PHARMACOLOGY · STUDY GUIDE

Exam 1 Study Guide

This guide covers the first two chapters. Chapter 1 gives you the tools you will use for every drug in the course: names and classes, what the body does to a drug, what the drug does to the body, safe administration, and the supplements patients forget to mention. Chapter 2 puts those tools to work on the antimicrobials. Work one chapter at a time. Read the "what to know" paragraphs, copy the tables out by hand, then try each worked example with the steps covered. Finish each chapter with its ten self-test items. Any dose or number in a worked example is a practice calculation only, not dosing advice. The cram sheet at the end puts the whole exam on one page.

WHAT THIS GUIDE COVERS

- Chapter 1 · chemical, generic and trade names, stems, classes and prototypes, ADME, bioavailability, protein binding, the first-pass effect, half-life and steady state, receptors, agonists and antagonists, potency versus efficacy, the therapeutic index, the rights of administration, routes, error prevention and herbal interactions.
- Chapter 2 · selective toxicity, spectrum, cidal versus static, culture and sensitivity, the antibacterial classes by target, resistance and stewardship, antifungals, antivirals, antiprotozoals, anthelmintics and the nursing care that applies to every antimicrobial.

CHAPTER 1

Pharmacological Principles and Herbal Supplements

Read it in full: **Chapter 1 in the textbook**. Practice it in the lab: **The Journey of a Drug (ADME)** and **What the Drug Does to the Body**.

What to know

Every drug has three names. The **chemical name** describes the molecule and you will almost never use it. The **generic name** is the drug's one true name, owned by no company and used on the order, the chart and in references: acetaminophen. The **trade name** is the brand a company

owns: Tylenol. Nurses work in generic names because a duplicate cannot hide there. A patient taking Tylenol, Percocet and a nighttime cold remedy may be taking acetaminophen three times.

Generic names carry **stems** that tell you the class on sight: -olol is a beta blocker, -pril an ACE inhibitor, -statin an HMG-CoA reductase inhibitor, -prazole a proton pump inhibitor, -cillin a penicillin and -sartan an angiotensin II receptor blocker.

A **drug class** shares a mechanism, so it shares effects, adverse effects and nursing care. A **therapeutic** classification says what the drug treats (antihypertensive). A **pharmacologic** classification says how it works (beta1-selective blocker). Metoprolol is both at once. Each class has a **prototype**, the member you learn cold and use as the template: morphine for the opioids, furosemide for the loop diuretics, metoprolol for the beta blockers. Faced with a new drug, ask first: what class, and what is the prototype?

Pharmacokinetics is what the body does to the drug, remembered as ADME. **Absorption** moves the drug from its site into the blood; it depends on route, blood flow, membrane crossing and food. **Bioavailability** is the fraction that reaches the circulation intact: 100% for IV, usually less by mouth. A patient in shock absorbs an IM or SubQ dose poorly.

Distribution carries the drug into tissues. Heart, brain, liver and kidney get it first. Only lipid-soluble drugs cross the blood-brain barrier easily; the placenta stops almost nothing. Many drugs ride on albumin and only the **free** (unbound) fraction is active, so a patient with low albumin has more free drug from the same dose and can be toxic at a normal dose.

Metabolism happens mostly in the liver through the CYP450 enzymes. It usually inactivates a drug, but a **prodrug** must be activated: codeine becomes morphine by CYP2D6. An **inducer** (rifampin, phenytoin, carbamazepine, St. John's wort) speeds metabolism so partner drugs fall too low. An **inhibitor** (azole antifungals, clarithromycin, amiodarone, grapefruit juice) slows it so partner drugs climb toward toxicity. The **first-pass effect** is liver metabolism of an oral dose before it reaches the body: oral morphine needs roughly three times the IV dose. IV, sublingual, transdermal, inhaled and partly rectal routes bypass it.

Excretion is mostly renal, so poor kidney function means accumulation and renally cleared drugs are adjusted to creatinine clearance or eGFR. The **half-life** is the time for plasma concentration to

