COMPETENCY BASED DYNAMIC CURRICULUM FOR THIRD BHMS PROFESSIONAL COURSE

(Applicable from Batch 2022-2023 onwards for 5 years or until further notification by National Commission for Homoeopathy whichever is earlier)

(Essentials of Pharmacology)

HOMOEOPATHY EDUCATION BOARD

NATIONAL COMMISSION FOR HOMOEOPATHY

MINISTRY OF AYUSH, GOVERNMENT OF INDIA

JAWAHAR LAL NEHRU BHARTIYA CHIKITSA AVUM HOMOEOPATHY ANUSANDHAN BHAVAN

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Course: Essentials of Pharmacology

Course Code: HomUG-Mod.Phar

Index

1. Preamble

Welcome, homeopathy students, to the world of pharmacology! This course will delve into the fascinating realm of medicines and their interactions with the human body. While homeopathy focuses on stimulating the body's natural healing response, understanding conventional medications is crucial for several reasons:

Complementary Care: Homeopathy can sometimes be used alongside traditional medications

Drug Interactions: Being aware of potential interactions between homeopathic remedies and conventional drugs is essential for safe patient care.

Patient Education: Many patients will be taking other medications and understanding how they work can empower you to better educate and guide them.

This course will equip you with a foundational knowledge of pharmacology, covering key areas such as:

Drug classifications and mechanisms of action

Pharmacokinetics: How drugs are absorbed, distributed, metabolized, and excreted

Pharmacodynamics: How drugs produce their effects on the body

Common medications used in various therapeutic areas

By the end of this course, you'll gain a deeper appreciation for the science of pharmacology and its valuable role in healthcare. We'll explore how this knowledge can complement your understanding of homeopathy and ensure you provide the safest and most informed care to your future patients.

Please note: This course is designed to provide a general overview of pharmacology. It is not intended to replace the knowledge and expertise of medical doctors or pharmacists.

2. Course outcomes

Upon successful completion of this pharmacology course, homeopathy students will be able to:

i. Demonstrate a foundational knowledge of major drug classifications and their mechanisms of action.
ii. Apply a scientific foundation to their understanding of medication and therapeutics, aligning with core principles of homeopathy.
iii. Demonstrate a comprehensive understanding of major drug classifications and their mechanisms of action.
iv. Explain the pharmacokinetics and pharmacodynamics of medications, including how drugs are absorbed, distributed, metabolized, excreted, and produce their effects in the body.
v. Identify common medications used in various treatment areas.
vi. Apply their understanding of Pharmacology to assess potential interactions between homeopathic remedies and conventional medications to ensure patient safety.
vii. Communicate medication information effectively to patients, empowering them to make informed decisions about their healthcare.
viii. Provide safe and complementary care to their patients by understanding conventional medications.
ix. Educate patients about potential interactions between medications.
x. Collaborate effectively with other healthcare providers when necessary.
xi. Treat and solve the adverse drug reactions of the patients with the homeopathy drugs.

Disclaimer: This course is designed to provide a general foundation in pharmacology. It is not a substitute for the expertise of medical doctors or pharmacists.

3. Course content

I. Module 1: Pharmacology

i. Introduction to Pharmacology
ii. Definition and Scope of Pharmacology
iii. Drug Nomenclature and Classification Systems
iv. Routes of Drug Administration

II. Module 2: Pharmacokinetics

i. Absorption, Distribution, Metabolism, and Excretion of Drugs (ADME)
ii. Factors Affecting Pharmacokinetics

III. Module 3: Pharmacodynamics

i. Mechanisms of Drug Action on Body Systems
ii. Dose-Response Relationships
iii. Factors Modifying Drug Action

IV. Module 4: Major Drug Classifications

A. ANS AND AUTACOID

i. Cholinergic and Anticholinergic drugs,
ii. Adrenergic and Antiadrenergic Drugs, T/t of Glaucoma
iii. Autacoids: Serotonin and drugs acting or Serotonergic System+ T/t of Migraine,
iv. Histamine and Antihistaminic

B. NSAID- Drugs used in RA and Gout

C. CNS

i. Anxiolytics
ii. Antiepileptics
iii. Antipsychotics and Antidepressants
iv. Opioid Analgesics

D. Respiratory system

i. Drugs for cough
ii. Bronchial asthma and COPD

E. Hormones

i. Insulin and oral Hypoglycemic drugs
ii. Adrenocortical steroids
iii. Estrogens, Progesterone and OCPs
iv. Vitamin D, Calcium and Drugs affecting Calcium Balance

F. CVS

i. T/t of Hypertension
ii. Angina, MI
iii. Cardiac Glycosides and Drugs for Heart failure
iv. Hypolipidemic drugs

G. Renal system- Diuretics and Antidiuretics

H. Blood- Hematinics, T/t of Iron deficiency anaemia and Megaloblastic anemia

I. GIT

i. Drugs for Peptic Ulcer and GERD
ii. Drugs for constipation and diarrhea
iii. Antiemetics

J. Chemotherapy

i. Sulfonamides and Cotrimoxazole,
ii. Quinolones,
iii. Beta Lactam Antibiotics,
iv. Tetracyclines, Chloramphenicol
v. Aminoglycosides
vi. Ant tubercular drugs and Antileprosy drugs
vii. Antimalarial drugs

K. Miscellaneous

i. Disinfectants
ii. Vitamins

4. Teaching hours

Year/Subject	Teaching hours- Lectures
III BHMS/ Essentials of Pharmacology	45

4.1.Term-wise teaching hours division:

Sr. No	Topics	Teaching Hours
	Term I	
1	**Module 1: Pharmacology (Introduction)**	**5**
2	**Module 2: Pharmacokinetics**	**5**
3	**Module 3: Pharmacodynamics**	**5**
4	**Module 4: Major Drug Classifications**	
i	ANS AND AUTACOIDS	4
ii	NSAID, Drugs used in RA and Gout	2
iii	CNS	2
iv	Respiratory System	3
	Term II	
v	Hormones	4

vi	CVS	2
vii	Renal System	2
viii	Blood	1
ix	GIT	4
x	Chemotherapy	4
xi	Miscellaneous	2
Total		**45**

5. Content mapping (Competencies tables):

Module 1: Pharmacology-

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
HomUG-Mod.Phar 1.1	Knowledge and scholarship	K	Introduction to Pharmacology	Explain the fundamental principles of pharmacokinetics (absorption, distribution, metabolism, excretion). Define key pharmacodynamic terms (agonists, antagonists, therapeutic index).	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	
				Recognize drug classifications and their mechanisms of action	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	

				Participate in clinical settings, reviewing patient medications under supervision	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	
HomUG-Mod.Phar 1.2	Knowledge and scholarship	K	Definition and Scope of Pharmacology	States the primary components of pharmacology (drug actions, mechanisms, therapeutic uses)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Lists the subdivisions of pharmacology and their relevance.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Explains how pharmacokinetics and pharmacodynamics influence drug therapy.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Describes how adverse effects or drug interactions impact patient care.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Interprets drug concentration-time curves or other pharmacokinetic data.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
HomUG-Mod.Phar	Knowledge and scholarship	K	Drug Nomenclature and Classification Systems	**olol**: Beta-blockers (e.g., propranolol, atenolol)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				pril: ACE inhibitors (e.g., lisinopril, enalapril)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Es-: Refers to an S-enantiomer (e.g., esomeprazole)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Levo-: Refers to a levorotatory isomer (e.g., levothyroxine)	C1	MK	Lecture, Group	Quiz, Written	SAQ, MCQ	Physiology

1.3							discussion	test, MCQ		
				cillin: Penicillin derivatives (e.g., ampicillin)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				statin: HMG-CoA reductase inhibitors (e.g., atorvastatin)	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
HomUG-Mod.Phar 1.4	Knowledge and scholarship	K	Routes of Drug Administration	**<u>Drug-Related Factors:</u>** Can the drug survive the environment of the GI tract (e.g., oral vs. IV)? Is the drug lipid or water-soluble? Does the drug require bypassing the liver (e.g., sublingual, parenteral)? Does the drug require rapid action (IV) or sustained release (transdermal)? **Consciousness**: Is the patient conscious and cooperative (oral vs. IV/IM)?	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	
				<u>Patient-Related Factors:</u> **Age**: Pediatrics and geriatrics may require specific routes (e.g., rectal for children) **Physical Condition**: Difficulty swallowing (requires non-oral routes like IV, SC). **Preferences and Compliance**: Does the patient prefer certain methods for better adherence (e.g., patches over injections)? **Vomiting/NPO (Nil Per Os)**: Oral route is contraindicated	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	

				Disease or Condition Factors: **Site of Action**: Does the drug act locally (topical, inhalational) or systemically (oral, IV)? **Urgency**: Emergency conditions often require IV for rapid effect **Target Organ**: Routes like intrathecal are used for CNS delivery due to the blood-brain barrier.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	

Module 2: Pharmacokinetics

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod.	Absorption, Distribution, Metabolism, and Excretion of Drugs (ADME)	K	Knowledge and scholarship	**Physicochemical Properties of the Drug** • Lipophilicity enhances crossing of cell membranes and distribution into fatty tissues. • Polarity limits drug penetration into lipid-rich	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Route of Administration	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Gastrointestinal Factors:	C1	MK	Lecture,	Quiz,	SAQ, MCQ	Physiolog

Phar 2.1				• pH of the stomach or intestine (affects ionization and solubility). • Gastric emptying time and motility. • Presence of food (may enhance or reduce absorption).			Group discussion	Written test, MCQ		y
				Drug Formulation: Immediate-release vs. sustained-release forms. Coating to protect against stomach acid (e.g., enteric-coated tablets).	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Blood Flow to Tissues: 1.Highly perfused organs (e.g., brain, liver, kidneys) get more drug initially. 2.Poorly perfused tissues (e.g., fat, bone) show slower distribution.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				Plasma Protein Binding: • Albumin binds acidic drugs; α1-acid glycoprotein binds basic drugs. • Only free (unbound) drug is pharmacologically active. **Tissue Binding**: • Some drugs accumulate in specific tissues (e.g., tetracyclines in bones/teeth, lipophilic drugs in adipose tissue) **Special Barriers**: • Blood-brain barrier: Permits lipophilic and small molecules; restricts polar drugs. • Placental barrier: Filters certain drugs, but not all.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				• Environments. **Site of Metabolism**: • Liver (primary site): Phase I and Phase II reactions. • Other tissues: Kidneys, lungs, intestines. **Phase I Reactions**: • Oxidation, reduction, hydrolysis. • Primarily mediated by cytochrome P450 enzymes. **Phase II Reactions**: • Conjugation reactions (e.g., glucuronidation, sulfation). • Make drugs more water-soluble for excretion. **First-Pass Metabolism**: • Drug is metabolized by the liver before reaching systemic circulation. • Reduces bioavailability (e.g., nitroglycerin, propranolol). **Enzyme Induction/Inhibition**: • Induction (e.g., rifampin, phenobarbital): Increases metabolism, reducing drug levels. • Inhibition (e.g., grapefruit juice, ketoconazole): Decreases metabolism, increasing drug levels. **Genetic Polymorphisms**: • Variability in enzyme activity (e.g., CYP2D6, CYP2C19) affects metabolism rates. • Primary Routes of Excretion **Hepatic (bile)**:						

				• Drug Ionization and Solubility • Renal Function						
Hom UG-Mod. Phar 2.2	Factors Affecting Pharmacokinetics		Knowledge and scholarship	**<u>Factors Affecting Absorption</u>** **Drug-Related Factors**: • **Solubility**: Lipophilic drugs absorb better through cell membranes. • **Ionization**: Non-ionized forms cross membranes more easily. • **Molecular Size**: Smaller molecules are absorbed more rapidly. • **Formulation**: Liquid > Capsule > Tablet (for speed of absorption). • **Chemical Stability**: Drugs degraded by gastric acid or enzymes have reduced absorption (e.g., insulin). **Route of Administration**: • Oral absorption depends on GI environment, while parenteral routes bypass it. **Gastrointestinal (GI) Factors**: • **pH**: Affects drug ionization (e.g., acidic drugs absorb better in the stomach, basic drugs in the intestine). • **Gastric Emptying**: Faster emptying enhances absorption. • **Presence of Food**: Some	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				drugs require food (e.g., fatty meals enhance lipophilic drug absorption), while others are inhibited. **Drug Transport Mechanisms**: • Passive diffusion, active transport, facilitated diffusion, or endocytosis. **Physicochemical Properties**: • Lipid solubility and polarity affect drug penetration into tissues. **Blood Flow to Tissues**: • Highly perfused organs (brain, liver, kidneys) receive drugs faster than less perfused areas (e.g., fat, bone). **Plasma Protein Binding**: • Bound drugs are pharmacologically inactive; only free drugs can act. • Albumin binds acidic drugs; α1-acid glycoprotein binds basic drugs. **Tissue Binding**: • Drugs like tetracyclines bind						

				to calcium in bones/teeth. **Special Barriers**: • **Blood-Brain Barrier (BBB)**: Limits polar and large molecules; favors lipophilic drugs. • **Placental Barrier**: Provides partial protection to the fetus but allows passage of some drugs. **Site of Metabolism**: Liver is the primary organ (Phase I and Phase II reactions), but other tissues (kidneys, lungs, intestines) also contribute. • **Phase I (Functionalization Reactions)**: o Oxidation, reduction, hydrolysis (e.g., CYP450 enzymes). • **Phase II (Conjugation Reactions)**: o Glucuronidation, sulfation, acetylation (make drugs water-soluble for excretion).						
				<u>Factors Affecting Metabolism</u> **Enzyme Activity**: • **Induction**: Enzyme inducers	C1	MK	Lecture, Group discussio	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				(e.g., rifampin, carbamazepine) increase metabolism and reduce drug efficacy. • **Inhibition**: Enzyme inhibitors (e.g., ketoconazole, grapefruit juice) decrease metabolism and increase drug levels. **Genetic Variability**: • Polymorphisms in CYP enzymes (e.g., CYP2D6, CYP2C19) cause individual variability in drug metabolism. **First-Pass Metabolism**: • Drugs extensively metabolized in the liver or gut wall before reaching systemic circulation (e.g., propranolol, nitroglycerin). **Age**: • Neonates have immature metabolic enzymes; elderly have reduced enzyme activity.			n			
				<u>Factors Affecting Excretion</u> **Route of Excretion**: **Renal Excretion (Primary)**: • **Glomerular Filtration**: Free drugs filtered based on molecular size.	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y

				• **Tubular Secretion**: Active process for drug elimination (e.g., penicillin). • **Tubular Reabsorption**: pH-dependent (acidic drugs excreted faster in alkaline urine). **Biliary Excretion**: • High molecular weight drugs excreted via bile; may undergo enterohepatic recycling. **Other Routes**: • Lungs (volatile drugs, e.g., anesthetics), sweat, saliva, breast milk. **Drug Properties**: • Lipophilic drugs require metabolism to water-soluble forms for excretion. • Ionized drugs are excreted more readily in urine. **Renal Function**: • Impaired kidney function (e.g., in elderly or renal disease) reduces drug clearance. **Hepatic Function**:						

				• Liver dysfunction reduces biliary excretion and metabolism. **Age**: • Neonates have immature kidneys; elderly may have reduced renal function **Age**: • Neonates and elderly patients have reduced metabolic and excretory capacity. **Gender**: • Hormonal differences can influence metabolism and excretion. **Genetic Variations**: • Pharmacogenetics impacts enzyme activity (e.g., poor vs. ultra-rapid metabolizers). **Disease States**: Liver disease, kidney disease, heart failure, or other conditions affect ADME processes						
				<u>Patient-Related Factors</u> **Drug-Drug Interactions**:	C1	MK	Lecture, Group discussio	Quiz, Written	SAQ, MCQ	Physiology

				• Competition for enzymes, protein-binding sites, or transporters. **Lifestyle Factors**: • Diet (e.g., grapefruit juice inhibits CYP enzymes). • Smoking or alcohol use (induces enzymes like CYP1A2).			n	test, MCQ		

Module 3: Pharmacodynamics

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom	Mechanisms of Drug Action on Body Systems	K	Knowledge and scholarship	**Receptor Interaction** Agonists, antagonists (e.g., β-blockers, opioids) **Enzyme Modulation** Inhibition, activation (e.g., statins, aspirin). **Ion Channel Modulation**	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

