

<b>POLICY TITLE</b>	<b>GENETIC TESTING FOR HEREDITARY HEMOCHROMATOSIS</b>
<b>POLICY NUMBER</b>	<b>MP 2.312</b>

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**I. POLICY**

Genetic testing for hereditary hemochromatosis (*HFE*) gene variants may be considered **medically necessary** in a patient with abnormal serum iron indices indicating iron overload. (See policy guidelines).

Genetic testing for *HFE* gene variants may be considered **medically necessary** in individuals with a family history of hemochromatosis in a first-degree relative. (See policy guidelines).

Genetic testing for hereditary hemochromatosis in screening of the general population is considered **investigational**. There is insufficient evidence to support a conclusion concerning the health outcomes or benefits associated with this procedure for this indication.

**POLICY GUIDELINES**

**Serum Iron Indices for Diagnosing Hereditary Hemochromatosis**

Elevated fasting transferrin saturation (the ratio of serum iron to total iron-binding capacity) is the most sensitive initial phenotypic screening test. A minimum cutoff value of 45% will detect almost all affected C282Y homozygotes.

Serum ferritin reflects body iron stores and generally rises later in the progression of iron overload. In the absence of other causes of hyperferritinemia (alcohol abuse, metabolic syndrome, inflammatory states [e.g., infection, cancer, active rheumatoid arthritis], acute and chronic hepatitis), serum ferritin is a good marker of the degree of iron overload.

The negative predictive value of a normal transferrin saturation and serum ferritin is 97%. In this situation, no further testing is recommended.

The 2011 practice guidelines from the American Association for the Study of Liver Diseases (AASLD) recommended human hemochromatosis (*HFE*) gene variant testing in patients with

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abnormal serum iron indices (i.e., serum ferritin and transferrin saturation), even in the absence of symptoms.

**Genetic Testing of an Individual with a Family History of Hereditary Hemochromatosis**

The 2011 practice guidelines from AASLD recommended screening (iron studies [serum ferritin and transferrin saturation] and *HFE* variant analysis) of first-degree relatives of patients with *HFE*-related hereditary hemochromatosis to detect early disease and prevent complications. For children of an identified proband, *HFE* testing of the other parent is generally recommended because, if results are normal, the child is an obligate heterozygote and need not undergo further testing because there is no increased risk of iron overload.

If C282Y homozygosity or compound heterozygosity is found in adult relatives of a proband, and if serum ferritin levels are increased, then therapeutic phlebotomy can be initiated. If ferritin level is normal in these patients, then yearly follow-up with iron studies is indicated. When identified, individuals with C282Y heterozygotes and H63D heterozygotes can be reassured that they are not at risk for developing progressive or symptomatic iron overload. Some individuals with H63D homozygotes can develop mild iron overload.

**GENETICS NOMENCLATURE UPDATE**

Human Genome Variation Society (HGVS) nomenclature is used to report information on variants found in DNA and serves as an international standard in DNA diagnostics. It is being implemented for genetic testing medical evidence review updates starting in 2017 (see Table PG1). HGVS nomenclature is recommended by HGVS, the Human Variome Project, and the HUmAn Genome Organization (HUGO).

The American College of Medical Genetics and Genomics (ACMG) and Association for Molecular Pathology (AMP) standards and guidelines for interpretation of sequence variants represent expert opinion from ACMG, AMP, and the College of American Pathologists. These recommendations primarily apply to genetic tests used in clinical laboratories, including genotyping, single genes, panels, exomes, and genomes. Table PG2 shows the recommended standard terminology - “pathogenic,” “likely pathogenic,” “uncertain significance,” “likely benign,” and “benign” - to describe variants identified that cause Mendelian disorders.

**Table PG1. Nomenclature to Report on Variants Found in DNA**

<b>Previous</b>	<b>Updated</b>	<b>Definition</b>
<b>Mutation</b>	Disease-associated variant	Disease-associated change in the DNA sequence
	Variant	Change in the DNA sequence
	Familial variant	Disease-associated variant identified in a proband for use in subsequent targeted genetic testing in first-degree relatives

**Table PG2. ACMG-AMP Standards and Guidelines for Variant Classification**

<b>Variant Classification</b>	<b>Definition</b>
<b>Pathogenic</b>	Disease-causing change in the DNA sequence
<b>Likely pathogenic</b>	Likely disease-causing change in the DNA sequence

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<b>Variant of uncertain significance</b>	Change in DNA sequence with uncertain effects on disease
<b>Likely benign</b>	Likely benign change in the DNA sequence
<b>Benign</b>	Benign change in the DNA sequence

ACMG: American College of Medical Genetics and Genomics; AMP: Association for Molecular Pathology.

