AVISE®

Interpretation Guide

Exagen®

AVISE Interpretation Guide

SLE Associated Markers

Marker (method)	AVISE CTD	AVISE Lupus	SLE Monitor	SLE Prognostic	AVISE APS	AVISE Vasculitis	Associated Disease	Sensitivity	Description
EC4d (FC)	●	●	●				Systemic Lupus Erythematosus (SLE)	46% [1]	Cell-bound complement activation products (CB-CAPs) EC4d & BC4d are measures of classical complement activation. CB-CAPs are primarily associated with SLE and 66% of patients have EC4d and/or BC4d elevated.[1] EC4d significantly correlates with fluctuations in SLE disease activity.[2]
BC4d (FC)	●	●					SLE	53% [1]	
TC4d (FC)	●						SLE	58% [3]	CB-CAP TC4d is a measure of classical complement activation. TC4d is positive in 58% of SLE patients and is also present in roughly 25% of SLE patients who test negative for EC4d and BC4d.
TIgG (FC)	●						SLE	31% [3]	TIgG and TIgM autoantibody formation against T Cell antigens is common in SLE. 40% of SLE patients will test positive for one or both T Cell autoantibodies.
TIgM (FC)	●						SLE	30% [3]	
C3 (IT)			●				SLE	33% [1]	C3/C4 proteins are integral components of the complement system. Low C3/C4 levels are associated with SLE and 44% of patients have one or both proteins abnormally low.
C4 (IT)			●				SLE	32% [1]	
Anti-C1q IgG (ELISA)			●	●			Lupus Nephritis/ Active SLE	58% [4]	Antibodies against the complement protein C1q are found in 58% of SLE patients with active lupus nephritis. Anti-C1q levels are associated with renal activity.[2] However, low positive levels of anti-C1q have also been found in up to 8% of patients with other diseases, such as infection, and normal healthy individuals.
Anti-dsDNA IgG (ELISA and IFA)	●	●					SLE	33% [1]	Anti-dsDNA positivity provides a high positive predictive value due to the high specificity for SLE. In the AVISE Lupus algorithm, patients are first screened using an anti-dsDNA ELISA assay. Those who test positive are then confirmed with the Crithidia luciliae IFA assay, a more specific method for detecting clinically significant anti-dsDNA in SLE. Approximately 50% of anti-dsDNA ELISA results are confirmed with Crithidia luciliae.
Anti-dsDNA (CIA)			●				SLE Disease Activity	46% [5,6]	The Chemiluminescent Immunoassay (CIA) method measures quantitative levels of anti-dsDNA, which significantly correlate with SLE disease activity. Due to its expanded dynamic range, CIA can detect very low levels of anti-dsDNA antibodies and correlates closely with the Farr assay.
Anti-Nuclear Antibodies IgG (ANA) (ELISA and IFA)	●	●					Autoimmune Diseases	89% in SLE[1]	A positive ANA test suggests the presence of autoantibodies, which may indicate an autoimmune disorder like SLE. However, it is not specific to one autoimmune disease and can also be positive in healthy individuals. Further testing is typically required to confirm a diagnosis and determine the specific condition.
Anti-Ribosomal P IgG (ELFA)				●			Neuropsychiatric Lupus	9% [7]	Antibodies against Ribosomal P are highly specific for SLE and can be present in anti-dsDNA or anti-Smith negative patients. Anti-Ribosomal P antibodies have been shown to associate with neuropsychiatric SLE manifestations.
Anti-Smith IgG (ELFA)	●	●					SLE	14% [1]	Anti-Smith is highly specific for SLE, but has comparatively low sensitivity for the disease.