UG-Mod. Phar **3.1**				Blockers, openers (e.g., calcium channel blockers, sodium channel blockers). **Neurotransmitter Systems** Reuptake inhibitors, enzyme inhibitors (e.g., SSRIs, MAO inhibitors). **Hormonal Systems** Agonists, antagonists, synthesis inhibitors (e.g., insulin, tamoxifen) **Immune Modulation** Vaccines, monoclonal antibodies (e.g., rituximab) **DNA/RNA Interaction** Anticancer agents, antivirals (e.g., cyclophosphamide, AZT). **Physical/Chemical Action** Osmotic agents, antacids (e.g., mannitol, sodium bicarbonate)						
Hom UG-	Dose-Response Relationships	K	Knowledge and scholarship	**<u>Agonists, Antagonists, and Modulators</u>** **Full Agonist**: • Produces the maximum possible response (e.g., epinephrine on beta-adrenergic receptors). **Partial Agonist**: • Produces a submaximal	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

Mod. Phar **3.2**				response, even at full receptor occupancy (e.g., buprenorphine). **Antagonist**: • Binds to receptors but does not activate them, blocking agonist effects (e.g., naloxone for opioid overdose). **Inverse Agonist**: • Produces the opposite effect of an agonist (e.g., propranolol as a beta-blocker). **Allosteric Modulators**: • Bind to a site other than the active site and modify receptor activity (e.g., benzodiazepines on GABA-A receptors).						
				<u>Variability in Dose-Response</u> **Inter-Individual Variability**: • Genetics, age, gender, disease states, and drug interactions can alter responses. **Tachyphylaxis**: • Rapid decrease in drug response with repeated doses	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				(e.g., nitrates for angina). **Tolerance**: • Gradual decrease in drug effect over time, requiring higher doses (e.g., opioids). **Hypersensitivity**: • Exaggerated response to a small dose.						
				<u>Drug Toxicity and Overdose</u> Indicators: **Toxic Dose (TD50)**: • Dose at which 50% of individuals experience toxic effects. **Lethal Dose (LD50)**: • Dose that is lethal to 50% of the population (typically studied in animals). **Margin of Safety**: • Difference between the therapeutic dose and the toxic dose. **Dose Adjustments**: • Based on individual patient	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				responses (e.g., warfarin dosing guided by INR levels).						
				<u>Applications in Clinical Practice</u> **Monitoring Therapeutic Effect**: • Measuring drug levels to ensure they are within the therapeutic range (e.g., digoxin, lithium). **Avoiding Toxicity**: • Monitoring for signs of toxicity and adjusting doses as needed.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
Hom UG-Mod. Phar 3.3	Factors Modifying Drug Action	K	Knowledge and scholarship	**<u>PhysiologicalFactors</u>** **Age**:**Neonates**: • Immature liver enzymes affect metabolism (e.g., chloramphenicol toxicity leads to "gray baby syndrome"). • Reduced renal clearance in newborns (e.g., aminoglycoside toxicity). **Elderly**: • Decreased liver and kidney function reduces drug clearance (e.g., accumulation of digoxin) **Gender**: • Hormonal differences	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				influence drug metabolism (e.g., women metabolize benzodiazepines slower than men). **Body Weight**: • Dosage adjustments are based on weight or body surface area (e.g., chemotherapy drugs).						
				<u>Pathological Factors</u> **Genetic Factors**: • Genetic polymorphisms affect drug metabolism (e.g., slow acetylators for isoniazid). **Liver Disease**: • Reduced metabolism of drugs (e.g., prolonged half-life of warfarin in hepatic dysfunction). **Kidney Disease**: • Impaired excretion of renally eliminated drugs (e.g., accumulation of aminoglycosides or lithium). **Cardiovascular Disease**: • Reduced perfusion delays drug distribution and elimination	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				(e.g., digoxin in heart failure). **Thyroid Function**: • Hyperthyroidism increases drug metabolism (e.g., rapid clearance of β-blockers). • Hypothyroidism decreases drug metabolism (e.g., longer half-life of digoxin)						
				<u>Pharmacogenetic Factors</u> **Indicators**: **Cytochrome P450 Enzymes**: • Variants like CYP2D6 or CYP3A4 alter drug metabolism (e.g., poor metabolizers of codeine may have reduced analgesic effects). **Drug Transporters**: • Genetic variations in P-glycoprotein (e.g., altered absorption of digoxin). **Enzyme Deficiency**: • Glucose-6-phosphate dehydrogenase (G6PD) deficiency leads to hemolysis with certain drugs (e.g., sulfonamides).	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				<u>Environmental Factors</u> **Indicators**: **Diet** • Grapefruit inhibits CYP3A4, increasing levels of drugs like statins. • High-fat meals delay drug absorption (e.g., delayed effect of antacids). **Smoking**: • Induces CYP1A2, increasing clearance of drugs like theophylline. **Alcohol**: • Acute alcohol use inhibits drug metabolism (e.g., potentiates sedatives). • Chronic alcohol use induces enzymes, accelerating metabolism (e.g., warfarin). **Temperature**: • Heat increases peripheral blood flow, enhancing drug absorption.	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y
				<u>Drug Interactions</u> **Indicators**:	C1	MK	Lecture, Group discussio	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y

				Pharmacokinetic Interactions: **Absorption**: • Antacids reduce absorption of tetracyclines. **Metabolism**: • Enzyme inducers like rifampin increase clearance of oral contraceptives. • Enzyme inhibitors like ketoconazole reduce metabolism of midazolam. **Excretion**: • Probenecid inhibits renal excretion of penicillin, prolonging its action. **Pharmacodynamic Interactions**: • Synergism (e.g., sulfonamides and trimethoprim). • Antagonism (e.g., naloxone reversing opioid effects).			n			
				<u>Tolerance and Dependence</u> **Indicators**: **Tolerance**: • Gradual decrease in drug effect with repeated use (e.g.,	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y

				opioids, nitrates). • Cross-tolerance (e.g., benzodiazepines and alcohol). **Dependence**: • Physical or psychological need for a drug (e.g., opioids, benzodiazepines).						
				<u>Route of Administration</u> **Indicators**: **Oral Route**: • First-pass metabolism reduces bioavailability (e.g., propranolol). **Intravenous Route**: • Rapid onset and complete bioavailability. **Intramuscular/Subcutaneous**: • Rate of absorption depends on blood flow and drug solubility	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology
				<u>Disease-Specific Factors</u> **Indicators**: **Infection and Inflammation**: • Alters drug distribution (e.g., reduced effectiveness of aminoglycosides in acidic	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				abscesses). **Malnutrition**: • Reduced plasma proteins (e.g., hypoalbuminemia increases free drug levels of phenytoin).						
				Psychological Factors Indicators: **Placebo Effect**: • Positive therapeutic response due to patient belief in treatment. **Nocebo Effect**: • Negative outcomes due to patient expectations of adverse effects.	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

Module 4: Major Drug Classifications

HomUG-Mod.Phar– 4.1 (ANS AND AUTACOIDS)

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formativ	Summative	

								e		
Hom UG-Mod. Phar 4.1.1	Cholinergic and Anticholinergic drugs	K	Knowledge and scholarship	**<u>Cholinergic Drugs</u>** • Define **Cholinergic and Anticholinergic Drugs:** Understand the pharmacological basis of these drugs, their receptors, and physiological actions. • Explain **Mechanisms of Action:** Describe how these drugs influence parasympathetic activity by either mimicking or blocking acetylcholine. • List **Clinical Applications:** Identify the therapeutic uses of cholinergic and anticholinergic drugs, such as in glaucoma, myasthenia gravis, COPD, or motion sickness. • Assess **Adverse Effects:** Recognize side effects such as dry mouth, tachycardia (anticholinergics), or diarrhea, bradycardia (cholinergics). • Compare **Drug Classes:** Differentiate between direct-acting, indirect-acting cholinergic drugs, and muscarinic antagonists.	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y
	Adrenergic and Antiadrenergic Drugs,	K	Knowledge and scholarship	**Miller Principles for Antiadrenergic Drugs:** • **Selective Blockade:** Selective	C1	MK	Lecture, Group discussio	Quiz, Written test, MCQ	SAQ, MCQ	Physiolog y

Hom UG-Mod. Phar **4.1.2**				α\alphaα or β\betaβ blockers minimize side effects and target specific conditions (e.g., β1\beta_1β1 blockers for heart conditions). • **Sympathetic Inhibition:** These drugs decrease heart rate, reduce cardiac output, and lower blood pressure by opposing SNS activity. • **Receptor Affinity and Potency:** Drugs with higher selectivity (e.g., atenolol for β1\beta_1β1) are preferred in specific populations to minimize adverse effects (e.g., bronchospasm). • **Clinical Uses:** Manage hypertension, arrhythmias, angina, heart failure, and conditions like pheochromocytoma. **Adverse Effects:** Monitor for bradycardia, hypotension, and fatigue, particularly in elderly or heart-compromised patients. **Antiadrenergic Drugs:** • Understand the roles of adrenergic receptors and how drugs modulate their activity. • Explain **Mechanisms of Action:** Describe how adrenergic drugs stimulate SNS activity, while antiadrenergic drugs inhibit it. • Classify **Adrenergic Agents:**			n			

				Differentiate between direct, indirect, and mixed-acting adrenergic agonists and selective/non-selective adrenergic blockers. • Identify **Therapeutic Applications:** Highlight uses in conditions such as asthma (β2\beta_2β2 agonists), hypertension (β1\beta_1β1 blockers), and shock (α\alphaα-agonists). • Discuss **Adverse Effects:** Recognize potential side effects like tachycardia (adrenergics) or bradycardia (antiadrenergics).						
Hom UG-Mod. Phar **4.1.3**	Autacoids: Serotonin and drugs acting or Serotonergic System+ T/t of Migraine,	K	Knowledge and scholarship	Selective Serotonin Reuptake Inhibitors (SSRIs): Serotonin-Norepinephrine Reuptake Inhibitors (SNRIs): 5-HT1_11 Agonists 5-HT3_33 Antagonists: 5-HT4_44 Agonists: Serotonin Modulators: Understand **the Serotonergic System:** • Describe serotonin's role in neurotransmission and its physiological functions in the	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				CNS and periphery. Classify **Serotonergic Drugs:** • Differentiate between SSRIs, SNRIs, triptans, serotonin receptor agonists/antagonists, and modulators. Explain **Mechanisms of Action:** • Discuss how drugs alter serotonin signaling to treat conditions like depression, migraines, and IBS. **Identify Clinical Applications:** • Match drug classes to appropriate therapeutic uses, such as SSRIs for depression or 5-HT1_11 agonists for migraines.						
Hom UG-Mod. Phar 4.1.4	Histamine and Antihistaminic	K	Knowledge and scholarship	Acute Treatments **Nonsteroidal Anti-Inflammatory Drugs (NSAIDs):** Ergot Alkaloids: Anti-Nausea Medications: Preventive Treatments Beta-Blockers	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				Anticonvulsants: **Calcium Channel Blockers:** CGRP (Calcitonin Gene-Related Peptide) Antagonists: Tricyclic Antidepressants (TCAs) **Understand Pathophysiology:** • Explain the neurovascular basis of migraines and the role of serotonin and CGRP pathways. **Differentiate Migraine Types:** • Identify different migraine presentations (e.g., with aura, without aura) and their implications for treatment. **Classify Therapies:** • Categorize acute and preventive migraine treatments based on their mechanisms of action.						

HomUG-Mod.Phar– 4.2 (NSAID)

Sl.No.	**Duration of Competency**	**Miller**	**Content**	**Specific Learning Objectives**	**Bloom/ Guilbert**	**Priority**	**TL MM**	**Assessment**		**Integration**
								Formative	Summative	

Hom UG-Mod. Phar 4.2.1	Drugs used in RA and Gout	K	Knowledge and scholarship	*1. Rheumatoid Arthritis* 1. **Understand Pathophysiology:** Explain the autoimmune nature of RA and the role of cytokines (e.g., TNF-α\alphaα, IL-6) in joint destruction. 2. **Classify RA Drugs:** Differentiate between NSAIDs, corticosteroids, DMARDs, and biologics. 3. **Select Appropriate Therapies:** Design treatment plans based on disease severity and progression. 4. **Monitor for Adverse Effects:** Recognize side effects of DMARDs (e.g., hepatotoxicity with methotrexate) and biologics (e.g., infection risks). *2. Gout* 1. **Identify Pathophysiology:** Explain the role of hyperuricemia and urate crystal deposition in gout	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

				pathogenesis. 2. **Classify Gout Drugs:** Differentiate between acute (NSAIDs, colchicine) and chronic (xanthine oxidase inhibitors, uricosurics) treatments. 3. **Manage Comorbidities:** Incorporate renal and cardiovascular considerations into gout therapy.						

HomUG-Mod.Phar– 4.3 (CNS)

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar **4.3.1**	Anxiolytics	K	Knowledge and scholarship	➢ **Treatment should be individualized** ➢ Minimize long-term use ➢ Balance between efficacy and safety ➢ **Combination with other treatments** ➢ Educate patients on safe use. ➢ Understanding **the pharmacology of anxiolytics**: Learners should be able to explain the different types of anxiolytics (e.g.,	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Neorology Medicine Physiology

				benzodiazepines, selective serotonin reuptake inhibitors (SSRIs), buspirone), their mechanisms of action, and their clinical indications.						
Hom UG-Mod. Phar 4.3.2	Antiepileptics	K	Knowledge and scholarship	• **Personalized Treatment** • Appropriate Monotherapy • Dose Titration and Monitoring • **Minimize Drug Interactions** • Long-Term Management and Monitoring • Consideration of Non-Pharmacological Treatments • Patient Education and Adherence • **Seizure-Free Goal** • **Discontinuation of AEDs**: i. Understanding **the pharmacology of AEDs**: Learners should be able to explain the mechanisms of action of various antiepileptic drugs, their pharmacokinetics, and how they control seizures in different types of epilepsy. ii. Identifying **appropriate drug choices**: Learners should be able to select the most appropriate AED for a given type of epilepsy or seizure disorder based on factors such as seizure type, patient age, comorbidities, and previous response to treatment. iii. Monitoring **drug levels and adjusting doses**: Learners should understand the	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				iv. importance of therapeutic drug monitoring and how to interpret serum drug levels to adjust dosages of AEDs to maintain efficacy while minimizing toxicity. Recognizing **side effects and drug interactions**: Learners should be able to identify the common and serious side effects associated with AEDs, including those related to cognitive function, liver and kidney toxicity, and hematologic effects. They should also understand the potential for drug-drug interactions and how these affect treatment decisions.						
Hom UG-Mod. Phar **4.3.3**	Antipsychotics and Antidepressants	K	Knowledge and scholarship	• Early and Accurate Diagnosis • Choice of Medication • Personalized Treatment • Minimizing Side Effects • Non-Pharmacological Support • Ongoing Monitoring and Adjustments • Patient Education and Adherence • Consideration for Tapering • First-Line Use of SSRIs and SNRIs: Selective serotonin reuptake inhibitors (SSRIs) and serotonin-norepinephrine reuptake inhibitors (SNRIs) • Tailored Treatment • Start Low, Go Slow • Pharmacology **of**	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiolog y

				Antipsychotics and Antidepressants: Learners should understand the mechanisms of action, pharmacokinetics, and clinical indications for both antipsychotics and antidepressants, including the differences between various drug classes (e.g., atypical vs. typical antipsychotics, SSRIs vs. SNRIs).						
Hom UG-Mod. Phar 4.3.4	Opioid Analgesics,	K	Knowledge and scholarship	Accurate Pain Assessment l: intensity, location, and underlying cause, is essential. Pain assessment tools, such as the Numeric Pain Rating Scale or Visual Analog Scale Appropriate Indications: • Acute pain (e.g., postoperative or trauma-related pain). • Chronic pain related to cancer or palliative care. • Severe pain unresponsive to non-opioid therapies. Individualized Treatment Plans: Risk Mitigation: Monitoring and Follow-Up Side Effect Management: • Constipation: Prophylactic use of laxatives. • Nausea/Vomiting: Use of antiemetics if needed. • Sedation: Adjust doses or switch medications if excessive.	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiolog y

				Avoiding Long-Term Use When Possible **Understanding Pharmacology**: Demonstrate knowledge of opioid receptor mechanisms, pharmacokinetics, and pharmacodynamics, including the differences between full agonists, partial agonists, and antagonists. • **Clinical Indications**: Identify appropriate clinical scenarios for opioid use and contraindications to their prescription. • **Recognizing Risks**: Understand the risks of opioid use, including tolerance, dependence, addiction, and respiratory depression. • Pain **Management**: Effectively assess and classify pain to guide the appropriate use of opioids. • Dosing **and Titration**: Accurately calculate and titrate opioid doses, ensuring adequate pain relief while minimizing side effects. • Side **Effect Management**: Develop strategies to anticipate, recognize, and manage opioid-induced side effects. • Naloxone **Use**: Educate patients and caregivers on the use of						

				naloxone for opioid overdose prevention. • Patient-**Centered Care**: Communicate effectively with patients about the benefits, risks, and goals of opioid therapy, fostering shared decision-making.						

