

Patient Name : Mrs Dummy  
 DOB/Age/Gender :  
 Patient ID / UHID : XXX/OFXXX  
 Referred By :  
 Sample Type :  
 Barcode No :

Bill Date :  
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 Sample Received :  
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 Report Status :



Test Description	Value(s)	Unit(s)	Reference Range
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**HEMATOLOGY REPORT**  
**Winter Special Pro Package**  
**Complete Blood Count (CBC)**

**RBC Parameters**

Hemoglobin	12.6	g/dL	12.0 - 15.0
Method : colorimetric			
RBC Count	3.9	10 <sup>6</sup> /μl	3.8 - 4.8
Method : Electrical impedance			
PCV	35	%	36 - 46
Method : Calculated			
MCV	89	fl	83 - 101
Method : Calculated			
MCH	32	pg	27 - 32
Method : Calculated			
MCHC	35.9	g/dL	31.5 - 34.5
Method : Calculated			
RDW (CV) *	14.4	%	11.6 - 14.0
Method : Calculated			
RDW-SD *	49.6	fl	35.1 - 43.9
Method : Calculated			

**WBC Parameters**

TLC	6.6	10 <sup>3</sup> /μl	4 - 10
Method : Electrical impedance and microscopy			

**Differential Leucocyte Count**

Neutrophils	56	%	40-80
Lymphocytes	38	%	20-40
Monocytes	4	%	2-10
Eosinophils	2	%	1-6
Basophils	0	%	<2

**Absolute Leukocyte Counts**

Method : Calculated

Neutrophils.	3.7	10 <sup>3</sup> /μl	2 - 7
Lymphocytes.	2.51	10 <sup>3</sup> /μl	1 - 3
Monocytes.	0.26	10 <sup>3</sup> /μl	0.2 - 1.0
Eosinophils.	0.13	10 <sup>3</sup> /μl	0.02 - 0.5
Basophils.	0	10 <sup>3</sup> /μl	0.02 - 0.5

**Platelet Parameters**

Platelet Count	231	10 <sup>3</sup> /μl	150 - 410
Method : Electrical impedance and microscopy			
Mean Platelet Volume (MPV) *	9.7	fL	9.3 - 12.1
Method : Calculated			

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PCT * Method : Calculated	0.22	%	0.17 - 0.32
PDW * Method : Calculated	16.6	fL	8.3 - 25.0
P-LCR * Method : Calculated	31.7	%	18 - 50
P-LCC * Method : Calculated	73	%	44 - 140
Mentzer Index * Method : Calculated	22.82	%	-

### Interpretation:

CBC provides information about red cells, white cells and platelets. Results are useful in the diagnosis of anemia, infections, leukemias, clotting disorders and many other medical conditions.

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**HEMATOLOGY REPORT**  
**Winter Special Pro Package**  
**Erythrocyte Sedimentation Rate (ESR)**

ESR - Erythrocyte Sedimentation Rate                      12                      mm/hr                      0 - 12  
 Method : MODIFIED WESTERGREN

**Interpretation:**

ESR is also known as Erythrocyte Sedimentation Rate. An ESR test is used to assess inflammation in the body. Many conditions can cause an abnormal ESR, so an ESR test is typically used with other tests to diagnose and monitor different diseases. An elevated ESR may occur in inflammatory conditions including infection, rheumatoid arthritis, systemic vasculitis, anemia, multiple myeloma, etc. Low levels are typically seen in congestive heart failure, polycythemia, sickle cell anemia, hypo fibrinogenemia, etc.

AGE	MALE	FEMALE
1 DAY	0-2	0-2
2 - 7 DAYS	0-4	0-4
8 - 14 DAYS	0-17	0-17
15 DAYS - 17 YEARS	0-20	0-20
18 - 50 YEARS	0-10	0-12
51 - 60 YEARS	0-12	0-19
61 - 70 YEARS	0-14	0-20
71 - 100 YEARS	0-30	0-35

**Reference-** Dacie and lewis practical hematology

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**HEMATOLOGY REPORT**  
**Winter Special Pro Package**  
**HbA1C (Glycosylated Haemoglobin)**

Glycosylated Hemoglobin (HbA1c) Method : IMMUNOTURBIDIMETRIC	4.99	%	< 5.7
Estimated Average Glucose *	96.51	mg/dL	Refer Table Below

**Interpretation:**

**Interpretation For HbA1c% As per American Diabetes Association (ADA)**

Reference Group	HbA1c in %
Non diabetic adults >=18 years	<5.7
At risk (Prediabetes)	5.7 - 6.4
Diagnosing Diabetes	>= 6.5
Therapeutic goals for glycemic control	Age > 19 years Goal of therapy: < 7.0 Age < 19 years Goal of therapy: <7.5

- Note:**
- Since HbA1c reflects long term fluctuations in the blood glucose concentration, a diabetic patient who is recently under good control may still have a high concentration of HbA1c. Converse is true for a diabetic previously under good control but now poorly controlled.
  - Target goals of < 7.0 % may be beneficial in patients with short duration of diabetes, long life expectancy and no significant cardiovascular disease. In patients with significant complications of diabetes, limited life expectancy or extensive co-morbid conditions, targeting a goal of < 7.0 % may not be appropriate.

**Comments :**

HbA1c provides an index of average blood glucose levels over the past 8 - 12 weeks and is a much better indicator of long term glycemic control as compared to blood and urinary glucose determinations ADA criteria for correlation between HbA1c & Mean plasma glucose levels.

HbA1c(%)	Mean Plasma Glucose (mg/dL)	HbA1c(%)	Mean Plasma Glucose (mg/dL)
6	126	12	298
8	183	14	355
10	240	16	413

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## HEMATOLOGY REPORT

### Anti Phospholipid Antibody (APLA) Panel

#### Activated Partial Thromboplastin Time (APTT)

APTT	33.7	Sec	23.8-34.8
Method : Viscosity-based clot detection			
Control (MNAPTT)	28.0		

#### Interpretation:

The APTT is a one-stage test. This is used for the diagnosis of bleeding disorders. APTT may be used in the patient to check treatment for those who are taking Heparin or other blood-thinning medicines. APTT measures the intrinsic system and common pathways. APTT used in the diagnosis of Hemophilia and Christmas disease.

#### Abnormal High results of APTT are due to:

1. All congenital deficiencies of Intrinsic system coagulation factors.
2. Cirrhosis, Drugs, Heparin therapy, Warfarin therapy.
3. Disseminated intravascular coagulopathy (DIC), Fibrin breakdown products.
4. Factor XII deficiency, Hemophilia A and B, Hypofibrinogenemia, Von Willebrand's disease.
5. Malabsorption, Vit K deficiency, Fibrin breakdown products, Leukemia.
6. In the case of streptokinase and urokinase.
7. Circulating anticoagulant inhibitors.

These may be specific for factor VIII.

1. These are seen as anti-factor VIII and anti-factor IX in 5% to 10% of hemophilic patients.
2. These are also in multiple plasma transfusions.
3. Drug reactions.
4. In the case of tuberculosis.
5. In autoimmune diseases like SLE and rheumatoid arthritis.

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**HEMATOLOGY REPORT**  
**Absolute Eosinophil Count (AEC)**

TLC	6.6		
Eosinophils	01		
Eosinophils.	0.06	10 <sup>3</sup> /μl	0.02 - 0.5

**Interpretation:**

Increased eosinophil count is often associated with allergic reaction, parasitic infestation, mycosis, collagen vascular disorders, some diffuse skin diseases, brucellosis and in hematopoietic diseases like CML, AML, Hodgkin's disease, T cell lymphoma, eosinophilic leukemia.

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**HEMATOLOGY REPORT**  
**Thrombophilia Profile- Comprehensive**  
**Anti Thrombin III Activity/Functional**

Anti Thrombin III Activity	118	%	Non Pregnant : 80-120
Method : Viscosity-based clot detection			First Trimester : 89-114
			Second Trimester : 88-112
			Third Trimester : 82-116

**Interpretation:**

**Interpretation :**

1. Anti Thrombin III is a protein molecule that inactivates several enzymes of the coagulation system.
2. Congenital ATIII deficiency leads to increased risk of venous and arterial thrombosis, with an onset of clinical manifestations typically appearing in young adulthood.
3. At present, the Anti-thrombin congenital deficiencies are classified into four categories as below:-

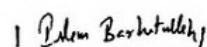
Type of Deficiency	Protein Amount	Biological Activity	Remarks
I	Decreased	Decreased	More frequent case
II RS (reactive site)	Normal	Decreased	Anomaly of the reactive site
II HBS (heparin binding site)	Normal	Normal when heparin is absent	Anomaly of the AT-heparin binding
II PE (pleiotropic effect)	Decreased	Decreased	Dysfunctional protein and at reduced quality

4. Acquired ATIII deficiency occurs in DIC, hemolytic uremic syndrome and venoocclusive disease (VOD) in patients undergoing bone marrow transplant, Nephrotic syndrome, Liver disease and L-asparaginase treatments

**Increased levels of antithrombin occur in :**

1. Acute hepatitis/cholestasis .
2. Kidney transplant .
3. Vitamin K deficiency .
4. Warfarin (coumadin) anticoagulation therapy (sometimes) Testing for antithrombin deficiency is not recommended unless the patients condition is stable.

