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ANTIBIOTIC RESISTANCE: WHY SOME DRUGS DON'T WORK AS INTENDED?

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Abstract

Antibiotics changed everything. For the first time in human history, a simple bacterial infection was no longer a death sentence. Pneumonia, sepsis, tuberculosis — diseases that had killed without mercy for centuries — became treatable within days. Then bacteria did what they have always done throughout billions of years of evolution. They adapted! Antibiotic resistance has quietly become one of the defining medical crises of this century. It does not arrive with the drama of an epidemic. It creeps, ward by ward, prescription by prescription, into the spaces where medicine once felt certain. Today, 1 in 6 bacterial infections worldwide resist the drugs prescribed to treat them. In India, nearly half of all Staphylococcus aureus isolates in hospitals are methicillin-resistant. In the United States, over 35,000 people die from resistant infections every year. Globally, The Lancet projects over 39 million deaths directly from AMR between 2025 and 2050.

This article examines resistance from its molecular foundations to its human consequences — how bacteria defeat antibiotics through enzymatic destruction, efflux pumps, target modification, and biofilm formation; how resistance genes travel silently between species; and which organisms are driving the greatest clinical burden today. We present emerging solutions with real human stories behind them: bacteriophage therapy, which saved Tom Patterson in 2016 when every antibiotic had failed, and CRISPR-based antimicrobials, now being engineered to cut resistance genes directly from bacterial DNA. We close with what antibiotic stewardship means in practice, for the clinician and patient alike.

The 92 million deaths researchers project could be prevented by 2050 will not be saved by declarations alone. They will be saved by doctors who understand why some drugs no longer work. Fleming warned us in 1945. This article is written for the generation that can still act on it.

Key words: Antibiotic resistance, antimicrobial resistance, MRSA, ESKAPE pathogens, Bacteriophage therapy, CRISPR antimicrobials, Antibiotic stewardship, NDM-1, WHO GLASS 2025, Horizontal gene transfer

Introduction

Imagine you approached the doctor with complaint of fever for 3 weeks. You say that you followed the prescription, taken the antibiotics but yet here you are- feeling exactly the same way as before. You will start to believe, “Hmm, maybe the drugs didn’t work or simply Are the doctors up to the task?”

You are not the first one and probably won’t be the last to have such thoughts. Diseases which can be cured easily are becoming much harder to cure or simply impossible. Millions of people are facing the same complications. It’s a life-threatening silent pandemic that slowly infiltrating our current generation. It is not that the drugs or the doctors are “bad”. There is a much important underlying cause. Its not simply as “Drugs vs disease”. Specifically, the term would be Antibiotic resistance.

It’s not a term for the distant future. It’s here, in this century and is reshaping how antibiotics works. According to statistics, 1 in 6 bacterial infections worldwide are now drug resistant and by the year 2050, deaths due to antibiotic resistance will be more rampant as compared to cancer.

For most of the human history, a simple bacterial infection could be death sentence, but then came antibiotics. Pneumonia, Tuberculosis, sepsis etc. became

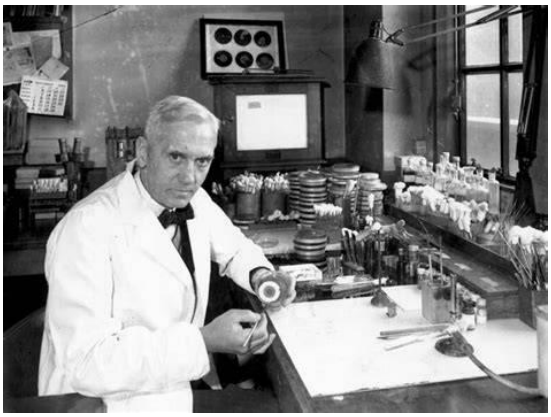
manageable, even ,completely curing them. This was and is our weapon against invisible enemies called bacteria. However, bacteria are ancient survivors that survived mass extinctions, even evolving to adapt extreme conditions such as temperature, geography etc. So, antibiotics became another hurdle for them.