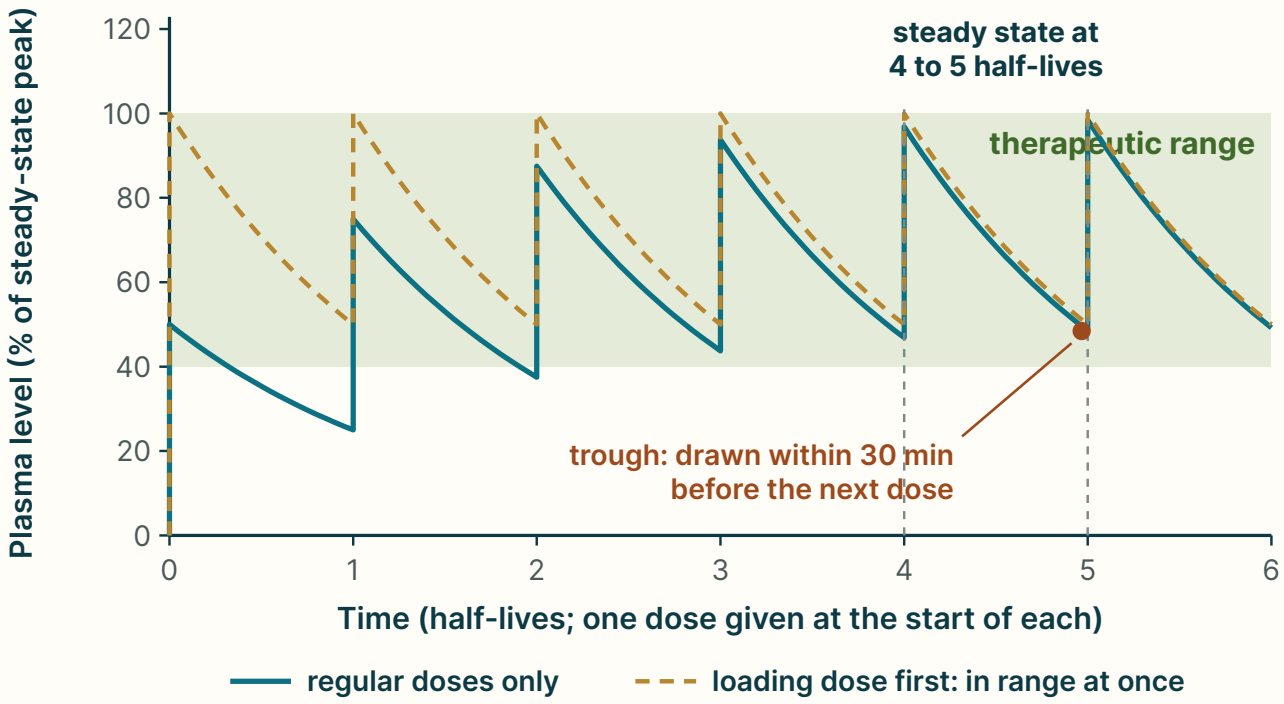
fall by half. It takes about four to five half-lives to reach **steady state** and about four to five to clear a drug. A **loading dose** fills the tank when the patient cannot wait. A **trough** is drawn within the 30 minutes before the next dose and shows accumulation.

Pharmacodynamics is what the drug does to the body. Most drugs bind a receptor the body already uses. **Affinity** is how tightly it binds; **intrinsic activity** is whether binding triggers a response. An **agonist** binds and activates. An **antagonist** binds and blocks, with no effect of its own; it is the antidote mechanism (naloxone). A **partial agonist** activates weakly with a ceiling (buprenorphine). Selectivity is dose dependent: metoprolol spills onto beta2 at high doses.

Potency is how many milligrams it takes. **Efficacy** is the maximum effect possible. Hydromorphone is more potent than morphine, not more efficacious. The **therapeutic index** is TD50 divided by ED50. A narrow index means little room for error: warfarin, digoxin, lithium, phenytoin, levothyroxine, theophylline, carbamazepine, vancomycin, aminoglycosides and insulin. Digoxin runs about 0.5 to 2 ng/mL, and hypokalemia (often from furosemide) makes toxicity more likely.

Safe administration rests on the **rights**: patient, drug, dose, route and time, plus documentation, reason, response, the right to refuse and education, checked three times. Right patient means two identifiers. Chart after giving, never before. IV is immediate and cannot be taken back. **High-alert** drugs (insulin, anticoagulants, opioids, concentrated electrolytes, chemotherapy, neuromuscular blockers, sedatives) need an independent double-check, and concentrated potassium chloride is never given IV push. Use a leading zero, never a trailing zero, and spell out "units". An unclear order is clarified, not given.

Supplements are regulated as **food** under DSHEA (1994), with no pre-market proof of safety, potency or purity. Interactions cluster three ways: CYP and P-glycoprotein induction (St. John's wort, which can drop cyclosporine, warfarin, oral contraceptives and antiretrovirals), bleeding (garlic, ginger, ginkgo, ginseng and feverfew), and additive effects (kava and valerian with CNS depressants; ginseng lowering glucose). Kava and black cohosh are linked to liver injury. Echinacea is avoided in immunosuppression. Grapefruit juice inhibits intestinal CYP3A4 for about 24 hours. Ask about everything bought without a prescription, and teach that most herbs stop one to two weeks before surgery.



Half-life sets the climb to steady state. Each dose adds the same amount while the body clears half every half-life, so levels plateau after about 4 to 5 half-lives. A loading dose skips the wait.

LOCK THESE IN

- Nurses work in generic names; stems such as -olol, -pril, -statin, -prazole, -cillin and -sartan name the class.
- ADME: absorption, distribution, metabolism, excretion. IV bioavailability is 100%.
- Only free drug is active; low albumin means more free drug and more toxicity risk.
- Inducers lower partner drug levels; inhibitors raise them. Grapefruit juice is an inhibitor.
- Four to five half-lives to steady state and four to five to clear. A loading dose skips the wait.
- Potency is milligrams; efficacy is the maximum effect. TI = TD50 ÷ ED50.
- Narrow TI list: warfarin, digoxin, lithium, phenytoin, levothyroxine, theophylline, carbamazepine, vancomycin, aminoglycosides, insulin.
- KCl is never IV push. Leading zero yes, trailing zero no. Spell out units.

