Name:	**Mr. Dummy**	Case ID:	**XXX**
Age:	**34 Years**	Sample Type:	**Blood**
Sex:	**Male**	Sample Receipt Date:	**13/05/2024**
Referring Clinician:	**Online booking**	Reporting Date:	**18/06/2024**
Test Requested:	**Whole Genome Sequencing (WGS)**		

CLINICAL INFORMATION/HISTORY

Mr. Dummy was referred for Pre-test counseling (over call/ WhatsApp) to discuss clinical implications, family history, advantages, and limitations of the whole genome genetic test.
He was referred for predictive genetic testing & counseling.
Previous Investigations:
Fertility Panel - Advance + Haemoglobinopathies report showed no evidence of Haemoglobinopathies.

RESULT SUMMARY

No Pathogenic, Likely Pathogenic variants relevant to predictive genetic testing were found.

VARIANTS RELEVANT TO INDICATION FOR TESTING

No variants were found in the sequence data which may be associated with the clinical history of the patient.

Gene & Transcript	Variant	Zygosity	Location	Disorder	Inheritance	ACMG Classification
No significant variant related to phenotype was detected.						

FINDINGS UNRELATED TO PHENOTYPE

This section provides information on variants identified which are unrelated to the provided phenotype.

ACMG Secondary Findings

No clinically relevant variants associated with the ACMG recommended secondary list of genes were found in the sequence data.

Incidental Findings

The following incidental finding was found which may not be associated with the diagnostic indication for which the sequencing test was performed.

Gene & Transcript	Variant	Zygosity	Location	Disorder	Inheritance	ACMG Classification
ABCA4 NM_000350.3	c.5882G>A p.Gly1961Glu	Heterozygous	Exon 42	{Macular degeneration, age-related, 2} [OMIM ID: 153800]	Autosomal Dominant	Likely pathogenic PM1, PS3, PP5 & PM5
				Cone-rod dystrophy 3 [OMIM ID: 604116]	Autosomal Recessive	
				Stargardt disease 1 / Retinal dystrophy, early-onset severe / Fundus flavimaculatus [OMIM ID: 248200]		
				Retinitis pigmentosa 19 [OMIM ID: 601718]		

DETAILED VARIANT INFORMATION (INCIDENTAL FINDINGS)

***ABCA4* Chr. 1:94008251 – Likely pathogenic:**

The missense variant NM_000350.3(ABCA4):c.5882G>A (p.Gly1961Glu) causes a change at the same amino acid residue as a previously established pathogenic variant. This variant was found in ClinVar (Variant 7888) with a classification of Pathogenic/Likely pathogenic/Pathogenic, low penetrance and a review status of (2 stars) criteria provided, multiple submitters, no conflicts. There is a moderate physicochemical difference between glycine and glutamic acid. 5 variants within 6 amino acid positions of the variant p.Gly1961Glu have been shown to be pathogenic, while none have been shown to be benign. This variant has been reported >20 individuals with Stargardt disease and other retinal phenotypes and segregated with disease in 5 affected individuals from 5 families, but has been associated with reduced penetrance [PMID:9295268, 22312191, 22661473, 22025579, 20696155, 16103129, 19217903, 19074458, 12037008, 12796258, 28327576, 29847635, 23769331, 22661473, 18285826, 26527198]. In vitro functional studies support an impact on protein function [PMID:11017087]. For these reasons, this variant has been classified as **Likely Pathogenic.**

{Macular degeneration, age-related, 2} [OMIM ID: 153800]:

Macular degeneration, age-related, 2 (ARMD2) is conferred by variation in the ABCR gene (ABCA4; 601691) on chromosome 1p22. Age-related macular degeneration-2 (ARMD2) is a complex disorder characterized by the accumulation of drusen in and under the retinal pigment epithelium (RPE) and the progressive atrophy of the macular RPE. These changes result in loss of photoreceptor function and vision impairment. Environmental risk factors include cigarette smoking, diet, and cholesterol level (summary by Allikmets et al., 1997).

