KEPUTUSAN DEKAN
SEKOLAH ILMU DAN TEKNOLOGI HAYATI
INSTITUT TEKNOLOGI BANDUNG
NOMOR: 79/IT1.C11/SK-KM/2022

TENTANG

PENETAPAN PENERIMA BANTUAN VISA, PENERBITAN ITAS ONLINE DAN TIKET PERJALANAN MAHASISWA PROGRAM *GANESHA TALENT ASSISTANTSHIP – INTERNATIONAL FELLOW* (GTA-IF) 2022 SEKOLAH ILMU DAN TEKNOLOGI HAYATI (SITH) INSTITUT TEKNOLOGI BANDUNG

DEKAN SEKOLAH ILMU DAN TEKNOLOGI HAYATI INSTITUT TEKNOLOGI BANDUNG,

Menimbang :

a. bahwa SITH ITB merupakan satuan akademik yang berfungsi untuk menyelenggarakan program Sarjana, Magister dan Doktor;
b. bahwa untuk meningkatkan motivasi belajar mahasiswa perlu diberikan bantuan untuk mehasiswa, dimana program beasiswa Ganesha Talent Assistantship – International Fellow (GTA)-IF adalah program beasiswa yang memberikan pembebasan seluruh biaya Pendidikan dan menyediakan bantuan biaya hidup bagi mahasiswa program pascasarjana doktoral ITB yang diberikan kepada mahasiswa asing yang memenuhi syarat;
c. bahwa mahasiswa SITH yang namanya tercantum dalam keputusan ini dinilai layak menerima bantuan dana kegiatan Ganesha Talent Assistantship - International Fellow (GTA)-IF dari SITH ITB;
d. bahwa sehubungan dengan butir a, b, c dan d di atas, maka perlu diterbitkan Keputusan Dekan Sekolah Ilmu dan Teknologi Hayati ITB.

Mengingat :

1. Undang-Undang RI Nomor 20 Tahun 2003 tentang Sistem Pendidikan Nasional;
2. Undang-Undang RI Nomor 12 Tahun 2012 tentang Pendidikan Tinggi;
3. Peraturan Pemerintah RI Nomor 65 Tahun 2013 tentang Statuta Institut Teknologi Bandung;
4. Peraturan Rektor ITB Nomor 060/SK/I.A1/KP 2013 tentang Pendelegasian Kewenangan Menandatangani Surat Keputusan dan Surat Tugas di Lingkungan Institut Teknologi Bandung;
5. Keputusan Majelis Wali Amanat ITB Nomor 005/SK/I1-MWA/KP/2020 tentang Pengangkatan Rektor Institut Teknologi Bandung Periode 2020-2025;
6. Keputusan Rektor ITB Nomor 015/SK/I1.A/KP/2020 tentang Pengangkatan Para Wakil Rektor Institut Teknologi Bandung Periode 2020-2025;
7. Keputusan Rektor ITB Nomor 212/IT1.A/SK/KP/2020 tentang Pengangkatan Dekan Fakultas/Sekolah di Lingkungan Institut Teknologi Bandung Periode 2020-2024.

M E M U T U S K A N:

Menetapkan :

PERTAMA : Memberikan dana bantuan Visa, Penerbitan Itas Online dan Tiket Perjalanan mahasiswa Program Ganesha Talent Assistantship - International Fellow (GTA)-IF Untuk Mahasiswa Program Doktor Biologi SITH sebagai berikut :

Nama	NIM	BANTUAN		
		Visa	Penerbitan ITAS Online	Tiket Perjalanan
Jawad	30622701	2.500.000,-	2.500.000,-	10.700.000

KEDUA : Sumber biaya yang timbul sehubungan dengan diterbitkannya Keputusan ini dibebankan kepada anggaran ITB atau sumber lain yang sah dan dianggarkan melalui Rencana Kerja dan Anggaran (RKA) SITH ITB.

KETIGA : Keputusan ini berlaku sejak tanggal ditetapkan sampai dengan tanggal 31 Desember 2022, dengan ketentuan apabila terdapat perubahan/kekeliruan akan diperbaiki sebagaimana mestinya.

