

Tuberculosis

A plain-English guide for you and your family

WHAT IS HAPPENING

Tuberculosis is a lung infection caused by a stubborn bacteria called *M. tuberculosis*. When you breathe in the bacteria, immune cells in your lungs called alveolar macrophages (the cleanup crew in the tiny air sacs where oxygen enters your blood) try to eat and kill them. In most people, the body builds tiny walled-off pockets called granulomas around the bacteria to trap them—like putting an intruder in a locked room. The bacteria stay alive inside these pockets but cannot spread or make you sick. This is called latent TB, and you feel fine. But if your immune system gets weak—from HIV, medicines that block a protein called TNF-alpha (used for arthritis or Crohn disease), diabetes, very young or old age, or malnutrition—the walls can crumble and the bacteria wake up and multiply. This is active TB. Now the bacteria damage lung tissue, causing a cough that lasts more than three weeks, coughing up blood, weight loss, drenching night sweats, and fever. The bacteria can also escape into the bloodstream and seed other organs: lymph nodes in the neck (scrofula), the brain's protective lining (meningitis), the spine (Pott disease), the stomach lining, or even spread everywhere at once (miliary TB). Every sign points back to one broken thing: macrophages cannot kill *M. tuberculosis*, so the bacteria keep multiplying and the body keeps fighting, burning energy and damaging tissue in the process.

What you might feel

- Cough that lasts three weeks or longer
- Coughing up blood or rust-colored coughed-up mucus (sputum) (hemoptysis)
- Fever, often with chills
- Drenching night sweats
- Unintentional weight loss and loss of appetite
- Fatigue and general weakness
- Chest pain, especially with deep breathing or coughing
- Cavities (holes) visible on chest X-ray
- In children: miliary TB (spread throughout the body) or tuberculous meningitis (infection of the brain's protective lining)

Call the doctor right away if...

- Coughing up blood (hemoptysis)
- Severe trouble breathing or shortness of breath at rest
- Confusion, stiff neck, or severe headache (signs of meningitis)
- Rapid weight loss (more than 10% of body weight in weeks)
- Known contact with someone who has active TB and new symptoms

What the care team will do

- Place patient in airborne isolation room (negative pressure room with door closed) and wear N95 respirator mask before entering
- Collect three coughed-up mucus (sputum) samples (morning is best) for acid-fast bacilli smear and culture before first antibiotic dose
- Administer all four RIPE medicines (rifampin, isoniazid, pyrazinamide, ethambutol) together at the same time each day, and watch the patient swallow every pill
- Give pyridoxine (vitamin B6) 25 to 50 mg by mouth daily with the isoniazid dose
- Check baseline liver function tests (ALT, AST, bilirubin) and teach patient to report yellowing eyes, dark urine, nausea, or right upper belly pain immediately
- Test vision and red-green color discrimination before starting ethambutol, then monthly during treatment
- Educate patient that urine, sweat, and tears will turn orange-red from rifampin, and soft contact lenses will stain permanently
- Continue airborne isolation until patient has three negative coughed-up mucus (sputum) smears on different days after starting treatment

Medicines you may be given — and why

Isoniazid (INH)

Isoniazid stops TB bacteria from building their protective outer wall. TB bacteria have a thick, waxy coat (called a mycolic acid layer) that acts like armor and lets them hide inside immune cells. Isoniazid blocks the enzyme (a tiny protein machine called InhA) that assembles this waxy armor. Without the armor, the bacteria can't grow or survive, and the body's immune cells can finally kill them.

Rifampin (RIF)

Rifampin stops TB bacteria from copying their DNA instructions into RNA, which is the step they need to make new proteins and grow. It does this by jamming the bacteria's RNA

polymerase (the machine that reads DNA and writes RNA). Without RNA, the bacteria can't make the proteins they need to survive. Rifampin is one of the few drugs that can kill both actively growing TB bacteria and slow-growing or dormant bacteria hiding in tissues, which is why it shortens treatment from 12–18 months down to 6 months.

