2.5.4

Biochemistry Department

Midcourse Improvement

Retest & Answer--sheets

2.5.4

Mid Course Improvement

2.5.4 MIDCOURSE IMPROVEMENT

The Institution has defined Policy Document to provide opportunities to students for midcourse improvement of performance through specific interventions

UG

Year	Timely administration of CIE	On time assessment and feedback	makeup assignments/tests please write number of students	remedial teaching/support please write number of students
2015-16	Yes	Yes	Yes (16)	Yes (16)
2016-17	Yes	Yes	Yes (4)	Yes (4)
2017-18	Yes	Yes	Yes (5)	Yes (5)
2018-19	Yes	Yes	Yes (3)	Yes (3)
2019-20	Yes	Yes	-	-

H. O. D.
Department of Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC,
Adgaon, Nashik .2 003

2.5.4 MIDCOURSE IMPROVEMENT

The Institution has defined Policy Document to provide opportunities to students for midcourse improvement of performance through specific interventions.

UG

Year	Timely administration of CIE	On time assessment and feedback	Makeup assignments/tests	Remedial teaching/support
2015-16	Yes	Yes	Yes (one assignment)	Yes (Mentorship)
2016-17	Yes	Yes	Yes (one assignment)	Yes (Mentorship)
2017-18	Yes	Yes	Yes (one assignment)	Yes (Mentorship)
2018-19	Yes	Yes	Yes (one assignment)	Yes (Mentorship) Remedial teaching
2019-20				

H. O. D.
Department of Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC,
Adgaon, Nashik 422003

Department of Biochemistry
Inward No.-Bio/ 19
Date: 28/8/19

Department of Physiology
Outward No. 24
Date: 29/8/19

MARATHA VIDYA PRASARAK SAMAJ'S
DR.VASANTRAO PAWAR MEDICAL COLLEGE & RESEARCH CENTER, ADGAON, NASHIK- 3
DEPARTMENT OF ANATOMY, PHYSIOLOGY & BIOCHEMISTRY

Date 28/8/2019

Time Table of Supplementary Examination

A) Theory Examination

Time Table No -01

Day	Date	Time	Paper
Friday	6/09/2019	10am to 12.30pm	Physiology-I
Saturday	7/09/2019	10am to 12.30pm	Physiology-II
Sunday	8/09/2019	10am to 12.30pm	Anatomy-I
Monday	9/09/2019	10am to 12.30pm	Anatomy-II
Tuesday	10/09/2019	10am to 12.30pm	Biochemistry-I
Wednesday	11/09/2019	10am to 12.30pm	Biochemistry-II

B) Practical Exam

Time Table No -2

Day	Date	Time	Department
Thursday	12/09/2019	9.00am onwards	Physiology
Friday	13/09/2019	9.00am onwards	Anatomy
Saturday	14/09/2019	9.00am onwards	Biochemistry

Prof & HOD
Dept. of Anatomy

HOD
Department of Anatomy
MVP'S DR. Vasantrao Pawar
Medical College, Nashik

Prof & HOD
Dept. of Physiology

HOD
Dept. of Physiology
MVPS Dr. VPMC, Nashik

29.08.19

Prof & HOD
Dept. of Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC.
Adgaon, Nashik-422 003

circulate & Notice board

Department of Biochemistry
Inward No.- Bio/ 25A
Date:- 5/9/18

MARATHA VIDYA PRASARAK SAMAJ'S
DR. VASANTRAO PAWAR MEDICAL COLLEGE & RESEARCH CENTER, ADGAON, NASHIK- 3
DEPARTMENT OF ANATOMY, PHYSIOLOGY & BIOCHEMISTRY

Date 05/09/18

Time Table of Supplementary Examination

A) Theory Examination

Time Table No -01

Date	Time	Paper
06/09/18	10am to 12.30pm	Physiology-I
07/09/18	10am to 12.30pm	Physiology-II
08/09/18	10am to 12.30pm	Anatomy-I
09/09/18	10am to 12.30pm	Anatomy-II
10/09/18	10am to 12.30pm	Biochemistry-I
11/09/18	10am to 12.30pm	Biochemistry-II

B) Practical Exam

Time Table No -2

Day	Date	Time	Department
Thursday	13/09/18	9.00am onwards	Physiology
Friday	14/09/18	9.00am onwards	Anatomy
Saturday	15/09/18	9.00am onwards	Biochemistry

Prof & HOD
Dept. of Anatomy

HOD
Department of Anatomy
MVP'S DR. Vasantrao Pawar
Medical College, Nashik

Prof & HOD
Dept. of Physiology

HOD
Dept. of Physiology
MVPS Dr. VPMC, Nashik

Prof & HOD
Dept. of Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr. Vasantrao Pawar
Medical College, Hospital & RC.
Adgaon, Nashik-422 003

circulate & Notice board

Department of Biochemistry
Inward No.-Bio/ 22A
Date:- 20/08/17

MARATHA VIDYA PRASARAK SAMAJ'S
DR.VASANTRAO PAWAR MEDICAL COLLEGE & RESEARCH CENTER, ADGAON, NASHIK- 3
DEPARTMENT OF ANATOMY, PHYSIOLOGY & BIOCHEMISTRY

Date - 20/08/17

Time Table of Supplementary Examination

A) Theory Examination

Time Table No -01

Date	Time	Paper
28/08/17	10am to 12.30pm	Physiology-I
29/08/17	10am to 12.30pm	Physiology-II
30/08/17	10am to 12.30pm	Anatomy-I
31/08/17	10am to 12.30pm	Anatomy-II
1/09/17	10am to 12.30pm	Biochemistry-I
2/09/17	10am to 12.30pm	Biochemistry-II

B) Practical Exam

Time Table No -2

Date	Time	Department
04/09/17	9.00am onwards	Physiology
05/09/17	9.00am onwards	Anatomy
06/09/17	9.00am onwards	Biochemistry

Prof & HOD
Dept. of Anatomy

HOD
Department of Anatomy
MVP'S DR. Vasantrao Pawar
Medical College, Nashik

Prof & HOD
Dept. of Physiology

HOD
Dept. of Physiology
MVPS Dr. VPMC, Nashik

Prof & HOD
Dept. of Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC.
Adgaon, Nashik-422 003

Circulate & Notice board

Department of Biochemistry
Date: 9/9/16

Department of Physiology
8/9/2016

Maratha Vidhya Prasarak Samaj's
Dr. Vasantrao Pawar Medical College, Adgaon, Nashik-3.
Department of Anatomy,Physiology & Biochemistry

Date 8 .9.2016

Subject: Supplementary Prelimanary Theory & Practical Examination **September – 2016**
Reference: Resolution meeting of board- I Anatomy ,Physiology & Biochemistry Dt.8.9.2016

Time table

1st Year MBBS 2015-16

It is to inform to all the Repeater students that Suplimentary,Preliminary,Examination for 1st MBBS 2015-16 batch (Repeater) will held as given below

A) Theory Examination

Time Table No. -1.

Day	Date	Time	Subject	Lect. Hall - I
Wednesday	21.9.2016	10.30 to1.00	Physiology I	Lect. Hall - I
Thursday	22.9.2016	10.30 to1.00	Physiology II	Lect. Hall - I
Friday	23.9.2016	10.30 to1.00	Anatomy I	Lect. Hall - I
Saturday	24.9.2016	10.30 to1.00	Anatomy II	Lect. Hall - I
Monday	26.9.2016	10.30 to1.00	Biochemistry I	Lect. Hall - I
Tuesday	27.9.2016	10.30 to1.00	Biochemistry II	Lect. Hall - I

Seating Arrangement: Lect.Hall-1

B) Practical Examination

Time Table No.2

Day	Date	Subject	Time	Batch
Wednesday	28.9.2016	Anatomy	9.00 Am to Onwards	All Repeater
Thursday	29.9.2016	Biochemistry	9.00 Am to Onwards	All Repeater
Friday	30.9.2016	Physiology	9.00 Am to Onwards	All Repeater

Instructions to the students:-

2) Disciplines – Mobiles, Pen drive, blue tooth...etc instruments are not allowed
- Any chits, books are not allowed
- Talking not allowed.
- Silence should be maintained

2) Student should submit journals on the day of their practical exam, to the lab in charge for Verification by Examiner & should be take back after Exam.

08.09.16
Prof. &HOD
Anatomy

for
Chairman
Prof. & HOD
Physiology

Prof.& HOD
Biochemistry
Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College Hospital & RC,
Adgaon, Nashik-422 003.

Copy to 1) Dean, MVPS Dr. V.P. Medical College, Nashik.
2) Anatomy,Physiology,Biochemistry Notice Board
3) Boy's & Girls Hostel
4) Library
5) Boys Girls Common Room

HOD
Dept. of Physiology
MVPS Dr. VPMC, Nashik

HOD
Department of Anatomy
M.V.P.S. Dr. Vasantrao Pawar
Medical College, Nashik-3

1103

13/8/15

Maratha Vidya Prasarak Samj's
Dr. Vasantrao Pawar Medical College Adgaon Nashik-3
Department of Anatomy, Physiology & Biochemistry

Date 13.8.2015

Subject: Suplimentary Prelimanary Theory & Practical Examination Auguest -2015
Reference : Resoluation meeting of board –I Anatomy,Physiology& Biochemistry Dt.13.8.2015

Time Table

1st Year MBBS -2014-15

It is to inform to all the Repeter students that Suplimentary Preliminary Examination for 1st MBBS 2014-15 batch (Repeater) will held as given below

A) Theory Examination

Time table No-01

Day	Date	Time	Subject	Lect Hall
Thursday	20.8.2015	10.00 am to 1.00 Pm	Anatomy -I	Lect. hall -I
Friday	21.8.2015	10.00 am to 1.00 Pm	Anatomy -II	Lect. Hall- I
Saturday	22.8.2015	10.00 am to 1.00 Pm	Biochemistry-I	Lect. hall -I
Monday	24.8.2015	10.00 am to 1.00 Pm	Biochemistry -II	Lect. hall -I
Tuesday	25.8.2015	10.00 am to 1.00 Pm	Physiology-I	Lect. hall -I
Wednesday	26.8.2015	10.00 am to 1.00 Pm	Physiology-II	Lect. hall -I

Seating Arrangement : Lect. Hall -I

B) Practical Exam.

Time Table No-02

Day	Date	Subject	Time	Batch
Thursday	27.8.2015	Anatomy	9.00 Am to onwards	All Repeater
Friday	28.8.2015	Biochemistry	9.00 Am to onwards	All Repeater
Saturday	29.8.2015	Physiology	9.00 Am to onwards	All Repeater

Instructions to the students

2) Disciplines – Mobiles, Pen drive, blue tooth ..etc instruments are not allowed
- Any chits , books are not allowed
- Talking not allowed
- Silence should be maintained.

2) Student should submit journals on the day of their practical exam, to the lab in charge for Verification by Examiner & should be take back after Exam.

Prof & HOD
Dept of Anatomy
Department of Anatomy
M.V.P.S. Dr. Vasantrao Pawar
Medical College, Nashik-3.

13.8.15
Chairman
Prof & HOD
Dept of Physiology

14.8.15
Prof & HOD
Dept of Biochemistry
Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC,
Adgaon, Nashik-422 003.

Copy to 1) The Dean, MVPS.Dr.VPMC.Nashik.
2) Anatomy,Physiology,Biochemistry Notice Board
3) Boy's & Girls Hostel
4) Library
5) Boys & Girls Common Room.

Maratha Vidya Prasarak Samaj's
Dr. Vasantrao Pawar Medical College, Adgaon, Nashik-3.
Department of Anatomy/ Physiology and Biochemistry

Date: - 22/07/2014.

IMPORTANT NOTICE

Subject – For the internal Assessment Examinations for 1st MBBS Students in Anatomy, Physiology & Biochemistry

Reference – 1. MUHS/ X-1/UG/5467/2014. dt.08/07/14.
2. University Notification No. 20/2012. dt. 30/05/2012. Batch -2013-14.

It is to notify to all the 1st MBBS Students of Batch – 2013-14 that, the failure students, Absent and those who have not filled MUHS Examination form of Summer 2014, for any reason, It is mandatory to have eligibility granted by MUHS. Hence there will be one internal assessment Examination.

Time Table of Examination is given Bellow –

Theory Examination

Day	Date	Subject	Time
Monday	4/8/14.	Anatomy I	10.30 to1.00
Monday	4/8/14	Anatomy II	02.00 to 4.30
Tuesday	5/8/14	Physiology I	10.30 to1.00
Tuesday	5/8/14	Physiology II	02.00 to 4.30
Wednesday	6/8/14	Biochemistry I	10.30 to1.00
Wednesday	6/8/14	Biochemistry II	02.00 to 4.30

*** Place: - Respective Department**

Practical Examination (9.00AM onwards)

Day	Date	Subject	Batch
Thursday	7/8/14	Anatomy	All Students
Friday	8/8/14	Physiology	All Students
Saturday	9/8/14	Biochemistry	All Students

All Students should sign the internal assessment for all Departments on 9th August 2014.

22/7/14.

Prof. HOD
Anatomy

HOD
Department of Anatomy
M.V.P.S. Dr. Vasantrao Pawar
Medical College, Nashik-3

for 22/7/14

Prof. HOD
Physiology

Professor & HOD
Department of Physiology
NDMVPS MEDICAL COLLEGE,
ADGAON, NASHIK (M.S.)

22.7.14

Prof. HOD
Biochemistry

Professor & Head
Department of Biochemistry
M.V.P.S. Dr.Vasantrao Pawar
Medical College, Hospital & RC.
Adgaon, Nashik-422 003.

Copy Submitted to the Dean, MVPS Dr. V.P. Medical College, Nashik.
Copy forwarded to: - The Chairman Academic Board 2, 3 & 4.
Copy forwarded to: - Time Table Committee. For circulation Notice Board Boys& Girls Hostel, Library,

Vasave Vikram Navsu

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adagon, Nashik-3.

Examination : Pre-Supplementary

Subject : Biochemistry

Section :

PAPER : I

Language of Answer : English

Serial No.

Date : 26 09 2016

P.Roll No. 114 (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet. I also know that no supplement will be provided to me.

Signature of the CANDIDATE

Signature of the JUNIOR SUPERVISOR

seen no complaints

3/10/2016

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1	MCQ			3.0												4.0
2	2.0	2.0	3.0	~~A 5~~ B	NA	2.0	3.0									15.0
3	4.5	NA	5.0													9.5
4																
5																
6																
7																
8																
9																
10																

Marks allotted By Examiner

Name of Teacher Dr. Nemade S.T. Signature :

TOTAL → ~~24.5~~ 28.5

No supplements shall be provided to any candidate in any case

VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWERSHEET

Name of Candidate : Vasave Vikram Navsu
Name of Examination : pre-supplementary.
Class : 1st M.B.B.S Semester :
Subject : Biochemistry Paper : I
Date : 26/09/2016

ROLL NO.

	1	5	1	1	4

0	☐	☐	☐	☐	☐	☐				
1	☐	■	☐	■	■	☐				
2	☐	☐	☐	☐	☐	☐				
3	☐	☐	☐	☐	☐	☐				
4	☐	☐	☐	☐	☐	■				
5	☐	☐	■	☐	☐	☐				
6	☐	☐	☐	☐	☐	☐				
7	☐	☐	☐	☐	☐	☐				
8	☐	☐	☐	☐	☐	☐				
9	☐	☐	☐	☐	☐	☐				

QUESTION :→	1	2	3	4	5	6	7	8	9	10
ANSWER :↓	×	×	×	×	×	×	✓	✓	✓	×
A	■	☐	☐	☐	☐	☐	☐	■	☐	■
B	☐	■	☐	☐	☐	■	☐	☐	■	☐
C	☐	☐	☐	☐	■	☐	■	☐	☐	☐
D	☐	☐	■	■	☐	☐	☐	☐	☐	☐

QUESTION :→	11	12	13	14	15	16	17	18	19	20
ANSWER :↓	✓	×	×	×	✓	✓	✓	×	×	✓
A	☐	■	☐	☐	■	☐	☐	☐	☐	■
B	☐	☐	☐	■	☐	■	☐	■	☐	☐
C	■	☐	■	☐	☐	☐	■	☐	☐	☐
D	☐	☐	☐	☐	☐	☐	☐	☐	■	☐

QUESTION :→	21	22	23	24	25	26	27	28	29	30
ANSWER :↓										
A	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
B	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
C	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐
D	☐	☐	☐	☐	☐	☐	☐	☐	☐	☐

ROLL NO. (In Words)
one, five, one, one four.

QUESTION BOOKLET VERSION :

This is to certify that the entries of Roll No., Question Booklet Version, Question Booklet Sr. No. & Subject have been verified.

CANDIDATE SIGNATURE

INVIGILATOR'S SIGNATURE

Date : 26/09/2016

USE BLUE BALL POINT PEN ONLY....

INSTRUCTIONS

1. FILL THE BLOCKS ☐ USING BLUE BALLPOINT PEN ONLY.
2. FILL ONLY ONE BLOCK FOR EACH QUESTION AS SHOWN BELOW.

Wrong			Correct
✓	☒	▬	■

3. FILL ONLY BLOCK PROVIDED DO NOT MAKE ANY STRAY MARKS NO THE ANSWER SHEET.
4. ROGH WORK MUST NOT BE DONE ON THIS ANSWER SHEET.
5. USE FREE SPACE IN THE QUESTION BOOKLET PROVIDED.

TOTAL MARKS :-

4/10

No supplements shall be provided to any candidate in any case

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adagon, Nashik-3.

Examination : pre-suppli

Subject : Biochemistry

Section :

PAPER : II

Language of Answer : English

Serial No. 15479

Date : 27 09 2016

P.Roll No. 114 (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet. I also know that no supplement will be provided to me.

Signature of the CANDIDATE

Signature of the JUNIOR SUPERVISOR

seen no complains

3/10/2016

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1	MCQs															06.0
2	NA	03	NA	03	02	NA	02									10.0
3	4.5	6.5	06													10.5
4																
5																
6																
7																
8																
9																
10																

Marks allotted By Examiner

Name of Teacher Dr. V.P. Suryakar Signature :

TOTAL → 26.50

R. VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWERSHEET

Name of Candidate : Vaseare Vikram Nawa

Name of Examination : Pre-suppli

Class : 1st M.B.B.S Semester :

Subject : Biochemistry Paper : II

Date : 29/09/2016

ROLL NO.

	1	5	1	1	4
0					
1		■		■	■
2					
3					
4					■
5			■		
6					
7					
8					
9					

ROLL NO. (In Words)

One, five, one, one, four.

QUESTION BOOKLET VERSION :

This is to certify that the entries of Roll No., Question Booklet Version, Question Booklet Sr. No. & Subject have been verified.

CANDIDATE SIGNATURE

INVIGILATOR'S SIGNATURE

Date : 29 / 9 / 2016

QUESTION :→	1	2	3	4	5	6	7	8	9	10
ANSWER :↓	X	✓	X	X	X	✓	✓	✓	✓	X
A	■			■			■	■		
B						■			■	
C		■	■		■					■
D										

QUESTION :→	11	12	13	14	15	16	17	18	19	20
ANSWER :↓	✓	✓	✓	✓	✓	✓	X	✓	X	X
A	■					■		■		
B		■			■					
C				■			■			■
D			■						■	

QUESTION :→	21	22	23	24	25	26	27	28	29	30
ANSWER :↓										
A										
B										
C										
D										

USE BLUE BALL POINT PEN ONLY....

INSTRUCTIONS

1. FILL THE BLOCKS ☐ USING BLUE BALLPOINT PEN ONLY.
2. FILL ONLY ONE BLOCK FOR EACH QUESTION AS SHOWN BELOW.

Wrong	Correct

3. FILL ONLY BLOCK PROVIDED DO NOT MAKE ANY STRAY MARKS NO THE ANSWER SHEET.
4. ROGH WORK MUST NOT BE DONE ON THIS ANSWER SHEET.
5. USE FREE SPACE IN THE QUESTION BOOKLET PROVIDED.

TOTAL MARKS :- 6/10

me: Vasave Vikram Navsu

ll No.: 114 Date: 29/09/2016

culty:

mination: suppli- practical

dent's Sign.:

Chanda 29/9/16 Q-B 29/9/16

Estimate Blood Glucose level in the given sample	20
Perform any five test on precipitation reactions of proteins	15
Spot	05

substrate beta-D-glucose to gluconic acid and hydrogen peroxide is liberated. - peroxidase enzyme acts on hydrogen peroxide to liberate nascent oxygen (O) Nascent oxygen then couples with 4-amino antipyrine and phenol to form red quinoneimine dye. The intensity of the colour is directly proportional to the concentration of glucose present in blood. The intensity of colour is measured colorimetrically at 530 nm.

Observation

Test	0.40 O.D
standard (S)	0.39
Blank (B)	0.02

Scanned with CamScanner

Name: Nikita G. Wakode

Roll No.: 116 (R) Date: 15/09/18

Faculty: Biochemistry.

Examination: Preliminary

15/9/18

Table Nos: - 1,

Q.A	Estimate Blood Glucose level in the given sample.	20
Q.B	Perform any five tests on Carbohydrate Solution	15
Q.C	Spots	05

Method - Glucose oxidase peroxidase method. (GOD - POD)

Other method -

Enzymatic method - 1) Hexokinase

2) Glucose oxidase peroxidase method.