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**HEMATOLOGY REPORT**  
**Anti Phospholipid Antibody (APLA) Panel**

**Lupus Anticoagulant**

Patient Value	34.5	sec	33.1-45.1
Control value	39.1	sec	
Screen Ratio	0.88		<1.20
DRVVT Screening	Negative		

**Interpretation:**

Interpretation:

Method: Dilute Russell viper venom method ( dRVV), electromechanical clot detection.

Remarks:

1. This is only a **screening test**.

2. If Screening test is positive, then a confirmatory test is necessary.

3. The presence of LA in the sample is confirmed when the Normalized Ratio ( calculated as ratio of dRVV screen ratio to dRVV confirmatory ratio) value is greater or equal to reference value.

Test description: Diluted RVV Screen test is performed with reagent containing a low concentration of phospholipids. If lupus anticoagulant (LA) is present, the

clotting time will be lengthened. dRVV confirmatory testing is done with reagent containing higher concentration of phospholipids, which neutralizes the LA (when

present in the sample) and corrects the clotting time to normal thereby confirming the presence of LA.

Notes:

1. As per ISTH( International society on thrombosis and hemostasis) guidelines , Lupus Anticoagulant detection must be done by using at least two clot based assays

employing separate clotting principles like Lupus sensitive APTT & dRVVT.

2. Results of this test should always be interpreted in conjunction with the patient's medical history, clinical presentation and other findings.

3. A positive LA can be seen in otherwise normal individuals and in certain viral or other infections.

4. Once a patient has been tested positive for LA, it is imperative that testing be repeated on a second occasion > 12 weeks after the initial testing.

5. Anticoagulation therapy effects such as Warfarin (especially when the effect is supratherapeutic), excess Heparin, direct thrombin inhibitors (DTI) (eg, Dabigatran

[Pradaxa]), Argatroban [Ancova], Bivalirudin [Angiomax]), direct factor Xa inhibitors (eg, Rivaroxaban [Xarelto], Apixaban [Eliquis], Edoxaban [Savaysa]) may result in

a false-positive assay performance for LA. Clinical correlation and repeat testing after discontinuation (>1 week) of anticoagulation therapy is suggested.

6. Although the dilute Russell viper venom time (dRVVT) reagents contain a heparin inhibitor (Polybrene) that is sufficient for neutralization of heparin (up to 1-2

U/mL), the results may not necessarily represent what would occur if no heparin were present in the specimen. Therefore, DRVVT results from heparinized plasma

should be interpreted with caution.

7. dRVVT assays, when performed in isolation, will not distinguish LA from heparin or inhibitors of factors V or VIII, which may cause false-positive results of LA testing.

Comments: Lupus Anticoagulants are heterogenous IgG or IgM autoantibodies which interfere with phospholipid dependent in vitro coagulation tests, particularly

activated partial thromboplastin time (APTT). These antibodies are associated with thrombosis (arterial & venous), recurrent abortions, neurological & neuropsychiatric

disorders. Various methods for testing Lupus Anticoagulants include Lupus sensitive APTT (PTT-LA), activated kaolin clotting time and dilute Russell Viper Venom

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time. Out of these the dRVVT assay is the most robust & specific because dRVVT is not influenced by deficiencies of intrinsic pathway or antibodies to factors VIII, IX or XI.

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**HEMATOLOGY REPORT**  
**Thrombophilia Profile- Comprehensive**  
**Protein C Activity / Functional**

Protein C Activity	90	%	70-130
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Method : Viscosity-based clot detection

**Interpretation:**

**Interpretation :**

Protein C is a major physiological anticoagulant The activated form (with protein S as a cofactor) degrades Factor Va and Factor VIIIa. An inherited decrease in protein C increases the patients risk for venous thromboembolism.

1. Artificially decreased functional protein C values may be seen in patients with abnormally elevated levels of factor VIII.
2. Artificially increased levels of functional protein C values may be seen in patients on heparin therapy.
3. Patients on oral anticoagulants will have decreased functional protein C values. Patients should be off oral anticoagulant therapy for two weeks for accurate measurement of functional protein C levels.

**HEMATOLOGY REPORT**  
**Thrombophilia Profile- Comprehensive**  
**PROTEIN S, (FREE) ANTIGEN.**

PROTEIN S, (FREE) ANTIGEN	72	%	71-113
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Method : Viscosity-based clot detection

For Pregnancy :  
 1st Trimester : 34 - 133  
 2nd Trimester : 29 - 113  
 3rd Trimester : 23 - 65

**Interpretation:**

**Note**

1. Determination of both Free & Bound Protein S are recommended to identify hereditary or acquired protein S deficiency.
2. Very high factor VIII (>400%) activity, Activated protein C resistance, Coumadin therapy & acute or chronic inflammation may cause spuriously low results.
3. Mutation in factor V at its protein Ca cleavage site may lead to diminished recovery of protein S.
4. Test conducted on citrated plasma.

**Comment**

Protein S is a vitamin K dependent plasma glycoprotein, exists in plasma as 40% free and 60% bound to C4b Binding protein. Free protein S possesses both ACP - dependent & independent anticoagulant properties. Its deficiency is associated with increased risk of thrombosis. The prevalence of protein S deficiency in thrombophilic population is 7 - 12%. The estimated thrombotic risk is 10-15 folds in patient with this deficiency. There is characteristic decrease of free protein S during pregnancy. At birth protein S activity is close to that of adult with total & free protein S being low.

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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Glucose Fasting (BSF)**

Glucose Fasting Method : Hexokinase	88	mg/dL	70 - 100
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**Interpretation:**

Status	Fasting plasma glucose in mg/dL
Normal	<100
Impaired fasting glucose	100 - 125
Diabetes	=>126

**Reference :** American Diabetes Association

**Comment :**

Blood glucose determinations in commonly used as an aid in the diagnosis and treatment of diabetes. Elevated glucose levels (hyperglycemia) may also occur with pancreatic neoplasm, hyperthyroidism, and adrenal cortical hyper function as well as other disorders. Decreased glucose levels (hypoglycemia) may result from excessive insulin therapy insulinoma, or various liver diseases.

**Note**

- 1.The diagnosis of Diabetes requires a fasting plasma glucose of > or = 126 mg/dL or a random / 2 hour plasma glucose value of > or = 200 mg/dL with symptoms of diabetes mellitus.
- 2.Very high glucose levels (>450 mg/dL in adults) may result in Diabetic Ketoacidosis.

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NABL-M(EL)T-00726

Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Liver Function Test (LFT)**

Bilirubin Total Method : Photometric	0.5	mg/dL	0.2 - 1.2
Bilirubin Direct * Method : Diazo Reaction	0.2	mg/dL	0.0 - 0.5
Bilirubin Indirect * Method : Calculation (T Bil - D Bil)	0.3	mg/dL	0.1 - 1.0
SGOT/AST Method : IFCC without P5P	12	U/L	5 - 34
SGPT/ALT Method : IFCC without P5P	9	U/L	0 to 55
SGOT/SGPT Ratio *	1.33	-	-
Alkaline Phosphatase Method : IFCC	53	U/L	40 - 150
Total Protein Method : Biuret	6.7	g/dL	6.4 - 8.3
Albumin Method : BCG	4.07	gm/dL	3.8 - 5.0
Globulin * Method : Calculation (T.P - Albumin)	2.63	g/dL	2.3 - 3.5
Albumin :Globulin Ratio * Method : Calculation (Albumin/Globulin)	1.55	-	1.0 - 2.1
Gamma Glutamyl Transferase (GGT) * Method : Photometric	15	U/L	9 to 36

**Interpretation:**

The liver filters and processes blood as it circulates through the body. It metabolizes nutrients, detoxifies harmful substances, makes blood clotting proteins, and performs many other vital functions. The cells in the liver contain proteins called enzymes that drive these chemical reactions. When liver cells are damaged or destroyed, the enzymes in the cells leak out into the blood, where they can be measured by blood tests Liver tests check the blood for two main liver enzymes. Aspartate aminotransferase (AST),SGOT: The AST enzyme is also found in muscles and many other tissues besides the liver. Alanine aminotransferase (ALT), SGPT: ALT is almost exclusively found in the liver. If ALT and AST are found together in elevated amounts in the blood, liver damage is most likely present. Alkaline Phosphatase and GGT: Another of the liver's key functions is the production of bile, which helps digest fat. Bile flows through the liver in a system of small tubes (ducts), and is eventually stored in the gallbladder, under the liver. When bile flow is slow or blocked, blood levels of certain liver enzymes rise: Alkaline phosphatase Gamma-utanyl transeptidase (GGT) Liver tests may check for any or all of these enzymes in the blood. Alkaline phosphatase is by far the most commonly tested of the three. If alkaline phosphatase and GGT are elevated, a problem with bile flow is most likely present. Bile flow problems can be due to a problem in the liver, the gallbladder, or the tubes connecting them. Proteins are important building blocks of all cells and tissues. Proteins are necessary for your body's growth, development, and health. Blood contains two classes of protein, albumin and globulin. Albumin proteins keep fluid from leaking out of blood vessels. Globulin proteins play an important role in your immune system. Low total protein may indicate: 1.bleeding 2.liver disorder 3.malnutrition 4.agammaglobulinemia High Protein levels Hyperproteinemia: May be seen in dehydration due to inadequate water intake or to excessive water loss (eg, severe vomiting, diarrhea, Addison's disease and diabetic acidosis) or as a result of increased production of proteins Low albumin levels may be caused by: 1.A poor diet (malnutrition). 2.Kidney disease. 3.Liver disease. High albumin levels may be caused by: Severe dehydration.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Kidney Function Test (KFT)**

