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To,

The Coordinator,

NAAC, Bengaluru.

Subject: Proof of Criteria to identify low performers and advanced learners and assessment methodology & special programmes for slow performers.

Reference:

2.2.1 The Institution assesses the learning levels of the students, after admission and organizes special Programmes for advanced learners and low performers.

Dear Sir/Madam,

2.2.1 Consolidated report of special programs for advanced learners and slow learners duly attested by the Head of the Institution.

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Strategies adopted for student improvement:

1. Remedial classes are organized to clarify doubts.
2. Re-explaining of critical topics for improving performance.
3. Daily attendance is reported to the parents through SMS and Phone calls.
4. Motivational classes are conducted to improve the mental ability of students to analyze problems and to encourage student to regularly attend classes.
5. Additional details are given in each class regarding the topic.
6. Students can discuss their personal issues with teachers for proper guidance.
7. Career counseling sessions are provided to students regarding various fields in which pharmacy students can do wonders.

All the staff members maintaining good relation with students and dealing with their Problems in a gentle manner.

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Methods to Identify Advanced learners and Slow learners:

Continuous monitoring and evaluation of the students is used to identify the low and the advanced performer based on the following.

- Group, one to one interaction of students and teachers during daily course delivery and during lab hours.
- Performance of the students in the class test, viva or group activities
- conducted during laboratory hours.
- Performance of the student in the sessional examinations/ in-semester assessments.
- Continuous monitoring of academic performance of each student by
- mentors.
- Feedback from the faculty members

Steps for Advanced learners

Advanced learners are continuously encouraged to strive for higher goals by providing them additional inputs for better career planning and growth like:

- Conducting preparation classes and assessment modules for the GPAT examination
- Topics on content beyond syllabus are taught. Encouraging them to participate in paper presentations
- Encouraging them to participate in classroom seminars, group discussion s, technical quizzes etc. for developing analytical, problem solving and presentation skills
- Motivating to access latest online journals, reference materials and help them to understand

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the emerging trends in their field of study

- Providing opportunity to develop their creativity by organizing intercollegiate and state level cultural, literary, technical and sports competitions.
- Encouraging them to take specialized training through certificate courses.
- Appointing them as student representatives at the department level committees to develop leadership skills.

The Institute responds to the learning needs of the low performers by:

- Indemnification of the problems encountered during learning by the student through mentoringsessions.
- Counseling of the students and providing solutions for problems during mentoringsessions.
- Arranging tutorial classes within small group of students for fewdifficultsubjects.
- **Remedial Sessions:** Remedial sessions for slow learners are organized on a weekly basis for specific subjects in order to ensure that slow learners can also progress in the course and stay at par with others. Slow learners requiring more practice and focus on English communication and aptitude proficiency are given additional classes to improve them in theseaspects.
- Arranging extra preparative lectures before sessionalexamination.
- Providing course material and the question bank to thestudents.
- Daily writing practice sessions are conducted to enable them to learn better.

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REMEDIAL CLASS TIME TABLE

Time: 03:45 to 04:45pm

TIME / DAY	B.PHARMACY II YEAR	B.PHARMACY III YEAR		B.PHARMACY IV YEAR
MONDAY	Physical Pharmacy K.JANAKI	Pharmacology –I B.MAMATHA		Instrumental Method of Analysis G.ANUSHA
TUESDAY	Pharmaceutical Organic Chemistry-II G.SURESH KUMAR	Industrial Pharmacy MD.JAFFER		Industrial Pharmacy –II V.DURGA MADHURI
WEDNESDAY	Pharmaceutical Engineering V.SRILATHA	Pharmacognosy-I N.VIJAYA REKHA		Pharmacy Practice M.VIJAYA LAXMI
THURSDAY	Pharmaceutical Microbiology TAHMENA BEGUM	Medicinal chemistry R.SUNEETHA		Novel Drug Delivery System R.SATYASRI
FRIDAY	Pharmaceutical Organic Chemistry-II G.SURESH KUMAR	Generic Product Development G.JAIPAL		Pharmaceutical Regulatory Sciences P.VENKATESH
SATURDAY	Physical Pharmacy K.JANAKI	Environmental sciences CH. LAVANYA		Pharmacy Practice M.VIJAYA LAXMI

