

Antiparasitics and anthelmintics

Two families of drugs grouped by what they target rather than a shared mechanism - antiprotozoals against single-celled parasites like Plasmodium, and anthelmintics against the worms that cause soil-transmitted helminthiasis, schistosomiasis and related infections.

Online guide:

<https://www.medguideglobal.com/pharmacology/antivirals-antifungals-antiparasitics/antiparasitics-and-anthelmintics>

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Quick revision

One Liner: Antiparasitic drugs are organized by target rather than by one shared mechanism: benzimidazoles block helminth microtubule formation, ivermectin paralyzes nematodes through glutamate-gated chloride channels, praziquantel disrupts calcium handling in flatworms, and the antimalarials interrupt the parasite's heme-detoxification and folate pathways inside red blood cells.

1. Albendazole and mebendazole (benzimidazoles) inhibit microtubule synthesis by preventing tubulin polymerization, which blocks glucose uptake in helminths and leads to their death. [7]
2. Ivermectin binds glutamate-gated chloride channels found in invertebrate nerve and muscle cells, causing hyperpolarization and paralysis of the parasite; mammalian glutamate receptors are not glutamate-gated chloride channels, which is part of the drug's selective toxicity. [7]
3. Praziquantel increases cell-membrane permeability to calcium in cestodes and trematodes, disrupting the parasite's tegument and musculature. [7]
4. Artemether-lumefantrine (Coartem) combines a fast-acting artemisinin derivative with a longer-acting partner drug and is CDC's preferred first-line treatment for uncomplicated *P. falciparum* malaria in the United States. [8]
5. Atovaquone-proguanil (Malarone) is a second-line option for uncomplicated malaria, generally well tolerated but with a longer parasite clearance time than artemether-lumefantrine. [8]
6. Ivermectin is the treatment of choice for both strongyloidiasis and onchocerciasis, and is also used for scabies, illustrating how one mechanism spans several very different clinical indications. [3][7]
7. Chloroquine's older mechanism - inhibiting heme detoxification (heme polymerase activity) and nucleic acid biosynthesis in the malaria parasite - remains a foundational exam concept even though resistance has limited its clinical use in many regions. [7]
8. Albendazole's own label carries a warning about fatal bone marrow suppression with prolonged or repeated dosing, which is why blood counts are monitored during extended courses such as hydatid disease treatment. [1]

Overview

1. Antiparasitic drugs are best organized by what kind of organism they target rather than by a single shared mechanism, because the two broad target groups - protozoa and helminths - are biologically very different. Antiprotozoal drugs act against single-celled organisms such as the Plasmodium species that cause malaria; anthelmintics act against multicellular worms, further divided into nematodes (roundworms), cestodes (tapeworms) and trematodes (flukes). A drug effective against one group is frequently useless against the other, which is why this class page groups drugs by the organism they were built for rather than presenting one unified mechanism story. [7]
2. Among the anthelmintics, the benzimidazoles (albendazole and mebendazole) are the mainstay for soil-transmitted helminth infections such as ascariasis, trichuriasis and hookworm, working by blocking microtubule formation in the worm and starving it of the glucose uptake microtubules normally support. Ivermectin, derived from the avermectin family of compounds produced by Streptomyces avermitilis, works through an entirely different channel-based mechanism and is the preferred drug for strongyloidiasis and onchocerciasis specifically, with an additional established

role in scabies. Praziquantel targets a third group of helminths altogether - the flatworms, meaning schistosomes and tapeworms - through a calcium-permeability mechanism unrelated to either of the other two anthelmintic classes. [7][3]

3. Antimalarial therapy in current U.S. practice for uncomplicated *Plasmodium falciparum* infection centers on two combination regimens. Artemether-lumefantrine (Coartem) is CDC's preferred option because the artemisinin component rapidly reduces parasite load; atovaquone-proguanil (Malarone) is a second-line option, generally well tolerated but with slower parasite clearance and treatment-failure risk that keeps it behind artemether-lumefantrine in the treatment hierarchy. Older agents such as chloroquine remain conceptually important - chloroquine's heme-detoxification-blocking mechanism is a classic teaching point - even though resistant *P. falciparum* has limited chloroquine's use in most malaria-endemic regions. [8][7]