Blood Urea Method : Urease	21	mg/dL	19 - 44.1
Creatinine Method : Photometric	0.8	mg/dL	0.57 - 1.11
Bun * Method : Urease	9.81	mg/dL	7.0 - 18.7
Bun/Creatinine Ratio *	12.26		
Urea / Creatinine Ratio *	26.25		
Uric Acid Method : Uricase	4.7	mg/dL	2.6 - 6.0
Calcium Serum Method : Arsenazo III	9.2	mg/dL	8.4 - 10.2
Phosphorus Method : Photometric	3.2	mg/dL	2.3 - 4.7
Sodium Method : Potentiometric	140	mmol/L	136 - 145
Potassium Method : Potentiometric	3.7	mmol/L	3.5 - 5.1
Chloride Method : Potentiometric	105	mmol/L	98 - 107

**Interpretation:**

Kidney function tests is a collective term for a variety of individual tests and procedures that can be done to evaluate how well the kidneys are functioning. Many conditions can affect the ability of the kidneys to carry out their vital functions. Some lead to a rapid (acute) decline in kidney function others lead to a gradual (chronic) decline in function. Both result in a buildup of toxic waste substances done on urine samples, as well as on blood samples. A number of symptoms may indicate a problem with your kidneys. These include : high blood pressure, blood in urine frequent urges to urinate, difficulty beginning urination, painful urination, swelling in the hands and feet due to a buildup of fluids in the body. A single symptom may not mean something serious. However, when occurring simultaneously, these symptoms suggest that your kidneys are not working properly. Kidney function tests can help determine the reason. Electrolytes (sodium, potassium, and chloride) are present in the human body and the balancing act of the electrolytes in our bodies is essential for normal function of our cells and organs. There has to be a balance. Ionized calcium this test if you have signs of kidney or parathyroid disease. The test may also be done to monitor progress and treatment of these diseases.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**

Lipid Profile

Total Cholesterol Method : Enzymatic - Cholesterol Oxidase	167	mg/dL	Desirable : <200 Borderline : 200-239 High : >240
Triglycerides Method : Colorimetric - Lip/Glycerol Kinase	82	mg/dL	<150
HDL Cholesterol Method : Accelerator Selective Detergent	55	mg/dL	>40
Non HDL Cholesterol * Method : Calculated	112	mg/dL	<130
LDL Cholesterol * Method : Calculated	95.6	mg/dL	<100
V.L.D.L Cholesterol * Method : Calculated	16.4	mg/dL	< 30
Chol/HDL Ratio * Method : Calculated	<b>3.04</b>	Ratio	3.5 - 5.0
HDL/ LDL Ratio * Method : Calculated	0.58	Ratio	0.5 - 3.0
LDL/HDL Ratio * Method : Calculated	1.74	Ratio	-

**Interpretation:**

Lipid level assessments must be made following 9 to 12 hours of fasting, otherwise assay results might lead to erroneous interpretation. NCEP recommends of 3 different samples to be drawn at intervals of 1 week for harmonizing biological variables that might be encountered in single assays.

National Lipid Association Recommendations (NLA-2014)	Total Cholesterol (mg/dL)	Triglyceride (mg/dL)	LDL Cholesterol (mg/dL)	Non HDL Cholesterol (mg/dL)
Optimal	<200	<150	<100	<130
Above Optimal			100-129	130 - 159
Borderline High	200-239	150-199	130-159	160 - 189
High	>=240	200-499	160-189	190 - 219
Very High	-	>=500	>=190	>=220

HDL Cholesterol	
Low	High
<40	>=60

**Risk Stratification for ASCVD (Atherosclerotic Cardiovascular Disease) by Lipid Association of India.**

Risk Category	Description
A. CAD with > 1 feature of high risk group	
Extreme risk group	B. CAD with >1 feature of very high risk group of recurrent ACS (within 1 year) despite LDL-C <or = 50 mg/dl or poly vascular disease

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# LABORATORY REPORT

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 DOB/Age/Gender :  
 Patient ID / UHID : XXX/OFXXX  
 Referred By :  
 Sample Type :  
 Barcode No :

Bill Date :  
 Sample Collected :  
 Sample Received :  
 Report Date :  
 Report Status :



NABL-M(EL)T-00726

Test Description	Value(s)	Unit(s)	Reference Range
<b>Very High Risk</b>	1.Established ASCVD 2.Diabetes with 2 major risk factors of evidence of end organ damage 3. Familial Homozygous Hypercholesterolemia		
<b>High Risk</b>	1. Three major ASCVD risk factors 2. Diabetes with 1 major risk factor or no evidence of end organ damage 3. CHD stage 3B or 4. 4 LDL >190 mg/dl 5. Extreme of a single risk factor 6. Coronary Artery Calcium - CAC > 300 AU 7. Lipoprotein a >= 50 mg/dl 8. Non stenotic carotid plaque		
<b>Moderate Risk</b>	2 major ASCVD risk factors		
<b>Low Risk</b>	0-1 major ASCVD risk factors		
<b>Major ASCVD (Atherosclerotic cardiovascular disease) Risk Factors</b>			
1. Age >=45 years in Males & >= 55 years in Females	3. Current Cigarette smoking or tobacco use		
2. Family history of premature ASCVD	4. High blood pressure		
5. Low HDL			

Newer treatment goals and statin initiation thresholds based on the risk categories proposed by Lipid Association of India in 2020.

Risk Group	Treatment Goals		Consider Drug Therapy	
	LDL-C (mg/dl)	Non-HDL (mg/dl)	LDL-C (mg/dl)	Non-HDL (mg/dl)
Extreme Risk Group Category A	<50 (Optional goal <OR = 30)	<80 (Optional goal <OR = 60)	>OR = 50	>OR = 80
Extreme Risk Group Category B	>OR = 30	>OR = 60	> 30	> 60
Very High Risk	<50	<80	>OR = 50	>OR = 80
High Risk	<70	<100	>OR = 70	>OR = 100
Moderate Risk	<100	<130	>OR = 100	>OR = 130
Low Risk	<100	<130	>OR = 130*	>OR = 160

\* After an adequate non-pharmacological intervention for at least 3 months.

References : Management of Dyslipidaemia for the Prevention of Stroke : Clinical practice Recommendations from the Lipid Association of India. Current Vascular Pharmacology,2022,20,134-155.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Iron Studies**

Iron Method : Ferene	49.8	µg/dL	50 - 170
TIBC,(Total Iron Binding Capacity) Method : Calculated	336.2	µg/dL	250 - 450
UIBC Method : Ferene	286.4	µg/dL	70 - 310
Transferrin Saturation Method : Method :Derived from IRON and TIBC values	14.81	%	14-50

**Interpretation:**

Increased levels due to iron ingestion or ineffective erythropoiesis. Decreased levels due to infection, inflammation, malignancy, menstruation and Fe deficiency. Needs to be taken into consideration with TIBC. Transferrin Saturation:- Low level Transferrin Saturation can indicate iron deficiency, erythropoiesis, infection, or inflammation. High level Transferrin Saturation can indicate recent ingestion of dietary iron, ineffective erythropoiesis, haemochromatosis or liver disease. High TIBC, UIBC, or transferrin usually indicates iron deficiency, but they are also increased in pregnancy and with the use of oral contraceptives. Low TIBC, UIBC, or transferrin may occur if someone has: Hemochromatosis, Certain types of anemia due to accumulated iron, Malnutrition, kidney disease that causes a loss of protein in urine.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**C-Reactive Protein (CRP), Quantitative**

CRP (Quantitative) Method : Immunoturbidimetry	2	mg/L	up to 5
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**Interpretation:**

**Increased CRP level:**

1. A high or increasing amount of CRP in the blood suggests the presence of inflammation but will not identify its location or the cause.
2. Suspected bacterial infection—a high CRP level can provide indication that patient has an infection.
3. Chronic inflammatory disease—high levels of CRP suggest a flare-up if you have a chronic inflammatory disease or that treatment has not been effective.

If the CRP level is initially elevated and drops, it means that the inflammation or infection is subsiding and/or responding to treatment.

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DOB/Age/Gender	:	Sample Collected	:
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Sample Type	:	Report Status	:
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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Rheumatoid Factor (RF), Quantitative**

RHEUMATOID FACTOR, Quantitative Method : Immunoturbidimetry	6.1	IU/mL	Negative <30 Weakly positive 30 to 50 Positive >50
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**Interpretation:**

Approximately 85% of patients with Rheumatoid arthritis have detectable RA. It may also be seen in other medical conditions like Sjogren's syndrome and SLE.

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Patient ID / UHID	: XXX/OFXXX	Sample Received	:
Referred By	:	Report Date	:
Sample Type	:	Report Status	:
Barcode No	:		

Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Vitamin B12 / Cyanocobalamin**

Vitamin - B12	303.6	pg/mL	197 - 771
Method : ECLIA			

**Interpretation:**

Low Values are a sign of a vitamin B12 deficiency. People with this deficiency are likely to have or develop symptoms.