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REMEDIAL CLASS TIME TABLE

Time: 03:45 to 04:45pm

TIME / DAY	PHARM-D II YEAR	PHARM-D III YEAR		PHARM-D IV YEAR
MONDAY	Pharmacotherapeutics-I Dr.D.Naga Latha	Pharmacotherapeutics-II Dr.A.M.Billah		Pharmacotherapeutics-III Dr.Rohit Kumar
TUESDAY	Pathophysiology B.Mamatha	Pharmaceutical Analysis M. Kavya		Hospital Pharmacy Dr.K.Shiva kumar
WEDNESDAY	Pharmaceutical Microbiology A.Kesava	Pharmaceutical Formulations V.Sreelatha		Clinical Pharmacy Dr.Ashwanth rao
THURSDAY	Pharmacognosy & Phytopharmaceuticals K.Hemanth	Pharmaceutical Jurisprudence Lalu Nayak		Biostatistics & Research Methodology Dr.A.S.Rao
FRIDAY	Pharmacology B.Laxmi	Medicinal Chemistry D.Sowmya		Biopharmaceutics & Pharmacokinetics Dr.D.Varun
SATURDAY	Community Pharmacy Dr.T.Pavithra	Pharmacology M.Pratyusha		Clinical Toxicology M.Vijaya laxmi

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CHAPTER WISE QUESTION BANK

Class: PHARM.D III YEAR

Subject: Medicinal chemistry

Modern concept of rational drug designing

1. Give the structures of two carrier linked prodrugs.
2. What is prodrug? Give examples.
3. Define prodrug with one example.
4. Write the structure of one prodrug and its active metabolites.
5. Mention the electronic parameters used in QSAR studies.
6. Mention the steric parameters used in QSAR.
7. What are lead molecules? How are they useful in drug discovery?
8. Enumerate the applications of QSAR.
9. What is CADD? Enlist the applications.
10. Define combinatorial chemistry. Enlist its applications.

Anti-infective agents

11. Write a note on urinary tract anti-infectives. Outline the synthesis of ciprofloxacin.
12. Add a note on synthetic antifungal agents. Give the synthesis of tolnaftate.
13. Name any four antiamoebic drugs. Give the synthesis of metronidazole.
14. What are anthelmintics? Write the synthesis of thiabendazole and albendazole
15. What are antifungal antibiotics? Explain their mechanism of action.
16. What are antitubercular agents? Write short notes on combination therapy fortuberculosis
17. Classify viral diseases and enumerate the drugs used in the therapy. Explaintheir mode ofaction. Give the structural and medicinal uses of zidovudine.
18. Classify antiviral agents? Give the structure and uses of Amantadine, Acyclovir
19. Give examples of drugs that act as DNA polymerase inhibitors. Describe theirchemistry and therapeutic uses.

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20. Give examples, MOA and clinical uses of Reverse transcriptase inhibitors.
21. Enumerate various classes of antiviral chemotherapeutic agents. Explain theMOA Idoxuridine
22. What are anti TB drugs? Enlist the problems associated with the treatment.Give the structure of Para amino salicylic acid and INH
23. What are antiviral drugs? Classify them with suitable examples.
24. Write concept of multi drug therapy for mycobactrial infections.
25. What is multi drug resistant TB? How it is treated.
26. Classify anti- TB agents? Write the structure of any two antitubercular drugs.
27. Write the structure and uses of p-amino salicylic acid, Isoniazid , Ethambutoland Pyrazinamide
28. Discuss the chemistry of Quinolones? Write the synthesis of Nitrofurantoin.
29. Give the examples of substituted Imidazoles as antifungal agents? Give thesynthesis of Miconazole
30. Name any four antiameobic agents? Give the synthesis of Metronidazole
31. Write a note on Polyene antibiotics as antifungal agents
32. Name any four synthetic antifungal agents. Outline the synthesis of Miconazole
33. Classify antifungal agents with examples? Write the synthesis of Tolnaftate
34. Classify Antiameobic agents with examples. Give the synthesis of Metronidazole
35. Classify anthelmintics with examples. Give the synthesis of Albendazole
36. Classify antitubercular agents with examples. Write the management of tuberculosis
37. Write the structure and therapeutic uses of any four antiviral agents
38. Classify Antiprotozoal agents giving one example with structure under each class.