Key terms and comparisons

Term	What it means	Bedside meaning
Bioavailability	Fraction of a dose reaching the circulation intact	IV 100%; oral usually less (absorption plus first pass)
First-pass effect	Liver metabolism of an oral dose before it reaches the body	Oral morphine about 3 times the IV dose; sublingual nitroglycerin bypasses it
Protein binding	Drug carried on albumin; only free drug acts	Low albumin: toxicity at a normal dose
Inducer vs inhibitor	Speeds vs slows CYP450 metabolism	Inducer: partner fails. Inhibitor: partner builds up
Half-life	Time for plasma level to fall by half	Sets the interval; 4 to 5 half-lives to steady state or to clear
Trough	Lowest level, drawn within the 30 min before the next dose	The safety check for aminoglycosides; many hospitals now dose vancomycin by AUC
Agonist / antagonist / partial	Activates / blocks / activates weakly with a ceiling	Morphine / naloxone / buprenorphine
Potency vs efficacy	Milligrams needed vs maximum effect	More potent is not more effective
Therapeutic index	TD50 ÷ ED50	Narrow index: levels, double-checks

Routes at a glance

Route	Onset	First pass?	Key nursing point
Oral	30 to 60 min	Yes	Never crush extended-release or enteric-coated tablets
Sublingual / buccal	1 to 5 min	No	Nitroglycerin; never with a PDE-5 inhibitor
Subcutaneous	Slow, sustained	No	Usually 1 mL or less; do not massage after heparin
Intramuscular	Moderate	No	Ventrogluteal site; fails in shock
Intravenous	Immediate	No	Irreversible; know rate, compatibility and antidote
Transdermal	Slow, steady	No	Remove the old patch first; date and initial
Inhaled	Fast, local	No	Bronchodilator before steroid; rinse mouth after steroid
Rectal	Variable	Partial	For vomiting or unconscious patients

Worked examples

WORKED EXAMPLE 1 · HALF-LIFE AND STEADY STATE

A drug has a half-life of 8 hours. A level of 64 mcg/mL is drawn right after a single IV dose. What is left at 8, 16, 24 and 32 hours, and about how long until steady state on regular dosing? (Practice numbers only.)

STEP 1 · HALVE ONCE PER HALF-LIFE

8 h: 32. 16 h: 16. 24 h: 8. 32 h: 4 mcg/mL.

STEP 2 · EXPRESS IT AS A FRACTION

After 4 half-lives, 4 ÷ 64 = 1/16 = 6.25% is left. After 5 half-lives it would be 3.125%.

STEP 3 · APPLY THE RULE

Steady state takes about 4 to 5 half-lives: 4 × 8 = 32 h to 5 × 8 = 40 h, so **about 32 to 40 hours**.

THE LESSON

The same 4 to 5 half-lives also clear the drug after the last dose. If a level is needed now, a loading dose is the answer, not a faster schedule.

WORKED EXAMPLE 2 · READING THE THERAPEUTIC INDEX

Drug A has a TD50 of 600 mg and an ED50 of 20 mg. Drug B has a TD50 of 3 mg and an ED50 of 1.5 mg. Which needs levels and a double-check?

STEP 1 · DIVIDE FOR DRUG A

600 ÷ 20 = a TI of **30**. Wide margin.

STEP 2 · DIVIDE FOR DRUG B

3 ÷ 1.5 = a TI of **2**. The toxic dose is only twice the effective dose.

STEP 3 · DECIDE

Drug B is the narrow-index drug. It behaves like digoxin or lithium: serum levels, careful dosing, and watching the things that push levels up, such as hypokalemia or poor kidney function.

WORKED EXAMPLE 3 · A ROUTE CHANGE IS NOT A STRAIGHT SWAP

A patient comfortable on IV morphine is switched to oral. The book says oral morphine needs roughly three times the IV dose. If the IV dose in this practice problem is 4 mg, what oral amount gives a similar effect, and why?

STEP 1 · APPLY THE RATIO

4 mg × 3 = **about 12 mg** by mouth (a practice figure; real conversions come from the prescriber and pharmacist).

STEP 2 · NAME THE CAUSE

The oral dose goes through the portal vein to the liver first, and much of it is destroyed there: the first-pass effect.

STEP 3 · CONNECT TO OTHER ROUTES

Sublingual, transdermal and IV routes skip the portal circulation, which is why nitroglycerin is placed under the tongue rather than swallowed.

WORKED EXAMPLE 4 · A HOME MEDICATION LIST

Mr. Okafor, 66, lists Tylenol, Percocet, a nighttime cold remedy, warfarin and "ginkgo for memory." What does the nurse do?

STEP 1 · TRANSLATE TO GENERIC NAMES

Tylenol is acetaminophen; Percocet contains acetaminophen; many nighttime cold remedies do too. He may be taking one drug three times and exceeding the maximum daily dose.

STEP 2 · SCREEN THE SUPPLEMENT

Ginkgo has antiplatelet activity. With warfarin it raises bleeding risk. It also lowers the seizure threshold.

STEP 3 · ACT

Document every product on the medication list, notify the prescriber about the duplicate acetaminophen and the ginkgo with warfarin, and teach him to tell every prescriber about every supplement.

WHY IT WORKS

The generic name is the only place a duplicate cannot hide, and the purse check finds the interaction no one prescribed.

TRAPS PEOPLE FALL INTO

- **Treating a trade name as the drug.** Translate every product to its generic name, or duplicates slip through.
- **Confusing potency with efficacy.** A smaller milligram dose does not mean a stronger maximum effect.
- **Swapping IV and oral doses one for one.** The first-pass effect means oral doses are often much larger for the same effect.
- **Drawing a trough early.** Early draws read falsely high. Draw within the 30 minutes before the next dose.
- **Assuming naloxone ends the danger.** It is shorter acting than most opioids, so the patient can re-sedate. Keep watching.
- **Calling a supplement "not a medication."** Supplements are drugs with real interactions. Ask about everything bought without a prescription.
- **Charting before giving.** Documentation comes after administration, or a dose gets given twice.