Chemical -

- folin Wu
- Nelson somogyi

Principle - The glucose in given sample, is reacted, with glucose oxidase method and form glucoronic acid.

$$\text{Glucose} \xrightarrow{GOD} \text{glucoronic acid} + H_2O_2 \uparrow$$

$$H_2O_2 \xrightarrow{POD} H_2O + [O]$$

$$[O] + 4\text{ Antipyrine} + \text{phenol} \longrightarrow \text{quinone compound}$$

The H_2O_2 by the enzyme peroxidase converted into H_2O and oxygen. Then nascent oxygen react with 4 antipyrine and phenol and gives quinone compound a pink (pink) colour compound.

~~Clin~~

Observation

Unknown	0.38
standard	0.36
Blank	0.02

Calculation -

$$\text{Blood glucose} = \frac{OD_u - OD_B}{OD_S - OD_B} \times \text{Conc. stand} \times \text{Vol}^{m} \text{ of std} \times \frac{100}{\text{Vol}^{m} \text{ of blood}}$$

$$= \frac{0.38 - 0.02}{0.36 - 0.02} \times \frac{0.01}{1} \times \frac{100}{0.01}$$

$$= \frac{0.36}{0.34} \times 10^2 \times 100 = \frac{18}{17} \times 10^2 \times 100$$

$$= 1.53 \times 10^2$$

$$= 153 \text{ mg \%}$$

Normal range.

Normal blood glucose level is 80 - 120 mg %.

fasting - 70 - 110 mg %.
Post-Prandial - 80 - 140 mg %
Random - 80 - 120 mg %

Clinical significance -

1) Hyperglycemia - Blood glucose level increases.
it is found in - severe diabetes mellitus.
- Hyperthyroidism
- Hyperadrenalism
- Pancreatitis.

Hypoglycemia - Blood glucose level decreases.
it is seen in :- Hypothyroidism
- Hypoadrenalism
- Increase in insulin secretion.

⊙ Result

The glucose level of given sample is 153 mg%
it is slightly more than normal

Q. B. Test on carbohydrate solution.

1] Molish test.

Test	Observation	Inference
3ml o.s. + 1drop α-naptho) + 1ml conc. ~~HNO_3~~ H_2SO_4.	Purple ring at junction between two liquids.	it is group test for carbohydrate. Given by all carbohydrate.

2] Benedicts test

3] Barfoed test

4] Fehling test

5] Selivanff test.

4] Fehling test

Fehling solution = Fehlings A + Fehling B

Test	Observation	Inference.
Take 2ml OS + 2ml fehling solution mix + boil.	brick red colour.	fehling solution is reducing agent. Direct reduction take place. Given by reducing sugar

3] Baetoed test.
Baetoed reagent used in this reaction.

Test	Observation	Inference.
3ml Original solution + 3ml baetoed reagent. Mix + boil	scandy red PPT at bottom	stepwise reduction occure in reactions due to this control reduction take place. Given by all monosaccharide.

- Baetoed reagent are reduce only monosaccharide hence it is used to distinguish monosaccharide and disaccharide.

2] Benedict Test.	Observation	Inference.
5 ml BR + 1ml OS heat and mix. + boil.	Brick red colour	All reducing sugar give this test positive.

-it is bed side test to detect glucose in urine.

5] Seliwonoff test

Test	Observation	Inference.
1ml OS + 3ml seliwonoff reagent heat + boil	No cherry red colour	distinguish betwn ketose and aldose Given sample carbohydrate is glucose.

- Given carbohydrate is glucose i.e, Aldolase.

Jathale 7/13

Name: Nikita G. Wakode

Roll No.: 116 (B) Date: 15/09/18

Faculty: Biochemistry

Examination: Preliminary

Student's Sign.:

5/5

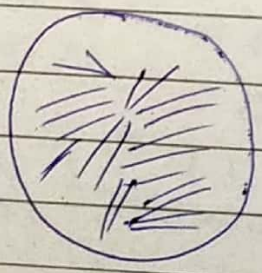
1) To detect the presence of bile pigment in sample urine

2) Esbatch reagent used for precipitation reactions of protein., to detect 24 hour urinary protein.

3) Colorimetry - it is use to measure colour intensity of sample.

4) Non-diabetogenic graph. (Normal GGT)
Blood glucose level is normal finally.

5) Glucosazone - Niddle shape



DR. VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWER SHEET

Name of Candidate : Nikita G. Nakode
Name of Examination : Preliminary Exam
Class : MBBS I Semester : II
Subject : Biochemistry Paper : I
Date : 10/09/18

ROLL NO.

				1	1	6
0						
1				■	■	
2						
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QUESTION :→	1	2	3	4	5	6	7	8	9	10
ANSWER :↓										
A	☒	■	☒	■	□	□	□	■	■	□
B	□	□	□	□	■	■	□	□	□	■
C	□	□	□	□	□	□	■	□	□	□
D	□	□	□	□	□	□	□	□	□	□

QUESTION :→	11	12	13	14	15	16	17	18	19	20
ANSWER :↓										
A	□	□	□	□	□	□	■	■	□	■
B	□	■	■	☒	□	□	□	□	□	□
C	■	□	□	□	□	■	□	□	■	□
D	□	□	□	□	■	□	□	□	□	□

QUESTION :→	21	22	23	24	25	26	27	28	29	30
ANSWER :↓										
A	□	□	□	□	□	□	□	□	□	□
B	□	□	□	□	□	□	□	□	□	□
C	□	□	□	□	□	□	□	□	□	□
D	□	□	□	□	□	□	□	□	□	□

TOTAL MARKS :-

17.5 / 10

ROLL NO. (In Words)
One hundred sixteen.

QUESTION BOOKLET VERSION :

This is to certify that the entries of Roll No., Question Booklet Version, Question Booklet Sr. No. & Subject have been verified.

CANDIDATE SIGNATURE

INVIGILATOR'S SIGNATURE

Date : 10/09/18.

USE BLUE BALL POINT PEN ONLY....

INSTRUCTIONS

1. FILL THE BLOCKS □ USING BLUE BALLPOINT PEN ONLY.
2. FILL ONLY ONE BLOCK FOR EACH QUESTION AS SHOWN BELOW.

Wrong			Correct
✓	☒	▬	■

3. FILL ONLY BLOCK PROVIDED. DO NOT MAKE ANY STRAY MARKS ON THE ANSWER SHEET.
4. ROUGH WORK MUST NOT BE DONE ON THIS ANSWER SHEET.
5. USE FREE SPACE IN THE QUESTION BOOKLET PROVIDED.

Wakode Nikita

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adgaon, Nashik-3.

Examination : Preliminary Exam

Subject : Biochemistry

Section : PAPER :

Language of Answer : English

Serial No. 6364

Date : 10 09 2018

P.Roll No. 11G (R) (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet. I also know that no supplement will be provided to me.

Signature of the CANDIDATE

Signature of the JUNIOR SUPERVISOR

15/09/18

Seen, Verified
No Complaints

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1	MCQ															7.5
2	3.0	2.0	2.5	3.0	3.0	1.5	NA									15.0
3	6.5	NA	4.5													11.0
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10																

Marks allotted By Examiner

Name of Teacher Dr. Nemade S.T Signature : Thanda

TOTAL → 33.50

No supplements shall be provided to any candidate in any case

a] Gout

1) Gout is metabolic disorder of purine metabolism.

2) Hyperuricemia is condition in which uric acid level increases in serum.

3) Normal range of uric acid is 3-7 ug/dL

4) At physiological pH uric acid is in more soluble form i.e. sodium urate.

5) In severe hyperurecimia, the crystals of uric acid i.e. sodium urate are form.

6) Crystals of sodium urate get deposited in soft tissue mainly joints.

7) This accumulation of uric acid crystals in joints called Tophi.

8) These leads to painful joints. i.e. arthritis.

Gout are of two types.

1) Primary gout
11) secondary gout

1) Primary gout -

- Primary gout develops due to some enzyme deficiency.

Deficient enzymes are -

1) PRPP synthase
11) PRPP Glutamylamidotransferase.
111) HGPRT enzyme.

① PRPP synthase –

- This enzyme regulate by negati feed back mechanism.
- Due to formation various aspirant enzyme which do not regulated by feed back mechanism., leads to over production of purines and leads to gout

② PRPP Glutamylamidotransferase –

- Due to lack of feed back regulation of this enzyme
- over synthesis of purines occure.

③ HGPRT –

- This a purine salvage pathway enzyme
- Deficiency of this enzyme causes Lesh Nyhan syndrome.
- This causes synthesis of purine nucleo and less utilization of it.

Secondary gout

secondary gout is produce due to various diseases which associate with hyperuricemia.

c] Diagnostic significance of enzyme —

- Certain enzymes have important diagnostic significance.

Enzymes		Diseases
Amylase	→	Pancreatitis.
Alkaline Aspartate (SGOT)	→	Heart diseases
Alkaline phosphate (SGPT)	→	Liver diseases.
LDH	→	Myocardial interaction.
Homogentisate	→	Alkaptonuria.
Phenylalanine hydroxylase	→	Phenylketone uria.

- By measuring serum level of enzymes we can detect the disease in early stages.

- Level of enzyme other than normal level in serum leads to detect related to that enzyme.

d] Immunoglobulins – (Antibodies)

- Immunoglobulins are special type of protein take part in ~~dens~~ defense system of body.

- structure of immunoglobulin

- Each immunoglobulin molecule have two Heavy chains and two light chains.
- They are held together by disulfide linkage. (S=S)
- Immunoglobulin is Y-shape molecule.
- Each chain have two region.
  (i) Constant
  (ii) Variable.

Variable region of each chain is used for binding with antigen.

Variable region is specific for specific antigen.

Immunoglobulin and γ-globulins are not synonymous.

Immunoglobulin is functional term and γ-globulin is a

Immunoglobulin have five types like
- Ig G
- Ig M
- Ig A
- Ig D etc.
- Ig E

① Ig G –
- Ig G is a single polypeptide chain
- It is the only immunoglobulin which can cross placenta.
- it is predominantly occurs upto 70 - 80%.

② Ig M –
- Ig M is largest immunoglobulin
- it is made up of pentameric polypeptide chains.
- This can not transfer transport placenta & blood vessels.

③ Ig A –
- Ig A this immunoglobulin is largly present in colostrum.
- It provide passive immunity to new born

e] Genetic code.

Genetic code very important for transcription, translation and ultimally protein synthesis.

Genetic code is codon of three ~~amino acid~~ nucleotide which determine amino acid.

Characteristic of Genetic code

(I) Specificity -
- Genetic code is specific for specific amino acid.
- They may have some exception

(II) Universal. -
- Genetic code is preserve during process of evolution.
- Hence, for specific amino acid specific ~~amino acid~~ genetic code is found all over.
- They may have some exception

(III) Degeneration -

- Gentic code is degenerative.
- due to one codon can code for more than one amino acid.

(IV) Unambigous.

- Genetic code is unambigous due to one amino acid ~~have~~ can code by more than one codon.
- This due to wobbal - hypothesis.

(V) Continuous -

- Genetic code is continuous sequence of nucleotide.
- This sequence is without any punctuation mark like quama, full stop, (or) any gap.

- Initiating codon → AUG predominantly sometime GUG.
- stop codon - UAA, UGA, UAG

b) RNA -

RNA i.e. Ribonucleic acid.

structure of RNA

1) Pentose → Ribose sugar.
   - 5 carbon atom compound

function of RNA

- RNA is important molecule for transcription & translation

- During transcription information from DNA to mRNA is transported.
- from mRNA with amino acid various protein synthesis occur.

RNA have various types

1) mRNA -
   it is massenger RNA which transport information from DNA from nucleus to cytosol.
2) t-RNA -
   Transfer RNA.
   - it transport amino acid

3) sRNA
- soluble RNA

4) hn RNA
it is heteronuclear RNA
it is converted to mRNA after modifica

5) r-RNA
- Ribosomal RNA
- it take part in protein synthesis.

1) Clinical condition - sickle cell anemia.

2) Molecular detect -

- In sickle cell anemia, mutation i.e. point mutation occur in globin chain.
- At the 6th position of globin chain, valine get inserted
- Due to this mutation, shape of RBC is changed from biconcave to sickle shape.
- In low O_2 tension person with sickle cell anemia have problem in ~~breathing~~ of hypoxia.
- Hypoxia is due to low oxygen supply to body.
-

Biochemical basis of sickling of RBC -

- Point mutation of globin chain.
- in sickle cell, α-globin chains are produce normal

β- globin chains have abnormality.

- At 6th position valline is inserted in β- globin chain. insted of glutathione.

Other lab investigation -

- Check RBC shape during hypoxic condition.
- is there, malerial parasite develop in that person or not because sickle cell anemia is registant to maleria.

Vitamin –

Vitamins are defined as vital amino acid.

It is very essential for normal growth of body and body function.

It require in very less amount for normal body function

Classify vitamins –

Vitamins are classified as

1) Water soluble – vitamin B and B complex and vitamin C.

2) fat soluble – A, a, d, E, K. vitamins.

Vitamin A –

- Retinol, Retinoic acid are considered as vitamers of vitamin A.
- Vitamin A is fat soluble vitamin
- β- carotene found in plants

Biochemical function -

- Vitamin A is important for vision, colour identification, growth, normal reproductive function.
- Vision -
- Vitamin play ~~impt~~ important role in normal vision through Wald's visual cycle
- Growth -
Vitamin A is play important role in tissue growth and tissue differentiation.
- Vitamin A is important for normal epithelium
- Vitamin A is important for reproduction
- Vitamin A is useful for various biochemical function
- β- carotene is natural antioxidant

Deficiency manifestations -

- Due to the deficiency of vitamin A there is defect in vision.
- Night blindness is very early symption of vitamin A deficiency.
- In night blindness, period of dark adaptation is increases.
- Vitamin A deficiency leads to keratinized skin, exfoliating skin, scalp.
- Vitamin A deficiency may lead to sterility.
- Vitamin A deficiency affect body growth.

RDA -

- For normal adult recommended dietary allowance of vitamin A is 600 IU
- Requirement increases during pregnancy in women.

Sources -

- Animal source - meat, milk, egg yolk, fish, fish liver oil.

Plant source - β-carotene.

3) Enzyme -

Defination - Enzyme is biocatalyst they increases the speed of reaction without getting converted or utilise.

Classification -

Hydrolyases - Hydrolyse the substeate.
Ex. pepsin, urease.

Lyases → cleave the molecules
Ex. - fumarase

Ligases - join together the two molecule
Ex. ~~ligases~~ succinate thiokanase.

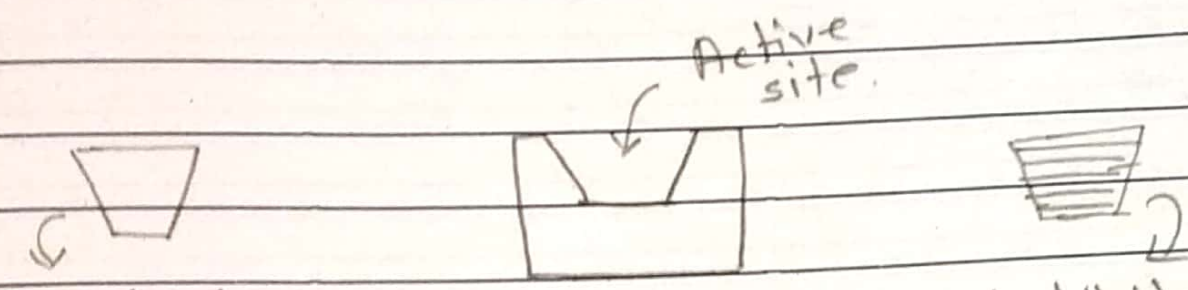
Transferases - Transfer the active group
Ex. Amino transferases

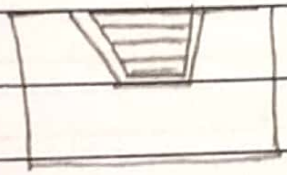
~~Oxidases~~ → ~~Oxidises the substeate by~~
Oxidoreductase → Ex. Cytocrome oxidase.
~~Reductases~~ → ~~Reduces the substeate.~~

- Enzyme alters ~~enzyme~~ activation energy of reaction. i.e. lowers the activation energy.

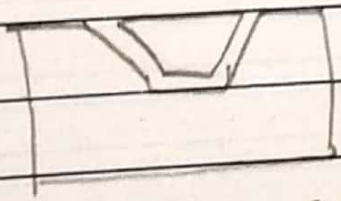
Competitive inhibition of enzyme →

- In competitive inhibition, inhibitor structure ressembles with substrate.
- Due to structural similarity with substrate, then inhibitor can bind to enzyme.
- If inhibitor bind to enzyme at active site of enzyme.
- ~~Shape of enzyme active site get chan~~
- No space for substrate to bind the enzyme.
- Due to this, substrate is inhibited by inhibitor to bind the enzyme and inhibit their action.
- Competitive inhibition can be overcome by increasing substrate concentration.
- Competitive inhibition may be reversible & irreversible.





enzyme - inhibitor complex
i.e. substeate action inhibited



enzyme - substeate complex.
i.e. substeaction action continues

DR. VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWER SHEET

Name of Candidate : Nikita G. Wakode

Name of Examination : Preliminary

Class : MBBS I Semester :

Subject : Biochemistry Paper : II

Date : 11/09/2018

ROLL NO. 116

ROLL NO. (In Words) One hundred sixteen

QUESTION BOOKLET VERSION :

QUESTION :→	1	2	3	4	5	6	7	8	9	10
ANSWER :↓										
A	■								■	
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C										■
D							■	■		

QUESTION :→	11	12	13	14	15	16	17	18	19	20
ANSWER :↓										
A	■	■						■	■	
B					■	■	■			■
C			■							
D				■						

QUESTION :→	21	22	23	24	25	26	27	28	29	30
ANSWER :↓										
A										
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C										
D										

This is to certify that the entries of Roll No., Question Booklet Version, Question Booklet Sr. No. & Subject have been verified.

CANDIDATE SIGNATURE

INVIGILATOR'S SIGNATURE

Date : 11/09/18

USE BLUE BALL POINT PEN ONLY....

INSTRUCTIONS

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2. FILL ONLY ONE BLOCK FOR EACH QUESTION AS SHOWN BELOW.

Wrong | Correct

3. FILL ONLY BLOCK PROVIDED. DO NOT MAKE ANY STRAY MARKS ON THE ANSWER SHEET.
4. ROUGH WORK MUST NOT BE DONE ON THIS ANSWER SHEET.
5. USE FREE SPACE IN THE QUESTION BOOKLET PROVIDED.

TOTAL MARKS :-

5.5 / 10

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adgaon, Nashik-3.

Examination : Preliminary Exam

Serial No. 6363

Subject : Biochemistry

Section :

PAPER : II

Language of Answer : English

Date : 11 09 2018

P.Roll No. 116 (R) (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet. I also know that no supplement will be provided to me.

Signature of the CANDIDATE

Signature of the JUNIOR SUPERVISOR

15/09/18

Seen, Verified
No Complaints

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1						ex										5.5
2	3	2	2	1.5	2.5	1	3									14
3	NA	3.5	4													7.5
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9																
10																

Marks allotted By Examiner

Name of Teacher Dr Ghuge G.D

Signature :

TOTAL → 27.00

No supplements shall be provided to any candidate in any case

2.

7] Significance of HMP shunt →

1) HMP shunt ~~is~~ pathway is another pathway for glycolysis.

2) HMP shunt is also called pentose phosphate pathway

3) In HMP shunt pathway ATPs are not formed.

Significance →

- HMP shunt produces two molecules namely pentoses and ~~NADPH~~. NADPH

- Pentose formed in HMP shunt is mainly ribose-5 phosphate.

- Ribose-5 phosphate further use for synthesis of nucleotides. like, NAD, FAD, Co A etc.

- These nucleotides are act as coenzymes for enzymes.

NADPH

- It act as reducing agent

- NADPH act as co-enzyme for many enzymes.

Phagocytosis of antigen bodies is done by with help of NADPH

NADPH is useful for energy production in ETC chain.

NADPH used in various biochemical reactions.

NADPH required for biosynthesis of fatty acids and steroids.

ATP is neither used nor produced in HMP shunt pathway.

6) Diagnosis -

- Test to detect ~~ureine in~~ gluose in ureine →
  - Benedicts test are used to detect ureine ~~in~~ gluose.

2) Clearance test -

Clearance test is ditined as removal of substance totally from serum ~~pee~~ in ~~minute~~. ~~1 min pee ml~~. ml pee 1 min.

$$\text{Clearance} = \frac{UV}{P}$$

- Creatine clearance -

- Creatine clearance is process in which removal of creatine from given blood sample ml pee min.

- Creatine clearance is used for accessement of livee function

- Amount of creatine ~~se~~ cleared from 1ml sample in 1 min. is clearance test.

- this test is useful for livee diseases.

4) Electrophoresis →

Electrophoresis is the process by which the substances having charges are separated based on their charge.