Today bacteria have evolved to neutralize, counter or completely disable the effects of antibiotics which are specifically made to kill them. Some strains are now completely resistant to every antibiotic. We will discuss how does using antibiotics doesn't exactly solve the problem in this article.

"It is not difficult to make microbes resistant to penicillin in the laboratory by exposing them to concentrations not sufficient to kill them, and the same thing has occasionally happened in the body. The time may come when penicillin can be bought by anyone in the shops. Then there is the danger that the ignorant man may easily underdose himself and by exposing his microbes to non-lethal quantities of the drug make them resistant. Moral: if you use penicillin, use enough."

~SIR ALEXANDER FLEMING, NOBEL LAUREATE

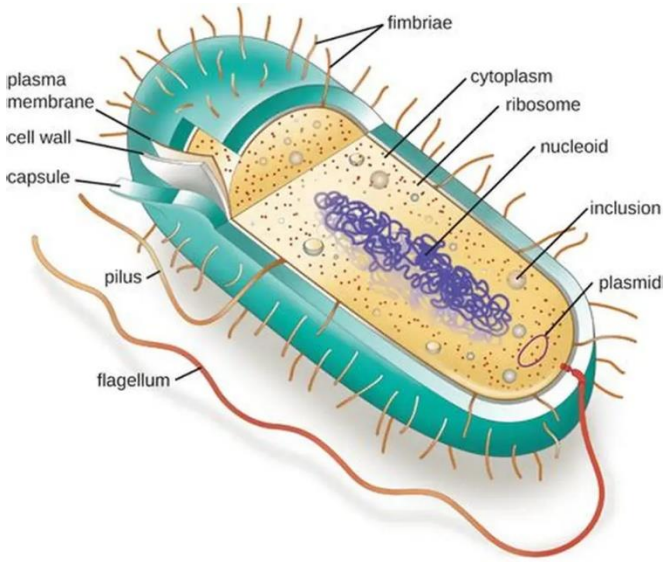
Antibiotic: The Revolution in medicine



In the morning of September 3, 1928, Alexander Fleming — Professor of Bacteriology at St. Mary's Hospital Medical School in London — returned to his laboratory after a month-long summer holiday with his family at The Dhoon, his country home in Barton Mills, Before his departure, he had left several culture plates of Staphylococcus aureus stacked on the corner of a bench, away from direct sunlight, to make space for his research student Stuart Craddock. It was a routine act of laboratory housekeeping — and one that would, by accident, change the course of human history.

What Fleming found upon his return was a contaminated Petri dish. A mould - later identified as Penicillium notatum (now reclassified as Penicillium rubens), thought to have drifted up from a mycology laboratory one floor below - had settled on one of his staphylococcal cultures and created something remarkable: a clear, bacteria-free halo surrounding its colonies, as though the mould were radiating an invisible

antibacterial field. As the American Chemical Society's landmark designation at the Alexander Fleming Laboratory Museum records: "Before its introduction there was no effective treatment for infections such as pneumonia, gonorrhoea or rheumatic fever. Hospitals were full of people with blood poisoning contracted from a cut or a scratch, and doctors could do little for them but wait and hope." In 1929, Fleming named the substance penicillin. In his own words, recounted by PBS NewsHour: "When I woke up just after dawn on September 28, 1928, I certainly didn't plan to revolutionize all medicine by discovering the world's first antibiotic, or bacteria killer. But I guess that was exactly what I did."



Fleming's discovery initially attracted little attention. He himself quickly concluded that it would be impossible to purify and stabilise the antibacterial substance for clinical use, and turned to another research. Penicillin and the Antibiotic Revolution For nearly a decade, penicillin lay dormant in the scientific literature. Then, in 1939, a team at the Sir William Dunn School of Pathology at the University of Oxford - led by Howard Florey and including Ernst Chain, Norman Heatley, and Edward Abraham - resumed intensive research. It was not until 1941 that penicillin was successfully taken from the laboratory to the clinic, and March 1942 that Anne Miller became the first civilian patient to be treated successfully with it, lying near death in New Haven Hospital following a miscarriage complicated by sepsis , Fleming, Florey, and Chain were jointly awarded the Nobel Prize in Physiology or Medicine. The antibiotic era had officially begun. And yet, standing at its very summit, Fleming himself sounded the alarm.