Causes of vitamin B12 deficiency include: Not enough vitamin B12 in diet (rare except with a strict vegetarian diet), Diseases that cause malabsorption (for example, celiac disease and Crohn's disease), Lack of intrinsic factor, Above normal heat production (for example, with hyperthyroidism), Pregnancy. Increased vitamin B12 levels are uncommon. Usually excess vitamin B12 is removed in the urine. Conditions that can increase B12 levels include: Liver disease (such as cirrhosis or hepatitis), Myeloproliferative disorders (for example, polycythemia vera and chronic myelocytic leukemia).

Vitamin B12: Low Levels can cause malabsorption, Lack of intrinsic factor, Above normal heat production (for example, with hyperthyroidism), Pregnancy. High Level Liver disease, Myeloproliferative disorders (for example, polycythemia vera and chronic myelocytic leukemia).

1. Out of 140 healthy indian population, 91% of Vitamin B 12 concentrations was at lower level: 59.00 pg/ml and upper level: 700.00 pg/ml

"Patients on Biotin supplement may have interference in some immunoassays. Ref: Arch Pathol Lab Med—Vol 141, November 2017. With individuals taking high dose Biotin (more than 5 mg per day) supplements, at least 8-hour wait time before blood draw is recommended."

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Vitamin D 25 Hydroxy**

Vitamin D 25 - Hydroxy Method : ECLIA	<b>21.45</b>	ng/mL	Deficiency : <30 ng/mL
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**Interpretation:**

25-Hydroxy vitamin D represents the main body reservoir and transport form. Mild to moderate deficiency is associated with Osteoporosis / Secondary Hyperparathyroidism while severe deficiency causes Rickets in children and Osteomalacia in adults. Prevalence of Vitamin D deficiency is approximately >50% specially in the elderly. This assay is useful for diagnosis of vitamin D deficiency and Hypervitaminosis D. It is also used for differential diagnosis of causes of Rickets & Osteomalacia and for monitoring Vitamin D replacement therapy.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**

**Thyroid Profile Total**

Triiodothyronine (T3) Method : ECLIA	1.1	ng/mL	0.80 - 2.00
TotalThyroxine (T4) Method : ECLIA	8.3	µg/dL	5.1 - 14.1
Thyroid Stimulating Hormone (Ultrasensitive) Method : ECLIA	5.2	mIU/L	0.27 - 4.20

**Interpretation:**

Pregnancy	Reference ranges TSH
1 st Trimester	0.1 - 2.5
2 ed Trimester	0.2 - 3.0
3 rd Trimester	0.3 - 3.0

Primary malfunction of the thyroid gland may result in excessive (hyper) or below normal (hypo) release of T3 or T4. In addition as TSH directly affects thyroid function, malfunction of the pituitary or the hypo - thalamus influences the thyroid gland activity. Disease in any portion of the thyroid-pituitary-hypothalamus system may influence the levels of T3 and T4 in the blood. In primary hypothyroidism, TSH levels are significantly elevated, while in secondary and tertiary hypothyroidism, TSH levels may be low. In addition, in the Euthyroid Sick Syndrome, multiple alterations in serum thyroid function test findings have been recognized in patients with a wide variety of non-thyroidal illnesses (NTI) without evidence of preexisting thyroid or hypothalamic-pituitary diseases. Thyroid Binding Globulin (TBG) concentrations remain relatively constant in healthy individuals. However, pregnancy, excess estrogen's, androgen's, antibiotic steroids and glucocorticoids are known to alter TBG levels and may cause false thyroid values for Total T3 and T4 tests.

TSH	T4	T3	INTERPRETATION
High	Normal	Normal	Mild (subclinical) hypothyroidism
High	Low	Low or normal	Hypothyroidism
Low	Normal	Normal	Mild (subclinical) hyperthyroidism
Low	High or normal	High or normal	Hyperthyroidism
Low	Low or normal	Low or normal	Nonthyroidal illness; pituitary (secondary) hypothyroidism
Normal	High	High	Thyroid hormone resistance syndrome (a mutation in the thyroid hormone receptor decreases thyroid hormone function)

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DOB/Age/Gender	:	Sample Collected	:
Patient ID / UHID	: XXX/OFXXX	Sample Received	:
Referred By	:	Report Date	:
Sample Type	:	Report Status	:
Barcode No	:		

Test Description	Value(s)	Unit(s)	Reference Range
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## BIOCHEMISTRY REPORT

### Anti Thyroid Peroxidase Antibodies (TPO)

ANTI THYROID PEROXIDASE ANTIBODY;(ANTI TPO), SERUM	< 3.00	IU/mL	< 5.61
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Method : CMIA

#### Interpretation:

#### Note:

Thyroid Peroxidase antibodies may be detected in individuals without clinically significant thyroid disease. They do not define the patient's thyroid functional status. Anti TPO is technically superior and a more specific method for measuring thyroid antibodies. It is especially useful in patients presenting with subclinical hypothyroidism where TSH is elevated but free T4 levels are normal.

#### Clinical Use

1. Confirm presence of Autoimmune thyroid disease

#### Increased Levels

1. Hashimoto thyroiditis
2. Graves disease
3. Postpartum thyroiditis
4. Primary hypothyroidism due to Hashimoto thyroiditis

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DOB/Age/Gender	:	Sample Collected	:
Patient ID / UHID	: XXX/OFXXX	Sample Received	:
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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Immunoglobulin E (IgE Total)**

IMMUNOGLOBULIN IgE TOTAL SERUM	87.69	IU/mL	28 - 140
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Method : ECLIA

**Interpretation:**

The level of serum IgE rises during childhood and reaches adult levels during the teens. IgE is the mediator of the allergic response. Patients with atopic disease, including allergic asthma, allergic rhinitis, and atopic dermatitis commonly have moderately elevated serum IgE levels. Total serum IgE levels may also be elevated in the presence of some clinical conditions that are not related to allergy. These clinical conditions include parasitic infections, immunodeficiency states, autoimmune diseases, Hodgkins disease, bronchopulmonary aspergillosis, IgE myeloma, and Sezary syndrome.

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Test Description	Value(s)	Unit(s)	Reference Range
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**BIOCHEMISTRY REPORT**  
**Winter Special Pro Package**  
**Apolipoproteins A1 & B**

Apolipoprotein A-1 (APO-A) Method : Tina-quant	152		104 - 202
Apolipoprotein B (APO-B) Method : Tina-quant	79	mg/dL	66 - 144
Apo B / Apo A1 Ratio	0.52		0.35 - 0.98

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*Sohini Sengupta*

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 Medical Laboratory Director  
 HOD (Biochemistry & Special Assays)

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 Processing Lab :- Redcliffe Lifetech Pvt. Ltd., H-55, Sector-63, Noida, Uttar Pradesh - 201301

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**SEROLOGY AND IMMUNOLOGY REPORT**  
**Anti Nuclear Antibody (ANA) By IFA (HEP-2)**

Anti Nuclear Antibody by IFA	Negative	Negative
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**Interpretation:**

**Guidelines (Sample screening Dilution - 1:100):**

- Negative : No Immunofluorescence
- + : Weak Positive
- ++ : Moderate Positive
- +++ : Strong Positive
- ++++ : Very strong Positive

**Test Description:** Antinuclear antibodies (ANAs) are unusual antibodies, detectable in the blood, that have the capability of binding to certain structures within the nucleus of the cells. ANAs indicate the possible presence of autoimmunity & provide, therefore, an indication of autoimmune illness. Fluorescence tech. are frequently used to actually detect the antibodies in the cells, thus ANA testing is sometimes referred to as fluorescent antinuclear antibody test (FANA). The ANA test is a sensitive screening test used to detect autoimmune diseases

**Technique:** Indirect Immunofluorescence.

The BIOCHIP Slide is a combination of Hep-20-10 cells and primate liver and has the following advantages.

1. It is a global standard tech. with a natural antigen spectrum capable of detecting more than 30 diagnostically relevant auto antibodies.
2. Hep 20-10 cell lines contain 40% mitotic cells, facilitating easier identification of rare patterns.
3. If the test is negative, detectable level of auto antibodies is ruled out. In case of a positive result, autoantibodies against any one or in some cases simultaneously against more than one antigens may be present and further monospecific tests or panel of profiles can be used to determine the specific autoantibodies present.

**NOTE-** All weak positive (+) results may be repeated after 6 - 8 weeks. **Associated Tests:** Monospecific ELISA to define single antigens, ANA Immunoblot assay.

**Abbreviations:** SLE: Systemic Lupus Erythematosus, SCL: Scleroderma, MCTD: Mixed Connective Tissue Disease; CFS: Chronic Fatigue Syndrome; AIH: Autoimmune Hepatitis, PBC: Primary Biliary Cirrhosis, PM: Polymyositis, DM: Dermatomyositis, SS: Systemic sclerosis, RA: Rheumatoid Arthritis.