Cone-rod dystrophy 3 [OMIM ID: 604116]:

Cone-rod dystrophy-3 (CORD3) is caused by homozygous or compound heterozygous mutation in the *ABCA4* (601691) on chromosome 1p22. Cone-rod dystrophy-3 (CORD3) is an autosomal recessive, clinically heterogeneous retinal disorder with typical findings of reduced visual acuity, impairment of the central visual field, color vision deficits, and fundoscopic evidence of maculopathy, with no or few midperipheral retinal pigment deposits. Cone degeneration appears early in life with a central involvement of the retina, followed by a degeneration of rods several years later (summary by Klevering et al., 2002 and Ducroq et al., 2002). Both cone and rod a- and b-wave electroretinogram (ERG) amplitudes are reduced (Fishman et al., 2003). Proband carries a heterozygous variant in the ABCA4 gene and hence, a carrier for the above-mentioned condition.

Stargardt disease 1/ Retinal dystrophy, early-onset severe/ Fundus flavimaculatus [OMIM ID: 248200]:
Stargardt disease-3 (STGD3; 600110) is caused by mutation in the ELOVL4 gene (605512) on chromosome 6q14, and Stargardt disease-4 is caused by mutation in the PROM1 gene (604365) on chromosome 4. Fundus flavimaculatus (FFM) is an allelic subtype of Stargardt disease that has been associated with mutation in the *ABCA4* gene and the PRPH2 gene (179605). FFM has a later age of onset. If loss of visual acuity begins in the first 2 decades, the designation Stargardt disease is preferred; if it begins later in life and has a more progressive course, the term FFM is preferred (Weleber, 1994). An early-onset severe form of retinal dystrophy (CORD3; 604116) is caused by homozygous null mutations in the *ABCA4* gene. Stargardt disease is one of the most frequent causes of macular degeneration in childhood. It has onset between 7 and 12 years, a rapidly progressive course, and a poor final visual outcome. Although visual acuity is severely reduced, peripheral visual fields remain normal throughout life. Degeneration limited to the macular area of the retina was described in multiple sibs by Ford (1961) and by Walsh (1957). Fundus flavimaculatus, which is a form of fleck fundus disease (see 228980), derives its name from the occurrence of many yellow spots rather uniformly distributed over the fundus. In some older patients the flecks fade with time as atrophy of the retinal pigment epithelium (RPE) increases. Round, linear, or pisciform lesions are distributed in the posterior pole, sometimes with extension to the equator, and with macular involvement. Network atrophy of the retinal pigment epithelium, and choroidal vascular atrophy are features. Central visual loss, loss of color vision, photophobia, paracentral scotoma, and slow dark adaptation are features. This is probably an autosomal recessive disorder. Klien and Krill (1967) observed a 'familial incidence...in 10 of 27 patients.' The 10 familial cases included 4 pairs of affected sibs with ostensibly normal parents who were, however, not examined in most instances. No parental consanguinity was described. In 1 instance the father and 2 daughters were affected. In the instance of an affected brother and sister, the father was black and the mother white. Krill and Deutman (1972) concluded that recessive macular dystrophy was the disorder described and beautifully illustrated by Stargardt (1909), and also was the disorder that Franceschetti (1963) renamed fundus flavimaculatus. Krill and Deutman (1972) suggested the possibility of a rarer, phenotypically indistinguishable, autosomal dominant form. Hadden and Gass (1976) presented evidence that fundus flavimaculatus is the same as the Stargardt form of macular dystrophy. Proband carries a heterozygous variant in the ABCA4 gene and hence, a carrier for the above-mentioned condition.

Retinitis pigmentosa 19 [OMIM ID: 601718]:
Retinitis pigmentosa-19 (RP19) can be caused by homozygous or compound heterozygous mutation in the ABCR gene (*ABCA4*; 601691) on chromosome 1p22. A retinal dystrophy belonging to the group of pigmentary retinopathies. Retinitis pigmentosa is characterized by retinal pigment deposits visible on fundus examination and primary loss of rod photoreceptor cells followed by secondary loss of cone photoreceptors. Patients typically have night vision blindness and loss of midperipheral visual field. As their condition progresses, they lose their far peripheral visual field and eventually central vision as well. RP19 is characterized by choroidal atrophy [Uniprot]. Proband carries a heterozygous variant in the ABCA4 gene and hence, a carrier for the above-mentioned condition.

Carrier Status in the genes related to disease

The following Likely Pathogenic variants were detected.

