(*University of Choice*)

MASINDE MULIRO UNIVERSITY OF SCIENCE AND TECHNOLOGY (MMUST)

MAIN CAMPUS

UNIVERSITY EXAMINATIONS
2019/2020 ACADEMIC YEAR

THIRD YEAR FIRST SEMESTER EXAMINATIONS

FOR THE DEGREE
OF
BACHELOR OF SCIENCE MEDICAL BIOTECHNOLOGY

MAIN EXAM

COURSE CODE: BMB 322

COURSE TITLE: MOLECULAR ONCOLOGY

DATE: 7TH DECEMBER 2020 **TIME: 2.00 -4.00 PM**

Instructions

Time 2 hours

Answer all questions

Section A comprise of 20 MCQs 1 mark each

Section B comprise 8 short answer questions: a total of 40 marks

Section C comprises of 3 long essay questions: 20 marks each

SECTION A: multiple choice questions (MCQs)

1. p53 is a tumour suppressor protein that has an important role during which part of the cell cycle:
    a. G1
    b. G2
    c. S
    d. M

2. Migration of tumor cells from primary site forming secondary tumour foci is known as:
    a. Metastasis
    b. Invasion
    c. Diapedesis
    d. Cancer

3. Which is NOT a typical mechanism by which a proto-oncogene is converted to an oncogene?
    a. Complete deletion of the proto-oncogene
    b. A point mutation in the proto-oncogene
    c. Amplification of the proto-oncogene
    d. A chromosomal translocation resulting in the up-regulation of the proto-oncogene

4. Proto-oncogenes are:
    a. normal cellular genes which promote cellular growth
    b. normal cellular genes which inhibit cellular growth
    c. mutated genes
    d. genes that promote apoptosis

5. All of the following mechanisms are involved in the conversion of proto-oncogenes to oncogenes except:
    a. Point mutations
    b. Chromosomal Translocation
    c. Gene amplification
    d. Loss of function mutations
6. Which of the following is a tumour suppressor gene?
    a. C-myc
    b. p53
    c. Ras
    d. c-erbB

7. Up to 80% of Burkits lympomas are associated with which of the following c-myc gene translocations:
    a. t (8, 14)
    b. t (8, 22)
    c. t (2, 8)
    d. t (14, 8)

8. Which of the following tumors exhibit 10-20 fold amplification of the L-myc gene?
    a. Adrenal carcinoma
    b. Small cell lung carcinoma
    c. Burkits lymphoma
    d. Chronic myelogenous leukaemia

9. Which of the following tumors exhibit up to 5-1000 fold amplification of the N-myc gene?
    a. Adrenal carcinoma
    b. Small cell lung carcinoma
    c. Epidermoid carcinoma
    d. Neuroblastoma

10. Which of the following exhibit a change of glutamine to lysine at amino acid number 61 of the N-ras protein?
    a. Lung carcinoma
    b. Adrenal carcinoma
    c. Retinoblastoma
    d. Colon cancer

11. Which of the following exhibit a change of glycine (gly) to cysteine (Cys) at amino acid number 12 of the K-ras protein?
    a. Lung carcinoma
    b. Adrenal carcinoma
    c. Retinoblastoma
    d. Bladder carcinoma

12. Conversion, or activation, of a proto-oncogene into an oncogene generally involves
    a. Gain-of-function mutations
    b. Loss of function mutations
    c. Tumorigenesis
    d. Metastasis
13. Which of the following factors is NOT antiapoptotic?
    a. Bcl-2
    b. Bcl-xL
    c. Mcl-1
    d. Bax

14. Which of the following molecules is not an immune checkpoint target for immunotherapy?
    a. Cytotoxic T-lymphocyte antigen-4 (CTLA-4),
    b. Programmed cell death-1 (PD-1)
    c. Programmed cell death ligand-1 (PD-L1)
    d. Caspase 3

15. Inherited mutations in BRCA1 or BRCA2 significantly increase risk of:
    a. Colon cancer
    b. Lung cancer
    c. Liver cancer
    d. Breast cancer

16. Microsatellite instability is the production of new alleles from:
    a. Unrepaired replication errors
    b. Pseudogenes
    c. Proto-oncogenes
    d. Dna repair genes

17. In microsatellite analysis of tumor tissues, the unstable microsatellite loci appear in the gel as:
    a. Point mutations

b. Insertions
c. Translocations
d. Extra products in tumor tissue compared to normal tissue

18. Which of the following molecules does **NOT** play a pro-angiogenic role in tumor neo-angiogenesis?
    a. Vascular endothelial growth factors (VEGF)
    b. Fibroblast growth factor-2 (FGF2)
    c. The platelet derived growth factor (PDGF)
    d. Thrombospondins (TSP)

19. PTEN is mutated in most cancers; what is its normal role in PI3K/Akt/mTOR signalling pathway
    a. Catalyse the dephosphorylation of PI3K
    b. Conversion of PIP3 to PIP2
    c. Phosphorylates Akt
    d. Dephosphorylates Akt

20. Microsatellite instability in tumors is detected by:
    a. comparing PCR amplicons of the microsatellite loci
    b. Immune histochemistry
    c. RNA sequencing
    d. DNA sequencing

SECTION B: SAQs: Answer all (40 marks)

1. Describe two mechanisms that lead to loss of heterozygosity (LOH) of tumor suppressor genes **(8marks)**

2. Describe three mechanisms that convert proto-oncogenes to oncogenes **(6 marks)**

3. Using colon cancer as an example, illustrate the role of proto-oncogene and tumour suppressor gene mutations in initiation and progression of carcinomas **(10 marks)**

4. Describe the intrinsic apopototic pathway **(8 marks)**

5 Explain FOUR main molecular mechanisms of cancer chemotherapy resistance **(8marks)**

SECTION C: LAQs: Answer any Three (60 marks)

1. Discuss the steps and mechanisms in tumor metastasis **(20 marks)**

2. Describe in detail the steps involved in second generation cancer Genome sequencing techniques **(20 marks)**

3. Outline the PI3K/Akt pathway and illustrate specific steps that are abnormal in tumors (**20 marks)**

4. Describe the molecular mechanisms involved in tumor angiogenesis **(20 marks)**