ENA Markers

Marker (method)	Associated Disease	Sensitivity	Description
Anti-CENP IgG (ELFA)	CREST Syndrome	20-60% [8]	Antibodies to CENP protein-B are found in 20-60% of patients with CREST syndrome, a limited form of systemic sclerosis.
Anti-Jo-1 IgG (ELFA)	Polymyositis/ Dermatomyositis (PM/DM)	20-30% [9]	Antibodies against Jo-1 are highly specific for DM/PM and are present in 25% of DM/PM patients. DM/PM patients who are positive for anti-Jo-1 may have interstitial pneumonitis and tend to have a severe form of the disease with a tendency to relapse.
Anti-RNP70 IgG (ELFA)	Mixed Connective Tissue Disease (MCTD)	90% [10]	Anti-RNP70 antibodies specifically target the 70 kDa protein of the U1-snRNP complex and are present in 90% of MCTD patients. Unlike anti-U1RNP, anti-RNP70 is more specific for MCTD and occurs in only 12% of SLE patients.
Anti-Scl-70 IgG (ELFA)	Systemic Sclerosis	28-70% [11]	Antibodies to Scl-70 are present in up to 70% of systemic sclerosis patients. Anti-Scl-70 is highly specific for diffuse cutaneous scleroderma and associates with interstitial lung disease (ILD).
Anti-SSA/Ro52 IgG (ELFA)	Myositis, SLE, Sjögren's Disease & Systemic Sclerosis	20-70% [12]	Anti-SSA/Ro52 antibodies are found in multiple autoimmune conditions. Anti-SSA/Ro52 has been shown to associate with ILD in patients with Sjögren's disease or systemic sclerosis.
Anti-SSA/Ro60 IgG (ELFA)	SLE, Sjögren's Disease, Myositis & Systemic Sclerosis	20-65% [12]	Anti-SSA/Ro60 antibodies are found in multiple autoimmune conditions. Anti-SSA/Ro60 is commonly found in both SLE and Sjögren's disease.
Anti-RNA Pol III IgG (ELFA)	Systemic Sclerosis	5-22% [13]	Anti-RNA Pol III antibodies are present in up to 22% of patients with systemic sclerosis, particularly diffuse cutaneous scleroderma. Anti-RNA Pol III is often present in the absence of other systemic sclerosis antibodies including anti-Scl-70, anti-centromere, and anti-PM/Scl.
Anti-SSB/La IgG (ELFA)	Sjögren's Disease	39% [1]	Anti-SSB/La antibodies are highly specific for Sjögren's disease and are present in 39% of Sjögren's disease patients.

