MEDICAL COVERAGE POLICY	
SERVICE:	**Exagamglogene autotemcel (Casgevy™)**
Policy Number:	**310**
Effective Date:	**6/1/2025**
Last Review:	**5/12/2025**
Next Review:	**5/12/2026**

Important note: *Unless otherwise indicated, medical policies will apply to all lines of business. Medical necessity as defined by this policy does not ensure the benefit is covered. This medical policy does not replace existing federal or state rules and regulations for the applicable service or supply. In the absence of a controlling federal or state coverage mandate, benefits are ultimately determined by the terms of the applicable benefit plan documents. See the member plan specific benefit plan document for a complete description of plan benefits, exclusions, limitations, and conditions of coverage. In the event of a discrepancy, the plan document always supersedes the information in this policy.*

SERVICE: Exagamglogene autotemcel (Casgevy™)

PRIOR AUTHORIZATION: Required

POLICY: Please review the plan's EOC (Evidence of Coverage) or Summary Plan Description (SPD) for details.

For Medicare plans, please refer to appropriate Medicare NCD (National Coverage Determination) or LCD (Local Coverage Determination). If there are no applicable NCD or LCD criteria, use the criteria set forth below.

For Medicaid plans, please confirm coverage as outlined in the Texas Medicaid Provider Procedures Manual | TMHP (TMPPM). Texas Mandate HB154 is applicable for Medicaid plans.

Baylor Scott & White Health Plan (BSWHP) may consider exagamglogene autotemcel (Casgevy™) medically necessary for the treatment of Sickle Cell Disease (SCD) or Transfusion-dependent β-thalassemia (TDT) when documentation is submitted showing ALL of the following universal criteria are met as well as criteria specific to each indication:

Universal Criteria Applied to All Requests

1. Exagamglogene is being prescribed by or in consultation with a board-certified hematologist; **AND**
2. Provider attests member will receive exagamglogene at an activated authorized treatment center; **AND**
3. Member is eligible for an autologous hematopoietic stem cell transplant (aHSCT); **AND**
4. Member does NOT have an available human leukocyte antigen (HLA)-matched related donor; **AND**
5. Member has NOT received any of the following:
    a. Hematopoietic stem cell transplant (HSCT)
    b. Exagamglogene or any other gene therapy
    c. Investigational cellular therapy

    AND
6. Exagamglogene will NOT be used concomitantly with other gene editing therapies; **AND**
7. Exagamglogene will be dosed and administered according to FDA approved labeling; **AND**
8. Member does NOT have any of the following:

a. White Blood Cell (WBC) count <3 × 10^9/L
b. Platelet count <50 × 10^9/L, not related to hypersplenism
c. Fetal hemoglobin (HbF) level >15%
d. Left ventricular ejection fraction (LVEF) <45% by echocardiogram
e. Baseline estimated glomerular filtration rate <60 mL/min/1.73 m2
f. Advanced liver disease, as defined by any one of the following:
    i. Alanine transaminase (ALT) >3 × the upper limit of normal (ULN)
    ii. Direct bilirubin value >2.5 × ULN
    iii. Baseline prothrombin time (INR >1.5 × ULN)
    iv. History of cirrhosis
    v. Any evidence of bridging fibrosis
    vi. Active hepatitis on liver biopsy
g. Clinically significant and active bacterial, viral, fungal, or parasitic infection
h. Any prior or current malignancy or myeloproliferative disorder
i. A significant immunodeficiency disorder
j. Positive for the presence of any of the following:
    i. Human immunodeficiency virus-1 (HIV-1) or Human immunodeficiency virus-2 (HIV-2) (positive antigen/antibody AND nucleic acid tests [NAT])
    ii. Hepatitis B virus (HBV) (positive Hepatitis B core antibody [HBcAb] AND NAT tests)
    iii. Hepatitis C virus (HCV; positive antibody [HCAb] AND NAT tests)
k. Intolerance, contraindication, or known sensitivity to plerixafor or busulfan
l. Prior anaphylactic reaction with excipients (dimethylsulfoxide [DMSO], dextran)
m. Pregnancy or breastfeeding
n. History of a significant bleeding disorder

Indication Specific Criteria

Sickle Cell Disease (SCD):