Cardiovascular agents

1. What are CVS drugs? Mention different types with suitable examples.
2. Define and classify lipid lowering agents. Explain the MOA of of HMG coA reductase inhibitors
3. Define and classify anti arrhythmic agents with examples.
4. Discuss briefly about antianginal agents.
5. What are anti arrhythmic agents? Outline the synthesis of Procainamide.
6. Give the Chemical name, structure and specific uses of Isosorbide dinitrite, Captopril.

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7. Classify antiarrhythmic agents with examples.
8. What are antianginal drugs? Give examples.
9. Give three chemical structures and uses of drugs belonging toAntihyperl
10. Classify antiarrhythmic agents with examples. Write the MOA and therapeuticuses of Verapamil.
11. Classify antihypertensive agents with examples. Write the synthesis ofDiltiazem.
12. Give the structure and uses of the following a) Captopril b) Methyldopa c) Phenytoin.
13. What are antiarrhythmic agents. Outline the synthesis of Procainamide.
14. Write the briefly about the chemistry and MOA of anticoagulants.
15. Outline the synthesis and mode of action of any one beta blocker.
16. Discuss mode of action of Antihyperlipidemic agents with examples.
17. What are antihypertensive agents. Classify them with examples.
18. Give an account of antihyperlipedimic agents including their structure and theirspecific uses.
19. Give the synthesis and uses of Warfarin and Propronolol.Write a note on antihyperlipedimic agents.
20. Give an account on membrane depressant drugs as antiarrthymic agents.
21. Write a note on antianginal agents.

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PATHOPHYSIOLOGY

Unit 1. Basic principles of cell injury and Adaptation

1. Write about Abnormalities in lipoproteinaemia?
2. Explain about Causes, Pathogenesis and morphology of cell injury?

Unit 2. Inflammation

3. Define Inflammation? Write about the inflammatory mediators?
4. Discuss in detail the healing of wounds on the skin.
5. Give the etiology of cell injury?
6. Write about the types and characteristic features of chronic inflammation?

Unit 3. Diseases of Immunity

7. Define Hypersensitivity? Explain about types of Hypersensitivity?
8. What is Amylodosis? Write about a note on Amylodosis?

Unit 4. Cancer

9. Write the differences between benign and malignant tumours. Explain about aetiology and pathogenesis of cancer?
10. Write a detailed account on the patterns and mechanism of spread of tumours?
11. Write about Types of shock, mechanisms, stages and management?
12. Discuss the types, causes and pathogenesis of hypertension?
13. Give a detailed account of Malaria?
14. Write a note on Typhoid?
15. Give the causes and types of Asthma?
16. Explain in Detail about Asthma?
17. What is Angina Pectoris? Explain the different types of angina pectoris?
18. Write a note on Acquired Immuno Deficiency Syndrome (AIDS)?

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19. Preventive measures to control AIDS?
20. Give note on Protein Calorie Malnutrition?
21. Write a note on Chronic Obstructive Airway Diseases?
22. Write in detail about causes and mechanism involved in shock?
23. Define radiation? Write a note on influence of radiation on biological systems?
24. Describe in detail the sources and pathological effects of radiation?
25. Explain effect of radiation on human body?
26. Discuss the following: A) Carbon monoxide in air pollution. B) Biological effects of starvation.
27. Write a short note on the following:

A) Parkinsonism

B) Schizophrenia

28. Explain the pathophysiology of following

A) Myocardial Infraction

B) Atherosclerosis

C) Angina pectoris

D) CHF

29. Define hypertension and a short note on pathophysiology?
30. Pathophysiology of asthma and peptic ulcers?
31. Write the pathophysiology of Renal failure and Cirrhosis?
32. Write in brief on A) HIV