Please view next page for co-relation table including various single antigens with their Immunofluorescence patterns and clinical associations

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Location	Pattern	Target Antigen	Clinical Association
Nucleus	Homogeneous	Double strand DNA Histones Nucleosome, RNA, Single Strand DN	SLE Drug Induced Lupus, SLE , RA SLE, MCTD, RA, PM, DM, SS
	Speckled	Sm U1-snRNP SSA/Ro SSB/La Ku Cyclin1(PCNA) Mitosin/Cyclin II	SLE MCTD, SLE, RA, sharp syndrome Sjogren`s syndromes (SS)/SLE/Neonatal Lupus PM/DM/SLE/SS SLE/Overlap Syndromes DM
	Dense Fine Speckled(DFS)	Lens epithelium-derived growth factor (LEDGF), DNA binding transcription coactivator p75.(DFS-70)	Healthy individuals, Various Inflammatory conditions like atopic dermatitis, interstitial cystitis, Asthma.
	Centomeres	Proteins of Kinetochores	CREST syndrome, PSS limited form
	Nuclear Dots	Sp-100 , NDP53	PBC, Rheumatic Disease
	Nuclear Membrane	Lamins, gp210, p62	CFS, Collagenoses, PBC, AIH
Nucleolus	Nucleolar homogeneous	PM-Scl Scl-70	PM, DM, PSS(Diffuse) PSS(Diffuse)
	Nucleolar speckled	RNA-Polymerase I / NOR-90	Progressive Systemic Sclerosis(Diffuse)
	Nucleolar Pattern	Fibrillarin	Progressive Systemic Sclerosis(Diffuse)
Cytoplasm	Cytoplasmic speckled	Mitochondrial Lysosomal Golgi Complex Ribosome P Jo -1 SRP, PL12, TIF1-Gamma	PBC, Unknown SS/SLE/RA SLE Polymyositis (PM), PM/ DM, Myositis
	Cytoplasmic filament	F-Actin Vimentin Tropomyosin Cytoplasmic Rings & rods	AIH Unknown Unknown HCV Infection- on therapy
Cell Cycle (mitotic cells)	Centriole Mid-Body Spindle Fibres	-- -- --	Unknown Unknown Rheumatic Disease

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### SEROLOGY AND IMMUNOLOGY REPORT

#### Torch Panel IgG & IgM (10 Parameters)

Toxoplasma IgG Method : CLIA	0.003	IU/mL	Non-Reactive <0.81 IU/mL Equivocal 0.81 - 1.2 IU/mL Reactive >1.2 IU/mL
Toxoplasma IgM Method : CLIA	0.001	AU/mL	Non-Reactive <6 AU/mL Equivocal 6 - 10 AU/mL Reactive >10 AU/mL
Rubella IgG Method : CLIA	<b>76.947</b>	IU/mL	Non-Reactive <5 IU/mL Equivocal 5 - 10 IU/mL Reactive >10 IU/mL
Rubella IgM Method : CLIA	0.001	AU/mL	Non-Reactive <5 AU/mL Equivocal 5 - 10 AU/mL Reactive >10.0 AU/mL
Cytomegalovirus, IgG Method : CLIA	<b>517.442</b>	AU/mL	Non-Reactive <10 AU/mL Equivocal 10 - 14 AU/mL Reactive >14 AU/mL
Cytomegalovirus ,IgM Method : CLIA	0.001	AU/mL	Non-Reactive <8 AU/mL Equivocal 8 - 12 AU/mL Reactive >12 AU/mL
Herpes simplex virus-1 IgG Method : CLIA	0.006	AU/mL	Non-Reactive <14 AU/mL Equivocal 14 - 19 AU/mL Reactive >19 AU/mL
Herpes simplex virus-1 IgM Method : CLIA	0.001	AU/mL	Non-Reactive <6 AU/mL Equivocal 6 - 10 AU/mL Reactive >10 AU/mL
Herpes simplex virus-2 IgG Method : CLIA	0.001	AU/mL	Non-Reactive <9 AU/mL Equivocal 9 - 13 AU/mL Reactive >13 AU/mL
Herpes simplex virus-2 IgM Method : CLIA	0.001	AU/mL	Non-Reactive <6 AU/mL Equivocal 6 - 10 AU/mL Reactive >10 AU/mL

#### **Interpretation:** **TORCH IgG**

1. Positive result indicates past infection with TORCH. Pregnant females with positive TORCH specific IgG antibodies are considered to be immune and hence risk of transmission of infection to fetus is minimal.
2. Equivocal results should be re-tested in 10-14 days.
3. Negative result indicates person has not been exposed to TORCH in the past. Pregnant females with negative TORCH specific IgG antibodies are considered at risk of transmission of infection to fetus. Patients with negative results in suspected disease should be re-tested after 10-14 days. False negative results can be due to immunosuppression or due to low/undetectable level of IgG antibodies.
4. To differentiate between recent and past infection, Toxoplasma, Rubella & CMV IgG avidity test is indicated.
5. Demonstration of rising antibody titer (four folds) in acute and convalescent sera taken 2-3 weeks apart are indicative of TORCH infection.
6. The result should be interpreted in conjunction with clinical finding and other diagnostic tests. The magnitude of the measured result is not indicative of the amount of antibody present.

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Test Description	Value(s)	Unit(s)	Reference Range
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### TORCH IgM

1. Positive result for TORCH IgM indicates possible acute infection with TORCH. False positive reaction due to rheumatoid factor and persistence of positive IgM (except Herpes Simplex virus) for upto 2 years is not uncommon.
2. An equivocal result requires repeat testing in 10-14 days.
3. Negative result indicates no serological evidence of infection with TORCH. False negative can be due to immunosuppression or due to low/undetectable level of IgM antibodies. A suspected diagnosis of acute TORCH infection should be confirmed by PCR analysis or repeat test after 10-14 days.
4. The diagnosis should not be established on the basis of single test and the results should be interpreted in conjunction with clinical findings.
5. The magnitude of the measured result is not indicative of the amount of antibody present.

### Comments

Perinatal infections account for 2-3% of all congenital anomalies. TORCH which includes Toxoplasma, Rubella, Cytomegalovirus & Herpes Simplex virus, are some of the most common infections associated with Congenital anomalies. Most of the TORCH infections cause mild maternal morbidity but have serious fetal consequences. Reliable recognition of acute infection is highly important in pregnant women. IgM-positive result alone does not accurately predict the risk of fetal infection; a positive IgM test should therefore be considered only as a starting point and a more thorough diagnostic evaluation is necessary to determine whether there is a risk of fetal infection. Primary CMV infection may result in establishment of persistent or latent infection. In man the infection is usually asymptomatic. Infections can be acquired through direct contact with individuals shedding the virus. Once HSV infection occurs, it persists in a latent state in sensory ganglia from where it may re-emerge to cause periodic recurrence of infection induced by many stimuli, which may or may not result in clinical lesions. Demonstration of Toxoplasma IgG in the serum of person with eye lesion helps in diagnosing ocular toxoplasmosis while persistent or increasing IgG antibody levels in the infant compared with the mother and/or positive result of Toxoplasma specific IgM or IgA are diagnostic of Congenital toxoplasmosis. Demonstration of rising antibody titer (four folds) in acute and convalescent sera taken 2-3 weeks apart are indicative of postnatal Rubella infection and to check response to Rubella vaccination. Single test results of CMV IgG are useful in screening organ transplant recipients and donors before transplantation and donors of blood products that are to be administered to premature infants and bone marrow transplant patients. Positive result of HSV (1+2) IgG indicates past infection with Herpes Simplex virus or administration of HSV immunoglobulins. Reliable recognition of acute infection is highly important in pregnant women. IgM-positive result alone does not accurately predict the risk of fetal infection; a positive IgM test should therefore be considered only as a starting point and a more thorough diagnostic evaluation is necessary to determine whether there is a risk of fetal infection.

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## SEROLOGY AND IMMUNOLOGY REPORT

### Anti Phospholipid Antibody (APLA) Panel

#### Anti Cardiolipin IgG Antibodies

Cardiolipin Antibody ACL- IgG <3.0 GPLU/ml < 12.0 GPLU/ml  
 Method : (Serum,EIA)

#### Interpretation:

RESULT IN GPLU/ml	REMARKS
< 11.9	Negative
≥ 12.0-17.9	Equivocal
≥ 18.0	Positive

#### Comments

Antibodies against cardiolipin belong to the group of anti-phospholipid antibodies specific for negatively charged phospholipids, components of biological membranes. Cardiolipin is an acidic phospholipid derived from glycerol. Antiphospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The prevalence of anti-cardiolipin antibodies in SLE is 24-50%. The occurrence of anti-cardiolipin antibodies in patients with SLE and related diseases is typical of a secondary anti-phospholipid syndrome (APS). In contrast, anti-cardiolipin antibodies in patients with no other autoimmune diseases characterize the primary anti-phospholipid syndrome (APS). Many studies have shown a correlation between these autoantibodies and an enhanced incidence of thrombosis, thrombocytopenia and habitual abortions (as a consequence of placental infarct). The exact mechanism by which pathogenic anti-phospholipid antibodies induce thrombosis is not yet fully revealed.

## SEROLOGY AND IMMUNOLOGY REPORT

### Anti Phospholipid Antibody (APLA) Panel

#### Anti Cardiolipin IgM Antibodies

Cardiolipin Antibody ACL- IgM 4.6 MPLU/ml < 12.0 MPLU/ml  
 Method : (Serum,EIA)

#### Interpretation:

RESULT IN MPLU/ml	REMARKS
< 11.9	Negative
≥ 12.0-17.9	Equivocal
≥ 18.0	Positive

#### Comments

Antibodies against cardiolipin belong to the group of anti-phospholipid antibodies specific for negatively charged phospholipids, components of biological membranes. Cardiolipin is an acidic phospholipid derived from glycerol. Antiphospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The prevalence of anti-cardiolipin antibodies in SLE is 24-50%. The occurrence of anti-cardiolipin antibodies in patients with SLE and related diseases is typical of a secondary anti-phospholipid syndrome (APS). In contrast, anti-cardiolipin antibodies in patients with no other autoimmune diseases characterize the primary anti-phospholipid syndrome (APS). Many studies have shown a correlation between these autoantibodies and an enhanced incidence of thrombosis, thrombocytopenia and habitual abortions (as a consequence of placental infarct). The exact mechanism by which pathogenic anti-phospholipid antibodies induce thrombosis is not yet fully revealed.

