



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

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: Biochemical Markers of Alzheimer's Disease

PRIOR AUTHORIZATION: Not Applicable

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

Note: Unless otherwise indicated (see below), this policy will apply to all lines of business.

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). If there are no applicable criteria to guide medical necessity decision making in the TMPPM, use the criteria set forth below.

BSWHP considers the use of the following biomarkers, tests and measurements in the diagnosis and management of Alzheimer's Disease to be experimental and investigational and NOT medically necessary.

Biomarkers, tests and measurements not medically necessary include, but are not limited to:

- AB42 or AB42:AB40 ratio
- Apolipoprotein E
- ATP-binding cassette transporter
- Bcl-2 rs956572 polymorphism testing
- Beta amyloid 42 (BA-24, A β 42) protein (cerebrospinal fluid and plasma)
- Beta-site amyloid precursor protein cleaving enzyme
- Cerebrospinal fluid (CSF) chitinase enzyme activity
- Circadian rhythm analysis
- CSF microRNAs (e.g., hsa-miR-27a-3p)
- CSF neurogranin
- CSF phosphorylated tau at threonine 181 (ptau181), tau/A β 42, and ptau181/A β 42
- CSF prion protein concentration



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

- CSF soluble amyloid precursor proteins (sAPP) level/CSF β -secretase activity
- CSF stathmin protein level
- CSF total tau (t-tau)
- CSF visinin-like protein-1 (VILIP-1) level
- Cognitive event-related potentials (cognitive evoked potentials)
- DNA methylation profiling (brain tissue or peripheral blood)
- Electronystagmography (in the absence of signs of vertigo or balance disorder)
- Genetic testing (e.g., presenilin-1 gene [PSEN1], presenilin-2 gene [PSEN2], apolipoprotein E epsilon 4 allele, amyloid precursor gene, etc.)
- Genetic variation of mitochondrial DNA
- Homocysteine (serum level)
- INNO-BIA AlzBio3 immunoassay kit (a multiplex immunoassay that allows simultaneous quantification of amyloid-beta, p-tau, and t-tau)
- Insulin degrading enzyme polymorphisms
- Long-term measurement of cortisol
- Macular thickness
- Microtubule-associated protein tau (MAPT)
- N-terminal pro-brain natriuretic peptide (NT-proBNP)
- Olfactory screening tests
- Plasma clusterin level
- Plasma prion protein concentration
- Plasma tau
- Pituitary adenylate cyclase-activating polypeptide (PACAP)
- Red blood cell omega-3 fatty acid level 06/06/2018
- Resting state eye-closed cortical electroencephalography
- Serum ceramides
- Serum insulin-like growth factor-1 (IGF-1 also known as somatomedin C)
- Serum microRNAs
- Serum neurofilament light concentration
- Tau protein, total tau, phosphor-tau
- Transforming growth factor-beta1 (TGF- β 1)
- TREM2 (triggering receptor expressed on myeloid cells 2)
- Tympanometry (in the absence of hearing loss)
- Urinary AD7c-NTP (neuronal thread protein/neural thread protein)
- Videopupullography and tropicamide drop test



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

BACKGROUND:

Alzheimer's disease (AD) is an irreversible, progressive brain disorder that occurs gradually and results in memory loss, unusual behavior, personality changes, and a decline in thinking ability. The Alzheimer's Foundation of America (AFA) reports that there are between 4.2 and 5.8 million people affected with AD. The incidence of AD is expected to climb as the population ages. Additionally, the Alzheimer's Association (AA) reports that there are between 200,000 and 500,000 people with early onset disease occurring before the age of 65. The mean duration from the onset of clinical symptoms to the death of the patient has been reported to be approximately 8.5 years.

Currently the diagnosis of AD is a clinical diagnosis, focusing on the exclusion of other causes of senile dementia. The United States Preventative task force continues to support the use of clinical findings in diagnosis. Psycho-behavioral instruments such as the Mini-Mental State Examination (MMSE) and the Functional Activities Questionnaire are in current use. The MMSE has a sensitivity that ranges from 71 to 96 percent and a specificity range from 56 to 72 percent for dementia. The task force also concluded that current therapies, primarily medication, could only slow AD progression two to seven months and had limited effects on activities of daily living. The benefits of early screening will not be fully realized until better treatment modalities are developed.