1. Member meets all universal criteria; **AND**
2. Member is 12 years of age or older, but less than or equal to 50 years of age; **AND**
3. Member has a Karnofsky performance status of ≥60% for subjects ≥ 16 years of age or Lansky performance status of ≥60% for subjects <16 years of age; **AND**
4. Member has a diagnosis of SCD with one of the following genotypes confirmed by molecular or genetic testing:
    a. β^S/β^S
    b. β^S/β^0
    c. β^S/β^+

    AND
5. Member has experienced hydroxyurea failure, intolerance, or has a contraindication; **AND**

6. Member has documented history of at least 2 severe vaso-occlusive episodes (sVOE) per year while receiving appropriate supportive care (e.g., pain management plan, hydroxyurea) during the previous two years

   Severe vaso-occlusive episode (sVOE) defined as any of the following:

   a. Acute pain events that required a visit to a medical facility and administration of pain medications (opioids or intravenous non-steroidal anti-inflammatory drugs) or RBC transfusions
   b. Acute chest syndrome, as indicated by the presence of a new pulmonary infiltrate associated with pneumonia-like symptoms, pain, or fever
   c. Priapism lasting >2 hours and requiring a visit to a medical facility
   d. Splenic sequestration, as defined by an enlarged spleen, left upper quadrant pain, and an acute decrease in hemoglobin concentration of ≥2 g/dL
   e. Acute hepatic sequestration, defined by a sudden increase in liver size associated with pain in the right upper quadrant, abnormal results of liver function test not due to biliary tract disease, and reduction in Hb concentration by ≥2 g/dL below the baseline value

AND

5. Member does not have a history of untreated Moyamoya disease or Moyamoya disease that puts the member at risk of bleeding

Transfusion-dependent β-thalassemia (TDT):

1. Member meets all universal criteria; **AND**
2. Member is 12 years of age or older, but less than or equal to 35 years of age; **AND**
3. Member has a Karnofsky performance status of ≥80% for subjects ≥ 16 years of age or Lansky performance status of ≥80% for subjects <16 years of age; **AND**
4. Member has a diagnosis of TDT with a non-β^0/β^0 or β^0/β^0 genotype confirmed by molecular or genetic testing (see Appendix A for examples); **AND**
5. Member has received at least 100 mL/kg/year or 10 units/year of packed red blood cells (pRBCs) during the previous two years

BSWHP considers only ONE treatment per lifetime medically necessary as repeat administration of exagamglogene autotemcel (Casgevy™) is experimental and investigational because the effectiveness of this strategy has not been established.

BSWHP considers exagamglogene autotemcel (Casgevy™) for the treatment of all other indications to be experimental and investigational because the effectiveness of this strategy has not been established.

All requests will be reviewed by a clinical pharmacist and medical director.

BACKGROUND:

Sickle Cell Disease Background

Sickle Cell Disease (SCD) is a single-gene disorder in which 1 DNA base-pair alteration in the gene coding for hemoglobin produces sickle hemoglobin (HbS) when inherited in an autosomal recessive fashion with a second HbS or when combined with other hemoglobin variants (e.g., HbC or β-thalassemia). When deoxygenated within capillary beds sickle hemoglobin forms long chains which distorts the red blood cell (RBC) into a sickle shape. Sickled RBCs have increased adhesion molecules compared to normal RBCs that facilitate binding to endothelial walls. In addition, Sickle cells hemolyze rapidly. Recurrent RBC sickling and hemolysis, combined with endovascular inflammation, result in acute and chronic organ damage at the cellular level, associated with acute, unpredictable, and potentially life-threatening complications.[1]

In the US, approximately 100,000 people have SCD. Children born in the US may be diagnosed shortly after birth through newborn screening programs. SCD is characterized by hemolytic anemia, acute and chronic pain, acute chest syndrome; increased incidence of stroke, nephropathy, and retinopathy; and a life span that is 20 years shorter than the general population.[1] A cure for SCD today is a stem cell transplant from a matched donor, but this option is only available to a small fraction of patients living with SCD because of the lack of available donors.[3]

Transfusion Dependent β-Thalassemia Background

Beta thalassemia is a hematologic disorder caused by a genetic defect in the *HBB* gene and characterized by ineffective erythropoiesis, which can lead to fewer red blood cells (RBCs) and severe anemia. It varies in severity, with some patients being dependent on regular RBC transfusions, known as transfusion-dependent beta thalassemia (TDT), and some being non–transfusion-dependent. Approximately 2000 patients in the United States are affected, 1500 of whom are transfusion-dependent. Beta thalassemia is most common in Mediterranean countries, North Africa, the Middle East, India, Central Asia, and Southeast Asia.[7]