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Test Description	Value(s)	Unit(s)	Reference Range
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### SEROLOGY AND IMMUNOLOGY REPORT

#### Anti Phospholipid Antibody (APLA) Panel

##### Beta 2 Glycoprotein IgG

Beta-2-Glycoprotein 1 -IgG 4.9 AU/ml  
 Method : (Serum,EIA)

Negative: < 11.9  
 Equivocal: 12.0-17.9  
 Positive: >= 18.0

#### Interpretation:

RESULT IN AU/ml	REMARKS
<11.9	Negative
≥12.0-17.9	Equivocal
≥ 18.0	Positive

#### COMMENTS

Antibodies against β2-glycoprotein I belong to the group of anti-phospholipid antibodies mainly targeted against complexes composed of negatively charged phospholipids (e.g. cardiolipin) and plasma proteins like β2-glycoprotein I, prothrombin, protein C or protein S. Reactivity against isolated β2-glycoprotein I is found, too. Thus β2-glycoprotein I is discussed to be an autoantigen on its own. β2-glycoprotein I, also called apolipoprotein H, is a 50 kDa beta-2 globulin which is associated in vivo with lipoprotein, platelets and phospholipids and which seems to inhibit the intrinsic coagulation pathway, the prothrombinase activity and the ADP-dependent platelet aggregation. Anti-phospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus and related diseases and are typical of the secondary development of an antiphospholipid syndrome (APS). Anti-phospholipid antibodies in patients with no other autoimmune diseases, on the other hand, characterize a primary APS. Many studies have shown a correlation between these autoantibodies and an enhanced incidence of thrombosis, thrombocytopenia and habitual abortions (as a consequence of placental infarction). The exact mechanism by which pathogenic anti-phospholipid antibodies induce thrombosis has not yet been revealed.

### SEROLOGY AND IMMUNOLOGY REPORT

#### Anti Phospholipid Antibody (APLA) Panel

##### Beta 2 Glycoprotein IgM

Beta-2-Glycoprotein IgM Antibody <3.0 AU/ml  
 Method : (Serum,EIA)

Negative: < 11.9  
 Equivocal: 12.0-17.9  
 Positive: >= 18.0

#### Interpretation:

RESULT IN AU/ml	REMARKS
<11.9	Negative
≥12.0-17.9	Equivocal
≥ 18.0	Positive

#### COMMENTS

Antibodies against β2-glycoprotein I belong to the group of anti-phospholipid antibodies mainly targeted against complexes composed of negatively charged phospholipids (e.g. cardiolipin) and plasma proteins like β2-glycoprotein I, prothrombin, protein C or protein S. Reactivity against isolated β2-glycoprotein I is found, too. Thus β2-glycoprotein I is discussed to be an autoantigen on its own. β2-glycoprotein I, also called apolipoprotein H, is a 50 kDa beta-2 globulin which is associated in vivo with lipoprotein, platelets and

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## SEROLOGY AND IMMUNOLOGY REPORT

### Anti Phospholipid Antibody (APLA) Panel

#### Anti Phospholipid IgG Antibodies

PHOSPHOLIPID ANTIBODY, IgG, SERUM 0.25 U/mL <12.00  
 Method : (EIA)

#### Interpretation:

RESULT IN U/ml	REMARKS
< 12	Negative
12.00-18.00	Borderline
>18.00	Positive

**NOTE-The assay is an aid in the diagnosis and risk estimation of thrombosis in patients with systemic lupus erythematosus and antiphospholipid syndrome (APS).**

Phospholipid-Screen-IgG is a solid phase enzyme immunoassay for the quantitative detection of IgG against phospholipids in human serum. Antibodies against phospholipids, components of the biological membranes, are specific for phospholipids such as Cardiolipin, Phosphatidyl -inositol, -ethanolamine, - serine, -choline and Sphingomyelin. Anti-phospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The occurrence of anti-phospholipid antibodies in patients with SLE and related diseases is typical for a secondary anti-phospholipid syndrome (APS). In contrast, anti-phospholipid antibodies in patients with no other autoimmune diseases characterize the primary APS.

## SEROLOGY AND IMMUNOLOGY REPORT

### Anti Phospholipid Antibody (APLA) Panel

#### Anti Phospholipid IgM Antibodies

PHOSPHOLIPID ANTIBODY, IgM, SERUM 0.32 U/mL <12.00  
 Method : (EIA)

#### Interpretation:

RESULT IN U/ml	REMARKS
< 12	Negative
12.00-18.00	Borderline
>18.00	Positive

**NOTE-The assay is an aid in the diagnosis and risk estimation of thrombosis in patients with systemic lupus erythematosus and antiphospholipid syndrome (APS).**

Phospholipid-Screen-IgM is a solid phase enzyme immunoassay for the quantitative detection of IgM against phospholipids in human serum. Antibodies against phospholipids, components of the biological membranes, are specific for phospholipids such as Cardiolipin, Phosphatidyl -inositol, -ethanolamine, - serine, -choline and Sphingomyelin. Anti-phospholipid antibodies are frequently found in sera of patients with systemic lupus erythematosus (SLE) and related diseases. The occurrence of anti-phospholipid antibodies in patients with SLE and related diseases is typical for a secondary anti-phospholipid syndrome (APS). In contrast, anti-phospholipid antibodies in patients with no other autoimmune diseases characterize the primary APS.

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### SEROLOGY AND IMMUNOLOGY REPORT

#### Anti Double Stranded DNA (DsDNA) BY IFA

Anti Double Stranded DNA (DsDNA) BY IFA Method : Indirect Immunofluorescence	NEGATIVE		Negative
Titre	1:10		

#### Interpretation:

1. Autoimmune reactivities are not by themselves diagnostic, but must be correlated with other laboratory & clinical findings
2. Test conducted on Serum

#### Comments

Anti ds-DNA antibodies are detected more frequently and at higher titres in Systemic lupus erythematosus (SLE) patients with Lupus nephritis. Presence of these antibodies or an increase in titre correlate with an increased risk of Lupus nephritis flare. Hence it is useful to monitor Anti ds -DNA antibody levels and initiate appropriate therapy when titres increase.

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**CLINICAL PATHOLOGY REPORT**  
**Winter Special Pro Package**  
**Urine Routine and Microscopic Examination**

**Physical Examination**

Volume *	10	ml	-
Colour *	Pale yellow	-	Pale yellow
Transparency *	Clear	-	Clear
Deposit *	Absent	-	Absent

**Chemical Examination**

Reaction (pH) Method : Double Indicator	6.5	-	4.5 - 8.0
Specific Gravity Method : Ion Exchange	1.010	-	1.010 - 1.030
Urine Glucose (sugar) Method : Oxidase / Peroxidase	Negative	-	Negative
Urine Protein (Albumin) Method : Acid / Base Colour Exchange	<b>Positive(Trace)</b>	-	Negative
Urine Ketones (Acetone) Method : Legals Test	Negative	-	Negative
Blood Method : Peroxidase Hemoglobin	Negative	-	Negative
Leucocyte esterase Method : Enzymatic Reaction	Negative	-	Negative
Bilirubin Urine Method : Coupling Reaction	Negative	-	Negative
Nitrite Method : Griess Test	Negative	-	Negative
Urobilinogen Method : Ehrlich's Test	Normal	-	Normal

**Microscopic Examination**

Pus Cells (WBCs) *	1-3	/hpf	0 - 5
Epithelial Cells *	2-4	/hpf	0 - 4
Red blood Cells *	Absent	/hpf	Absent
Crystals *	Absent	-	Absent
Cast *	Absent	-	Absent
Yeast Cells *	Absent	-	Absent
Amorphous deposits *	Absent	-	Absent
Bacteria *	Absent	-	Absent
Protozoa *	Absent	-	Absent

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🌐 [www.redcliffelabs.com](http://www.redcliffelabs.com)

All Lab results are subject to clinical interpretation by qualified medical professional and this report is not subject to use for any medico-legal purpose.

<b>Patient Name:</b>	Dummy	<b>Booking ID:</b>	XXX
<b>Age:</b>		<b>Sample Type:</b>	EDTA Whole Blood
<b>Gender:</b>		<b>Sample Collection Date:</b>	
<b>Referring Clinician:</b>	NA	<b>Sample Receiving Date:</b>	
<b>Test Requested:</b>	Factor II Prothrombin gene mutation analysis by PCR, Sanger Sequencing		<b>Reporting Date:</b>

## FACTOR II PROTHROMBIN GENE MUTATION ANALYSIS

### CLINICAL INDICATION

N/A

### RESULT SUMMARY

**NEGATIVE**  
(Not Detected)

### KEY FINDING

Target gene mutation	Mutation detection status	Relevance
Factor II - G20210A	Not Detected	None

### RESULT INTERPRETATION

No mutation was detected for the Prothrombin Factor II gene variant G20210A.

