Time Table Semester – II (Venue: New Class Room)

Day	9:00-10:00	10:00-11:00	11:15-12:15	12:15-1:15	1:45-5:45
Monday	Human Physiology MBS 205 **(Dr. Adila Parveen)**	Organic Chem. II MBS 201 **(Dr. Manisha Tiwari)**	Immunology MBS 204 **(Dr. Anju Katyal)**	Molecular Biology and Biotechnology MBS 202 **(Dr. Ajay K. Yadav)**	**Batch 1** Molecular Biology and Biotechnology **(Prof. B. C. Das, Dr. Harsimrut Kaur & Dr. Ajay Yadav)** **Batch 2** Immunology **(Dr. Anju Katyal & Prof. K. Natrajan)**
Tuesday	Molecular Biology and Biotechnology MBS 202 **(Prof. Daman Saluja)**	Immunology MBS 204 **(Dr. Anju Katyal)**	Application of Statistics & Mathematics for Biology MBS 203 **(Dr. L. Satyanarayana/Umesh)**		
Wednesday	Organic Chem II MBS 201 **(Dr. Manisha Tiwari)**	Immunology MBS 204 **(Dr. Anju Katyal)**	Molecular Biology and Biotechnology MBS 202 **(Prof. Daman Saluja)**	Human Physiology MBS 205 **(Dr. L. R. Singh)**	**Batch 2** Organic Chemistry **(Dr. Manisha Tiwari)** **Batch 1** Immunology **(Dr. Anju Katyal & Prof. K. Natrajan)**
Thursday	Human Physiology MBS 205 **(Dr. Adila Parveen)**	Molecular Biology and Biotechnology MBS 202 **(Dr. Ajay Yadav)**	Immunology MBS 204 **(Prof. Natarajan)**	Organic Chem II MBS 201 **(Dr. P. M. Luthra)**	**Batch 1** Organic Chemistry **(Dr. P. M. Luthra)** **Batch 2** Molecular Biology and Biotechnology **(Prof. B. C. Das, Dr. Harsimrut Kaur & Dr. Ajay Yadav)**
Friday	Organic Chem II MBS 201 **(Dr. P. M. Luthra)**	Human Physiology MBS 205 **(Dr. Manisha Yadav)**	Application of Statistics & Mathematics for Biology MBS 203 **(Dr. L. Satyanarayana/Umesh)**		**2:45-4:45 pm Students Seminar**

The theory classes will start from 7th January 2013.

Organic Chemistry II: **Dr. Manisha Tiwari and Dr. P. M. Luthra**
Human Physiology: **Dr. L. R. Singh and Dr. Manisha Yadav, Dr. Adila Parveen**
Molecular Biology and Biotechnology: **Prof. Daman Saluja and Dr. Ajay K. Yadav**
Immunology: **Prof. K. Natarajan and Dr. Anju Katyal**
Applications of Statistics & Mathematics for Biology: **Dr. L Satyanarayanan and Umesh**

Course Co-ordinator

Dr L. R. Singh
10/11/17

DS-NOV-2017/43

Dr. B.R. Ambedkar Center for Biomedical Research
University of Delhi, Delhi-110 007

M.Sc. in

Biomedical Sciences
Semester - I

Final Date-sheet (Theory Exams)
Morning Time: 9:30- 12:30

Date/Day (Time)	Subject	Code	Co-ordinator
30th Nov 2017 Thursday **(Morning)**	Cell Biology	MBS-103	Dr. A.K. Yadav
4th Dec 2017 Monday **(Morning)**	Organic Chemistry-I	MBS-101	Dr. M. Tiwari
7th Dec 2017 Thursday **(Morning)**	Medical Microbiology	MBS-104	Dr. A. Katyal
11th Dec 2017 Monday **(Morning)**	Genetics	MBS-105	Prof V. Brahmachari
15th Dec 2017 Friday **(Morning)**	Biochemistry	MBS-102	Dr. L. R. Singh

7/11/17
विशेष कार्याधिकारी (परीक्षा) / O.S.D.(Examin
दिल्ली विश्वविद्यालय / University of Delh
दिल्ली-110007 / Delhi-110007