Antibiotic came from two Greek words: “Anti” meaning “Against”, Bios meaning “life”. Antibiotics are medications that fight bacterial infections in people and animals. In simple terms, they generally work by killing the bacteria or making it hard to replicate and grow.

Antibiotics can be:

Oral (by mouth): Pills, capsules or liquid.

Topical: Cream, spray or ointment that you can put on the skin.

Through injection (IV): Usually for serious infections.

A limited number of antibiotics also possess antiprotozoal activity. Antibiotics are not effective against viruses and fungi. In medicine, antibiotics (such as penicillin) are those produced naturally (one microorganism fighting another), whereas non-antibiotic antibacterials (such as sulfonamides and antiseptics) are fully synthetic.

The pharmacology of antibiotics involves destroying bacterial cells by either preventing cell reproduction or altering a necessary cellular function or process. Antimicrobial agents are classically grouped into two main categories based on their in vitro effect on bacteria: bactericidal and bacteriostatic. Common teaching often states that bactericidal antibiotics "kill" bacteria, whereas bacteriostatic antibiotics "prevent the growth" of bacteria. The true definition is not so simple. To accurately define each category, the minimum inhibitory concentration (MIC) and minimum bactericidal concentration (MBC) must be understood. The lowest concentration that inhibits visible bacterial growth at 24 hours is the MIC. The MBC is the antibiotic concentration that reduces bacterial density by 1000-fold at 24 hours.

Bacteriostatic activity is further defined by an MBC-to-MIC ratio greater than 4, whereas an MBC-to-MIC ratio less than or equal to 4 is bactericidal. The clinical implications of antibiotic efficacy depend heavily on many factors not limited to: pharmacokinetic and pharmacodynamic principles, the particular bacteria, bacterial load, and site of infection. This is further complicated by the ability of some bacteriostatic antibiotics to exhibit bactericidal activity against particular bacteria. Therefore, bacteriostatic antibiotics also kill bacteria, but the laboratory definition makes it seem as if they do not. For example, a bacteriostatic antibiotic such as linezolid can be bactericidal against Streptococcus pneumoniae. This concept works in reverse: bactericidal antimicrobials may also be bacteriostatic against certain bacterial strains and under certain conditions. Conflicting data exist as to whether the necessity for bactericidal antibiotics is needed for severely ill or immunosuppressed patients.

The Mechanism

To understand why Antibiotics, fail, we have to understand how they work. As discussed above, Antibiotics generally works on Bacteria. Bacteria are surrounded by a rigid outer wall of peptidoglycan. The wall is necessary because without it the bacteria will swell, rupture and die. Antibiotics target specific structures or functions

