بسم الله الرحمن الرحيم

Past Papers

MID – Lecture 13 to 16

Cytology and Molecular

﴿ وَإِن تَتَوَلَّوْا يَسْتَبْدِلْ قَوْمًا غَيْرَكُمْ ثُمَّ لَا يَكُونُوا أَمْثَالَكُم ﴾

اللهم استعملنا ولا تستبدلنا

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First we will start with (13) past Qs

then there will be (14) test bank Qs

(all Qs will be by default past unless it is written to be test bank)

- Note: There are no past paper questions on the topic of "cell cycle" and "Cancer," so you should solve test bank questions to assess your understanding.

Q1 : What group of enzymes phosphorylates most of the carbons on inositol?

A)Phospholipases

b) Phosphoinositide kinases

c) Phosphorylases

d) Phosphodiesterases

e) phosphatases

Answer : b

Q2 : How is signaling by an activated Gα subunit terminated?

A. The GTP bound to the protein is broken down into GMP.

B. The GDP bound to the protein is broken down into GTP.

C. The GTP bound to the protein is broken down into GDP.

D. The GDP bound to the protein is converted to GTP by adding a phosphate group

E. .The Gα subunit releases GDP and binds to GTP.

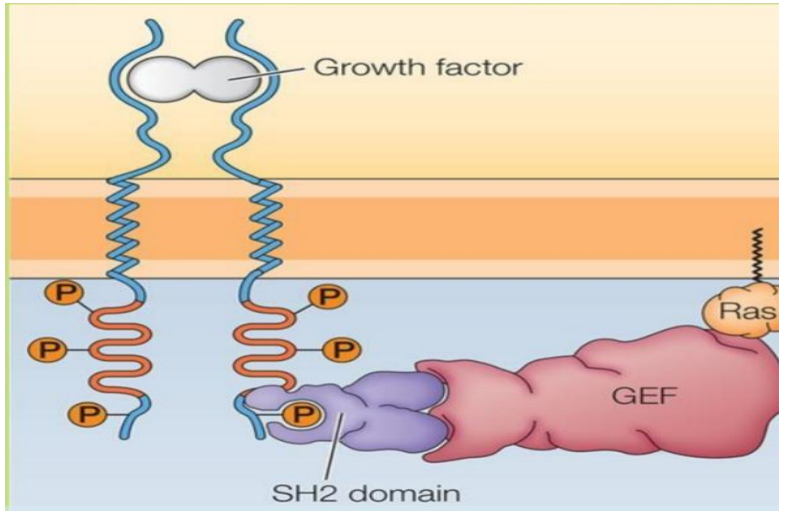
Answer : C

Q3 :SH2 domain is found in:

- A) Ras protein
- B) RTK- linked adaptor proteins
- C) Sos
- D) RTK
- E) GBCR

Answer : B

Q4 : What is the function of GEF particle?



A. Inhibit RAS-GTP complex
B. stimulate the exchanging (GDP To GTP) on Ras
C. Synthesis of heme group
D. A+C
E. All of the above

Note: This question is derived from the idea of another question that was presented in a different format.

Answer : B

Q5 : Most protein kinases transfer phosphate groups to which amino acid(s)?

1) glutamate
2) threonine
3) serine
4) Tryptophan

a) 1 b) 2 c) 3 d) 4 e) 2 and 3

Answer : e

Q6 : What is the largest protein superfamily encoded by animal genomes?

- A) G-protein coupled receptors
- b) RTKs
- c) steroid receptors
- d) tubulin superfamily
- e) ligand-gated channels

Answer : A

Q7 :Place the following events in the proper order.

a) 4 – 5 – 2 – 6 – 3 – 1

b) 5 – 4 – 2 – 6 – 3 – 1

c) 4 – 6 – 2 – 5 – 3 – 1

d) 4 – 5 – 2 – 3 – 1 – 6

Step	Event
1	Activation of one or more cellular signaling proteins.
2	Dissociation of Gα from the G protein complex.
3	Production of a second messenger, like cAMP.
4	Replacement of GDP by GTP on the Gα after interaction with an activated GPCR.
5	Conformational change in the Gα subunit causing a decreased affinity for the Gβγ subunit.
6	Gα-subunit with its attached GTP activates an effector like adenylyl cyclase.