4. Because these drugs act on organisms with very different biology from bacteria or human cells, their toxicity profiles are correspondingly distinct from antibacterials: albendazole's principal serious risk is bone marrow suppression with prolonged dosing, ivermectin's central concern in some settings is neurologic toxicity with concurrent heavy *Loa loa* infection, and praziquantel's main precaution is exacerbation of central nervous system disease in neurocysticercosis or during the acute phase of schistosomiasis. Each of these is discussed drug-by-drug in the adverse-effects section below rather than folded into a single class-wide toxicity statement. [1][4]

Classification and drug examples

Intro: Antiparasitics divide first into antiprotozoal and anthelmintic drugs, and the anthelmintics divide further by which type of worm they target - a structure driven by target biology rather than chemical class alone.

1. Name: Benzimidazole anthelmintics

1. Note: First-line for soil-transmitted helminth infections; act by blocking microtubule formation.

1. Name: Albendazole

Albenza

1. Route: Oral

1. Note: Also used for neurocysticercosis and hydatid (echinococcal) disease at doses requiring blood-count monitoring. [1]

2. Name: Mebendazole

1. Emverm

2. Vermox

2. Route: Oral

2. Note: Used for pinworm, whipworm, roundworm and hookworm infections. [2] [7]

2. Name: Avermectin anthelmintic

2. Note: A single-drug subclass acting through glutamate-gated chloride channels, distinct from the benzimidazole mechanism.

Name: Ivermectin

Stromectol

Route: Oral (also topical for lice/rosacea, not covered here)

Note: Drug of choice for strongyloidiasis and onchocerciasis; also used for scabies. [3] [3]

3. Name: Anti-trematodal / anti-cestodal agent

3. Note: Targets flatworms (flukes and tapeworms) through a calcium-permeability mechanism distinct from the nematode-targeted drugs above.

Name: Praziquantel

Biltricide

Route: Oral

Note: Treats schistosomiasis (all *Schistosoma* species) and liver-fluke infections (clonorchiasis, opisthorchiasis); also used for tapeworm infections. [4] [4]

4. Name: Antimalarial agents

4. Note: Antiprotozoal drugs targeting Plasmodium species; current U.S. first- and second-line choices for uncomplicated P. falciparum.

1. Name: Artemether/lumefantrine

Coartem

1. Route: Oral

1. Note: CDC's preferred treatment for uncomplicated P. falciparum malaria in the United States; taken with fatty food to improve absorption. [5]

2. Name: Atovaquone/proguanil

Malarone

2. Route: Oral

2. Note: Second-line treatment option, also used for malaria chemoprophylaxis; not recommended for treatment in patients with severe renal impairment. [6]

3. Name: Chloroquine

Chloroquine phosphate

3. Route: Oral

3. Note: A 4-aminoquinoline effective only against chloroquine-sensitive parasites; widespread resistance limits its current role. [7] [8]

Mechanism of action

Summary: Anthelmintic and antiprotozoal drugs act on structurally unrelated targets because they act on structurally unrelated organisms. Benzimidazoles block tubulin polymerization, starving helminths of the microtubule-dependent glucose uptake they need to survive. Ivermectin opens glutamate-gated chloride channels specific to invertebrate nerve and muscle, producing paralysis. Praziquantel increases calcium permeability across the flatworm tegument. The antimalarials act inside the infected red blood cell or the parasite's own metabolism: chloroquine blocks the parasite's detoxification of heme released during hemoglobin digestion, while the artemisinins and the atovaquone-proguanil combination interrupt free-radical generation and mitochondrial electron transport respectively. Target: Helminth microtubules (benzimidazoles), invertebrate glutamate-gated chloride channels (ivermectin), flatworm tegument calcium permeability (praziquantel), and Plasmodium heme detoxification or mitochondrial function (antimalarials)

Cidal Static: Anthelmintics are generally parasitocidal at the organism level (they kill or paralyze and expel the worm); antimalarials are parasitocidal against the erythrocytic stage of Plasmodium responsible for clinical illness

Kill Kinetics: Anthelmintic courses are typically short (single dose to a few days) because the target is a whole organism rather than a replicating population; antimalarial combination regimens are dosed over 3 days to clear the rapidly multiplying blood-stage parasite before recrudescence can occur

