



Fetal Red Blood Cell Antigen Genotyping Using Maternal Plasma

Effective: October 1, 2026

Next Review: June 2027

Last Review: August 2026

IMPORTANT REMINDER

Medical Policies are developed to provide guidance for members and providers regarding coverage in accordance with contract terms. Benefit determinations are based in all cases on the applicable contract language. To the extent there may be any conflict between the Medical Policy and contract language, the contract language takes precedence.

PLEASE NOTE: Contracts exclude from coverage, among other things, services or procedures that are considered investigational or cosmetic. Providers may bill members for services or procedures that are considered investigational or cosmetic. Providers are encouraged to inform members before rendering such services that the members are likely to be financially responsible for the cost of these services.

DESCRIPTION

The use of cell-free fetal DNA in maternal blood has been proposed as a noninvasive method to determine fetal red blood cell antigen genotypes, including *RHD* genotype.

MEDICAL POLICY CRITERIA

Notes:

- Combination cell-free fetal DNA tests that may include RhD testing are addressed in a separate policy, see Cross References below.
- Clinical documentation to support criteria is required (See Required Documentation)

Medically Necessary

- I. Fetal RhD antigen genotyping using cell-free DNA may be considered **medically necessary** when all of the following criteria are met (A. – C.):
 - A. Pregnancy may be at risk for alloimmunization due to maternal RhD negative status or the presence of maternal red cell antigen antibodies; and
 - B. Paternal antigen typing is unavailable or heterozygous; and

- C. Invasive testing (i.e., amniocentesis or chorionic villus sampling) is declined or contraindicated.

Not Medically Necessary

- II. Fetal RhD antigen genotyping using cell-free DNA is considered **not medically necessary** when Criterion I. is not met.

Investigational

- III. Testing for other, non-RhD, fetal red blood cell antigens, including but not limited to Fy^a (Duffy), K (Kell), C, c, and E antigens, using cell-free DNA is considered **investigational**.

NOTE: A summary of the supporting rationale for the policy criteria is at the end of the policy.

REQUIRED DOCUMENTATION

SUBMISSION OF GENETIC TESTING DOCUMENTATION

All of the following information must be submitted for review prior to the genetic testing:

- Name of genetic test and performing laboratory
- Relevant billing codes
- Date of sample collection/blood draw
- Clinical documentation to support rationale for testing, including:
 - Alloimmunization risk
 - Information on paternal antigen typing, and
 - Reason amniocentesis will not be performed.

CROSS REFERENCES

1. [Noninvasive Prenatal Testing to Determine Fetal Aneuploidies, Microdeletions, Single-Gene Disorders, and Twin Zygosity](#), Genetic Testing, Policy No 44

BACKGROUND

During pregnancy, antigen-negative individuals who are exposed to antigen-positive red blood cells (RBCs) can develop specific antibodies against those antigens, which can cross the placenta and cause fetal anemia. If undiagnosed and untreated, alloimmunization can cause significant perinatal morbidity and mortality. Determining the antigen status, particularly the Rh status, of the fetus may guide subsequent management of the pregnancy. The use of cell-free fetal DNA in maternal blood has been proposed as a noninvasive method to determine fetal antigen genotype.

RED BLOOD CELL ANTIGENS

The surface of RBCs are covered with antigen molecules. These include the standard blood group antigens associated with ABO blood types, Rh antigens, and many others. These antigens can stimulate an immune response in individuals who do not produce the same antigens.

The (Rhesus) Rh system includes more than 100 antigen varieties found on RBCs. RhD is the most common and the most immunogenic. When people have the RhD antigen on their RBCs, they are considered RhD-positive; if their RBCs lack the antigen, they are considered RhD-negative. The RhD-antigen is inherited in an autosomal dominant fashion, and a person may be heterozygous (Dd) (~60% of Rh-positive people) or homozygous (DD) (approximately 40% of Rh-positive people). Homozygotes always pass the RhD antigen to their offspring, whereas heterozygotes have a 50% chance of passing the antigen to their offspring. A person who is RhD-negative does not have the Rh antigen. Although nomenclature refers to RhD-negative as dd, there is no small d antigen (i.e., they lack the *RHD* gene and the corresponding RhD antigen).

RhD-negative status varies among ethnic groups and is 15% in Whites, 5 to 8% in African Americans, 5% to 8%, and 1% to 2% in Asians and Native Americans, respectively.