AVISE Interpretation Guide

	Marker (method)	AVISE CTD	AVISE Lupus	SLE Monitor	SLE Prognostic	AVISE APS	AVISE Vasculitis	Associated Disease	Sensitivity	Description
ENA Markers	Anti-U1RNP IgG (ELFA)	●						MCTD	95-99% [10]	Anti-U1RNP antibodies are highly sensitive for MCTD but are also present in 30-40% of SLE patients. The absence of anti-U1RNP antibodies is used to rule out MCTD.
	Anti-Histone IgG * (ELISA)							Drug-Induced Lupus (DIL)	95% [14]	Up to 95% of DIL and 50% of SLE patients exhibit elevated levels of histone antibodies. Histone antibodies have also been found in RA, DM, and Sjögren's disease placing added importance on clinical presentation.
	Marker (method)							**Associated Disease**	**Sensitivity**	**Description**
RA Markers	Anti-Cyclic Citrullinated Peptide IgG (ELFA)	●	●					Rheumatoid Arthritis (RA)	70-90% [15]	Antibodies to cyclic citrullinated peptides (CCP) aid in the diagnosis of RA. Anti-CCP antibodies are highly specific for RA.
	Anti-PAD4 IgA, IgG (ELISA)	●						RA	14%-50% [18]	Anti-PAD4 autoantibodies are highly specific (>95%) for RA against healthy individuals [18]. Together, IgA and IgG antibodies are observed in 50% of seropositive RA and 14% of seronegative RA. Anti-PAD4 can develop pre-clinically and are associated with a heightened risk for severe joint damage. [16, 17]
	Anti-RA33 IgA, IgG, & IgM (ELFA)	●						RA	16-32% [19]	Anti-RA33 autoantibodies are highly specific (>95%) for RA against healthy individuals. Collectively, IgG, IgM, and IgA antibodies are present in 32% of seropositive RA patients and 16% of seronegative RA patients.
	Rheumatoid Factor IgA & IgM (ELFA)	●						RA	70-90% [15]	Rheumatoid factor (RF) antibodies are detected in 70%-90% of patients with rheumatoid arthritis (RA). The specificity for RA improves with higher RF titers and the presence of multiple positive isotypes, particularly IgM and IgA. The detection of the IgA isotype has been associated with a more severe disease prognosis.
	Anti-Carbamylated Protein (CarP) IgG * (ELISA)							RA	33% [20]	Antibodies to Carbamylated Proteins (CarP) serve as markers of more severe prognosis in RA, independent of anti-CCP or RF status. Studies have shown anti-CarP found in early RA associates with future erosive damage. The clinical significance of positive anti-CarP in the absence of RA has not been established.
	Marker (method)							**Associated Disease**	**Sensitivity**	**Description**
APS Markers	Anti-β2-Gylcoprotein I IgA, IgG, & IgM ** (ELFA)	●			●	●		Antiphospholipid Syndrome (APS)	45% [21]	Antibodies to beta-2 glycoprotein 1 (ß2 GP1) exhibit higher specificity than anti-cardiolipin. In 3-10% of APS patients, ß2 GP1 antibodies may be the only positive test. Positive results should be confirmed after 12 weeks to ensure persistency of antibodies. IgA ß2 GP1 antibodies are less common than IgG or IgM and can occur in isolation.
	Anti-Cardiolipin IgA, IgG, & IgM ** (ELFA)	●			●	●		APS	97% [22]	Antibodies to cardiolipin are present in SLE patients (30-40%) and APS. The prevalence of anti-cardiolipin in APS is high, but the specificity is lower than other anti-phospholipid antibodies. Positive results should be confirmed after 12 weeks.
	Anti-Phosphatidylserine /Prothrombin (PS/PT) IgG & IgM (ELISA)				●	●		APS	22-37% [23]	Antibodies to PS/PT are markers for APS that have been found to significantly correlate with lupus anticoagulant (LAC).[20] Unlike LAC, anti-PS/PT testing is unaffected by anti-coagulant therapy.
	Marker (method)							**Associated Disease**	**Sensitivity**	**Description**
Thyroid Markers	Anti-Thyroglobulin IgG (ELFA)	●						Hashimoto's Thyroiditis & Graves' Disease	60-85% [24]	Anti-thyroglobulin antibodies are found in 60-85% of patients with Hashimoto's thyroiditis and 30-80% of patients with Graves' disease.
	Anti-Thyroid Peroxidase IgG (ELFA)	●						Hashimoto's Thyroiditis & Graves' Disease	71-97% [24]	Anti-thyroid peroxidase antibodies are found in > 90% of patients with Hashimoto's thyroiditis & 71-97% of patients with Graves' disease. Over 95% of thyroiditis patients have thyroglobulin IgG and/or thyroid peroxidase antibodies.
	Marker (method)							**Associated Disease**	**Sensitivity**	**Description**
Vasculitis Markers	ANCA (IFA)						●	ANCA-Associated Vasculitis	77% [25]	The c-ANCA pattern produces a granular cytoplasmic pattern with interlobular accentuation on ethanol fixed neutrophils. c-ANCA patterns are associated with necrotizing segmental glomerulonephritis and GPA. [25,26]
									85% [25]	The p-ANCA pattern produces perinuclear staining with or without nuclear extension. The p-ANCA pattern is commonly detected in patients with MPA and about 40% of patients with Eosinophilic Granulomatosis with Polyangiitis (EGPA). [26,27]
	Anti-PR3 IgG (CIA)						●	Granulomatosis with Polyangiitis (GPA)	81% [25]	Anti-PR3 antibodies are primarily associated with GPA and to a lesser extent, found in MPA (10%) and EGPA.[25,29] However, anti-PR3 antibodies can also be seen in connective tissue disease, IBD, some infections, malignancy, and as a reaction to drugs. Therefore, results should be interpreted with care in light of the clinical findings and workup. [28]
	Anti-MPO IgG (CIA)						●	Microscopic Polyangiitis (MPA)	85% [25]	Anti-MPO antibodies are primarily associated with MPA and to a lesser extent, found in GPA (6%) and EGPA.[25,29] However, anti-MPO antibodies can also be seen in connective tissue disease, inflammatory bowel disease (IBD), some infections, malignancy, and as a reaction to drugs. Therefore, results should be interpreted with care in light of the clinical findings and workup.[28]
	Anti-GBM IgG (CIA)						●	Goodpasture's Syndrome (GPS)	96% [29]	Anti-GBM antibodies are often associated with Goodpasture's disease and anti-GBM nephritis.[29] A significant proportion of patients with anti-GBM disease are also positive for ANCA.

* Available as a stand alone test order.
** IgA antibodies only available in AVISE APS and SLE Prognostic test orders.

AVISE Interpretation Guide

AVISE HCQ

A test to aid in assessing adherence to HCQ and individual exposure to HCQ as measured in whole blood.