**GENETIC COUNSELING**

Experts recommend formal genetic counseling for patients who are at risk for inherited disorders and who wish to undergo genetic testing. Interpreting the results of genetic tests and understanding risk factors can be difficult for some patients; genetic counseling helps individuals understand the impact of genetic testing, including the possible effects the test results could have on the individual or their family members. It should be noted that genetic counseling may alter the utilization of genetic testing substantially and may reduce inappropriate testing; further, genetic counseling should be performed by an individual with experience and expertise in genetic medicine and genetic testing methods.

**II. PRODUCT VARIATIONS**

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This policy is only applicable to certain programs and products administered by Capital BlueCross and subject to benefit variations as discussed in Section VI. Please see additional information below.

**FEP PPO:** Refer to FEP Medical Policy Manual MP-2.04.80 Genetic Testing for Hereditary Hemochromatosis. The FEP Medical Policy manual can be found at: [https://www.fepblue.org/benefit-plans/medical-policies-and-utilization-management-guidelines/medical-policies.](https://www.fepblue.org/benefit-plans/medical-policies-and-utilization-management-guidelines/medical-policies)

**III. DESCRIPTION/BACKGROUND**

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**Iron Overload Syndromes**

Iron overload syndromes may be hereditary, secondary to another disease (e.g., iron-loading anemias, parenteral iron overload, chronic liver disease, or dysmetabolic iron overload syndrome), or due to other miscellaneous conditions (e.g., neonatal iron overload, aceruloplasminemia, congenital atransferrinemia).

Iron overload, if untreated, can lead to secondary tissue damage in a wide range of organs resulting in chronic liver disease (hepatic fibrosis, cirrhosis, hepatocellular carcinoma), endocrine dysfunction (diabetes, hypogonadism), arthralgia or arthritis (typically involving the second and third metacarpophalangeal joints), and cardiomyopathy (with either symptomatic cardiac failure or arrhythmias).

**Hereditary Hemochromatosis**

Hereditary hemochromatosis (HH), an autosomal recessive disorder, is the most commonly identified genetic disorder in white people, with an estimated prevalence of 1 in 250. However, fully expressed disease with end-organ manifestations is seen in less than 10% of affected

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individuals. Factors that influence phenotypic expression of human hemochromatosis (*HFE*; high iron-related HH [i.e., the clinical appearance of iron overload]) are not clearly defined. Low clinical penetrance may be due to a complex interplay of genetic status and other factors such as age, sex, environmental influences, and comorbid diseases.

HH leads to inappropriate iron absorption from the intestine and progressive increase in intracellular iron concentrations. Untreated HH leads to premature death, usually by liver complications.

***Diagnosis***

Patients with hemochromatosis may present with nonspecific systemic symptoms or specific organ-related symptoms, or they may be asymptomatic. Clinical diagnosis of hemochromatosis is based on documentation of increased iron stores as demonstrated by abnormal serum iron indices, specifically elevated transferrin saturation and elevated serum ferritin concentration. Liver biopsy has been used to confirm diagnosis but is now generally limited to determining the degree of hepatic fibrosis and cirrhosis during disease management. Most patients with a diagnosis of hemochromatosis will exhibit a familial pattern, thereby confirming the diagnosis of HH. However, the familial pattern may not be obvious due to the large percentage of undiagnosed patients in some families, and further evaluation of family members may be required to establish whether a familial pattern is present.

General population screening for HH has been proposed because of the high prevalence of disease, absence of or nonspecific early clinical findings, specificity of findings once they appear, low cost of diagnosis and treatment, and high cost and low success rate of late diagnosis and treatment. However, because penetrance is low, and the natural history of asymptomatic individuals is unpredictable, support for population-based screening is lacking. A 2006 U.S. Preventive Services Task Force (USPSTF) review of the literature suggested that 38% to 50% of individuals with C282Y homozygotes may develop iron overload, with 10% to 33% eventually developing hemochromatosis-associated morbidity.<sup>1</sup> The American Academy of Family Physicians, Centers for Disease Control and Prevention, and USPSTF recommended against population-based general screening.

***Treatment***

Treatment to remove excess iron with serial phlebotomy is simple and effective, and if started before irreversible end-organ damage, restores normal life expectancy. While there has never been a randomized controlled trial comparing phlebotomy with no phlebotomy in the treatment of HH, there is evidence from nonrandomized studies that initiation of phlebotomy before the development of cirrhosis and/or diabetes will significantly reduce HH-associated morbidity and mortality.