Answer : A

Q8 :Cytochrome c, cytoplasmic factors and procaspase 9 form

- A-executioner caspases
- B- executioner procaspases
- C- initiator caspase 8
- D- apoptosomes
- E-A&B

Answer : D

Q9 : Which of the following is correct about apoptosis ?

- A-the extrinsic pathway of apoptosis is initiated by DNA damage
- B- diabetes type 2 is linked to elevated apoptosis
- C- phosphatidylserine is moved to the outer leaflet of the PM to attract . . . . Macrophages
- D- apoptosis isn’t needed in the embryonic development
- E- none of the above

Answer : C

Q10 : What is the name of an extracellular messenger protein that is named for its ability to kill tumor cells and also serves as an apoptotic stimulus?

- a) tumor angiogenesis factor
- b) tumor death factor
- c) tumor necrosis factor
- d) necromancer factor
- e) tumorigenic factor

Answer : C

Q11 : What process is responsible for organelle turnover in the cell and carries out the regulated destruction of the cell's own organelles for the purpose of recycling the components of which they are made?

- A. Autolysis
- B. Autophagolysosome
- C. Apoptosis
- D. Autophagy
- E. Autonomy

Answer : D

Q 12: The molecule which marks the cell for destruction macrophages is:

- a) phosphatidylcholine
- b) phosphatidylserine
- c) phosphatidylethanolamine
- d) phosphatidylinositol
- E)c+d

Answer : b

Q13 : What is not true about programmed cell death?

- A) Apoptosis is needed during embryonic development and during adult
- B) Macrophages engulf and clear apoptotic cells
- C) Extrinsic apoptotic pathway involves activation of procaspase 9
- D) Necroptosis ends by cell lysis and inflammation of surrounding tissue
- E) Intrinsic apoptotic pathway involves mitochondria and activation ofprocaspase 9

Answer : C

Test bank Qs

Test bank Q

Q14 : Which factor regulates the G1 to S phase transition?

- A) p53 protein
- B) CDK1
- C) Cyclin D
- D) RB
- E) CDK4

Answer : D

Test bank Q

Q15 :What is the role of Ras in the Ras/Raf/MAPK pathway?

- A) Deactivates Raf
- B) Activates Raf by GTP hydrolysis
- C) Acts as a GTPase
- D) Activates Raf when bound to GTP
- E) Prevents ERK phosphorylation

Answer : D

Test bank Q

Q16 : How do Ras mutations contribute to cancer?

- A) Inhibit Raf activation
- B) Cause constant Ras/MAPK activation, leading to uncontrolled proliferation
- C) Prevent MAPK deactivation
- D) Promote apoptosis
- E) Activate p53

Answer : B

Test bank Q

Q17 :How does p53 mutation affect the Ras/Raf/MAPK pathway in cancer?

- A) Enhances apoptosis
- B) Inhibits Ras activation
- C) Allows unchecked cell division
- D) Switches off the pathway
- E) Activates Raf

Answer : c

Test bank Q

Q18 : What if continuous activation of the Ras/Raf/MEK/ERK pathway and PTEN mutations lead to hyperactivation of the PI 3-kinase/Akt/mTORC1 pathway? How would this affect the crosstalk between the pathways?

- A) It would block mTORC1 activation due to negative feedback from Akt.
- B) Ras activation would enhance PI 3-kinase activity, leading to increased Akt and mTORC1 activation
- C) Hyperactivation of PI 3-kinase would inhibit ERK signaling.
- D) Ras activation would prevent Akt activation.
- E) Hyperactivation of PI 3-kinase would reduce Ras signaling.

Answer : B

Test bank Q

Q19: What would happen if a mutation in the gene encoding IκB leads to the loss of its ability to inhibit NF-κB activation?