Ditetapkan di Bandung
pada tanggal 27 September 2022

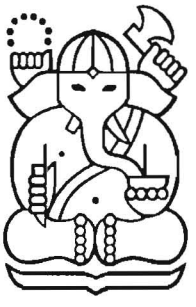
DEKAN,

ENDAH SULISTYAWATI, S.Si., Ph.D.
NIP 19691119 199512 2 001

Tembusan Yth.:
1. Rektor;
2. Para Wakil Rektor dan Sekretaris Institut;
3. Ketua Satuan Penjaminan Mutu;
4. Dekan Sekolah Pascasarjana;
5. Kepala Biro Administrasi Umum dan Informasi;
6. Kepala Kantor Hukum;
7. Masing-masing yang bersangkutan.

PROPOSAL

Ganesha Talent Assistantship – International Fellow (GTA-IF) 2022



Fakultas/Sekolah : Sekolah Ilmu dan Teknologi Hayati

INSTITUT TEKNOLOGI BANDUNG
2022

IDENTITAS PENGUSUL

1. Pengusul : Sekolah Ilmu dan Teknologi Hayati

2. Penanggung Jawab :
   Nama : Dr. Endah Sulistyawati
   Alamat :
   Telepon Kantor : 022 - 251 1571
   Telepon Genggam (*Whatsapp*) : 08122041599
   e-mail : dekan@sith.itb.ac.id

Bandung, 9 September 2022
Menyetujui,

Dekan Sekolah Ilmu dan Teknologi Hayati

Endah Sulistyawati, S.Si., Ph.D.
NIP. 196911191995122001

Nama Kegiatan:
Ganesha Talent Assistantship – International Fellow (GTA-IF) 2022

Introduction and Theoretical Framework:

Despite numerous advances in our knowledge of cancer our ability to develop clinically effective therapies based on this understanding has met with limited success [1]. Current therapies can control tumor growth initially, but most patients ultimately relapse. One prominent example is lung cancer, the leading cause of cancer-related mortality with over 1 million deaths each year (2). Non- small cell lung cancer (NSCLC) accounts for approximately 85% of all lung cancers. Although NSCLC patients with epidermal growth factor receptor (EGFR) mutations respond to EGFR inhibitors initially, most patients experience a relapse within 1 year (3,4).

The third most typical cancer kind and the leading cause of cancer-related fatalities in the United States is lung cancer. Males are most likely to get it, and in the United States, Black men are around12% more likely than White men to do so, according to the American Cancer Society (ACS). Healthy cells undergo modifications as a result of cancer. Without perishing, the cells continue to expand. NSCLC and SCLC are the two primary subtypes of lung cancer. Under a microscope, you can notice that their cell sizes are different. Depending on how they appear under a microscope, the two primary kinds of lung carcinoma are small-cell lung cancer (SCLC) and non-small cell lung cancer (NSCLC). SCLC is less frequent than NSCLC. NSCLC accounts for about 84 percent of lung cancer incidences in the US. Three different subtypes exist: Adenocarcinoma, Squamous Cell Carcinoma, and Large Cell Carcinoma are three cancer types.

In the United States, SCLC accounts for around 13% of instances of lung cancer. Compared to NSCLC, this kind typically grows more quickly. Stage of NSCLC, Imaging scans do not reveal the malignancy, however mucus or phlegm may contain malignant cells. Only the outermost layers of the cells lining the airways contain aberrant cells. There is a tumor in the lung, but it is less than 4 centimeters (cm) in size and has not migrated to other body areas. The tumor is 7 cm or smaller and may have metastasized to adjacent lymph nodes and organs. Lymph nodes,