1. Step: 1

1. Title: Benzimidazoles block helminth microtubule assembly

1. Detail: Albendazole and mebendazole inhibit microtubule synthesis by preventing tubulin polymerization; because microtubules are needed for the worm's own glucose uptake, this leads to energy depletion and death of the parasite. [7]

2. Step: 2

2. Title: Ivermectin opens glutamate-gated chloride channels

2. Detail: Ivermectin binds selectively to glutamate-gated chloride channels in nematode nerve and muscle cells, increasing chloride permeability, hyperpolarizing the cell, and producing paralysis of the parasite's pharyngeal and somatic musculature. [7]

3. Step: 3

3. Title: Praziquantel disrupts calcium handling in flatworms

3. Detail: Praziquantel increases the permeability of the flatworm's cell membrane to calcium, disrupting ion transport

in cestodes and trematodes and damaging the tegument, which exposes the parasite to the host immune system. [7]

4. Step: 4

4. Title: Chloroquine blocks heme detoxification in Plasmodium

4. Detail: As the malaria parasite digests host hemoglobin inside the red blood cell, it must detoxify the released heme by polymerizing it into inert hemozoin; chloroquine inhibits this heme polymerase activity, allowing toxic free heme to accumulate and kill the parasite. [7]

5. Step: 5

5. Title: Artemether and atovaquone-proguanil interrupt separate parasite processes

5. Detail: Artemether's artemisinin component is activated by iron released during hemoglobin digestion and generates reactive intermediates that damage parasite proteins; lumefantrine, its longer-acting partner drug, is combined with it to reduce recrudescence. Atovaquone separately collapses the parasite's mitochondrial membrane potential by inhibiting the cytochrome bc1 complex, while proguanil's active metabolite inhibits parasite dihydrofolate reductase. [5][6]

Spectrum of activity

1. Subclass Id: benzimidazoles

1. Coverage: Soil-transmitted helminths, including *Ascaris lumbricoides*, *Trichuris trichiura*, *Ancylostoma duodenale* and *Necator americanus*; albendazole additionally covers neurocysticercosis (*Taenia solium* larvae) and hydatid disease (*Echinococcus granulosus* larvae). [1][2]

2. Subclass Id: avermectins

2. Coverage: *Strongyloides stercoralis* (intestinal strongyloidiasis) and *Onchocerca volvulus* (onchocerciasis) are ivermectin's core FDA-recognized indications; ivermectin is also used against *Sarcoptes scabiei* (scabies). [3]

3. Subclass Id: antitrematodal-anticestodal

3. Coverage: All *Schistosoma* species causing schistosomiasis, and the liver flukes *Clonorchis sinensis* and *Opisthorchis viverrini*; praziquantel is also used against tapeworm infections. [4]

4. Subclass Id: antimalarials

4. Coverage: *Plasmodium falciparum* is the primary target of current combination therapy in the United States; artemether-lumefantrine and atovaquone-proguanil are both used for chloroquine-resistant *P. falciparum* specifically. [8]

Major clinical uses

1. Drug: Albendazole

1. Organism: *Taenia solium* larvae; *Echinococcus granulosus* larvae; soil-transmitted helminths

1. Indication: Parenchymal neurocysticercosis; hydatid (cystic echinococcal) disease of liver, lung and peritoneum

1. Line: first-line

1. Note: Blood counts are monitored at the start of, and every 2 weeks during, each treatment cycle because of bone-marrow-suppression risk. [1]

2. Drug: Mebendazole

2. Organism: *Enterobius vermicularis*, *Trichuris trichiura*, *Ascaris lumbricoides*, *Ancylostoma duodenale*, *Necator americanus*