There are three major categories of *HBB* mutations: β^0, no functional beta-globin production; β^+, reduced functional beta-globin production; and β^E, reduced functional beta-globin production and production of a variant Hb (HbE). While patients are broadly defined as having either β^0/β^0 or non-β^0/β^0 genotypes, patients with either genotype may be transfusion-dependent.[7]

Patients with TDT require transfusions every 2–5 weeks. They experience severe symptoms related to anemia (e.g. fatigue, weakness, shortness of breath) and iron overload from the transfusions that can also damage the heart, liver, and other endocrine organs. TDT can also cause ineffective erythropoiesis, which contributes to bone deformities and growth retardation in children. One study found that the median age of death for patients with TDT was 45 years (interquartile range [IQR]: 29–52 years) and the mean age was 43.9 years.[7,8]

At this time, HSCT with a human leukocyte antigen (HLA)-matched sibling donor is recommended for patients with TDT, as it is a potentially curative option. Overall disease-free survival is >85% in children

and 65% in adults. Both older age and presence of organ toxicity due to iron overload can negatively impact outcomes. Risks related to the stem cells include non-engraftment/graft failure, graft-versus-host disease (GVHD), and mortality. In addition, this treatment requires myeloablative therapy, which carries the risk of infertility and infections, and has a mortality risk as well. Because most patients do not have a suitable donor, there is a need for additional curative therapies.[7]

About Exagamglogene

Exagamglogene autotemcel (Casgevy™) is an autologous genome edited hematopoietic stem cell-based gene therapy indicated for the treatment of SCD in patients 12 years and older with recurrent vaso-occlusive crises (VOCs).[2]

Exagamglogene is a cellular gene therapy consisting of autologous CD34+ Hematopoietic Stem Cells (HSC) edited by CRISPR/CAs9-technology at the erythroid specific enhancer region of the BCL11A gene to reduce BCL11A expression in erythroid lineage cells, leading to increased fetal hemoglobin (HbF) protein production.[2]

Exagamglogene is prepared from the patient's own HSCs, which are obtained via apheresis procedure(s). The autologous cells are enriched for CD34+ cells, and then genome edited ex vivo by introducing the CRISPR/Cas9 ribonucleoprotein (RNP) complex by electroporation. The guide RNA included in the RNP complex enables CRISPR/Cas9 to make a precise DNA double-strand break at a critical transcription factor binding site (GATA1) in the erythroid specific enhancer region of the BCL11A gene. As a result of the editing, GATA1 binding is disrupted and BCL11A expression is reduced. This reduction in BCL11A expression conversely results in an increase in gamma-globin expression and downstream fetal hemoglobin formation.[2]

The edited CD34+ cells are formulated into a suspension in a sterile cryo-preservative medium and cryopreserved. Exagamglogene is shipped as a frozen suspension in patient-specific vial(s). The product is thawed prior to infusion, and administered as a HSC transplant.[2]

After Exagamglogene infusion, the edited CD34+ cells engraft in the bone marrow and differentiate to erythroid lineage cells with reduced BCL11A expression. Reduced BCL11A expression results in an increase in γ-globin expression and HbF protein production in erythroid cells. In patients with severe sickle cell disease, HbF expression reduces intracellular hemoglobin S (HbS) concentration, preventing the red blood cells from sickling and addressing the underlying cause of disease, thereby eliminating VOCs. In patients with transfusion-dependent β-thalassemia, γ-globin production improves the α-globin to non-α-globin imbalance thereby reducing ineffective erythropoiesis and hemolysis and increasing total hemoglobin levels, addressing the underlying cause of disease, and eliminating the dependence on regular red blood cell (RBC) transfusions.[2]

Sickle Cell Disease Clinical Studies

Trial 1 (NCT03745287, CLIMB-121) is an ongoing single-arm, multi-center trial evaluating the safety and efficacy of a single dose of Exagamglogene in adult and adolescent patients with sickle cell disease. Eligible patients underwent mobilization and apheresis to collect CD34+ stem cells for

Exagamglogene manufacture, followed by myeloablative conditioning and infusion of Exagamglogene. Patients were then followed in Trial 1 for 24 months after Exagamglogene infusion.[2]

At the time of the interim analysis, a total of 63 patients enrolled in the trial, of which 58 (92%) patients started mobilization. A total of 44 (76%) patients received Exagamglogene infusion and formed the full analysis set (FAS). Thirty-one patients from the FAS (70%) had adequate follow-up to allow evaluation of the primary efficacy endpoint and formed the primary efficacy set (PES).[2]