HomUG-Mod.Phar– 4.4 (Respiratory System)

Sl.No.	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar **4.4.1**	Drugs for cough	K	Knowledge and scholarship	**1. Accurate Diagnosis of the Cough's Underlying Cause** • A thorough assessment is necessary to identify the underlying cause of the cough, such as a respiratory infection (e.g., viral or bacterial), asthma, post-nasal drip, or more severe conditions like chronic obstructive pulmonary disease (COPD) or heart failure. • Treatment should focus on the underlying cause when possible (e.g., antibiotics for bacterial infections,	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				bronchodilators for asthma), rather than merely masking the symptom. **2. Classification of Cough Type** • **Dry Cough**: Typically non-productive, can be treated with **antitussives** like **dextromethorphan** or **codeine**. • **Productive Cough**: Involves the production of mucus or phlegm and is usually treated with **expectorants** like **guaifenesin** to facilitate mucus clearance. • **Acute vs. Chronic Cough**: Coughs lasting less than three weeks are usually acute and often self-limited, whereas coughs persisting for more than eight weeks may indicate an underlying chronic condition. **3. Symptomatic Treatment** • Use **antitussives** for suppressing persistent dry cough that is uncomfortable or interfering with dailyactivities. Use **expectorants** or mucolytics for productive coughs to aid in the clearance of mucus.						

				Knowledge-Based Outcomes • **Pharmacological Understanding**: Demonstrate an understanding of the pharmacokinetics, pharmacodynamics, and mechanisms of action of drugs used for cough, including antitussives (e.g., dextromethorphan, codeine) and expectorants (e.g., guaifenesin). • **Cough Etiology**: Identify and differentiate the causes of acute and chronic cough and understand the role of drugs in treating these causes based on the type of cough (dry vs. productive). **Drug Safety and Side Effects**: Recognize the potential side effects and safety concerns of commonly used cough medications, such as opioids, and other non-opioid alternatives, ensuring safe use in different patient populations						
Hom UG-Mod. Phar **4.4.2**	Bronchial asthma and COPD	K	Knowledge and scholarship	*1. Accurate Diagnosis and Differentiation* • **Asthma** is typically characterized by reversible airway obstruction and inflammation, often triggered by allergens, irritants, or	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				exercise. • **COPD** is characterized by progressive, irreversible airflow limitation, most commonly caused by long-term exposure to smoking or environmental pollutants. • Early diagnosis, based on clinical symptoms, spirometry, and patient history, is essential for effective treatment. *2. Individualized Pharmacological Treatment* • **Asthma** treatment often involves controlling inflammation and bronchoconstriction, with a focus on symptom control and preventing exacerbations. • **COPD** treatment emphasizes symptom management, reducing exacerbations, and slowing disease progression. • The choice of medication should be tailored to the patient's age, disease severity, comorbidities, and frequency of exacerbations. *3. Stepwise Approach to Asthma Management* • **Step 1 (Mild Asthma)**: Use **short-acting beta-agonists (SABA)** like **albuterol** for						

				symptom relief. • **Step 2 (Persistent Asthma)**: Add **inhaled corticosteroids (ICS)** like **beclometasone** or **budesonide** to control inflammation. • **Step 3 (Moderate Asthma)**: Use **long-acting beta-agonists (LABA)** like **salmeterol** in combination with ICS to enhance bronchodilation and reduce inflammation. • **Step 4 (Severe Asthma)**: Add **leukotriene receptor antagonists (LTRAs)** like **montelukast** or consider **biologics** like **omalizumab** or **dupilumab** for severe cases. *4. COPD Pharmacological Management* • **Short-Acting Bronchodilators**: **SABA** (e.g., **salbutamol**) and **short-acting muscarinic antagonists (SAMA)** (e.g., **ipratropium**) provide quick relief for symptoms. • **Long-Acting Bronchodilators**: **LABA** (e.g., **formoterol**) and **long-acting muscarinic antagonists (LAMA)** (e.g., **tiotropium**) are used for long-term management of airflow obstruction.						

				• **Inhaled Corticosteroids (ICS)**: Used in combination with bronchodilators for patients with frequent exacerbations (e.g., **fluticasone**). *5. Monitoring and Adjusting Treatment* • Monitor **lung function** (using **spirometry**), symptoms, and exacerbation frequency to assess the efficacy of treatment and adjust medications accordingly. • For asthma, adjust treatment in response to symptom control or frequency of exacerbations, considering a **step-up** or **step-down** approach based on disease control. *For COPD, monitor symptoms, exacerbation history, and comorbidities to adjust the therapeutic regimen.* **Knowledge-Based Outcomes** • **Pathophysiology**: Demonstrate understanding of the underlying pathophysiological mechanisms of **asthma** and **COPD**, including inflammation, airway remodeling, and bronchoconstriction.						

				• **Pharmacological Principles**: Understand the classes of drugs used in asthma and COPD, including bronchodilators, corticosteroids, and other adjunctive therapies, and their mechanisms of action. • **Diagnosis and Differentiation**: Differentiate between asthma and COPD based on clinical presentation, diagnostic tests (spirometry, chest imaging), and patient history. • **Exacerbation Management**: Recognize the appropriate use of medications in managing asthma and COPD exacerbations, including the role of systemic corticosteroids, bronchodilators, and antibiotics. • **Inhaler Technique and Patient Education**: Educate patients on the proper use of inhalers, ensuring correct technique to maximize drug efficacy, and provide guidance on lifestyle modifications and non-pharmacological interventions.						

HomUG-Mod.Phar– 4.5 (Hormones)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar 4.5.1	Insulin and oral Hypoglycemic drugs	K	Knowledge and scholarship	*1. Accurate Diagnosis and Classification of Diabetes* • **Type 1 Diabetes (T1D)**: An autoimmune disorder where the pancreas produces little to no insulin, requiring lifelong insulin therapy. • **Type 2 Diabetes (T2D)**: Characterized by insulin resistance and eventual pancreatic beta-cell dysfunction, managed with a combination of lifestyle changes and pharmacological therapy. *2. Insulin Therapy in Diabetes Management* **Insulin for Type 1 Diabetes**:	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				• **Basal Insulin**: Provides a constant level of insulin, mimicking the body's natural insulin secretion (e.g., **insulin glargine**, **insulin detemir**). • **Bolus Insulin**: Used to control postprandial blood glucose levels (e.g., **insulin aspart**, **insulin lispro**). • **Continuous Insulin Infusion (Insulin Pump)**: For some Type 1 diabetic patients, especially those who need more precise control of their blood sugar. **Insulin for Type 2 Diabetes**: • Initially, T2D patients may be managed with **oral hypoglycemic agents** but may eventually require insulin as the disease progresses and pancreatic function declines. • **Combination Therapy**: Insulin may be combined with **oral hypoglycemic drugs** like **metformin** or **sulfonylureas** for enhanced control. *3. Oral Hypoglycemic Agents (OHAs) in Type 2 Diabetes* **Biguanides (e.g., Metformin)**: • First-line therapy for T2D. • Works by decreasing hepatic						

				glucose production and improving insulin sensitivity, without increasing insulin secretion (reducing the risk of hypoglycemia). **Sulfonylureas (e.g., Glibenclamide, Glimepiride)**: • Stimulate the pancreas to secrete more insulin. • Typically used when metformin alone is not enough to control blood sugar. **Thiazolidinediones (TZDs) (e.g., Pioglitazone)**: • Improve insulin sensitivity by acting on the **peroxisome proliferator-activated receptor gamma (PPAR-γ)** in the muscles and adipose tissue. **Dipeptidyl Peptidase-4 Inhibitors (DPP-4 inhibitors) (e.g., Sitagliptin, Saxagliptin)**: • Enhance the action of incretin hormones, which increase insulin release and decrease glucagon secretion in a glucose-dependent manner. **SGLT2 Inhibitors (e.g.,**						

				Empagliflozin, Dapagliflozin): • Work by inhibiting sodium-glucose cotransporter 2 in the kidneys, leading to increased glucose excretion in the urine and lowering blood glucose levels. **GLP-1 Receptor Agonists (e.g., Exenatide, Liraglutide)**: • Increase insulin secretion in response to meals, reduce glucagon secretion, and delay gastric emptying, leading to weight loss. *4. Monitoring and Adjusting Treatment* • **Self-Monitoring of Blood Glucose (SMBG)**: Regular blood glucose testing is essential to assess treatment efficacy and guide adjustments. • **Hemoglobin A1c (HbA1c)**: A key long-term marker used to assess the effectiveness of treatment. The target for most diabetic patients is usually an A1c below 7%, but individual goals may vary. • **Renal Function**: Regularly monitor **renal function** in patients on drugs like **metformin**, **SGLT2**						

				inhibitors, or **insulin** to avoid complications, particularly in those with compromised kidney function. *5. Prevention and Management of Hypoglycemia* • **Insulin Therapy**: Hypoglycemia is a common risk, especially with **insulin** or **sulfonylureas**. Patients should be educated on recognizing early signs of hypoglycemia (e.g., sweating, dizziness) and how to correct it (e.g., consuming glucose). • **Oral Hypoglycemics**: Drugs like **SGLT2 inhibitors** and **DPP-4 inhibitors** are less likely to cause hypoglycemia compared to insulin or sulfonylureas. • **Patient Education**: Teach patients about diet, medication timing, and how exercise can affect blood sugar levels, helping them avoid hypoglycemic episodes. *6. Management of Comorbidities* • **Cardiovascular Disease**: Many diabetic patients have comorbid cardiovascular conditions. **SGLT2 inhibitors** and **GLP-1 agonists** have						

				shown cardiovascular benefits. • **Kidney Disease**: Monitor kidney function regularly, especially in patients receiving **metformin**, **SGLT2 inhibitors**, or **insulin**. *7. Long-Term Management and Lifestyle Support* • Diabetes is a chronic condition requiring long-term management. **Lifestyle support** (including diet, exercise, and weight management) should be integrated into therapy. Encourage patient engagement and regular follow-ups to ensure proper management and prevent complications. **Knowledge-Based Outcomes** 1. **Pharmacodynamics and Pharmacokinetics**: Demonstrate an understanding of the mechanisms of action, pharmacokinetics, and therapeutic use of **insulin** and **oral hypoglycemic drugs** such as **metformin**, **sulfonylureas**, **SGLT2 inhibitors**, and **GLP-1 receptor agonists**. 2. **Diabetes Classification and**						

				Pathophysiology: Understand the different types of diabetes (Type 1, Type 2) and the pathophysiological mechanisms underlying **insulin resistance** and **beta-cell dysfunction**. **Drug Safety**: Recognize the adverse effects, contraindications, and precautions of insulin and oral hypoglycemics, including the risk of **hypoglycemia** and **kidney impairment**						
Hom UG-Mod. Phar **4.5.2**	Adrenocortical and Androgenic steroids	K	Knowledge and scholarship	**Adrenocortical Steroids** *1. Differentiating Glucocorticoids and Mineralocorticoids* ***Glucocorticoids (e.g., Prednisone, Dexamethasone)**:* • Primary role: Anti-inflammatory and immunosuppressive effects. • Used for: Asthma, rheumatoid arthritis, autoimmune diseases, and allergies. • Adverse effects: Long-term use can lead to osteoporosis, diabetes, and adrenal suppression. **Mineralocorticoids (e.g., Fludrocortisone)**: • Primary role: Regulation of	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				electrolyte and water balance via actions on renal tubules. • Used for: Addison's disease and other conditions involving adrenal insufficiency. • Adverse effects: Sodium retention, hypokalemia, and hypertension. *2. Rational Use in Therapy* Select drugs based on their **potency**, **duration of action**, and **specific indications**: • **Short-acting glucocorticoids**: Hydrocortisone. • **Intermediate-acting**: Prednisone, Methylprednisolone. • **Long-acting**: Dexamethasone, Betamethasone. *3. Managing Adverse Effects* • Monitor for **iatrogenic Cushing's syndrome** with long-term glucocorticoid therapy. • Prevent complications such as **osteoporosis** by supplementing calcium and vitamin D. • Regularly check for **hypertension**, **hyperglycemia**, and						

				infections. *4. Anti-Inflammatory and Immunosuppressive Therapy* • Glucocorticoids are used to manage inflammatory and autoimmune diseases such as **lupus**, **rheumatoid arthritis**, and **inflammatory bowel disease**. • Their use in transplantation helps prevent graft rejection through immunosuppressive effects**for Androgenic Steroid Drugs** *1. Therapeutic Indications* • **Primary or Secondary Hypogonadism**: Use testosterone or its esters to restore normal androgen levels. • **Anabolic Effects**: Promote nitrogen retention and muscle growth in catabolic states such as severe burns or chronic illnesses. ***Other Uses**: Certain androgenic steroids are used for endometriosis, hereditary angioedema, or as part of hormone therapy in transgender men.*						

				Knowledge-Based Outcomes **Pharmacology**: Understand the **mechanism of action**, **pharmacokinetics**, and **therapeutic uses** of glucocorticoids, mineralocorticoids, and androgenic steroids.						
Hom UG-Mod. Phar **4.5.3**	Estrogens, Progesterone and OCPs	K	Knowledge and scholarship	**Estrogens and Progesterone** *1. Physiological Roles* **Estrogens** • Regulate the development of female secondary sexual characteristics. • Essential for the menstrual cycle and maintaining bone density. • Common examples:**Estradiol**, **Ethinyl Estradiol**, and **Conjugated Estrogens**. **Progesterone**: • Regulates the menstrual cycle and maintains pregnancy. • Plays a role in the endometrial transformation required for implantation. • Common examples: **Progesterone**, **Medroxyprogesterone**	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				acetate, and **Norethindrone**. *3. Therapeutic Applications* ***Estrogens**:* • Used for **hormone replacement therapy (HRT)** in menopausal women. • Management of **hypogonadism** in females. • Treatment of **osteoporosis** and certain cancers (e.g., prostate cancer in men). **Progesterone**: • Used in combination with estrogens in HRT to prevent **endometrial hyperplasia**. • Indicated for **abnormal uterine bleeding**, **amenorrhea**, and **endometriosis**. *3. Rational Use* • Combine estrogens and progestins in women with an intact uterus to prevent **endometrial carcinoma**. • Use the **lowest effective dose** for the shortest duration to minimize risks like thromboembolism or cancer. • For menopausal women, use HRT only for **vasomotor**						

				symptoms, vaginal atrophy, or osteoporosis prevention. *4. Monitoring and Adverse Effects* **Estrogens**: • Risks include **thromboembolism**, **breast cancer**, and **endometrial hyperplasia**. • Monitor liver function, lipid profile, and coagulation parameters. **Progesterone**: • Can cause **mood changes**, **weight gain**, and **irregular bleeding**. • Monitor for symptoms of depression and cardiovascular risks. **Oral Contraceptive Pills (OCPs)** *1. Types of OCPs* **Combined Oral Contraceptives (COCs)**: • Contain both estrogen (e.g., **Ethinyl Estradiol**) and a progestin (e.g., **Levonorgestrel**, **Drospirenone**). • Mechanism: Suppress						

				ovulation, thicken cervical mucus, and alter the endometrium. **Progestin-Only Pills (POPs)**: • Contain only progestins. • Mechanism: Thicken cervical mucus and inhibit ovulation. • Preferred for women who are breastfeeding or have contraindications to estrogens. *2. Therapeutic Applications* **Contraception**: • Primary use of OCPs to prevent pregnancy. **Non-Contraceptive Uses**: • Management of **menstrual disorders** like dysmenorrhea or menorrhagia. • Treatment of **endometriosis**. • Reduction in the risk of ovarian and endometrial cancers. *3. Selection of OCPs* Choose based on patient needs, medical history, and side effect profile. • **Low-dose COCs**: Preferred						

				for minimizing estrogen-related side effects. • **POPs**: Recommended for women with risk factors like thromboembolism. *4. Safety and Monitoring* Screen for contraindications to OCPs, including: • **Thromboembolic disorders**. • **Breast cancer** or other estrogen-dependent cancers. • **Uncontrolled hypertension** or **migraine with aura**. • Monitor for **breakthrough bleeding**, **mood changes**, and **weight gain**. *5. Managing Side Effects* • Adjust the estrogen or progestin dose for symptoms like **nausea**, **headaches**, or **irregular bleeding**. Educate on the importance of adherence and steps to take if a dose is missed. *Knowledge-Based Outcomes* 1. **Pharmacodynamics and Pharmacokinetics**: Understand the mechanisms of action, absorption,						

				metabolism, and elimination of estrogens, progesterone, and OCPs. **Indications and Contraindications**: Learn the therapeutic uses of these drugs, along with contraindications like a history of thromboembolism or breast cancer.						
Hom UG-Mod. Phar **4.5.4**	Vitamin D, Calcium and Drugs affecting calcium Balance	K	Knowledge and scholarship	**1.Physiological Roles** **Vitamin D**: • Regulates calcium and phosphate absorption from the gut. • Facilitates bone mineralization and remodeling. • Active forms: **Calcitriol** (1,25-dihydroxyvitamin D) and **Cholecalciferol** (Vitamin D3). **Calcium**: • Essential for bone structure, muscle contraction, nerve transmission, and blood clotting. • Daily requirement depends on age, gender, and physiological state (e.g., pregnancy, lactation).	C1	MK	Lecture, Group discussio n	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiolog y