Type of electrophoresis
- Gel electrophoresis
- Paper electrophoresis
- electrode electrophoresis.

In serum, various biological components contain ionizable compound.

By using electrophoresis we can separate them from serum.

Components of electrophoretic tank -
- Electrophoresis tank
- electrodes - cathode, anode
- Gel
- electric wire.
- separation plate between two electrodes.

In electrophoresis process, the speed of ions to deposition on electrode or to dissolve into solution is dependent on

- concentration of ions in solution,
- Temperature is also a influcin factor in electrophoresis.

5] Tumor markers. –

Tumor markers support the diagnosing the cancer, besides this it also help in treatment of disease.

Tumor markers are special protein produce by affected cell in body.

With the help of tumor markers, early diagnosis of disease is done.

Some time tumor markers are not specific. for specific cancers.

- it used to detect presence of cancer.

① Carcino embryonic antigen (CEA)
- Complex glycoprotein normally produce in embryonic tissue, liver, gut.

- Presence CEA is detected in several cancers
- serum CEA also detected in several other disorders. like alcoholic cirrhosis, emphysema.

Alpha-fetoprotein -

- it is chemically glycoprotein synthesized from yolk sac in fetal life.

- elevation AFP in serum mainly indicate the cancers of liver, & germ cells of testis.

3] Radioisotopes.

- Radioisotopes are isotopes which are same molecular weight but different atomic number.
- Ex. radioisotopes of iodine are I^{138} I^{132}

Radioisotopes ~~are~~ have significance in diagnosing of various diseases.

These isotopes are used various dease treatment Various tretment of cancer.

Radioisotopes are also used in detection of heart blockages, any blockages in vessels.

The radioisotope of same molecule is different in their action.

Phosphorus, iodine, nitrogen, carbon have their radioisotope.

Radioisotope are used largely in various biological reaction for detection of substrate and product reaction.

- Radioisotopes are used in nuclear reactions also

- With the help of radioisotopes we can diagnose the disease in early stage and can prevent severe stages of deasease

1) Phospholipid –

Phospholipids are important compounds which are made up of lipid in association of phorous besides amino group, & etc.

Phospholipids are used in synthesis of various biological products in body.

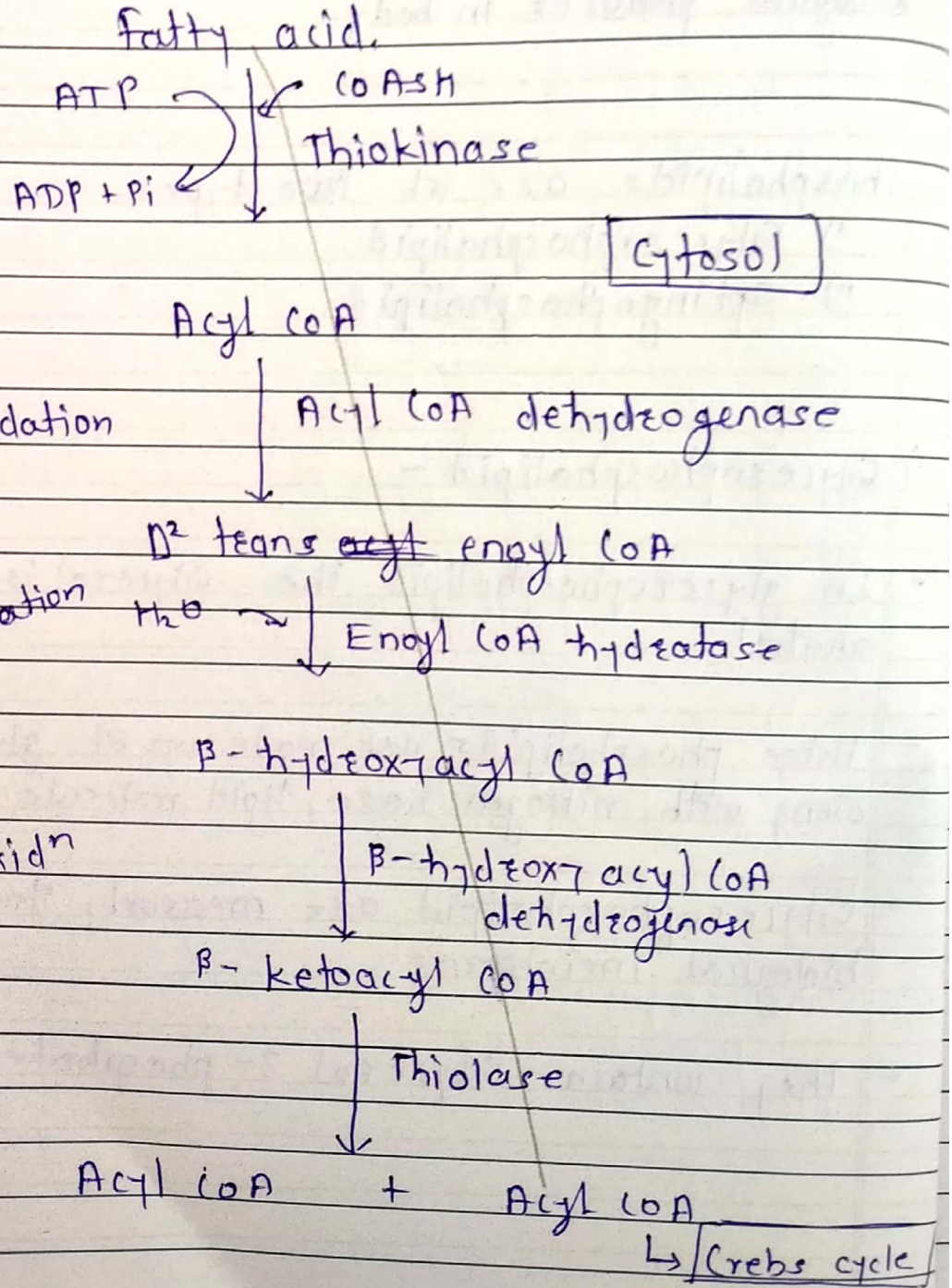
Phospholipids are of two types.
1) Glycerophospholipid
11) Sphingophospholipid.

1) Glycerophospholipid –

- In glycerophospholipid the Glycerol is the alcohol.
- These phospholipids are made up of glycerol along with nitrogen base, lipid molecule + ... etc.
- Glycerophospholipid are measurably found in biological membranes.
- They contain Glycerol 3- phosphate

Energetics of β-oxidation.

β-oxidation of fatty acid generates large quantity of ATPs.



β-oxidation		ATP
7 $FADH_2$	→	10.5
7 NADH	→	17.5

1) Phosphatidic acid - simplest form of phospholipid.
- it is intermediate betwn synthesis of triacyl-glycerol from phospholipid.

2) lecithin - Most abundant phospholipid.
- lecithin is phosphatidic acid with choline as base.
- lecithin is storage form of bodies choline.
- Dipalmatoyl lecithin act as surfactant of lung.

3) Cephalin -
- in cephalin ethanolamine is nitrogen base
- Cephalin is differ from lecithin in only base.

4) Phosphatidyl serine → Amino acid serine is present in this phospholipid.

2) spingophospholipid

- spingomyelins
- spingomyelin is alcohol in spingophospholipid. it is amino alcohol.
- they do not contain glycerol at all
- Ceramide act as second messenges by regulating programmed cell death.

functions of phospholipid -

1) In association of protein phospholipid form structural components of membrane
2) it is important for synthesis of lipoprotein, thus participate in transport of lipids.
3) Phospholipid participate in reverse cholesterol transport

3.

2] Disorders of β-oxidation –

SIDs syndrome –

- sudden infant death syndrome is syndrome in which sudden death of ~~heat~~ healthy infants occure.

- SIDs syndrome is mainly cause by deficiency of medium acyl chain in infant.

- Due to lack of medium acyl chain, β-oxidation of fatty acids is affected.

- After feeding to newborn glucose level in their body is more but after sometime glucose level and glucose metabolism slow down.

- Hence, simultaneously, oxidation fatty acid is also increase to provide energy demand of body.

- Due to deficiency of medium acyl chain β-oxidation of fatty acid is affected and energy level decrease

- Energy level to brain decreases and death may occure.

fatty acids

ATP → ADP + Pi ← Thiokinase

Acyl CoA

(cytosol)

Acyl CoA

oxidation | Acyl CoA dehydrogenase

Δ^2 transenoyl CoA (Mitocondria)

Hydration H_2O → Enoyl CoA hydratase

β-hydroxy acyl CoA

oxidation | β-hydroxy acyl CoA dehydrogenase

β-ketoacyl CoA

Thiolase

Acyl CoA + Acyl CoA

crebs cycle

Energetics of β-oxidation

7 $FADH_2$ 10.5 ATP

7 NADH 17.5 ATP.

3.

Metabolic acidosis -

- Metabolic acidosis is ~~an~~ metabolic disorder.
- Measure cause of metabolic acidosis is decrease in bicarbonate ion in blood.
- decrease in bicarbonate ion is due to decrease in their synthesis or utilize in buffering action with H^+ ion.
- Metabolic acidosis, pH of body fluid decreases.
- Metabolic acidosis may be seen in severe diabetes mellitus.
- Cause of metabolic acidosis is excessive production of organic acids which combine with $NaHCO_3^-$ and deplet alkali reserve.

Anion gap and metabolic acidosis -
increased production and accumulation of organic acid causes elevation in anion gap.

Metabolic acidosis usually compensated by Hyperventilation of lung.

Mentenance of blood pH

1) Blood buffers
2) Respiratory mechanism
3) Renal mechanism.

1) Blood buffer -

Buffer defined as solution of a weak acid and its salt with steong base

- Buffer resist change in pH

Bicarbonate buffer ~~system~~ -

- sodium bicarbonate & carbonic acid are most predominant buffer system.

2) Respiratory mechanism

- it provide rapid mechanism ~~of~~ for maintance of acid - base balance.

- regulate concentration of carbonic acid in blood.

3) Renal mechanism

Renal mechanism is peermanant solution for regulation of blood pH.

kidney play important role in acid-base balance.

Urine pH is normally lower than ~~bo~~ blood pH, it indicate that the kidney aciditie the urine in kidney.

- kidney, the renal tubule excrete H^+ ion in urine, helps in lowering H^+ ion conc.

Yash Patil

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adgaon, Nashik-3.

Examination : prylcum supply exam

Serial No. 9627

Subject : Biochemistry

Section : A, B, C PAPER : II

Language of Answer : english

Date : 11 09 2019 P.Roll No. (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet..I also know that no supplement will be provided to me.

Signature of the CANDIDATE

Signature of the JUNIOR SUPERVISOR

No supplements shall be provided to any candidate in any case

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1																6
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Marks allotted By Examiner

Name of Teacher Dr. Ghuge Signature :

TOTAL → 28.00

DR. VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWER SHEET

Name of Candidate : Pal's Yash

Name of Examination : _____

Class : I MBBS Semester : _____

Subject : Biochemistry Paper : _____

Date : 11/9/19

ROLL NO.

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ANSWER :↓	✗	✓	✗	✓	✓	✗	✓	✓	✗	✓
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ANSWER :↓	✓	✗	✗	✓	✓	✗	✗	✓	✓	✓
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MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre
Vasantdada Nagar, Adgaon, Nashik-3.

Examination : Preliminary exam
Subject : Biochemistry
Section : A, B, C PAPER : I
Language of Answer : english

Serial No. 9629

Date : 10 09 2019

P.Roll No. (in figure)

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Signature of the JUNIOR SUPERVISOR

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1	MCQ															3.0
2	3.0	2.0	3.5	3.0	2.0	3.0	NA									15.5
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Marks allotted By Examiner

Name of Teacher Dr. Nemade S.T. Signature :

TOTAL → 24.00

DR. VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWER SHEET

Name of Candidate : patil yash Jayawant

Name of Examination : Prelim exam

Class : _____ Semester : _____

Subject : Biochemistry Paper : I

Date : 14/9/19

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ll No. _____ Date : 14/9/19

uity I st MBBS

m: Practical Exam Biochemistry

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14.9.19

4

.A **Estimate serum uric acid content in the given sample. Express your result in mg %.** **20**

.B **Find out abnormal constituents in the given urine sample & write urine report** **15**

.C **Spots** **05**

timate serum uric uric acid content in the ven sample express your result in mg %.

um uric is easy for the blood
n level content it modified the
d

used a time endpoint method to measure the concentration of uric acid present in serum or urine. Uric acid The change in absorbance is directly proportional to the concentration of uric acid in sample

M.V.P.S. Dr. Vasantrao Pawar Medical College

Yash Patil.

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adgaon, Nashik-3.

Examination : I ST MBBS

Serial No. 6362

Subject : Biochemistry

Section :

PAPER : I

Language of Answer : English

Date : 10 09 2018

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Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
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Marks allotted By Examiner

Name of Teacher Dr Nemade S.S Signature : Shande

TOTAL → 6.00

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M.V.P.S. Dr. Vasantrao Pawar Medical College

VASANTRAO PAWAR MEDICAL COLLEGE, HOSPITAL & RESEARCH CENTRE, NASHIK.

MCQ ANSWER SHEET

ne of Candidate : patil yash

ne of Examination : 1st MBBS

ss : _____ Semester : _____

ject : Biochemistry Paper : I

e : 10/9/18

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Bio Chem I

SECTION A

MCQ

1. B
2. B
3. C
4.
5. C
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7. A
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10. A
11. C
12. B.
13. C.
14.
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16. A
17.
18. A
19.
20. B.

SECTION-B

(2). Function of Plasma Proteins.

→ The Plasma Proteins are:
(a) Albumin
(b) Globulin
(c) Fibrinogen.

⇒ Functions of Albumin:

→ Albumin's primary function is the maintainance of Colloidal Osmotic Pressure. Albumin makes the Biggest contribution to the Plasma Oncotic Pressure.

→ Second Important function of Albumin is to Bind and Transport metabolites which are poorly soluble in water such as
- Fatty Acids
- Bilirubin.
- Inorganic constituent of Plasma like Calcium.
- Certain Steroid Hormones.
- Drugs.

* Nutritive function:- Degradation of Albumin provides essential amino acid during Malnutrition.

Fibrinogen (Blood Clotting Factor I)

It is a glycoprotein and constitutes about 4% of total plasma Proteins. It is synthesized in liver and secreted in Blood where it is involved in Blood Coagulation.

→ During Blood Coagulation the fibrinogen is converted to fibrin which polymerized to form fibrin clot.

→ Plasma level of fibrinogen decreases in severe Hepatic Disease.

Globulins

Globulins are group of Proteins in Blood they are made in liver By Immune system. Globulins Play Important Role in liver function, Blood clotting and fighting Infection.

Types of Globulins

α1, α2, Beta Globulin

Alpha and Beta Globulins are transport Protein, serve as globulin which other substances are formed and Perform other Diverse functions.

Gamma Globulin Have a vital Role in Natural and Acquired immunity to Infection.

(2) What are Isoenzymes? Explain with suitable examples.

→ Isoenzymes or Isozymes are multiple forms (Isomers) of the same enzyme that catalyze the same Biochemical Reaction. Isoenzymes different and chemical and physical properties like electrophoretic & kinetic properties.

for Eg:-

1) Lactate Dehydrogenase (LDH)
2) Creatine kinase

Lactate Dehydrogenase. (LDH)

Lactate Dehydrogenase is a tetrameric enzyme that catalyses the oxidation of L-lactate to Pyruvate

LDH has five Isoenzyme

LDH 1
LDH 2
LDH 3
LDH 4
LDH 5.

Clinical Application of LDH

The LDH isenzyme analyses

-) significant elevation of LDH1 and LDH2 (LDH1 > LDH2) occurs within 24 to 48 hours after Myocardial Infarction.
-) Predominant Infarction of LDH, and LDH3 occur in Leukemia.
-) Elevation of LDH3 occur after Damage to the liver or skeletal muscle.

Creatine kinase (CK).

Creatine kinase are Isoenzymes or dimers that are made up of two types of Polypeptide chains, which may be Either.
- CK1 (BB) - It is Present in the Brain
- CK2 (MB) - Cardiac tissue is the only tissue which has the mixed (MB, CK2 isoenzyme).
- CK3 (MM) - It is present in Skeletal Muscle.

Clinical Application.

-) CK1 may be elevated in Neonates.
-) Increased level of CK2 in Blood is characteristic Damage of Heart from Myocard. Infarction. Because cardiac tissue is only tissue which has the mixed MB (CK2) isoenzyme

(3) Balanced Diet.

A Balanced diet is defined as one which contains a variety of food in such quantities and proportions that the need for energy, amino acids, vitamins, minerals, fats, carbohydrates and other nutrients is adequately met for maintaining health, vitality and general well being. and makes a small provision for extra Nutrients to withstand short duration of illness.

Balanced diet Suggested By ICMR

Food Item	Men Quantity / Pers.	Women quantity / Per.
cereals	~~50~~ 520	44
Pulses	50	45
Leafy vegetables	40	100
Milk	200	15
Oil and fat	45	2

Page No.
Date

(c) Salient features of genetic code.

The information needed to direct the synthesis of protein is contained in the DNA in the form of genetic code.

Characteristics of genetic code.

→ Number of Codons

There are 64 possible codon sequences of nucleotide bases A, G, C and used to produce the three base codons.

Nonsense Codons

Three of 64 possible codons triplets UAA, UAG, and UGA does not code for any amino acids, they are called Nonsense codons.

→ The code is universal:-

That is the meaning each codon the same in almost all known organisms.

- UGA codes for tryptophan instead of serving as a stop codon
- AUA codes for methionine instead of isoleucine.
- CUA code for threonine instead of Leucine.

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Date

→ Codons that designate the same amino acids called Synonyms. Two Amino Acids methionine (AUG) and tryptoham (UGG) each have only one codon.

(c) Factors affecting enzyme activity.

-> Various factors affecting Enzyme activity. Ex...

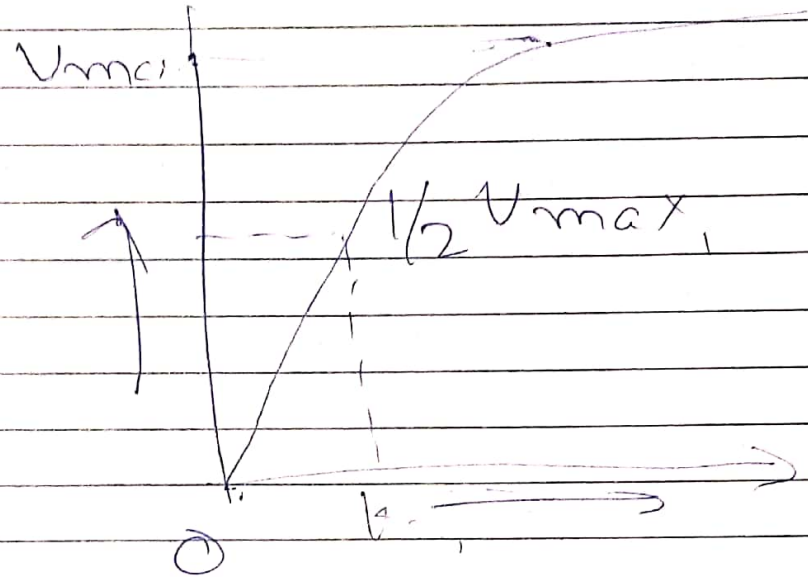
(a) Substrate concentration
(b) Enzyme concentration
(c) Temperature
(d) PH.
(e) Product concentration
(f) Activators and Coenzymes
(g) Time
(h) Physical agents
(i) Inhibitors.

Effect of Substrate Concentration.

For a given given quantity of enzyme, the velocity of the Reaction increases as the concentration of substrate is increased.

At relatively low concentration of substrate, V0 increased almost linearly with an increase in Substrate Concentration (S) a condition known as first order of kinetics.

At higher substrate concentrations, V0 increases by smaller amounts in response to increase in Substrate concentration.

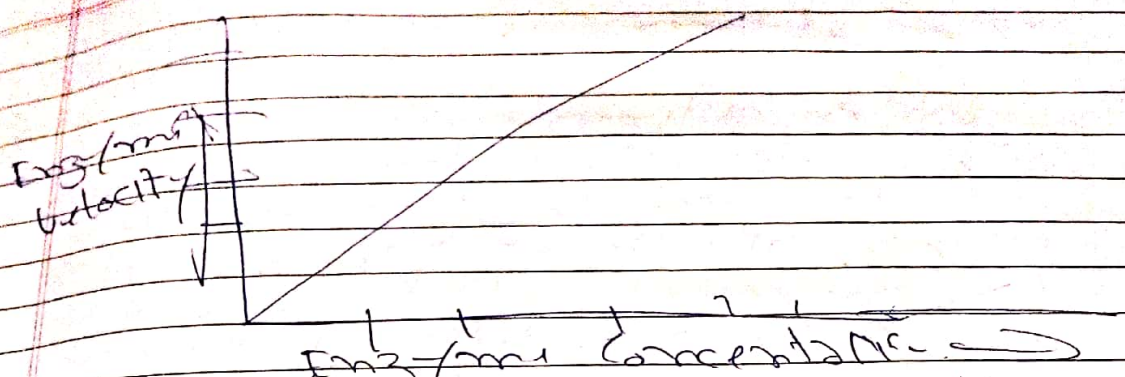


V_0 = Initial Velocity.
V_{max} = Maximum Velocity.
K_m = 1/2 V_{max} =
S = Substrate Concentration =

Effect of enzyme concentration.