B) UTI

C) TB

33. Detail note on Syphilis and Gonorrhoea.

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20-1-2020 * Tetracyclines * UN-1 3

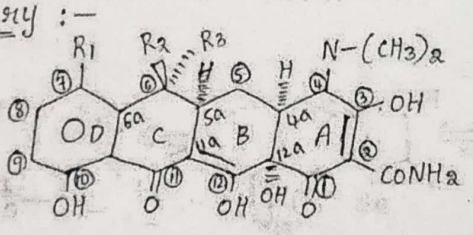
* Introduction :-

Obtained by fermentation from Streptomyces species or by chemical transmission of chemical structure.

⇒ They are derivatives of an octahydro naphthacene, a hydro carbon system that comprises 4- fused 6-membered ring.

⇒ The 1st member of tetracycline group of an antibiotics is chlortetracycline from culture of S. aurefaciens, oxytetracycline was isolated from S. rimosus.

* Chemistry :-



⇒ The stereoche is very complex. Carbon 4, 4a, 5, 5a, 6 & 12a are potentially chiral depending on subst'n. oxytetracycline & doxycycline possess 5-a hydroxy subst'n have 6-chiral 'c' atoms & other tetracyclines have only '5' chiral 'c' atoms.

⇒ The basic function is 4-a-dimethyl amine moiety.

⇒ The conjugated phenolic enone system is extending from C_{10} to C_{12} is associated with PKa at approxiamately 7.5, where as conjugated trione system extending from C_1 to C_3 in ring A is nearly acidic as acetic acid, PKa is 3. (32) (30)

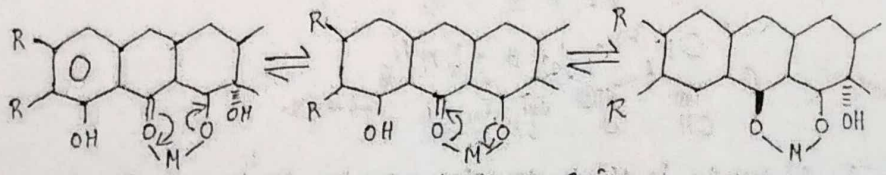
* Chemical Properties :-

⇒ The tetracyclines are Amphoteric substances (they are acidic or basic) with 3 Pka values (2.8-3.4, 7.2-7.8, and 9.1-9.7) and these are having an iso electric point at approxiamately pH-5.

⇒ It exists mainly as zwitter ion in neutral sol'n. They are yellow in colour & have bitter taste.

* Chelation :-

Imp feature of the chemical & clinical prpt's of tetra cyclines. The acidic functions of the tetracyclines are capable of forming salts through chelation with metal ions.



M - Divalent / Trivalent ion

⇒ The salts of poly valent metal ions Ca^{2+}, Mg^{2+}, Al^{2+} are all quite insoluble at neutral pH-5. This insolubility not only inconvinient for the prepn of soln's but also interfere with blood levels on oral adm'n.

⇒ The tetracyclines are incompatible with co-administered multivalent ion which antacids & with Hematonics.

* Epimerisation :-

⇒ The tetracyclines in their ability to under epimerization in soln's of intermediate pH range. This isomers are

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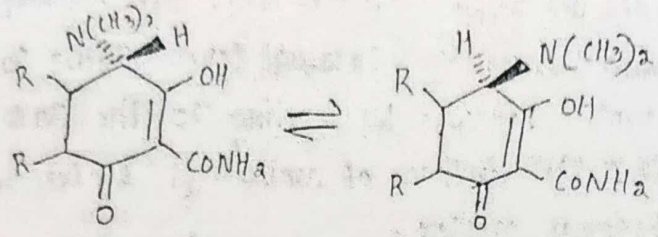
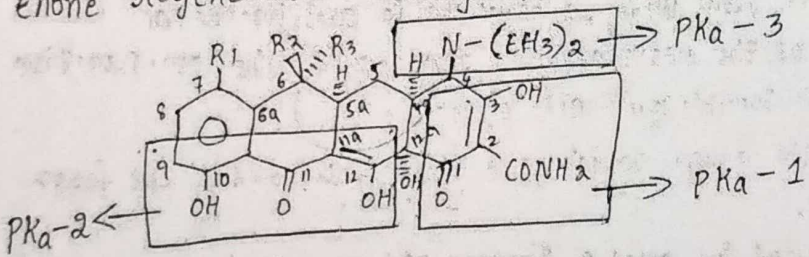
called as epitetracyclins. They exhibit less activity than the natural isomers.