				2. Therapeutic Applications **Vitamin D**: • Treats **rickets**, **osteomalacia**, **hypocalcemia**, and **secondary hyperparathyroidism**. • Used for **osteoporosis prevention and treatment**. **Calcium**: • Supplementation for **hypocalcemia**, **osteoporosis**, and dietary insufficiency. • Adjunct in **cardiac arrest** (calcium chloride or calcium gluconate). **3. Rational Use** • Ensure adequate **calcium intake** (dietary or supplemental) alongside vitamin D therapy. • Use the **active form (Calcitriol)** in cases of **renal impairment**, where vitamin D activation is impaired. • Tailor dosage to patient needs, age, and comorbidities: o Vitamin D: Typically 400–800 IU/day for adults, higher doses for deficiency. o Calcium: 1,000–1,200 mg/day for						

				adults. **4. Monitoring and Adverse Effects** • Monitor **serum calcium** and **vitamin D levels** to avoid **hypercalcemia** and **toxicity**. • Watch for symptoms of excess, such as **nausea**, **constipation**, and **renal calculi**. In high-risk populations (e.g., elderly), ensure supplements do not exceed tolerable upper intake levels. **Knowledge-Based Outcomes** 1. **Mechanisms of Action**: Understand how vitamin D, calcium, and calcium-modulating drugs affect bone remodeling and calcium homeostasis. 2. **Indications and Dosage**: Learn the appropriate indications, dosages, and formulations for conditions like **osteoporosis**, **rickets**, and **hypercalcemia**. **Adverse Effects**: Recognize common side effects, including **hypercalcemia**, **renal complications**, and **osteonecrosis**.						

HomUG-Mod.Phar– 4.6 (CVS)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar **4.6.1**	T/t of Hypertension	K	Knowledge and scholarship	**Hypertension Management** *1. Etiology and Pathophysiology* Identify the cause of hypertension: • **Primary (essential)** hypertension: Idiopathic but influenced by genetic and lifestyle factors. • **Secondary** hypertension: Caused by conditions such as renal artery stenosis, endocrine disorders, or	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				medication use. • Understand the pathophysiological mechanisms: • **Increased systemic vascular resistance**, **volume overload**, and **sympathetic nervous system activation**. *2. Therapeutic Goals* • The primary goal is to reduce blood pressure to recommended targets: o **<140/90 mmHg** for most adults. o **<130/80 mmHg** for patients with diabetes, chronic kidney disease, or a history of cardiovascular disease. • Prevent complications such as stroke, myocardial infarction, and organ damage. *3. Pharmacological Treatment Options* Hypertension treatment requires patient-specific drug selection based on comorbidities, age, and ethnicity: 1. **First-Line Agents**: • **Thiazide Diuretics** (e.g.,						

				Hydrochlorothiazide, Chlorthalidone): Reduce blood volume by increasing urinary excretion of sodium and water. • **ACE Inhibitors** (e.g., Enalapril, Ramipril): Inhibit the renin-angiotensin-aldosterone system (RAAS), reducing vasoconstriction and aldosterone secretion. • **Angiotensin II Receptor Blockers (ARBs)** (e.g., Losartan, Valsartan): Block angiotensin II receptors, reducing vasoconstriction. • **Calcium Channel Blockers (CCBs)** (e.g., Amlodipine, Nifedipine): Cause vasodilation by inhibiting calcium influx in vascular smooth muscle. 2. **Second-Line and Add-On Agents**: • **Beta-Blockers** (e.g., Metoprolol, Atenolol): Reduce heart rate and cardiac output. • **Aldosterone Antagonists** (e.g., Spironolactone, Eplerenone): Effective in resistant hypertension. • **Alpha-Blockers** (e.g., Doxazosin): Relax vascular smooth muscle						

				to lower BP. 3. **Other Agents**: • **Centrally Acting Drugs** (e.g., Clonidine, Methyldopa): Reduce sympathetic outflow. • **Direct Vasodilators** (e.g., Hydralazine, Minoxidil): Reserved for severe or refractory hypertension. --- *4. Non-Pharmacological Measures* Emphasize lifestyle modifications for all patients: • **Dietary Approaches to Stop Hypertension (DASH)** diet: Rich in fruits, vegetables, and low-fat dairy. • **Salt reduction**: <2.3 grams of sodium/day. • **Regular exercise**: At least 150 minutes of moderate-intensity aerobic activity per week. • **Weight loss**: Aim for BMI <25 kg/m². • **Smoking cessation** and limiting alcohol intake. **Knowledge-Based Outcomes** 1. **Understanding**						

				Pathophysiology: Describe the mechanisms contributing to hypertension and the pharmacological targets of antihypertensive drugs. 2. **Therapeutic Principles**: Understand the indications, mechanisms, and contraindications of different antihypertensive drugs. 3. **Risk Stratification**: Learn to assess cardiovascular risk and tailor treatment to reduce complications. **Skill-Based Outcomes** **Blood Pressure Measurement**: Accurately measure and interpret blood pressure values using standard techniques.						
Hom UG-Mod. Phar **4.6.2**	Angina, MI	K	Knowledge and scholarship	**Angina** *1. Types of Angina* • **Stable Angina**: Caused by a fixed coronary artery obstruction. • **Unstable Angina**: Part of acute coronary syndrome (ACS), often due to plaque rupture. • **Prinzmetal (Variant) Angina**: Caused by coronary	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				vasospasm. *2. Goals of Therapy* • Alleviate symptoms by improving oxygen supply-demand balance. • Prevent progression to MI or sudden cardiac death. • Improve quality of life and physical activity tolerance. *3. Drug Classes for Angina* 1. **Nitrates** (e.g., Nitroglycerin, Isosorbide Dinitrate): o Mechanism: Reduce preload and afterload by venodilation; improve coronary blood flow. o Indication: Acute relief (sublingual nitroglycerin) and long-term prophylaxis (oral or transdermal nitrates). o Adverse Effects: Headache, hypotension, reflex tachycardia. 2. **Beta-Blockers** (e.g., Metoprolol, Atenolol): o Mechanism: Reduce heart rate and contractility, lowering myocardial						

				oxygen demand. ○ Indication: First-line for stable angina; reduce mortality in post-MI patients. ○ Adverse Effects: Bradycardia, fatigue, bronchospasm. 3. **Calcium Channel Blockers (CCBs)** (e.g., Amlodipine, Verapamil): ○ Mechanism: Reduce afterload by vasodilation and decrease myocardial oxygen demand. ○ Indication: Prinzmetal angina, stable angina in patients intolerant to beta-blockers. ○ Adverse Effects: Hypotension, peripheral edema, constipation. 4. **Antiplatelet Agents** (e.g., Aspirin, Clopidogrel): ○ Mechanism: Prevent platelet aggregation and thrombus formation. ○ Indication: Prevent acute coronary syndromes. ○ Adverse Effects: Bleeding, gastrointestinal irritation. 5. **Ranolazine**:						

				○ Mechanism: Inhibits late sodium currents, reducing ischemia. ○ Indication: Chronic stable angina refractory to standard therapy. ○ Adverse Effects: QT prolongation. **Drugs Used in Myocardial Infarction (MI)** *1. Pathophysiology* • MI is caused by complete or partial coronary artery occlusion, leading to ischemia and myocardial necrosis. *2. Goals of Therapy* • Restore coronary perfusion and limit infarct size. • Prevent complications such as arrhythmias, heart failure, and reinfarction. • Reduce mortality and improve long-term outcomes. *3. Drug Classes for MI* 1. **Thrombolytics (Fibrinolytics)** (e.g., Alteplase, Streptokinase): ○ Mechanism: Dissolve thrombi by activating						

				plasminogen to plasmin. ○ Indication: STEMI when percutaneous coronary intervention (PCI) is unavailable. ○ Adverse Effects: Bleeding, intracranial hemorrhage. 2. **Antiplatelet Agents**: ○ **Aspirin**: Irreversible COX-1 inhibition reduces thromboxane A2, preventing platelet aggregation. ○ **P2Y12 Inhibitors** (e.g., Clopidogrel, Ticagrelor): Block ADP-mediated platelet activation. ○ Used in dual antiplatelet therapy (DAPT) for ACS and post-PCI. 3. **Anticoagulants** (e.g., Enoxaparin, Heparin): ○ Mechanism: Prevent clot propagation by inhibiting clotting factors. ○ Indication: Acute phase of MI to reduce thrombotic risk. ○ Adverse Effects: Bleeding, heparin-induced thrombocytopenia (HIT). 4. **Beta-Blockers**:						

				○ Reduce myocardial oxygen demand, preventing arrhythmias and infarct expansion. 5. **ACE Inhibitors/ARBs**: ○ Prevent ventricular remodeling and reduce afterload. ○ Indication: Post-MI with heart failure or reduced ejection fraction. ○ Adverse Effects: Cough (ACE inhibitors), hyperkalemia. 6. **Statins** (e.g., Atorvastatin, Rosuvastatin): ○ Mechanism: Lower LDL cholesterol and stabilize plaques. ○ Indication: All MI patients irrespective of baseline cholesterol levels. 7. **Nitrates**: ○ Relieve ischemic pain by improving coronary perfusion. 8. **Aldosterone Antagonists** (e.g., Spironolactone): Indication: Post-MI with heart failure or reduced ejection fraction. **Knowledge-Based Outcomes**						

				1. **Pathophysiology Understanding**: Describe the mechanisms underlying angina and MI, including the role of oxygen supply-demand mismatch and thrombosis. 2. **Drug Mechanisms and Indications**: Understand the mechanism of action, indications, and contraindications of drugs used in angina and MI. 3. **Guideline Awareness**: Familiarity with current guidelines for managing stable angina, unstable angina, and MI. **Skill-Based Outcomes** 4. **Drug Selection**: Select the most appropriate therapy for angina or MI based on patient-specific factors, such as comorbidities and contraindications. 5. **Acute Management Skills**: Administer appropriate acute therapies for MI (e.g., thrombolysis, dual antiplatelet therapy). 6. **Monitoring and Adjustment**: Monitor therapy effectiveness (e.g., relief of chest pain, BP control) and adjust based on patient response and side						

				effects. **Attitude-Based Outcomes** 7. **Patient-Centered Care**: Prioritize patient preferences and risk factors in designing treatment plans. 8. **Preventive Focus**: Educate patients on lifestyle changes (e.g., smoking cessation, diet, exercise) to prevent angina and recurrent MI. 9. **Ethical Prescribing**: Avoid polypharmacy and adhere to evidence-based practices for reducing cardiovascular morbidity and mortality.						
Hom UG-Mod. Phar **4.6.3**	Cardiac Glycosides and Drugs for Heart failure	K	Knowledge and scholarship	**Cardiac Glycosides** *1. Mechanism of Action* • **Digoxin**, the primary cardiac glycoside, works by: o Inhibiting the **Na^+/K^+-ATPase pump**, leading to increased intracellular sodium. o Increased sodium decreases calcium extrusion via the sodium-calcium exchanger, leading to	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				higher intracellular calcium and stronger myocardial contractions (positive inotropy). ○ Slowing conduction through the atrioventricular (AV) node and increasing vagal tone (negative chronotropy). *2. Indications* • Symptomatic relief in **chronic heart failure** (especially in patients with reduced ejection fraction). • Management of **atrial fibrillation** with rapid ventricular rates, especially in heart failure patients. *3. Adverse Effects* • **Cardiac**: Arrhythmias (e.g., ventricular tachycardia, AV block). • **Gastrointestinal**: Nausea, vomiting, diarrhea. • **Neurological**: Visual disturbances (e.g., yellow vision), confusion. • **Toxicity**: Narrow therapeutic index necessitates careful monitoring of drug levels						

				(normal range: 0.5–2 ng/mL). *4. Monitoring* • Regular assessment of digoxin levels, renal function, and electrolytes (hypokalemia and hypomagnesemia increase toxicity risk). **Drugs Used in Heart Failure** Heart failure management varies based on the type: • **Heart Failure with Reduced Ejection Fraction (HFrEF)**: EF ≤40%. • **Heart Failure with Preserved Ejection Fraction (HFpEF)**: EF >50%. *1. Goals of Therapy* • Alleviate symptoms (e.g., dyspnea, fatigue). • Reduce hospitalizations and mortality. • Prevent disease progression and improve quality of life. *2. Drug Classes* 1. **First-Line Drugs**: **Angiotensin-Converting Enzyme**						

				(ACE) Inhibitors (e.g., Enalapril, Ramipril): • Mechanism: Reduce afterload and preload by blocking the renin-angiotensin-aldosterone system (RAAS). • Benefits: Decrease mortality and slow disease progression. • Adverse Effects: Cough, hyperkalemia, angioedema. **Angiotensin II Receptor Blockers (ARBs)** (e.g., Losartan, Valsartan): • Alternative to ACE inhibitors in patients with intolerance. • **Beta-Blockers** (e.g., Bisoprolol, Carvedilol, Metoprolol): • Mechanism: Block sympathetic overactivation. • Benefits: Reduce mortality and improve left ventricular function. • Adverse Effects: Bradycardia, fatigue, hypotension. **Mineralocorticoid Receptor Antagonists (MRAs)** (e.g., Spironolactone, Eplerenone): • Mechanism: Block aldosterone, reducing fluid retention and fibrosis. • Benefits: Mortality reduction in HFrEF.						