2. Indication: Pinworm, whipworm, roundworm and hookworm gastrointestinal infections

2. Line: first-line

2. Note: Chewable tablet formulation, indicated from age 1 (Vermox) or age 2 (Emverm) depending on the product. [2]

3. Drug: Ivermectin

3. Organism: *Strongyloides stercoralis*; *Onchocerca volvulus*; *Sarcoptes scabiei*

3. Indication: Intestinal strongyloidiasis; onchocerciasis; scabies

3. Line: first-line

3. Note: Semisynthetic avermectin derived from *Streptomyces avermitilis* fermentation products. [3]

4. Drug: Praziquantel

- 4. Organism: Schistosoma species; Clonorchis sinensis; Opisthorchis viverrini
- 4. Indication: Schistosomiasis (all species); clonorchiasis and opisthorchiasis (liver flukes)
- 4. Line: first-line
- 4. Note: Approved for patients aged 1 year and older. [4]
- 5. Drug: Artemether/lumefantrine
- 5. Organism: Plasmodium falciparum
- 5. Indication: Acute, uncomplicated malaria in patients 2 months of age and older weighing 5 kg or more
- 5. Line: first-line (preferred)
- 5. Note: Not approved for severe/complicated malaria or for malaria prevention. [5][8]
- 6. Drug: Atovaquone/proguanil
- 6. Organism: Plasmodium falciparum
- 6. Indication: Acute, uncomplicated malaria; also used for malaria chemoprophylaxis in travelers
- 6. Line: second-line for treatment
- 6. Note: Generally well tolerated but parasite clearance is slower than with artemether-lumefantrine, with treatment-failure risk. [6][8]

Pharmacokinetics

- 1. Drug: Albendazole
- 1. Route: Oral
- 1. Absorption: Poor and variable when taken without food; significantly increased with a fatty meal
- 1. Csf Penetration: See prescribing reference
- 1. Metabolism: Rapid hepatic first-pass to the active metabolite albendazole sulfoxide
- 1. Elimination: Biliary/renal
- 1. Dose Adjust: Hepatic impairment; blood counts monitored during extended courses [1]
- 2. Drug: Ivermectin
- 2. Route: Oral
- 2. Absorption: Well absorbed on an empty stomach
- 2. Csf Penetration: See prescribing reference
- 2. Metabolism: Hepatic
- 2. Elimination: Predominantly fecal
- 2. Dose Adjust: Caution in areas of concurrent heavy Loa loa infection because of reported severe neurologic reactions in that setting [3]
- 3. Drug: Praziquantel
- 3. Route: Oral
- 3. Absorption: Well absorbed orally, with extensive first-pass metabolism
- 3. Csf Penetration: See prescribing reference
- 3. Metabolism: Hepatic (CYP-mediated)
- 3. Elimination: Renal, mostly as metabolites
- 3. Dose Adjust: Hepatic impairment prolongs half-life; strong CYP inducers (e.g., rifampin) can reduce plasma levels substantially [4]
- 4. Drug: Artemether/lumefantrine
- 4. Route: Oral
- 4. Absorption: Improved substantially by co-administration with fatty food
- 4. Csf Penetration: See prescribing reference
- 4. Metabolism: Hepatic, CYP3A4 (artemether) and CYP3A4 (lumefantrine)
- 4. Elimination: Hepatic/fecal
- 4. Dose Adjust: Take with fatty food; avoid strong CYP3A4 inhibitors/inducers per label interaction guidance [5]

5. Drug: Atovaquone/proguanil

5. Route: Oral

5. Absorption: Atovaquone absorption is markedly improved with food or a milky drink

5. Csf Penetration: See prescribing reference

5. Metabolism: Proguanil is hepatically converted to its active metabolite cycloguanil; atovaquone undergoes minimal metabolism

5. Elimination: Atovaquone: biliary/fecal; proguanil/cycloguanil: renal

5. Dose Adjust: Not recommended for treatment in severe renal impairment; take with food or a milky drink [6]

1. This page gives no dose regimens by design. Dosing for anthelmintics and antimalarials depends on body weight, indication, renal and hepatic function, and (for antimalarials) local resistance patterns, and belongs in a current prescribing reference used by the treating clinician.