Chapter 1 self-test · ten items

1. Which name for a drug is the one that belongs to no company and appears on the order?

A. The brand name
B. The trade name
C. The generic name
D. The chemical name

C. The generic name The generic (nonproprietary) name never changes and is used on orders, charts and references. Trade and brand names are owned by a manufacturer; the chemical name describes the molecule.

2. A drug name ends in -sartan. What class does the stem point to?

A. Gastric proton pump inhibitor
B. Angiotensin II receptor blocker
C. Beta1-selective adrenergic blocker
D. Angiotensin converting enzyme inhibitor

B. Angiotensin II receptor blocker The -sartan stem marks an angiotensin II receptor blocker. ACE inhibitors end in -pril, beta blockers in -olol, and proton pump inhibitors in -prazole.

3. A patient with an albumin of 2.0 g/dL starts a usual dose of a highly protein-bound drug. What is the main concern?

A. More free drug and a risk of toxicity
B. Less free drug and a weaker effect
C. Faster renal clearance of the drug
D. No change, since albumin is irrelevant

A. More free drug and a risk of toxicity Only unbound drug is active. With less albumin to carry it, more drug circulates free, so a normal dose can act like a large one.

4. Which drug is a prodrug that must be converted by CYP2D6 to work?

A. Morphine
B. Furosemide
C. Naloxone
D. Codeine

D. Codeine Codeine is inactive until CYP2D6 converts it to morphine. Poor metabolizers get little relief; ultrarapid metabolizers can be overdosed.

5. A patient on an oral contraceptive starts rifampin. What should the nurse expect?

A. Higher levels because rifampin blocks renal excretion
B. Higher contraceptive levels from enzyme inhibition
C. Lower contraceptive levels from enzyme induction
D. No interaction, since rifampin is an antibiotic only

C. Lower contraceptive levels from enzyme induction Rifampin is a CYP450 inducer. It speeds metabolism of partner drugs, so contraceptives can fail. A backup method is taught.

6. A drug has a half-life of 6 hours. About how long does it take to reach steady state?

A. 24 to 30 hours
B. 6 to 12 hours
C. 12 to 18 hours
D. 48 to 60 hours

A. 24 to 30 hours Steady state takes 4 to 5 half-lives: $4 \times 6 = 24$ and $5 \times 6 = 30$ hours.

7. Which drug acts as a partial agonist at the mu receptor?

A. Morphine
B. Buprenorphine
C. Hydromorphone
D. Naloxone

B. Buprenorphine Buprenorphine activates the mu receptor weakly with a ceiling. Naloxone is an antagonist; morphine and hydromorphone are full agonists.

8. Which drug in this list has a narrow therapeutic index?

A. Cefazolin
B. Amoxicillin
C. Omeprazole
D. Lithium

D. Lithium Lithium sits on the narrow-index list with warfarin, digoxin, phenytoin, insulin and others. The other three have wide margins.

9. An order reads "KCl 20 mEq IV push now." What does the nurse do?

A. Do not give it and clarify the order
B. Give it slowly over two minutes
C. Give it after a second nurse checks
D. Dilute it in a 10 mL syringe and push

A. Do not give it and clarify the order Concentrated potassium chloride is never given IV push. It must be diluted and infused. An order that does not make sense is clarified, not given.

10. Ms. Varga takes cyclosporine after a liver transplant and has begun an herbal mood remedy, St. John's wort. Which outcome is the nurse most worried about?

A. Serotonin syndrome from the cyclosporine
B. High cyclosporine levels and kidney injury
C. Bleeding from antiplatelet effects
D. Low cyclosporine levels and graft rejection

D. Low cyclosporine levels and graft rejection St. John's wort induces CYP3A4 and P-glycoprotein, driving cyclosporine below its range. Rejection is documented. Notify the prescriber at once.

CHAPTER 2

Antimicrobial Drugs

Read it in full: **Chapter 2 in the textbook**. Practice it in the lab: **Antibiotic Targets on a Bacterial Cell** and **Reading the Culture & Sensitivity**.

What to know

Selective toxicity makes antimicrobials possible: the drug attacks something the microbe has and the patient lacks. Bacteria have a peptidoglycan wall, 70S ribosomes (30S plus 50S) and make their own folate. Human cells have no wall, 80S ribosomes, and get folate from food. Where microbe and host overlap, toxicity appears: human mitochondrial ribosomes look bacterial, which is why linezolid suppresses marrow and aminoglycosides injure the inner ear.

Narrow-spectrum drugs hit a few organisms; **broad-spectrum** drugs hit many. Broad is a starting guess, not a goal. It wipes out normal flora, inviting **superinfection** (C. difficile, thrush, vaginal yeast) and resistance. **Bactericidal** drugs kill (beta-lactams, aminoglycosides, fluoroquinolones, vancomycin, metronidazole). **Bacteriostatic** drugs stall growth (tetracyclines, macrolides, clindamycin, sulfonamides, linezolid). A cidal drug is generally needed in endocarditis, meningitis and neutropenia, where the immune system cannot finish the job.