				• Adverse Effects: Hyperkalemia, gynecomastia (spironolactone). **2.Symptomatic Relief**: **Loop Diuretics** (e.g., Furosemide, Torsemide): • Mechanism: Increase urinary excretion of sodium and water. • Indication: Relieve fluid overload symptoms (e.g., pulmonary congestion, peripheral edema). • Adverse Effects: Hypokalemia, hypovolemia. • **Thiazide Diuretics** (e.g., Hydrochlorothiazide): • Used in mild fluid retention or combination therapy. **3.Novel Therapies**: • **Sodium-Glucose Cotransporter-2 (SGLT2) Inhibitors** (e.g., Dapagliflozin, Empagliflozin): • Benefits: Mortality reduction, symptom improvement, regardless of diabetes status. **4.Other Agents**: **Ivabradine**:						

				• Mechanism: Reduces heart rate by inhibiting the If current in the sinoatrial node. • Indication: HFrEF with resting heart rate >70 bpm despite beta-blocker use. **Hydralazine and Isosorbide Dinitrate**: • Indication: HFrEF in African-American patients or those intolerant to ACE inhibitors/ARBs. **5.Anticoagulation and Antiplatelets**: Indicated in HF patients with atrial fibrillation or thromboembolic risk. **Knowledge-Based Outcomes** 1. **Understanding Mechanisms**: Describe the pharmacological actions of cardiac glycosides and other HF drugs. 2. **Pathophysiology**: Understand the role of neurohormonal dysregulation in HF and the impact of pharmacotherapy. 3. **Evidence-Based Practices**: Learn to apply current guidelines in the pharmacological management of HF.						

				Skill-Based Outcomes 4. **Drug Selection**: Select appropriate drugs for HFrEF, HFpEF, and symptom management. 5. **Toxicity Monitoring**: Identify and manage digoxin toxicity and other drug-related adverse effects. **Patient Counseling**: Educate patients on medication adherence, recognizing symptoms of fluid overload, and dietary restrictions.						

Hom UG-Mod. Phar 4.6.4	Hypolipidemic drugs	K	Knowledge and scholarship	**1. Classes of Hypolipidemic Drugs**
				A. HMG-CoA Reductase Inhibitors (Statins)
				
				• **Examples**: Atorvastatin, Rosuvastatin, Simvastatin.
				• **Mechanism**: Inhibit HMG-CoA reductase, the rate-limiting enzyme in cholesterol synthesis, reducing LDL cholesterol.
				• **Benefits**:
				  ○ Decrease LDL by 20–60%.
				  ○ Modest increase in HDL and decrease in triglycerides.
				  ○ Anti-inflammatory and plaque-stabilizing effects.
					C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				• **Indications**:
				  ○ Primary and secondary prevention of atherosclerotic cardiovascular disease (ASCVD).
				  ○ Hypercholesterolemia.
				
				B. Fibrates
				
				• **Examples**: Fenofibrate, Gemfibrozil.
				• **Mechanism**: Activate peroxisome proliferator-activated receptor-alpha (PPAR-α), increasing fatty acid oxidation and lipoprotein lipase activity, leading to reduced triglycerides.
				• **Benefits**:
				  ○ Decrease triglycerides by 30–50%.
				  ○ Modest increase in HDL.
				• **Indications**: Hypertriglyceridemia, prevention of pancreatitis.
				
				C. Bile Acid Sequestrants
				
				• **Examples**: Cholestyramine, Colestipol.
				• **Mechanism**: Bind bile acids in the intestine, preventing reabsorption and promoting						

				cholesterol excretion.
				• **Benefits**:
				  ○ Lower LDL by 10–20%.
				  ○ May slightly increase HDL.
				• **Indications**: Hypercholesterolemia, particularly in statin-intolerant patients.
				
				D. Cholesterol Absorption Inhibitors
				
				• **Example**: Ezetimibe.
				• **Mechanism**: Inhibits Niemann-Pick C1-Like 1 (NPC1L1) protein in the intestine, reducing dietary and biliary cholesterol absorption.
				• **Benefits**:
				  ○ Reduces LDL by 18–25%.
				  ○ Additive effect with statins.
				• **Indications**: Hypercholesterolemia, adjunct to statins.
				
				E. PCSK9 Inhibitors
				
				• **Examples**: Evolocumab, Alirocumab.
				• **Mechanism**: Monoclonal antibodies that inhibit PCSK9, increasing LDL receptor recycling and reducing LDL levels.						

				• **Benefits**:
				  ○ Lower LDL by 50–70%.
				  ○ Reduce cardiovascular events.
				• **Indications**: Severe hypercholesterolemia, familial hypercholesterolemia, and patients with ASCVD.
				
				F. Nicotinic Acid (Niacin)
				
				• **Mechanism**: Reduces hepatic synthesis of triglycerides and VLDL, increasing HDL.
				• **Benefits**:
				  ○ Reduces triglycerides and LDL.
				  ○ Significantly increases HDL.
				• **Adverse Effects**: Flushing, hyperglycemia, hepatotoxicity.
				• **Indications**: Mixed dyslipidemias.
				
				G. Omega-3 Fatty Acids
				
				• **Examples**: Eicosapentaenoic acid (EPA), Docosahexaenoic acid (DHA).
				• **Mechanism**: Reduce hepatic triglyceride synthesis.
				• **Benefits**:
				  ○ Decrease triglycerides.
				  ○ Anti-inflammatory						

				and anti-arrhythmic properties. • **Indications**: Hypertriglyceridemia. **2. Principles of Therapy** • **Lifestyle Modifications**: Emphasize diet, exercise, and smoking cessation alongside pharmacotherapy. **3. Monitoring and Safety** • **Lipid Levels**: Monitor baseline and follow-up lipid profiles to assess efficacy. • **Liver Function**: Regularly check liver enzymes, especially with statins and niacin. • **Muscle Toxicity**: Monitor for myalgia or rhabdomyolysis in statin-treated patients. • **Glycemic Control**: Be cautious of hyperglycemia in susceptible individuals on statins or niacin. **Adverse Effects of Hypolipidemic Drugs** • **Statins**: Myopathy, rhabdomyolysis, hepatotoxicity, increased diabetes risk. • **Fibrates**: Myopathy						

				(especially with statins), gallstones. • **Bile Acid Sequestrants**: Gastrointestinal discomfort, vitamin deficiencies (A, D, E, K). • **Niacin**: Flushing, pruritus, hepatotoxicity. • **PCSK9 Inhibitors**: Injection site reactions, rare hypersensitivity.						

HomUG-Mod.Phar– 4.7 (Renal System)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
HomUG-Mod.Phar 4.7.1	Diuretics and Antidiuretics	K	Knowledge and scholarship	**Diuretics: Classes and Mechanism of Action** Diuretics are classified based on their site of action in the nephron and their mechanism of action. *A. Thiazide Diuretics* • **Examples**: Hydrochlorothiazide,	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				Chlorthalidone. • **Mechanism**: Inhibit sodium-chloride symporters in the distal convoluted tubule, promoting sodium and water excretion. • **Effects**: Decrease blood volume and lower blood pressure. • **Indications**: Hypertension, mild heart failure, edema due to kidney disease, and nephrolithiasis (calcium-containing stones). • **Adverse Effects**: Hypokalemia, hyponatremia, hyperglycemia, hyperuricemia. *B. Loop Diuretics* • **Examples**: Furosemide, Bumetanide, Torsemide. • **Mechanism**: Inhibit the sodium-potassium-chloride co-transporter in the thick ascending limb of the loop of Henle, preventing sodium reabsorption. • **Effects**: Cause significant diuresis, lowering fluid overload rapidly. • **Indications**: Acute heart failure, pulmonary edema, chronic kidney disease, severe edema. • **Adverse Effects**: Hypokalemia, dehydration,						

				hypotension, ototoxicity.
				
				C. Potassium-Sparing Diuretics
				
				• **Examples**: Spironolactone, Eplerenone, Amiloride, Triamterene.
				• **Mechanism**:
				  ○ **Aldosterone antagonists** (e.g., Spironolactone, Eplerenone) block the effects of aldosterone in the collecting ducts, preventing sodium retention and potassium excretion.
				  ○ **Epithelial sodium channel blockers** (e.g., Amiloride, Triamterene) inhibit sodium reabsorption in the collecting tubules.
				• **Effects**: Reduce sodium retention without causing significant potassium loss.
				• **Indications**: Hyperaldosteronism, heart failure (as adjunct), hypertension, and cirrhosis with ascites.
				• **Adverse Effects**: Hyperkalemia, gynecomastia (with spironolactone), metabolic acidosis.						

				D. Carbonic Anhydrase Inhibitors • **Examples**: Acetazolamide. • **Mechanism**: Inhibit carbonic anhydrase in the proximal convoluted tubule, preventing bicarbonate reabsorption and increasing urine output. • **Effects**: Mild diuresis, alkalinization of urine. • **Indications**: Glaucoma, metabolic alkalosis, altitude sickness. • **Adverse Effects**: Metabolic acidosis, kidney stones, hypokalemia. *E. Osmotic Diuretics* • **Examples**: Mannitol. • **Mechanism**: Increase osmolarity of the filtrate, preventing water reabsorption in the proximal tubule and descending loop of Henle. • **Effects**: Draw water into the renal tubules, promoting diuresis. • **Indications**: Cerebral edema, increased intraocular pressure, acute renal failure. • **Adverse Effects**: Dehydration, hyperkalemia, electrolyte imbalances. **2. Antidiuretics: Mechanism and Use**						

				Antidiuretics, or **diuretic inhibitors**, are used to reduce urine output, typically by increasing water reabsorption in the kidneys. *A. Antidiuretic Hormone (ADH) Analogs* • **Examples**: Desmopressin, Vasopressin. • **Mechanism**: Mimic the action of endogenous ADH, promoting water reabsorption in the collecting ducts by activating V2 receptors. • **Indications**: Diabetes insipidus, nocturnal enuresis. • **Adverse Effects**: Hyponatremia, water retention, hypertension. *B. Vasopressin Receptor Antagonists* • **Examples**: Tolvaptan, Conivaptan. • **Mechanism**: Block the V2 receptors, inhibiting water reabsorption and promoting diuresis. • **Indications**: Syndrome of inappropriate antidiuretic hormone (SIADH), hypervolemic hyponatremia. **Adverse Effects**: Hypernatremia, thirst, dehydration.						

HomUG-Mod.Phar– 4.8 (Blood)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar 4.8.1	Hematinics	K	Knowledge and scholarship	T/t of Iron deficiency anemia and Megaloblastic anemia	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Physiology

HomUG-Mod.Phar– 4.9 (GIT)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-	Drugs for Peptic Ulcer and GERD	K	Knowledge and	**Peptic Ulcer Disease (PUD)** Peptic ulcers are lesions in the mucosal	C1	MK	Lecture, Group discussio	Quiz, Written	SAQ, MCQ	Gastro Depratment

Mod. Phar **4.9.1**			scholarship	lining of the stomach, duodenum, or esophagus caused by an imbalance between aggressive factors (e.g., gastric acid, pepsin) and protective factors (e.g., mucus, bicarbonate). Common causes include Helicobacter pylori infection, long-term NSAID use, and excess gastric acid production. **A. Drug Classes Used for PUD** 1. **Proton Pump Inhibitors (PPIs)** ○ **Examples**: Omeprazole, Lansoprazole, Esomeprazole, Pantoprazole, Rabeprazole. ○ **Mechanism**: PPIs irreversibly inhibit the H+/K+ ATPase (proton pump) in parietal cells, which reduces gastric acid secretion. ○ **Indications**: First-line therapy for peptic ulcers, H. pylori eradication (as part of combination therapy), GERD, Zollinger-Ellison syndrome. ○ **Adverse Effects**: Long-term use may lead to vitamin B12 deficiency,			n	test, MCQ		Medicine Physiology

				hypomagnesemia, osteoporosis, and increased susceptibility to Clostridium difficile infections. 2. **H2-Receptor Antagonists (H2RAs)** ○ **Examples**: Ranitidine, Famotidine, Cimetidine, Nizatidine. ○ **Mechanism**: H2RAs block histamine receptors (H2 receptors) on parietal cells, reducing gastric acid secretion. ○ **Indications**: Used in the treatment of mild to moderate PUD, GERD, and Zollinger-Ellison syndrome. They are also used in the prevention of stress ulcers in critically ill patients. ○ **Adverse Effects**: Headache, dizziness, fatigue, and gastrointestinal symptoms. Cimetidine has significant drug interactions due to its inhibition of cytochrome P450						

				enzymes. 3. **Antacids** ○ **Examples**: Magnesium hydroxide, Aluminum hydroxide, Calcium carbonate, Sodium bicarbonate. ○ **Mechanism**: Antacids neutralize gastric acid, increasing gastric pH and providing symptomatic relief. ○ **Indications**: Short-term relief of symptoms in PUD and GERD, as adjunctive therapy in combination with other agents. ○ **Adverse Effects**: Constipation (aluminum-based), diarrhea (magnesium-based), and metabolic alkalosis with overuse of sodium bicarbonate. 4. **Mucosal Protective Agents** ○ **Examples**: Sucralfate, Misoprostol. ○ **Mechanism**: Sucralfate forms a protective barrier over the ulcer, while Misoprostol, a						

				prostaglandin analog, increases mucus and bicarbonate secretion to protect the gastric mucosa. ○ **Indications**: Used in the healing of ulcers, particularly NSAID-induced ulcers (Misoprostol), and in combination with other drugs for H. pylori eradication. ○ **Adverse Effects**: Sucralfate can cause constipation and nausea, while Misoprostol may cause diarrhea, abdominal cramping, and is contraindicated in pregnancy due to its abortifacient effects. 5. **Antibiotics (for H. pylori infection)** ○ **Examples**: Amoxicillin, Clarithromycin, Metronidazole, Tetracycline. ○ **Mechanism**: Antibiotics target and eradicate Helicobacter pylori, which is a primary cause of peptic ulcers. ○ **Indications**: Used in						

				combination therapy with PPIs for the eradication of H. pylori in PUD. ○ **Adverse Effects**: Nausea, vomiting, diarrhea, and the risk of developing antibiotic resistance. **2. Gastroesophageal Reflux Disease (GERD)** GERD is a chronic condition where gastric contents, including acid, reflux into the esophagus, causing symptoms such as heartburn, regurgitation, and potential esophageal damage (e.g., esophagitis, Barrett's esophagus). **A. Drug Classes Used for GERD** 1. **Proton Pump Inhibitors (PPIs)** ○ **Indications**: First-line therapy for GERD to heal esophagitis, provide symptomatic relief, and prevent complications like strictures or Barrett's esophagus. ○ **Adverse Effects**: Similar to those seen in PUD therapy, including risk of long-term						

				complications like vitamin B12 deficiency and osteoporosis. 2. **H2-Receptor Antagonists (H2RAs)** ○ **Indications**: Used for mild GERD symptoms or in patients who do not require more potent acid suppression (PPIs). ○ **Adverse Effects**: As noted above, particularly with cimetidine due to its interaction with cytochrome P450 enzymes. 3. **Antacids** ○ **Indications**: Provide short-term relief from heartburn symptoms in GERD. ○ **Adverse Effects**: As noted in PUD treatment, particularly with overuse leading to metabolic alkalosis, constipation, or diarrhea. 4. **Prokinetic Agents** ○ **Examples**: Metoclopramide, Domperidone. ○ **Mechanism**: These drugs increase lower						

				esophageal sphincter tone and enhance gastric emptying, reducing acid reflux. ○ **Indications**: Used in GERD with delayed gastric emptying or in patients with esophageal motility issues. ○ **Adverse Effects**: Metoclopramide can cause extrapyramidal symptoms, tardive dyskinesia, and sedation. 5. **Alginate-based Products** ○ **Examples**: Gaviscon. ○ **Mechanism**: Alginate forms a gel-like barrier that floats on the stomach contents, preventing acid from refluxing into the esophagus. ○ **Indications**: Used for mild to moderate GERD and in combination with other agents. **Adverse Effects**: Typically well-tolerated but may cause bloating or discomfort. **Knowledge-Based Outcomes** 1. **Mechanisms of Action**:						