An interim analysis (IA) was conducted with 31 patients eligible for the primary efficacy analysis, i.e., the primary efficacy set (PES). The median (min, max) total duration of follow up was 19.3 (0.8, 48.1) months from the time of Exagamglogene infusion in FAS. There were no cases of graft failure or graft rejection.[2]

The primary efficacy outcome was the proportion of VF12 responders, defined as patients who did not experience any protocol-defined severe VOCs for at least 12 consecutive months within the first 24 months after Exagamglogene infusion in Trial 1. The proportion of patients who did not require hospitalization due to severe VOCs for at least 12 consecutive months within the 24-month evaluation period (HF12) was also assessed. The evaluation of VF12 and HF12 began 60 days after the last RBC transfusion for post-transplant support or SCD management. The median (min, max) time to the last RBC transfusion was 19 (11, 52) days following Exagamglogene infusion for patients in the primary efficacy set.[2]

The interim analysis occurred at the time when the alpha spending was approximately 0.02 for a one-sided test, when 31 patients were evaluable for VF12 responder status. The VF12 response rate was 29/31 (93.5%, 98% one-sided CI: 77.9%, 100.0%). The 29 VF12 responders did not experience protocol defined severe VOCs during the evaluation period with a median duration of 22.2 months at the time of the interim analysis. One VF12 responder, after initially achieving a VF12 response, experienced an acute pain episode meeting the definition of a severe VOC at Month 22.8 requiring a 5-day hospitalization; this patient was reported to have a parvovirus B19 infection at the time. Of the 31 patients evaluable for VF12 response, one patient was not evaluable for HF12 response; the remaining 30 patients (100% [98% one-sided CI: 87.8%, 100.0%]) achieved the endpoint of HF12.[2]

Transfusion-dependent β-thalassemia Clinical Studies

Trial 2 (NCT03655678) is an ongoing open-label, multi-center, single-arm trial to evaluate the safety and efficacy of Exagamglogene in adult and adolescent patients with transfusion-dependent β-thalassemia. Eligible patients underwent mobilization and apheresis to collect CD34+ stem cells for Exagamglogene manufacture, followed by myeloablative conditioning and infusion of Exagamglogene. Patients were then followed in Trial 2 for 24 months after Exagamglogene infusion. Patients who complete or discontinue from Trial 2 are encouraged to enroll in Trial 3 (NCT04208529), an ongoing long-term follow-up trial for additional follow-up for a total of 15 years after Exagamglogene infusion.[2]

At the time of the interim analysis, a total of 59 patients enrolled in the trial, of which 59 (100%) patients started mobilization. A total of 52 (88%) patients received Exagamglogene infusion and formed the full analysis set (FAS). Thirty-five patients from the FAS (67%) had adequate follow-up to allow evaluation

of the primary efficacy endpoint and formed the primary efficacy set (PES). The key demographics and baseline characteristics for all patients administered Exagamglogene in Trial 2 are shown in Table 10, below. The baseline characteristics and demographics are consistent between the PES and the FAS.[2]

An interim analysis (IA) was conducted with 35 patients eligible for the primary efficacy analysis, i.e., the primary efficacy set (PES). The median (min, max) total duration of follow-up was 23.8 (16.1, 48.1) months from the time of Exagamglogene infusion in the PES. There were no cases of graft failure or graft rejection.[2]

The primary outcome was the proportion of patients achieving transfusion independence for 12 consecutive months (TI12), defined as maintaining weighted average Hb ≥9 g/dL without RBC transfusions for at least 12 consecutive months any time within the first 24 months after Exagamglogene infusion in Trial 2, evaluated starting 60 days after the last RBC transfusion for post-transplant support or TDT disease management.[2]

The interim analysis occurred at the time when the alpha spending was approximately 0.017 for a one-sided test, when 35 patients were evaluable for TI12 responder status. The TI12 responder rate was 32/35 (91.4%, 98.3% one-sided CI: 75.7%, 100%). All patients who achieved TI12 remained transfusion-independent, with a median (min, max) duration of transfusion-independence of 20.8 (13.3, 45.1) months and normal mean weighted average total Hb levels (mean [SD] 13.1 [1.4] g/dL). The median (min, max) time to last RBC transfusion for patients who achieved TI12 was 30 (11, 91) days following Exagamglogene infusion. Three patients did not achieve TI12. These patients had reductions in annualized RBC transfusion volume requirements of 79.8%, 83.9% and 97.9%, and reductions in annualized transfusion frequency of 78.6%, 67.4% and 94.6%, respectively, compared to baseline requirements.[2]

