Case ID	: **18-P-1150**		**Sample Type**	: **FFPE TISSUE BLOCK**
Name	: R. PEDDI RAJU		**Date & Time Collected**	:
Sex/Age	: **MALE / 63 Years**		**Date & Time Received**	: **31-Jan-2018 14:51**
Bill. Loc.	: **Surya Speciality Lab, Hyderabad**		**Date & Time Reported**	: **03-Feb-2018**
Ref. By	: **Dr. Vindhya Vasini MD DM (Omega)**			
Indication	:			

EGFR MUTATION ANALYSIS REPORT

RESULT

Deletion in Exon 19 detected in EGFR gene.

Block used: H-272/18

Mutation description	Exon	Mutation detected	Recommendation for treatment with GEFITINIB
G719S	18	No	Currently limited data supporting sensitivity to gefitinib
G719A	18	No	
G719C	18	No	
Double mutations	18	No	
S768I	18	No	
Deletion in exon 19	19	Yes	Data supporting sensitivity to gefitinib
Insertion in exon 20	20	No	Currently no data supporting sensitivity to gefitinib
T790M	20	No	
L858R	21	No	Data supporting sensitivity to gefitinib
L861Q	21	No	

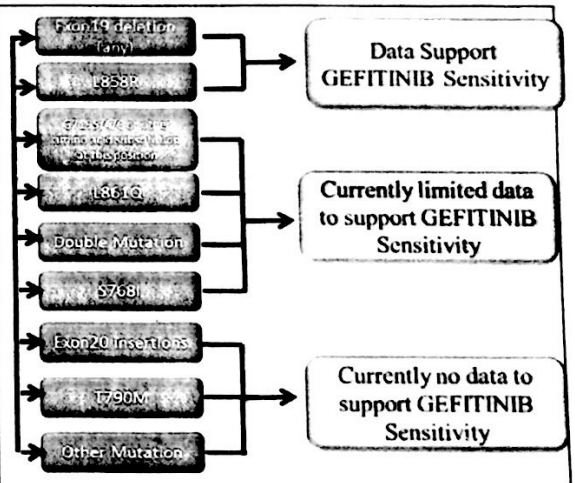
METHOD SUMMARY:

DNA was isolated from Paraffin tissue block, and PCR was performed to amplify exon 18, 19, 20 and 21 of EGFR gene. EGFR mutations are then identified by analysis of the PCR products using automated DNA sequencing technique.

CLINICAL BACKGROUND:

EGFR mutations are found in four exons of the EGFR gene, exons 18 to 21. Exon 19 deletions and exon 21 L858R mutations account for ~90% of all mutations. Screening technologies such as sequencing normally assess all four exons. On the other hand, targeted technologies (non-sequencing methods) tend to assess only specific common mutations.

Tumour cells harbouring EGFR mu
tations become "oncogene (EGFR) addicted" and undergo an apoptosis in response to gefitinib exposure which translates in the clinic into significant tumour shrinkage in the majority of the patients with such tumours. The science and clinical data in the area of EGFR mutations are still emerging. Based on previous experience from randomised phase III studies (IPASS, INTEREST, V-15-32, ISEL) and external literature from before March 2009, exons 18-21 of the EGFR gene should be analysed to support the treatment decision for a patient with advanced NSCLC.



Dr. Pratap N. Mukhopadhyaya
Ph.D (Sc.)

Dr. Arpan Acharya
M. Tech, PhD(Biotech)

Dr.Salil N.Vaniawala
M.Sc Ph.D.(Human Genetics)
Consulting Geneticist

2nd Floor, President Plaza - A, Near R.T.O. Circle, Nanpura, Ring Road, Surat - 395 001 (GUJARAT - INDIA)
Ph. : +91 261 - 2471515, 2471616
M : +91 95861 77755
REFERENCE LAB IN MOLECULAR GENETIC DISEASES
E-mail : contact@sngenelab.com
www.sngenelab.com

	:	18-P-1150			
me	:	R. PEDDI RAJU			
Sex/Age	:	MALE / 63 Years	Sample Type	:	FFPE TISSUE BLOCK
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Indication	:		Date & Time Reported	:	03-Feb-2018

Table showing various reported EGFR Gene mutations

1	Exon 19 deletions; L858R	~90%	Yes	Exon 19 deletions and the L858R mutation constitute ~90% of the EGFR mutations identified to date. In patients with tumors that are positive to these mutations, the current data supports sensitivity to gefitinib.
2	T790M/exon 19 deletions; T790M/L858R; G719X; L861Q; S768I	~7%	Limited*	NSCLC patients can have tumors that are positive for more than one EGFR mutation type. These are known as double mutations and are predominantly seen with T790M & an exon 19 deletion or T790M & L858R. In patients with tumors positive for these mutations where T790M is present, or tumors with other rare mutations listed here, there is very limited data to support sensitivity or resistance to gefitinib.
3	T790M alone; exon 20 insertions; other Mutations	~3%	None*	The exon 20 point mutation T790M (T790M alone), and the exon 20 insertions make up ~3% of the EGFR TK mutations identified to date. In patients with tumors positive for these mutations, there are currently no data to support sensitivity to gefitinib. Some screening methodologies such as sequencing may identify novel EGFR mutations where there will be no clinical or preclinical data to guide use of gefitinib.

** Due to lack of data it is difficult to draw definitive conclusions on the sensitivity or lack of sensitivity to gefitinib in these mutation types, because they only constitute ~10% of all EGFR TK mutations. Few patients in global AstraZeneca studies have been identified with these types of EGFR TK mutations*

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Ph.D (Sc.)

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2nd Floor, President Plaza -A, Near R.T.O. Circle, Nanpura, Ring Road, Surat - 395 001 (GUJARAT - INDIA)

+91 261-2471515, 2471616
+91 95861 77755

✧ REFERENCE LAB IN MOLECULAR GENETIC DISEASES ✧

E-mail : contact@sngenelab.com
www.sngenelab.com