2. Food dramatically affects absorption for several drugs in this class - albendazole, artemether-lumefantrine and atovaquone-proguanil all specify taking the dose with a fatty meal or milky drink to achieve adequate exposure, a pharmacokinetic detail with direct practical consequences for treatment success. [1][5][6]

Adverse effects

1. Effect: Gastrointestinal upset

1. Detail: Nausea, abdominal pain and diarrhea are reported with benzimidazoles, praziquantel and the antimalarial combinations. [1][4][5]

2. Effect: Headache and dizziness

2. Detail: Common with praziquantel and with the antimalarial combinations. [4][5]

3. Effect: Skin and mucosal reactions with ivermectin in onchocerciasis treatment

3. Detail: Pruritus and other reactions can occur as dying microfilariae provoke a local inflammatory response (a Mazzotti-type reaction), rather than representing a direct ivermectin toxicity. [3]

1. Effect: Bone marrow suppression (albendazole)

1. Detail: Fatalities have been reported from bone marrow suppression, including aplastic anemia and agranulocytosis, particularly with prolonged or repeated albendazole courses.

1. Action: Complete blood counts are checked at the beginning of each 28-day treatment cycle and every 2 weeks during therapy. [1]

2. Effect: Central nervous system exacerbation (praziquantel)

2. Detail: Praziquantel can exacerbate CNS pathology in patients with neurocysticercosis or cause potentially life-threatening clinical deterioration if given during the acute phase of schistosomiasis.

2. Action: Consideration of seizure history and disease stage before treatment; corticosteroids may be co-administered for neurocysticercosis per the treating clinician's judgment. [4]

3. Effect: Severe neurologic reactions with ivermectin in heavy Loa loa infection

3. Detail: Rare but serious encephalopathy-type reactions have been reported when ivermectin is used in patients with very high Loa loa microfilarial loads, a consideration specific to co-endemic regions.

3. Action: Screening for Loa loa co-infection is relevant in mass drug administration programs in co-endemic areas. [3]

4. Effect: Hemolysis in G6PD deficiency (antimalarials)

4. Detail: Some antimalarial regimens carry hemolysis risk in G6PD-deficient patients; this is a standard pre-treatment consideration for antimalarial therapy generally.

4. Action: G6PD status is a relevant consideration before starting antimalarial treatment where indicated by the specific regimen. [7]

5. Effect: QT interval effects (antimalarials)

5. Detail: Antimalarial combination regimens are used with attention to cardiac risk factors and concomitant QT-prolonging drugs.

5. Action: Cardiac history and concomitant medications are reviewed before starting combination antimalarial therapy.

[5]

1. Drug: Albendazole

1. Effect: The clearest bone-marrow-suppression signal among the anthelmintics covered here, tied specifically to prolonged or repeated dosing rather than single-dose use. [1]

2. Drug: Praziquantel

2. Effect: The only drug in this class with an explicit warning about worsening the disease process itself (CNS pathology in neurocysticercosis; deterioration in acute schistosomiasis) rather than an independent organ toxicity. [4]

3. Drug: Ivermectin

3. Effect: Its adverse-reaction profile in onchocerciasis treatment is dominated by host reactions to dying microfilariae rather than direct drug toxicity. [3]

4. Drug: Atovaquone/proguanil

4. Effect: Generally the best-tolerated of the antimalarial options discussed here, which is part of why it remains a viable second-line and prophylactic choice despite slower parasite clearance in treatment. [8]

Contraindications, precautions and interactions

Praziquantel: concurrent ocular cysticercosis is a reason to avoid treatment, since destruction of the parasite in the eye can cause irreversible damage. [4]

1. A history of seizures or known neurocysticercosis is a caution with both albendazole and praziquantel, given the risk of a treatment-triggered CNS inflammatory reaction as cysts are killed. [1][4]

2. Pregnancy status is reviewed before starting most agents in this class; atovaquone-proguanil is not recommended for malaria prophylaxis or preferred for treatment during pregnancy per CDC guidance, while artemether-lumefantrine is a CDC-recognized option in the second and third trimesters and, when other options are unavailable, the first trimester. [8]

3. Severe renal impairment is a caution for atovaquone-proguanil use, particularly for prophylaxis. [6]

4. Heavy Loa loa microfilarial load is a caution before mass ivermectin administration in co-endemic regions because of reported severe neurologic reactions. [3]

1. With: Strong CYP3A4 inducers (e.g., rifampin)

1. Effect: Can reduce plasma levels of praziquantel and of artemether/lumefantrine substantially, risking treatment failure. [4][5]

2. With: Other QT-prolonging drugs

2. Effect: Reviewed before combination antimalarial therapy given the cardiac considerations built into current dosing guidance. [5]