A **culture and sensitivity** names the organism and reports the **MIC**, the lowest concentration that stops growth, with a susceptible, intermediate or resistant label. Read the label, not just the number. Cultures take 24 to 72 hours, so **empiric** therapy starts first and is **de-escalated** to a narrow drug when results return. Draw cultures before the first dose, but in sepsis the antibiotic still goes in within the hour. Surgical prophylaxis is timed within 60 minutes before incision.

Cell wall drugs are bactericidal. The **beta-lactams** (penicillins, cephalosporins, carbapenems and the monobactam aztreonam) bind penicillin-binding proteins. Cephalosporins climb by generation: cefazolin (first, surgical prophylaxis), ceftriaxone (third, meningitis and pneumonia), cefepime (fourth, Pseudomonas), ceftaroline (fifth, MRSA). Carbapenems are reserved for resistant gram-negatives and carry seizure risk. Aztreonam is the option in true penicillin anaphylaxis. **Vancomycin** is nephrotoxic and ototoxic, needs AUC (or trough, per facility protocol) levels and creatinine, and is infused over at least 60 minutes. **Daptomycin** needs weekly CPK and is inactivated by lung surfactant, so it is not for pneumonia.

Protein synthesis drugs split by subunit: "buy AT 30, CELL at 50." At 30S: aminoglycosides (gentamicin; nephrotoxic and ototoxic, peak and trough, daily creatinine) and tetracyclines (doxycycline; photosensitivity, teeth staining, avoid under 8 and in pregnancy except doxycycline for suspected tick-borne rickettsial illness; separate from dairy, antacids and iron). At 50S: macrolides (QT prolongation), clindamycin (the classic C. difficile trigger) and linezolid (thrombocytopenia, optic neuropathy, serotonin syndrome with SSRIs).

Nucleic acid drugs: fluoroquinolones block DNA gyrase and carry warnings for tendon rupture, aortic aneurysm, neuropathy, CNS effects, QT and glucose swings; separate them from calcium, iron and antacids. Rifampin blocks RNA polymerase, turns body fluids orange, is hepatotoxic and a strong CYP inducer. Metronidazole damages anaerobe DNA and causes a disulfiram-like reaction with alcohol during and 3 days after therapy. **Metabolic** drugs: TMP-SMX blocks two folate steps; watch for rash progressing to SJS, hyperkalemia, crystalluria and a rising INR, and avoid it in the first trimester and at term. Isoniazid needs pyridoxine to prevent neuropathy and is hepatotoxic.

Antibiotics **select for** resistance; they do not create it. Genes spread by conjugation, transformation and transduction. Four mechanisms: enzymes (beta-lactamase, ESBL,

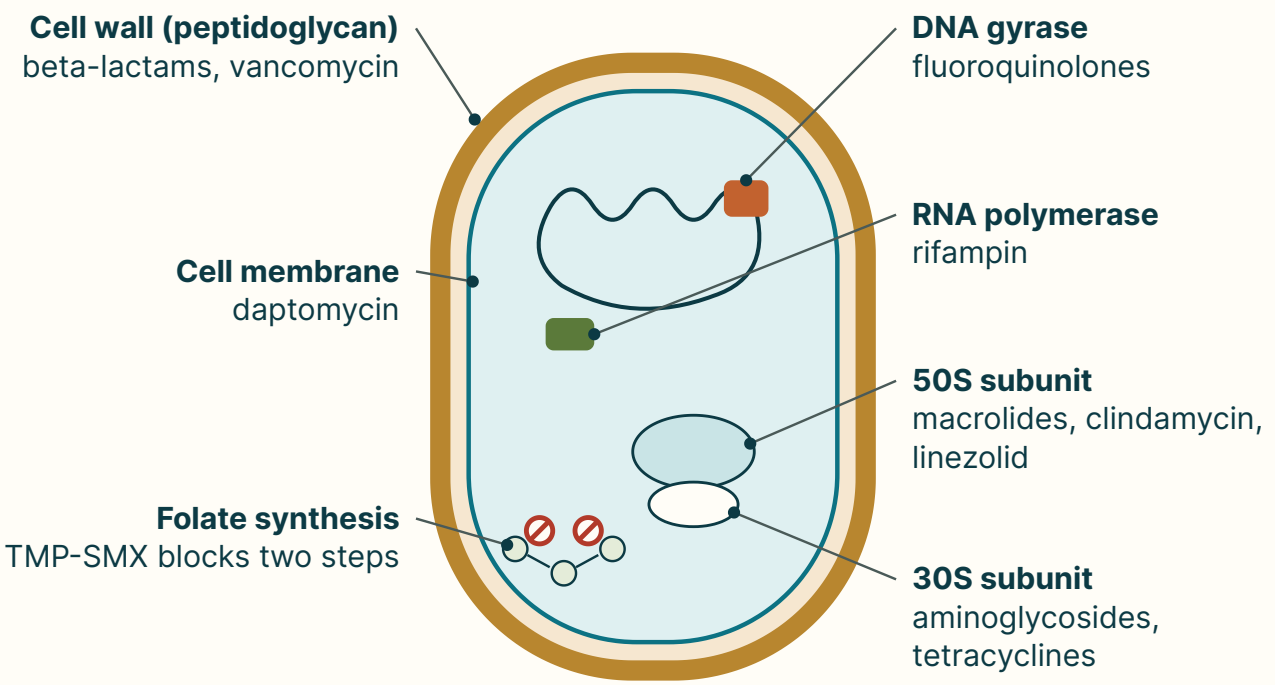
carbapenemase), target change (MRSA's mecA builds PBP2a; VRE's vanA changes D-Ala-D-Ala to D-Ala-D-Lac), efflux pumps and reduced permeability. MRSA defeats every traditional beta-lactam; options are vancomycin, linezolid, daptomycin and ceftaroline. ESBL pushes toward carbapenems. C. difficile is treated with oral vancomycin or fidaxomicin, and soap and water are used because alcohol gel does not kill spores. Oral and IV vancomycin are not interchangeable.

