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3rd year Test Examination, 2017

**Molecular Biology and Biotechnology
Paper-VIII**

Full marks: 50

Time: 2hours

USE SEPARATE ANSWER SHEETS FOR EACH UNIT

Unit-I

Marks: 25

A. Answer any FIVE:

5*1

- (1) Enhancers are tissue specific but NOT orientation dependent – T/F
- (2) Genes transcribed by RNA Pol III often have internal promoters – T/F
- (3) RNA Pol III transcribes 5S RNA – T/F
- (4) Xeroderma Pigmentosa is caused due to mutation in DNA repair pathway – T/F
- (5) De-amination of Cytosine leads to formation of _____
- (6) Two Dimensional DIGE is a technique used in _____ studies
- (7) Give one example each for a DNA alkylating agent and a Base analog.
- (8) What is the function of DNA photolyase?

B. Briefly answer any FIVE:

5*2

- (1) Name a disease associated with tri-nucleotide repeat expansion. What is ‘Anticipation’?
- (2) What are the 2 kinds of tautomeric shifts observed in nitrogenous bases of DNA? In total, how many types of base substitutions (transition+transversion) can occur?
- (3) Show the subunits and RNA components of prokaryotic 70S ribosome by a flowchart?
- (4) What is the function of RNase P? How does it differ from other protein-enzymes?
- (5) What do you understand by the term ‘mutation’? Between expected and observed error rate in DNA replication, which one is more?
- (6) Name the 2 excision repair pathways. What are ‘AP’ sites? In which chromosomal event do you find Holliday junction?
- (7) Enlist any 4 Hallmarks of Cancer OR Illustrate post-replication DNA mismatch repair.
- (8) How can you correlate physical maps of chromosomes with genetic and cytological maps?

C. Answer any TWO:

2*5

- (1) Name an assay used to characterize protein binding sites on gene promoter. What is -10 consensus sequence on prokaryotic promoter popularly known as? By one simple sketch show what you understand by sense/ antisense, +/- or template/ non-template strand in DNA. (1+1+3)
- (2) What is so special about Group I splicing mechanism? In Spliceosome mediated splicing, the intron is released in the form of _____. What does snRNP stand for? In Systemic Lupus Erythematosus, autoantibodies are generated against _____. In Group II splicing, a 2’-5’ phosphodiester bond formation is observed (T/F) (1+1+1+1+1)
- (3) Mutations in genes coding for enzymes often manifest as recessive diseases. Explain why? Name 3 such diseases encountered in phenylalanine – tyrosine metabolic pathway? (2+3)
- (4) Draw a simple flowchart/ figure to depict the Lederberg’s replica plating experiment. Why do you think rat liver extract increased the sensitivity of Ames test? (3.5 + 1.5)

D. Answer any five:

5*1

1. EBI stands for _____.
2. **What is EC50 ?**
3. SAR's stands for _____.
4. **If two sequences in an alignment share a common ancestor, mismatches can be interpreted as _____.**
5. **Small ligand binding sites are usually _____.**
6. **The lower the E-value, or the closer it is to zero, the more "significant" the match is. T/F**
7. HTU stands for _____.
8. What is Topology?

E. Answer any five:

5*2

1. **What is Global Alignment ?**
2. **Why do we insert gaps between the residues?**
3. **State the difference between Gap Opening Penalties and Gap Extension Penalties?**
4. **Pattern - C-x(2,4)-C-x(3)-[LIVMFYWC]-x(8)-H-x(3,5)-H.... explain it.**
5. **What is cladogram?**
6. **What Is Lead compound?**
7. **What Is Pharmacokinetics?**
8. **How do we modify various functional groups in the lead compound ?**

F. Answer any two:

5*2

1. **Describe Blast algo.**
2. **Align the following sequences using Dynamic programming algorithm**
Seq1 TAGA Match= 1, Mismatch= 0, Gap= 0

Seq2 GA
3. **Define Rooted tree with diagram. What is Paralog? When do we use Maximum Parsimony method? 3+1+1**
4. **What is Pharmacophore? How do we chemically modify a lead compound? 2+3**