				Understand how PPIs, H2RAs, and other drugs reduce gastric acid secretion, promote ulcer healing, and protect the gastrointestinal mucosa. 2. **Indications and Contraindications**: Identify when to use PPIs, H2RAs, antacids, and mucosal protectants based on the underlying etiology of PUD or GERD. 3. **Adverse Effects and Drug Interactions**: Recognize the potential side effects of each class of drug (e.g., bleeding risks with mucosal protectants, infection risks with PPIs, or drug interactions with cimetidine). **Skill-Based Outcomes** 4. **Therapeutic Decision Making**: Select appropriate drug therapy based on the clinical scenario, balancing the severity of the condition with the potential for adverse effects and long-term complications. 5. **Monitoring and Adjustment**: Monitor patient progress, adjust therapy as needed based on symptom control, adverse effects, and response to treatment (e.g., for H. pylori						

				eradication or healing of ulcers). 6. **Patient Education**: Educate patients on the importance of adherence to therapy, lifestyle modifications (e.g., diet, weight management), and potential side effects. **Attitude-Based Outcomes** 7. **Patient-Centered Care**: Address patient concerns regarding long-term treatment (e.g., PPI use) and emphasize the importance of managing GERD and PUD to prevent complications. 8. **Rational Prescribing**: Prescribe drugs for PUD and GERD based on current guidelines, considering the risk of complications, especially with long-term therapy. 9. **Preventive Approach**: Advocate for preventive measures such as H. pylori screening and eradication, lifestyle modifications, and careful monitoring of patients at risk for complications.						
Hom UG-Mod.	Drugs for constipation and diarrhoea	K	Knowledge and scholarship	**Drugs for Constipation** Constipation is characterized by	C1	MK	Lecture, Group discussio	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

Phar **4.9.2**				infrequent, difficult, or painful bowel movements, often associated with hard stools. Treatment typically involves dietary and lifestyle changes, but pharmacotherapy is used in more severe or chronic cases. *A. Drug Classes Used for Constipation* 1. **Bulk-Forming Laxatives** ○ **Examples**: Psyllium, Methylcellulose, Polycarbophil. ○ **Mechanism**: These drugs increase stool volume by absorbing water, which softens the stool and promotes peristalsis. ○ **Indications**: First-line therapy for chronic constipation, especially in patients with low fiber intake. ○ **Adverse Effects**: Bloating, flatulence, and abdominal discomfort. Rarely, they can cause obstruction if not taken with adequate water. 2. **Stool Softeners** ○ **Examples**: Docusate sodium, Docusate calcium. ○ **Mechanism**: These agents increase the			n			

				water content of the stool, making it softer and easier to pass. ○ **Indications**: Used for mild constipation, particularly in patients with hemorrhoids or post-operative patients who should avoid straining. ○ **Adverse Effects**: Generally well tolerated but may cause mild abdominal cramping. 3. **Osmotic Laxatives** ○ **Examples**: Lactulose, Polyethylene glycol (PEG), Magnesium hydroxide. ○ **Mechanism**: These agents draw water into the colon through osmosis, softening the stool and promoting bowel movement. ○ **Indications**: Used in chronic constipation, fecal impaction, and in patients with hepatic encephalopathy (lactulose). ○ **Adverse Effects**: Bloating, flatulence, diarrhea, and						

				electrolyte imbalances (especially with magnesium-based agents). 4. **Stimulant Laxatives** ○ **Examples**: Bisacodyl, Senna, Castor oil. ○ **Mechanism**: These drugs stimulate the smooth muscle of the colon, enhancing peristalsis and accelerating bowel movements. ○ **Indications**: Used in cases of acute constipation or as a second-line treatment for chronic constipation. ○ **Adverse Effects**: Cramping, dehydration, and long-term use may lead to dependence or bowel atony. 5. **Chloride Channel Activators** ○ **Examples**: Lubiprostone, Linaclotide. ○ **Mechanism**: These agents increase chloride secretion into the intestinal lumen, enhancing water secretion and improving stool						

				consistency. ○ **Indications**: Used for chronic idiopathic constipation (CIC) and constipation-predominant irritable bowel syndrome (IBS-C). ○ **Adverse Effects**: Nausea, diarrhea, bloating, and abdominal discomfort. 6. **Guanylate Cyclase-C Agonists** ○ **Examples**: Plecanatide, Linaclotide. ○ **Mechanism**: These drugs increase cyclic GMP levels in the intestines, stimulating fluid secretion and promoting bowel motility. ○ **Indications**: Used for chronic constipation and IBS-C. ○ **Adverse Effects**: Diarrhea, flatulence, and abdominal pain. **2. Drugs for Diarrhea** Diarrhea is defined as the passage of loose or watery stools more than three times a day and can be caused by infections, medications, or						

				gastrointestinal disorders. The goal of treatment is to restore fluid balance, relieve symptoms, and treat the underlying cause. *A. Drug Classes Used for Diarrhea* 1. **Antidiarrheal Agents** ○ **Examples**: Loperamide, Diphenoxylate with atropine (Lomotil). ○ **Mechanism**: These drugs work by slowing intestinal motility, allowing more time for fluid absorption in the colon. ○ **Indications**: Used for acute diarrhea (including traveler's diarrhea), chronic diarrhea (e.g., in IBS), and to reduce the frequency of stool in conditions like inflammatory bowel disease (IBD). ○ **Adverse Effects**: Constipation, bloating, and abdominal cramps. Loperamide should be avoided in cases of dysentery or bacterial infections involving the gut.						

				2. **Adsorbents** ○ **Examples**: Activated charcoal, Kaolin, Pectin. ○ **Mechanism**: Adsorbents bind to toxins or pathogens in the gut, preventing them from causing diarrhea. ○ **Indications**: Used for mild diarrhea and toxin-related diarrhea (e.g., from bacterial infections). ○ **Adverse Effects**: Constipation, bloating, and reduced effectiveness of other medications if used concurrently. 3. **Bismuth Subsalicylate** ○ **Examples**: Pepto-Bismol. ○ **Mechanism**: It has antimicrobial, anti-inflammatory, and antacid properties, which help reduce diarrhea, nausea, and abdominal discomfort. ○ **Indications**: Used for acute diarrhea (including traveler's diarrhea), indigestion, and nausea. ○ **Adverse Effects**: Blackened stools,						

				tongue discoloration, and in large doses, salicylate toxicity. 4. **Probiotics** ○ **Examples**: Lactobacillus, Saccharomyces boulardii. ○ **Mechanism**: Probiotics restore the natural balance of gut flora, which can be disrupted in diarrhea. ○ **Indications**: Used for antibiotic-associated diarrhea, gastroenteritis, and inflammatory bowel disease (IBD). ○ **Adverse Effects**: Generally well tolerated but may cause bloating and gas in some individuals. 5. **Octreotide** ○ **Mechanism**: Octreotide is a somatostatin analog that inhibits the secretion of various gastrointestinal hormones, slowing motility and reducing secretions. ○ **Indications**: Used for diarrhea caused by neuroendocrine tumors,						

				chemotherapy, and certain gastrointestinal disorders. ○ **Adverse Effects**: Nausea, abdominal cramps, and flatulence. 6. **Antibiotics (for Infectious Diarrhea)** ○ **Examples**: Ciprofloxacin, Metronidazole, Rifaximin. ○ **Mechanism**: These drugs treat bacterial infections causing diarrhea by targeting the pathogen directly. ○ **Indications**: Used for diarrhea caused by specific bacterial infections, such as Salmonella, Shigella, or Clostridium difficile. ○ **Adverse Effects**: Diarrhea, nausea, and potential development of antibiotic resistance. **3. Monitoring and Safety** • **Constipation**: Regular monitoring for adverse effects with long-term laxative use, especially stimulant laxatives,						

				to avoid dependency and bowel atony. **Diarrhea**: Close monitoring of hydration status, especially in children and elderly patients, to prevent complications like electrolyte imbalances and dehydration. **Knowledge-Based Outcomes** 1. **Mechanisms of Action**: Understand how each class of drug for constipation and diarrhea works to alleviate symptoms and restore normal bowel function. 2. **Indications and Contraindications**: Recognize the appropriate use of bulk-forming agents, laxatives, antidiarrheals, and probiotics based on the underlying cause and clinical scenario. 3. **Adverse Effects and Drug Interactions**: Identify potential side effects and interactions of these drugs (e.g., electrolyte imbalances with osmotic laxatives, constipation with antidiarrheals). **Skill-Based Outcomes** 4. **Therapeutic Decision**						

				Making: Be able to select the appropriate pharmacotherapy based on the type of constipation or diarrhea and its underlying cause (e.g., osmotic laxatives for chronic constipation, loperamide for acute diarrhea). 5. **Monitoring and Adjustment**: Monitor patients for adverse effects, treatment response, and the resolution of underlying causes (e.g., adjusting therapy based on stool frequency and consistency). 6. **Patient Education**: Provide guidance on proper use of medications, potential side effects, and lifestyle modifications (e.g., increasing fluid and fiber intake for constipation). **Attitude-Based Outcomes** 7. **Patient-Centered Care**: Ensure that treatment decisions are tailored to the individual patient, taking into account their preferences, lifestyle, and specific medical conditions. 8. **Rational Prescribing**: Prescribe drugs for constipation and diarrhea based on the clinical severity,						

				while minimizing risks of adverse effects, especially in vulnerable populations (e.g., the elderly). 9. **Preventive Approach**: Advocate for preventive measures like diet modification (increasing fiber intake), hydration, and regular exercise for constipation, and proper sanitation to prevent infections that cause diarrhea.						
Hom UG-Mod. Phar **4.9.3**	Antiemetics	K	Knowledge and scholarship	**Mechanisms of Nausea and Vomiting** Nausea and vomiting are complex physiological processes that involve multiple pathways and brain regions, including: • **Central mechanisms**: Activation of the **vomiting center** in the medulla oblongata via signals from the chemoreceptor trigger zone (CTZ), vestibular system, and higher brain centers. • **Peripheral mechanisms**: Inflammation or irritation of the gastrointestinal tract, involving neurotransmitters like serotonin (5-HT), dopamine, and substance P. *Key Neurotransmitters Involved:* 1. **Serotonin (5-HT)**: Plays a central role in chemotherapy-	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				induced nausea and vomiting (CINV) and post-operative nausea. 2. **Dopamine (D2 receptors)**: Involved in motion sickness, CINV, and gastroparesis. 3. **Histamine (H1 receptors)**: Responsible for motion sickness and vestibular nausea. 4. **Substance P (NK1 receptors)**: Involved in chemotherapy-induced and post-operative nausea. **2. Drug Classes Used for Nausea and Vomiting (Antiemetics)** 1. **Serotonin (5-HT3) Antagonists** ○ **Examples**: Ondansetron, Granisetron, Dolasetron, Palonosetron. ○ **Mechanism**: These drugs block serotonin receptors in the central and peripheral nervous system, specifically 5-HT3 receptors, which are involved in triggering nausea and vomiting. ○ **Indications**: Primarily used for **chemotherapy-induced nausea and vomiting (CINV)**,						

				postoperative nausea, and **radiation therapy-induced nausea**. ○ **Adverse Effects**: Headache, constipation, dizziness, and potential QT prolongation. 2. **Dopamine (D2) Antagonists** ○ **Examples**: Metoclopramide, Prochlorperazine, Domperidone. ○ **Mechanism**: These drugs block dopamine receptors in the CTZ and gastrointestinal tract, which helps control nausea and vomiting. ○ **Indications**: Used for **gastrointestinal disorders** (e.g., gastroparesis), **postoperative nausea**, **motion sickness**, and **CINV**. ○ **Adverse Effects**: Extrapyramidal symptoms (EPS) like dystonia, parkinsonism, and tardive dyskinesia, sedation, and increased prolactin levels (domperidone is less likely to cause						

				EPS). 3. **Histamine (H1) Antagonists** ○ **Examples**: Diphenhydramine, Meclizine, Promethazine. ○ **Mechanism**: These drugs block histamine receptors in the vestibular system, helping prevent nausea related to motion sickness and vertigo. ○ **Indications**: Used for **motion sickness**, **vertigo**, **postoperative nausea**, and as adjunct therapy in **CINV**. ○ **Adverse Effects**: Sedation, dry mouth, blurred vision, and urinary retention. 4. **Neurokinin (NK1) Receptor Antagonists** ○ **Examples**: Aprepitant, Fosaprepitant, Rolapitant. ○ **Mechanism**: These drugs block substance P at the NK1 receptors in the brain, reducing the signal that triggers vomiting. ○ **Indications**: Mainly						

				used in combination with 5-HT3 antagonists and corticosteroids for **CINV**, especially for highly emetogenic chemotherapy regimens. ○ **Adverse Effects**: Fatigue, dizziness, diarrhea, and liver enzyme elevation. 5. **Corticosteroids** ○ **Examples**: Dexamethasone. ○ **Mechanism**: The exact mechanism of action is unclear, but corticosteroids may reduce inflammation and affect the central nervous system's response to nausea. ○ **Indications**: Used in combination with 5-HT3 antagonists and NK1 receptor antagonists for **CINV** and **postoperative nausea**. ○ **Adverse Effects**: Increased appetite, weight gain, insomnia, and elevated blood glucose levels. 6. **Cannabinoids** ○ **Examples**: Dronabinol,						

				Nabilone. ○ **Mechanism**: These agents act on the cannabinoid receptors in the brain, which play a role in the regulation of nausea and vomiting. ○ **Indications**: Used for **CINV** in patients who do not respond to standard antiemetics or in cases of **appetite stimulation** in patients with cancer or HIV/AIDS. ○ **Adverse Effects**: Euphoria, sedation, dizziness, dry mouth, and psychosis in susceptible individuals. 7. **Anticholinergic Agents** ○ **Examples**: Scopolamine. ○ **Mechanism**: These drugs block acetylcholine receptors in the vestibular system and gastrointestinal tract, helping prevent nausea and vomiting caused by motion sickness. ○ **Indications**: Used for **motion sickness** and **postoperative**						

				nausea. **Adverse Effects**: Dry mouth, blurred vision, urinary retention, and confusion. **Knowledge-Based Outcomes** 1. **Mechanism of Action**: Understand how different classes of antiemetic drugs act on the various pathways involved in nausea and vomiting, such as serotonin, dopamine, histamine, and substance P. 2. **Clinical Indications**: Recognize the appropriate indications for the use of specific antiemetic drugs, including **CINV**, **PONV**, **motion sickness**, and **gastrointestinal disorders**. 3. **Adverse Effects**: Identify the potential adverse effects and safety concerns associated with antiemetic drugs, such as sedation, extrapyramidal symptoms, and electrolyte imbalances. **Skill-Based Outcomes** 4. **Therapeutic Decision-Making**: Be able to select the most appropriate antiemetic						

				treatment based on the type of nausea/vomiting (e.g., **CINV**, **motion sickness**) and the patient's clinical condition. 5. **Combination Therapy**: Develop the ability to combine antiemetics (e.g., **5-HT3 antagonists** with **NK1 antagonists** and **corticosteroids**) to provide effective control of nausea and vomiting in complex conditions like **CINV**. 6. **Monitoring and Adjustment**: Monitor patients for adverse effects, especially in chemotherapy and postoperative settings, and adjust treatment based on the patient's response and side effects. **Attitude-Based Outcomes** 7. **Patient-Centered Care**: Ensure that antiemetic therapy is tailored to the individual patient's needs, considering factors such as chemotherapy regimens, comorbidities, and personal preferences. 8. **Rational Prescribing**: Prescribe antiemetics based on the severity and cause of nausea and vomiting, while minimizing side effects and drug interactions, especially in vulnerable populations.						

				9. **Patient Education**: Provide thorough education to patients about the appropriate use of antiemetics, potential side effects, and when to seek medical attention for any adverse reactions.						

HomUG-Mod.Phar– 4.10 (Chemotherapy)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod. Phar 4.10.1	Sulfonamides + Cotrimoxazole	K	Knowledge and scholarship	**Sulfonamides** *Definition* Sulfonamides are bacteriostatic agents that inhibit the synthesis of dihydropteroic acid, an essential precursor in the folate pathway of bacteria. *Mechanism of Action* • Sulfonamides act as **competitive inhibitors** of the enzyme **dihydropteroate synthase**. • They block the incorporation of para-aminobenzoic acid	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				(PABA) into folic acid, preventing bacterial replication. *Examples* 1. **Short-acting sulfonamides**: Sulfisoxazole. 2. **Intermediate-acting sulfonamides**: Sulfamethoxazole. 3. **Long-acting sulfonamides**: Sulfadoxine. 4. **Topical sulfonamides**: Sulfacetamide (eye infections), Silver sulfadiazine (burn infections). *Clinical Uses* • Urinary tract infections (UTIs). • Nocardiosis. • Trachoma. • Toxoplasmosis (in combination with pyrimethamine). • Burn wound infections (topical silver sulfadiazine). *Adverse Effects* • **Hypersensitivity reactions**: Rash, Stevens-Johnson syndrome. • **Hematologic effects**:						

				Hemolytic anemia (especially in G6PD-deficient patients). • **Kernicterus** in neonates due to bilirubin displacement. • **Crystalluria**: Formation of crystals in urine leading to renal damage. **2. Cotrimoxazole** *Definition* Cotrimoxazole is a combination of **sulfamethoxazole (SMX)** and **trimethoprim (TMP)** in a 5:1 ratio. The combination provides synergistic bactericidal activity. *Mechanism of Action* • **Sulfamethoxazole**: Inhibits dihydropteroate synthase, blocking folic acid synthesis. • **Trimethoprim**: Inhibits **dihydrofolate reductase**, further blocking folate metabolism. • Together, they inhibit two consecutive steps in folate synthesis, leading to bacterial cell death. *Clinical Uses* • **Respiratory infections**: Pneumocystis jirovecii pneumonia (PCP).						