Natural ⇌ Epimerisation

⇒ The α- Stereo orientation of the C4 - dimethyl amino moiety of the tetracyclines essential for their activity.

⇒ The presence of tricarbonyl system allows involving loss of C4 - 'H'. Reprotonation from the top of the enone regenerates tetracyclines.



PKa Values :-	PKa-1	PKa 2	PKa 3
Tetracycline	3.3	7.7	9.5
Chlortetracycline	3.3	7.4	9.3
Oxytetracycline	3.3	7.3	9.7
Doxycycline	3.4	7.7	9.7
Minocycline	2.8	7.8	9.3

* MoA & Spectrum of activity :-

⇒ The tetracyclines are having clinical imp. They interfere with protein binding biosyn's at ribosomal level leading to bacteriostasis

⇒ They inhibit protein biosyn's by binding to the 30s ribosome subunit & the binding of amino acyl t RNA to the m RNA ribosomal complex.

⇒ They are broad spectrum antibiotic active against gram +ve & -ve bacteria.

Ex :- Spirochetes, Microplasma, Rickettsiae and Chlamydia.

* Uses :-

They possess very wide bacteriostatic antibacterial activity bcz of the resistance phenomena & the comparitive frequency of troublesome side effects.

⇒ They are the drugs which are rarely used for the first choice today.

⇒ They are used for oral & topical therapy for achne in low doses & also used for uninary tract infections, rickettsia infections, Microplasma pneumonia, prophylaxis, prevention of diarrhoea & cholera.

⇒ Widely used for agricultural purposes.

* Classification :-

. Classified into 3 types.

1) Natural tetracyclins.
2) Synthetic / protetracyclins.
3) Semisynthetic.

(35)

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① Natural tetracyclins :-

Ex :- Tetracycline, chlortetracycline, Bromotetracycline, Demethyl tetracycline, demethyl chlor tetracycline.

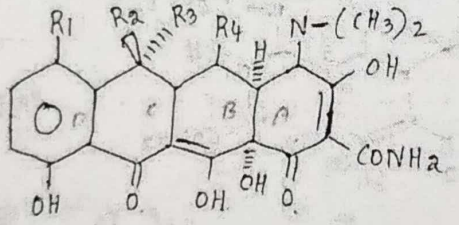
② Semisynthetic tetracyclins :-

Ex :- Doxycycline, Minocycline, Methacycline, Meclocycline, Sancycline.

③ Synthetic tetracyclins :-

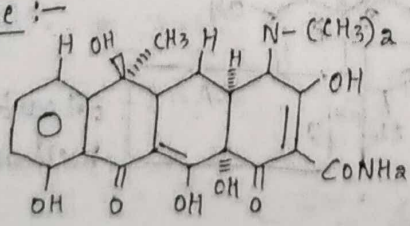
Ex :- Rolitetracycline, Lymetetracycline, Clomocycline, Apicycline, pipacycline.

* Structures of imp tetracyclines :-



	R1	R2	R3	R4
Tetracycline —	-OH	-OH	$-CH_3$	-H
Chlortetracycline —	-Cl	-OH	$-CH_3$	-H
oxycycline —	-H	-OH	$-CH_3$	-OH
Doxycycline —	-H	$-CH_3$	-H	-OH
Minocycline —	$-N-(CH_3)_2$	-H	-H	-H