Stewardship is nursing work: cultures first, a real allergy history (about 9 in 10 people labeled penicillin allergic are not), a day 3 review, IV to PO when the patient is eating and stable, the right duration, teaching and hand hygiene. Vancomycin flushing is a rate problem, not an allergy; slow the infusion and do not chart it as an allergy.

Fungi are eukaryotes, so antifungals are rougher. Targets are **ergosterol** and **beta-glucan**. Amphotericin B binds ergosterol: rigors ("shake and bake") and nephrotoxicity with potassium and magnesium loss; conventional and liposomal doses are not interchangeable. Azoles block ergosterol synthesis: liver toxicity, QT and CYP interactions; voriconazole adds visual changes. Echinocandins hit the glucan wall, are the best tolerated, IV only and first-line for invasive candidemia. Terbinafine needs LFTs. Nystatin is swish and swallow.

Antivirals target viral enzymes and mostly suppress rather than cure. Acyclovir is activated by viral thymidine kinase; hydrate and infuse IV doses over at least an hour. Ganciclovir suppresses marrow. Oseltamivir works best within about 48 hours, but hospitalized, severe or high-risk patients are treated at any point. For HIV, integrase inhibitors are first-line, ritonavir is a strong CYP3A4 inhibitor, abacavir needs **HLA-B*5701** screening, and adherence prevents resistance. HCV direct-acting antivirals cure most patients in 8 to 12 weeks; screen for hepatitis B first.

Antiparasitics are chosen by life stage. Artemether-lumefantrine is first-line for uncomplicated falciparum malaria, taken with fatty food. Primaquine and tafenoquine clear liver hypnozoites but need **G6PD** screening first. Metronidazole covers giardiasis, amebiasis and trichomoniasis (treat the partner). Albendazole and mebendazole block worm microtubules, ivermectin opens worm chloride channels, praziquantel causes calcium influx. Pinworm: treat the household and repeat in two weeks.



"Buy AT 30, CELL at 50." Human cells have no wall,
80S ribosomes and take folate from food: that is selective toxicity.

Antibacterial targets. Each class hits something the bacterium has and you lack: the wall, the 70S ribosome, its own folate pathway, or its DNA enzymes.

LOCK THESE IN

- Selective toxicity: the smaller the microbe-host gap, the more toxic the drug.
- Narrow-spectrum is the goal; broad is a bridge to de-escalation after C&S.
- 30S: aminoglycosides, tetracyclines. 50S: chloramphenicol, macrolides, clindamycin, linezolid.
- Vancomycin: infuse over at least 60 min, levels and creatinine; flushing is a rate problem.
- Fluoroquinolones: tendon rupture, aortic aneurysm, QT; separate from calcium, iron and antacids.
- Metronidazole: no alcohol during and 3 days after. Rifampin: orange fluids, CYP inducer.
- MRSA: vancomycin, linezolid, daptomycin, ceftaroline. C. diff: oral vancomycin or fidaxomicin, soap and water.
- Screen before the first dose: HLA-B*5701 for abacavir, G6PD for primaquine.

Key antibacterial classes

Class	Prototype	Target	Nursing alert
Penicillins	penicillin G, amoxicillin	Cell wall (PBPs)	Ask what the "allergy" was; observe 30 min after first dose
Cephalosporins	cefazolin, ceftriaxone	Cell wall	Low cross-reactivity; no ceftriaxone with calcium IV in neonates
Carbapenems	meropenem	Cell wall	Reserve drug; seizure risk, worse in renal impairment
Glycopeptide	vancomycin	Cell wall	Over 60 min or more; nephrotoxic, ototoxic; AUC or trough per protocol
Aminoglycosides	gentamicin	30S	Nephrotoxic and ototoxic; peak and trough; report tinnitus
Tetracyclines	doxycycline	30S	Sun, teeth, upright 30 min, no dairy or iron with dose
Macrolides	azithromycin	50S	QT prolongation; erythromycin and clarithromycin inhibit CYP3A4
Lincosamide	clindamycin	50S	Report new diarrhea: C. difficile
Oxazolidinone	linezolid	50S	Weekly CBC; serotonin syndrome with SSRIs
Fluoroquinolones	ciprofloxacin	DNA gyrase	Tendon, aorta, nerves, QT, glucose
Rifamycin	rifampin	RNA polymerase	Orange fluids; hepatotoxic; CYP inducer
Sulfonamide combo	TMP-SMX	Folate pathway	Rash to SJS, K+, fluids, INR; not first trimester or term