- Velocity of Reaction is Directly proportional to the amount of enz present as long as the substrate is not ~~limiting~~ limiting.

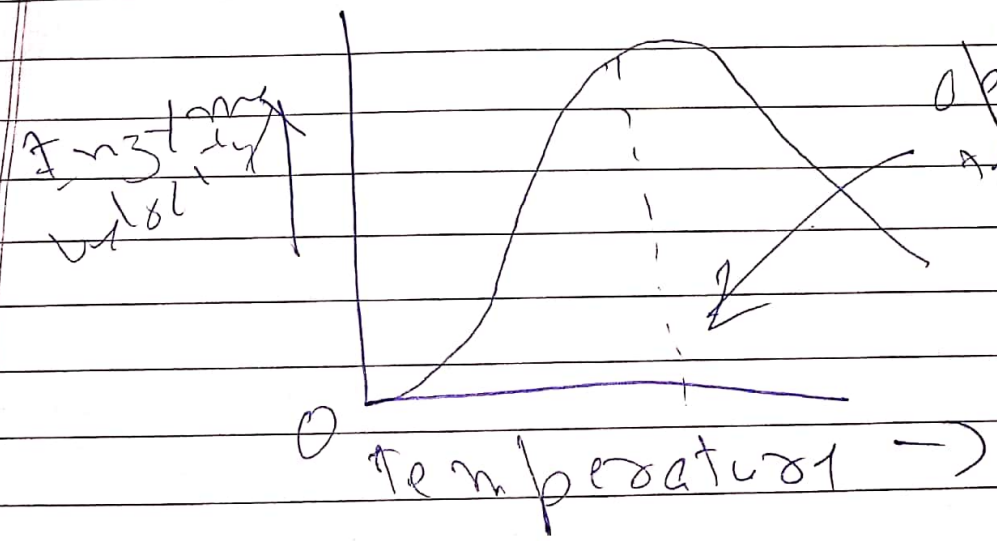
→ the Substrate must be present a concentration Sufficient to ensure that all of the enzyme molecules have substrate bound to their active site.



Effect of enzyme concentration on enzyme activity.

Effect of Temperature

- Enzyme catalyzed reactions show an increase in rate with increasing temperature only within a relatively small and low temperature range.



⇒ Each enzyme shows the high. activity at a particular temperature called optimum temperature.

Page No.
Date

Effect of time.

Under optimum conditions of pH and temperature, time required for an enzyme reaction is less. The time required for an enzyme complex of an enzyme reaction increases with changes in temperature and PH from its optimum

g) Case History

i) The Probable Diagnosis Seems to be Gout.

ii) Different types of Gout are.

a) Primary Gout.

b) Secondary Gout.

a) Primary Gout

Patient with Primary Gout shows deposition of urate crystals in soft tissues that affect joints and leads to painful arthritis.

b) Secondary Gout

Secondary Gout relates variety of Diseases that cause elevated Destruction of cells or decreased level of uric acid.

Elevated Destruction of cells is accompanied by increased degradation of nucleic acids to uric acids to which occurs in Leukemia, polycythemia, psoriasis and cancer, starvation, trauma, etc).

Decreased elimination of uric acid occurs in chronic Renal Disease due to Reduced Glomerular filtration Rate.

(iii) Allopurinol is the drug, which inhibits xanthine oxidase completely. the enzyme Respo for converting purines into uric ~~acid~~ acid.

Page No.
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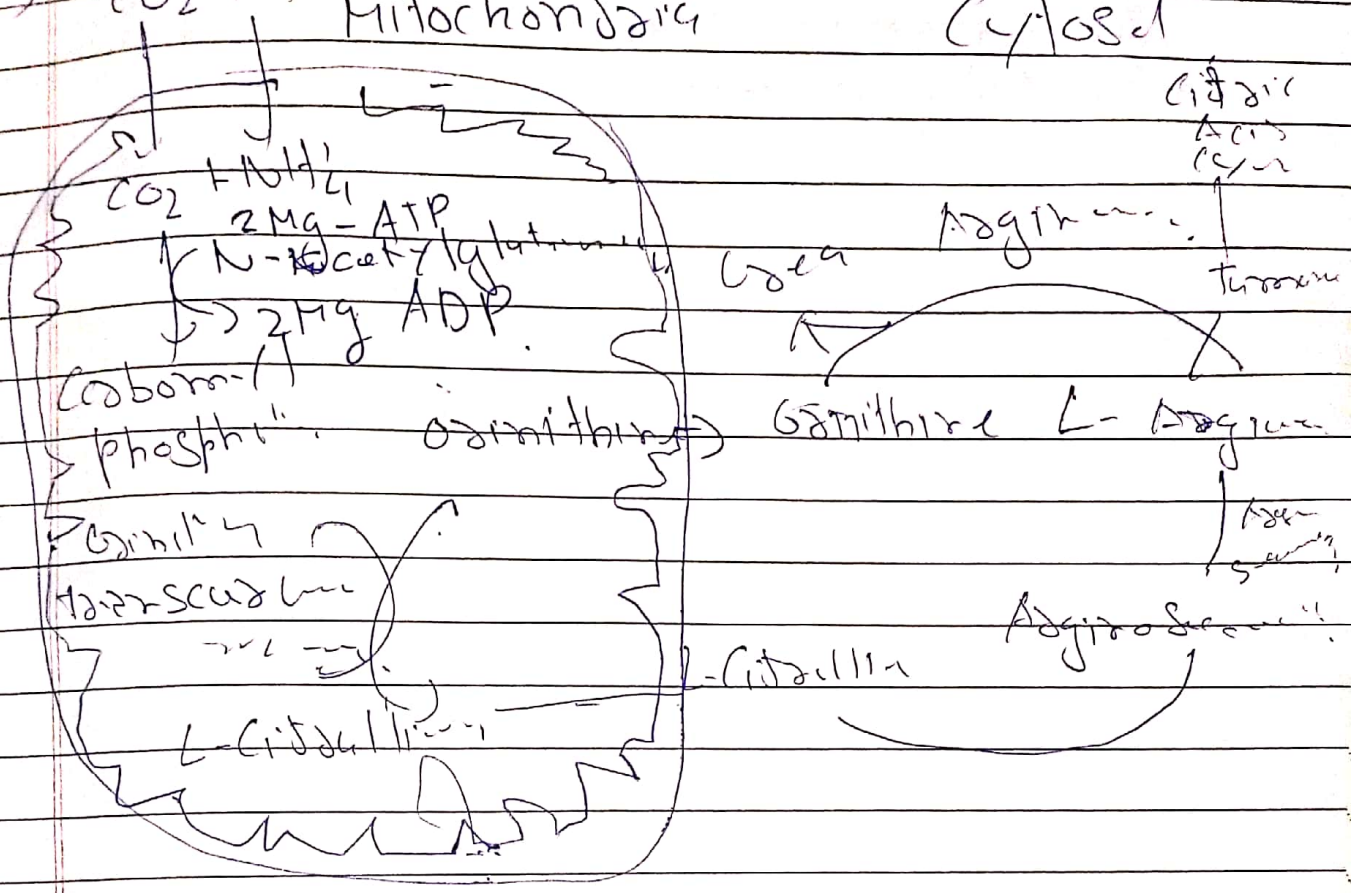
SECTION-C

(2) Describe Urea cycle. Add a Disadd- of Urea Cycle

-) $CO_2 + NH_3$



Reactions of Urea Cycle.

1) formation of Carbamyl Phosphate

2) formation of Citrulline

3) formation of Argino Succinate.

4) formation of Arginine and Fumarate.

5) formation of Urea and Ornithine.

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Date

Disorders of Urea Cycle

1) Hyperammonemia type-I
2) Hyperammonemic type-II
3) Citrullinemia
4) Argino succinic Aciduria
5) Arginimemia.

(3) Describe Vit D

-> Vit D is also known as 'calciferol because of its Role in calcium Metabolism and antiRachitic factor it prevents Rickets.

~~Vit D~~

BDA

- Daily Requirement of Vit D is 200-400 I.U.

functions

Vit D (calcitriol) plays an important Role in the Regulation of ~~Ca~~ Calcium and phosphorus, metabolism.

It maintains the normal Blood level of Calcium and phosphor- by acting on Intestine, kidney and Bones.

Deficiency Manifestation.

Rickets in Children
Osteomalacia in Adults

Rickets

Rickets is characterized by formation of soft and pliable Bones Due to poor minerals and calcium deficiency.

Due to softness, the weight bearing Bones are Bent and deformed.

→ the main feature of Rickets is a large head with protruding forehead, pigeon chest Bow legs (curved legs) Knock knees)

→ Curvature of Spine.

Osteomalacic.

→ the Deficiency of Vit D in adults causes Osteomalacia.

= Osteomalacia characterized by low plasma levels of Calcium and phosphorus, and high alkaline phosphatase.

Page No.
Date

Biochem - II

Reference - Kurlbek 3rd

SECTION - A

MCQ

1) B
2) C
3) B
4) A
5) A
6) B
7) A
8) A
9) B
10) B
11) D.
12) B
13) C.
14) D.
15) A
16) A
17) A
18) C
19) B
20) B.

Page No.
Date

SECTION B

(a) classification of phospholipids with examples

→

Phospholipids are class of lipids that are major component of all cell membranes. they can be form lipid Bilayers of their amphilic characteristic. the structure of phospholipid Molecule generally consist of two hydrophobic fatty acid tails and hydrophilic head consisting of a phosphate group.

Phospholipid can be divided into four major categories

1) Lysolecithin or Inositol phosphatide
2) SphingoMyelin
3) Phosphatidylserine
4) Phosphatidylethanolamine
5) Lecithin.

1) SphingoMyelin

Sphingomyelin is a type of phospholipid found in animal cell membranes, especially in the Membranous Myelin sheath that surrounds some nerve cell axons It consists of phosphocolin and Ceramide or a Phosphoethanolamine head group. therefore Sphingomyelins

can also be classified as Sphingo.
Phospholipids.

Lysolecithin

Lysolecithins are also called Lysophosph
dylcholines (LPC, lysoPC) of chemical
compounds which are Derived from
Phosphatidylcholine Phosphatidyl) choline

Phosphatidylserine

Phosphatidylserine is a phospholipid
is a component of the cell Membra
It plays a key role in cell cycle
signalling, specifically in Relation to
apoptosis. It is a key pathway for
viruses to enter cells via apoptopic
Mimicry.

Lecithin

Lecithin is a Generic term to
Designate any group of yellow-brow
fatty substances occuring Animal
and plant tissues which are
& amphiphilic they attract both wa
and fatty substances, and are us
for smoothening food textures
emulsifying homogenizi
Liquid mixtures and Repelling
Sticking materials.

(B) Metabolic changes in starvation.

Starvation is deprivation of food and thereby deprivation of exogenous supply of calories to meet the energy demands of the body for basal metabolism and other activities.

Metabolic changes during short term short term starvation.

Changes in carbohydrate metabolism and role of the liver

i) the first phase of starvation begins four to five hours after a meal.
→ within 2 hr after meal, blood glucose levels begin to fall.
∴ Consequently, insulin levels decline and glucagon, epinephrine levels rise.
∴ the first priority of metabolism in starvation is to provide sufficient glucose to the brain, other tissues that are absolutely dependent on glucose.

→ In this phase, the main source of Blood Glucose is i) Liver glyco

Metabolic changes during Prolonged Starvation

The process which take place in short term starvation second to on immediately Indefinitely although gluconeogenesis provides Glucose efficiently for the Body energy requirements, it will soon deplete the substantial proportions of Body protein and is known to death ensues when to 30 to 50% Body protein is lost.

Changes in Carbohydrate Metabolism

the Blood Glucose levels drop to about 100 mg/dl to about 70mg after 24hrs but are thereafter maintained.

→ Conservation of Body proteins accomplished By a Reduction in G production by Gluconeog...

Changes in fat Metabolism

During prolong fasting, Adipose continued to Breakdown its fats to fatty acids and Glycerol.

→ these fatty acids serve as source of fuel for the Body

Page No
Date

Changes in Protein Metabolism

During first few Days of Starvation there is a Rapid breakdown of muscle proteins, provide amino acids, alanine and Glutamine for Gluconeogenesis.

After several weeks of Starvation the Rate of muscle Breakdown decr Due to Decreased need of Glucose as a fuel for Brain which has be as Ketone Bodies as a source of energy.

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(1) Dehydration

Dehydration may be defined as a loss of water from the body that exceeds that of intake, as a result body's water content gets reduced and body is in negative water balance. Dehydration may be of two types.

- Dehydration due to pure water deficiency without loss of electrolytes called simple dehydration.
- Simple Dehydration or pure water dehydration is due to depravation of water without corresponding loss of electrolyte. Simple Dehydration is associated with Hypernatraemia.

Causes of Dehydration

Simple dehydration result from depravation of water either due to no or inadequate intake of water due to excessive loss of water from body eg. in diabetes Insipidus

Symptoms of Dehydration

- Symptoms of simple dehydration are immense thirst intense thirst mental confusion, fever and alguria (decreased

- S/symptoms of Deh/dration due to combined deficit of water & electrolytes are dry skin, Dry/mucus membrane, sunken eyeballs, increased Blood urea analysis with increasing severity, hypotension & shock may occur.

Treatment

- Treatment of Simple Deh/dration the patient is asked to drink plenty of water.

- Treatment of Deh/dration due to combined deficient of water and electrolyte is Isotonic solution of Sodium chloride (Normal Saline) is given Intravenously.

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Date

12) Electrophoresis

A charged particle placed in an electric field migrates towards the anode or cathode depending on the net charge ~~of~~ carried by the particle.

Def^n:-

Electrophoresis is the process of separating the charged constituents of a solution by a means of an electric current.

Classification of electrophoresis

1) Zone Electrophoresis:

a) Paper Electrophoresis
b) Gel Electrophoresis
c) Thin layer electrophoresis
d) Cellulose Acetate electrophoresis

Moving Boundary Electrophoresis

a) Capillary Electrophoresis
b) Isotachophoresis
c) Electric focussing

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Clinical Applications of Electrophoresis

→ The electrophoresis procedure is used in clinical laboratory to separate of serum proteins, Lipoproteins, isoenzymes, hemoglobin and other classes of macromolecules.

Immuno electrophoresis is used to determine specific classes of immunoglobulins.

Page No
Date

1) Case History

a) Renal Glycosuria

2) → a) Diabetes Mellitus.
   - b) Glycosuria.

3) a) Normal Blood Glucose level
   ~~80-130~~ 80 - 130 mg/dl.

b) Normal Renal threshold - 180mg

SECTION-C

(a) Enumerate Liver function tests and describe the tests Based on excretory function.

-) Liver Function tests

tests Based on Excretory function.

It includes measurement of.

a) Serum Bilirubin
b) Urine Bilirubin
c) Urine Bile Salts.
d) Bromosulphathalein (or BSP) dye tests.

Serum Bilirubin estimation

Van Den Bergh Reaction.

Estimation of Serum Bilirubin is Based on * Van Den Bergh Reac

- Conjugated bilirubin being water soluble can react directly with aqueous solution of Diazo Reagent so called direct Bilirubin
- Whereas the unconjugated Bil.. water insoluble and does not react in aqueous solution. It requires addition methyl alcohol to

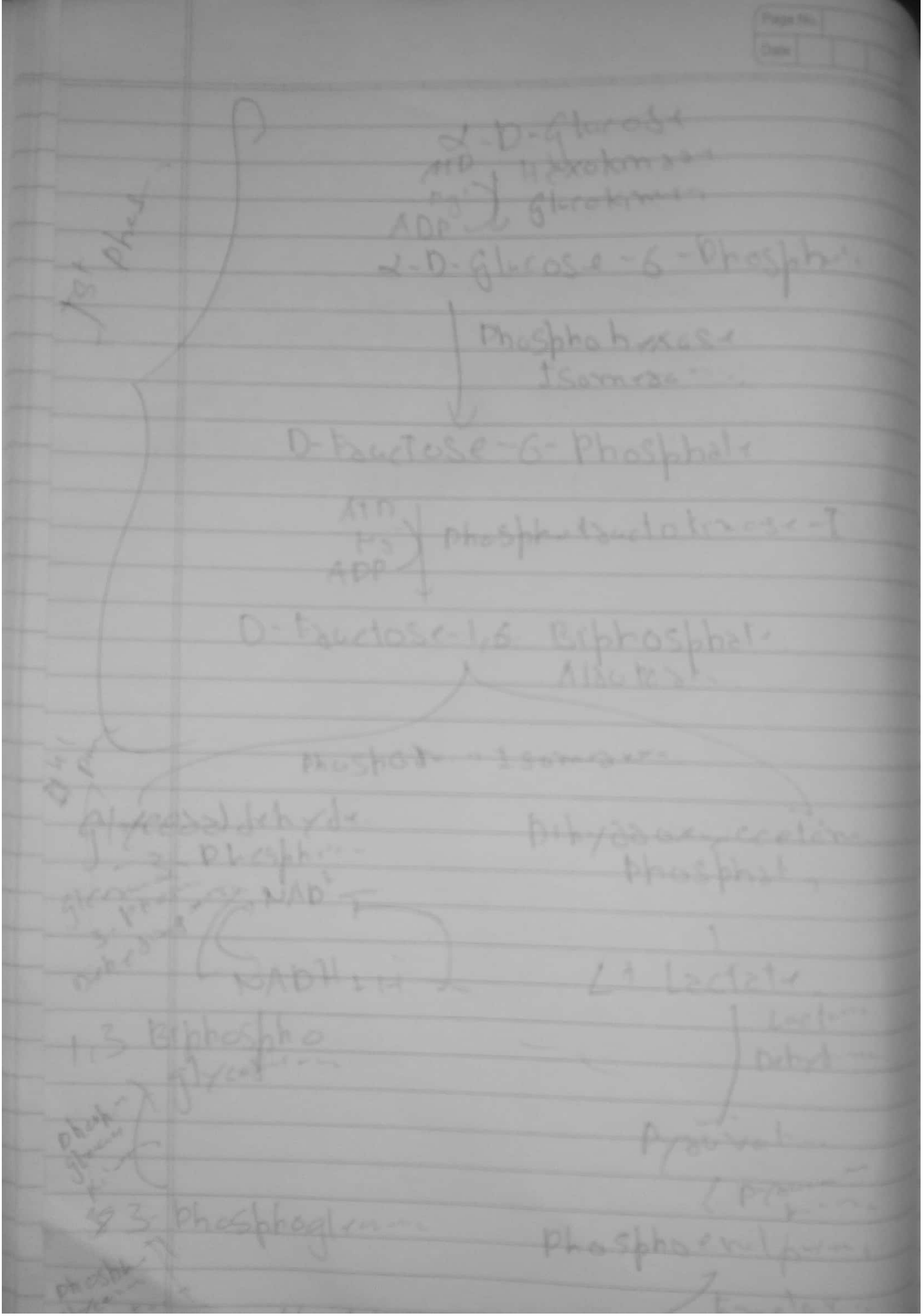
React with Diazo Reagent in the determination method and call it Indirect Bilirubin.

-> If both conjugated and unconjugated bilirubin are present in small amounts, a purple color is produced immediately and the color is intensified on addition

(2)

→ Def:

Glycolysis is the sequence of Rea... th.t converts glucose into pya... in the presence of oxygen (aerob... or lactate in the absence of oxyge... (aneaob...) with the production of ATP. this pathway is called ~~Int~~ Embden Meyerhoff pathway.



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Energetics of glycolysis

1) Under Aerobic conditions 8 molecules of ATP are produced
2) In Anaerobic glycolysis 2 molecules of ATP are produced per molecule of glucose.

Name - Yash Jagannath patil

Exam - Preliminary Exam

Date - 12/6/2020

Subject - Biochemistry I

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section A

MCQs

Q1)

1] - LDH-5

2] . Hydrogen bond

3] poly (a) tail

4] primers

5] Liver

6] Transferases

7] IgG

8] Erythromycin

9] PLP

10] HGPRTase

11] a_2B_2

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12] - 3 ATP

13] - VIT B12

14] - Oligomycin

15] - Amylase

16] Hexokinase

17] - FAD

18] - purine metabolism

19] - creatine

20] - ribosomes

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Balanced Diet suggested by ICMR

Food Item	Adult man			Adult woman		
	sedentary work	Moderate work	Heavy work	sedentary work	Moderate work	Hea wor
	Quantity g rum/day			Quantity gm/day		
cereals	460	520	670	410	446	675
pulses	40	60	60	40	45	50
Leafy vegetables	40	40	40	100	100	100
other vegetables	60	70	80	40	40	60
Root and tubers	50	60	80	50	50	60
Milk	150	200	250	100	150	200
Oil and fat	40	45	65	20	25	40
sugar and jaggery	30	35	55	20	20	40

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Section B

Q2)

Balanced Diet

C] The dietary pattern varies widely in different parts of the world. It is generally developed according to the

- kinds of Food products (which depends upon the climatic condition)
- Economic capacity
- Religion
- customs
- Taste and habbits of people

ICMR has suggested balance diet for different age group, sex and under various occupations for physical activity.

nutrition disorders

when balanced diet is not consumed by a person for a sufficient length of time, it leads to nutritional deficiencies or disorders, this nutritional status is called malnutrition.