1) Tetracycline :-



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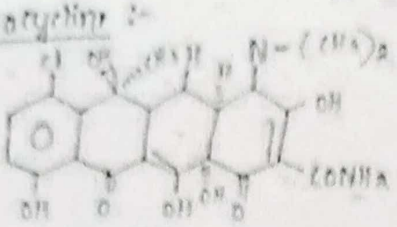
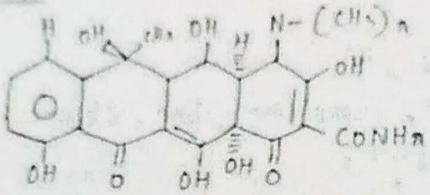
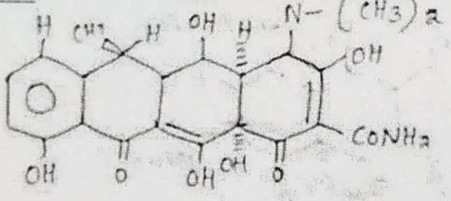
2) Chlortetracycline :-



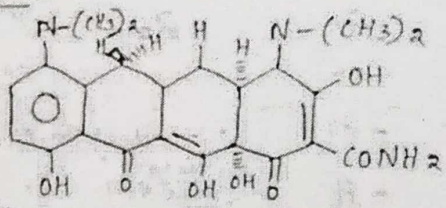
3) Oxytetracycline :-



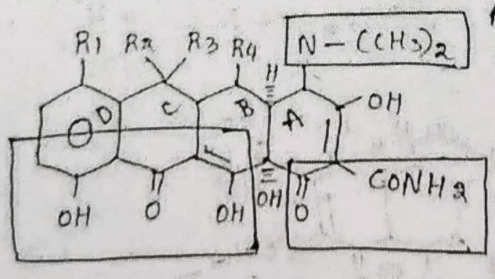
4) Doxycycline :-



5) Minocycline :-



* SAR of tetracyclines :-



1) Functional grps at positions 5,6 & 7 on rings B,C & D in tetracyclines can be removed without drastically on their antimicrobial Pft's.

2) conversion of carboxamide grp ($CONH_2$) to nitriles causes loss of activity. Some carboxamide & methyl grps are highly active.

3) Epimerization of C_3 & C_4 dehydrogenation C_5 and C_{11a} results in the loss of activity.

4) The mono & tri methyl amino are relatively inactive.

* Adverse effects :-

Toothstaining, photoxicity, Kidney damage, Nausea, Vomiting, diarrhoe & some CNS effects (dizziness & Vertigo).

⇒ Rapid admt'n on prolonged IV use can lead to the thrombophlebitis.

* Resistance of tetracyclines :-

Resistance results from an unusual ribosomal protection process involving elaboration & bacterial proteins. These proteins associated with the ribosomes thus allowing protein biosyn's to proceed even in the presence of bound tetracycline

⇒ Another imp resistance mechanism involves 'R' factor mediated energy requiring active efflux of tetracyclines from bacterial cells.

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STUDY MATERIAL FOR SLOW LEARNERS

UV-VISIBLE SPECTROSCOPY

principle:

* UV spectroscopy – Definition | visible Spectroscopy
* Compounds which are Colourless
  ↓
  absorb radiation in UV region
  (200-400 nm)
* Compounds which are Coloured
  ↓
  absorb radiation in Visible region
  (400-800nm)

(I)

* In both UV & Visible:

  Only Valence electrons absorbs the Energy
  ↓ (thereby)
  Molecule undergoes
  ↓
  transition from
  ⇓
  Ground state ⟶ Excited state

* Any molecule has either n, π or σ
  (or)
  Combination of these electrons absorbs the characteristic radiation & undergoes transition from
  G.S ⟶ E.S.