HCQ Level	Interpretation & Consideration
Supratherapeutic (>1200 ng/mL)	Level associated with clinical efficacy. HCQ is likely absorbed effectively. However, levels >1200 ng/ml have a minimal added benefit compared to levels in the therapeutic range (750 ng/ml - 1200 ng/ml). Thus, adjusting HCQ doses to maintain blood levels in the therapeutic range may maximize efficacy while limiting toxicity.
Therapeutic (750-1200 ng/mL)	Levels associated with clinical efficacy. HCQ is likely to be absorbed effectively.
Subtherapeutic (200-<750 ng/mL)	Patient may be partially adherent to therapy. Patients with HCQ < 750 ng/mL can be at greater risk for disease flare. Thus, adjusting HCQ doses and/or discussing barriers to compliance may maximize efficacy.
Underexposed (<200 ng/mL)	Patient is likely non-adherent to HCQ therapy.

AVISE MTX

A test to aid in assessing adherence to MTX and individual exposure to active MTX metabolites in red blood cells.

MTXPG Level	Interpretation & Consideration
Therapeutic (>60 nmol/L)	Patient is metabolizing MTX effectively. Level is consistent with clinical efficacy.
Intermediate (20-60 nmol/L)	Patient may need more exposure to MTX.
Sub-therapeutic (<20 nmol/L)	Patient may not be metabolizing MTX effectively or patient may be non-adherent with therapy.

Methodology Definitions:

FC: Flow Cytometry

ELISA: Enzyme-Linked Immunosorbent Assay

IFA: Immunofluorescence Assay

ELFA: Enzyme-Linked Fluorescence Assay

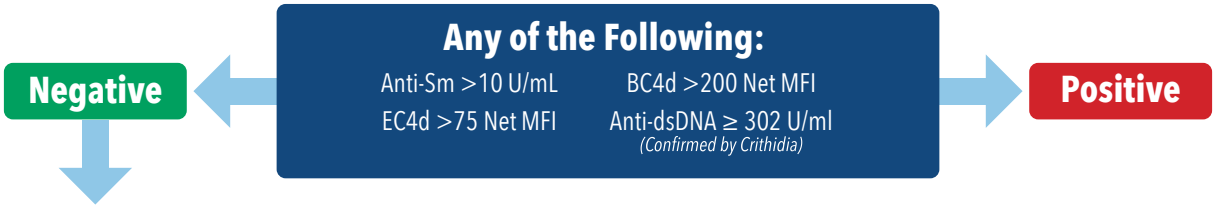
IT: Immunoturbidimetry

CIA: Chemiluminescent Immunoassay

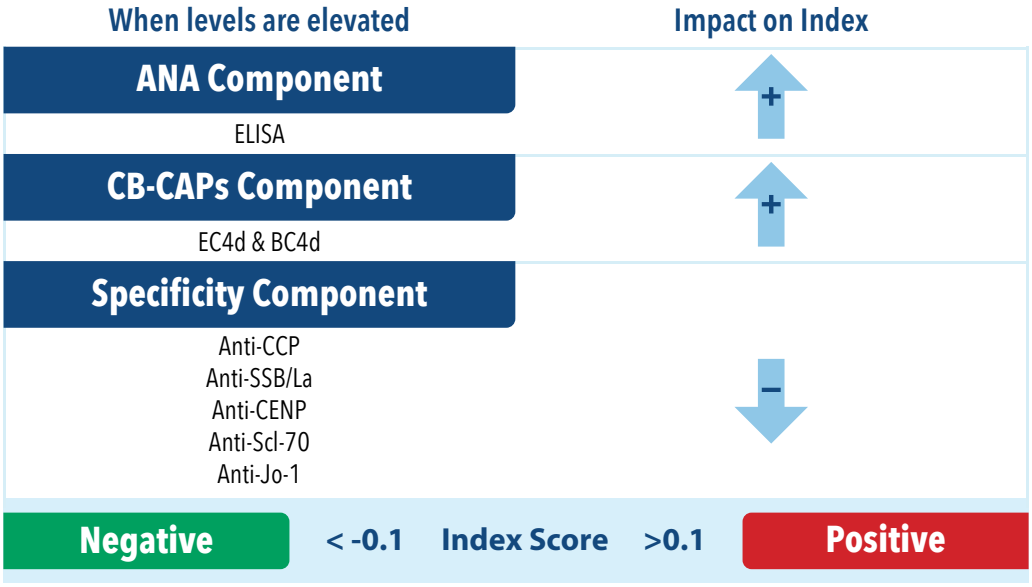
A deeper look at the AVISE® Lupus two-tier algorithm

The AVISE CTD two-tier algorithm is a proprietary diagnostic method used to assess the patient's likelihood of systemic lupus erythematosus (SLE). This is done by deriving an index score from the calculation of ANA, highly specific lupus markers (anti-Smith, anti-dsDNA, BC4d and EC4d), and other disease specific autoantibodies.

