FAKULTI : FAKULTI ALAM BINA DAN UKUR

TAJUK : PERMOHONAN PELANJUTAN PEMBETULAN TESIS

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<th>BUTIRAN PELAJAR</th>
<th>KETERANGAN PELAJAR</th>
<th>ULASAN DAN TINDAKAN FAKULTI</th>
<th>KELULUSAN</th>
</tr>
<tr>
<td>
NAMA : MONGKOL KHAN

NO K/P @ ISID : 201203M10057

NO MATRIK : PB113079

PROGRAM : DOKTOR FALSAFAH (SENIBINA)

JENIS PENGAJIAN : PENYELIDIKAN

BENTUK PENDAFTARAN : SEPENUH MASA

PENYELIA : PROF. LAr DR. HASANUDDIN BIN LAMIT

BIL SEM : 18/16

STATUS : PEPERIKSAAN

PEMERIKSA LUAR : PROF. DR. ALDRIN BIN ABDULLAH (USM)

PEMERIKSA DALAM : PROF. DR. SY AHMAD ISKANDAR BIN SY ARIFFIN

PENGERUSI : PROF. LAr DR. ISMAIL BIN SAID

TAJUK TESIS : SOUTHEAST ASIAN TRADITIONAL STREETS AS PLACES OF CULTURAL SIGNIFICANCE

MUKASURAT : 1/1
</td>
<td>
1.1 Pelajar telah menjalani peperiksaan dan kronologi seperti di bawah :
<table>
<tr><th>Bil</th><th>Tarikh</th><th>Perkara</th></tr>
<tr><td>1</td><td>6/2/2020</td><td>Pelajar telah menghantar tesis ke Fakulti untuk tujuan peperiksaan lisan.</td></tr>
<tr><td>2</td><td>30/6/2020</td><td>Peperiksaan lisan dijalankan dan pelajar mendapat keputusan b2/6 bulan serta perlu disemak oleh panel pemeriksa dalam dan luar. Tarikh akhir pelajar perlu menghantar pembetulan tesis ke Fakulti adalah pada 28 Disember 2020.</td></tr>
<tr><td>3</td><td>9/12/2020</td><td>Fakulti menerima permohonan pelajar untuk pelanjutan penghantaran pembetulan tesis melalui surat rasmi yang telah disokong oleh penyelia.</td></tr>
</table>
1.2 Pelajar telah memohon pelanjutan penghantaran tesis bagi tujuan pemeriksaan semula tesis. Permohonan penangguhan di atas sebab yang berikut :

i. Pelajar perlu menguruskan rawatan ayahnya yang menghidap blood cancer yang menyebabkan beliau tidak boleh fokus terhadap penyelidikan dan penulisan tesis.
ii. Pelajar juga sedang menjalani kes perbicaraan di mahkamah bagi sebuah kes yang berlaku dalam tahun 2015 semasa pelajar bertugas sebagai arkitek di salah sebuah universiti di Thailand.
</td>
<td>
1.1 Pelajar telah memohon untuk pelanjutan tempoh penghantaran pembetulan tesis selama 3 bulan dan mendapat sokongan dari penyelia.

1.2 Penyelia memaklumkan bahawa pelajar telah berjaya membuat pembetulan sebanyak 30% daripada keseluruhan bab.

1.3 Mesyuarat JKA (PS) Fakulti pada 14 Disember 2020 telah menyokong permohonan pelajar ini dilanjutkan sehingga 11 Mac 2021.

1.4 Dokumen seperti di lampiran.
</td>
<td>
Disokong / ~~Tidak disokong~~

Dekan/TD (Akademik)
& Cop rasmi
PROF. Ir. DR. HISHAMUDDIN BIN MOHD ALI
Dean
Faculty of Built Environment and Surveying
Universiti Teknologi Malaysia
81310 UTM Johor Bahru, Johor, Malaysia
Tarikh : _____

Setuju / Tidak Setuju
(Ulasan)

_____

_____

_____

Pengerusi Mesyuarat
Jawatankuasa Akademik
Pengajian Siswazah Universiti
(JAPSU) &
Cop Rasmi
Tarikh : _____
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</tr>
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CABUTAN MINIT MESYUARAT JAWATANKUASA AKADEMIK (PENGAJIAN SISWAZAH)
BIL. 10/2020
FAKULTI ALAM BINA DAN UKUR
TARIKH : 14 DISEMBER 2020
MASA : 2.30 PETANG
TEMPAT : SECARA DALAM TALIAN *(ONLINE WEBEX MEETING)*

8.0 PERMOHONAN PELANJUTAN PEMBETULAN TESIS

Mesyuarat bersetuju memperakukan permohonan pelanjutan tesis bagi pelajar seperti berikut :

Maklumat Pelajar	Perakuan Fakulti
Nama Pelajar : Mongkol Khan No. Matrik : PB113079 Program : Doktor Falsafah (Senibina) Bil. Sem : 18/16 Penyelia Utama : Prof. Lar Dr. Hasanuddin bin Lamit	Permohonan pelanjutan pembetulan tesis dilanjutkan selama tiga (3) bulan

***Tindakan**: PP Akademik PG*

Confidential

Faculty of Architecture,
Urban design and Creative Arts
Mahasarakham University
Maha Sarakham 44150

7 December 2020

To: Dean of faculty and whom it may concern

Requested for postponing the Thesis correction

My name is Mongkol Khan, Matrix no. PB113079. PhD candidate, Faculty of Built environment and surveying.