In 1988, the National Institute of Neurological and Communicative Disorders and Stroke (NINCDS) and the Alzheimer's and Related Disorders Association (ADRDA) published clinical criteria for the diagnosis of AD. These organizations defined three categories: Possible AD, Probable AD and Definite AD.

The only difference between Probable and Definite AD is that the Definite AD category requires a brain biopsy confirming the presence of characteristic neurofibrillary tangles. While Definite AD is invariably only confirmed at autopsy, in approximately 85% of those with a diagnosis of Probable AD, pathological findings are found to be consistent.

The clinical criteria currently used for AD in patients with probable AD provide a sensitivity of approximately 85% when compared to autopsy confirmed cases (Definite AD). Therefore, a biomarker should have a sensitivity approaching or exceeding this value. A biomarker should have a specificity of 75% to 85% or greater, and the positive predictive value should be 80% or more.

There are currently no biomarkers that meet the above criteria. To date, all studies have focused on the use of biomarkers with Probable AD. The clinical utility of biomarkers may be greatest in patients with Possible AD, where the diagnosis is more uncertain. Few studies have focused on the use of biomarkers in patients with Possible AD with any follow up to determine the sensitivity and specificity of these markers in earlier stages of the disease.

The use of biomarkers will continue to be of interest to distinguish early AD from other causes of mild cognitive impairment, such as normal aging, vascular dementia or alcohol-related cognitive disorders. Research in patients with incipient AD is challenging because of the long follow-up required, and the



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

possibility that any control group will also have patients with unrecognized incipient AD. There are inadequate data to determine how the results of these tests could be used to improve patient management, particularly given the limited treatment options. If ongoing research developing drugs targeting early stages of AD comes to fruition, then biomarkers to identify treatment candidates may have an impact on patient management.

The Alzheimer's Association and the National Institute on Aging (NIA), an agency of the U.S. National Institutes of Health (NIH), agree there are currently no validated biomarkers for Alzheimer's disease. They have jointly issued four new criteria and guidelines to diagnose Alzheimer's disease. Three of the four new criteria and guidelines that came out as a result of the research that the Alzheimer's Association and the National Institute on Aging (NIA) completed in April 2011 focus on three stages of Alzheimer's disease: 1) dementia due to Alzheimer's, 2) mild cognitive impairment (MCI) due to Alzheimer's, and 3) preclinical (presymptomatic) Alzheimer's. The 4th guideline updates criteria for documenting and reporting Alzheimer's related changes observed during an autopsy.

According to the research completed by these two agencies in 2011 "In the future, biomarker evidence may provide additional diagnostic certainty, but much more research is needed to identify the most accurate biomarkers and confirm their usefulness. " Please see [http://www.alz.org/research/diagnostic_criteria/for more information](http://www.alz.org/research/diagnostic_criteria/for_more_information).

There are several widely investigated biomarkers (AB42 or AB42:AB40 ratio, total tau, phosphor-tau) for the molecular and degenerative process of AD that appear to be supportive of a diagnosis of AD but are not yet recommended as clinical tools to diagnose, predict or monitor the progress of AD.

MANDATES: None

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:		
CPT Not Covered:	For this indication: 83015, 83018, 83520, 83825	
HCPCS Codes:		
ICD-10 Codes:	F01.50 - F03.91	Dementia
	F07.0 - F07.9	Personality and behavioral disorders due to known physiological condition
	G30.0 - G30.9	Alzheimer's disease
	Z13.858	Encounter for screening for other nervous system disorders (screening for dementia)



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

POLICY HISTORY:

Status	Date	Action
New	12/6/2010	New policy
Reviewed	12/6/2011	Reviewed.
Reviewed	10/25/2012	Reviewed.
Reviewed	10/3/2013	Checked for updated Hayes rating.
Reviewed	07/24/2014	No changes
Reviewed	08/11/2015	No changes
Reviewed	08/18/2016	No changes
Reviewed	07/18/2017	Updated "Overview" section and coding.
Reviewed	05/29/2018	No changes
Reviewed	08/22/2019	Added list of studies not medically necessary
Reviewed	09/24/2020	Re-formatted for SWHP/FirstCare
Reviewed	09/23/2021	No changes
Reviewed	09/22/2022	No changes
Updated	12/29/2023	Updated Overview section with updated investigational biomarkers not yet recommended for clinical use. Formatting changes, added hyperlinks to NCD and TMPPM, beginning and ending note sections updated to align with CMS requirements and business entity changes.
Reviewed	01/13/2025	Ending note section updated to align with business entity changes.
Updated	08/11/2025	Removed, "Medicare NCD or LCD specific InterQual criteria may be used when available."