TIER 1 - *Tier 1 criteria is highly specific for SLE*



TIER 2 - *If Tier 1 is Negative move to Tier 2*



Tier 2 generates an index value based on the following components

- ANA component
- CB-CAPs component
- Autoantibody specificity component

AVISE Interpretation Guide

AVISE® CTD results report

AVISE® CTD
Comprehensive Connective Tissue Disease Assessment

Order ID	Specimen	Test Order	Patient	
739813	*Collected* 07/21/2025	*Created* 07/22/2025	*Gender* Female	**Sample, Susan S.**
Provider Exagen Provider MD	*Received* 07/22/2025	*Reported* 07/24/2025	*MRN* AB123450	*DOB* 01/01/1996

AVISE Lupus Result: **Tier 1 Positive**

Negative | Indeterminate | Positive | Tier 1 Positive
-5 -4 -3 -2 -1 0 1 2 3

INDEX INTERPRETATION: Tier 1 positive results are associated with an increased likelihood of SLE and are greater than 96% specific for SLE against other autoimmune CTDs. These results are driven by elevated markers that are highly specific for SLE (anti-dsDNA, anti-smith, and/or strong positive EC4d/BC4d). Results should be interpreted by a provider conjunction with all available clinical findings.

Lupus Index Score Biomarkers	Value	Interpretation	Reference Range
+ Anti-dsDNA IgG (ELISA)	865.61 IU/mL	**POSITIVE**	<201 - Negative I 201-<302 - Equivocal I ≥302 - Positive
Confirmation by Crithidia luciliae (IFA)		Positive anti-dsDNA confirmed by Crithidia	
Anti-Smith IgG (ELFA)	1.2 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive
+ CB-CAP: EC4d - Erythrocyte-bound C4d (FC)	18 Net MFI	**POSITIVE**	<15 - Negative I 15-75 - Positive I >75 - Strong Positive
CB-CAP: BC4d - B-lymphocyte-bound C4d (FC)	52 Net MFI	Negative	<61 - Negative I 61-200 - Positive I >200 - Strong Positive
++ANA IgG (ELISA)	120.88 Units	**STRONG POSITIVE**	<20 - Negative I 20-<60 - Positive I ≥60 - Strong Positive
Anti-SSB/La IgG (ELFA)	1.7 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive
Anti-Scl-70 IgG (ELFA)	0.8 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive
Anti-Centromere Protein B (CENP) IgG (ELFA)	1.0 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive
Anti-Jo-1 IgG (ELFA)	<0.3 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive
Anti-CCP IgG (ELFA)	1.4 U/mL	Negative	<7 - Negative I 7-10 - Equivocal I >10 - Positive

T Cell Biomarkers	Value	Interpretation	Reference Range
+ CB-CAP: TC4d (FC)	215 Net MFI	**POSITIVE**	<200 - Negative I ≥200 - Positive
+ T Cell autoantibody: TIgG (FC)	190 Net MFI	**POSITIVE**	<170 - Negative I ≥170 - Positive
+ T Cell autoantibody: TIgM (FC)	250 Net MFI	**POSITIVE**	<230 - Negative I ≥230 - Positive

ANA (Immunofluorescence)	Value	Interpretation	Reference Range
+ ANA by HEp-2 (IFA)	Titer: 1:640	**POSITIVE**	<1:80 - Negative I ≥1:80 - Positive
	Nuclear Pattern: Homogeneous		
	Cytoplasmic Pattern: Not Observed		

Comments:

- EC4d and TC4d are markers of classical complement activation with greater than 95% specificity against healthy individuals. In the positive range, EC4d is present in 46% of SLE patients and in low percentages of patients with Sjögren's disease, myositis, systemic sclerosis, RA, APS, and vasculitis. EC4d correlates with SLE disease activity. When positive, TC4d is observed in 58% of SLE patients. It is also present in 28% of Sjögren's disease, 13% of spondyloarthropathies, 13% of PsA, and 5% of RA patients. [Kyttaris et al. 2025]. **A positive result alone does not establish a diagnosis. Results must be interpreted by a healthcare provider in the context of all available clinical findings.**
- TIgG and TIgM autoantibodies are 95% specific against healthy individuals. When positive, TIgG is observed in 31% of SLE patients. It is also present in 10% of Sjögren's disease and 10% of RA patients. When positive, TIgM is observed in 30% of SLE patients. It is also present in 14% of Sjögren's disease, 4% of PsA, 7% of fibromyalgia, and 6% of RA patients. Additionally, TIgM is elevated in up to 21% of patients exposed to COVID-19, though levels typically do not persist beyond six months in mild (non-hospitalized) cases. [Kyttaris et al. 2025 and Perez-Diez et al. 2024]. **A positive result alone does not establish a diagnosis. Results must be interpreted by a healthcare provider in the context of all available clinical findings.**

