**Title:** Genetic Testing for Cystic Fibrosis

**Description/Background**

Cystic fibrosis (CF) is a multisystem genetic disease that affects the body’s ability to move salt (chloride) and water in and out of cells. This causes tenacious mucus in the lungs, mucus plugs in the pancreas, and characteristically high sweat chloride levels. Symptoms vary and include very salty-tasting skin, persistent coughing, frequent lung infections, wheezing, shortness of breath, poor growth or weight gain in spite of good appetite, frequent greasy and/or bulky stools, difficulty with bowel movements and male infertility. Extensive lung damage and malnutrition can occur as a result.

There is no known cure for this progressive illness, but health problems can be managed. Treatment revolves around keeping the airway clear and maintaining adequate nutrition.

CF is an inherited autosomal recessive disorder. The CF transmembrane conductance regulator (CFTR) gene, which is mapped to chromosome 7, is responsible for making the protein that helps to move salt and water out of the cells. An individual must inherit two non-functioning copies of the CFTR gene, one from each parent, to have CF. If only one copy of the gene pair is mutated, the individual is a CF carrier. Carriers do not have the disease but can pass the defective copy of the mutated gene to their children. In Caucasians of European descent about 1 in 31 persons is a carrier.

When both parents carry the mutated gene:
- 1 in 4 children (25%) will have CF
- 1 in 2 children (50%) will be a carrier, but will not have CF
- 1 in 4 children (25%) will not be a carrier or have CF.

Genetic carrier testing for the CFTR gene is performed to predict the risk of disease, to identify carriers and to formulate prenatal diagnosis/prognosis.
The U.S. Food and Drug Administration (FDA) has issued recommendations and guidance on pharmacogenetic tests and genetic tests for heritable markers, as well as special controls guidance documents for some specific types of genetics tests, including CFTR gene mutation detection systems.

**Regulatory Status**

The FDA (2017) expanded the approved gene mutations from 10 to 33 for determining responsiveness of Kalydeco (ivacaftor) when used to treat cystic fibrosis. The list of cystic fibrosis transmembrane conductance regulator (CFTR) Gene Mutations that produce CFTR protein and are responsive to Kalydeco are as follows:

- A1067T
- A455E
- D110E
- D110H
- D1152H
- D1270N
- D579G
- E193K
- E56K
- F1052V
- F1074L
- G1069R
- G1244E
- G1349D
- G178R
- G551D
- G551S
- K1060T
- L206W
- P67L
- R1070W
- R117C
- R117H
- R347H
- R352Q
- R74W
- S1255P
- S549N
- S549R
- S945L
- S977F

Orkambi® (lumacaftor/ivacaftor) received FDA approval (2018) for the treatment of cystic fibrosis (CF) in patients age two years and older who are homozygous for the F508del mutation in the CFTR gene.

Symdeko (tezacaftor/ivacaftor) received FDA approval (2019) for treatment of pediatric patients ages 6 years and older with cystic fibrosis who have two copies of the most common type of mutation – F508del mutation; OR who have at least one of the mutations in the CFTR gene that is responsive to the active ingredients in Symdeko based on in vitro data and/or clinical evidence.

Trikafta™ (elexacaftor/ivacaftor/tezacaftor) received FDA approval (2019) for the treatment of cystic fibrosis in patients aged 12 years and older who have at least one F508del mutation in the CFTR gene. Trikafta is the first triple combination therapy available to treat patients with the most common cystic fibrosis mutation, which is estimated to represent 90% of the cystic fibrosis population.

**Medical Policy Statement**

The safety and effectiveness of genetic testing for cystic fibrosis have been established. Genetic testing may be considered a useful diagnostic tool when indicated and should be performed in conjunction with appropriate pre-and post-test genetic counseling.
Inclusionary and Exclusionary Guidelines (Clinically based guidelines that may support individual consideration and pre-authorization decisions)

Inclusions:
- Individuals planning pregnancy who have a family history of CF and the reproductive partners of those with CF.
- The prenatal population and those in the early stages of pregnancy when the test results will be used to make informed decisions regarding childbearing or a need for fetal diagnosis.
- Individuals, who have not undergone newborn screening, have an inconclusive sweat chloride test and there remains a suspicion of CF, when the results of the testing shall result in a definitive plan of patient management.
- Diagnostic testing in male infertility due to congenital bilateral absence of the vas deferens and carrier testing of their partners.
- Prenatal ultrasound findings that indicate an increased risk for CF (e.g. echogenic bowel or dilated loops of bowel)
- Gene mutation testing in patients with cystic fibrosis when used to guide medication regimens per FDA approved* indications

Genetic testing should be performed in conjunction with appropriate pre-and post-test genetic counseling.