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 make modifications 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. AA – Definition of Alzheimer's (2007). Alzheimer's Foundation of America (accessed - 2007 June 1). Available at <http://www.alzfdn.org>.
2. Andreasen, N., Vanechelen, E., et al. Cerebrospinal fluid levels of total-tau, phosphor-tau and A beta 42 predicts development of Alzheimer's disease in patients with mild cognitive impairment. Acta Neurology Scandinavica Supplement (2003) 179:47-51.
3. Arai, H., Terajima, M., et al. Tau in cerebrospinal fluid: a potential diagnostic marker in Alzheimer's disease. Annals of Neurology (1995 October) 38(4):649-652



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

4. Attems, J., and K. Jellinger. Olfactory tau pathology in Alzheimer disease and mild cognitive impairment. *Clinical Neuropathology* (2006 November-December) 25(6):265- 71.
5. Attems, J., Lintner, F., et al. Olfactory involvement in aging and Alzheimer's disease: an autopsy study. *Alzheimer's Disease* (2005 April) 7(2):173-80.
6. Attems, J., and K. Jellinger. Only cerebral amyloid angiopathy correlates with Alzheimer pathology: A pilot study. *Acta Neuropathology* (2004 February) 107(2):83-90.
7. Blasko, I., Lederer, W., et al. Measurement of thirteen biological markers in CSF of patients with Alzheimer's disease and other dementias. *Dementia Geriatric Cognitive Disorder* (2006 October) 21(1):9-15
8. Bouwman, F.H., Schoonenboom, S.N., et al. CSF biomarkers and medial temporal lobe atrophy predict dementia in mild cognitive impairment. *Neurobiology of Aging* (2007 July) 28(7):1070-4.
9. Buerger, K., Zinkowski, R., et al. Differential diagnosis of Alzheimer disease with cerebrospinal fluid levels of tau protein phosphorylated at threonine 231. *Archives of Neurology* (2002 August) 59(8):1267-72.
10. Busser, J., Gelddmacher, D.S., et al. Ectopic cell cycle proteins predict the sites of neuronal cell death in Alzheimer's disease brain. *Journal of Neuroscience* (1998 April 15) 18(8):2801-7.
11. Carta, P., Flore, C., et al. Neuroendocrine and neurobehavioral effects with exposure to low doses of mercury from habitual consumption of marine fish. *Medical Lavara* (2002 May-June) 93(3):215-24.
12. De La Monte, S., and J. Wands. The AD7c-NTP neuronal thread protein biomarker for detecting Alzheimer's disease. *Frontier of Bioscience* (2002 April) 7:989-96.
13. Galasko, D., Clark, C., et al. Assessment of CSF levels of tau protein in mildly demented patients with Alzheimer's disease. *Neurology* (1997 December) 48(3):632-635.
14. Ganguli, M., Dodge, H.H., et al. Mild cognitive impairment, amnesic type: An epidemiologic study. *Neurology* (2004 July) 63(1):115-21.
15. Genetic Testing for Alzheimer's Disease: APOE Epsilon 4 Allele. Chicago, Illinois: Blue Cross Blue Shield Association – Technology Evaluation Center Assessment Program (1999 June) 14(7):1-61.
16. Goodman, I., Golden, G., et al. A multi-center blinded prospective study of urine neural thread protein measurements in patients with suspected Alzheimer's disease. *Journal of the American Medical Director Association* (2007 January) 8(1):21-30.
17. Green, R.C., Clarke, V.C., et al. Early detection of Alzheimer disease: methods, markers, and misgivings. *Alzheimer's Disease and Associated Disorder* (1997) 11(Supplement)5:S1-5.
18. Hampel, H., Buerger, K., et al. Measurement of phosphorylated tau epitopes in the differential diagnosis of Alzheimer disease: a comparative cerebral spinal fluid study. *Archives of General Psychiatry* (2004 January) 61(1):95-102.
19. Hampel, H., Goernitz, A., et al. Advances in the development of biomarkers for Alzheimer's disease: from CSF total tau and A-beta (1-42) proteins to phosphorylated tau protein. *Brain Research Bulletin* (2003 August) 61(3):243-53.
20. Hampel, H., Mitchell, A., et al. Core Biological marker candidates of Alzheimer's disease-perspectives for diagnosis, prediction of outcome and reflection of biological activity. *Journal of Neurological Transmission*. (2004 March) 111(3):27-72.
21. Hansson O., Zetterberg, H., et al. Association between CSF biomarkers and incipient Alzheimer's disease in patients with mild cognitive impairment: a follow-up study. *Lancet Neurology* (2006 March) 5(4):228-34.
22. Hayes, Inc, Health Technology Assessment May 19, 2003 Biochemical Testing For Alzheimer's Disease, Accessed 11/29/2023
23. Hock, C., Drasch, G., et al. Increased blood mercury levels in patients with Alzheimer's disease. *Journal of Neural Transmission* (1998) 105(1):59-68.
24. Hock, C., Golombowski, S., et al. Histological markers in nasal mucosa in patients with Alzheimer's disease. *European Neurology* (1998 July) 40(1):31-6.
25. Ibach, B., Binder, H., et al. Cerebrospinal fluid tau and beta-amyloid in Alzheimer patients, disease controls and an age-matched random sample. *Neurobiology of Aging* (2006 September) 27(9):1202-11.