3. With: Fatty food

3. Effect: Not a drug interaction in the traditional sense, but co-administration with fatty food or a milky drink substantially changes absorption for albendazole, artemether-lumefantrine and atovaquone-proguanil, and is part of correct administration. [1][5][6]

Resistance mechanisms

1. Mechanism: Beta-tubulin mutations (benzimidazole resistance)

1. Detail: Because benzimidazoles work by binding parasite tubulin, mutations in beta-tubulin that reduce drug binding are the principal resistance mechanism described for this drug class, more extensively documented in veterinary than human helminth populations but conceptually the same target-based mechanism. [7]

2. Mechanism: Chloroquine resistance in Plasmodium falciparum

2. Detail: Mutations affecting a parasite transporter reduce chloroquine accumulation in the parasite's digestive vacuole, preventing the drug from reaching the concentration needed to block heme detoxification; this resistance is now widespread enough that chloroquine is no longer first-line therapy in most malaria-endemic regions. [7]

3. Mechanism: Artemisinin partial resistance

3. Detail: Delayed parasite clearance associated with certain Plasmodium falciparum kelch13 gene mutations has been observed in parts of Southeast Asia and, more recently, parts of Africa, which is why artemisinins are always used as part of a combination (with a partner drug like lumefantrine) rather than alone. [8]

Comparison tables

1. Title: Which anthelmintic for which worm

1. Caption: The three anthelmintic mechanisms line up with three distinct worm groups; matching mechanism to organism is the fastest way to recall which drug applies to a given infection.

1. Key: drug

1. Label: Drug

1. Is Row Header: true

2. Key: target_organism

2. Label: Target organism group

3. Key: mechanism

3. Label: Mechanism

1. Drug: Albendazole / mebendazole

1. Target Organism: Soil-transmitted nematodes (roundworm, whipworm, hookworm)

1. Mechanism: Microtubule/tubulin polymerization inhibition [7]

2. Drug: Ivermectin

2. Target Organism: Strongyloides, Onchocerca (nematodes); also scabies mite

2. Mechanism: Glutamate-gated chloride channel opening, paralysis [7]

3. Drug: Praziquantel

3. Target Organism: Schistosoma, liver flukes, tapeworms (flatworms)

3. Mechanism: Increased calcium permeability, tegument disruption [7]

2. Title: First- versus second-line malaria treatment

2. Caption: CDC positions artemether-lumefantrine ahead of atovaquone-proguanil for uncomplicated P. falciparum treatment mainly on speed of parasite clearance, not on overall safety.

1. Key: drug

1. Label: Regimen

1. Is Row Header: true

2. Key: line

2. Label: Treatment line

3. Key: note

3. Label: Distinguishing feature

1. Drug: Artemether/lumefantrine (Coartem)

1. Line: Preferred first-line

1. Note: Fastest parasite clearance because of the artemisinin component [8]

2. Drug: Atovaquone/proguanil (Malarone)

2. Line: Second-line for treatment

2. Note: Generally well tolerated; slower clearance, treatment-failure risk [8]

High-yield exam pearls

1. Pearl: Match mechanism to worm type: microtubule inhibition for nematodes (benzimidazoles), chloride-channel paralysis for nematodes via a different route (ivermectin), and calcium-permeability disruption for flatworms (praziquantel).

1. Why: This is the fastest way to predict which drug applies to which helminth on an exam without memorizing every indication separately. [7]

2. Pearl: Ivermectin is the drug of choice for strongyloidiasis and onchocerciasis specifically - not a general-purpose anthelmintic first choice for soil-transmitted helminths.
2. Why: Students often default to ivermectin for any worm infection; the benzimidazoles remain first-line for ascariasis, trichuriasis and hookworm. [3]
3. Pearl: Praziquantel can worsen neurocysticercosis or acute schistosomiasis by provoking inflammation as the parasite dies.
3. Why: This is a genuinely counterintuitive point - an effective drug causing apparent clinical deterioration - that is a favorite exam trap. [4]
4. Pearl: Artemether-lumefantrine is always a combination, never artemisinin-class monotherapy, specifically to prevent recrudescence and slow the emergence of artemisinin partial resistance.
4. Why: The combination-therapy principle mirrors the multidrug logic used in tuberculosis treatment and is frequently tested as a general antiparasitic/antimicrobial theme. [8]
5. Pearl: Chloroquine blocks heme detoxification (heme polymerase), not folate metabolism - that folate-pathway mechanism belongs to proguanil instead.
5. Why: The two mechanisms are easy to swap on exams because both drugs are 'antimalarials'; they act on entirely different parasite pathways. [7]
6. Pearl: Albendazole's most serious risk is hematologic (bone marrow suppression), not hepatic or neurologic, and is tied to prolonged/repeated dosing rather than single-dose deworming.
6. Why: Single-dose albendazole for soil-transmitted helminths carries a very different risk profile from the extended dosing used for hydatid disease or neurocysticercosis. [1]