In a study of 794 subjects comprising 304 SLE patients, 285 patients with other rheumatic diseases and 205 normal healthy controls, positivity for Tier 1 markers (anti-dsDNA by ELISA, confirmed with Crithidia by IFA, anti-Sm by Enzyme Linked Flourescent Immunoassay (ELFA) or elevated EC4d and BC4d by Flow Cytometry yielded a sensitivity of 46% and a specificity of 97% for SLE vs. other autoimmune rheumatic diseases (ORD). Among the 164 SLE subjects negative in Tier 1, a positive index score composite of ANA (by ELISA), EC4d/BC4d and positivity for anti-CCP, SS-B/La, CENP, Jo-1 or Scl-70 (by ELFA) resulted in sensitivity of 62% for SLE and specificity of 89% for SLE vs. ORD. The overall two-tier algorithm result yielded 80% sensitivity for SLE and 86% specificity vs. ORD diseases (98% specificity for SLE vs. healthy individuals).

Page 1 of 3
STR0002 v2

Patients: Positive individual biomarker results may the context of all clinical information.

Rheumatoid Arthritis Biomarkers
Anti-CCP IgG (ELFA)
Anti-PAD4 IgA (ELISA)
Anti-PAD4 IgG (ELISA)
Anti-RA33 IgA (ELFA)
Anti-RA33 IgG (ELFA)
Anti-RA33 IgM (ELFA)
Rheumatoid Factor IgA (ELFA)
Rheumatoid Factor IgM (ELFA)

Sjögren's Disease Biomarkers
Anti-SSA/Ro52 IgG (ELFA)
Anti-SSA/Ro60 IgG (ELFA)
Anti-SSB/La IgG (ELFA)

Mixed Connective Tissue Disease Biomarkers
Anti-U1RNP IgG (ELFA)
Anti-RNP70 IgG (ELFA)

Antiphospholipid Syndrome Biomarkers
+ Anti-Cardiolipin IgG (ELFA)
Anti-Cardiolipin IgM (ELFA)
Anti-β2 Glycoprotein 1 IgG (ELFA)
Anti-β2 Glycoprotein 1 IgM (ELFA)

Systemic Sclerosis Biomarkers
Anti-Scl-70 IgG (ELFA)
Anti-RNA Pol III IgG (ELFA)
Anti-Centromere Protein B (CENP) IgG (ELFA)

Myositis Biomarkers
Anti-Jo-1 IgG (ELFA)

Thyroid Biomarkers
Anti-Thyroglobulin IgG (ELF
Anti-Thyroid Peroxidase Ig

Case Remarks:

Signed by: Prashanti Re

References
1. Kyttaris V, et al. Front Immu
2. Concoff A, Warsi T, Taghav
3. Concoff A, Warsi T, Taghav
4. Ramsey-Goldman R, et al.
5. Arriens C, et al. Lupus Sci
6. Ramsey-Goldman R, et al.
7. Hui-Yuen J, et al. Lupus. 20
8. Sieghart D, Platzer A, Stude
9. Wallace D, et al. Lupus Sci
10. Putterman C, et al. Lupus S

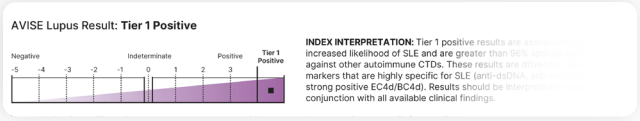
Exagen

This test was developed and its perfor such clearance or approval is not nece Laboratory improvement Amendments

RA33 autoantibodies are highly specific (>95%) for RA against healthy IgM, and IgA RA33 autoantibodies were collectively present in 32% of healthy individuals. In a cohort of 379 RA patients (220 seropositive a

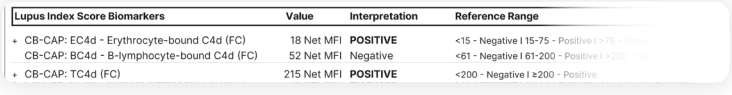
Lupus index and interpretation

The AVISE Lupus algorithm and its interpretation turn isolated data points into meaningful information to help rule in/out lupus.