Gene & Transcript	Variant	Zygosity	Location	Disorder	Inheritance	ACMG Classification
PROP1 NM_006261.5	c.301_302delAG p.Leu102Cysfs*8	Heterozygous	Exon 2	Pituitary hormone deficiency, combined, 2 [OMIM ID: 262600]	Autosomal Recessive	Likely pathogenic PVS1, PM2 & PP5
HYDIN NM_001270974.2	c.2092delC p.Leu698Serfs*15	Heterozygous	Exon 16	Ciliary dyskinesia, primary, 5 [OMIM ID: 608647]	Autosomal Recessive	Likely pathogenic PM2 & PVS1

DETAILED VARIANT INFORMATION (CARRIER STATUS)

***PROP1* Chr. 5:177994146 – Likely pathogenic:**

The frameshift deletion NM_006261.5(PROP1):c.301_302delAG (p.Leu102Cysfs*8) has been reported to ClinVar as Pathogenic/Likely pathogenic with a status of (2 stars) criteria provided, multiple submitters, no conflicts (Variation ID 8098 as of 2024-06-06). The p.Leu102Cysfs*8 variant is not reported in any individuals in 1kG All. The p.Leu102Cysfs*8 variant is observed in 42/246,046 (0.0171%) alleles from individuals of gnomAD All background. This variant is predicted to cause loss of normal protein function through protein truncation causing a frameshift mutation. The frame shifted sequence continues 8 residues until a stop codon is reached. This variant has been previously classified as pathogenic, indicating that the region is critical to protein function. There are 18 downstream pathogenic loss of function variants, with the furthest variant being 109 residues downstream of this variant. This indicates that the region is critical to protein function. The p.Leu102Cysfs*8 variant is a loss of function variant in the gene PROP1, which is intolerant of Loss of Function variants, as indicated by the presence of existing pathogenic loss of function variant NP_006252.4:p.M1V and 29 others. For these reasons, this variant has been classified as **Likely Pathogenic.**

Pituitary hormone deficiency, combined, 2 [OMIM ID: 262600]:

Pituitary hormone deficiency, combined, 2 (CPHD2) is caused by homozygous or compound heterozygous mutation in the PROP1 gene (601538) on chromosome 5q35. Congenital hypopituitarism is characterized by multiple pituitary hormone deficiency, including somatotroph, thyrotroph, lactotroph, corticotroph or gonadotroph deficiencies. Congenital hypopituitarism is rare compared with the high incidence of hypopituitarism induced by pituitary adenomas, transsphenoidal surgery or radiotherapy [Orphanet]. Voutetakis et al. (2004) used long-term MRI findings to characterize the morphologic abnormalities of the pituitary gland in 15 patients with CPHD caused by PROP1 gene mutations. Small pituitary gland was detected in 7 patients (25.2 +/- 14.4 years of age), normal pituitary size in 3 patients (10.2 +/- 5.8 years of age), and pituitary enlargement in 5 patients (6.5 +/- 2.7 years of age). The pituitary enlargement consisted of a nonenhancing mass lesion interposed between the normally enhancing anterior lobe and the neurohypophysis. The pituitary stalk was displaced anteriorly, whereas the neurohypophysis was orthotopic, displaying a normal signal. Spontaneous regression of the mass lesion with normalization of the pituitary stalk position was observed in 3 patients. The authors concluded that while a small pituitary gland is usually observed in older subjects, a significant number of young patients with PROP1 gene mutations demonstrate pituitary enlargement with subsequent regression.

DETAILED VARIANT INFORMATION (CARRIER STATUS)

***HYDIN* Chr. 16:71064824 – Likely pathogenic:**

The frameshift deletion NM_001270974.2(HYDIN):c.2092delC (p.Leu698Serfs*15) has not been reported previously on a disease database like ClinVar or in the disease database literature, to our knowledge. The p.Leu698Serfs*15 variant is not reported in any individuals in 1kG All. The p.Leu698Serfs*15 variant is not reported in any individuals in gnomAD. This variant is predicted to cause loss of normal protein function through protein truncation causing a frameshift mutation. The frame shifted sequence continues 15 residues until a stop codon is reached. This variant is a frameshift variant which occurs in an exon of HYDIN upstream of where nonsense mediated decay is predicted to occur. There are 28 downstream pathogenic loss of function variants, with the furthest variant being 4216 residues downstream of this variant. This indicates that the region is critical to protein function. The p.Leu698Serfs*15 variant is a loss of function variant in the gene HYDIN, which is intolerant of Loss of Function variants, as indicated by the presence of existing pathogenic loss of function variant NP_001257903.1:p.R383* and 10 others. For these reasons, this variant has been classified as **Likely Pathogenic.**