***Genetics***

Most patients with HH have variants in the *HFE* gene, located on the short arm of chromosome 6. The *HFE* gene was identified and cloned in 1996. The most common variant in the *HFE* gene is C282Y, a missense variant that changes cysteine at position 282 in the HFE protein to

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tyrosine. Homozygosity for the C282Y variant is associated with 60% to 90% of all cases of HH. Additionally, 3% to 8% of affected individuals are heterozygous for this variant. Penetrance for elevated serum iron indices among C282Y homozygotes is variable. However, penetrance for characteristic clinical end points (i.e., end-organ damage) is quite low. There is no test that can predict whether an individual with a C282Y homozygote will develop clinical symptoms. A specific variant in *PCSK7*, which is associated with iron metabolism, has been investigated as a possible predictor of cirrhosis risk in HH patients homozygous for the *HFE* C282Y variant.

Another significant *HFE* variant is referred to as H63D, which changes histidine at position 63 to aspartic acid. Homozygosity for H63D is insufficient to cause clinically significant iron overload in the absence of modifying factors. However, compound heterozygosity for C282Y/H63D has been associated with increased hepatic iron concentrations; approximately 1% to 2% of patients with this genotype will develop clinical evidence of iron overload, usually in the presence of another liver disease.

The clinical significance of a third *HFE* variant, S65C (serine at position 65 changed to cysteine), appears to be minimal. This rare variant displays very low penetrance. Compound heterozygosity for C282Y/S65C may confer a low risk for mild HH. Individuals who are heterozygous for S65C and either the wild-type (normal) or H63D alleles do not seem to be at an increased risk for HH. Other variants in *HFE* and in non-*HFE* genes (e.g., transferrin receptor 2 [*TFR2*]) resulting in iron overload syndromes are rare.

1. *HFE*-related HH is now frequently identified by genetic testing in asymptomatic probands and in asymptomatic relatives of patients who are known to have the disease.<sup>2</sup> Therefore, a genetic diagnosis can be made in subjects who have not yet developed phenotypic expression; these subjects have a genetic susceptibility to developing iron overload but may never do so. A 2000 consensus conference of the European Association for the Study of Liver Diseases led to the recognition of different stages and progression of hemochromatosis.<sup>11</sup> These stages were defined as Stage 1: Patients with “genetic susceptibility” who have the genetic disorder but no increase in iron stores.
2. Stage 2: Patients who have the genetic disorder and phenotypic evidence of iron overload but no tissue or organ damage.
3. Stage 3: Patients who have the genetic disorder with iron overload and iron deposition to the degree that tissue and organ damage occur.

**Regulatory Status**

Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; laboratory-developed tests (LDTs) must meet the general regulatory standards of the Clinical Laboratory Improvement Amendments (CLIA). Laboratories that offer LDTs must be licensed by CLIA for high-complexity testing. To date, the U.S. Food and Drug Administration has chosen not to require any regulatory review of this test.

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**IV. RATIONALE**

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**Summary of Evidence**

For individuals who have abnormal iron indices or clinical signs of iron overload who receive genetic testing for the human hemochromatosis (*HFE*) gene, the evidence includes retrospective and prospective observational studies. Relevant outcomes are test accuracy, test validity, and change in disease status. These studies have established high analytic validity of genetic testing. Studies have demonstrated that current genetic testing detects the large majority of hereditary hemochromatosis (HH) disease, but that, among those with positive tests (HH homozygotes), penetrance for clinical disease is low. There is no direct evidence of the clinical utility of genetic testing, but, along with prior knowledge on the effectiveness of treatment for clinical iron overload, there is a strong chain of evidence that supports definitive genetic diagnosis of persons with early signs of HH. The evidence is sufficient to determine that the technology results in a meaningful improvement in the net health outcome.

For individuals who are asymptomatic with a first-degree relative with HH who receive genetic testing for *HFE*, the evidence includes retrospective and prospective observational studies. Relevant outcomes are test accuracy, test validity, and change in disease status. These studies have established high analytic validity of genetic testing. Studies have demonstrated that current genetic testing detects the large majority of HH disease, but that among those with positive tests (HH homozygotes), penetrance for clinical disease is low. There is no direct evidence of the clinical utility of genetic testing, but, along with prior knowledge on the effectiveness of treatment for clinical iron overload, there is a strong chain of evidence that supports definitive genetic diagnosis of persons who are first-degree relatives of persons with HH. The evidence is sufficient to determine that the technology results in a meaningful improvement in the net health outcome.