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Additional Allowance during pregnancy and lactation

Food items	During pregnancy gm/day	Calories KCl	During lactation gm/day	Calories KCl
cereals	35	118	60	203
pulses	15	118	60	203
Milk	15	83	100	83
fat	-	-	10	90
sugar	10	40	10	90
Total	-	293	-	521

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Enzymes are biological materials with catalytic properties ie. they increase the rate of chemical reactions in biological and in vitro (in the laboratory) system that otherwise proceed very slowly.

The study of enzymes and of the changes in the enzyme activity that occure in body fluids has become a valuable diagnostic tool

Defn

Enzymes are biological catalyst produced by living tissues they are protein (except a small group of RNA acting as Ribozyme) that have the property of accelerating specific chemical reactions without being consumed in the process

Coenzyme And activators

- some enzyme require additional nonprotein component for its optimum activity this additional component is called coenzyme.
- many vitamins will be consider for function as coenzymes.

coenzyme derived from vitamins.

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Common coenzyme and their functions

vitamin	coenzyme	functions of coenzymes
Thiamine (B1)	TPP (thiamine pyrophosphate)	oxidative decarboxylation and transketolase
riboflavin (vit B2)	FAD and FMN Flavin adenine dinucleotide and flavin mononucleotide	oxidation and reduction reaction.
niacin	NAD^+ (nicotinamide adenine dinucleotide) $NADP^+$ (nicotinamide adenine dinucleotide phosphate)	oxidation and reduction reaction
pyridoxine	Biotin	carboxylation reactions
Folic acid	THF (tetrahydrofolate)	carrier of one carbon group
pantothenic acid	coenzyme A	Acyl carrier
cyanocobalamine	methylcobalamine and deoxyadenosylcobalamin	transfer of CH_3 group and Isomerization

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Enzyme Requiring or containing Inorganic elements

Enzyme	cofactor (Activator)
Ferroxidase (ceruloplasmin) Ascorbic acid oxidase	copper
carbonic anhydrase DNA - polymerase, porphobilinogen synthase, carboxypeptidase	zinc
cytochrome oxidase catalase	iron
Glucose 6 phosphatase, Hexokinase	magnesium
Glutathione peroxidase arginase	selenium
pyruvate carboxylase	manganese
xanthine oxidase	molybdenum

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Plasma contains a variety of proteins with different functions. At present time over 100 different plasma protein have been described. there are many proteins whose functions demains to be determined

Plasma protein

1] Albumin

2] Globulin

3] Fibrinogen

The normal value of plasma proteins are

Total protein - 6-8 gm %
serum albumin - 3.5 - 6 gm %
serum globulin - 2 to 3.5 gm %
Fibrinogen - 200 to 400 mg %
A/G Ratio - 1.2:1 to 2.5:1

Synthesis of plasma protein

- All the albumin and fibrinogen are essentially synthesized by the liver only
- 50-80 % of the globulin is formed in the liver.

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- The remaining globulins are formed almost entirely in the lymphoid tissues.

Major classes of plasma proteins, their functions and Diagnostic importance

classes	Examples	principles functions
Albumin		Exert colloidal osmotic pressure and transport functions.
Globulin	α_1-protease inhibitor (API) or α_1-antitrypsin (AAT)	- Antiprotease, natural inhibitor of proteolytic enzyme elastase
	ceruloplasmin	· Transport of copper
	Heptoglobin	conversation of iron by binding free hemoglobin
	α_2-macroglobulin (AMG)	- natural antiprotease, inhibits thrombin, trypsin, pepsin
	Hemopexin	binds heme and prevent loss of iron
	Transferrin	transport of iron

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	C-reactive protein (CRP)	Body's defense mechanism
	β2- microglobulin (BMG)	Body's defense mechanism
	Immunoglobulins IgG, IgA, IgM IgE	Body defense mechanism
fibrinogen		Blood coagulation

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b) Isoenzyme or isozymes are multiple forms (isomers) of the same enzyme that catalyze the same biochemical reactions.

Isoenzymes shows different chemical and physical properties like electrophoretic mobility and kinetic properties.

- Not all enzymes have isoenzymes. Infact, it was found that only those enzymes, which are active in polymeric form demonstrate isoenzyme.

1) Lactate dehydrogenase
2) Creatine kinase (CK) formerly called creatine phosphokinase (CPK)

Isoenzymes Examples

Enzymes	Iso-Enzyme forms
Acid phosphatase	Prostate, erythrocytes, platelets, liver, spleen, kidney and bone marrow
Alkaline phosphatase	Bone, liver, placenta intestine and kidney

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Amylase	salivary and pancreatic
Hexokinase	liver (glucokinas) and muscles

3] Lactate dehydrogenase

- Lactate dehydrogenase is a tetrameric enzyme that catalyzes oxidation of lactate to pyruvate
- LDH has five Isoenzymes
  - LDH 1
  - LDH 2
  - LDH 3
  - LDH 4
  - LDH 5

∴ since LDH is a tetramer made up of two types of polypeptide M (muscle) type H (Heart) type, five combination are possible with varying ratio

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- Five isoenzymes of LDH can be detected by electrophoresis as they have different electrophoretic mobility.

- LDH_1 is a fatest moving fraction moving towards anode and LDH_5 is the slowing moving isoenzyme.

2] Creatine kinase

Creatine kinase isoenzymes are dimers that are made up of two types of polypeptide chains, which may be either M (muscle) type or B (Brain) type.

- CK_1 (BB) - It is present in the brain

- CK_2 (MB) - cardiac tissues is the only tissue which has the mixed MB (CK_2)

- CK_3 (MM) - It is present in the skeletal muscles

- CK_1 may be elevated in neonates particularly in damaged brain or very low birth weight neoborn

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Importance of Lactate dehydrogenase (LDH) and creatine kinase)

Type	Composition	Location	Diagnostic importance
LDH_1	HHHH	Heart, RBC	myocardial infraction
LDH_2	HHHM	Heart, RBC	Megaloblastic anemia
LDH_3	HHMM	Brain	Leukemia, malignancy
LDH_4	HMMM	Lung spleen	pulmonary infraction
LDH_5	MMMM	Liver, muscle	liver disease muscle damage
CK1	BB	Brain	neurological injury
CK2	BM	Heart	Myocardial infraction
CK3	MM	Skeletal muscle	Muscular dystrophies and myopathies

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e] Generic code

The information needed to direct the synthesis of protein is contained in the mRNA in the form of a generic code

The generic code is a system of nucleotide sequences of mRNA that determines the sequence of amino acids in protein

characteristic of generic code

1) number of codons; there are 64 possible codon sequences because four nucleotide bases. A, G, C and U are used to produce the three base codons, there are therefore 4^3 or 64 possible codon sequences.

2) Stop termination or nonsense codons
three of the 64 possible nucleotide triplets, UAA, UAG and UGA do not code for any amino acids they are called nonsense codons that normally single signal termination of polypeptide chain these nonsense codons name amber, opal, ochre

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- The code is degenerate but unambiguous.

- As there are 61 codons for 20 amino acids, one amino acids are has more than one codon and code is referred to as degenerate

- codons that designate the same amino acid are called synonyms. two amino acids methionine (AUG) and tryptophan (UGC) each have only one codon

- The code is almost universal; that is the meaning of each codon is the same in almost all known organism

- UGA code for tryptophan instead of serving as a stop codon

- AUA codes for methionine instead of isoleucine

- CUA codes for threonine instead of leucine

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	U	C	A	G	
U	UUU Phe UUC UUA Leu UUG	UCU UCC UCA UCG Ser	UAU Tyr UAC UAA* UAG*	UGU Cys UGC UGA* UGG Trp	U C A G
C	CUU CUC CUA CUG Leu	CCU CCC CCA CCG Pro	CAU His CAC CAA Gln CAG	CGU CGC CGA CGG Arg	U C A G
A	AUU AUC AUA AUG	ACU ACC ACA ACG Thr	AAU Asn AAC AAA Lys AAG	AGU Ser AGC AGA AGG Arg	U C A G
G	GUU GUC GUA GUG Val	GCU GCC GCA GCG Ala	GAU Asp GAC GAA Glu GAG	GGU GGC GGA GGG Gly	U C A G

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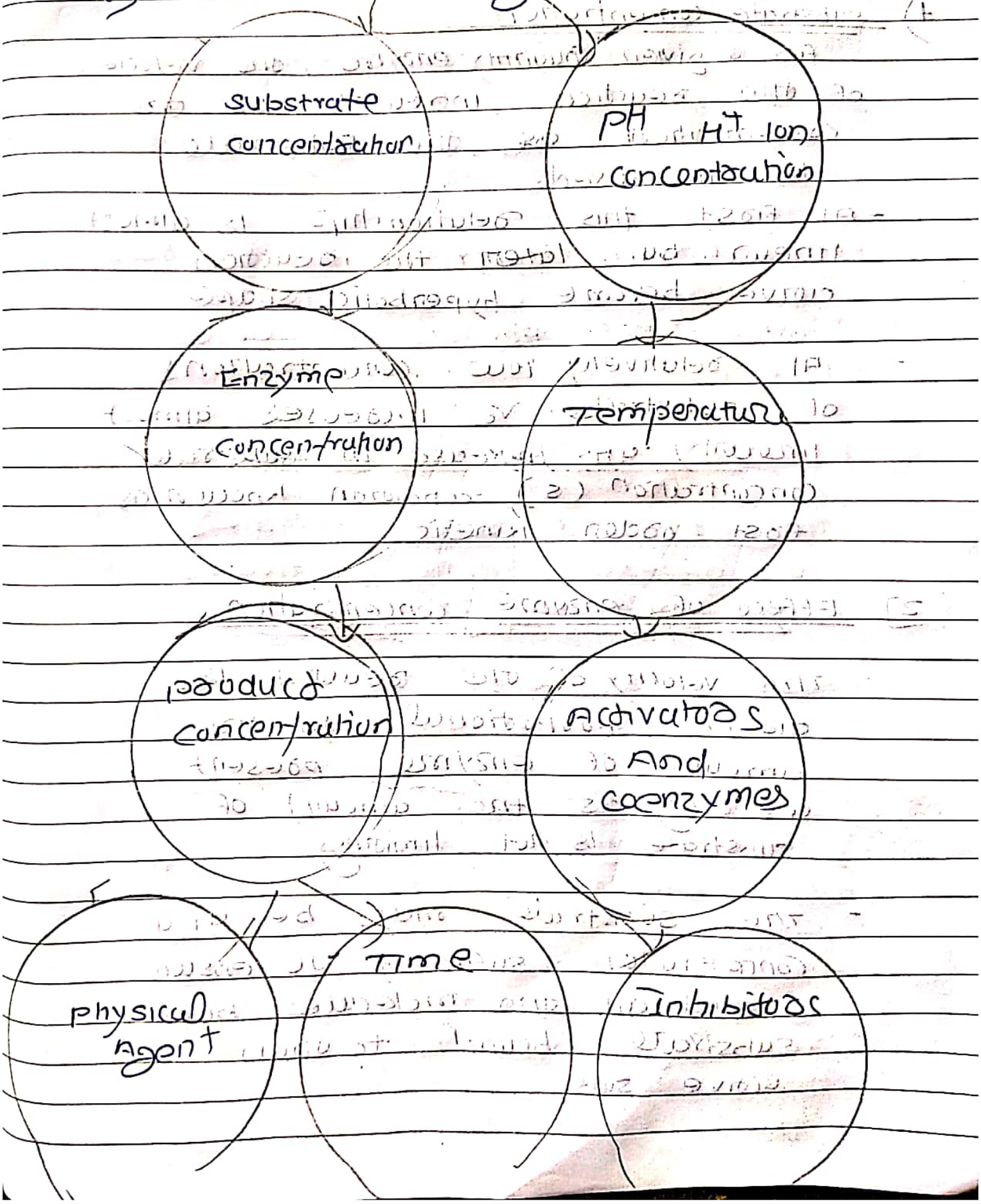
E) Factors affecting enzyme Activity



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1) Substrate concentration

- For a given quantity enzyme → the velocity of the reaction increases as the concentration of the substrate is increased.
- At first this relationship is almost linear but later the reaction curve become hyperbolic shape.
- At relatively low concentrations of substrate, V_0 increases almost linearly with an increase in substrate concentration (S) condition known as first order kinetic.

2] Effect of enzyme concentration

- The velocity of the reaction is directly proportional to the amount of enzyme present as long as the amount of substrate is not limiting.
- the substrate must be at a concentration sufficient to ensure that all the molecules have substrate bound to their active site.

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3] Effect of Hydrogen ion concentration pH

- Each enzyme has an optimum pH, ie pH at which the enzyme activity is maximum. below or above this pH enzyme activity is decreased. the optimum pH
- As pH affects the enzyme activity in enzyme studies buffer are used to keep the enzyme at an optimum or most favorable pH

4] Effect of Temperature

- Enzyme catalyzed reactions show increase in rate with increasing temperature only within a relatively small and low temperature range.
- Each enzyme shows the highest activity at a particular temperature called optimum temperature
- A bell shaped curve is obtained when we plot the enzyme velocity Vs
- Increase in velocity is due to increase in the kinetic energy.

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5] Effect of products

- accumulation of products of one reaction causes the inhibition of enzyme activity for some enzymatic reactions
- this form of control limit the rate of formation of the product when the product is under used

6] Effect of coenzyme and activators

- The activity of many enzymes is depends on the activators like Mg^{2+}, Mn^{2+}, Zn^{2+}, Ca^{2+}, Co^{2+}, Cu^{2+} and coenzyme for the minimum activity

7] Effect of time

under optimum conditions of pH and temperature, time required for an enzyme reaction is less the time required for the completion of an enzyme reaction increases when changes in temperature and pH from its optimum

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8) Effect of physical agent

- Physical agent like light rays can inhibits or accelerate certain enzyme reaction. eg. the activity of salivary amylase is increased by Red and blue ~~light~~ light.

9) Effect of inhibitors

- the sustances which stop the enzymatic reaction are called inhibitors. presence of these substances in reaction medium decreases the rate of enzyme reactions.

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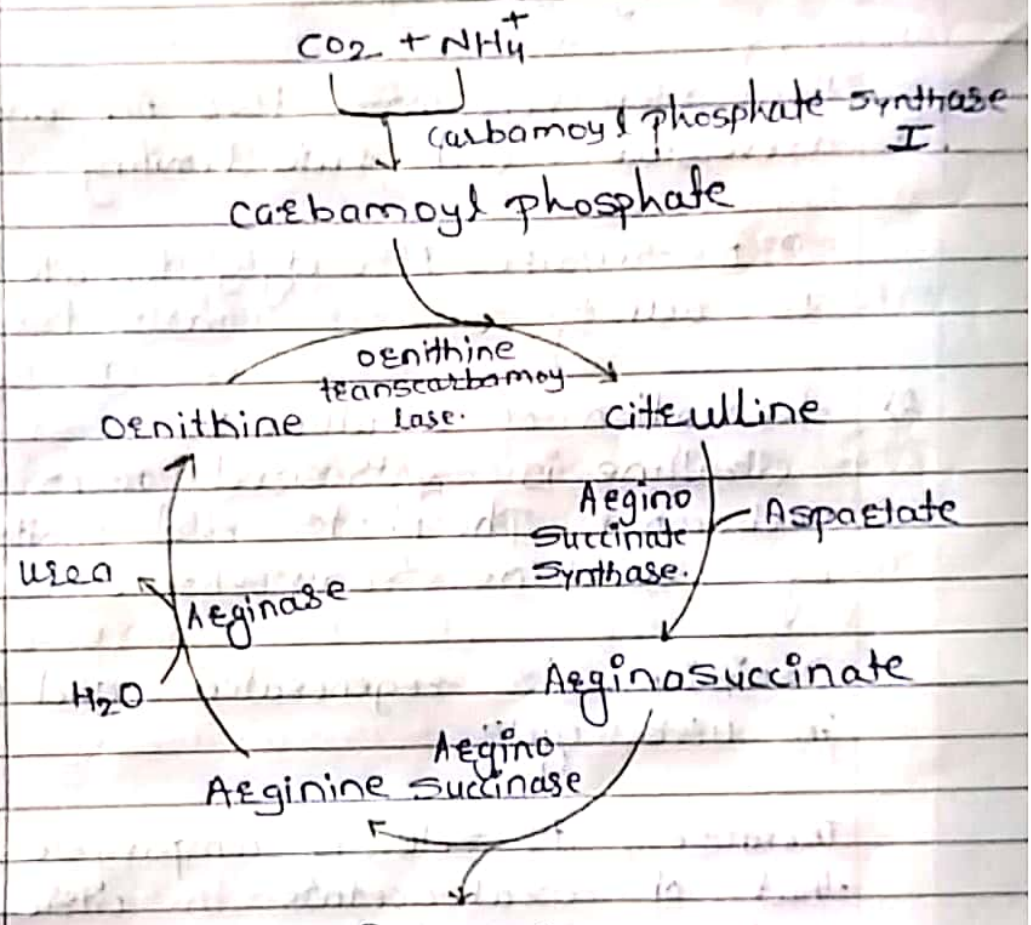
Section - C

Q.B

b) ——— →?

Urea cycle



Urea is the end product of protein metabolism.
The nitrogen of amino acids converted to ammonia is toxic to the body. It is converted to urea and detoxified.

Urea is synthesized in amino-liver and transported to kidneys for excretion

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Urine.

1) Synthesis of carbamoyl phosphate :-
carbamoyl phosphate synthase I of mitochondria catalyses the condensation of NH_4^+ ions with CO_2 to form carbamoyl phosphate.

This step consumes two ATP and is irreversible, and rate-limiting

CPS I requires N-Acetylglutamate for its activity.

2) formation of citrulline :-
citrulline is synthesized from carbamoyl phosphate and ornithine by ornithine transcarbamoylase.

Ornithine is regenerated and used in urea cycle.

Therefore, its role is comparable to that of oxaloacetate in citric acid cycle.

3) Synthesis of Arginosuccinate :
Arginosuccinate synthase condenses citrulline with aspartate to produce arginosuccinate.

The second amino acid group of urea is incorporated in this reaction.

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4) Cleavage of arginosuccinate :-
Argino succinase cleaves arginosuccinate to give arginine and fumarate. Arginine is the immediate precursor for urea.

5) Formation of urea :-
Arginase is the fifth and final enzyme that cleaves arginine to yield urea and ornithine.

Ornithine, so regenerated, enters mitochondria for its reuse in the urea cycle.

Arginase is mostly found in the liver, while the rest of the enzymes of urea cycle are also present in other tissues.

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(4)

Disorderes of urea cycle :-

- Five disorderes associated with each of the enzymes of urea cycle have been reported

1) Hyperammonemia type-I
2) Hyperammonemia type-II
3) Citrullinemia
4) Argininosuccinic aciduria
5) Argininemia.

Since urea synthesis converts toxic ammonia to non-toxic urea, all defects in urea synthesis result in hyperammonemia and ammonia intoxication.

This intoxication is more severe when the metabolic block occures at Reaction I or II, since it accumulates ammonia itself.

Deficiency of later enzymes result in the accumulation of other intermediates of the urea cycle, which are less toxic and therefore severity of symptoms is less.

Symptoms :-
1) Ammonia intoxication
2) Protein induced vomiting
3) Intermittent ataxia
4) Irritability

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c) ——————→?

RDA :-

The daily requirement of Vitamin D is 400 international units or 10 mg of cholecalciferol.

In countries with good sunlight the RDA for vitamin D is 200 IU

Dietary Sources

good sources of vitamin D include fatty fish, fish lives oils, egg yolk etc. Milk is not a good soueces of vitamin D.

Vitamin D can be peovided to the body in three ways.

1. Exposure of skin to sunlight for synthesis of vitamin D.
2. Consumption of Natural foods.
3. By irradiating foods that contain precursors of vitamin D and fortification of foods.

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Functions :-

Vitamin D plays an essential role as a hormone in the regulation of calcium and phosphorus Metabolism.

1) On Intestine :-
It increases the plasma calcium and phosphorus concentration by stimulating the absorption of calcium and phosphorus from the intestine by enhancing the synthesis of calcium binding proteins calbindins.

2) On kidney :-
It stimulates the reabsorption of calcium and phosphorus from the kidney and decreases their excretion.

3) On bone :-
It is believed that calcitriol has both anabolic and catabolic role on bone.

- calcitriol promotes the mineralization of bones by deposition of calcium and phosphorus.
- calcitriol along with PTH stimulating the mobilization of calcium and Phosphorus from bone by stimulating the synthesis of osteocalcin.

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This causes elevation of plasma calcium and phosphorus levels.

Deficiency Manifestation :-

Deficiency of vitamin D causes rickets in growing children and osteomalacia in adults.