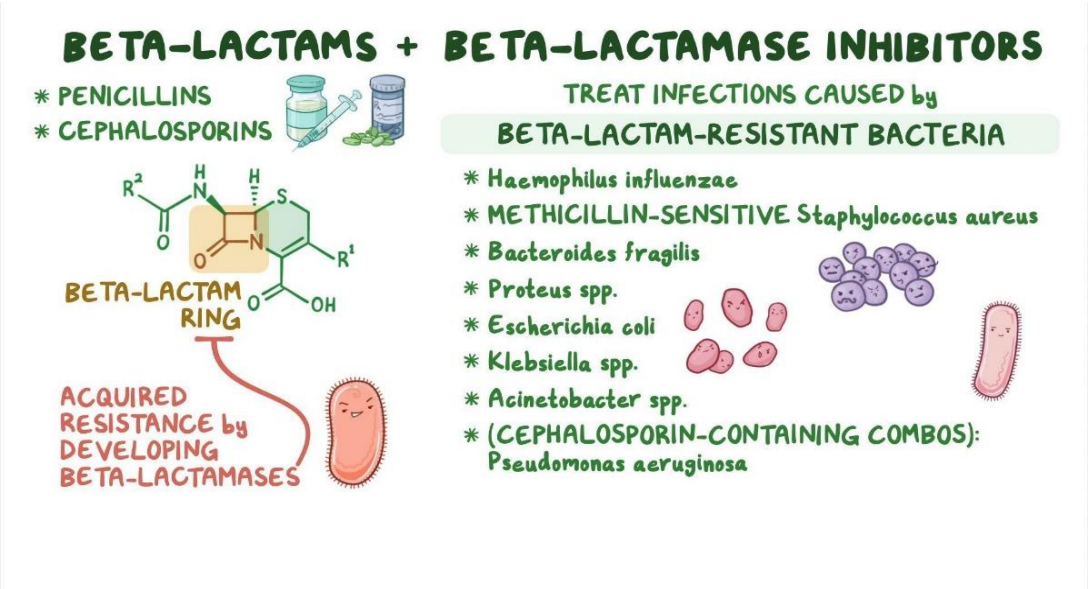
in bacteria such as the cell wall, ribosome synthesis, DNA or RNA synthesis etc. For example, Penicillin, Cephalosporins and Vancomycin all work by interfering with the cell wall synthesis. A bacterium that cannot maintain its cell wall integrity, will simply fall apart.

This is one of the most effective mechanisms and the one most studied because human cells don't have peptidoglycan walls. Some antibiotics like Polymyxins punch holes in this membrane , causing the bacterial contents to leak. However, these kinds of antibiotics should be used as last resort as they can also affect human cell membranes at higher concentrations.

The other way is blocking protein synthesis. Every living cell needs proteins to survive whether that be enzymes, structural components or signaling molecules. Bacteria build these proteins with the help of ribosomes (rRNA). Some of the antibiotics which uses this mechanism are Tetracyclines, Macrolides and Aminoglycosides. The reason why these drugs don't affect us because bacteria has structurally different ribosomes (Prokaryotes: 70S). But here is the neat part, some bacteriostatic don't kill the bacterium outright, they just inhibit the protein synthesis which stalls the time for our immune system to finish it off.

Lastly, some antibiotics don't attack the bacteria's structure at all, they block their nutrient supply. Sulphonamides block bacteria from synthesizing folate, a chemical essential for DNA replication.

The Antibiotic Resistance: The battle between antibiotics and bacteria



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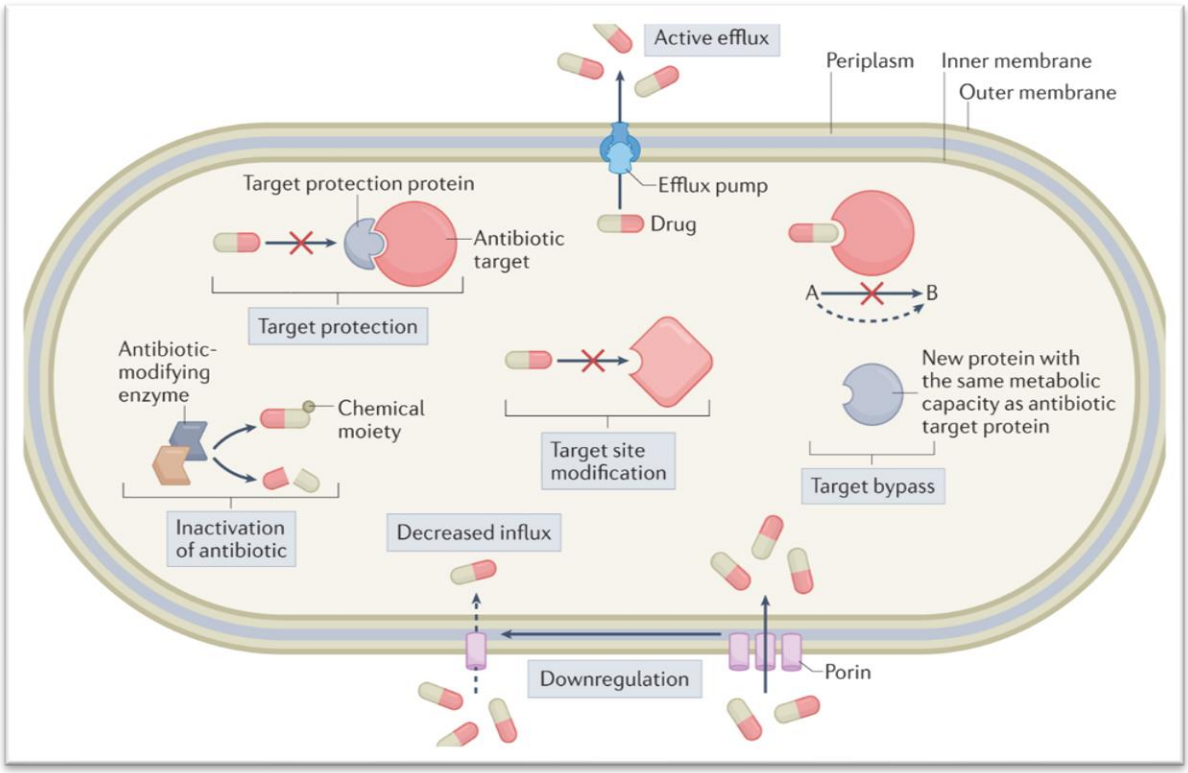
We have discussed how different antibiotics attack specific structures or processed such as Cell wall, protein synthesis, DNA/RNA synthesis etc. But bacteria have found a way to counter everyone of them. To understand resistance, we need to learn about one fundamental reality regarding bacteria. A single bacterium divides every 20 minutes. In one day, billions of bacteria and with every division comes the possibility of mutation. Billions might die in the process due to natural selection but those who survive produce offspring having the same traits.