Ciliary dyskinesia, primary, 5 [OMIM ID: 608647]:

Ciliary dyskinesia, primary, 5 (CILD5) is caused by homozygous mutation in the HYDIN gene (610812) on chromosome 16q22. Primary ciliary dyskinesia-5 (CILD5) is an autosomal recessive disorder characterized by early onset of a progressive decline in lung function due to an inability to clear mucus and particles from the airways. Affected individuals have recurrent infections of the sinuses, ears, airways, and lungs. Sperm motility is also decreased. Individuals with CILD5 do not have situs inversus (summary by Olbrich et al., 2012).

RECOMMENDATIONS

Based on the clinical features and the observed genetic findings the following have been recommended:

1. Genetic counseling is recommended to discuss the potential clinical implications of this result.
2. **Clinical/ Genotype-phenotype correlation is strongly recommended.**
3. **Sanger validation of the identified variant(s) in the proband and segregation analysis in the parents, affected and unaffected family members and close relatives is advised.**
4. **Re-analysis of whole genome sequencing data can be done if additional phenotype is provided, and results may change/differ on re-analysis depending on the provided phenotype.**
5. **Blood karyotype can be offered to rule out gross chromosomal abnormalities.**
6. **If the clinician suspects for copy number variations as a cause of the patient's phenotype then additional testing with chromosomal microarray is recommended with better sensitivity and specificity for the detection of copy number variants.**
7. If the above results do not correlate completely with patient phenotype, additional testing is advised based on the clinician's recommendation.

REPORTED VARIANTS STATISTICS:

Gene/Transcript	Variant	Depth	Allelic Depth	Alternate Allele Fraction	dbSNP rsID
PROP1 NM_006261.5	c.301_302delAG	45X	21X	0.47	rs193922688
HYDIN NM_001270974.2	c.2092delC	35X	20X	0.57	NA
ABCA4 NM_000350.3	c.5882G>A	61X	28X	0.46	rs1800553

DATA STATISTICS

Total data generated (Gb)	**239**
Reads that passed alignment (%)	**96.5**
Data > Q30 (%)	**92.1**

METHODOLOGY

The whole genome sequencing for the sample was performed using Illumina NovaSeq platform at a mean depth of ~30-35X, with mean Q30 >=90%. The individual's DNA was extracted and fragmented. The fragments from the whole genome were amplified, size selected and sequenced with 150*2 read length. Reads from the sequence output were quality trimmed & aligned to the human reference genome (GRCh38) using the DRAGMAP Aligner. Duplicate reads identification and removal, base quality recalibration and re-alignment of reads based on indels were done using inbuilt **DRAGEN bio-IT pipeline**. Variants to the reference were called using the Haplotype caller in DRAGEN mode. The variants were annotated and filtered using the **Golden Helix VarSeq** and **Varsome** analysis workflow implementing the ACMG guidelines for interpretation of sequence variants. This includes comparison against the gnomAD population catalogue, the 1000 Genomes Project Consortium's publication of 2,500 genomes, the NCBI ClinVar database of clinical

assertions on variant's pathogenicity and multiple lines of computational evidence on conservation and functional impact. All variants with minor allele frequency (MAF) of less than 1% in gnomAD database, and disease-causing variants reported in HGMD & in ClinVar are considered. The investigation for relevant variants is focused on coding exons and the intronic nucleotides in the vicinity of the genes with clear gene-phenotype evidence (based on OMIM information). All potential modes of inheritance patterns are considered. In addition, provided family history and clinical informations are used to evaluate identified variants with respect to their pathogenicity and causality. This test has not been cleared or approved by the U.S. Food and Drug Administration (FDA). The FDA has determined that such clearance or approval is not necessary.

VARIANT ASSESSMENT PROCESS

The following databases and in-silico algorithms are used to annotate and evaluate the impact of the variant in the context of human disease: 1000 genomes, gnomAD, ClinVar, OMIM, dbSNP, NCBI RefSeq Genes, ExAC Gene Constraints, VS-SIFT, VS-PolyPhen2, PhyloP, GERP++, GeneSplicer, MaxEntScan, NNSplice, PWM Splice Predictor. Analysis was reported using the HGVS nomenclature (www.hgvs.org/mutnomen) as implemented by the VarSeq transcript annotation algorithm. The reported transcript matches those used most frequently by the clinical labs submitting to ClinVar.