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

26. Jeynes, B., and J. Provias. The possible role of capillary cerebral amyloid angiopathy in Alzheimer lesion development: a regional comparison. *Acta Neuropathology* (2006 October) 112(4):417-27.
27. Kahle, P.J., Jakowec, M., et al. Combined assessment of tau and neuronal thread protein in Alzheimer's disease CSF. *Neurology* (2000 October) 54(7):1498-504.
28. Kanai, M., Matsubara, E., et al. Longitudinal study of cerebrospinal fluid levels of tau, A beta1-40, and A beta1-42(43) in Alzheimer's disease: a study in Japan. *Annals of Neurology* (1998 July) 44(1):7-26.
29. Kim, K., Jung, G., et al. Increased Urinary F(2)-isoprostanes levels in the patients with Alzheimer's disease. *Brain Research Bulletin* (2004 July) 64(1):47-51.
30. Kurz, A., Riemenschneider, M., et al. Tau protein in cerebrospinal fluid is significantly increased at the earliest clinical stage of Alzheimer's disease. *Alzheimer's Disease and Associated Disorders* (1998 December) 12(4):372-7.
31. Lace, G., Wharton, S., et al. A brief history of tau: the evolving view of the microtubule-associated protein tau in neurodegenerative diseases. *Clinical Neuropathology* (2007 March-April) 26(2):43-58.
32. Maddalena, A., Papassotiropoulos, A., et al. Biochemical diagnosis of Alzheimer disease by measuring the cerebrospinal fluid ratio of phosphorylated tau protein to beta-amyloid peptide 42. *Archives of Neurology* (2003 September) 60(9):1202-6.
33. Mattsson N, Zetterberg H, Hansson O et al. CSF biomarkers and incipient Alzheimer disease in patients with mild cognitive impairment. *JAMA* 2009; 302(4):385-93.
34. Mecocci, P., Cherubini, A., et al. Tau protein in cerebrospinal fluid: a new diagnostic and prognostic marker in Alzheimer's disease? *Alzheimer's Disease and Associated Disorder* (1998 September) 12(3):211-4.
35. McEvoy, Linda K & Brewer, James B. Biomarkers for the clinical evaluation of the cognitively impaired elderly: amyloid is not enough. *Imaging Med.* 2012; 4(3): 343-357.
36. Motter, R., Vigo-Pelfrey, C., et al. Reduction of beta-amyloid peptide 42 in the cerebrospinal fluid of patients with Alzheimer's disease. *Annals of Neurology* (1995 October) 38(4):643-648. Available at <http://onlinelibrary.wiley.com/doi/10.1002/ana.410380413/abstract>
37. Mutter, J., Naumann, J., et al. Alzheimer disease: mercury as pathogenesis factor and apolipoprotein E as moderator. *Neuro Endocrinology Letter* (2004 October) 25(5):31-9.
38. Munzar, M., Levy, S., et al. Clinical study of a urinary competitive ELISA for neural thread protein in Alzheimer Disease. *Neurological Clinical Neurophysiology* (2002 September) 2002(1):2-8.
39. NIA – National Institutes on Aging Progress Report on Alzheimer's Disease (1998 November) 99-3616: 1-51. Alzheimer's Disease Education and Referral (ADEAR) Center. Available at <http://www.alzheimers.org>
40. NIH – Differential Diagnosis of Dementing Diseases #63. Consensus Statement from the NIH Consensus Development Program (1987 July 6-8) 6(11):1-27
41. Paraskevas, G., Kapaki, E., et al. The diagnostic value of cerebrospinal fluid tau protein in dementing and nondementing neuropsychiatric disorders. *Journal of Geriatric Psychiatry and Neurology* (2005 September) 18(3):163-73.
42. Parnetti, L., Lanari, A., et al. Diagnosing prodromal Alzheimer's disease: Role of CSF biochemical markers. *Mechanical Ageing Development* (2006 February) 127(2):129-32.
43. Parnetti, L., Lanari, L., et al. Cerebrospinal fluid in early detection and in differential diagnosis of dementia disorders in routine clinical practice. *Neurological Science* (2003 October) 24(3):199-200
44. Reimenschneider, M., Lautenschlager, N., et al. Cerebrospinal fluid tau and beta-amyloid 42 proteins identify Alzheimer disease in subjects with mild cognitive impairment. *Archives of Neurology* (2002 November) 59(11):1729-34.
45. Schoonenboom, N.S.M., Van De Flier, W.M., et al. CSF and MRI markers independently contribute to the diagnosis of Alzheimer's disease. *Neurobiology of Aging* 2008 May; 29(5); 669-675