				• **Urinary tract infections (UTIs)**: First-line treatment. • **Gastrointestinal infections**: Shigellosis, Traveler's diarrhea (caused by *E. coli*). • **Nocardiosis** and toxoplasmosis (alternative to sulfonamides and pyrimethamine). *Adverse Effects* • Same as sulfonamides (hypersensitivity, hematologic effects, crystalluria). • **Trimethoprim-specific effects**: Hyperkalemia (due to potassium-sparing effects), megaloblastic anemia, leukopenia (due to folate deficiency). **Knowledge-Based Outcomes** 1. **Mechanisms of Action**: o Understand how sulfonamides and trimethoprim target bacterial folate synthesis. 2. **Spectrum of Activity**: o Recognize the broad spectrum of sulfonamides and the synergistic effects of Cotrimoxazole. 3. **Therapeutic Applications**:						

				○ Know the specific indications for sulfonamides and Cotrimoxazole, including bacterial, parasitic, and opportunistic infections. 4. **Adverse Effects**: ○ Identify common side effects and contraindications, including their use in neonates, pregnant women, and G6PD-deficient individuals. **Skill-Based Outcomes** 5. **Rational Drug Selection**: ○ Select appropriate sulfonamide or Cotrimoxazole therapy based on infection type, organism susceptibility, and patient factors. 6. **Therapeutic Monitoring**: ○ Monitor for clinical efficacy and adverse effects, including renal function, electrolyte levels, and signs of hypersensitivity. 7. **Drug Interactions**: ○ Manage potential						

				interactions, such as the displacement of warfarin or methotrexate by sulfonamides, increasing their toxicity. **Attitude-Based Outcomes** 8. **Patient Education**: o Counsel patients on the importance of hydration to prevent crystalluria and adherence to therapy to avoid resistance. 9. **Antimicrobial Stewardship**: o Avoid unnecessary use to reduce the development of resistance, particularly with Cotrimoxazole in Pneumocystis infections. 10. **Holistic Care**: • Consider patient comorbidities (e.g., renal impairment, G6PD deficiency) and adjust therapy accordingly.						
Hom UG-Mod.	Quinolones	K	Knowledge and scholarship	**Quinolones** *Definition*	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

Phar **4.10.2**				Quinolones are synthetic antibiotics that inhibit bacterial DNA replication by targeting DNA gyrase (topoisomerase II) and topoisomerase IV. **2. Mechanism of Action** 1. **DNA Gyrase Inhibition**: o Primarily in gram-negative bacteria, it prevents the unwinding of supercoiled DNA, which is essential for replication. 2. **Topoisomerase IV Inhibition**: o Predominantly in gram-positive bacteria, it interferes with the separation of replicated DNA strands during cell division. **3. Classification and Examples** *First-Generation Quinolones* • **Examples**: Nalidixic acid. • **Spectrum**: Limited to gram-negative bacteria. • **Use**: Uncomplicated urinary tract infections (UTIs).						

				Second-Generation Fluoroquinolones • **Examples**: Ciprofloxacin, Norfloxacin, Ofloxacin. • **Spectrum**: Broad spectrum, effective against gram-negative bacteria and some gram-positive organisms. • **Use**: UTIs, gastroenteritis, prostatitis, bone/joint infections. *Third-Generation Fluoroquinolones* • **Examples**: Levofloxacin. • **Spectrum**: Improved activity against gram-positive bacteria (e.g., Streptococcus pneumoniae). • **Use**: Respiratory tract infections (e.g., community-acquired pneumonia, bronchitis). *Fourth-Generation Fluoroquinolones* • **Examples**: Moxifloxacin, Gemifloxacin. • **Spectrum**: Broad spectrum, effective against anaerobes and atypical pathogens. • **Use**: Respiratory tract infections, intra-abdominal infections. **4. Adverse Effects**						

				1. **Gastrointestinal**: o Nausea, vomiting, diarrhea. 2. **Central Nervous System (CNS)**: o Headache, dizziness, insomnia, seizures (rare). 3. **Tendinopathy**: o Risk of tendonitis and Achilles tendon rupture, especially in older adults or those on corticosteroids. 4. **QT Prolongation**: o Particularly with moxifloxacin, leading to arrhythmias. 5. **Photosensitivity**: o Increased sensitivity to sunlight. 6. **Resistance Development**: o Widespread use has led to significant bacterial resistance. **6. Contraindications and Precautions** 1. **Pregnancy and Breastfeeding**: o Contraindicated due to potential cartilage damage in the fetus or neonate. 2. **Children (<18 years)**: o Avoid unless benefits outweigh risks (e.g.,						

				severe infections like anthrax). 3. **Drug Interactions**: o Antacids, iron, and calcium reduce absorption. Increased risk of seizures with NSAIDs or theophylline. *Knowledge-Based Outcomes* 1. **Mechanisms of Action**: o Explain how quinolones inhibit bacterial DNA synthesis by targeting DNA gyrase and topoisomerase IV. 2. **Spectrum of Activity**: o Differentiate between generations and their bacterial targets (e.g., gram-negative, gram-positive, atypicals). 3. **Therapeutic Uses**: o Identify clinical conditions where quinolones are first-line or alternative treatments. 4. **Adverse Effects**: o Recognize side effects and the patient populations at higher risk. *Skill-Based Outcomes*						

				5. **Rational Drug Selection**: o Prescribe appropriate quinolones based on infection type, bacterial susceptibility, and patient factors. 6. **Therapeutic Monitoring**: o Monitor for adverse effects such as tendinopathy, QT prolongation, or photosensitivity. 7. **Resistance Management**: o Avoid overuse or inappropriate prescribing to minimize antimicrobial resistance. *Attitude-Based Outcomes* 8. **Patient Counseling**: o Educate patients on proper usage, adherence, and the need to avoid direct sunlight. 9. **Ethical Prescribing**: o Limit use to infections where quinolones are clearly indicated, reducing unnecessary exposure.						

				10. **Safety Awareness**: • Prioritize patient safety by considering contraindications and drug interactions. **8. Clinical Pearls** • Ciprofloxacin is highly effective for UTIs and gastrointestinal infections but has limited activity against gram-positive cocci. • Levofloxacin and moxifloxacin are preferred for respiratory infections due to better activity against *S. pneumoniae*. • Resistance is a growing concern; restrict use to confirmed or strongly suspected bacterial infections.						
Hom UG-Mod. Phar 4.10.3	Beta Lactam Antibiotics	K	Knowledge and scholarship	*Penicillins* • **Natural Penicillins**: Penicillin G, Penicillin V. ○ Spectrum: Gram-positive bacteria (*Streptococcus spp.*, *Treponema pallidum*). • **Penicillinase-Resistant Penicillins**: Methicillin, Nafcillin, Oxacillin. ○ Spectrum:	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				Penicillinase-producing *Staphylococcus aureus*.
				• **Aminopenicillins**: Amoxicillin, Ampicillin.
				  ○ Spectrum: Broader gram-negative activity (e.g., *E. coli*, *H. influenzae*).
				• **Extended-Spectrum Penicillins**: Piperacillin, Ticarcillin.
				  ○ Spectrum: Includes *Pseudomonas aeruginosa*.
				
				2. Cephalosporins
				
				• Classified into five generations:
				  ○ **1st Generation**: Cefazolin, Cephalexin (Gram-positive activity).
				  ○ **2nd Generation**: Cefuroxime (Broader gram-negative coverage).
				  ○ **3rd Generation**: Ceftriaxone, Ceftazidime (Expanded gram-negative activity, some penetrate CNS).
				  ○ **4th Generation**: Cefepime						

				(*Pseudomonas* coverage). ○ **5th Generation**: Ceftaroline (*MRSA* coverage). *3. Carbapenems* • Examples: Imipenem, Meropenem, Ertapenem. • Spectrum: Broadest among beta-lactams, covering gram-positive, gram-negative, and anaerobes. *4. Monobactams* • Example: Aztreonam. • Spectrum: Gram-negative aerobes, including *Pseudomonas*. *5. Beta-Lactamase Inhibitors* • Examples: Clavulanic acid, Sulbactam, Tazobactam. • Used in combination with beta-lactams to inhibit bacterial beta-lactamase enzymes. **4. Clinical Applications** *Gram-Positive Infections* • Penicillin: *Streptococcus*,						

				Enterococcus, *Listeria*. • Cephalosporins (1st generation): Skin infections caused by *Staphylococcus aureus*. *Gram-Negative Infections* • Aminopenicillins: *H. influenzae*, *E. coli*. • Cephalosporins (3rd and 4th generations): Severe gram-negative infections. • Carbapenems: ESBL-producing organisms. *Anaerobic Infections* • Carbapenems and penicillins with beta-lactamase inhibitors. *Special Cases* • Meningitis: Ceftriaxone or Cefotaxime (good CNS penetration). • *Pseudomonas* infections: Piperacillin-tazobactam, Ceftazidime, Cefepime. MRSA: Ceftaroline. *Knowledge-Based Outcomes*						

				1. **Mechanism of Action**: o Understand how beta-lactams inhibit bacterial cell wall synthesis. 2. **Spectrum of Activity**: o Differentiate between various classes and their antibacterial coverage. 3. **Resistance Mechanisms**: o Recognize common mechanisms of resistance and strategies to overcome them. 4. **Adverse Effects**: o Identify common and serious side effects associated with beta-lactams. *Skill-Based Outcomes* 5. **Rational Drug Selection**: o Prescribe appropriate beta-lactams based on infection type, organism susceptibility, and patient-specific factors. 6. **Therapeutic Monitoring**: o Monitor for signs of efficacy and toxicity, including hypersensitivity and						

				GI disturbances. 7. **Combination Therapy**: o Use beta-lactamase inhibitors appropriately to enhance the efficacy of beta-lactam antibiotics. *Attitude-Based Outcomes* 8. **Antimicrobial Stewardship**: o Avoid unnecessary or prolonged use to reduce resistance development. 9. **Patient Education**: o Counsel patients on completing the course of antibiotics and recognizing signs of adverse effects. 10. **Ethical Prescribing**: • Balance effective treatment with the need to minimize resistance and preserve antibiotics for future use. **8. Clinical Pearls** • Cephalosporins have a **generation-specific spectrum**, with newer generations covering more gram-negative organisms. • Carbapenems are **last-resort**						

				antibiotics, reserved for multidrug-resistant infections. • Avoid beta-lactams in patients with a history of **severe hypersensitivity** reactions. • Combining beta-lactams with beta-lactamase inhibitors significantly broadens their spectrum against resistant bacteria.						
Hom UG-Mod. Phar **4.10.4**	Tetracyclines, Chloramphenicol	K	Knowledge and scholarship	**Tetracyclines** **Mechanism of Action** Tetracyclines inhibit bacterial protein synthesis by binding to the **30S ribosomal subunit**, preventing the attachment of aminoacyl-tRNA to the mRNA-ribosome complex. This action halts bacterial growth, making them bacteriostatic. **Classification and Examples** 1. **Short-acting**: Tetracycline. 2. **Intermediate-acting**: Demeclocycline. 3. **Long acting**: Doxycycline, Minocycline. **Clinical Uses** 1. **Respiratory Infections**: o Atypical pathogens (*Mycoplasma*						

				pneumoniae, *Chlamydia pneumoniae*). 2. **Zoonotic Infections**: o Rickettsial diseases (e.g., Rocky Mountain spotted fever), Lyme disease (*Borrelia burgdorferi*). 3. **Sexually Transmitted Infections**: o Chlamydia, syphilis (alternative to penicillin). 4. **Acne**: o Long-term therapy for severe acne. 5. **Malaria Prophylaxis**: o Doxycycline. **Adverse Effects** 1. **Gastrointestinal**: o Nausea, vomiting, diarrhea. 2. **Photosensitivity**: o Increased sensitivity to sunlight. 3. **Teeth Discoloration**: o Avoid in children <8 years and pregnant women. 4. **Hepatotoxicity**: o Rare but severe in high doses. 5. **Fanconi Syndrome**: o Result of using						

				expired tetracycline. **Resistance Mechanisms** 1. **Efflux Pumps**: o Actively expel tetracyclines from bacterial cells. 2. **Ribosomal Protection Proteins**: o Prevent tetracycline binding. **2. Chloramphenicol** **Mechanism of Action** Chloramphenicol inhibits bacterial protein synthesis by binding to the **50S ribosomal subunit**, preventing peptide bond formation during translation. This action is primarily bacteriostatic but may be bactericidal at high concentrations against certain pathogens. **Clinical Uses** 1. **Serious Infections**: o Meningitis (*Haemophilus influenzae*, *Neisseria meningitidis*). o Typhoid fever (*Salmonella typhi*). 2. **Rickettsial Infections**: o Alternative for						

				tetracyclines in pregnant women or children. 3. **Topical Use**: o Eye infections (conjunctivitis). **Adverse Effects** 1. **Bone Marrow Suppression**: o Reversible suppression (dose-dependent). o Aplastic anemia (idiosyncratic and potentially fatal). 2. **Gray Baby Syndrome**: o In neonates due to immature liver enzymes, leading to toxicity. 3. **Gastrointestinal**: o Nausea, vomiting, diarrhea. **Resistance Mechanisms** 1. **Chloramphenicol Acetyltransferase**: o Inactivates the drug via acetylation. 2. **Efflux Pumps**: *Reduce intracellular drug concentration.* *Knowledge-Based Outcomes*						

				1. **Mechanisms of Action**: o Explain how tetracyclines target the 30S ribosome and chloramphenicol targets the 50S ribosome to inhibit protein synthesis. 2. **Spectrum of Activity**: o Recognize their broad-spectrum activity, covering gram-positive, gram-negative, atypical, and intracellular pathogens. 3. **Clinical Applications**: o Identify appropriate conditions for their use, such as rickettsial infections, typhoid fever, and acne. 4. **Adverse Effects**: o Understand the risks of bone marrow suppression, gray baby syndrome, and photosensitivity. *Skill-Based Outcomes* 5. **Rational Drug Selection**: o Select tetracyclines or chloramphenicol based on the infection type, pathogen						

				susceptibility, and patient factors. 6. **Therapeutic Monitoring**: o Monitor for toxicity, such as hepatotoxicity (tetracyclines) or bone marrow suppression (chloramphenicol). 7. **Resistance Management**: o Prescribe only when indicated to avoid resistance development. *Attitude-Based Outcomes* 8. **Antimicrobial Stewardship**: o Promote responsible use to prevent resistance. 9. **Patient Counseling**: o Educate patients on completing therapy, avoiding sunlight exposure (tetracyclines), and recognizing signs of adverse effects. 10. **Safety Considerations**: • Avoid use in vulnerable populations (e.g., pregnant women, neonates, and children) unless absolutely necessary.						