For individuals who are asymptomatic in the general population who are asymptomatic with no family history of hereditary hemochromatosis who receive genetic testing for *HFE*, the evidence includes observational studies of screening in population samples. Relevant outcomes are test accuracy, test validity, and change in disease status. These studies have established population prevalence of genetic HH, and serve as partial evidence to estimate penetrance of disease. The low prevalence of HH homozygosity in the general population and incomplete penetrance of clinical disease do not support a chain of evidence for clinical utility of genetic testing in an unselected population. The evidence is insufficient to determine the effects of the technology on health outcomes.

**V. DEFINITIONS**

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**FIRST-DEGREE RELATIVE** refers to a parent, sibling, or child.

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**VI. BENEFIT VARIATIONS**

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The existence of this medical policy does not mean that this service is a covered benefit under the member's health benefit plan. Benefit determinations should be based in all cases on the applicable health benefit plan language. Medical policies do not constitute a description of benefits. A member's health benefit plan governs which services are covered, which are excluded, which are subject to benefit limits and which require preauthorization. There are different benefit plan designs in each product administered by Capital BlueCross. Members and providers should consult the member's health benefit plan for information or contact Capital BlueCross for benefit information.

**VII. DISCLAIMER**

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*Capital BlueCross's medical policies are developed to assist in administering a member's benefits, do not constitute medical advice and are subject to change. Treating providers are solely responsible for medical advice and treatment of members. Members should discuss any medical policy related to their coverage or condition with their provider and consult their benefit information to determine if the service is covered. If there is a discrepancy between this medical policy and a member's benefit information, the benefit information will govern. If a provider or a member has a question concerning the application of this medical policy to a specific member's plan of benefits, please contact Capital BlueCross' Provider Services or Member Services. Capital BlueCross considers the information contained in this medical policy to be proprietary and it may only be disseminated as permitted by law*

**VIII. CODING INFORMATION**

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**Note:** This list of codes may not be all-inclusive, and codes are subject to change at any time. The identification of a code in this section does not denote coverage as coverage is determined by the terms of member benefit information. In addition, not all covered services are eligible for separate reimbursement.

**Covered when medically necessary:**

CPT Codes ®							
81256							

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ICD-10-CM Diagnosis Code	Description
E83.10	Disorder of iron metabolism, unspecified
E83.110	Hereditary hemochromatosis
E83.111	Hemochromatosis due to repeated red blood cell transfusions
E83.118	Other hemochromatosis
E83.119	Hemochromatosis, unspecified

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E83.19	Other disorders of iron metabolism
R79.0	Abnormal level of blood mineral
Z83.49	Family history of other endocrine, nutritional and metabolic diseases

**IX. REFERENCES**

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1. Whitlock EP, Garlitz BA, Harris EL, et al. Screening for hereditary hemochromatosis: a systematic review for the U.S. Preventive Services Task Force. *Ann Intern Med.* Aug 01 2006; 145(3): 209-23. PMID 16880463
2. Bacon BR, Adams PC, Kowdley KV, et al. Diagnosis and management of hemochromatosis: 2011 practice guideline by the American Association for the Study of Liver Diseases. *Hepatology.* Jul 2011; 54(1): 328-43. PMID 21452290
3. Adams PC, Speechley M, Kertesz AE. Long-term survival analysis in hereditary hemochromatosis. *Gastroenterology.* Aug 1991; 101(2): 368-72. PMID 2065912
4. Niederau C, Fischer R, Purschel A, et al. Long-term survival in patients with hereditary hemochromatosis. *Gastroenterology.* Apr 1996; 110(4): 1107-19. PMID 8613000
5. Stickel F, Buch S, Zoller H, et al. Evaluation of genome-wide loci of iron metabolism in hereditary hemochromatosis identifies PCSK7 as a host risk factor of liver cirrhosis. *Hum Mol Genet.* Jul 15 2014; 23(14): 3883-90. PMID 24556216
6. Kanwar P, Kowdley KV. Metal storage disorders: Wilson disease and hemochromatosis. *Med Clin North Am.* Jan 2014; 98(1): 87-102. PMID 24266916
7. Sood R, Bakashi R, Hegade VS, et al. Diagnosis and management of hereditary haemochromatosis. *Br J Gen Pract.* Jun 2013; 63(611): 331-2. PMID 23735405
8. Vujic M. Molecular basis of HFE-hemochromatosis. *Front Pharmacol.* 2014; 5: 42. PMID 24653703
9. Radio FC, Majore S, Binni F, et al. TFR2-related hereditary hemochromatosis as a frequent cause of primary iron overload in patients from Central-Southern Italy. *Blood Cells Mol Dis.* Feb-Mar 2014; 52(2-3): 83-7. PMID 24055163
10. Ekanayake D, Roddick C, Powell LW. Recent advances in hemochromatosis: a 2015 update : a summary of proceedings of the 2014 conference held under the auspices of Hemochromatosis Australia. *Hepatology Int.* Apr 2015; 9(2): 174-82. PMID 25788196
11. Adams P, Brissot P, Powell LW. EASL International Consensus Conference on Haemochromatosis. *J Hepatol.* Sep 2000; 33(3): 485-504. PMID 11020008
12. Blue Cross and Blue Shield Association Technology Evaluation Center (TEC). Genetic testing for HFE gene mutations related to hereditary hemochromatosis. *TEC Assessments.* Dec 6 2001; Volume 16:Tab 22. PMID
13. Bryant J, Cooper K, Picot J, et al. A systematic review of the clinical validity and clinical utility of DNA testing for hereditary haemochromatosis type 1 in at-risk populations. *J Med Genet.* Aug 2008; 45(8): 513-8. PMID 18310265