In the White population, almost all RhD-negative individuals are homozygous for a deletion of the *RHD* gene. However, in the African-American population, only 18% of RhD-negative individuals are homozygous for an *RHD* deletion, and 66% of RhD-negative African Americans have an inactive *RHD* pseudogene (*RHDψ*).^[1] There are also numerous rare variants of the D antigen, which are recognized by weakness of expression of D and/or by absence of some of the epitopes of D. Some individuals with variant D antigens can make antibodies to one or more epitopes of the D antigen, if exposed to RhD-positive RBCs. In addition to *RHD* and *RHDψ* variants, variants in the homologous gene *RHCE* can produce C and E antigens. Other RBC antigens families include the Duffy, Kell, Kidd, Lewis antigens.

ALLOIMMUNIZATION

Alloimmunization refers to the development of antibodies in a patient whose blood cells are antigen-negative and who is exposed to antigen-positive red blood cells (RBCs). This most commonly occurs from fetal-placental hemorrhage and entry of fetal blood cells into the maternal circulation.

The management of a Rh-negative pregnant patient who is not alloimmunized and is carrying a known Rh-positive fetus or the fetal Rh status is unknown, involves administration of Rh immune globulin at standardized times during the pregnancy to prevent the formation of anti-Rh antibodies. If the patient is already alloimmunized, management involves monitoring the levels of anti-Rh antibody titers for the development of fetal anemia. Both noninvasive and invasive tests to determine fetal Rh status exist.

By 30 days of gestation, the RhD antigen is expressed on the red blood cell (RBC) membrane, and alloimmunization can occur when fetal Rh-positive RBCs enter maternal circulation, and the Rh-negative mother develops anti-D antibodies.^[2] Once anti-D antibodies are present in a pregnant woman's circulation, they can cross the placenta and cause destruction of fetal RBCs.

The production of anti-D antibodies in RhD-negative women is highly variable and significantly affected by several factors, including the volume of fetomaternal hemorrhage, the degree of the maternal immune response, concurrent ABO incompatibility, and fetal homozygosity versus heterozygosity for the D antigen. Therefore, although ~10% of pregnancies are Rh-incompatible, <20% of Rh-incompatible pregnancies actually lead to maternal alloimmunization.

Small fetomaternal hemorrhages of RhD-positive fetal RBCs into the circulation of an RhD-negative woman occurs in nearly all pregnancies, and incidence of fetomaternal hemorrhage increases as the pregnancy progresses: 7% in the first trimester, 16% in the second trimester, and 29% in the third trimester, with the greatest risk of RhD alloimmunization occurring at birth (15% to 50%). Transplacental hemorrhage accounts for almost all cases of maternal RhD alloimmunization.

Fetomaternal hemorrhage can also be associated with miscarriage, pregnancy termination, ectopic pregnancy, invasive in-utero procedures (e.g., amniocentesis), in utero fetal death, maternal abdominal trauma, antepartum maternal hemorrhage, and external cephalic version. Other causes of alloimmunization include inadvertent transfusion of RhD-positive blood and RhD-mismatched allogeneic hematopoietic stem-cell transplantation.

There are other antigens on RBCs in addition to RhD, including the Duffy (Fy^a, Fy^b) and Kell antigens, that can lead to alloimmunization, but these are much more rare.

Consequences of Alloimmunization

IgG antibody-mediated hemolysis of fetal RBCs, known as hemolytic disease of the fetus and newborn, varies in severity and can have a variety of manifestations. The anemia can range from mild to severe with associated hyperbilirubinemia and jaundice. In severe cases, hemolysis may lead to extramedullary hematopoiesis and reticuloendothelial clearance of fetal RBCs, which may result in hepatosplenomegaly, decreased liver function, hypoproteinemia, ascites, and anasarca. When accompanied by high-output cardiac failure and pericardial effusion, this condition is known as hydrops fetalis, which without intervention, is often fatal. Intensive neonatal care, including emergent exchange transfusion, is required.

Cases of hemolysis in the newborn that do not result in fetal hydrops can still lead to kernicterus, a neurologic condition observed in infants with severe hyperbilirubinemia due to the deposition of unconjugated bilirubin in the brain. Symptoms that manifest several days after delivery can include poor feeding, inactivity, loss of the Moro reflex, bulging fontanelle, and seizures. The 10% of infants who survive may develop spastic choreoathetosis, deafness, and/or mental retardation.