02/11/2017
7-11-17

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DS-NOV-2017/44

Dr. B.R. Ambedkar Center for Biomedical Research
University of Delhi, Delhi-110 007

M.Sc. in Biomedical Sciences
Semester - III

Final Date-sheet (Theory Exams)
Morning Time: 9:30- 12:30; Afternoon Time: 2:00 – 5:00

Date/Day (Time)	Subject	Code	Co-ordinator
25th Nov 2017 Saturday **(Morning)**	Human Physiology-II	MBS-301	Dr. L. R. Singh
30th Nov 2017 Thursday **(Afternoon)**	Pharmacology & Toxicology	MBS-305	Dr. K. Srivastava
4th Dec 2017 Monday **(Afternoon)**	Principles of Medicinal Chemistry	MBS-302	Dr. P. M. Luthra
9th Dec 2017 Saturday **(Morning)**	Analytical and Biomedical Techniques & Instrumentation	MBS-303	Dr. M. Chopra
13th Dec 2017 Wednesday **(Morning)**	Molecular Oncology	MBS-304	Prof D. Saluja

विशेष कार्याधिकारी (परीक्षा) / O.S.D. (Examination)
दिल्ली विश्वविद्यालय / University of Delhi
दिल्ली–110007 / Delhi-110007

02/11/2017

7-11-17

Dr. B.R. Ambedkar Center for Biomedical Research
University of Delhi, Delhi-110 007

M.Sc. in Biomedical Sciences
Semester - I

Final Date-sheet (Practical Exams) Time: 9:30- 4:30

Date/Day	Subjects	
20th Nov 2017 Monday	Biochemistry (LRS+AD+DS), Medical Microbiology (AK+KN), Genetics (VB+A)	Batch – I
21st Nov 2017 Tuesday	Biochemistry (LRS+AD+DS), Medical Microbiology (AK+KN), Genetics (VB+A)	Batch – II

M.Sc. in Biomedical Sciences
Semester - III

Final Date-sheet (Practical Exams), Time: 9:30- 4:30

Date/Day	Subjects	
16th Nov 2017 Thursday	Human Physiology (RS+LRS+AD), Instrumentation (MC+LRS+KN), Toxicology and Pharmacology (PML+KS)	Batch – I
17th Nov 2017 Friday	Human Physiology (RS+LRS+AD), Instrumentation (MC+LRS+KN), Toxicology and Pharmacology (PML+KS)	Batch – II

Dalya

02/11/2017

[This question paper contains 5 printed pages]

S. No..... Your Roll No.....

M.Sc. Biomedical Sciences/IV Sem./2016
Paper: Genome Biology (MBS 402)

Time: 3 hours **Maximum Marks : 70**

(Write your Roll No. on the top immediately on receipt of this question paper)

Question Number 1 is compulsory; Out of the remaining six questions answer any Four.

1. Answer the following (Compulsory question) **10x1**

a. What is candidate gene approach?
b. What are Tag SNPs?
c. What is the use of Tag SNP in capturing nucleotide variation in haplotypes?
d. What is the significance of LOD score in human genetics?
e. Name two restriction enzymes you would use to generate genomic DNA fragments for cloning in BAC vectors. Justify your choice.
f. Which of the following techniques is most suitable for discovery of novel splice variants:

   i. In situ Hybridization
   ii. RNAseq
   iii. Microarrays
   iv. Quantitative RT-PCR

g. Gene A encodes a transcription factor TFA, which binds to the promoter of Gene B, inducing its expression. The product of Gene B is a microRNA called miR-B which can repress the translation of the mRNA of GeneC. Recently, it was shown that the product of Gene C is a long non-coding RNA, called lnc1, that regulates neuronal development. The promoter of Gene C was shown to harbour functional binding sites for TFA. On over-expression of TFA, the GFP expression from a reporter plasmid carrying the promoter of GeneC fused to gene for GFP, is heavily induced. From these word arguments, Gene A, B and C is most likely to be connected by the following Network Motif:

   i. Negative auto-regulatory loop
   ii. Positive auto-regulatory loop
   iii. Incoherent feed-forward loop
   iv. Coherent feed-forward loop

h. Why is linkage disequilibrium (LD) greater in Y chromosome, parts of X chromosome and the centromere proximal regions of autosomes?
i. Describe two advantages that make zebrafish a good model organism for studying cardiovascular biology? Justify your choice.
j. What is incomplete penetrance and why is it commonly seen in dominant diseases?