Common exam traps

1. Trap: Assuming ivermectin is first-line for all nematode infections.
1. Correction: Ivermectin's core FDA-recognized anthelmintic indications are strongyloidiasis and onchocerciasis; the benzimidazoles remain first-line for the common soil-transmitted helminths (Ascaris, Trichuris, hookworm). [3][2]
2. Trap: Treating praziquantel-induced worsening of neurologic symptoms in neurocysticercosis as a drug failure.
2. Correction: It reflects the expected inflammatory response to dying cysts, a labelled risk requiring caution rather than evidence the drug is not working. [4]
3. Trap: Assuming chloroquine remains a reliable first-line antimalarial everywhere.
3. Correction: Widespread Plasmodium falciparum resistance has removed chloroquine from first-line status in most malaria-endemic regions; current CDC-preferred options for uncomplicated P. falciparum are artemether-lumefantrine and, second-line, atovaquone-proguanil. [8]
4. Trap: Confusing artemether's mechanism (iron-mediated free-radical generation from hemoglobin digestion) with lumefantrine's role (a longer-acting partner drug reducing recrudescence).
4. Correction: The two components of Coartem have distinct roles: artemether provides fast initial killing, lumefantrine provides sustained exposure to prevent recurrence. [5]
5. Trap: Treating single-dose deworming with albendazole as carrying the same bone-marrow-suppression risk as extended courses for hydatid disease or neurocysticercosis.
5. Correction: The FDA-labelled hematologic monitoring requirement is tied to prolonged or repeated 28-day treatment cycles, not brief single-dose regimens for common soil-transmitted helminths. [1]

Self-test questions

1. Question: Which mechanism is shared by albendazole and mebendazole?
1. Glutamate-gated chloride channel opening
2. Inhibition of tubulin polymerization (microtubule synthesis)
3. Increased calcium permeability of the tegument
4. Inhibition of heme polymerase

1. Explanation: Both are benzimidazoles that inhibit microtubule synthesis by preventing tubulin polymerization, blocking the helminth's glucose uptake and leading to its death. [7]
2. Question: Ivermectin's mechanism of action is best described as:
 1. Blockade of folate synthesis in the parasite
 2. Binding glutamate-gated chloride channels in invertebrate nerve/muscle, causing paralysis
 3. Inhibition of parasite RNA polymerase
 4. Disruption of the parasite cell wall
2. Explanation: Ivermectin binds selectively to glutamate-gated chloride channels found in invertebrate nerve and muscle cells, increasing chloride permeability and causing hyperpolarization and paralysis. [7]
3. Question: Which drug is specifically associated with worsening neurologic disease when used to treat neurocysticercosis?
 1. Ivermectin
 2. Mebendazole
 3. Praziquantel
 4. Atovaquone/proguanil
3. Explanation: Praziquantel's label warns it can exacerbate CNS pathology due to neurocysticercosis, since killing the cysts provokes an inflammatory response. [4]
4. Question: Why is artemether never used as monotherapy for malaria?
 1. It has no antimalarial activity alone
 2. Monotherapy risks recrudescence and encourages the emergence of artemisinin partial resistance, so it is paired with a longer-acting partner drug like lumefantrine
 3. It is too toxic to give without a second drug
 4. It only works against Plasmodium vivax
4. Explanation: Artemisinin-class drugs act quickly but clear rapidly from the body, so pairing them with a longer-acting partner drug like lumefantrine prevents recrudescence and slows the development of artemisinin resistance. [8]
5. Question: What is the principal serious adverse effect that drives blood-count monitoring during prolonged albendazole therapy?
 1. QT prolongation
 2. Bone marrow suppression
 3. Optic neuritis
 4. Hepatic cytochrome induction
5. Explanation: Albendazole's label describes fatalities from bone marrow suppression with prolonged or repeated dosing, which is why complete blood counts are checked at the start of and during each treatment cycle. [1]
6. Question: Between artemether-lumefantrine and atovaquone-proguanil for uncomplicated P. falciparum malaria, what is the main reason CDC prefers the former as first-line?
 1. Atovaquone-proguanil is not FDA-approved for treatment
 2. Artemether-lumefantrine achieves faster parasite clearance because of its artemisinin component
 3. Atovaquone-proguanil cannot be used in adults
 4. Artemether-lumefantrine has no food interaction
6. Explanation: CDC guidance identifies artemether-lumefantrine as preferred because the artemisinin component rapidly reduces parasite load, while atovaquone-proguanil, though generally better tolerated, has slower parasite clearance and a higher treatment-failure risk. [8]