Result classification	Comment
Homozygous mutation detected	Both copies of the gene carry mutation
Heterozygous mutation detected	One copy of the gene carries mutation
Not Detected	Mutation not detected

### COMMENT

- ✓ Please correlate clinically.
- ✓ For about this report, or for assistance in locating nearby genetic counseling services, please contact the Laboratory: [geneticcounselors@redcliffelabs.com](mailto:geneticcounselors@redcliffelabs.com), or [ccsupport@redcliffelabs.com](mailto:ccsupport@redcliffelabs.com).

### CLINICAL SIGNIFICANCE

Thrombosis is the formation of a blood clots inside a blood vessel, obstructing the blood flow of the cardiovascular system. Several thrombosis associated single nucleotide polymorphisms (SNPs) have been identified and reported to significantly increase the risk of deep venous thrombosis (DVT). Prothrombin (Factor II) gene (G20210A) has been found to be associated with increased Prothrombin levels and an increase in the risk for venous thrombosis in heterozygous. Higher concentrations of Prothrombin lead to increased rates of thrombin generation, resulting in excessive growth of fibrin clots. Heterozygosity for 20210G>A is associated with a 3-fold increased risk of venous thrombosis. Affected individuals may be candidates for anti-thrombotic prophylaxis. Homozygous are rare but two copies of the mutation would increase that risk. When heterozygosity for 20210G>A is combined with heterozygosity for the Factor V Leiden mutation, the relative risk for thrombosis increases further. Combination with non-genetic risk factors such as use of oral contraceptives, also leads to substantial elevations in relative risk.

### TEST INFORMATION

This assay is based on DNA extracted from blood followed by PCR and targeted mutation Sanger Sequencing. The Test may be used for evaluation of patients with early onset VTE, as a thrombosis risk factor in patients prior to major surgery, to determine the cause of recurrent second or third trimester pregnancy loss, screening for risk of thrombosis before Oral contraceptive use and estrogen replacement therapy. Prothrombin G20210A mutation occurs in the noncoding region of the Factor II gene and is the second most common cause of inherited thrombophilia after FVL mutations. This test was developed and its analytical performance characteristics have been determined **by Redcliffe labs**. It has not been cleared or approved by FDA.

### TEST LIMITATIONS

- ✓ Test results may vary if appropriate sample collection and transportation to lab not followed as per protocol.
- ✓ Mutations below the detection limits of the assay may not be detected. Typical detection limit for Sanger Sequencing assays is >10-20%.
- ✓ This test is laboratory developed and its performance were evaluated at National Reference Lab, Redcliffe Labs.
- ✓ PCR is a highly sensitive technique; reasons for apparently contradictory results may be due to improper quality control during sample collection, selection of inappropriate specimen and/or presence of PCR inhibitors.
- ✓ This test detects only ONE variants in Factor II gene and report includes variants that meets a level of evidence threshold for cause or contribute to disease.
- ✓ If this mutation is not found by the testing procedure, it does not mean that the risk of carrying or developing deep vein thrombosis is not present. It simply means that this specific mutation has not been found, although other mutations may be present.
- ✓ False positive or false negative results may occur for reasons that include genetic variants, blood transfusions, bone marrow transplantation, somatic or tissue-specific mosaicism.
- ✓ Gene transcript used for clinical reporting generally represents the canonical transcript, which is usually the longest coding transcript with strong/multiple supporting evidence.

**DISCLAIMER**

- ❖ Test has been performed assuming that the sample received belongs to the above-named individual(s) and that any stated relationships between individuals are accepted as true.
- ❖ The results should be interpreted in the context of the patient's medical evaluation. Mutation identified in this gene does not guarantee activity of the drug in a given indication due to presence of contraindicated mutation in gene.
- ❖ The mutation information provided should only be utilized as a guide or aid and the decision to select any therapy option based on the information reported here resides solely with the discretion of the treating physician.
- ❖ This report should only be used as an aid and the treating physician should employ sound clinical judgment in arriving at any decision for patient care or treatment.

**REFERENCES**

1. Hussein et al., 2012. . Journal of Thrombosis and Thrombolysis, DOI: 10.1007/s11239-012-0731-9
2. Varga EA and Moll S. Prothrombin 20210 Mutation (Factor II Mutation). Circulation. (2004). 110:e15-e18.
3. Segers K, Dahlbäck B, Nicolaes GA. Coagulation factor V and thrombophilia: background and mechanisms. Thromb Haemost. (2007). 98(3):530-542.
4. Castoldi E, Simioni P, et al. Combinations of 4 mutations (FV R506Q, FV H1299R, FV Y1702C, PT 20210G/A) affecting the prothrombinase complex in a thrombophilic family. Blood. (2000). 96(4):1443-1448.



Reviewed by  
Imran Haider  
Senior Scientific Officer  
Onco-Genomics



Approved by  
Dr. Himani Pandey  
Postdoc-SGPGIMS Lucknow  
Lab Head-Clinical Genomics

<b>Patient Name:</b>	Dummy	<b>Booking ID:</b>	XXX
<b>Age:</b>	32 Years	<b>Sample Type:</b>	EDTA Whole Blood
<b>Gender:</b>	Female	<b>Sample Collection Date:</b>	09.03.2024
<b>Referring Clinician:</b>	NA	<b>Sample Receiving Date:</b>	11.03.2024
<b>Test Requested:</b>	Factor V Leiden Mutation Analysis by PCR,Sanger Sequencing	<b>Reporting Date:</b>	14.03.2024

## FACTOR V LEIDEN MUTATION ANALYSIS

### CLINICAL INFORMATION

N/A

### RESULT SUMMARY

**POSITIVE**  
**(Mutation Detected)**

### KEY FINDINGS

Target gene mutation	Mutation detection status	Relevance
Factor V - H1299R	Heterozygous mutation detected	Clinically significant
Factor V -R506Q	Not detected	None
Factor V -Y1702C	Not detected	None

### RESULT INTERPRETATION

Heterozygous mutation detected for Factor V gene variant H1299R. However, no mutation detected for Factor V gene R506Q and Factor V gene Y1702C in given specimen.

Result	Comment
Homozygous mutation detected	Both copies of the gene carry mutation
Heterozygous mutation detected	One copy of the gene carries mutation
Not Detected	Mutation not detected

### COMMENT

- ✓ Please correlate clinically.
- ✓ For about this report, or for assistance in locating nearby genetic counseling services, please contact the Laboratory: [geneticcounselors@redcliffelabs.com](mailto:geneticcounselors@redcliffelabs.com), or [ccsupport@redcliffelabs.com](mailto:ccsupport@redcliffelabs.com).

### CLINICAL INTERPRETATION

Thrombosis is the formation of a blood clots inside a blood vessel, obstructing the blood flow of the cardiovascular system. Several thrombosis associated single nucleotide polymorphisms (SNPs) have been identified and reported to significantly increase the risk of venous thrombosis. Three SNPs (R506Q, H1299R and Y1702C) in the Factor V gene are the most important genetic risk factors for inherited thrombophilia. Factor V mutation increases the relative risk of thrombosis by 5-10 fold in the heterozygous condition and by 50-100 fold in the homozygous individual. The lifetime risk for DVT is 12-20% for Heterozygote and 80% for Homozygote. Factor V mutation is a risk factor for venous as well as arterial thrombosis. It is the most common genetic risk factor for thrombosis and accounts for >90 percent of APC resistance.

### TEST INFORMATION

This assay is based on DNA extracted from blood followed PCR and Sanger Sequencing. It is used as a thrombosis risk factor in patients prior to major surgery, to determine the cause of recurrent second or third trimester pregnancy loss, screening for risk of thrombosis before oral contraceptive use, estrogen replacement therapy and for presymptomatic evaluation of individuals with a family history of thrombosis or a family member identified to have FV mutations. A mutational defect in factor V causes APC (Activated Protein C) resistance which can be homozygous or heterozygous. Factor V Leiden mutation is a risk factor for venous as well as arterial thrombosis.

### TEST LIMITATIONS

- ✓ Test results may vary if appropriate sample collection and transportation to lab not followed as per protocol.
- ✓ Mutations below the detection limits of the assay may not be detected. Typical detection limit for Sanger Sequencing assays is >10-20%.
- ✓ This test is laboratory developed and its performance were evaluated at National Reference Lab, Redcliffe Labs.
- ✓ PCR is a highly sensitive technique; reasons for apparently contradictory results may be due to improper quality control during sample collection, selection of inappropriate specimen and/or presence of PCR inhibitors.
- ✓ This test detects mutations only three target variants in Factor V gene and report includes only variants that meets a level of evidence threshold for cause or contribute to disease.
- ✓ Gene transcript used for clinical reporting generally represents the canonical transcript, which is usually the longest coding transcript with strong/multiple supporting evidence.

### DISCLAIMER

- ❖ Test has been performed assuming that the sample received belongs to the above-named individual(s) and that any stated relationships between individuals are accepted as true.
- ❖ The results should be interpreted in the context of the patient's medical evaluation. Mutation identified in this gene does not guarantee activity of the drug in a given indication due to presence of contraindicated mutation in gene.
- ❖ The mutation information provided should only be utilized as a guide or aid and the decision to select any therapy option based on the information reported here resides solely with the discretion of the treating physician.
- ❖ This report should only be used as an aid and the treating physician should employ sound clinical judgment in arriving at any decision for patient care or treatment.