Rickets :-

Rickets is characterized by formation of soft and pliable bones due to poor mineralization and calcium deficiency.

Due to softness, the weight bearing bones are bent and deformed.

osteomalacias :-

The deficiency of vitamin D in adults causes osteomalacias.
This is a condition similar to that of rickets.

osteomalacia characterized by demineralization of previously formed bones, Demineralization of bones makes them soft and susceptible to fractures.

Renal Rickets :-

In renal failure synthesis of calcitriol in kidney is impaired.

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As a result, the deficiency of calcitriol occurs which leads to hypocalcemia and hypophosphatemia.

It can be treated by oral or intravenous administration of calcitriol.

Vitamin D Resistant Rickets:-

There are various possible causes of this condition and all involve a defect in the metabolism or mechanism of action of 1,25 dihydroxycholecalciferol.

- Due to defective vitamin D receptor
- Due to defective 1, 1-hydroxylase activity in kidney
- Due to liver disease and kidney failure.

Hypervitaminosis D.

High doses of Vitamin D over a long period are toxic.

- Nausea, vomiting, anorexia, increased thirst, loss of weight, etc.
- Hypercalcemia is seen due to: increased bone reabsorption and intestinal absorption of calcium.
- The prolonged hypercalcemia causes calcification of soft tissues and organs such as kidneys and may lead to formation of stones in the kidneys.

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Name :- Yash Jaganath Patil

Exam :- Priliminary Exam

Date :- 13/6/2020

Subject - Biochemistry-II.

Section - A.

Q.1 ⟶ ?

1. b
2. c
3. b
4. a
5. a
6. b
7. a
8. a
9. b
10. b
11. d
12. b
13. c
14. d
15. a

16. a

17. a

18. c

19. b

20. b

Section-B

Q.

A) ~~classification~~ ?

classification-

- Glycerophospholipids
- Sphingophospholipids

① glycerophospholipids :-

Phospholipids derived from glycerol are called glycerophospholipids.

classification.

1. Phosphatidylcholine (lecithin).
2. phosphatidylethanolamine (cephalin).
3. phosphatidylserine
4. phosphatidylinositol
5. Plasmalogens
6. Lysophospholipids
7. cardiolipin (diphosphatidylglycerol).

(2) Sphingophospholipids :-
Phospholipids derived from alcohol sphingosine instead of glycerol are called sphingophospholipids, e.g. Sphingomyelin.

Sphingomyelin :-

Sphingomyelin is only phospholipid in membranes that is not derived from glycerol.

Intead the alcohol in sphingomyelin is sphingosine, an amino alcohol.

Sphingomyelin is one of the principal structural lipids of membranes of nerve tissue.

In Sphingomyelin the amino group of the sphingosine is linked to a fatty acid to yeild ceramide.

In addition, the primary hydroxy group of sphingosine is esterified with phosphorylcholine.

b) ——————→?

Metabolic changes :-

1) The first phase of staevation begins four to five hours after a meal.

2) Within about 1 hr after a meal blood glucose levels begin to fall.

3) consequently insulin levels decline and glucagon epinephrine levels rise

4) The brain, the erythrocytes, the bone marrow, the renal medulla and peripheral nerves have to be supplied with glucose, for their energy needs.

5) However the tissues such as muscle can readily use free fatty acids, released from adipose tissue.

6) In this phase, the main source of blood glucose is liver glycogen.

7) The liver first uses glycogenolysis the gluconeogenesis to maintain blood glucose levels and provide sufficient glucose to the brain and other glucose requiring

tissues.

8) The fall of glucose and insulin concentration and rise of glucagon stimulates glycogenolysis to maintain the blood glucose levels.

9. glucagon stimulates c-AMP formation, essential for degradation of glycogen.

10. The liver glycogen is capable of maintaining the blood glucose ~~level~~ concentration at normal values for 12 to 16 hours.

11. As the liver glycogen store begins to be depleted, gluconeogenesis becomes active which ensures a continuous supply of glucose to the brain and other tissues.

The source of substrates for gluconeogenesis are:
- pyruvate
- lactate which is a product of glycolysis in red blood cell and exercising muscle.
- Glucogenic amino acids released from muscle.
- Glycerol released by degradation of triacylglycerols.

changes in Protein Metabolism & role of muscles

↓

changes in fat Metabolism and adipose tissue.

Metabolic changes during Prolonged Starvation.

① changes in carbohydrate Metabolism

↓

② changes in fat Metabolism

↓

③ changes in protein Metabolism.

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c) ——————→?

The plasma calcium concentration is normal individual is 8-11 mg%.

Regulation

Homeostasis is plasma calcium is dependent on the:

- function of three main organs:

i) Bone
ii) kidney
iii) intestine

- function of three main hormone:

i) Parathyroid hormones:
ii) vitamin D or calcitriol.
iii) calcitonin.

- The four major process are.

1) The absorption of calcium from the intestine, mainly through the action of vitamin D.

2) Reabsorption of calcium from the kidney, mainly through the action of parathyroid hormone and vitamin D.

3) Demineralization of bone mainly through action of parathyroid

but facilitated by vitamin D.

4. Mineralization of bone through the action of calcitonin.

Role of parathyroid hormone:-
It is secreted by the parathyroid in response to drop in the blood calcium level.

Action on bon :- PTH stimulates mobilization of calcium and phosphate from bone by stimulating osteoclast activity.

Action on kidney :- In kidney PTH increases the tubular reabsorption of calcium and decreases renal excretion of calcium.
PTH increases excretion of phosphate by inhibiting its renal reabsorption.

Action on intestine :- Action of PTH on intestine is indirect via the formation of calcitriol, active from of vit. D.

Role of vit. D :-
- Absorption of calcium & phosphorus from intestine
- Reabsorption of calcium & phosphorus from intestine kidney.
- Mobilization of calcium and phosphorus from the bone.

(11)

D. D ——→?

Dehydration :-

Dehydration may be defined as ~~loss of~~ a state in which loss of water exceeds that of intake, as a result of which body's water content gets reduced and the body is in negative water balance.

Dehydration.

Dehydration due to Pure water deficiency	Dehydration due to combined deficiency of water and electrolyte, sodium.

① Dehydration due to pure water deficiency, without loss of electrolytes, called simple dehydration.

Simple dehydration is due to deprivation of water without corresponding loss of electrolytes.

Simple dehydration is associated with ~~sir~~ hypernatremia, i.e ↑ level of sodium and increase in ECW osmolality due to loss of water from the body.

② Dehydration due to combined deficiency of water and electrolyte, Sodium.

Dehydration due to combined water and electrolytes sodium deficiency is more common than simple dehydration.

causes :-
simple dehydration :-
i) Excessive loss of water from body.
ii) inadequate intake of water.

combined deficiency :-
i) Vomiting
ii) diarrhea
iii) excessive sweating.
iv) Salt wasting renal disease
v) Adrenocortical insufficiency.

Symptoms :-
1) intense thirst,
2) Mental confusion
3) fever and
4) oliguria
5) wrinkled skin
6) dry mucous membranes
7) muscle cramps.
8) sunken eyeballs
9) increased blood urea Nitrogen.
10) increasing severity
11) weakness
12) hypotension.

(13)

E. ——→ D

Electrophoresis :-

A charged particles placed in an electric field migrates towards the anode or cathode, depending on the net charge carried by the particle.

Defn :-
Electrophoresis is the process of separating the charged constituents of a solution by means of an electric current.

Electrophoresis apparatus :-

- The electrophoresis apparatus is designed so that the circuit between the two poles is bridged by the support medium holding the sample, and the current flow is partially carried by the components of the sample.

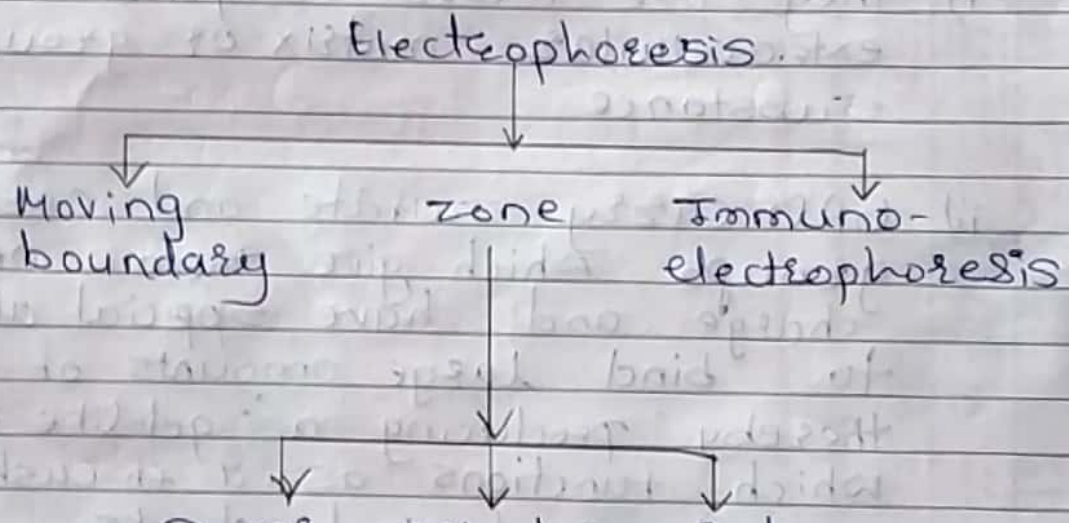
- An electrophoresis chamber consists of two compartments separated from each other by a dividing wall, on side contains the anode and the other, the cathode.

- Each side is filled to the same level with a buffer is often used for serum protein separation.

- A "bridge," across the top of the dividing wall holds a membrane or other support material so that each end of it is in contact with the buffer in one of the compartment.

- first membrane is immersed in buffer, blotted and placed in the chamber, and then sample is applied.

classification :-



F. ——— s?

glucoseaminoglycans :-

It is found in the :-
- Synovial fluid of joints
Vitreous humor of the eye.
Arterial walls.
Bones
cartilage.

functions :-

i) They are major components of the extracellular matrix or ground substance

ii) GAGs carry sulfate and carboxyl groups which give them a negative charge and have special ability to bind large amounts of water. thereby producing a gel-like matrix which functions as a ~~th~~ cushion against mechanical shocks.

iii) They act as "molecular sieves". determining which substances enter and leave cells.

iv) They also give resilience to cartilage, permitting compression and ~~re~~ expansion.

v. They lubricate joints both at the surface of cartilage and in synovial fluid.

vi. The viscous lubricating properties of mucous secretions are also due to the presence of glycosamino glycans, which led to the original naming of these compounds as mucopolysaccharides.

Types :-

- Hyaluronic acid
- Condroitin sulfate
- Keratan sulfate
- Dermatan sulfate
- Heparin
- Heparin sulphate.
- Uronic acid composition
- Amino sugar composition
- linkage between amino sugar and uronic acid
- chain length of the ~~disch~~ disaccharide polymer.
- presence & absence of sulfate groups and their position of attachments to the sugar.
- The nature of the core protein to which they are attached.
- Their tissue and subcellular distribution and their biological function.

SAPNA
PAGE NO
DATE : / /20

Section - C

1. Liver function Tests.

classification.

- Tests based on excretory function.
  - serum bilirubin
  - urine bilirubin
  - urine bile salts
  - Bromosulphophthalein dye tests.
- Based on detoxification funcn.
  - Blood ammonia & bilirubin
  - Hippuric acid Test.
- Based on synthetic funcn.
  - plasma proteins, albumins & globulins
  - prothrombin time.
- Based on metabolic function.
  - Galactose tolerance test
  - Serum protein estimation.
  - serum ammonia estimation.
- Serum enzymes.
  - serum alanine transaminase
  - Serum aspartate transaminase
  - Serum alkaline phosphatase.

Liver function test based on excretory function.

- An important physiologic role of the liver is the removal of toxic endogenous and exogenous substances from the blood.

① Tests based on bilirubin metabolism:
Bilirubin is the excretory end product of heme.
It is conjugated in the liver to form bilirubin diglucuronide

Bilirubin test in serum in two forms.
i) conjugated or direct bilirubin which is water soluble.
ii) unconjugated or indirect bilirubin which is water insoluble.

A. Serum bilirubin estimation :-
Van Den Bergh Reaction.
Estimation of serum bilirubin is based on Van Den Bergh reaction.

In this reaction, when serum bilirubin is allowed to react with vanden Bergh's diazo reagent a purple colored azobilirubin is formed.

B. Urine Bilirubin :-
In normal individuals, bilirubin is not excreted in the urine.
When it is present in the urine it

indicates some disease of the liver.

- only conjugated bilirubin is soluble in water and is excreted in urine but not the unconjugated which is water insoluble.

- In urine, conjugated bilirubin can be detected by fouchet's test.

C. Urobilinogen in urine is
The amount of urobilinogen present in urine depends on the amount of bilirubin entering the intestine.

Urine urobilinogen is estimated semi-quantitatively by Ehrlich's aldehyde reagent.

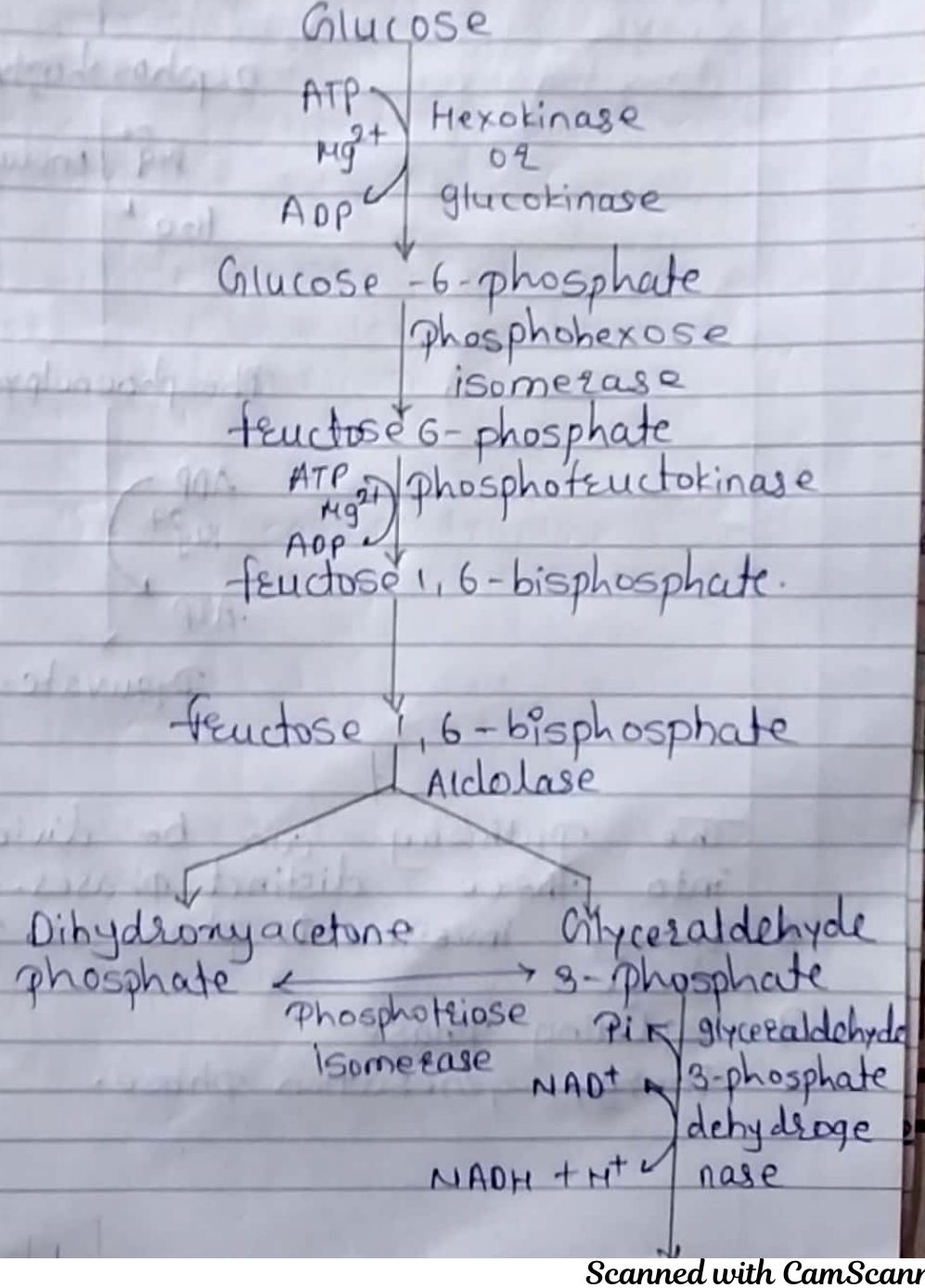
D. Dye excretion Test :-
In addition to excreting bilirubin the liver is capable of eliminating various dyes or drugs by the same excretory pathway as bilirubin.

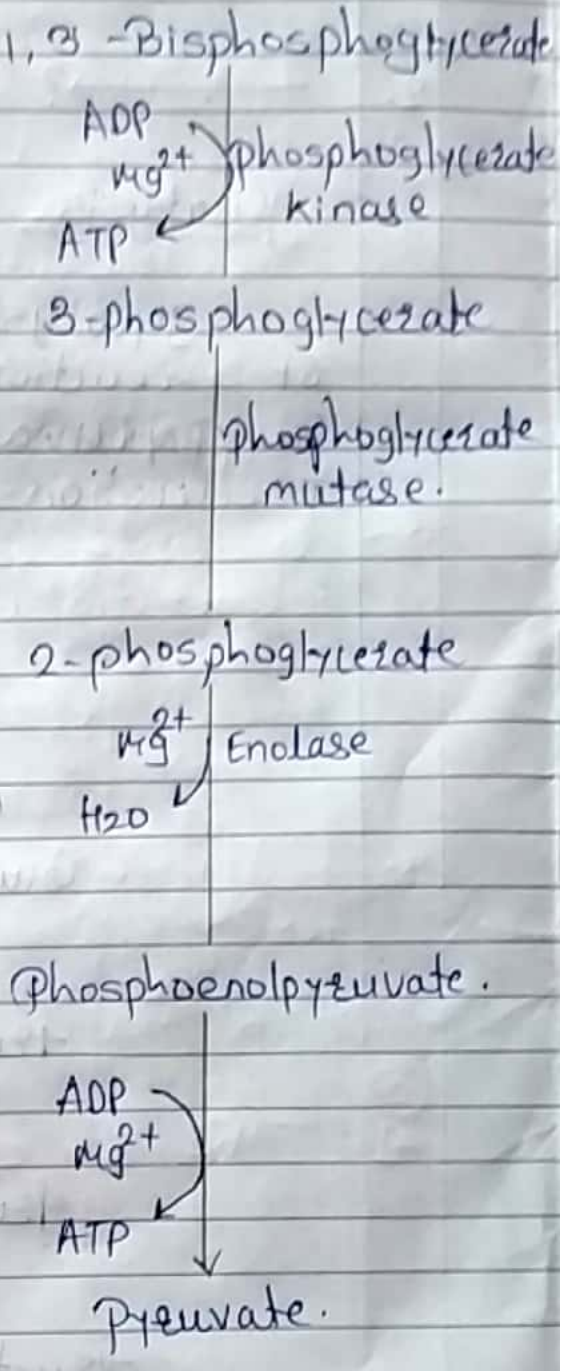
Bromosulfophthalein excretion Test.
A 5% solution of BSP is injected intravenously and a sample of blood is tested 45 minutes later for percentage of injected dye remaining in the blood.

2. ——————>

glycolysis

glycolysis is defined as the sequence of reactions converting glucose to pyruvate or lactate with production of ATP.





The pathway can be divided into three distinct phases.

A. Energy investment phase or priming stage
B. Splitting phase
C. Energy generation phase.

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PAGE NO.
DATE / /20

① Energy investment phase

1. glucose is phosphorylated to glucose 6-phosphate by hexokinase or glucokinase.

This is an irreversible reaction. dependent on ATP and Mg^{2+}.

2. Glucose 6-phosphate undergoes isomerization to give fructose 6-phosphate in the presence of the enzyme phosphohexose isomerase and Mg^{2+}.

3. fructose 6-phosphate is phosphorylated to fructose 1,6-bisphosphate by phosphofructokinase.

②

4] The six carbon fructose 1,6 biphosphate is split to two three-carbon compounds, glyceraldehyde 3-phosphate and dihydroxyacetone phosphate by the enzyme aldolase.

5] The enzyme phosphotriose isomerase catalyses the reversible interconversion of glyceraldehyde 3-phosphate and dihydroxyacetone phosphate. thus, two molecules of glyceraldehyde 3-phosphate are obtained from one molecule of glucose.

C. Energy generation phase

6 Glyceraldehyde 3- phosphate dehydroge -nase converts glyceraldehyde 3-phos -phate to 1,3- bisphosphglycerate. This step is important as it is involved in the formation of NADH + H^+ and a high energy compound 1,3- bisphosphate glycerate. Todoac -etate and arsenate inhibit the enzyme glyceraldehyde 3-phosphate dehydrogenase. In

M.V.P.S.
Dr.Vasantrao Pawar Medical College, Nashik
Department of Biochemistry
Compensatory Mid Terminal Exam 1st MBBS – 2018-19

Date : 24/04/2019

Time : 9:30 am to 10 am (Marks : 12+ 48)

Instruction: - 1. Every question carries ¼ Marks.
2. Avoid overwriting or striking of answer once written.
3. MCQ answer sheet will be collected exactly after 30 Min.