*See Regulatory Status section above for more information

Exclusions:
- Complete analysis of the CFTR gene by DNA sequencing is not appropriate for routine carrier screening

CPT/HCPCS Level II Codes (Note: The inclusion of a code in this list is not a guarantee of coverage. Please refer to the medical policy statement to determine the status of a given procedure.)

Established codes:
81220  81221  81222  81223  81224  88299

Other codes (investigational, not medically necessary, etc.):
N/A

Note: The above code(s) may not be covered by all contracts or certificates. Please consult customer or provider inquiry resources at BCBSM or BCN to verify coverage.

Rationale

According to the Cystic Fibrosis Foundation Patient Registry, there are more than 1,700 known mutations of the CFTR gene. Somewhere in the proximity of 30,000 people in the United States and 70,000 people worldwide are living with CF. Approximately 1,000 new cases are diagnosed each year. CF is one of the most common genetic diseases in Caucasians, with an
incidence of about 1 in 2,500-3,500 American live births. Hispanics have a 1 in 4,000-10,000 incidence. CF occurs in African Americans in approximately 1 in 15,000-20,000 births. CF is rare in native Asians, estimated to occur in less than 1 in 100,000. There are more than ten million Americans that are unknowing, asymptomatic carriers of the CF gene mutations.

In 2010 it became mandatory for all newborns to be screened, via heel prick, for CF. Nearly two-thirds of individuals are diagnosed through newborn screening, 75% of individuals are diagnosed before the age of two.

Individuals may have severe pulmonary and/or gastrointestinal disease. Some have relatively mild disease and presentation during adolescence and young adulthood. Outcomes range from early death from pulmonary complications, mild atypical disease in the second and third decades and rare normal length of life. One-half of those with cystic fibrosis die by the age of thirty. Despite advances in treatment, there is currently no cure for CF. Ninety percent of persons with CF die from pulmonary complications. Major goals of traditional treatment of CF are to improve pulmonary, gastrointestinal and pancreatic outcomes.

Scotet et al (2020) reviewed the changes in the incidence and survival of CF and assessed the impact of the discovery of the responsible gene (the CFTR gene) on these changes. Authors concluded that significant advances in the management of cystic fibrosis in recent decades have dramatically changed the epidemiology and prognosis of this serious disease, which is no longer an exclusively pediatric disease. The incidence of CF appears to be decreasing in most countries and patient survival, which can be monitored by various indicators, has improved substantially, with an estimated median age of survival of approximately 50 years today. Cloning of the CFTR gene 30 years ago and efforts to identify its many mutations have greatly improved the management of CF. Implementation of genetic screening policies has enabled earlier diagnosis (via newborn screening), in addition to prevention within families or in the general population in some areas (via prenatal diagnosis, family testing or population carrier screening). In the past decade, in-depth knowledge of the molecular bases of CF has also enabled the emergence of CFTR modulator therapies which have led to major clinical advances in the treatment of CF. All of these phenomena have contributed to changing the face of CF. The advent of targeted therapies has paved the way for precision medicine and is expected to further improve survival in the coming years.

Cystic Fibrosis and Fetal Echogenic Bowel
Cystic fibrosis is associated with a condition known as fetal echogenic bowel, which refers to increased echogenicity or brightness of the fetal bowel noted on ultrasound examination. Echogenic bowel has been reported to be found in 50% to 78% of fetuses affected with CF. Intestinal echogenicity was initially described as a normal variant, but it has recently been associated with various fetal abnormalities, including CF. Ultrasound detection of fetal echogenic bowel is associated with an increased risk of CF in pregnancies otherwise at low risk for this disease.

In a 2002 study, Scotet et al reviewed prenatal testing (in Brittany, France, an area with one of the highest incidences of CF in the world) for CF in pregnancies which showed an echogenic bowel. Over a 10 year period (1991-2000), 346,554 pregnancies, which did not end with a spontaneous abortion, had an ultrasound examination. Echogenic bowel was diagnosed during the second trimester in 142 cases, which were referred for a prenatal test for CF. Of the 142 cases, 14 fetuses were identified as carrying two CFTR mutations. This implies an incidence of CF in this population of approximately 1 in 10 (9.9%). The authors concluded that these results
confirm that the ultrasound detection of fetal echogenic bowel is associated with an increased risk of CF in pregnancies otherwise at low risk.