CODES:

Important note: *Due to the wide range of applicable diagnosis codes and potential changes to codes, an inclusive list may not be presented, but the following codes may apply. Inclusion of a code in this section does not guarantee that it will be reimbursed, and patient must meet the criteria set forth in the policy language.*

CPT Codes:	96413 - Chemotherapy administration, intravenous infusion technique; up to 1 hour, single or initial substance/drug 96409 – Chemotherapy administration, intravenous, push technique, single or initial substance/drug	
HCPCS Codes:	J3392 - Injection, exagamglogene autotemcel, per treatment	
ICD10 codes:	D56.1	Beta thalassemia
	D57.00	Hb-Ss Disease With Crisis, Unspecified
	D57.01	Hb-Ss Disease With Acute Chest Syndrome
	D57.02	Hb-Ss Disease With Splenic Sequestration
	D57.03	Hb-Ss Disease With Cerebral Vascular Involvement
	D57.04	Hb-Ss Disease With Dactylitis
	D57.09	Hb-Ss Disease With Crisis With Other Specified Complication

	D57.1	Sickle-Cell Disease Without Crisis
	D57.20	Sickle-Cell/Hb-C Disease Without Crisis
	D57.211	Sickle-Cell/Hb-C Disease With Acute Chest Syndrome
	D57.212	Sickle-Cell/Hb-C Disease With Splenic Sequestration
	D57.213	Sickle-Cell/Hb-C Disease With Cerebral Vascular Involvement
	D57.214	Sickle-Cell/Hb-C Disease With Dactylitis
	D57.218	Sickle-Cell/Hb-C Disease With Crisis With Other Specified Complication
	D57.219	Sickle-Cell/Hb-C Disease With Crisis, Unspecified
	D57.40	Sickle-Cell Thalassemia Without Crisis
	D57.411	Sickle-Cell Thalassemia, Unspecified, With Acute Chest Syndrome
	D57.412	Sickle-Cell Thalassemia, Unspecified, With Splenic Sequestration
	D57.413	Sickle-Cell Thalassemia, Unspecified, With Cerebral Vascular Involvement
	D57.414	Sickle-Cell Thalassemia, Unspecified, With Dactylitis
	D57.418	Sickle-Cell Thalassemia, Unspecified, With Crisis With Other Specified Complication
	D57.419	Sickle-Cell Thalassemia, Unspecified, With Crisis
	D57.42	Sickle-Cell Thalassemia Beta Zero Without Crisis
	D57.431	Sickle-Cell Thalassemia Beta Zero With Acute Chest Syndrome
	D57.432	Sickle-Cell Thalassemia Beta Zero With Splenic Sequestration
	D57.433	Sickle-Cell Thalassemia Beta Zero With Cerebral Vascular Involvement
	D57.434	Sickle-Cell Thalassemia Beta Zero With Dactylitis
	D57.438	Sickle-Cell Thalassemia Beta Zero With Crisis With Other Specified Complication
	D57.439	Sickle-Cell Thalassemia Beta Zero With Crisis, Unspecified
	D57.44	Sickle-Cell Thalassemia Beta Plus Without Crisis
	D57.451	Sickle-Cell Thalassemia Beta Plus With Acute Chest Syndrome
	D57.452	Sickle-Cell Thalassemia Beta Plus With Splenic Sequestration
	D57.453	Sickle-Cell Thalassemia Beta Plus With Cerebral Vascular Involvement
	D57.454	Sickle-Cell Thalassemia Beta Plus With Dactylitis
	D57.458	Sickle-Cell Thalassemia Beta Plus With Crisis With Other Specified Complication
	D57.459	Sickle-Cell Thalassemia Beta Plus With Crisis, Unspecified
	D57.80	Other Sickle-Cell Disorders Without Crisis
	D57.811	Other Sickle-Cell Disorders With Acute Chest Syndrome
	D57.812	Other Sickle-Cell Disorders With Splenic Sequestration
	D57.813	Other Sickle-Cell Disorders With Cerebral Vascular Involvement
	D57.814	Other Sickle-Cell Disorders With Dactylitis
	D57.818	Other Sickle-Cell Disorders With Crisis With Other Specified Complication
	D57.819	Other Sickle-Cell Disorders With Crisis, Unspecified
ICD10 Not covered:		