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14. Eckerstrom C, Frandberg S, Lyxe L, et al. Evaluation of a screening program for iron overload and HFE mutations in 50,493 blood donors. *Ann Hematol.* Oct 2020; 99(10): 2295-2301. PMID 32844323
15. Bulaj ZJ, Ajioka RS, Phillips JD, et al. Disease-related conditions in relatives of patients with hemochromatosis. *N Engl J Med.* Nov 23 2000; 343(21): 1529-35. PMID 11087882
16. McLaren GD, Gordeuk VR. Hereditary hemochromatosis: insights from the Hemochromatosis and Iron Overload Screening (HEIRS) Study. *Hematology Am Soc Hematol Educ Program.* 2009: 195-206. PMID 20008199
17. Kowdley KV, Brown KE, Ahn J, et al. ACG Clinical Guideline: Hereditary Hemochromatosis. *Am J Gastroenterol.* Aug 2019; 114(8): 1202-1218. PMID 31335359
18. U.S. Preventive Services Task Force (USPSTF). Hemochromatosis: Screening (inactive). 2006; <https://www.uspreventiveservicestaskforce.org/BrowseRec/InactiveTopic/219>. Accessed June 4, 2021.
19. Blue Cross Blue Shield Association Medical Policy Reference Manual. 2.04.80, Genetic Testing for Hereditary Hemochromatosis June 2021.

**X. POLICY HISTORY**

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<b>MP 2.312</b>	<b>CAC 8/28/12</b> New Policy. Adopting BCBSA. Previously silent on this testing. New medically necessary statement for a patient with abnormal serum iron indices indicating iron overload or with a family history of hemochromatosis in a first-degree relative. Investigational for screening in the general population. Policy guidelines added.
	<b>CAC 7/30/13</b> Consensus review. References updated but no change to policy statements. FEP variation revised to refer to the FEP manual. Admin code review completed.
	<b>CAC 3/25/14</b> Consensus review. References updated but no change to the policy statements. Rationale added. Codes reviewed.
	<b>CAC 3/24/15</b> Consensus review. Added Medicare variation referencing LCD L3360 Biomarkers Overview. Updated rationale and reference list. No change to policy statements. Policy coded.
	<b>11/2/15</b> Administrative change. LCD number changed from L33640 to L35062 due to Novitas update to ICD-10.
	<b>CAC 3/29/16</b> Consensus review. No changes to the policy statements. References and rationale updated. Coding reviewed.
	<b>1/1/17 Administrative update.</b> Variations reformatted.
	<b>CAC 5/23/17</b> Consensus review. No change to policy statements. References and rationale updated. Coding Reviewed.
	<b>1/1/18 Admin Update:</b> Medicare variations removed from Commercial Policies.
	<b>2/7/18</b> Consensus review. The policy is revised with updated genetics nomenclature. “Mutations” changed to “variants” in policy statements. Policy statements otherwise unchanged.

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<b>2/1/2019</b> Consensus review. Condensed rational. Updated references. No changes to policy statement.
<b>4/2/2019</b> Consensus coding Review. No changes made.
<b>2/21/2020</b> Consensus review. No change to policy statement. Coding reviewed.
<b>6/4/2021 Consensus Review.</b> No change to policy statement. References and policy guidelines updated and coding reviewed.

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