Similarly, De Oronzo (2011) reported that in prospective evaluations of pregnancies at risk for CF, hyperechogenic bowel has been documented in up to 60% of affected fetuses. The report notes that “the association of echogenic bowel with fetuses affected with CF is thought to be caused by changes in the consistency of meconium in the small intestine as a result of abnormalities in pancreatic enzyme secretion. This can result in detectible sonographic findings, such as diffuse echogenic bowel, focal echogenic bowel with calcifications, a hyperechoic mass, or bowel dilation.” Additionally, “CF has been reported to affect 0.8% to 13.3% of fetuses with echogenic bowel, markedly higher than the rate of CF expected in a white population in which the carrier frequency is 1 in 25.”

**Cystic Fibrosis and Male Infertility**

In addition to the aforementioned complications of CF, male fertility is significantly reduced with CF. The condition known as congenital bilateral absence of the vas deferens (CBAVD) exists in 75-80% of the patients with associated defects in the CFTR gene, and 97-98% of males with CF are infertile due to CBAVD and resultant obstructive azoospermia. Males with CBAVD can have CFTR gene mutations, but they may have no other manifestations of CF.

Tomaiuolo et al (2011) evaluated the link between CFTR mutations and infertility by sequencing the CFTR gene in 294 subjects (190 males) affected by infertility. As a control group, they studied 1000 (353 males) unrelated, unselected subjects from the general population. The frequency of CFTR mutations was significantly higher in obstructive and secretory azoospermic patients than in the general population. Some patients, primarily those with CBAVD, were compound heterozygous for two mutations. The researchers concluded that “all subjects affected by obstructive or secretory azoospermia should undergo molecular analysis and counselling for CF. Molecular analysis seems to be less mandatory in other types of male/female infertility.” Furthermore, the researchers found that the CFTR TG12-T5-V470 variant haplotype was associated with both severe oligospermia and tubal infertility, implicating the CFTR protein in both spermatogenesis and tubal functionality.

In 2012, Chen et al reviewed a number of studies and clinical evidence over the past two decades which evaluate the roles of CFTR mutations and CBAVD as they relate to male infertility. The reviewers noted that the clinical evidence shows increased mutation frequency or reduced CFTR expression in men with CBAVD or sperm abnormalities, such as azoospermia teratospermia and oligoasthenospermia. Evidence also reveals a critical role of CFTR in sperm capacitation by directly or indirectly mediating HCO(3) entry that is essential for capacitation. The authors concluded that CFTR is a key regulator of male fertility, a defect of which may result in different forms of male infertility other than CBAVD.

Asadi et al (2018) investigated the frequency of the most common mutations of the CFTR gene (DF508, G542X, N1303K, G551D, and W1282X) in a population of infertile men with nonobstructive azoospermia (NOA) and CBAVD. Blood samples were obtained from 50 NOA, 50 CBAVD, and 100 normal males (control). Genomic DNA was isolated from whole blood leukocytes, and the presence of common mutations of the CFTR gene was assessed by an amplification refractory mutation system-polymerase chain reaction (ARMS-PCR). Restriction fragment length polymorphism (PCR-RFLP) was also used to analyze IVS8-Tn polymorphism. It was found that 16%, 8%, and 8% of patients with CBAVD were heterozygote for DF508, G542X, and N1303K, respectively. The frequency of the 5T allele was 34% and higher than
the normal group (p < 0.001). None of the common CFTR gene mutations were detected in NOA patients, and no significant difference was found in the distribution of the 5T allele between the NOA patients and the control group (5 vs. 3 p = 0.721). The authors concluded that the CFTR gene mutations and IS8-Tn polymorphisms are correlated with CBAVD; however, extensive investigations to determine the exact relationship between the gene mutations and other forms of male infertility were recommended.

Pharmacogenetics
Identification of the mutated CFTR gene allows for target therapy as follows:

- **Kalydeco™** is an orally self-administered CFTR potentiator. It is a unique medication that improves CF symptoms by facilitating increased chloride transport by potentiating the channel-open probability of the G551D-CFTR protein. It is the first treatment for CF patients that targets the underlying cause of CF.
- **Orkambi™** is a combination therapy of ivacaftor (Kalydeco) and lumacaftor (VX-809) for treatment of individuals age 12 and older who have two copies of the F508del mutation in their CFTR gene. The F508del mutation causes an abnormal protein to be produced that disrupts water and chloride transport in the body.
  - Lumacaftor is a cystic fibrosis transmembrane conductance regulator (CFTR) corrector, which helps CFTR protein reach the cell surface.
  - Kalydeco (ivacaftor), known as a CFTR potentiator, keeps the CFTR protein channels on the cell surface open longer to increase the flow of salt and water into and out of the cell.
- If the patients genotype is unknown, an FDA cleared CF mutation test should be used to detect the presence of the F508del mutation on both alleles of the CFTR gene.

