate the possibility of disease. Test results should be interpreted in context of clinical findings, family history, and other laboratory data. Misinterpretation of results may occur if the information provided is inaccurate or incomplete.

For predictive testing of asymptomatic individuals, it is often useful to first test an affected family member. Identification of a pathogenic variant in an affected individual allows for more informative testing of at-risk individuals.

Technical Limitations:

Next-generation sequencing may not detect all types of genetic variants. The variant detection software has lower detection efficiency for insertion/deletion variants as compared to single nucleotide variants. Therefore, small deletions and insertions greater than 8 nucleotides in length may not be detected by this test. Copy Number variations (CNV) are not currently reported for any of the genes on this panel. Additionally, rare polymorphisms may be present that could lead to false-negative or false-positive results. In some cases, DNA variants of undetermined significance may be identified. If results do not match clinical findings, consider alternative methods for analyzing these genes, such as Sanger sequencing or large deletion/duplication analysis. If the patient has had an allogeneic blood or bone marrow transplant or a recent (i.e., <6 weeks from time of sample collection) heterologous blood transfusion, results may be inaccurate due to the presence of donor DNA. Call 800-533-1710 for instructions for testing patients who have received a bone marrow transplant.

Reclassification of Variants—Policy:

At this time, it is not standard practice for the laboratory to systematically review likely deleterious alterations or variants of uncertain significance that are detected and reported. The laboratory encourages health care providers to contact the laboratory at any time to learn how the status of a particular variant may have changed over time. Consultation with a healthcare provider, or team of healthcare providers, with expertise in genetics and primary immunodeficiencies, is recommended for interpretation of this result.

A list of benign and likely benign variants detected is available from the lab upon request.

Contact the laboratory if additional information is required regarding the transcript or human genome assembly used for the analysis of results.

Clinical Reference


6. Albert MH, Notarangelo LD, Ochs HD: Clinical spectrum, pathophysiology and treatment of the Wiskott-Aldrich


Performance

Method Description

Next-generation sequencing (NGS) is performed using an Illumina instrument with paired-end reads. The DNA is prepared for NGS using a custom Agilent SureSelect Target Enrichment System. Data is analyzed with a bioinformatics software pipeline. Supplemental Sanger sequencing may be performed occasionally in regions where NGS is insufficient for data capture or not specific enough to correctly identify a variant. (Unpublished Mayo method)

The following genes are evaluated in this multigene panel:

AP3B1 (HP2), CSF3R, CXCR4, ELANE (ELA2), G6PC3, GATA2, GFI1, HAX1, LAMTOR2 (MAPBPIP), RAC2, SBDS, SLC37A4, TAZ, USB1 (C16orf57), VPS13B (COH1), VPS45, WAS, WIPF1

PDF Report

No

Day(s) and Time(s) Test Performed

Varies

Analytic Time

4 weeks

Maximum Laboratory Time

8 weeks

Specimen Retention Time

Extracted DNA: 2 months

Performing Laboratory Location

Rochester

Fees and Codes

Fees

- Authorized users can sign in to Test Prices for detailed fee information.
- Clients without access to Test Prices can contact Customer Service 24 hours a day, seven days a week.
Prospective clients should contact their Regional Manager. For assistance, contact Customer Service.

**Test Classification**
This test was developed and its performance characteristics determined by Mayo Clinic in a manner consistent with CLIA requirements. This test has not been cleared or approved by the U.S. Food and Drug Administration.

**CPT Code Information**
81443

**LOINC® Information**

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<th>Test Order Name</th>
<th>Order LOINC Value</th>
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<td>Congenital Neutropenia PID Panel</td>
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