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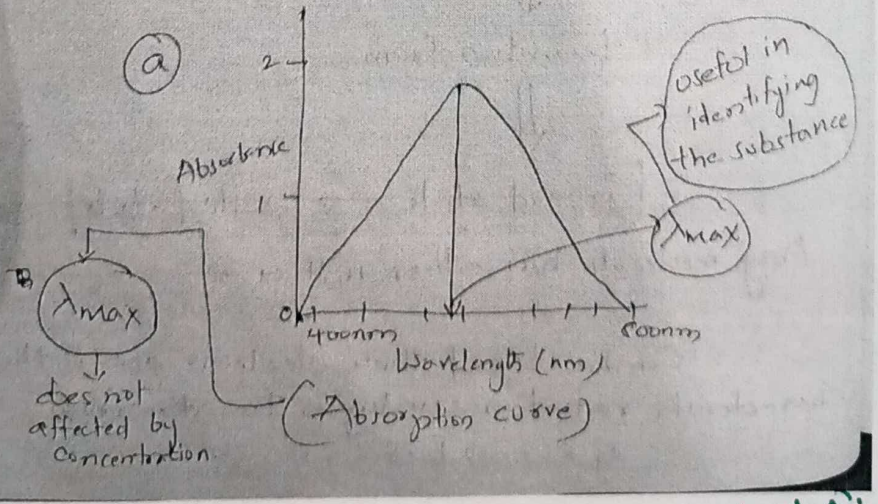
* This absorption is characteristic
  ↓ E
  depends on the nature of electrons.

* The Intensity of absorption
  ↓
  depends on → Concentration
  → pathlength

(ii) Coloured substances
↓
absorb light of different wavelength
↓
in different manner
↓
we get an Absorption curve
(Absorbance (vs) Wavelength)

(a)



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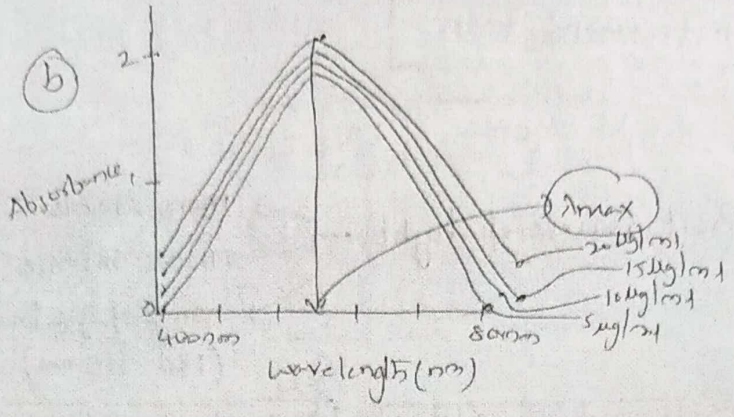
(b)



Absorption curve using

↓

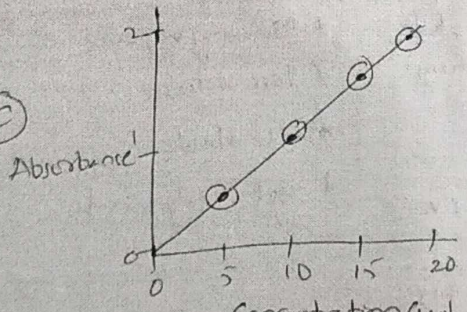
different Concentrations of same substance

⇓

Absorbance of a solution ↑ses with Concentration of a substance

Note (*) But there is "no change in λmax"

(c)



When we plot a graph between Conc (vs) Absorbance

⇓

← we get a.

Calibration Curve

(or)

Standard Curve

↳ is useful for determining the "Concentration"

(or)

amount of drug substance in the given sample solution.

A.M.

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Instrumentation.

① Light source : [UV]

Best source of light → more stable
→ more intense
→ range of spectrum (120 – 360 nm)

(a) Hydrogen discharge lamp

* Hydrogen under high pressure
* more stable
* widely used & robust
* gives radiation from 120 – 350 nm

(b) Xenon discharge lamp

* Xenon at 10 – 30 atm. pressure
* less stable than HDL
* widely used & robust
* Intensity greater than HDL

(c) Deuterium lamp

* Similar to HDL, but filled with "deuterium"
* more intense
* more stable
* widely used & Expensive

(d) Mercury arc lamp

* Mercury vapours
* less intense
* less stable
* Not widely used.

① Light source [visible]

(a) Tungsten lamp

* Tungsten filament in a vacuum bulb.
* more stable
* less intense
* widely used

Carbon arc lamp

* Carbon arc with a thick coating.
* less stable than T.L
* Very high Intensity
* Not widely used.