additional lung tissue, and the surrounding region have all been affected by the cancer's spread. Far-off body parts like the bones or brain have been affected by the cancer's spread. SCLC's own factors. Limited and widespread phases are used to describe whether the cancer is internal or external to the lungs. Although it may already be present in some nearby lymph nodes, the cancer at the restricted stage only affects one side of the chest. These findings underscore the urgent need for both combination therapies and also new approaches to treat cancerous tumors. One such approach may be to target tumor-initiating cells (TICs). Data from leukemias, germ cell tumors, and a number of solid tumors support the notion that cancers are maintained by a subpopulation of self-renewing and evolving TICs. This is also popularly known as the cancer stem cell (CSC) model (5). Although the validity of the CSC model is an issue of controversy in melanoma (6), many other solid tumors appear to follow the CSC model (7). Recently it was proposed that at earlier stages of tumorigenesis, rare TIC clones differentiate into nonmalignant progeny to form the bulk of the tumor, whereas at advanced stages, TIC clones constitute the bulk of the tumor (8). Studies with mouse models of lung cancer have also begun to reconcile the connection between the evolving genotype of TIC clones and the surface phenotype of TICs (9). Thus, accumulated findings suggest that targeting TICs may be a promising approach for eradicating tumors early. However, progress in the targeting of TICs to improve cancer therapy has been hindered by a lack of understanding of the molecular pathways that are critical to TICs. Recent studies have led to an emerging appreciation of the importance of metabolic reprogramming in cancer (10). Most recently, the embryonic isoform of pyruvate kinase PKM2, in collaboration with phosphoglycerate mutase, was found to regulate the shift from oxidative phosphorylation to glycolysis in cancer cells (11). These findings have led to a resurgence of interest in the Warburg effect—the phenomenon whereby cancer cells, like embryonic cells, preferentially use glycolysis even under aerobic conditions (12). Besides glycolysis, an arm of metabolism that results in sarcosine production has also been implicated in prostate cancer (13). These data suggest that metabolic reprogramming is crucial for tumorigenesis, and much remains to be uncovered. Here we show that glycine metabolism and the metabolic enzyme glycine decarboxylase (GLDC) drive TICs and tumorigenesis in NSCLC. Using CD166 as a surface marker and NOD/SCID Il2rg _/_ mice as xenotransplantation recipients, we isolated lung TICs from a broad range of primary NSCLC tumors (stages I–III).

Primary lung TICs express high levels of LIN28B, GLDC, and many other glycine/serine metabolism enzymes. Both LIN28B and GLDC were required for lung TIC proliferation and tumor growth. Overexpression of GLDC alone, and other glycine/serine enzymes, promotes cellulartransformation both in vitro and in vivo. Metabolomic analysis shows that GLDC over expression induces dramatic changes in glycolysis and glycine metabolism, leading to changes in pyrimidine metabolism for cancer cell proliferation. In human patients, aberrant upregulation of GLDC is significantly associated with higher mortality from lung cancer, and aberrant GLDC expression is observed in multiple cancer types. Our findings establish a link between glycine metabolism and tumorigenesis and may provide novel targets for advancing anticancer therapy.

Problem Statement:

Despite numerous advances in our knowledge of cancer our ability to develop clinically effective therapies based on this understanding has met with limited success. metabolic enzyme glycine decarboxylase (GLDC) is critical for TICs in non-small cell lung cancer (NSCLC). TICs from primary NSCLC tumors express high levels of the oncogenic stem cell factor LIN28B and GLDC, which are both required for TIC growth and tumorigenesis. Overexpression of GLDC and other glycine/serine enzymes. but not catalytically inactive GLDC, promotes cellular transformation and tumorigenesis.

Purpose of the Study:

Identification of the factors critical to the tumor-initiating cell (TIC) state may open new avenues incancer therapy Here we show that the metabolic enzyme glycine decarboxylase (GLDC) is criticalfor TICs in non-small cell lung cancer (NSCLC). TICs from primary NSCLC tumors express highlevels of the oncogenic stem cell factor LIN28B and GLDC, which are both required for TICgrowth and tumorigenesis. Overexpression of GLDC and other glycine/serine enzymes, but notcatalytically inactive GLDC,promotes cellular transformation and tumorigenesis. We found that GLDC induces dramatic changes in glycolysis and glycine/serine metabolism, leading to changes in pyrimidine metabolism to regulate cancer cell proliferation. This link between glycine metabolism and tumorigenesis may provide novel targets for advancing anticancer therapy.

Kebutuhan Dana:

Komponen Biaya	Biaya (Rp)	Jumlah	Total Biaya (Rp)
Living Allowance	4.000.000	4 bulan	16.000.000
Tiket Pakistan-Indonesia Ekonomi	10.708.330	1 kali	10.708.330
Visa (TELEX dan VITAS)	2.500.000	1 kali	2.500.000
Penerbitan ITAS Online	2.500.000	1 kali	2.500.000
TOTAL BIAYA			31.708.330

Data mahasiswa peserta:

No	Nama	NIM	Email	Bank	Nomor Rekening
1	Jawad	30622701	jawadafridi585@gmail.com	BNI	1471299958