- A) NF-κB would be continuously activated, leading to excessive transcription of target genes and potentially contributing to chronic inflammation or cancer.
- B) NF-κB would be permanently inactivated, preventing the transcription of target genes.
- C) The feedback loop would enhance NF-κB activity, increasing IκB production and further inhibiting NF-κB.
- D) Loss of IκB function would have no effect on NF-κB activity, as the pathway can function without feedback regulation.
- E) NF-κB activation would be reduced due to an increase in IκB levels, leading to decreased target gene transcription.

Answer : A

Test bank Q

Q20 : What happens to p53 levels in the presence of DNA damage?

- A) p53 is degraded by the proteasome
- B) p53 is phosphorylated, preventing its degradation and increasing its levels
- C) p53 is ubiquitinated and degraded
- D) p53 remains unaffected
- E) p53 accumulates due to an increase in cell division

Answer : B

Test bank Q

Q21: What is the role of BRCA1 in DNA repair?

- A) It repairs single-strand breaks directly
- B) It activates p53 to arrest the cell cycle and induce DNA repair
- C) It prevents apoptosis in response to DNA damage
- D) It inhibits ATM to stop DNA repair
- E) It causes mutations in p53 that lead to cancer

Answer : B

Test bank Q

Q22 : How do the E6 and E7 proteins of HPV contribute to carcinogenesis?

- A) E6 inhibits the action of p21, and E7 promotes BRCA1 activity
- B) E6 promotes p53 degradation, and E7 inactivates the Rb protein
- C) E6 promotes the repair of DNA double-strand breaks, and E7 induces apoptosis
- D) E6 enhances p53 activity, and E7 inhibits cell division
- E) E6 and E7 repair DNA damage through activation of ATM and ATR

Answer : B

Test bank Q

Q23 : How might a drug that inhibits the activity of ATM, a key DNA damage sensor, influence the outcome of cancer therapies that rely on DNA damage (such as radiation therapy)?

- A) The drug would make cancer cells more susceptible to DNA damage, enhancing therapy efficacy
- B) The drug would reduce DNA repair efficiency, leading to increased genomic instability and tumor progression
- C) The drug would enhance DNA repair mechanisms, reducing the effects of radiation therapy
- D) The drug would prevent apoptosis, allowing tumor cells to survive therapy
- E) The drug would activate p53, enhancing DNA repair and tumor suppression

Answer : B

Test bank Q

Q24: How does Herceptin (trastuzumab) work in the treatment of HER2-positive breast cancer?

- A) Stimulates cancer cell growth
- B) Blocks growth signals by binding to HER2
- C) Increases cancer cell resistance to chemotherapy
- D) Promotes tumor spread by stimulating blood vessel formation
- E) Stimulates the immune system to attack cancer cells

Answer : B

Test bank Q

Q25: How do mutations in PML/RAR affect acute promyelocytic leukemia?

- A) Stimulate differentiation and prevent proliferation
- B) Block differentiation, keeping cells in an actively proliferating state
- C) Stimulate proliferation without affecting differentiation
- D) Enhance immune response against cancer cells
- E) Cause cancer cell destruction through apoptosis

Answer : B

Test bank Q

Q26 : What occurs when IκB is phosphorylated?

- A) NF-κB is inhibited
- B) NF-κB is activated and enters the nucleus
- C) NF-κB stays in the cytoplasm
- D) IκB is deactivated, leading to differentiation
- E) NF-κB inhibits gene expression

Answer : B

Test bank Q

Q27 : What determines if ERK signaling leads to proliferation or differentiation?

- A) Type of cell
- B) Duration of ERK activity
- C) Presence of IκB inhibitors
- D) NF-κB activation
- E) Level of NF-κB phosphorylation

Answer : B

اللهم نسألك لأهل غزة النّصر على من عاداهم، عاجلاً غير آجل يا رب العالمين

For any feedback, scan the code or click on it.

Corrections from previous versions:

Versions	Question #	Before Correction	After Correction
V1 → V2			
V2 → V3			