Section –A

Q.1 Multiple choice questions.

1) The glycosaminoglycan which serves as an anticoagulant is :-
a) Heparin
b) Hyaluronic acid
c) Chondroitin sulphate
d) Dermatan sulphate

2) ($\alpha_1 \rightarrow \beta_2$) Glycosidic bond is present in :
a) Maltose
b) Lactose
c) Sucrose
d) Glycogen

3) The lipoprotein which carries dietary triglycerides from intestines to the liver is :-
a) Chylomicrons
b) VLDL
c) LDL
d) HDL

4) Respiratory distress syndrome (RDS) is caused by defect in synthesis of :-
a) Phosphatidyl ethanolamine
b) Phosphatidyl serine
c) Dipalmitoyl lecithin
d) Phosphatidyl inositol

5) All of the following are methods of plasma protein seperation, EXCEPT :-
a) Salting out
b) Cohn's fractionation
c) Chromatography
d) Electrophoresis

6) Ketogenic amino acid is:-
a) Glycine
b) Histidine
c) Leucine
d) Methionine

7) Bence Jones proteins are excreted in :-
a) Nephrotic syndrome
b) Multiple myeloma
c) Nephritis
d) Liver cirrhosis

8) One of the following proteins is NOT present in serum :-
a) Albumin
b) Prothrombin
c) α_1 antitrypsin
d) Ceruloplasmin

9) Immunoglobulin that crosses the placenta is :-
a) Ig A
b) Ig M
c) Ig G
d) Ig D

10) The earliest biomarker to increase in serum after myocardial infarction is:-
a) Lactate Dehydrogenase
b) Creatine kinase-2
c) Alanine Transaminase
d) Aspartate Transaminase

11) Diagnostic enzyme for hepatitis is:-
a) Lipase
b) Amylase
c) Alanine transaminase
d) Acid phosphatase

12) The DNA strands are held together by :-
a) Phosphodiester bond
b) Hydrogen bond
c) Glycosidic bond
d) Van der Waals forces

13) Following are the examples of high energy compounds, EXCEPT :-
a) ATP
b) Phosphoenol pyruvate
c) Creatine Phosphate
d) AMP

14) Naturally occurring uncoupler is :-
a) Dicumarol
b) 2, 4 Dinitrophenol
c) Thermogenin
d) Dinitrocresol

15) Which one of the following vitamins have antioxidant property?
a) Vitamin K
b) Riboflavin
c) Tocopherol
d) Cholecalciferol

16) Deficiency of intrinsic factor of Castle leads to :-
a) Iron deficiency anaemia
b) Pernicious anaemia
c) Macrocytic anaemia
d) Microcytic anaemia

17) Biotin is involved in :-
a) Oxidation-Reduction
b) Carboxylation
c) Decarboxylation
d) Dehydration

18) Pantothenic acid forms a coenzyme which is :-
a) PLP
b) Cobamide
c) Coenzyme A
d) Coenzyme Q

19) Wernicke Korsakoff syndrome in alcoholics is caused by the deficiency of :-
a) Niacin
b) Retinol
c) Thiamine
d) Riboflavin

20) The functionally active form of vitamin D is :-
a) Cholecalciferol
b) Ergocalciferol
c) Dehydrocholesterol
d) Calcitriol

21) All of the following vitamins have antioxidant property, EXCEPT:-
a) Carotene
b) Ascorbic acid
c) Tocopherol
d) Cholecalciferol

22) The form of vitamin A present in rhodopsin is :-
a) All trans retinol
b) 11-cis retinol
c) All trans retinal
d) 11-cis retinal

23) The defective enzyme in Alkaptonuria is :-
a) Tyrosinase
b) Tyrosine transaminase
c) Phenylalanine hydroxylase
d) Homogentisate oxidase

24) The amino acid required for synthesis of heme is :-
a) Lysine
b) Glycine
c) Glutamine
d) Glutamic acid

M.V.P.S.
Dr.Vasantrao Pawar Medical College, Nashik
Department of Biochemistry
Compensatory Mid Terminal Exam 1st MBBS – 2018-19

Date: 24/04/2019

Time:09:30 to 12:30 pm **(Marks: 12 + 48)**

Section – B

Q.2. Short answer questions (Any six) **(6 x 4 = 24)**

a) What are heteropolysaccharides? Describe their functions with examples.

b) Structure and functions of RNA.

c) Diagnostic significance of enzymes.

d) Co Enzymes

e) Essential fatty acids.

f) A 15 year old boy presented with acute abdominal pain in emergency. His investigations showed Hb – 9.1gm% and presence of sickle shaped RBCs on blood smear.

1) Name the clinical condition.

2) Mention the molecular defect involved.

3) Explain the biochemical basis of the sickling of RBCs.

4) Name the other lab investigation to confirm the diagnosis.

g) Phospholipids.

Section – C

Q.3 Long answer questions (Any three) **(3 x 8 = 24)**

1) Define and Classify vitamins. Describe Vitamin D under following headings.

-- Biochemical functions, Deficiency manifestations, RDA & Sources.

2) Formation and fate of ammonia. Add a note on congenital metabolic disorders of urea cycle.

3) Define enzyme classify them with examples. Add a note on competitive type of enzyme inhibition.

4) Describe ETC in detail. Add a note on sites on ATP synthesis & inhibiters of ETC.

Dr. Vasantrao Pawar Medical College, Hospital and Research Centre Adgaon Nashik

Department of Biochemistry

Mark list of Compensatory Terminal Examination (Theory) 2018 -2019

Roll no.	Name of the Students	Q. A 12	Q. B 24	Q. C 24	TOTAL 60
24	DEVAKATE MANASI ANANDRAO	9.5	15.5	14	39
47	JAIN SHRISHTI ASHOK KUMAR	8.5	13	14	35.5

HOD

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Dr. Vasantrao Pawar Medical College, Hospital and Research Centre Adgaon N

Department of Biochemistry

Mark list of Compensatory Terminal Examination (Practical) 2018 -2019

Dated on 24/04/2018

Roll no.	Name of the Students	Q. A 20	Q. B 15	Q. C 5	TOTAL 40
47	JAIN SHRISHTI ASHOK KUMAR	13	10	5	28

HOD

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M.V.P.S.
Dr.Vasantrao Pawar Medical College, Nashik
Department of Biochemistry
Compensatory Mid terminal Exam MBBS – 2016-17

Date : 07 /04/2017

Time : 10 to 1 (Marks : 12+ 48)

Instruction: - 1. Every question carries ½ Marks.
2. Avoid overwriting or striking of answer once written.
3. MCQ answer sheet will be collected exactly after 30 Min.

Section –A

Q.1 Multiple choice questions.

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   a) Heparin
   b) Hyaluronic acid
   c) Chondroitin sulphate
   d) Dermatan sulphate

2) ($\alpha_1 \rightarrow \beta_2$) Glycosidic bond is present in :
   a) Maltose
   b) Lactose
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   d) Glycogen

3) The lipoprotein which carries dietary triglycerides from intestines to the liver is :-
   a) Chylomicrons
   b) VLDL
   c) LDL
   d) HDL

4) Respiratory distress syndrome (RDS) is caused by defect in synthesis of :-
   a) Phosphatidyl ethanolamine
   b) Phosphatidyl serine
   c) Dipalmitoyl lecithin
   d) Phosphatidyl inositol

5) All of the following are methods of plasma protein seperation, EXCEPT :-
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   b) Cohn's fractionation
   c) Chromatography
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   a) Glycine
   b) Histidine
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   a) Nephrotic syndrome
   b) Multiple myeloma
   c) Nephritis
   d) Liver cirrhosis

8) One of the following proteins is NOT present in serum :-
   a) Albumin
   b) Prothrombin
   c) α_1 antitrypsin
   d) Ceruloplasmin

9) Immunoglobulin that crosses the placenta is :-
   a) Ig A
   b) Ig M
   c) Ig G
   d) Ig D

10) The earliest biomarker to increase in serum after myocardial infarction is:-
   a) Lactate Dehydrogenase
   b) Creatine kinase-2
   c) Alanine Transaminase
   d) Aspartate Transaminase

11) Diagnostic enzyme for hepatitis is:-
a) Lipase
b) Amylase
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12) The DNA strands are held together by :-
a) Phosphodiester bond
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d) Van der Waals forces

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a) Dicumarol
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c) Thermogenin
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a) PLP
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a) Niacin
b) Retinol
c) Thiamine
d) Riboflavin

20) The functionally active form of vitamin D is :-
a) Cholecalciferol
b) Ergocalciferol
c) Dehydrocholesterol
d) Calcitriol

21) All of the following vitamins have antioxidant property, EXCEPT:-
a) Carotene
b) Ascorbic acid
c) Tocopherol
d) Cholecalciferol

22) The form of vitamin A present in rhodopsin is :-
a) All trans retinol
b) 11-cis retinol
c) All trans retinal
d) 11-cis retinal

23) Scarvy is due to deficiency of which one of the following vitamin. :-
a) Vitamin A
b) Vitamin D
c) Vitamin K
d) Vitamin C

24) The amino acid required for synthesis of heme is :-
a) Lysine
b) Glycine
c) Glutamine
d) Glutamic acid

M.V.P.S.
Dr.Vasantrao Pawar Medical College, Nashik
Department of Biochemistry
Supplementary Terminal Exam 1st MBBS – 2016-17

Date: 07/04/2017

Time: 10:00 to 1:00 pm **(Marks: 12 + 48)**

Section – B

Q.2. Answer the following short questions (Any six) **(6x4 =24)**

a) Essential fatty acids

b) Hetropolysaccharides.

c) t RNA

d) Iso enzymes & their diagnostic significance.

e) Rickets

f) ETC

g) Functional classification of plasma protein.

Section – C

Q.3 Answer the following long questions (Any three) **(3 x 8 = 24)**

A) Define and Classify vitamins. Describe Vitamin A under following headings.

-- Biochemical functions, Deficiency manifestations, RDA & Sources.

B) Define & describe TCA. Explain amphoteric nature of TCA cycle. Add note on energetic.

C) Define Enzyme. Describe factors affecting enzyme activity. Add note on competitive enzyme inhibition with examples.

D) Define Phospholipids. Describe classification phospholipids with examples. Add a note on functions of phospholipids.

M.V.P.S.Dr.Vasantrao Pawar Medical College Adgaon Nashik

Department of Biochemistry

Mark list of Compensatory Terminal Examination (Theory) 2016 -2017

Roll no.	Name of the Students	Q. A 12	Q. B 24	Q. C 24	TOTAL 60	Sign.
2	AGRAWAL AASTHA MUKESH	6.5	16	18	40.5	
105	SHINDE MANALI MADHUKAR	8.5	12	18	38.5	
115	THAKUR MAHIMA MANOJ	10.5	18	15	43.5	

HOD

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Adgaon, Nashik-

M.V.P.S.Dr.Vasantrao Pawar Medical College Adgaon Nashik

Department of Biochemistry

Mark list of Compensatory Terminal Examination (Practical) 2016 -2017

Roll no.	Name of the Students	Q. A 20	Q. B 15	Q. C 5	TOTAL 40	Sign.
2	AGRAWAL AASTHA MUKESH	15	11	5	31	
105	SHINDE MANALI MADHUKAR	12	10	3.5	25.5	

HOD

Department of Biochemistry

Professor & Head
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Medical College, Hospital & RC
Adgaon, Nashik-422 003

Name: Aastha Agrawal

Roll No.: 02 Date: 7/4/2017

Examination: Biochemistry Practical

Student's Sign.: Aastha

7.4.17

1

Estimate Blood Glucose level in the given sample. **20**

Perform any five tests on precipitation reactions of proteins. **15**

Spots **05**

~~Spots~~

~~Note~~

Quantitative

- To estimate blood glucouse level by glucose oxidase/ peroxidase method.

s methods - Folin wu method.

mple collection - collect sample in flouride bulb with anticoagulant.

ample used - plasma.

rinciple -

Glucose oxidase acts on a sp specific substrate of B-D glucose to form glucoronic acid and hydrogen peroxide is released. Hydrogen peroxide gets acted upon by

peroxide to form nascent oxygen. This nascent oxygen couples with phenol and 4 aminoantipyrine to form red quioneamine dye. ~~which is~~ The intensity of colour is directly proportional to concentration of glucose in blood. It is estimated colorimetrically at 530 nm and compared with a glucose standard which is treated similarly.

- Observations.

	O.D.
Standard	0.21
Blank	0.02
Unknown.	0.23

- Calculations -

$$= \frac{O.D_U - O.D_B}{O.D_S - O.D_B} \times \text{conc. of std.} \times \text{Vol. of std.} \times \frac{100}{\text{Vol. of plasma}}$$

$$= \frac{U - B}{S - B} \times 100$$

$$= \frac{0.23 - 0.02}{0.21 - 0.02} \times 100$$

$$= \frac{0.21}{0.19} \times 100$$

$$= \frac{21}{19} \times 100$$

Blood glucose = 110 mg/dL

Normal range of blood glucose is 80-120 mg/dL.
Fasting range is 70-110 mg/dL.

Result = Blood glucose is 110 mg/dL.

Interpretation
Upper Hyperglucaemia - diabetes mellitus.
Hypoglucaemia - diabetes insipidus.

7.4.17

2] Qualitative.

Test	Observation.	Inference.
Alkaloidal test 2mL O.S. + 2mL sulphosalicylic acid.	white ppt.	Protein is present.
Alkaloidal test 2mL O.S + 1% acetic acid.	white ppt.	Protein is present.
Alcohol test. 2mL O.S. + 1 mL absolut alcohol	Scanty white ppt.	Protein is present.
~~Lead Acetate Test.~~ 2mL O.S + lead acetate.	white ppt.	Protein is present.
Heat Coagulation Fill 2/3rd of test tube. & Heat. Add glacial acetic acid.	upper part show protein coagulation.	Protein is present.

7.4.17

Name: ~~Aastha Agrawal~~

Roll No.: 02 Date: 7/4/17

Faculty:

Examination: Biochemistry Practical

Student's Sign.: ~~Astha~~

5

Fehl's Test.

is a group test for carbohydrates.

idine Reagent.

for detection of blood in abnormal urine.

meter.

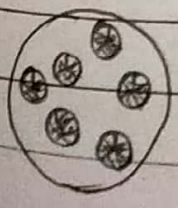
for determination of pH.

glucose - Normal

blood glucose ~~of~~ is 80-140 mg/dL.

is 70-110 mg/dL.

azone.



Scanned with CamScanner

Lastha Agrawal

Compensatory Exam

MVP's Dr. Vasantrao Pawar Medical College Hospital & Research Centre

Vasantdada Nagar, Adgaon, Nashik-3.

Examination : Terminal

Subject : Biochemistry

Section : PAPER :

Language of Answer : English

Serial No. 1754

Date : 07 04 2017

P.Roll No. 02 (in figure)

Pledge : I hereby declare that I have gone through the "Special Instructions to Candidate" printed on page number two and my Roll No., PRN Subject printed / written on page no. One of the Anwer Booklet. I also know that no supplement will be provided to me.

Lastha

Signature of the CANDIDATE

RDKalav 7/4/17

Signature of the JUNIOR SUPERVISOR

Lastha

Seen, Verified
No Complaints

Que	A	B	C	D	E	F	G	H	I	J	K	L	M	N	O	Total
1	MCQ															06.5
2	3	3.5	2	2.5	2.5		2.5									18
3	6		6	6												
4																
5																
6																
7																
8																
9																
10																

TOTAL → 40.50

Marks allotted By Examiner

Signature :

Name of Teacher

No supplements shall be provided to any candidate in any case

Lastha Agrawal

SECTION-B

Q.2] Short Questions.

a] Essential fatty acids.

- Essential fatty acids are those compounds which are needed in our diet but cannot be synthesised by the body, hence they have to be provided by the diet.
- Examples - linoleic acid, linolenic acid and arachadonic acid.
- Linoleic acid is a precursser of arachadonic acid and arachadonic acid becomes an essential fatty acid only when there is deficiency of linoleic acid.
- These fatty acids have become essential because the human body does not have the enzyme needed for the introduction of double bond beyond 9 to 10 carbon atoms. Hence they have to be provided through the diet.
- Functions of essential fatty acids-
  i) Help is transport of cholesterol.
  ii) Formation of lipoproteins.
  iii) Prevention of a fatty liver.
  iv) Formation of other compounds like ecasanoids.
  v) They form an important component of the cell

membrane structure.

⇒ Deficiency

- Deficiency of these fatty acids cause phrynoderma, or toad skin.
- The limbs show horny eruptions on the posterior aspects.
- Wound healing becomes slow.
- Hair loss is observed.
- There is loss of apetite.

b] Heteropolysaccharides.

- These are carbohydrate compounds.
- Polysaccharides are divided into 2 classes -
  i) Homopolysaccharides
  ii). Heteropolysaccharides.
- Heteropolysaccharides are composed of different single units and on hydrolysis give various different singular units.

i) Starch.
- It is formed by amylose and amylopectin.
- Amylose forms the straight chains
- Amylopectin is needed for branching of the compound.

Amylose shows $\alpha(1\rightarrow4)$ glycosidic linkage whereas amylopectin shows $\alpha(1\rightarrow6)$ glycosidic linkage.

Starch is present in potato and rare rice and various other food substances.

Their digestion starts in the mouth by the activity of salivary amylase.

Glycogen

Heteropolysaccharides are also called glycosaminoglycan.

They contain mucous and therefore also called as mucoproteins.

Contain, ~~amino~~ amino sugars and uronic acid.

mucopolysaccharides are GAGs (glycosaminoglycan).

Hyaluronic acid.

Formed form D-glucoronic acid and N-acetylglucosamine units.

Present as synovial fluid, vitreous humor.

The hyaluronic acid provides lubrication ~~to the~~ of the structures and prevents friction.

Chondroitin sulphate.

D-glucoronic acid and N-acetylgalatosamine 4 sulfate units form chondroitin sulphate.

Found in collagen fibres, bones, cells.

It helps in keeping the shape of the structure maintained.

iii) Dermatan sulphate.

- L-Iduronic acid and N-acetylgalactosamine units form dermatan sulphate.
- Seen in skin, vessels.
- It too provides mechanical support by maintaining the shape of the cells.

iv) Keratan sulphate.

- It is found in cornea
- It maintains the clarity of cornea.

v) Heparin.

- Found in blood
- It plays an important role in the anticoagulation of blood inside the vessels and hence prevents blockage of blood vessels.

SECTION-C

3] A] • Vitamins are organic compounds needed in small quantities in the body for basic ~~biogt~~ biological functioning and growth of the body.

• Classification -

i) Fat Soluble ii) Water Soluble.

i) Fat soluble vitamins -
- Vit. A
- Vit. D
- Vit. E
- Vit. K

ii) Water soluble vitamins -
a) energy releasing
b) non-energy releasing.

a) energy releasing -
- Vit B_1 (thiamine)
- Vit. B_2 (riboflavin)
- Vit. B_3 (niacin)
- Vit. B_5 (panthothenic acid)
- Vit. B_6 (pyridoxne)
- Vit. B_7

b) non energy releasing -
- Vit. C
- Vit. B_{12}
- Vit. B_9

Vitamins

- Fat soluble
  - Vit. A
  - Vit. D
  - Vit. E
  - Vit. K
- water soluble
  - energy releasing B-complex vitamins,
    - B_1
    - B_2
    - B_3
    - B_5
    - B_6
    - B_7
  - Others
    - Vit. C (ascorbic acid)
    - Vit. B_{12} (cobalamine)
    - Vit. B_9

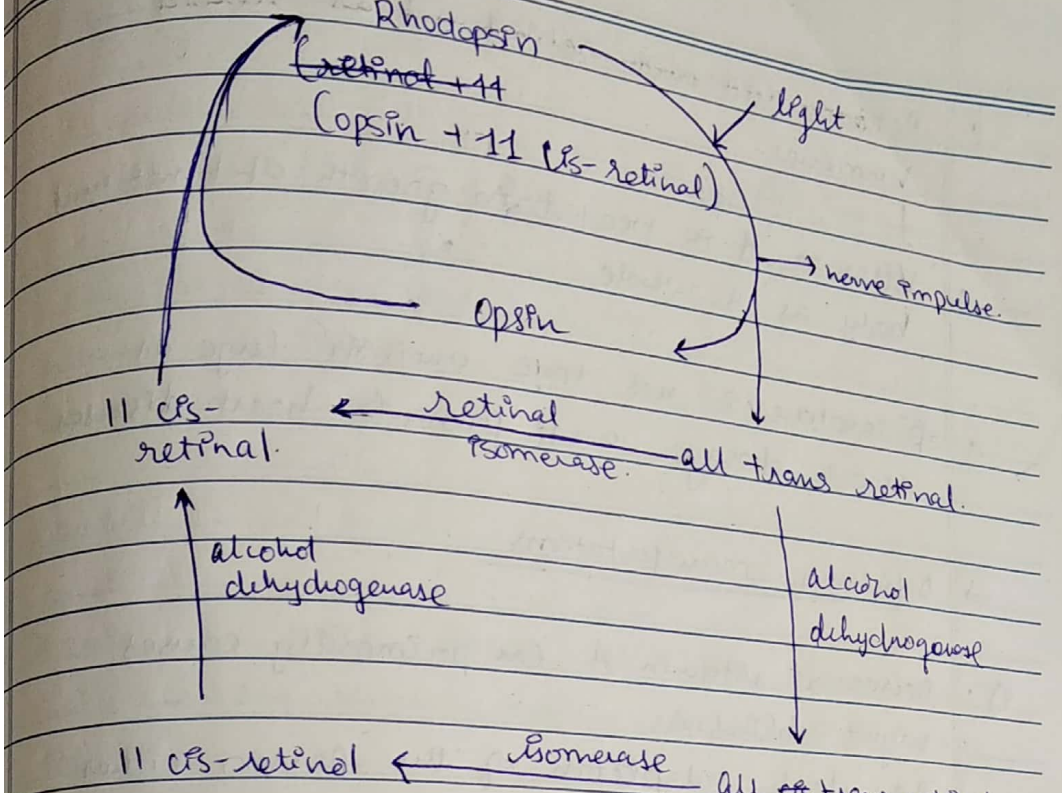
* Vitamin A *

- Vitamin A is a fat soluble vitamins.
- Retinol, retinal and retinoic acid ~~the~~ are the vitamers of vit. A.