The result of disease from alloimmunization, hemolytic disease of the fetus or newborn, was once a major contributor to perinatal morbidity and mortality. However, with the widespread adoption of antenatal and postpartum use of Rh immune globulin in developed countries, the result has been a major decrease in frequency of this disease. In developing countries without prophylaxis programs, stillbirth occurs in 14% of affected pregnancies, and 50% of pregnancy survivors either die in the neonatal period or develop cerebral injury.^[3]

Prevention of Alloimmunization

There are four currently in use Rh immune globulin products available in the U.S., all of which undergo micropore filtration to eliminate viral transmission.^[3] To date, no reported cases of viral infection related to Rh immune globulin administration have been reported in the U.S.^[3] Theoretically, the Creutzfeldt-Jakob disease (CJD) agent could be transmitted by the use of Rh immunoglobulin. Local adverse reactions may occur, including redness, swelling, and mild pain at the site of injection, and hypersensitivity reactions have been reported.

The American College of Obstetricians and Gynecologists (ACOG) and the American Association of Blood Banks (AABB) recommend the first dose of Rh_o(D) immune globulin (e.g.,

RhoGAM®) be given at 28 weeks' gestation, (or earlier if there's been an invasive event), followed by a postpartum dose given within 72 hours of delivery.

Diagnosis of Alloimmunization

The diagnosis of alloimmunization is based on detection of antibodies to specific RBC antigens in the maternal serum.

The most common test for determining antibodies in serum is the indirect Coombs test.^[2] Maternal serum is incubated with known RhD-positive RBCs. Any anti-RhD antibody that is present in the maternal serum will adhere to the RBCs. The RBCs are then washed and suspended in Coombs serum, which is antihuman globulin. RBCs coated with maternal anti-RhD will agglutinate, which is referred to as a positive indirect Coombs test. The indirect Coombs titer is the value used to direct management of pregnant alloimmunized women.

Management of Alloimmunization during Pregnancy

A patient's first alloimmunized pregnancy involves minimal fetal or neonatal disease. Subsequent pregnancies are associated with more severe degrees of fetal anemia. Treatment of an alloimmunized pregnancy requires monitoring of maternal anti-D antibody titers and serial ultrasound assessment of middle cerebral artery peak systolic velocity of the fetus.

If severe fetal anemia is present near term, delivery is performed. If severe anemia is detected remote from term, intrauterine fetal blood transfusions may be performed.

DETERMINING FETAL RHD STATUS

ACOG recommends that all pregnant women should be tested at the time of their first prenatal visit for ABO blood group typing and Rh-D type and be screened for the presence of anti-RBC antibodies. These laboratory tests should be repeated for each subsequent pregnancy. The AABB also recommends that antibody screening be repeated before administration of anti-D immune globulin at 28 weeks' gestation, postpartum, and at the time of any event during pregnancy.

If the mother is determined to be Rh-negative, the paternal Rh status should also be determined at the initial management of a pregnancy. If paternity is certain and the father is Rh-negative, the fetus will be Rh-negative, and further assessment and intervention are unnecessary. If the father is RhD-positive, he can be either homozygous or heterozygous for the D allele. If he is homozygous for the D allele (i.e., D/D) then the fetus is RhD-positive. If the paternal genotype is heterozygous for Rh status or is unknown, determination of the Rh-status of the fetus is the next step.

Invasive and noninvasive testing methods to determine the Rh status of a fetus are available.

Invasive procedures use polymerase chain reaction (PCR) assays to assess the fetal cellular elements in amniotic fluid by amniocentesis or by chorionic villus sampling (CVS). Although CVS can be performed earlier in a pregnancy, amniocentesis is the preferred method because CVS is associated with disruption of the villi and the potential for larger fetomaternal hemorrhage and worsening alloimmunization if the fetus is RhD-positive. The sensitivity and specificity of fetal *RHD* typing by PCR are reported as 98.7% and 100%, respectively, with positive and negative predictive values of 100% and 96.9%, respectively.^[4]

Noninvasive prenatal testing (NIPT) involves molecular analysis of cell-free fetal DNA (cffDNA) in the maternal plasma or serum. Lo (1998) showed that about 3% of cell-free DNA in the plasma of first trimester pregnant women is of fetal origin, with this percentage rising to 6% in the third trimester.^[5] Fetal DNA cannot be separated from maternal DNA, but if the pregnant woman is RhD-negative, the presence of specific exons of the *RHD* gene, which are not normally present in the circulation of an RhD-negative patient, predicts an RhD-positive fetus. Measurement of cffDNA has been proposed as an alternative to obtaining fetal tissue by invasive methods, which are associated with a risk of miscarriage.^[1]