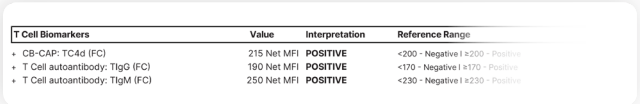
Cell-Bound Complement Activation Products (CB-CAPs)

AVISE CTD incorporates CB-CAPs biomarkers, which offer higher sensitivity for diagnosing SLE than many conventional biomarkers including C3/C4 levels.



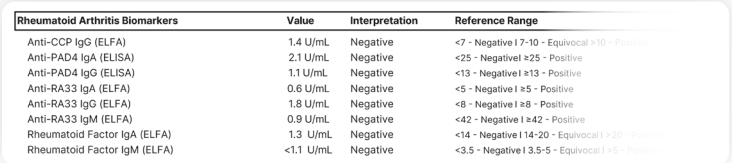
T Cell lupus biomarkers

The newly available T Cell biomarkers provide unique and unmatched diagnostic accuracy to further substantiate an SLE diagnosis when conventional markers are negative.



Rheumatoid arthritis biomarkers

The addition of new highly specific RA biomarkers, including anti-RA33 and anti-PAD4, aids in the early diagnosis of seronegative RA patients.



AVISE®

References:

1. Putterman C, Furie R, Ramsey-Goldman R, et al. Cell-bound complement activation products in systemic lupus erythematosus: comparison with anti-double-stranded DNA and standard complement measurements. Lupus Sci Med. 2014;1(1):e000056. doi:10.1136/lupus-2014-000056
2. Buyon J, Furie R, Putterman C, et al. Reduction in erythrocyte-bound complement activation products and titres of anti-C1q antibodies associate with clinical improvement in systemic lupus erythematosus. Lupus Sci Med. 2016;3(1):1-8. doi:10.1136/lupus-2016-000165
3. Kyttaris V, Concoff A, Warsi T, et al. Multi-centered Clinical Validation of T Cell-bound C4d (TC4d) and T Cell Autoantibodies (TIgG and TIgM): Sensitive and Specific Biomarkers of SLE with Enhanced Accuracy Compared to Conventional SLE Tests [abstract]. Arthritis Rheumatol. 2024; 76 (suppl 9).
4. Yin Y, Wu X, Shan G, Zhang X. Diagnostic value of serum anti-C1q antibodies in patients with lupus nephritis: A meta-analysis. Lupus. 2012;21(10):1088-1097.- doi:10.1177/0961203312451202
5. Merrill J, Petri M, Buyon J, et al. Erythrocyte-bound C4d in combination with complement and autoantibody status for the monitoring of SLE. Lupus Science & Medicine. 2018.
6. Mahler M. Bentow C. O'Malley T, et al. Performance Characteristics of Different Anti-Double-Stranded DNA Antibody Assays in the Monitoring of Systemic Lupus Erythematosus. J Immunol Res. 2017;2017:1720902.
7. Hanly JG, Urowitz MB, Su L, Romero-Diaz J. Autoantibodies as biomarkers for the prediction of neuropsychiatric events in systemic lupus erythematosus Ann Rheum Dis. 2011;70(12):2240. doi:10.1136/ard.2010.148502corr1.
8. Russo K, Hoch S, Dima C, Varga J, Teodorescu M. Circulating anticentromere CENP-A and CENP-B antibodies in patients with diffuse and limited systemic sclerosis, systemis lupus erythematosus, and rheumatoid arthritis. J Rheumatol. 2000;27(1):142-148.
9. Zampieri S, Ghirardello A, Iaccarino L, Tarricone E, Gambari PF, Doria A. Anti-Jo-1 antibodies. Autoimmunity. 2005;38(1):73-78. doi:10.1080/08916930400022640
10. Hoffman R.W., GreidingerE.I. (2002) Mixed Connective Tissue Disease. In: Tsokos G.C. (eds) Modern Therapeutics in Rheumatic Diseases. Humana Press, Totowa, NJ. doi:10.1007/978-1-29259-239-5_23
11. Basu D, Reveille JD. Anti-scl-70. Autoimmunity. 2005;38(1):65-72. doi:10.1080/08916930400022947
12. Robbins A, et al. Diagnostic Utility of Separate Anti-Ro60 and Anti-Ro52/TRIM21 Antibody Detection in Autoimmune Diseases. Front Immunol. 2019;10:444. doi: 10.3389/
13. Maes L, et al. Anti-PL/Scl-100 and RNA-Pol III antibodies in scleroderma. Clin Chim Acta. 2010; 411(13-14): 965-71. doi: 10.1016/j.cca.2010.03.018