⇒ Biochemical functions.

i) . Vitamin A forms a very important component is the visual cycle.
- Dark adaptation of eyes due to rods is done with the help of vit. A.
- Rods ~~produce~~ help in dark vision while cones provide for coloured vision.
- ~~Ro~~ Rhodopsin contains opsin and 11 cis-retinal which helps in the vision.
- Wald's cycle or visual pathway.

~~Asthma Asthmol~~



Due to light, nerve impulse gets generated as opsin breaks from rhodopsin and causes a conformational change in the retinal unit.

This all trans-retinal immediately forms 11 cis-retinal and couples with opsin to again form rhodopsin.

Not all retinal is converted to 11 cis retinal, some is converted to ~~11~~ all trans retinol and further steps of visual cycle occur to finally form ~~Athop~~ rhodopsin.

β-carotene of vitamin A is a strong antioxidant.

Vitamin A helps in maintaining proper health of epithelial cells. ~~And~~

- Retinoic acid and retinol act as Steroid hormones.
- Vitamin A is needed in growth of bones and body as a whole.
- β carotene is not toxic even in large or excess dosage and prevents heart diseases.

⇒ Deficiency manifestations

i) • Decreased vitamin A ~~fat~~ primarily causes night blindness.
- The dark adaptation of the person either increases or ~~do~~ stops existing.
- vitamin A is needed for rhopsin and hence its deficiency initially causes problem in seeing at night or in dark.

ii) • If not cured, it causes xerophthalmia.
- Conjuctiva starts to dry and there are visual problems. Degradation occurs.

iii) • Keratomalacia exists in cases of severe deficiency and not treatment.
- There is conjuctival ~~keratina~~ keratinisation which may lead to opacity and hence total blindness in the eyes.

RDA (recommended dietary allowance).

For males ~~it is~~ RDA is 1000 RE (retinol equivalents).

For females RDA is 800 RE

During pregnancy and lactation the need for vitamin A increases and the RDA ~~also~~ shows an increase.

Sources

Vitamin A rich foods are liver, kidney, carrot. Other accessory sources include milk, cereals, pulses, meat etc.

C]. Enzymes are protein biocatalysts which are thermolabile and help in the normal functioning of the body.

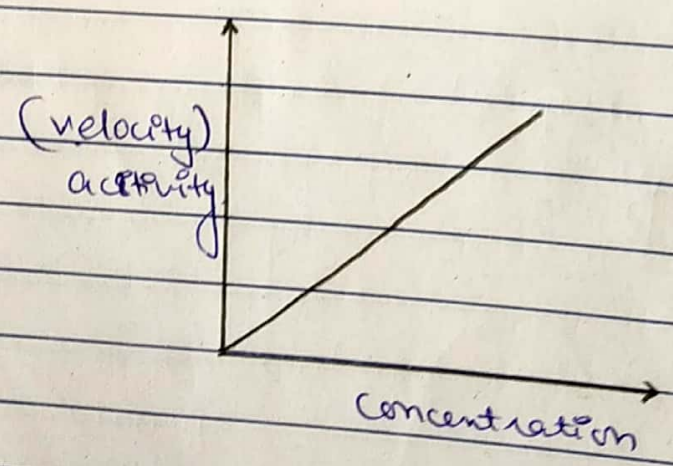
- Ribozyme is an enzyme which non proteinous in nature.

⇒ Factors affecting enzyme activity.

*) Enzyme activity depends on many factors ~~for~~ as the following -

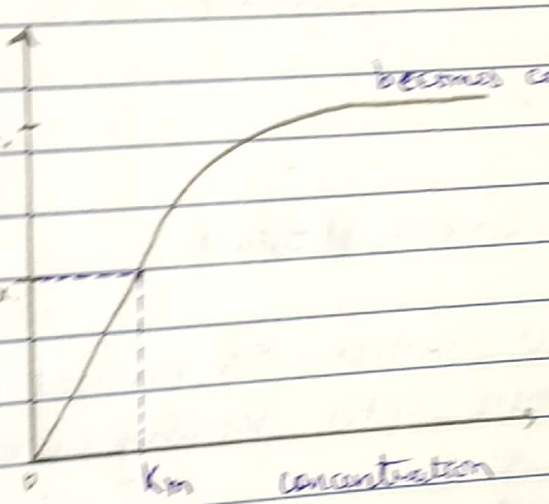
P) Enzyme concentration.

- Enzyme concentration is directly proportional to the rate of activity.
- More enzymes lead to more number of active sites and hence increased activity.



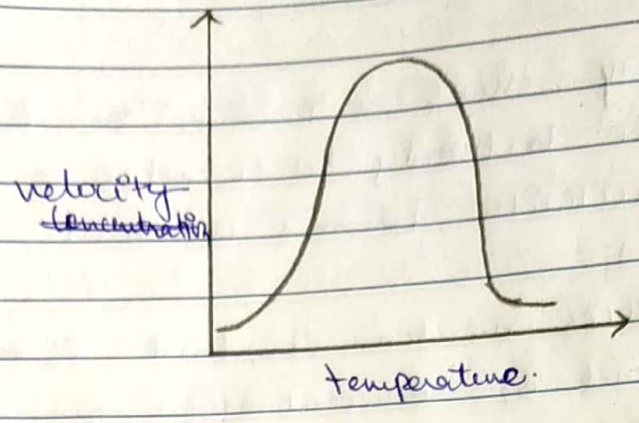
iii) Substrate concentration

- Enzyme activity shows an increase due to more substrate but only to a certain level after which the activity becomes constant.
- Km or Michelles menten constant is defined as the amount of concentration produced at half of the maximum velocity
- Graph is a rectangular parabola.



iv) Effect of temperature

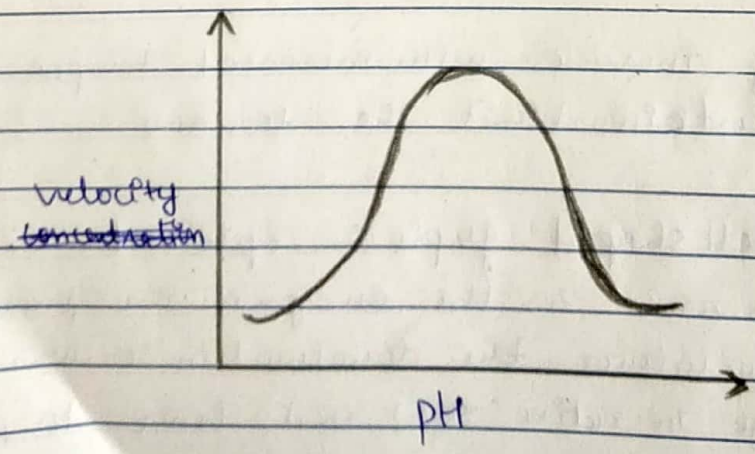
- Activity increases with increased temperature upto a certain limit the decrease
- Bell shaped graph is obtained
- Activity decreases as the enzymes are proteins and at high temperatures the denaturation occurs which causes change in active sites and hence to prevent denaturation, activity decreases.



- Q_{10} is the value which shows the amount of activity increased by increasing 10°C of the temperature.

iv) Effect of pH.

- Bell shaped curve is obtained.
- Activity first increases with increased pH and then decreases after reaching an optimum.
- Mostly neutral and slightly alkaline pH is optimum for enzyme activity.



v) Effect of products formed.

- When the products keep getting formed continously, to maintain equilibrium, the reaction move towards left.
Hence activity of enzyme is reduced.

- Product formed should be removed to maintain the reaction in forward / right direction.

vi) Metal activators

- Certain enzymes need metal ions for their functioning.
  - metalloenzymes.
    cytochrome.
  - aldolase needs Mg^{2+} for action - metal activator.

vii) Effect of time.

- If all other factors are constant, the ~~fast~~ reaction proceeds fast and less time is required.

viii) Effect of UV lights.

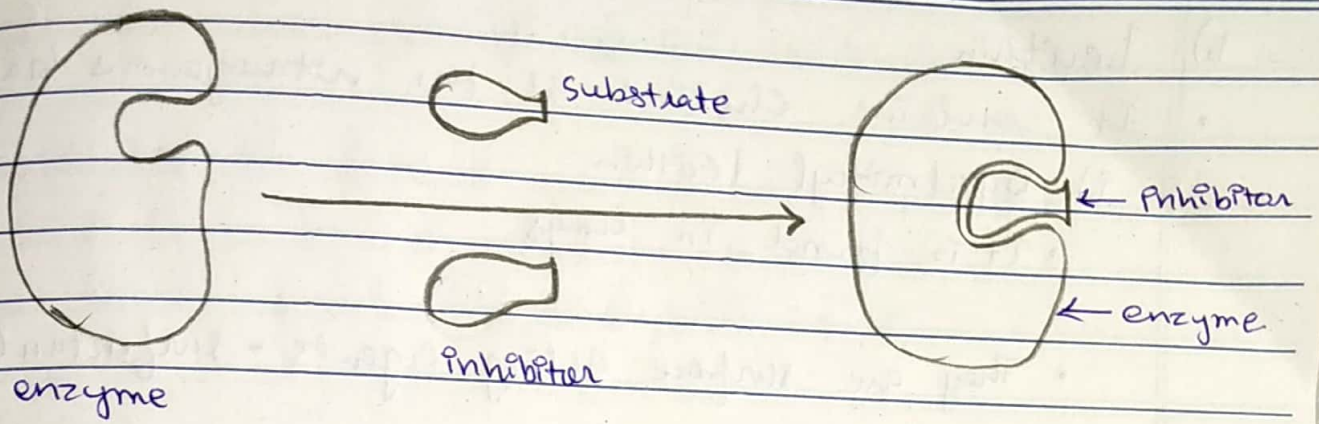
- Activity is increased on exposure to direct light.

Competitive Enzyme Inhibition

- Inhibitors are substances which decrease the enzymatic activity.
- Competitive inhibition is a type of reversible inhibition where the inhibitor binds non-covalently and the complex formed and reaction occured can be reversed.
- In competitive inhibition, the inhibitor is a structural analog of the substrate and hence finds it easy to get attached to the active site.
- The K_m value in competitive inhibition increases while the V_{max} value remains unchanged.
- Example -

i) For succinate dehydrogenase enzyme, succinate is the substrate but malvonic acid acts as the inhibitor.

ii) Ethanol is used in methanol poisoning as it is a structural analog of methanol and competitively binds at the site of alcohol dehydrogenase.



Competitive Enzyme Inhibition.

D] PHOSPHOLIPIDS

- Phospholipids are compounds lipids found in the body which have fatty acids with alcohol and presence of a prosthetic group of phosphorous.

- Classified as -
  i) Glycerophospholipids
  ii) Sphingophospholipids.

i) Glycerophospholipids.

- These contain the alcohol glycerol and hence the name.
- They are abundantly present in the body.

a) Phosphatidic acid.

- This forms the main derivative of the other glycerophospholipids.
- Contains nitrogenous base.

b) Lecithin

- It contains Choline as the nitrogenous base

i) Dipalmitoyl lecithin

- It is found in lungs
- They are surface acting agents - surfactants.
- Deficiency of dipalmitoyl lecithin cause RDS - respiratory distress syndrome. in the newborns.
- It prevents the collapse of the ~~to~~ lungs and hence is a necessary component.

ii) Lysolecithin

- It shows change in carbon C_1 and C_2.

c) Cephalin

- It is similar to lecithin but with a change in the base
- It contains ethanolamine as the nitrogenous base.

d) Plasmalogens

- They are esters ~~compounds~~ compounds.
- They are plasmatidalesters which are similars to ~~plasmatdit~~ plasmatidylesters but only with an ester &

. They also have ethanolamine as their base

e) Cardiolipin

. These were first found in the heart and hence the name.

. They are the only antigenic phospholipid available naturally.

. They present disorders of the heart

f) Sphingophospholipid.

. They contain sphingosine as the alcohol

. ⇒ Sphingomyelins are very abundant in the body They are needed for myelination of the axon fibres hence play an important ~~&~~ role.

. Ceramides is another example of sphingolipids.

⇒ Functions of phospholipids.

Phospholipids are very largely used in human body.

i) Main component of ~~m~~ cell membrane structure.

ii) Some have antigenic property, which helps in defence ~~u~~ of body against foreign antigens and

therefore provides immunity.

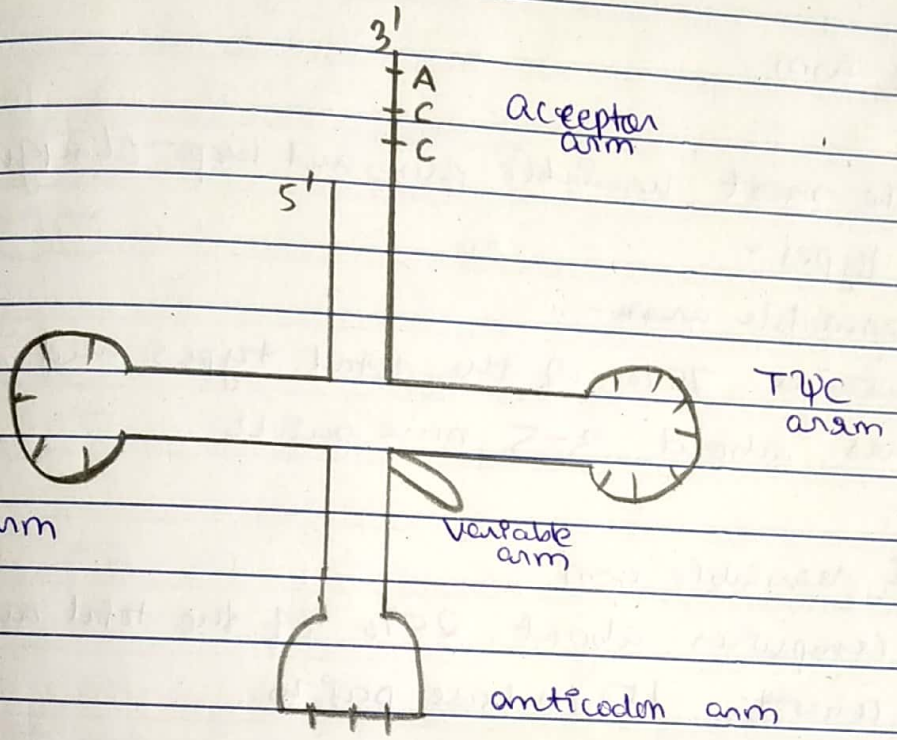
iii) Phospholipids acting as surfactant decrease lung surface tension in lungs and it prevents collapse of the lung alveoli.

iv) Myelination of nerve fibers is one very important function of the phospholipids.

Section - B

Q) t-RNAs.

- Transfer RNA or t-RNA are present in about 20 species for the transfer of amino acids.



- The t-RNA is a soluble RNA.
- It has 5 arms which have various different uses -

i) Acceptor arm.

- It is marked CCA (5'→3') and ~~acc~~ is the acceptor site for amino acids.

ii) Anticodon arm.

- It ~~has~~ receives the ~~an~~ 3 base pairs which are transfered later.

iii) D arm

- It is so named due to dihydrouridine presence

iv) TΨC arm

- Psendouridine presence is seen here.

v) Variable arm

- This is the most variable arm and keeps changing.
- Has 2 types -

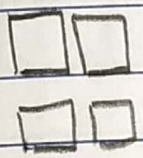
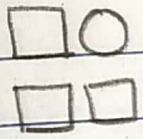
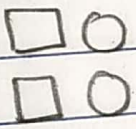
a) Type I variable arm -
   - contains 75% of the total types and has about 3-5 base pairs.

b) Type II variable arm
   - comprises about 25% of the total and consists 15-20 base pairs

d] • Isoenzymes are forms of enzymes which catalyse the same reactions but are chemically and physically different.

i) Lactate dehydrogenase.

• Tetrameric enzyme, shows 5 isoenzymes.

		Present
LH_4		Heart and RBC
L_1H_3		RBC and heart
L_2H_2		Brain & kidney
L_3H		Liver and skeletal muscle
L_4H		Liver and skeletal muscle.

ii) Creatine phosphokinase

• It is a dimeric form.

• It is an early marker for myocardial infarction.

CPK-1	BB	Brain
CPK-2	BM	Heart
CPK-3	MM	Skeletal muscle

iii) Alkaline phosphatase.

- It has 6 isoenzymes, which differ due to carbohydrates.
- It helps in diagnostic function of obstructive jaundice and rickets.

e] Rickets

- It is a disorder seen in children due to deficiency of vitamin D.
- In adults, osteomalacia is observed.
- Rickets shows bowing of legs as a very prominent sign.
- long bones which take body pressure become weak.
- Vitamin D is need for calcium, phosphate levels in body.
  Since the deficiency these levels gets disturbed.

Alkaline phosphatase is used for diagnostic function of rickets.

Weakness and loss of apetite are other sign signs and symptoms.

Proper administration of Vitamin D restores the body calcium and phosphate levels.

Calcitriol is the active form of Vitamin D and if it decreases in plasma, parathyroid hormone increases and hence the levels of calcium in bones, intestine and plasma increase.
But due to deficiency of Vitamin D, proper calcium uptake is not possible and hence disorders occurs.

Functional classification is as follows-

- contractile proteins
  - actin, myosin
- genetic proteins
  - nucleoprotein.

- structural proteins
  - albumin, globulin
- transport proteins
  - haemoglobin
- membrane proteins
  - lipoproteins, phosphoproteins

i) Contractile proteins help in contraction and relaxation of muscles, hence ~~@~~ bringing about movement.

ii) genetic proteins are needed for basic transfer to information.

iii) Structural protein show globular, fibrous forms

iv) Transport protein carry ~~substan~~ substances from one area to another through blood in the body

v) Membrane protein provide structural shape and maintains the support needed for membrane structure.

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MCQ ANSWER SHEET

Name of Candidate : Aastha Agrawal.
Name of Examination : Terminal (Compensatory)
Class : 1st MBBS Semester :
Subject : Biochemistry Paper :
Date : 7/4/17

ROLL NO.

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0	■	□	□	□	□	□
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4	□	□	□	□	□	□
5	□	□	□	□	□	□
6	□	□	□	□	□	□
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QUESTION :→	1	2	3	4	5	6	7	8	9	10
ANSWER :↓										
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B	□	□	□	□	□	■	□	□	□	■
C	□	■	□	■	■	□	□	■	■	□
D	□	□	■	□	□	□	□	□	□	□

QUESTION :→	11	12	13	14	15	16	17	18	19	20
ANSWER :↓										
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B	□	□	□	■	□	■	□	□	□	□
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D	■	□	□	□	■	□	□	□	□	■

QUESTION :→	21	22	23	24	25	26	27	28	29	30
ANSWER :↓										
A	□	□	□	□	□	□	□	□	□	□
B	□	□	□	■	□	□	□	□	□	□
C	■	□	□	□	□	□	□	□	□	□
D	□	■	■	□	□	□	□	□	□	□

ROLL NO. (In Words)
Two

QUESTION BOOKLET VERSION :

This is to certify that the entries of Roll No., Question Booklet Version, Question Booklet Sr. No. & Subject have been verified.

CANDIDATE SIGNATURE

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INSTRUCTIONS

1. FILL THE BLOCKS ☐ USING BLUE BALLPOINT PEN ONLY.
2. FILL ONLY ONE BLOCK FOR EACH QUESTION AS SHOWN BELOW.

Wrong | Correct

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4. ROUGH WORK MUST NOT BE DONE ON THIS ANSWER SHEET.
5. USE FREE SPACE IN THE QUESTION BOOKLET PROVIDED.

TAL MARKS :-

6.5/12