The large quantity of maternal DNA compared to fetal DNA in the maternal circulation complicates the inclusion of satisfactory internal controls to test for successful amplification of fetal DNA. Therefore, reactions to detect Y chromosome-linked gene(s) can be included in the test, which will be positive when the fetus is a male.^[1] When Y chromosome-linked genes are not detected, tests for polymorphisms may be performed to determine whether the result is derived from fetal but not maternal DNA.

REGULATORY STATUS

There are currently no U.S. Food and Drug Administration (FDA)-cleared *RHD* genotyping tests. Thus, genotyping is offered as a laboratory-developed test. Clinical laboratories may develop and validate tests in-house and market them as a laboratory service; such tests must meet the general regulatory standards of the Clinical Laboratory Improvement Act (CLIA). The laboratory offering the service must be licensed by CLIA for high-complexity testing.

EVIDENCE SUMMARY

Human Genome Variation Society (HGVS) nomenclature^[6] is used to describe variants found in DNA and serves as an international standard. It is being implemented for genetic testing medical evidence review updates starting in 2017. According to this nomenclature, the term “variant” is used to describe a change in a DNA or protein sequence, replacing previously-used terms, such as “mutation.” Pathogenic variants are variants associated with disease, while benign variants are not. The majority of genetic changes have unknown effects on human health, and these are referred to as variants of uncertain significance.

Fetal *RHD* genotyping is best evaluated in the framework of a diagnostic test, as the test provides diagnostic information that assists in treatment decisions. Validation of the clinical use of any diagnostic test focuses on three main principles:

1. The analytic validity of the test, which refers to the technical accuracy of the test in detecting a mutation that is present or in excluding a mutation that is absent;
2. The clinical validity of the test, which refers to the diagnostic performance of the test (sensitivity, specificity, positive and negative predictive values) in detecting clinical disease; and
3. The clinical utility of the test, i.e., how the results of the diagnostic test will be used to change management of the patient and whether these changes in management lead to clinically important improvements in health outcomes.

This evidence review focuses on the clinical validity and utility of testing.

CLINICAL VALIDITY

For the evaluation of clinical validity, studies that reported on the accuracy of the marketed version of the technology, included a suitable reference standard, and described patient/sample clinical characteristics and selection criteria were considered for inclusion.

Systematic Reviews

Mustafa (2025) published a systematic review and meta-analysis of 84 cohort studies (77 prospective, seven retrospective) on the diagnostic accuracy of cffDNA for fetal *RHD* genotyping.^[7] Alloimmunized pregnancies were included in 10 studies, non-alloimmunized pregnancies were included in 66 studies, and a combination of alloimmunized and non-alloimmunized pregnancies were included in eight studies. A total of 77,187 fetal antigen samples were evaluated in the included studies. Several laboratory methods were used including polymerase chain reaction, next-generation sequencing, and matrix-assisted laser desorption/ionization-time of flight. Overall, the sensitivity and specificity of cffDNA were 99% and 99%, respectively. Sensitivity and specificity for each of the studied antigens (RhD, RhC, Rhc, RhE, Kell, and Duffy (Fya)) were similar to the overall cohort.

A systematic review and meta-analysis by Yang (2019) evaluated the diagnostic accuracy of high-throughput cffDNA testing to determine fetal RhD status.^[8] Study eligibility criteria for the review included a prospective cohort design, inclusion of women who were RhD-negative and not known to be sensitized, and the use of cord blood testing as a comparison standard. Eight studies were included, two of which were judged to be at high risk of bias. The results of the meta-analysis showed a false negative rate of 0.34% (95% confidence interval [CI] 0.15 to 0.76), and a false positive rate of 3.86% (95% CI 2.54 to 5.82) when inconclusive results were treated as positives, which dropped to 1.26% (95% CI 0.87 to 7.83) when inconclusive results were excluded.

Mackie (2017) published a systematic review and meta-analysis of studies on the diagnostic accuracy of cffDNA-based non-invasive prenatal testing.^[9] Thirty of the 117 included cohort studies in the analysis evaluated RhD status. The overall sensitivity and specificity were 99.3% and 98.4% respectively. Real-time PCR exhibited higher sensitivity when compared to conventional PCR. There was no difference in specificity. Ten of the 30 studies reported inconclusive results.