Frequently asked questions

1. Question: Why are anthelmintics grouped by the worm they treat instead of by chemical class alone?
1. Answer: Because the three main anthelmintic mechanisms in wide use - microtubule inhibition, glutamate-gated chloride channel opening, and calcium-permeability disruption - line up closely with three distinct worm biology groups

(soil-transmitted nematodes, Strongyloides/Onchocerca, and flatworms respectively). Knowing the target organism usually tells you which mechanism, and therefore which drug, applies. [7]

2. Question: Is ivermectin the right first choice for any worm infection?

2. Answer: No. Ivermectin's core anthelmintic indications are strongyloidiasis and onchocerciasis specifically. For the more common soil-transmitted helminth infections - roundworm, whipworm and hookworm - the benzimidazoles albendazole and mebendazole remain first-line. [3][2]

3. Question: Why would a drug make neurocysticercosis symptoms temporarily worse?

3. Answer: Praziquantel (and albendazole, used similarly for this indication) kills the larval cysts in brain tissue, and the resulting inflammatory response to the dying parasite can transiently worsen neurologic symptoms or provoke seizures. This is a labelled, anticipated risk managed by the treating clinician rather than evidence the drug has failed. [4]

4. Question: Why is chloroquine less commonly used for malaria today?

4. Answer: Chloroquine blocks the malaria parasite's ability to detoxify heme released during hemoglobin digestion, but widespread resistance among Plasmodium falciparum strains - driven by reduced drug accumulation in the parasite - has made it unreliable in most malaria-endemic regions. Current U.S. treatment guidance for uncomplicated P. falciparum favors artemether-lumefantrine first, atovaquone-proguanil second. [7][8]

5. Question: Does atovaquone-proguanil work as well as artemether-lumefantrine for malaria?

5. Answer: It is a recognized, generally well-tolerated second-line option, but CDC guidance notes it clears parasites more slowly and carries a higher risk of treatment failure than artemether-lumefantrine, which is why the latter is preferred first-line for uncomplicated P. falciparum malaria in the United States. [8]

References

[1] ALBENDAZOLE tablet, film coated - prescribing information. DailyMed, U.S. National Library of Medicine . <https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=9ac95554-94c9-1f38-8f49-ec11979e4ba4>

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[3] STROMEKTOL (ivermectin) tablet - prescribing information. DailyMed, U.S. National Library of Medicine . <https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=681888c9-af79-4b7d-ae80-c3f4f6f1effd>

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[5] COARTEM (artemether and lumefantrine) tablet - prescribing information. DailyMed, U.S. National Library of Medicine . <https://dailymed.nlm.nih.gov/dailymed/drugInfo.cfm?setid=7866ec19-dfac-47d4-a53f-511a12643cbf>

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[7] Antiparasitic Drugs. StatPearls Publishing / NCBI Bookshelf 2023. <https://www.ncbi.nlm.nih.gov/books/NBK544251/>

[8] Treatment of Uncomplicated Malaria. Centers for Disease Control and Prevention . <https://www.cdc.gov/malaria/hcp/clinical-guidance/treatment-of-uncomplicated-malaria.html>

For education only. This guide does not replace professional medical advice. Do not use it to choose or change treatment.