Summary
Cystic fibrosis is a complex disease. Symptoms can vary significantly by type and severity (mild to severe) from one person to another. Some mutations have been associated with specific areas of the body including the lungs, pancreas, liver and other organs. Characterizing the gene mutation may help to identify which system is most likely to be affected and may assist in guiding appropriate treatment.

The severity of future symptoms cannot be fully predicted from an understanding of the specific gene mutation. However, screening can identify the presence of those mutations which are known to be associated with milder symptoms.

A family history of CF or CF carriers increases the chance of a person carrying a mutation that has the potential to be passed to their children. Carriers do not have symptoms, thus the CFTR mutation can be passed to offspring without anyone having the disease or a known family history. Unless a child is born with CF, many people who are carriers may not be aware of their ability to pass along the CFTR gene.

Genetic testing for cystic fibrosis plays a key role in allowing parents to make informed decisions regarding childbearing and guides health providers in determining effective treatment modalities. For childbearing, results of carrier testing provide the certainty of the predicted risk that offspring will be affected by a severe mutation for any of the disorders included in the panel. Such information can be used to make informed reproductive decisions such as preimplantation, genetic diagnosis, in vitro fertilization, not having a child, invasive prenatal testing, adoption, or pregnancy termination.
Supplemental Information

PRACTICE GUIDELINES AND POSITION STATEMENTS

The National Society of Genetic Counselors (2013) issued practice guidelines for molecular testing for CF carrier status. Some of the recommendations are as follows:

- Carrier testing for CF should be offered to all women of reproductive age, regardless of ancestry; preferably preconceptionally. CF carrier testing should also be offered to any individual with a family history of CF and to partners of mutation carriers and people with CF.
- Pre-test risk assessment should include an estimate of CF carrier frequency based on the individual's family history, ethnic background, and the predicted residual risk to have a child with CF if the test is negative.
- Carrier testing panels should include the mutations recommended by the American College of Obstetricians and Gynecologists' and the American College of Medical Genetics. For individuals of non-Northern European descent, pan-ethnic panels that include additional mutations more commonly identified in minority populations are appropriate to consider: Focus general population CF screening practices on identifying carriers of established disease-causing CFTR mutations.
- When both parents are known carriers for CF, available prenatal and pre-implantation diagnostic testing should be offered. Prenatal facilitation of a monitoring plan should begin for couples at risk or who continue a pregnancy known to have CF, and postnatal evaluation through sweat testing and state NBS programs, should be discussed.
- If a client is found to carry an R117H mutation, it is important to ensure the testing laboratory performs reflex testing for poly T status along with studies to determine the cis/trans orientation of the poly T alleles. In the absence of an R117H mutation, assessment of the intron 8 poly T or TG tracts is not recommended for routine CF carrier testing.

The National Institutes of Health Consensus Development Program genetic testing recommendations for CF are as follows:

- Individuals with a family history of CF and the reproductive partners of those with CF should be offered genetic testing.
- CF genetic testing should be offered to the prenatal population and couples currently planning a pregnancy, particularly those in high-risk populations.
- CF testing for the general population is not advocated.

The American College of Obstetricians and Gynecologists' Committee on Genetics (2017) updated current guidelines for cystic fibrosis screening practices. The committee made the following recommendations:

- Cystic fibrosis carrier screening should be offered to all women who are considering pregnancy or are currently pregnant.
- Complete analysis of the CFTR gene by DNA sequencing is not appropriate for routine carrier screening.
- For couples in which both partners are unaffected but one or both has a family history of CF, genetic counseling and medical record review should be performed to determine if CFTR mutation analysis in the affected family member is available.
• If a woman’s reproductive partner has CF or apparently isolated congenital bilateral absence of the vas deferens, the couple should be provided follow-up genetic counseling by an obstetrician-gynecologist or other health care provider with expertise in genetics for mutation analysis and consultation.