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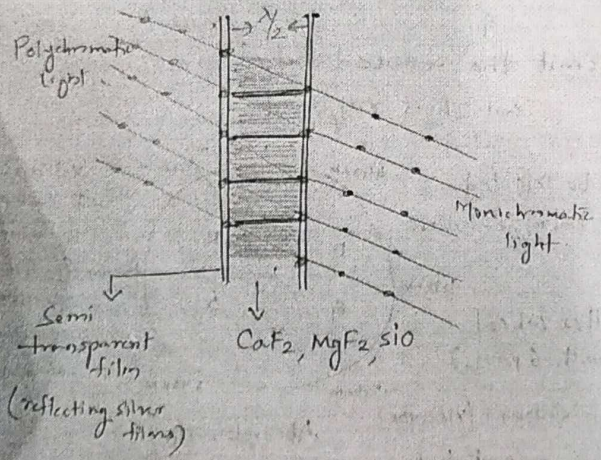
(v) The Colour of the filter is opposite to the Colour of the solution
(i.e) (Complimentary in nature)

(vi) We can select the required filter in a Colorimeter, based upon the Colour of the Solution.

(b) **Interference filters**

(Fabry-perot filter)



* It has dielectric Spacer film made up of CaF_2, MgF_2, SiO, between two parallel reflecting silver films.

* The thickness of dielectric Spacer film can be
  ($\lambda/2$) $1/2\lambda$ – 1st order
  @ $\lambda/2$ – 2nd order
  $3\lambda/2$ – 3rd order etc..

* Mechanism:

The radiation reflected by 2nd film

↕

Incoming radiation

⇓

CONSTRUCTIVE INTERFERENCE

↳ to give monochromatic radiation

↓

followed by equation

$$\lambda = 2\eta b/m$$

λ = wavelength of light
η = dielectric Constant
b = Layer thickness
m = Order no. (0, 1, 2, 3 – etc)

Beer-Lambert's law:

* UV- visible spectroscopy
  - ↳ based on fundamental law of absorption

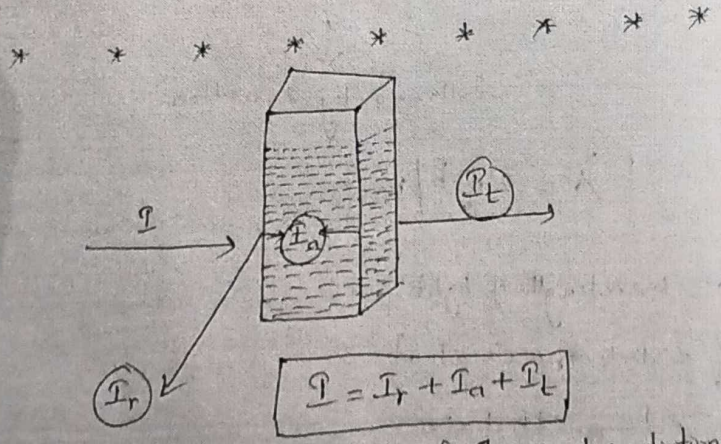
⇓

Beer-Lambert's law

⇒ This law governs the absorption of radiation

↓

by an absorbing medium (Dilute solution)



$$I = I_r + I_a + I_t$$

I = Intensity of Incident radiation
I_r = Intensity of reflected radiation
I_a = Intensity of absorbed radiation
I_t = Intensity of transmitted radiation

* I_r = negligible (1%)

So, $\boxed{I = I_a + I_t}$

Relation b/w Intensity of incident light (I)
absorbed light (I_a)
transmitted light (I_t)

⇓

given by

Beer-Lambert's law

Beer's law: $I = I_0 \cdot e^{-kc}$

$$-\frac{dI}{dc} \propto I \quad \text{——— (1)}$$

Lambert's law: $I = I_0 \cdot e^{-kt}$

$$-\frac{dI}{dt} \propto I \quad \text{——— (2)}$$

Beer-Lambert's law:

$A = act$ ——— (1+2) (3)

$A = \epsilon ct$