As guided from Postgraduate office regarding the student status after VIVA session, presented on 30 June 2020 via online system. The candidate received the result as B2 for correction and amending within 6 months and expected to submit the amended thesis by **30 December 2020**.

However, as consulted several times with supervisor, Prof. Hasanuddin Lamit regarding the personal difficulties faced by 2 situations that seriously affected to my work, my health and mentality, therefore I could not focus and spent enough time for the thesis correction and the coming deadline due to:

1. Father's health condition as stated by the doctor as he is affected his blood by cancer (Lymphoma) at the age of 80. therefore, he needs concern and brings to the hospital every 2 weeks. I have to spend time with my brother to take care of and facilitate him the best we can.

2. Serious problem regarding to the Criminal court in Thailand, as I have been trapped with the committee of the university since 2005. Until late 2019 to early of 2020, it's become the case at the confliction between the ex-president and present committee. At that time, I am on duty as an architect for the university and become one of the members and felt into the situation that I have to prove my incident and offenses under the section to the court.

Above all, I have lost my concentration and focus on thesis correction and amending. With highly respect to UTM restriction and guidelines for thesis procedure for graduation. I still have strong will to graduate as PhD student from UTM which I took some years from the beginning until the present.

As a result of this respectively requested to postpone the deadline for thesis submission after for at least minimum of one month and maximum of three months.

Sincerely yours,

(Mongkol Khan)
mongkol.khan@gmail.com
+66 818338685

LABORATORIES
201 Route 17 North, 2nd Floor
Rutherford, New Jersey 07070
Phone: 201 528 9187
Fax: 201 933 0787

Patient Name: **E7505003**
Sex: ☒Male ☐Female
Date of Birth: 07/01/1940
Specimen: Peripheral Blood
Collected: 06/24/2020
Received: 06/27/2020
Reported: 07/10/2020
Clinical Hx: Screening

Accession Number: **BM29-800008**
Ordering Physician: D822BC00001
Client: AstraZeneca
Client ID No: 7505
Client Address: NA

FOCUS::CLL/SLL NGS ASSAY REPORT

INTERPRETATION

Variant of unknown significance detected in TP53 gene. See variant details below.

SUMMARY

Pathogenic Variants

None detected.

Likely Pathogenic Variants

None detected.

Variants of Unknown Significance(VOUS)

Gene	Transcript	cDNA	Protein	AVF
TP53	NM_000546.5	c.470_475delTCCGCG	p.V157_R158del	88%

TP53 - p.V157_R158del (c.470_475delTCCGCG)

CLL NGS Gene list

ATM (ex2-69), BIRC3 (ex6-8), CARD11 (ex2-8), MYD88 (ex3-5), NOTCH1 (ex25-28, 34), SF3B1 (ex13-19), TP53 (ex2-11)

LABORATORIES
201 Route 17 North, 2nd Floor
Rutherford, New Jersey 07070
Phone: 201 528 9187
Fax: 201 933 0787

Patient Name: E7505003, .

Accession Number: BM29-800008

BACKGROUND

Risk stratification in Chronic Lymphocytic Leukemia/Small Lymphocytic Lymphoma (CLL/SLL) currently comprises the use of a combination of clinical and molecular features. [1] Molecular analyses generally include assessment of the mutation status of the clonal IGHV rearrangement, gain or loss of specific genomic loci, and more recently somatic mutations in relevant genes: TP53, NOTCH1, SF3B1, BIRC3, ATM, and MYD88. [1] For untreated CLL/SLL patients, 7-10% exhibit mutations in TP53, 5-12% in NOTCH1, 4-10% in SF3B1, 2-5% in BIRC3, 4-10% in ATM, and 1-5% in MYD88, often with exclusivity. [2-4] Mutations in TP53, NOTCH1, SF3B1, BIRC3, and ATM are found to be associated with more aggressive disease and unfavorable outcome. [6-14] On the other hand, MYD88 mutations are predominantly found in cases with mutated clonal IGHV rearrangement, associated with favorable outcome. [4,12] CARD11 mutations are suggested to prognostic value in mature B-cell lymphomas in response to certain treatments that are also indicate for CLL/SLL. [13,14]