Antifungals and antivirals

Drug	Target	Watch for
amphotericin B	Ergosterol (pores)	Rigors, nephrotoxicity, low K+ and Mg2+; check formulation
fluconazole, voriconazole	Ergosterol synthesis	LFTs, QT, CYP interactions; vision changes (voriconazole)
micafungin, caspofungin	Beta-glucan wall	Best tolerated; IV only
acyclovir	Viral DNA polymerase	Hydrate; IV over at least 1 h
oseltamivir	Neuraminidase	Best within about 48 h; severe or high risk at any time
abacavir	Reverse transcriptase (NRTI)	HLA-B*5701 before the first dose
primaquine, tafenoquine	Liver hypnozoites	G6PD screen first

Worked examples

WORKED EXAMPLE 1 · CULTURE FIRST, THEN NARROW

Ms. Delgado, 58, arrives with fever, low blood pressure and a suspected urinary source. The team orders blood and urine cultures and a broad empiric antibiotic. Walk through the next 72 hours.

STEP 1 · TIMING

Draw the cultures fast, then hang the antibiotic. In sepsis the drug goes in within the first hour; the draw must not delay it.

STEP 2 · WHILE WAITING

Give doses on time and evenly spaced. Watch the fever curve, white count, blood pressure and creatinine.

STEP 3 · DAY 3 REVIEW

The culture grows an organism susceptible to a narrow drug. Flag it: the broad drug should be **de-escalated**.

STEP 4 · IV TO PO

When she is eating and stable, ask whether an oral option fits.

WHY IT MATTERS

Staying broad after the report wastes flora, invites C. difficile and selects for resistance.

WORKED EXAMPLE 2 · TIMING A TROUGH

A patient receives an IV antibiotic every 12 hours, with doses at 0900 and 2100. When is the trough drawn, and what happens if it is drawn at 2130?

STEP 1 · FIND THE NEXT DOSE

The next dose after 0900 is at 2100.

STEP 2 · BACK UP 30 MINUTES

The trough is drawn about 30 minutes before the next dose: **about 2030**.

STEP 3 · SPOT THE ERROR

At 2130 the new dose is already running, so the level is not a trough at all. It may read high and lead to a wrong dose change.

STEP 4 · CHART IT

Record the exact draw time. A level at the wrong time is worse than none, because someone will act on it.

WORKED EXAMPLE 3 · INFUSION RATE FOR A SLOW ANTIBIOTIC

Vancomycin must run over at least 60 minutes. A practice order gives 250 mL to infuse over 2 hours. What is the pump rate, and does it meet the minimum time?

STEP 1 · SET UP

Rate = volume ÷ time = 250 mL ÷ 2 h.

STEP 2 · CALCULATE

250 ÷ 2 = **125 mL/h**.

STEP 3 · CHECK THE RULE

Two hours is longer than the 60 minute minimum, so the rate is acceptable.

IF FLUSHING APPEARS

Stop, give an antihistamine per order, restart slower. Chart the event, not "allergy."

WORKED EXAMPLE 4 · NEW DIARRHEA ON CLINDAMYCIN

Mr. Haddad, 71, on day 5 of clindamycin, has four watery stools since morning. What is the nurse thinking and doing?

STEP 1 · NAME IT

Clindamycin is the classic C. difficile trigger. This is likely superinfection after normal flora were destroyed.

STEP 2 · ACT

Notify the prescriber, send stool, start contact precautions.

STEP 3 · CLEAN CORRECTLY

Use soap and water. Alcohol gel does not kill spores.

STEP 4 · AVOID

Do not give antimotility agents. Expect the offending antibiotic to stop and oral vancomycin or fidaxomicin to start.

TRAPS PEOPLE FALL INTO

- **Thinking broad-spectrum is better.** Broad is a guess; narrow is the goal once the organism is known.
- **Charting vancomycin flushing as an allergy.** It is a rate reaction. Slow the infusion and document what happened.
- **Assuming any cephalosporin covers MRSA.** Only ceftaroline does. Cefazolin and ceftriaxone do not.
- **Giving IV vancomycin for C. difficile.** Only oral vancomycin reaches the gut lumen.
- **Using alcohol gel after a C. diff room.** Spores survive alcohol. Wash with soap and water.
- **Skipping the screen.** HLA-B*5701 before abacavir and G6PD before primaquine are decided before the first dose.
- **Withholding doxycycline from a child with suspected tick-borne illness.** It is first-line at any age there; short courses do not cause meaningful staining.

Chapter 2 self-test · ten items

1. Why is penicillin so much safer than linezolid?

A. Penicillin is cleared faster by the liver

B. Penicillin is given only in small doses

C. Human cells have no cell wall to attack

D. Linezolid is taken by mouth, not IV

C. Human cells have no cell wall to attack Penicillin targets wall cross-linking, which humans lack. Linezolid hits a ribosome that resembles human mitochondrial ones, so marrow suppression follows.

2. Which situation most clearly calls for a bactericidal drug?

A. Prophylaxis before a dental cleaning
B. Endocarditis in a patient with a vegetation
C. Mild acne treated over several months
D. A simple skin infection in a healthy adult

B. Endocarditis in a patient with a vegetation Bacteriostatic drugs rely on the immune system to finish the job. Vegetations, meningitis and neutropenia leave no one to finish it.

3. A culture report returns on day 3 showing a susceptible organism. What should happen to the broad empiric drug?

A. Add a second broad drug for safety
B. Stop all antibiotics until symptoms return
C. Continue it to complete a full course
D. De-escalate to a narrow drug that covers it

D. De-escalate to a narrow drug that covers it Empiric therapy is a bridge. Once the organism and its susceptibilities are known, therapy narrows to spare flora and limit resistance.