LIMITATIONS

This report only includes variants that meet a level of evidence threshold for cause or contribution to disease. Certain classes of genomic variants are also not covered using the NGS testing technology, including triplet repeat expansions, copy number alterations, translocations and gene fusions or other complex structural rearrangements. More evidence for disease association of genes and causal pathogenic variants is discovered every year, and it is recommended that genetic variants are re-interpreted with updated software and annotations periodically.

VARIANT CLASSIFICATION BASED ON ACMG RECOMMENDATIONS

Genetic test results are reported based on the recommendations of American College of Medical Genetics (ACMG) as described below [1]

Variant	A change in a gene. This could be disease causing (pathogenic) or not disease causing (benign).
Pathogenic	A disease-causing variation in a gene which can explain the patients' symptoms.
Likely pathogenic	A variant which is very likely to contribute to the development of disease. However, the scientific evidence is currently insufficient to prove this conclusively. Additional evidence is expected to confirm this assertion of pathogenicity
Variant of uncertain significance	A variant which is difficult to classify either as pathogenic (disease causing) or benign (non-disease causing) based on current available scientific evidence.

ACMG Criteria for classifying Variants.

Very Strong (PVS1)	
PVS1	Null variant (nonsense, frameshift, canonical ±1 or 2 splice sites, initiation codon, single or multi-exon deletion) in a gene where LOF is a known mechanism of disease.
Strong (PS)	
PS1	Same amino acid change as a previously established pathogenic variant regardless of nucleotide change
PS2	De novo variant (both maternity and paternity confirmed) in a patient with the disease and no family history.

PS3	Well-established in vitro or in vivo functional studies supportive of a damaging effect on the gene or gene product.
PS4	The prevalence of the variant in affected individuals is significantly increased compared with the prevalence in controls.
Moderate (PM)	
PM1	Located in a mutational hot spot and/or critical and well-established functional domain (e.g., active site of an enzyme) without benign variation
PM2	Absent from controls (or at extremely low frequency if recessive) in reputed databases.
PM3	Variant (one of the compound heterozygous), is segregating with a pathogenic variant with known phase after testing of parents.
PM4	An in-frame deletions/insertions in non-repeat region or stop-loss can alter the protein length.
PM5	A novel missense change at the same amino acid residue where a pathogenic missense variant has already been determined.
PM6	De novo, without testing in the family.
Supporting (PP)	
PP1	A variant in known gene for a disease which is co-segregating in multiple affected family members
PP2	Missense variants are a common mechanism of disease in a gene which has low benign missense variants.
PP3	A deleterious effect of the variant is predicted by multiple lines of computational evidence (conservation, evolutionary, splicing impact, etc.)
PP4	Patient's phenotype or family history is highly specific for a disease with a single genetic etiology.
PP5	Reputable source recently reported the variant as pathogenic, but the evidence is not available to the laboratory to perform an independent evaluation.

DISCLAIMER

- In accordance with the Pre-Conception and Pre-Natal Diagnostic Testing (PCPNDT) Act, 2003- Govt. of India; Lab does not disclose the gender of the fetus.
- Prenatal genetic testing or pre-implantation genetic diagnosis is not recommended for the variants reported as variants of uncertain significance (VUS).
- Interpretation of variants in this report is performed to the best knowledge of the laboratory based on the information available at the time of reporting. The classification of variants can change over time and the laboratory cannot be held responsible for this. Re-analysis of variants in previously issued reports considering new evidence is not routinely performed but may be available upon request.
- Negative results do not completely exclude the risk/carrier status for these disorders tested (residual risk)
- The sensitivity of this assay to detect large deletions/duplications of more than 10bp or copy number variations (CNV) is 70-75%. The CNVs detected must be confirmed by an alternate method.
- Due to inherent technological limitations of the assay, not all bases of the genome can be covered by this test. Accordingly, variants in regions of insufficient coverage may not be identified and/or interpreted. Therefore, it is possible that pathogenic

variants are present in one or more of the genes analyzed but have not been detected. The variants not detected by the assay that was performed may impact the phenotype.