1. Zelenetz et al. NCCN Clinical Practice Guidelines in Oncology: non-Hodgkin's lymphomas. J Natl Compr Canc Netw, 2013;11:257-7
2. Puente et al. Whole-genome sequencing identifies recurrent mutations in chronic lymphocytic leukaemia. Nature, 2011;475:101-5
3. Fabbri et al. Analysis of the chronic lymphocytic leukemia coding genome: role of NOTCH1 mutational activation. J Exp Med. 2011;208:1389-401
4. Wang et al. SF3B1 and other novel cancer genes in chronic lymphocytic leukemia. N Engl J Med, 2011;365:2497-506
5. Zenz et al. TP53 mutation and survival in chronic lymphocytic leukemia. J Clin Oncol, 2010;28:4473-9
6. Rossi et al. Disruption of BIRC3 associates with fludarabine chemorefractoriness in TP53 wild-type chronic lymphocytic leukemia. Blood, 2012;119:2854-62
7. Guarini et al. ATM gene alterations in chronic lymphocytic leukemia patients induce a distinct gene expression profile and predict disease progression. Haematologica, 2012;97:47-55
8. Mansouri et al. NOTCH1 and SF3B1 mutations can be added to the hierarchical prognostic classification in chronic lymphocytic leukemia. Leukemia, 2013;27:512-4
9. Rossi et al. Clinical impact of small TP53 mutated subclones in chronic lymphocytic leukemia. Blood, 2014;123:2139-47
10. Stilgenbauer et al. Gene mutations and treatment outcome in chronic lymphocytic leukemia: results from the CLL8 trial. Blood, 2014;123:3247-54
11. Messina et al. Genetic lesions associated with chronic lymphocytic leukemia chemo-refractoriness. Blood, 2014;123:2378-88
12. Landau et al. Evolution and impact of subclonal mutations in chronic lymphocytic leukemia. Cell, 2013;152:714-26
13. Bohers et al. E Targetable activating mutations are very frequent in GCB and ABC diffuse large B-cell lymphoma. Genes Chromosomes Cancer. 2014; 53:144-53
14. Carbone et al, Diffuse large B cell lymphoma: using pathologic and molecular biomarkers to define subgroups for novel therapy. Ann Hematol. 2014;93:1263-77...

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Accession Number: BM29-800008

METHODOLOGY

Genomic DNA was extracted from peripheral blood or bone marrow and quantitated. For next-generation sequencing, library preparation was performed by a PCR-based multiplexed DNA amplification procedure using AmpliSeq library preparation kit (Illumina) on a custom designed panel. The target regions of interest comprised 171 amplicons for 7 genes (ATM, BIRC3, CARD11, MYD88, NOTCH1, SF3B1 and TP53). The PCR amplified libraries were normalized using a bead-based method and pooled prior to bi-directional sequencing on a Miseq system (Illumina). Sequencing data was analyzed using the on-instrument MiSeq Local Run Manager (LRM) software (v3.2) including base calling, automated alignment with human genome build GRCh37/hg19 reference, and somatic variant calling using Somatic Variant Caller algorithm. Variant annotation and filter was performed using Agilent Alissa v5.2.7, which compiles information from the following public databases: RefSeq, COSMIC, ClinVar, dbSNP, ExAc, 1000 Genomes Project, and HGVSc. Synonymous variants and nucleotide polymorphisms considered benign are not reported. Non-synonymous single/multiple nucleotide variants (pathogenic or of uncertain significance) and short insertions/deletions in covered exons and splice junctions are reported. Depending on variant allele frequency, type, and coordinate, variants are confirmed by conventional Sanger sequencing, real time PCR, fragment analysis, repeat independent NGS analysis. Functional impact of variants was based on Polyphen-2 and SIFT predictions, published literature, and somatic origin as reported in COSMIC and for TP53 a custom database (http://p53.curie.fr). The analytical sensitivity of this assay is 5% at >500x read depth and 20% at 100-500x read depth. Specificity of the assay is expected to be >99%.

This NGS panel is designed to detect mutations in 170 amplicons from 7 genes. The 7 genes are not sequenced entirely. Other mutations in each gene outside of the amplicons sequenced will not be detected. This test does not detect large insertions/deletions (over 25bp), genomic copy number variants, or chromosome translocations. This test cannot differentiate between somatic and germ line mutations.

Annotation sources

CIViC	Version 13	CIViC - Clinical Interpretations of Variants in Cancer - release 01-Jan-2020
ClinVar	Version 18	NCBI ClinVar 2020-01
OMIM	Version 13	OMIM 2020-01-28
VariantFunction	Version 33	Transcript based Variant annotation
1000GenomesPh	Version 2	1000 Genomes Phase 3 release v5 (10 September 2014), including GRCh38 data
ExAC	Version 2	ExAC release 1.0 - including GRCh38 from liftover data
gnomAD	Version 2	gnomAD release 2.0.2 - with additional multi-allelic insertions and GRCh38 statistics from the lift-over vcf
COSMIC	Version 16	COSMIC release v90
dbSNP	Version 6	dbSNP build 151
ESP6500	Version 3	Variants in the ESP6500SI-V2 dataset of the exome sequencing project (ESP), annotated with
dbNSFP	Version 4	dbNSFP v3.0b2: Database of functional predictions for non-synonymous SNPs
1000Genomes	Version 1	1000 Genomes Phase1 release v3.20101123

Electronically approved by:

Boaz Kurtis, M.D.
Date: 07/10/2020 11:34 AM EST

End of Report