Hom UG-Mod. Phar **4.10.5**	Aminoglycosides	K		**Aminoglycosides Overview** **Mechanism of Action** • Aminoglycosides irreversibly bind to the **30S ribosomal subunit**, leading to: 1. Misreading of mRNA. 2. Production of defective proteins. 3. Disruption of bacterial cell membrane integrity, resulting in bactericidal activity. **Unique Feature** • Aminoglycosides exhibit a **concentration-dependent killing effect** and **post-antibiotic effect (PAE)**, meaning bacterial suppression continues even after drug levels drop below the minimum inhibitory concentration (MIC). Common Aminoglycosides **3. Clinical Applications** **1. Gram-Negative Infections** • Effective against						

				Enterobacteriaceae (e.g., *E. coli*, *Klebsiella*), *Pseudomonas aeruginosa*, and other gram-negative aerobes. **2. Combination Therapy** • Used synergistically with beta-lactams or glycopeptides for: 1. **Endocarditis**(*Enterococcus* spp., *Staphylococcus* spp.). 2. **Serious gram-positive infections** (e.g., MRSA). **3. Tuberculosis** • Streptomycin as a second-line agent. **4. Topical/Local Use** • Neomycin for skin infections. • Gentamicin in ophthalmic or otic preparations. **4. Adverse Effects** 1. **Ototoxicity** (Cochlear and Vestibular): o Irreversible damage to auditory and vestibular function. o Risk increases with prolonged therapy,						

				high doses, and renal impairment. 2. **Nephrotoxicity**: o Reversible renal tubular damage. o Risk factors include high trough levels, prolonged therapy, and co-administration with other nephrotoxic drugs. 3. **Neuromuscular Blockade**: Rare but can cause respiratory paralysis, especially in patients with myasthenia gravis. **Knowledge-Based Outcomes** 1. **Mechanism of Action**: o Explain how aminoglycosides disrupt bacterial protein synthesis by binding to the 30S ribosomal subunit. 2. **Spectrum of Activity**: o Recognize their primary activity against aerobic gram-negative bacteria and synergistic potential with other antibiotics. 3. **Pharmacokinetics**: o Understand their poor oral bioavailability, renal elimination, and concentration-						

				dependent killing. 4. **Adverse Effects**: o Identify key toxicities (ototoxicity, nephrotoxicity) and their risk factors. **Skill-Based Outcomes** 5. **Rational Prescribing**: o Select appropriate aminoglycosides based on infection type, pathogen susceptibility, and patient-specific factors. 6. **Therapeutic Monitoring**: o Use therapeutic drug monitoring (TDM) to maintain effective peak levels while minimizing toxic trough levels. 7. **Resistance Management**: o Apply appropriate combination therapy to minimize resistance. **Attitude-Based Outcomes** 8. **Patient-Centered Care**: o Educate patients on potential toxicities and monitor closely for early signs of adverse effects.						

				9. **Antimicrobial Stewardship**: o Avoid overuse or misuse of aminoglycosides aminoglycosides to prevent resistance and toxicity. 10. **Ethical Use**: • Reserve aminoglycosides for serious infections to preserve their efficacy. **Clinical Pearls** • Monitor renal function (serum creatinine, urine output) and auditory/vestibular function during therapy. • Avoid aminoglycosides in patients with pre-existing renal or auditory impairment unless absolutely necessary. • Use combination therapy for gram-positive infections to enhance efficacy and reduce resistance.						
Hom UG-Mod. Phar 4.10.6	Antitubercular drugs and Antileprosy drugs	K	Knowledge and scholarship	**Antitubercular Drugs** **Mechanism of Action and Classification** Antitubercular drugs are classified into **first-line** and **second-line agents** based on their efficacy, safety, and	C1	MK	Lecture, Group discussion	Quiz, Written test, MCQ	SAQ, MCQ	Medicine Physiology

				tolerability. *Second-Line Drugs* Used for multidrug-resistant TB (MDR-TB): • Fluoroquinolones (e.g., Levofloxacin, Moxifloxacin). • Aminoglycosides (e.g., Amikacin, Kanamycin). • Linezolid, Bedaquiline, Delamanid. **Clinical Uses** 1. **Active TB**: o Initial phase: Combination of 4 drugs (INH, RIF, PZA, EMB) for 2 months. o Continuation phase: Combination of 2 drugs (INH and RIF) for 4-7 months. 2. **Latent TB**: o Monotherapy (e.g., INH for 6-9 months) or combination (RIF + INH for 3 months). 3. **Drug-Resistant TB**: o Individualized regimens with second-line drugs. **Adverse Effects**						

				1. **Isoniazid**: Peripheral neuropathy (prevented with pyridoxine), hepatotoxicity. 2. **Rifampin**: Hepatotoxicity, red-orange body fluids, drug interactions (CYP inducer). 3. **Pyrazinamide**: Hyperuricemia, hepatotoxicity. 4. **Ethambutol**: Optic neuritis, reversible with discontinuation. 5. **Streptomycin**: Ototoxicity, nephrotoxicity. **Resistance Mechanisms** 1. Mutations in drug targets (e.g., katG for INH, rpoB for RIF). 2. Efflux pumps. 3. Enzymatic inactivation of drugs. **2. Antileprosy Drugs** **Mechanism of Action and Classification** Antileprosy drugs are used in combination therapy to prevent resistance and ensure effective eradication of *Mycobacterium leprae*. **Clinical Uses** 1. **Multibacillary Leprosy**						

				(MB): o Dapsone + Rifampin + Clofazimine for 12 months. 2. **Paucibacillary Leprosy (PB)**: o Dapsone + Rifampin for 6 months. 3. **Reactions in Leprosy**: o **Type I Reaction**: Use corticosteroids. o **Type II Reaction (Erythema Nodosum Leprosum)**: Use thalidomide or corticosteroids. **Adverse Effects** 1. **Dapsone**: Hemolysis (especially in G6PD deficiency), methemoglobinemia. 2. **Rifampin**: Hepatotoxicity, red-orange body fluids. **Clofazimine**: Skin pigmentation (reddish-brown), gastrointestinal symptoms. **Knowledge-Based Outcomes** 1. **Mechanisms of Action**: o Describe how antitubercular and antileprosy drugs target bacterial cell walls, protein						

				synthesis, and metabolic pathways. 2. **Drug Regimens**: o Understand standard treatment regimens for TB (e.g., DOTS strategy) and leprosy. 3. **Adverse Effects**: o Recognize potential toxicities and their prevention (e.g., pyridoxine for INH-induced neuropathy). 4. **Drug Resistance**: o Explain the mechanisms and management of drug-resistant TB. **Skill-Based Outcomes** 5. **Rational Prescribing**: o Prescribe appropriate combinations of drugs to avoid resistance and ensure efficacy. 6. **Monitoring**: o Monitor liver function (for hepatotoxicity), vision (ethambutol), and other toxicities during therapy. 7. **Public Health Implementation**: o Apply national TB control program						

				guidelines and strategies for leprosy eradication. **Attitude-Based Outcomes** 8. **Patient Education**: o Educate patients on the importance of adherence to long-term therapy to prevent resistance. 9. **Antimicrobial Stewardship**: o Promote the rational use of drugs in TB and leprosy management. 10. **Community Engagement**: Advocate for early diagnosis and treatment in endemic regions to reduce transmission.						
Hom UG-Mod. Phar **4.10.7**	Antimalarial drugs	K	Knowledge and scholarship	Classification of Antimalarial Drugs • Based on Mechanism of Action Blood Schizonticides Tissue Schizonticides Gametocides Sporontocides • Key Antimalarial Agents Chloroquine Artemisinin Derivatives Quinine/Quinidine						

				Mefloquine Primaquine Proguanil and Pyrimethamine Atovaquone **Clinical Applications** **Treatment of Malaria** 1. **Uncomplicated Malaria**: o *P. vivax* and *P. ovale*: Chloroquine + Primaquine. o *P. falciparum* (chloroquine-resistant): Artemisinin-based Combination Therapy (ACT). 2. **Severe Malaria**: o IV Artesunate is the drug of choice. o Alternatives: IV Quinine or Quinidine. 3. **Radical Cure**: o Primaquine or Tafenoquine to eradicate hypnozoites in relapsing malaria (*P. vivax* and *P. ovale*). **Prophylaxis of Malaria** 1. **Casual Prophylaxis** (prevents hepatic infection): Atovaquone-Proguanil.						

				2. **Suppressive Prophylaxis** (prevents blood-stage infection): Mefloquine, Doxycycline. **Drug-Resistant Malaria** • ACTs (e.g., Artemether-Lumefantrine, Artesunate-Mefloquine). **Mechanisms of Resistance** 1. **Chloroquine**: Mutations in the *PfCRT* (Plasmodium falciparum chloroquine resistance transporter) gene. 2. **Artemisinin**: Delayed parasite clearance due to mutations in the *K13* gene. 3. **Pyrimethamine/Proguanil**: Mutations in the dihydrofolate reductase (DHFR) enzyme Adverse Effects **Standard Learning Outcomes (SLO)** **Knowledge-Based Outcomes** 1. **Mechanisms of Action**: o Explain how antimalarial drugs target specific stages of the parasite lifecycle.						

				2. **Drug Selection**: o Describe first-line and alternative therapies for uncomplicated, severe, and relapsing malaria. 3. **Resistance Patterns**: o Understand mechanisms of resistance and how they influence therapy. 4. **Adverse Effects**: o Recognize common toxicities and preventive measures (e.g., testing for G6PD deficiency). **Skill-Based Outcomes** 5. **Rational Prescribing**: o Prescribe appropriate antimalarials based on species, resistance patterns, and patient factors. 6. **Monitoring and Safety**: o Monitor for adverse effects, such as QT prolongation and hemolysis. 7. **Preventive Strategies**: o Develop prophylactic regimens tailored to travel destinations						

				and patient risks. **Attitude-Based Outcomes** 8. **Patient Education**: o Counsel patients on the importance of adherence to treatment to prevent resistance. 9. **Global Health Awareness**: o Advocate for malaria prevention and treatment programs in endemic regions. 10. **Antimicrobial Stewardship**: Use antimalarials judiciously to avoid resistance development						

HomUG-Mod.Phar– 4.11 (Miscellaneous)

Sl.No	Duration of Competency	Miller	Content	Specific Learning Objectives	Bloom/ Guilbert	Priority	TL MM	Assessment		Integration
								Formative	Summative	
Hom UG-Mod.	Disinfectants,	K	Knowledge and scholarship	**Classification of Disinfectants** **Based on Chemical Composition**						

Phar **4.11.1**				Ethanol/Isopropanol Sodium Hypochlorite Glutaraldehyde Hydrogen Peroxide Quaternary Ammonium Compounds **Mechanism of Action** • **Targeting Microbial Structures**: Disinfectants act on cell walls, membranes, or intracellular components to inhibit or kill microbes. • **Broad-Spectrum Activity**: Effective against bacteria, viruses, fungi, and spores (depending on the type and concentration). Commonly Used Disinfectants and Applications **Principles of Disinfectant Use** **Selection Based on Purpose** 1. **High-Level Disinfection**: Required for medical instruments that contact mucous membranes (e.g., glutaraldehyde, peracetic acid). 2. **Intermediate-Level Disinfection**: For surfaces that come in						

				contact with intact skin (e.g., alcohols, chlorine compounds). 3. **Low-Level Disinfection**: Suitable for general cleaning of floors, walls, and furniture (e.g., QACs). **Concentration and Contact Time** • **Alcohols**: Effective at 60–90%, with optimal contact time of 10–15 minutes. • **Sodium Hypochlorite**: Typically used in 0.1–0.5% solutions for surface disinfection. • **Hydrogen Peroxide**: Usually applied in 3% solutions for general use. Adverse Effects and Precautions **Standard Learning Outcomes (SLO)** **Knowledge-Based Outcomes** 1. **Classification**: o Categorize disinfectants based on chemical composition and use. 2. **Mechanism of Action**: o Explain how disinfectants kill or inhibit microbes. 3. **Application**:						

				○ Recognize the appropriate disinfectant for specific situations (e.g., surgical instruments vs. general surfaces). **Skill-Based Outcomes** 4. **Preparation**: ○ Demonstrate correct preparation and dilution of disinfectants. 5. **Application Techniques**: ○ Apply disinfectants effectively to achieve optimal microbial control. 6. **Monitoring**: ○ Evaluate the effectiveness of disinfection and identify potential failures. **Attitude-Based Outcomes** 7. **Judicious Use**: ○ Encourage responsible use of disinfectantsdisinfectants to prevent resistance and environmental harm.						

				8. **Safety Practices**: Adhere to safety guidelines to protect users and patients.						
Hom UG-Mod. Phar **4.11.2**	Vitamins	K	Knowledge and scholarship	Vitamins Classification and Sources Fat-Soluble 1. Deficiency Disorders Vitamin A Vitamin D Vitamin E Vitamin K Vitamin C B-complex **Supplementation Principles** • **Deficiency-Based**: Supplementation is used to address deficiencies. • **Therapeutic Use**: High-dose vitamins for specific conditions (e.g., Vitamin D for osteoporosis). **Safety Considerations**: Avoid hypervitaminosis, particularly for fat-soluble vitamins.						

				Knowledge-Based Outcomes 1. Understand the role of vitamins in metabolism and disease prevention. 2. Identify deficiency disorders and their management. **Skill-Based Outcomes** 1. Prescribe appropriate vitamin supplements for specific needs. 2. Educate patients about dietary sources of vitamins. **Attitude-Based Outcomes** 1. Promote balanced nutrition to prevent deficiencies. Encourage responsible use of vitamin supplements.						

6.Teaching Learning Methods

- Lectures (including AV aid), Small group discussion, Integrated lectures, Library reference, Self directed learning etc.
- While lectures can provide a foundation, they shouldn't be the sole method Incorporate active learning strategies such as engage students through case studies, problem-based learning (PBL). PBL challenges students to solve real-world scenarios.
- Utilize online resources, explore online learning modules, simulations, and interactive quizzes to reinforce concepts at the student's pace.

7. Details of assessment

7.1. Overall Scheme of Assessment (Summative)

Sr. No	Professional Course	Term I (1-6 Months)		Term II(7-12 Months)		
1	Third Professional BHMS	PA I (end of 3 months)	TT I (end of 6 months)	PA II (end of 9 months)	FUE (end of 12 months)	
		05 Marks Viva	25 Marks Viva voce	05 Marks Viva	50 marks theory	50 marks (Viva+ IA)

PA: Periodical Assessment; TT: Term Test; FUE: Final University Examinations; IA: Internal Assessment

7.2.Number of papers and Marks Distribution for Final University Examination (FUE)

Sr. No.	Course Code	Papers	Theory	Viva Voce	Internal Assessment*	Grand Total
1	Hom.UG-Mod. Phar-I	01	50 marks	40 marks	10 marks (Marks of PA I + TT I + PA II)	100 marks

***Method of Calculation of Internal Assessment Marks for Final University Examination:**

Marks of IA- **(**Marks of PA-1 + Marks of TT + Marks of PA-2) / 35 X 10

7.3.Paper Layout
Summative assessment (FUE):
Theory- 50 marks

MCQ	**5 marks (5 questions each of 1mark)**
SAQ	**15 marks (3 questions each of 5 marks)**
LAQ	**30marks (3 questions each of 10 marks)**

7.4.Theme-wise distribution of questions for theory paper:

Theme	Topics	Marks	MCQ's	SAQ's	LAQ's
A	Introduction to Pharmacology	2	02	0	0
B	Pharmacokinetics	5	0	01	0
	Pharmacodynamics				
C	ANS and Autacoids	10	0	0	01
D	NSAID	2	02		
	CNS	10	0	0	01
	Respiratory system				
F	Renal system	1	01	0	0
G	Blood	5		01	
H	GIT	10	0	0	01
I	Hormones	5	0	01	0
	CVS				
	Chemotherapy				
	Miscellaneous				
	Total	50	05	03	03

7.5.Question paper blueprint

A Question Serial Number	B Type of Question	Question Paper Format (Refer Table 2 for themes)
Q.1	Multiple choice Questions (MCQ) 5 Questions 1 mark each All compulsory	Theme A Theme D Theme F
Q.2	Short Answer Questions (SAQ) 3 Questions 5 marks each All compulsory	Theme B Theme G Theme I
Q.3	Long Answer Questions (LAQ) 3 Questions 10 marks each All compulsory	Theme C Theme E Theme H

8. List of recommended Books

- Rang & Dale's Pharmacology
- Goodman & Gilman's The Pharmacological Basis of Therapeutics
- K.D. Tripathi – Essentials of Medical Pharmacology
- Katzung's Basic & Clinical Pharmacology

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