14. Dalle Vedove C, Simon JC, Girolomoni G. Drug-induced lupus erythematosus with emphasis on skin manifestations and the role of anti-TNF agents. J Dtsch Dermatol Ges. 2012;10(12):889-897. doi:10.1111/j.1610-0387.2012.08000.x
15. Rantapää-Dahlqvist S, de Jong BA, Berglin E, et al. Antibodies against cyclic citrullinated peptide and IgA rheumatoid factor predict the development of rheumatoid arthritis. Arthritis Rheum. 2003;48(10):2741-2749. doi:10.1002/art.11223
16. Kolfenbach J, et al. Arthritis Rheum. 2010 Sep;62(9):2633-9. doi: 10.1002/art.27570
17. Harris M, et al. Arthritis Rheum. 2008 July; 58 (7)1958–67. doi: 10.1002/art.23596
18. Data on file.
19. Concoff A, Warsi T, Taghavi S, et al. Anti-RA33 Autoantibodies Are Unique, Sensitive Biomarkers for the Identity cation of Seronegative Rheumatoid Arthritis in a U.S. Cohort [abstract]. Arthritis Rheumatol. 2024; 76 (suppl 9).
20. Truchetet ME, et al. Association of the presence of anti-carbamylated protein antibodies in early arthritis with a poorer clinical and radiological outcome. Arthritis & Rheumatology. 2017;69(12): 2292-2302. doi: 10.1002/art.40237
21. Monica Galli, Davide Luciani, Guido Bertolini, Tiziano Barbui; Anti– 2-glycoprotein I, antiprothrombin antibodies, and the risk of thrombosis in the antiphospholipid syndrome. Blood.2003; 102 (8): 2717–2723. doi: 10.1182/blood-2002-11-3334
22. Akhter E, Shums Z, Norman GL, Binder W, Fang H, Petri M. Utility of antiphosphatidylserine/prothrombin and IgA antiphospholipid assays in systemic lupus erythematosus. J Rheumatol.2013;40(3):282-286. doi:10.3899/jrheum.120084
23. Petri MA, Conklin J, O'Malley T, Dervieux T. Platelet-bound C4d , low C3 and lupus anticoagulant associate with thrombosis in SLE. Lupus Sci Med. Published online 2019:6-11. doi:10.1136/lupus-2019-000318
24. Engler H, Riesen WF, Keller B. Anti-thyroid peroxidase (anti-TPO) antibodies in thyroid diseases, non-thyroidal illness and controls. Clinical validity of a new commercial method for detection of anti-TPO (thyroid microsomal) autoantibodies. Clin Chim Acta. 1994;225(2):123-136. doi:10.1016/0009-8981(94)90040-x
25. Damoiseaux J, Csernok E, Rasmussen N, et al. Detection of antineutrophil cytoplasmic antibodies (ANCAs): A multicentre European Vasculitis Study Group (EUVAS) evaluation of the value of indirect immunofluorescence (IFF) versus antigen-specific immunoassays. Ann Rheum Dis. 2017;76(4):647-653. doi:10.1136/annrheumdis-2016-209507
26. Savige J, Gillis D, Benson E, et al. International Consensus Statement on Testing and Reporting of Antineutrophil Cytoplasmic Antibodies (ANCA). Am J Clin Pathol. 999;111(4):507-13. doi:10.1093/ajcp/111.4.507
27. Mahler M, Radice A, Yang W, et al. Clinica Chimica Acta Development and performance evaluation of novel chemiluminescence assays for detection of anti-PR3 and anti-MPO antibodies. Clin Chim Acta. 2012;413(7-8):719-726. doi:10.1016/j.cca.2012.01.004
28. Wallace ZS, Miloslavsky EM. Management of ANCA associated vasculitis. BMJ. 2020;368(March):1-16. doi:10.1136/bmj.m421
29. Mahler M, Radice A, Sinico RA, et al. Performance evaluation of a novel chemiluminescence assay for detection of anti-GBM antibodies: an international multicenter study. 2012;3(May 2011):24.

Explore our real-world data and clinical studies.

www.AviseTest.com | 888.452.1522

Exagen®