### REFERENCES

1. Castoldi E, Lunghi B, et al. A missense mutation (Y1702C) in the coagulation factor V gene is a frequent cause of factor V deficiency in the Italian population. *Haematologia*. (2001). 86(6):629-633.
2. Ornstein DL and Cushman M. Factor V Leiden. *Circulation*. (2003). 107:e94-e97.
3. Ornstein DL, Cushman M, et al. The factor V HR2 haplotype and the risk of venous thrombosis: a meta-analysis. *Journal of Hematology*. (2003). 88(10):1182-1189.
4. Segers K, Dahlbäck B, Nicolaes GA. Coagulation factor V and thrombophilia: background and mechanisms. *Thromb Haemost*. (2007). 98(3):530-542.
5. Castoldi E, Simioni P, et al. Combinations of 4 mutations (FV R506Q, FV H1299R, FV Y1702C, PT 20210G/A) affecting the prothrombinase complex in a thrombophilic family. *Blood*. (2000). 96(4):1443-1448.



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Lab Head-Clinical Genomics

Patient Name:	Dummy	CRM ID:	XXX
Age/DOB:	32 Years	Sample Type:	EDTA Whole Blood
Sex:	Female	Collection Date:	09.03.2024
Referring Clinician:	NA	Receiving Date:	11.03.2024
Test Requested:	Methylenetetrahydrofolate Reductase (MTHFR)-2 Variants(C677T,A1298C)	Reporting Date:	14.03.2024

### MTHFR GENE MUTATION (C677T, A1298C) BY PCR, SANGER

#### CLINICAL DIAGNOSIS & SYMPTOMS

N/A

#### RESULTS

POSITIVE	
<i>MTHFR</i> : c.1298A>C (p.Glu429Ala)	Heterozygous mutation detected
<i>MTHFR</i> : c.677C>T (p.Ala222Val)	Heterozygous mutation detected

Result classification	Comment
Homozygous mutation detected	Both copies of the gene carry mutation
Heterozygous mutation detected	One copy of the gene carries mutation
Not Detected	Mutation not detected

#### CLINICAL SIGNIFICANCE

The mutation for MTHFR (Methyltetrahydrofolate reductase) is associated with hyperhomocysteinemia which is an independent risk factor for Stroke, Myocardial infarction, Peripheral arterial disease and venous thrombosis. Indian studies suggest that heterozygosity for MTHFR C 677T is also associated with elevated homocysteine levels. MTHFR C677T or A1298C carriers are not at increased risk for thrombosis in the absence of hyperhomocysteinemia.

Homozygous MTHFR C 677T or A1298C carriers are at increased risk for hyperhomocysteinemia if they become deficient in vitamins B6, B12 or folic acid. Hyperhomocysteinemia is a relatively weak risk factor for both venous thromboembolism and arterial thrombosis.

## METHODOLOGY

Targeted sequencing and mutation analysis was performed by Polymerase Chain Reaction (PCR) followed by automated DNA sequencing of the amplicon using BigDye Terminator Chemistry on an ABI Genetic Analyzer 3500XL platform. Sequencing data were aligned to NCBI database to analyze the mutations

## COMMENT

A genetic polymorphism commonly associated with severe MTHFR deficiency is defined by a C to T substitution (cytosine to thymine) at position 677 (C677T) of the MTHFR gene, which leads to the incorporation of amino acid alanine (A) instead of valine (V) at position 222 of the MTHFR protein. The altered MTHFR is known as “thermolabile MTHFR”. Homozygous and heterozygous carriers of this mutation both show reduced MTHFR activity. In particular, homozygous carriers suffer from significantly increased blood levels of homocysteine. C677T mutation in its homozygous form alone or as a compound heterozygote, which involves both C677T and an A1298C condition (where an Adenine (A) residue changes to a Cytosine (C) residue at the 1298th position) lead to the disruption of the MTHFR gene and causes a drastic reduction of the MTHFR enzyme. This in turn, leads to an elevation of Homocysteine in the blood. Homocysteine is an important substance in the blood as elevated levels of Homocysteine has been found to be the causative agent of various diseases such as; Cerebrovascular disease cerebral vein thrombosis, coronary artery disease, myocardial infarction, venous thrombosis neural tube defects leading to dementia and Alzheimer’s disease osteoporosis, diabetes, complications in pregnancy.

## LIMITATIONS

- A positive result is specific for a particular MTHFR C677T & A1298C variant, diagnosis and clinicopathologic correlation is necessary in all cases.

- The sensitivity of detection for Sanger sequencing is generally recognized as being approximately 15% to 20% mutant allele frequency.
- PCR is a highly sensitive technique; common reasons for paradoxical results are contamination during specimen collection, selection of inappropriate specimen and inherent PCR inhibitors in the sample.

## REFERENCES:

1. Arruda, VR, et al. The mutation Ala677. Val in the Methylene Tetrahydro Folate Reductase gene: a risk factor for arterial disease and venous thrombosis. *Thrombosis and Haemostasis* 77(5) (1997).
2. . Dahlback B et al. Resistance to activated protein C, the FV: Q506 allele, and venous thrombosis. *Ann Hematol.* 1996; 72:166-176.
3. Bagley PJ et al. *Proc Natl Acad Sci U S A* 1998; 95:13217- 13220.
4. Botto LD, Yang Q. 5,10-Methylenetetrahydrofolate reductase gene variants and congenital anomalies: a HuGE review. *Am J Epidemiol.* 2000 May 1; 151 (9): 862 – 877.

## CONDITIONS OF REPORTING

- Test results released pertain to the specimen submitted .
- All test results are dependent on the quality of the sample received by the Laboratory .
- Laboratory investigations are only a tool to facilitate in arriving at a diagnosis and should be clinically correlated by the Referring Physician .
- Certain tests may require further testing at additional cost for derivation of exact value. Kindly submit request within 72 hours postreporting.
- Test results may show inter laboratory variations.
- If Sample collection date is not stated on test requisition form, the current date will be printed by default as the date of collection.
- Test results are not valid for medico legal purposes.



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# Terms and Conditions of Reporting

1. The presented findings in the Reports are intended solely for informational and interpretational purposes by the referring physician or other qualified medical professionals possessing a comprehensive understanding of reporting units, reference ranges, and technological limitations. The laboratory shall not be held liable for any interpretation or misinterpretation of the results, nor for any consequential or incidental damages arising from such interpretation.
2. It is to be presumed that the tests performed pertain to the specimen/sample attributed to the Customer's name or identification. It is presumed that the verification particulars have been cleared out by the customer or his/her representation at the point of generation of said specimen / sample. It is hereby clarified that the reports furnished are restricted solely to the given specimen only.
3. It is to be noted that variations in results may occur between different laboratories and over time, even for the same parameter for the same Customer. The assays are performed and conducted in accordance with standard procedures, and the reported outcomes are contingent on the specific individual assay methods and equipment(s) used, as well as the quality of the received specimen.
4. This report shall not be deemed valid or admissible for any medico-legal purposes.
5. The Customers assume full responsibility for apprising the Company of any factors that may impact the test finding. These factors, among others, includes dietary intake, alcohol, or medication / drug(s) consumption, or fasting. This list of factors is only representative and not exhaustive.

**Disclaimer: Method given in report are only indicative and can be changed depending upon type of machine and kit available at time of testing.**

**Not all tests at all locations are under NABL scope. Availability of tests under NABL scope varies from lab to lab.**

Name  
Mrs Dummy

Patient ID  
XXX

Gender  
F

Age  
32

## Health Advisory

● Normal (N) ● Low (L) ● Borderline (BL) ● High (H)

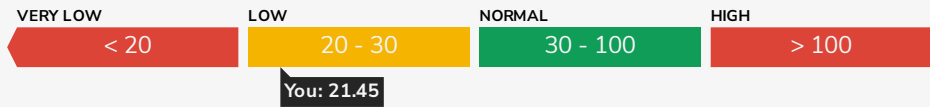


### Vitamins Profile

Vitamins are considered essential nutrients because they perform hundreds of roles in your body. They help maintain bones, heal wounds, and strengthen your immune system. They also convert food into energy, and repair cellular damage

Vitamin D (25-Hydroxy): 21.45 ng/mL

● BORDERLINE



#### Diet and Lifestyle Tips :



Avoid very high-SPF sunscreen: Your skin naturally produces vitamin D on being exposed to sun but applying sunscreen on your skin can decrease this natural production. It is recommended that you should get a balanced amount of sunshine.



Consider supplements. Ask your doctor if Vitamin D supplements are right for you.

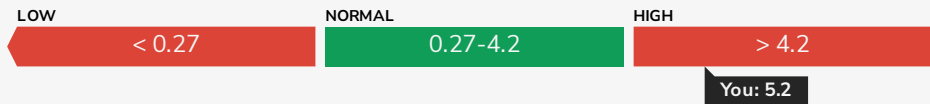


### About Thyroid

This panel is used to check the imbalance in your thyroid gland. A healthy thyroid gland is very important for metabolism, controlling body temperature, regulation of mood, muscle strength and regulation of body weight

TSH: 5.2 mIU/L

● HIGH



#### Common reasons for abnormal results :



Hormonal changes from use of oral contraceptive pills.



Injury to the thyroid following radiation therapy



Autoimmune disorders such as Hashimoto's thyroiditis



## About Redcliffe Labs

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