The EU project EuroGentest and the European Cystic Fibrosis Network (2009) issued recommendations and best practice guidelines for molecular genetic diagnosis of CF and CFTR-related disorders. Indications for CFTR testing were outlined:

• Diagnostic testing in typical CF presentation
• Diagnostic testing in atypical clinical presentation and/or borderline sweat test
• Diagnostic testing in male infertility with CBAVD
• Diagnostic testing in other CFTR-RD in adults
• Diagnostic testing in fetuses with bowel hyperechogenicity and/or loop dilatation
• Prenatal diagnosis
• CF carrier testing in individuals with a positive family history
• CF carrier screening in individuals without a family history
• CF carrier testing in infertile couples

**Government Regulations**

**National:**
National Medicare does not have a policy specifically addressing genetic testing for CF. Medicare’s indications for genetic testing are to rule out a constitutional or acquired chromosomal abnormality. Constitutional abnormalities refer to those present at birth, prenatally or postnatally. Acquired chromosome abnormalities refer to those that are typically acquired after birth by a subpopulation of cells that is involved in a premalignant or malignant condition. Medicare may review genetic testing for CF on a case-by-case basis.

**Local:**
No Local Determination specifically addressing genetic testing for CF is available.

Local Coverage Article: **Response to Comments: Molecular Pathology Procedure** (A55334) Original Effective Date: 12/01/16

Requests were received in support of coverage for CPT Codes 81220, 81221, 81222, 81223,81224 (cystic fibrosis transmembrane conductance regulator) (e.g.cystic fibrosis) gene analysis, common variants (e.g. ACMG/ACOG guidelines). NGS agrees and will provide coverage for a beneficiary who has or may have cystic fibrosis to guide therapeutic decision-making.

Local Coverage Article: **Billing and Coding: MolDX: Testing of Multiple Genes** (A57880); Effective date: 12/26/19

A panel of genes is a distinct procedural service from a series of individual genes. All services billed to Medicare must be reasonable and necessary. As such, if a provider or supplier submits a claim for a panel, then the patient’s medical record must reflect that the panel was reasonable and necessary. Alternatively, if a provider or supplier bills for a number of individual
genes, then the patient’s medical record must reflect that each individual gene is reasonable and necessary.

When more than two codes from this list are submitted for the same beneficiary on the same date of service, the claims processing system will reject every code submitted after the first two services. However, if a lab runs more than two distinct procedural services from this list on a single date of service, then the lab must use the 59 modifier with each additional service billed as an attestation that it is a distinct procedural service.

### Code Description

- **81220** CFTR gene com variants
- **81221** CFTR gene known fam variants
- **81222** CFTR gene dup/delet variants
- **81223** CFTR gene full sequence
- **81224** CFTR gene intron poly t

Local Coverage Article: **Billing and Coding: MolDX: Pharmacogenomics Testing** (A58395); Effective 7/26/20

Only one test may be performed per date of service; the test should be the most likely to identify the necessary alleles/variants for the drug/drugs in question. This applies to both single gene tests and multigene panels. Multigene panels can be performed when (as defined in the policy):

- More than one gene is reasonable and necessary for the safe use of the drug being considered or in use; or
- More than one drug is in consideration or use that is associated with a gene-drug interaction

A multigene panel must include all relevant genes and variants for its intended use to be reasonable and necessary.

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<td>Kalydeco</td>
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(The above Medicare information is current as of the review date for this policy. However, the coverage issues and policies maintained by the Centers for Medicare & Medicare Services [CMS, formerly HCFA] are updated and/or revised periodically. Therefore, the most current CMS information may not be contained in this document. For the most current information, the reader should contact an official Medicare source.)

### Related Policies

- Genetic Testing and Counseling
- Genetic Testing - Carrier Screening for Genetic Diseases
- Kalydeco™ (ivacaftor) (P&T Policy)
- Orkambi™ (lumacaftor/ivacaftor) (P&T policy)

### References
The articles reviewed in this research include those obtained in an Internet based literature search for relevant medical references through 12/3/20, the date the research was completed.
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Next Review Date: 1st Qtr, 2022
BLUE CARE NETWORK BENEFIT COVERAGE POLICY: GENETIC TESTING FOR CYSTIC FIBROSIS

I. Coverage Determination:

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II. Administrative Guidelines:

- The member's contract must be active at the time the service is rendered.
- Coverage is based on each member's certificate and is not guaranteed. Please consult the individual member’s certificate for details. Additional information regarding coverage or benefits may also be obtained through customer or provider inquiry services at BCN.
- The service must be authorized by the member's PCP except for Self-Referral Option (SRO) members seeking Tier 2 coverage.
- Services must be performed by a BCN-contracted provider, if available, except for Self-Referral Option (SRO) members seeking Tier 2 coverage.
- Payment is based on BCN payment rules, individual certificate and certificate riders.
- Appropriate copayments will apply. Refer to certificate and applicable riders for detailed information.
- CPT - HCPCS codes are used for descriptive purposes only and are not a guarantee