- It is also possible that a pathogenic variant is present in a gene that was not selected for analysis and/or interpretation in cases where insufficient phenotypic information is available.
- Genes with pseudogenes, paralog genes and genes with low complexity may have decreased sensitivity and specificity of variant detection and interpretation due to inability of the data and analysis tools to unambiguously determine the origin of the sequence data in such regions.
- The mutations have not been validated/confirmed by Sanger sequencing.
- Incidental or secondary findings (if any) that meet the ACMG guidelines [2] can be given upon request.
- The report shall be generated within turnaround time (TAT), however, such TAT may vary depending upon the complexity of test(s) requested. Laboratory under no circumstances will be liable for any delay beyond aforementioned TAT.
- It is hereby clarified that the report(s) generated from the test(s) do not provide any diagnosis or opinion or recommend any cure in any manner. Laboratory hereby recommends the patient and/or the guardians of the patients, as the case may be, to take assistance of the clinician or a certified physician or doctor, to interpret the report(s) thus generated. Laboratory hereby disclaims all liability arising in connection with the report(s).
- In a very few cases genetic tests may not show the correct results, e.g., because of the quality of the material provided to the laboratory. In cases where any test provided by the laboratory fails for unforeseeable or unknown reasons that cannot be influenced by the laboratory in advance, the laboratory shall not be responsible for the incomplete, potentially misleading, or even wrong result of any testing if such could not be recognized by the laboratory in advance.
- This is a laboratory developed test and the development and the performance characteristics of this test was determined by the laboratory.

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4. Sherry ST, Ward MH, Kholodov M, Baker J, Phan L, Smigielski EM, Sirotkin K. dbSNP: the NCBI database of genetic variation. Nucleic Acids Res. 2001 Jan 1;29(1):308-11.
5. GnomAD database - https://gnomad.broadinstitute.org/.

PMID	CITATION
9295268	Mutation of the Stargardt disease gene (ABCR) in age-related macular degeneration. *Allikmets R et al., Science. 1997 Sep 19*

PMID	CITATION
22312191	Familial discordance in Stargardt disease. *Burke TR et al., Mol Vis. 2012*
22661473	Retinal phenotypes in patients homozygous for the G1961E mutation in the ABCA4 gene. *Burke TR et al., Invest Ophthalmol Vis Sci. 2012 Jul 03*
22025579	High-throughput retina-array for screening 93 genes involved in inherited retinal dystrophy. *Song J et al., Invest Ophthalmol Vis Sci. 2011 Nov 25*
20696155	Loss of peripapillary sparing in non-group I Stargardt disease. *Burke TR et al., Exp Eye Res. 2010 Nov*
16103129	ABCA4 mutations causing mislocalization are found frequently in patients with severe retinal dystrophies. *Wiszniewski W et al., Hum Mol Genet. 2005 Oct 01*
19217903	G1961E mutant allele in the Stargardt disease gene ABCA4 causes bull's eye maculopathy. *Cella W et al., Exp Eye Res. 2009 Jun 15*
19074458	ABCA4 disease progression and a proposed strategy for gene therapy. *Cideciyan AV et al., Hum Mol Genet. 2009 Mar 01*
11017087	Biochemical defects in ABCR protein variants associated with human retinopathies. *Sun H et al., Nat Genet. 2000 Oct*

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Conditions for Reporting

1. It is presumed that the specimen belongs to the patient named or identified, such verification being carried out at the point of generation of said specimen.
2. A test might not be performed due to following reasons:
   a. Specimen quantity not sufficient (Inadequate collection/spillage during transit).
   b. Specimen quality not acceptable (Hemolysis/clotted/lipemic).
   c. Incorrect sample type.
   d. Test canceled either on request of patient or doctor.
3. In any of the above cases a fresh specimen will be required for testing and reporting.
4. The results of the tests may vary from lab to lab, time to time for the same patient.
5. The reported results are dependent on individual assay methods, equipment, method sensitivity, specificity and quality of the specimen received.
6. Partial representation of the report is not allowed.
7. The reported tests are for the notification of the referring doctor, only to assist him/her in the diagnosis and management of the patient.
8. Report with status "Preliminary" means one or more tests are yet to be reported.
9. This report is not valid for Medico Legal Purpose.
10. Applicable Jurisdiction will be of "Delhi" for any dispute/claim concerning the test(s) & results of the test(s).

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.

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.