2. Answer the following:

a. Explain the strategy used for classifying cell cycle mutants of yeast into different linkage groups? **3**

b. Mutations in the gene coding for Androgen receptor mapping on the X chromosome can cause two entirely different diseases. Triplet repeat expansion leading to polyglutamine expansion leads to SBMA(Spinal and Bulbar Muscular Atrophy) and point mutations in the same gene leads to Androgen Insensitivity syndrome and feminization of XY individuals. What are possible reasons for the origin of these entirely different phenotypes though the mutation is in the same gene.

4

c. Indicate the distinguishing feature(s) of a pedigree exhibiting a mitochondrial inheritance using a hypothetical pedigree.

4

d. What is locus heterogeneity?Illustrate it with a hypothetical pedigree.

2+2

3. Answer the following:

a. What are the ways in which an miRNA can regulate the expression of a gene, directly or indirectly?

4

b. How can you utilize Cre-lox system to induce the stage specific (developmental) and tissue specific expression of a transgene in mouse.

3

c. How can epigenetic modifications of histones affect drug metabolism?

3

d. Discuss the genesis of IGVdb.

3

e. How are the outcomes of the HapMap project used in meta-analysis of association studies?

2

4. Explain the experimental strategy to address the following problems:

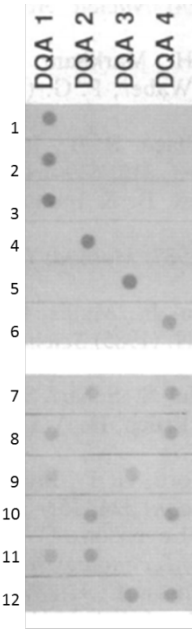
a. A patient of Down syndrome reported to the clinic, but trisomy 21 was not detected in his Karyotype. You are asked to assess the copy number of genes mapping on chromosome 21 in this patient. Briefly outline the strategy you would take.

4

b. List four questions to answer by experiments using ChIP- sequencing (ChIP-seq)as the most suitable technique. What kind of sequencing platform will you use and why?

4+1+1

c. The following blot represents a technique called the Reverse Dot Blotting that has been utilized for polymorphism screening. The following probes (DQA1 to 4) have been used in order to detect variable alleles (arising due to the presence of different SNPs). Data for 12 individuals is given below. DQA1 identifies the ancestral gene whereas the other three probes identify variations caused by different SNPs. DQA4 allele is known to be associated with Cardiomyopathies when in homozygous condition but in a heterozygous state, either its association is diminished (when with DQA3) or enhanced (when with DQA1/2).



i. Which of these individuals are likely to suffer from cardiomyopathies? Order them on the basis of the severity?

2.5

ii. How would you explain the enhancing or the depleting effect of other SNPs with DQA4 allele?

2.5

5. Answer the following:

a. How is IBS different from IBD? Explain giving a hypothetical example.

3

b. Define linkage disequilibrium. What are the commonly used measures to calculate it? Explain.

3

c. Two loci with two alleles each (**A**, **a** and**B**, **b**) are located on the same chromosome. If the frequency of chromosomes in a sample of 500 individuals is as follows: AB: 620, Ab: 180, aB: 80, ab: 120, what is the level of linkage disequilibrium between the two genes?

4

d. Discuss the advantages and limitations of using TDT as a method of association studies.

3

e. Find a minimum set of haplotypes to explain the given genotypes G_1 and G_2 for two SNPs (A/C and G/T): G_1:SNP1- AC, SNP2- GT and G_2:SNP1- CC, SNP2- GG

2

6. Answer the following:

a. Define Network motifs.

2

b. What is Threshold cycle value in the context of quantitative RT-PCR? How is it related to expression level.

2

c. Discuss the relative merits and demerits of the following normalization methods and controls.