POLICY HISTORY:

Status	Date	Action
New	6/10/2024	New Policy
Update	5/12/2025	Added TDT indication (FDA approved), Added TDT specific criteria, Added CPT code: 96409, Updated HCPCS code: J3590 to J3392, Added ICD10 code: D56.1, Updated treatment center criteria to attestation only, Updated Medicaid language to align with standard language, Updated all other indications note to align with standard language, Updated and reformatted

		background to include TDT, Reformatted to include "Universal Criteria" and "Indication Specific Criteria", Removed "for SCD" from universal criteria, Reformatted background, Updated references to AMA style
Update	8/11/2025	Removed, Medicare NCD/LCD Interqual statement for clarity.

Appendix A: Examples of non-β^0/β^0 Or β^0/β^0 genotypes[4,5]

1. β^0/β^0
2. β^0/β^+
3. β^E/β^0
4. β^E/β^+
5. β^+/β^+
6. $\beta^0/\beta^{+(IVS\text{-}I\text{-}110)}$
7. $\beta^{+(IVS\text{-}I\text{-}110)}/\beta^{+(IVS\text{-}I\text{-}110)}$

REFERENCES:

The following scientific references were utilized in the formulation of this medical policy. BSWHP will continue to review clinical evidence related to this policy and may modify it at a later date based upon the evolution of the published clinical evidence. Should additional scientific studies become available, and they are not included in the list, please forward the reference(s) to BSWHP so the information can be reviewed by the Medical Coverage Policy Committee (MCPC) and the Quality Improvement Committee (QIC) to determine if a modification of the policy is in order.

1. Kavanagh PL, Fasipe T, Wun T. Sickle cell disease. JAMA. 2022;328(1):57. doi:10.1001/jama.2022.10233
2. Food and Drug Administration. FDA label Casgevy™ (Exagamglogene autotemcel).
3. Vertex Pharmaceuticals Newsroom. Vertex and CRISPR Therapeutics announce US FDA approval of CASGEVY™ (exagamglogene autotemcel) for the treatment of sickle cell disease. https://news.vrtx.com/news-releases/news-release-details/vertex-and-crispr-therapeutics-announce-us-fda-approval. Accessed April 16, 2025.
4. Frangoul H, Altshuler D, Cappellini MD, et al. CRISPR-CAS9 gene editing for sickle cell disease and β-thalassemia. N Engl J Med. 2021;384(3):252-260. doi:10.1056/nejmoa2031054
5. Taher AT, Musallam KM, Cappellini MD. β-Thalassemias. N Engl J Med. 2021;384(8):727-743. doi:10.1056/NEJMra2021838
6. Locatelli F, Lang P, Wall D, et al. Exagamglogene autotemcel for transfusion-dependent β-thalassemia. N Engl J Med. 2024;390(18):1663-1676. doi:10.1056/NEJMoa2309673
7. IPD Analytics. Gene therapies for sickle cell disease and beta thalassemia. RxInsights: Hematology. 2023.
8. Jobanputra M, Paramore C, Laird SG, McGahan M, Telfer P. Co-morbidities and mortality associated with transfusion-dependent beta-thalassaemia in patients in England: A 10-year retrospective cohort analysis. Br J Haematol. 2020;191(2):262-270. doi:10.1111/bjh.17091

***Note**:*

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Health Maintenance Organization (HMO) products are offered through Scott and White Health Plan dba Baylor Scott & White Health Plan, and Scott & White Care Plans dba Baylor Scott & White Care Plan. Insured PPO and EPO products are offered through Baylor Scott & White Insurance Company. Scott and White Health Plan dba Baylor Scott & White Health Plan serves as a third-party administrator for self-funded employer-sponsored plans. Baylor Scott & White Care Plan and Baylor Scott & White Insurance Company are wholly owned subsidiaries of Scott and White Health Plan. These companies are referred to collectively in this document as Baylor Scott & White Health Plan.

RightCare STAR Medicaid is offered through Scott and White Health Plan in the Central Texas Medicaid Rural Service Area (MRSA); FirstCare STAR is offered through SHA LLC dba FirstCare Health Plans (FirstCare) in the Lubbock and West MRSAs; and FirstCare CHIP is offered through FirstCare in the Lubbock Service Area.