Zhu (2014) published a meta-analysis of studies on the diagnostic accuracy of noninvasive fetal *RHD* genotyping using cell-free fetal DNA.^[10] The investigators identified 37 studies conducted in RhD-negative pregnant women that were published by the end of 2013. The studies included a total of 11,129 samples, and 352 inconclusive samples were excluded. When all data were pooled, the sensitivity of fetal *RHD* genotyping was 99% and the specificity was 98%. Diagnostic accuracy was higher in samples collected in the first trimester (99.0%) than those collected in the second (98.3%) or third (96.4%) trimesters.

Nonrandomized Studies

A prospective study by Chitty (2014) was published evaluating the diagnostic accuracy of antenatal testing for fetal RhD status.^[11] Samples from 2,288 Rh-negative women who initiated prenatal care before 24 weeks of gestation were analyzed using *RHD* genotyping. Overall, the sensitivity of the test was 99.34% and the specificity was 94.91%. The likelihood of correctly detecting RhD status in the fetus increased with gestational age, with high levels of accuracy after 11 weeks. For example, for samples taken before 11 completed weeks of gestation, the sensitivity was 96.85% and the specificity was 94.40%, and at 14 to 17 weeks' gestation,

sensitivity was 99.67% and specificity was 95.34%. These findings of increased accuracy as pregnancies advanced differ from that of the Zhu (2014) meta-analysis, which found highest diagnostic accuracy in the first trimester.

A study published by Wikman (2012) reported the results of a prospective, population-based study involving 4,118 RhD-negative, non-alloimmunized pregnant women from 83 maternity care centers.^[12] Median gestational age was 10 weeks (range 3 to 40 weeks), with 75.5% of patients undergoing testing in the first trimester, 18.8% in the second, 4.3% in the third, and 1.4% unknown. Extracted DNA samples from each woman were analyzed in triplicate. Reanalysis had to be performed in 211 (5.1%) cases with inconclusive results in the first analysis. A positive or negative fetal RhD was reported for 96% of the samples, with 165 (4%) remaining inconclusive. A second sample was then obtained from 147 of the 165 pregnancies with inconclusive results: 14 (0.8%) remained inconclusive, all resulting from a weak or silent maternal *RHD* gene. Blood group serology of the newborns was used as the gold standard, and blood group serology results were missing for 466 pregnancies, leaving 3,652 newborns for whom the validity of *RHD* genotyping could be assessed. The false-negative rate (*RHD* genotyping was Rh-negative, but newborn was determined to be Rh-positive) was 55 of 2,297 (2.4%) and the false-positive rate (*RHD* genotyping was Rh-positive, but newborn was determined to be Rh-negative) was 15 of 1,355 (1.1%). After exclusion of the samples obtained before the eighth week of gestation, the false-negative rate was 23 of 2,073 (1.1%) and the false-positive rate was 14 of 1,218 (1.1%). Both sensitivity and specificity were close to 99% if the samples were not collected before gestational week eight. The authors note that a limitation of their study was the lack of a positive control for fetal DNA.

Other studies have replicated previous findings that fetal *RHD* genotyping can be accurately determined using cfDNA from maternal plasma, although not all Rh-positive fetuses are identified.^[13-20]

The Unity Fetal Antigen™ test, which assesses RhD, K1, Fy^a, C, c, and E antigens, demonstrated 100% sensitivity and specificity in a validation study in 1,683 clinical samples.^[21] Rego (2024) published a company-funded, prospective, validation study with samples from 156 patients with alloimmunized pregnancies.^[22] Of these, 15.4% were Hispanic, 9.0% were non-Hispanic Black, 65.4% were non-Hispanic White, 4.5% were Asian, and 1.3% had more than one race/ethnicity. The authors reported 100% concordance between NIPT test results and neonatal genotype obtained from buccal swabs for 465 antigen calls: K1 (n=143), E (n=124), C (n=60), Fy^a (n=50), c (n=47), and D (RhD) (n=41). Neonatal phenotype was not assessed.

CLINICAL UTILITY

No published data are identified showing that this type of testing leads to improved health outcomes. This type of testing could lead to the avoidance of the use of anti-D immune globulin (e.g., RhoGAM) in Rh-negative mothers with Rh-negative fetuses. However, the false negative rate of the test, while low, is not zero, and a certain percentage of Rh-negative women will develop alloimmunization to Rh-positive fetuses. Other issues that still need to be defined include the optimal timing of testing during the pregnancy.

