Taipei Veterans General Hospital

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Sample Information

Patient Name: 謝路明
Gender: Male
ID No.: M120164248
History No.: 48643750
Age: 64

Ordering Doctor: DOC3064F 陳育民
Ordering REQ.: D71E81C
Signing in Date: 2022/08/11

Path No.: S111-97836
MP No.: F22082
Assay: Oncomine Focus Assay
Sample Type: FFPE
Block No.: S111-27949A
Percentage of tumor cells: 70%

Reporting Doctor: DOC5466K 葉奕成 (Phone: 8#5466)

Note:

Sample Cancer Type: Non-Small Cell Lung Cancer

Report Highlights

0 Relevant Biomarkers
0 Therapies Available
0 Clinical Trials

Relevant Non-Small Cell Lung Cancer Variants

Gene	Finding	Gene	Finding
ALK	None detected	NTRK1	None detected
BRAF	None detected	NTRK2	None detected
EGFR	None detected	NTRK3	None detected
ERBB2	None detected	RET	None detected
KRAS	None detected	ROS1	None detected
MET	None detected		

Relevant Biomarkers

No relevant biomarkers found in this sample.

Prevalent cancer biomarkers without relevant evidence based on included data sources
MYC amplification

Variants (Exclude variant in Taiwan BioBank with >1% allele frequency)

Copy Number Variations

Gene	Locus	Copy Number
MYC	chr8:128748885	14.94

Biomarker Descriptions

MYC (MYC proto-oncogene, bHLH transcription factor)

Background: The MYC gene encodes the MYC proto-oncogene (c-MYC), a basic helix-loop-helix transcription factor that regulates the expression of numerous genes that control cell cycle progression, apoptosis, metabolic pathways, and cellular transformation[1,2,3,4]. MYC is part of the MYC oncogene family that includes related transcription factors MYCN and MYCL that regulate transcription in 10-15% of promoter regions[5]. MYC functions as a heterodimer in complex with the transcription factor MAX[2,6].

Alterations and prevalence: Recurrent somatic alterations are observed in both solid and hematological cancers. Recurrent somatic mutations in MYC, including codon T58, are infrequent and hypothesized to increase the stability of the MYC protein[7,8]. MYC gene amplification is particularly common in diverse solid tumors. MYC amplification is observed in 30% of serous ovarian cancer, 20% of uterine serous carcinoma, 15% of esophageal and breast cancers, and is common (1-10%) in numerous other cancer types[9,10,11]. MYC is the target of the t(8;14)(q24;32) chromosomal translocation in Burkitt's lymphoma that places MYC coding sequences adjacent to immunoglobulin region regulatory sequences, which results in increased MYC expression[12,13].

Potential relevance: Currently, no therapies are approved for MYC aberrations. Due to the high frequency of somatic MYC alterations in cancer, many approaches are being investigated in clinical trials including strategies to disrupt complex formation with MAX, including inhibition of MYC expression and synthetic lethality associated with MYC overexpression[1,14,15,16].

Clinical Trials in Taiwan region:

Signatures

Testing Personnel: Shin-Ying Lin 2022.08.11 16:56:48 +08'00'

Laboratory Supervisor: 2022.08.11 16:57:01 +08'00'

Pathologist: Yi-Chu Yeh 2022.08.11 16:57:15 +08'00'

References

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