3x2=6

i. Normalization by a constitutive/ house-keeping gene, e.g. : beta-Actin.
ii. Quantile normalization.
iii. Normalization to Spike-in controls

d. What is Case-control population sampling and how does “population stratification” affect case-control population sampling.

3

e. What is the aim of GWAS studies? Where would you apply it, in case of monogenic or polygenic disease?

2

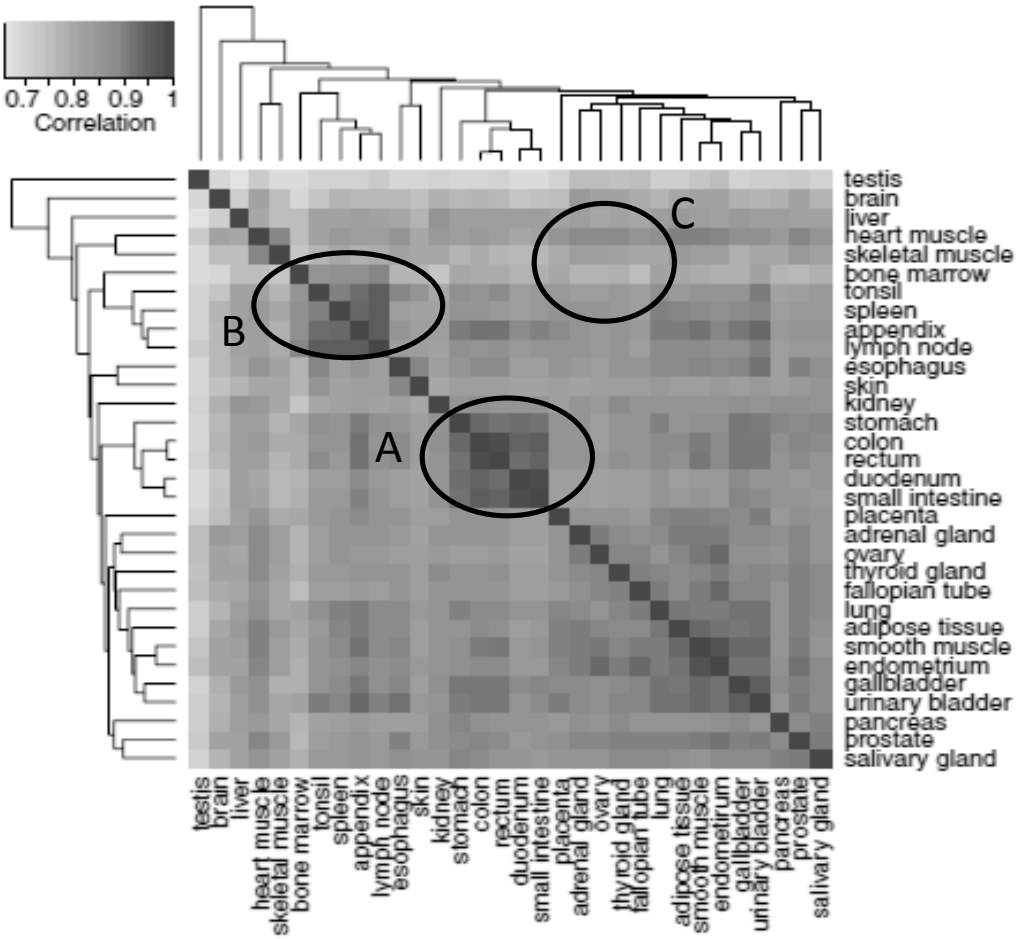
7. Answer the following

a. Name a commonly used oligonucleotide based antisense technique for knocking down gene expression in zebrafish embryos. Illustrate the method with an example.

1+ 3

b. The heat map below shows pairwise correlation between 32 tissues based on transcript levels of 20,344 genes. Interpret the regions marked by circles, A, B and C.

6



c. Name one heavy atom used in SILAC? What is the nature of the substrate that is labeled in this technique.

1

d. Mention any two applications of SILAC?

1

e. A kinase named Protein A is known to phosphorylate the proteins P, Q and Z. Protein A is over-expressed in Glioblastoma. How will you study the increase in phosphorylation of the substrate proteins using SILAC?

3