Pyrazinamide (PZA)

Pyrazinamide is unique because it works best in acidic, low-oxygen environments—exactly the conditions inside macrophages and in the center of granulomas (the walled-off clumps of immune cells and bacteria in the lungs). In these acidic pockets, pyrazinamide is converted by a bacterial enzyme (pyrazinamidase) into pyrazinoic acid, which disrupts the bacteria's energy production and membrane transport. This kills slow-growing, semi-dormant TB bacteria that other drugs miss. Pyrazinamide shortens treatment by clearing these hidden bacteria early, which is why it is used only in the first 2 months of therapy.

Ethambutol (EMB)

Ethambutol blocks the bacteria's ability to build arabinogalactan, a key sugar-based scaffold in the TB bacteria's cell wall. It does this by inhibiting arabinosyl transferases (enzymes that assemble this scaffold). Without arabinogalactan, the bacteria's thick waxy wall becomes incomplete and weak, and the bacteria can't grow or survive. Ethambutol is included in the first 2 months of treatment mainly to protect against drug resistance—if the TB bacteria turn out to be resistant to isoniazid (which isn't known until culture results come back after several weeks), ethambutol helps keep the other drugs working.

Pyridoxine (Vitamin B6)

Pyridoxine is vitamin B6, which nerves need to stay healthy and function properly. Isoniazid lowers vitamin B6 levels in the body by interfering with its metabolism. Without enough B6, nerves can't make the proteins and neurotransmitters they need, and they become damaged, causing tingling, numbness, or burning pain in the hands and feet (peripheral neuropathy). Giving pyridoxine alongside isoniazid prevents this nerve damage by keeping B6 levels normal.

Rifapentine (used with isoniazid in the 3HP regimen for latent TB)

Rifapentine works the same way as rifampin—it jams the TB bacteria's RNA polymerase enzyme so the bacteria can't make RNA or proteins—but it stays in the body much longer (has a longer half-life). This allows it to be given once a week instead of daily. In the 3HP regimen for latent TB infection (infection without active disease), rifapentine is combined with isoniazid and given once weekly for 12 weeks. This is much shorter and more convenient than 6–9 months of daily isoniazid, and people are more likely to complete it. Rifapentine kills dormant TB bacteria hiding in the body before they can wake up and cause active TB disease.

Bedaquiline (for multidrug-resistant TB, MDR-TB)

Bedaquiline blocks the bacteria's ATP synthase, the enzyme that makes ATP (the energy currency of cells). TB bacteria need ATP to survive, grow, and maintain their thick protective wall. By shutting down ATP production, bedaquiline starves the bacteria of energy, causing them to die. Bedaquiline is one of the few drugs that works against multidrug-resistant TB (MDR-TB) and extensively drug-resistant TB (XDR-TB), strains that do not respond to isoniazid and rifampin. It is always used in combination with other second-line TB drugs, never alone.

Pretomanid (for highly resistant TB, used with bedaquiline and linezolid in the BPaL regimen)

Pretomanid is activated inside TB bacteria and creates toxic nitrogen radicals (reactive molecules) that damage the bacteria's DNA, cell wall, and proteins. It works against both actively growing bacteria and dormant bacteria hiding in low-oxygen environments (like the center of granulomas). Pretomanid is used in the BPaL regimen (bedaquiline, pretomanid, linezolid) for extensively drug-resistant TB (XDR-TB) or treatment-intolerant MDR-TB. This is a 6-month, all-oral regimen that replaced much longer, more toxic injectable regimens. Pretomanid must be used with bedaquiline and linezolid—never alone.

Linezolid (for multidrug-resistant TB, part of BPaL and other MDR-TB regimens)

Linezolid stops TB bacteria from making new proteins by blocking the ribosome (the bacteria's protein-making machine) at an early step. It binds to the 50S subunit of the bacterial ribosome and prevents the formation of the initiation complex, so protein synthesis never gets started. Without proteins, the bacteria can't grow, repair themselves, or survive. Linezolid works against multidrug-resistant and extensively drug-resistant TB and is a key part of the BPaL regimen (with bedaquiline and pretomanid) and other MDR-TB regimens.

Where this information comes from

- WHO Global Tuberculosis Report 2024
- ATS/CDC/IDSA Clinical Practice Guidelines for Treatment of Drug-Susceptible Tuberculosis 2016
- CDC Tuberculosis Guidelines 2023
- Cochrane Reviews on Tuberculosis Diagnosis and Treatment

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