A systematic review by Runkel (2020) evaluated the evidence for the benefit of cfDNA testing for fetal RhD status in RhD-negative pregnant women and reported a lack of studies investigating patient-relevant outcomes.^[23] They additionally performed a meta-analysis of diagnostic accuracy studies and reported a high sensitivity and specificity for the testing.

EVIDENCE SUMMARY

For individuals who are pregnant and have Rhesus D (RhD)-negative blood type who receive noninvasive *RHD* genotyping of the fetus using cell-free DNA from maternal plasma, the evidence for clinical validity includes two meta-analyses and additional prospective studies and one retrospective cohort for clinical utility. Relevant outcomes are test validity, morbid events, medication use, and treatment-related morbidity.

The clinical validity of fetal *RHD* genotyping is high, in that the test has shown a high degree of accuracy in correctly predicting fetal RhD status. However, the test does not identify all Rh-positive fetuses, which may lead to alloimmunization of the Rh-negative mothers in these cases. Evidence that demonstrates how the results from cffDNA analysis in maternal blood are used to alter treatment decisions and improve health outcomes compared to conventional testing are lacking. Therefore, the clinical utility of fetal *RHD* genotyping is unknown, and it is uncertain whether it will lead to improved health outcomes.

PRACTICE GUIDELINE SUMMARY

AMERICAN ASSOCIATION OF BLOOD BANKS (AABB)

AABB does not have specific practice guidelines or recommendations on the use of fetal *RHD* or other RBC antigen genotyping.

AMERICAN COLLEGE OF OBSTETRICIANS AND GYNECOLOGISTS (ACOG)

The American College of Obstetricians and Gynecologists Practice Bulletins 192 (2018, reaffirmed in 2024) and 181 (2017) address management and prevention of RhD alloimmunization, respectively.^[24, 25] The Bulletins note that although the detection of fetal RhD using molecular analysis of maternal plasma or serum can be assessed in the second trimester with an accuracy greater than 99%, it is not recommended nor widely used as a clinical tool.

The 2024 update on paternal and fetal genotyping for managing alloimmunization recommends fetal RhD antigen testing when the paternal genotype is heterozygous or unknown, and cffDNA is described as an "alternative tool" for patients who are unwilling to undergo amniocentesis.^[26]

A Practice Advisory statement on RhD immune globulin shortages (2024) stated that:^[27]

"Although current ACOG guidance does not recommend routine use of noninvasive prenatal testing (NIPT) to determine fetal Rh(D) status based on cost-effectiveness analyses, the use of NIPT to prioritize use of RhIg and conserve RhIg supply is a reasonable consideration in the practice setting that is experiencing RhIg shortages."

SUMMARY

More research is needed to know how well fetal red blood cell (RBC) antigen genotyping with maternal plasma works for improving health outcomes compared to current standard of care. However, clinical guidelines recommend fetal RBC antigen testing when the paternal genotype is heterozygous or unknown, and testing can be performed with cell-free fetal DNA when individuals are unwilling or unable to have an amniocentesis. Therefore, fetal *RHD*

genotyping using maternal plasma may be considered medically necessary when policy criteria are met. Fetal *RHD* genotyping using maternal plasma is not recommended in other situations, and is therefore considered not medically necessary when criteria are not met.

Evidence regarding cell-free fetal DNA testing for other RBC antigen genotypes (e.g., Fy^a (Duffy), K (Kell), C, c, and E antigens) is extremely limited and this testing is therefore considered investigational.

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CODES

Codes	Number	Description
CPT	0536U	Red blood cell antigen (fetal RhD), PCR analysis of exon 4 of RHD gene and housekeeping control gene GAPDH from whole blood in pregnant individuals at 10+ weeks gestation known to be RhD negative, reported as fetal RhD status
	81403	Molecular pathology procedure, Level 4 <i>RHD (Rh blood group, D antigen)</i> (eg, hemolytic disease of the fetus and newborn, Rh maternal/fetal compatibility), deletion analysis (eg, exons 4, 5 and 7, pseudogene), performed on cell-free fetal DNA in maternal blood (For human erythrocyte gene analysis of RHD, use a separate unit of 81403)
HCPCS	None	

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