MEDICAL COVERAGE POLICY

SERVICE: Biochemical Markers of Alzheimer's Disease

Policy Number: 029

Effective Date: 02/01/2025

Last Review: 01/13/2025

Next Review: 01/13/2026

46. Stefani, A., Martoana, A., et al. CSF markers in Alzheimer disease patients are not related to the different degree of cognitive impairment. *Neurological Science* (2006 December) 251(1-2):124-8.
47. Sunderland, T., Linker, G., et al. Decreased beta-amyloid 1-42 and increased tau levels in cerebrospinal fluid of patients with Alzheimer disease. *Journal of the American Medical Association* (2003 April) 289(16):2094-103.
48. Takeda, M., Okochi, M., et al. Biological markers as outcome measures for Alzheimer's interventions-real problems and future possibilities. *Institute Psychogeriatric* (2007 March) 19 (3):391-400.
49. Tsuboi, Y., Wszolek, Z., et al. Tau Pathology in the olfactory bulb correlates with Braak stage, Lewy body pathology and apolipoprotein epsilon4. *Neuropathological Applied Neurobiology* (2003 October) 29(5):503-
50. USPSTF – Screening for Dementia: Recommendations and Rationale (2003). U.S. Preventative Services Task Force.
51. Update on Dementia Edited by Davide Vito Moretti, ISBN 978-953-51-2655-3, Print ISBN 978-953-51-2654-6, 556 pages, Publisher: InTech, Chapters published September 28, 2016 under CC BY 3.0 license DOI: 10.5772/61983
52. UpToDate Clinical features and diagnosis of Alzheimer Disease, Oct 8, 2021
53. Vemuri P, Wiste HJ, Weigand SD et al. MRI and CSF biomarkers in normal, MCI, and AD subjects: diagnostic discrimination and cognitive correlations. *Neurology* 2009; 73(4):287-93.
54. Vemuri P, Wiste HJ, Weigand SD et al. MRI and CSF biomarkers in normal, MCI, and AD subjects: predicting future clinical change. *Neurology* 2009 Jul 28; 73(4):294-301.
55. Weil, M., Bressler, J., et al. Blood Mercury levels and neurobehavioral function. *Journal of the American Medical Association* (2005 April) 294(6):1875-82.
56. What is Alzheimer's? (2007). Alzheimer's Association
57. Williams, S., Chambers, K., et al. Relationship of neurofibrillary pathology to cerebral amyloid angiopathy in Alzheimer's disease. *Neuropathological Applied Neurobiology* (2005 August) 31(4):414-21.
58. Yokel, R. A. Blood-brain Barrier Flux of aluminum, manganese, iron and other metals suspected to contribute to metal-induced neurodegeneration. *Journal of Alzheimer's Disease* (2006 November) 10(2-3):223-253.
59. Yokoo, E., Valente, J., et al. Low level methyl mercury exposure affects neuropsychological function in adults. *Environmental Health* (2003 June) 2(1):8.

Note:

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 MRSA; and FirstCare CHIP is offered through FirstCare in the Lubbock Service Area.