4. Which pair both bind the 30S ribosomal subunit?

A. Gentamicin and doxycycline
B. Linezolid and erythromycin
C. Azithromycin and clindamycin
D. Clindamycin and gentamicin

A. Gentamicin and doxycycline Aminoglycosides and tetracyclines act at 30S. Macrolides, clindamycin and linezolid act at 50S.

5. A patient on ciprofloxacin reports new heel pain. What is the priority?

A. Apply heat and recheck the heel in a week
B. Continue the drug and offer a cushioned heel pad
C. Hold the drug and report possible tendinitis
D. Give an NSAID and encourage daily walking

C. Hold the drug and report possible tendinitis Fluoroquinolones can cause Achilles tendinitis and rupture, especially over 60 or on steroids. Hold and notify.

6. What should a patient avoid during metronidazole therapy and for 3 days after?

A. Sunlight
B. Dairy
C. Caffeine
D. Alcohol

D. Alcohol Metronidazole causes a disulfiram-like reaction with alcohol: flushing, vomiting and tachycardia.

7. Why does MRSA resist every traditional beta-lactam at once?

A. mecA builds an altered binding protein, PBP2a
B. It lacks a peptidoglycan cell wall entirely
C. It makes an enzyme that destroys vancomycin
D. It pumps out methicillin but not other drugs

A. mecA builds an altered binding protein, PBP2a All beta-lactams work by binding PBPs. PBP2a ignores penicillins, cephalosporins and carbapenems alike. Ceftaroline is the engineered exception.

8. Which antifungal is the best tolerated and first-line for invasive candidemia?

A. Terbinafine
B. Micafungin
C. Amphotericin B
D. Voriconazole

B. Micafungin Echinocandins attack beta-glucan, a target humans lack, so they are well tolerated. They are IV only.

9. What test is done before the first dose of abacavir?

A. G6PD level
B. Serum creatinine
C. CD4 count only
D. HLA-B*5701

D. HLA-B*5701 A positive HLA-B*5701 predicts a potentially fatal hypersensitivity reaction, so the drug is not given.

10. A family is treated for pinworm. Which teaching is correct?

A. Give one dose and wash linens in cold water
B. Collect the tape test at bedtime after a bath
C. Treat everyone and repeat the dose in two weeks
D. Treat only the child who has symptoms

C. Treat everyone and repeat the dose in two weeks Pinworm spreads through the household. Treat all members, repeat in two weeks, wash linens hot, and collect the tape test in the morning before bathing.

Cram sheet

Everything from chapters 1 and 2 in one place. Cover the right column and see if you can rebuild each row.

Principles

Item	Fact	Note
Names	Chemical, generic, trade	Work in generic names
Stems	-olol, -pril, -statin, -prazole, -cillin, -sartan	Beta blocker, ACE inhibitor, statin, PPI, penicillin, ARB
ADME	Absorption, distribution, metabolism, excretion	IV bioavailability 100%
First pass	Oral morphine about 3 times IV	Bypassed by IV, SL, transdermal, inhaled
Inducers	rifampin, phenytoin, carbamazepine, St. John's wort	Partner drug falls
Inhibitors	azoles, clarithromycin, amiodarone, grapefruit	Partner drug rises
Half-life rule	4 to 5 half-lives to steady state and to clear	Loading dose skips the wait
Trough	About 30 min before next dose	Vancomycin, aminoglycosides
TI	TD50 ÷ ED50	Narrow: warfarin, digoxin, lithium, phenytoin, insulin and more
Digoxin	About 0.5 to 2 ng/mL	Hypokalemia raises toxicity; apical pulse 1 full min, hold under 60
Safety	Two identifiers; chart after giving	KCl never IV push; leading zero, no trailing zero
Supplements	St. John's wort induces; garlic, ginger, ginkgo, ginseng, feverfew bleed	Kava and black cohosh: liver; stop herbs 1 to 2 weeks before surgery

Antimicrobials

Item	Fact	Note
Cidal	Beta-lactams, aminoglycosides, fluoroquinolones, vancomycin, metronidazole	Needed in endocarditis, meningitis, neutropenia
Static	Tetracyclines, macrolides, clindamycin, sulfonamides, linezolid	Need a working immune system
Ribosome	30S: AG, tetracyclines; 50S: chloramphenicol, macrolides, clindamycin, linezolid	"buy AT 30, CELL at 50"
Cultures	Before first dose; 24 to 72 h	Sepsis: antibiotic within the first hour
Prophylaxis	Within 60 min before incision	Cefazolin is the classic
MRSA	vancomycin, linezolid, daptomycin, ceftaroline	Contact precautions
VRE / ESBL / CRE	linezolid or daptomycin / carbapenem / newer combinations	Infection prevention
C. difficile	Oral vancomycin or fidaxomicin	Soap and water; no antimotility drugs
Antifungals	Ampho B, azoles, echinocandins, terbinafine, nystatin	Ergosterol and beta-glucan
Antivirals	Acyclovir, oseltamivir (about 48 h), ART, HCV DAAs	Abacavir: HLA-B*5701
Antiparasitics	ACT with fatty food; primaquine needs G6PD	Pinworm: household, repeat in 2 weeks

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