Some of the ways how bacteria develop resistance are by preventing antibiotics reaching their target site (e.g., changing the permeability of the cell wall or active efflux of the drug), changing the structure of the target cells or entirely replacing them or producing enzymes that neutralize the antibiotic.

Bacteria can acquire resistance genes from other bacteria when microorganisms join together and transfer DNA to each other (conjugation via sex pili), Free floating resistant plasmids are picked up by them or DNA remnant are scavenged from dead bacteria.

However, natural selection is not the only problem, misuse or overuse of antibiotics can also drastically accelerate the process. When antibiotics are used incorrectly, one

or more effects might happen- short time, low dose, inadequate strength or simply wrong medication- bacteria are not killed and can express and pass the resistant gene to even more bacteria. This results in stronger infections, increased intensity of illness and possibly death.

This phenomenon is not only limited to humans. Antibiotics are also used in industries, cattle, poultry etc, to promote similar effect and their growth. Therefore, this also caused the bacteria to develop resistant genes.

According to World Health Organisation, "widespread use of antimicrobials for disease control and growth promotion in animals has been paralleled by an increase in resistance in those bacteria (such as Salmonella and Campylobacter) that can spread from animals, often through food, to cause infections in humans."

But here's the most important question, "How are people exposed to resistant bacteria from animals?"

Cattle and poultry pass the bacteria through their faeces or on skin. One of such examples is Escherichia coli. Faeces contaminated with Antibiotic resistant bacteria can possibly migrate to neighbouring locations or even across long distances. But this is only one way, there are different modes of transmission:

For Animal to animal, it could be direct contact (shared pens, grooming, licking, biting etc), faecal contamination (as discussed above), feed & water (contaminated feed or troughs), vertical transmissions (mothers pass resistant bacteria to offspring).

For Animal to humans, consumption (eating undercooked meat, contaminated water, raw milk or even eggs; Salmonella, Campylobacter and E. coli are most common here), direct contact (pets, livestock or wildlife), environment (manure remnants in rivers, lakes or crops). Farmers, veterinarians and workers in slaughterhouse are at much higher risk.

For Human to human, direct contact (hugging, kissing, sex, holding hands etc.), indirect contact (shared food, using another's belongings, touching contaminated surfaces). Contrary to belief, Hospitals are at more risk of resistant strains (e.g., ESBL E. coli, MRSA).

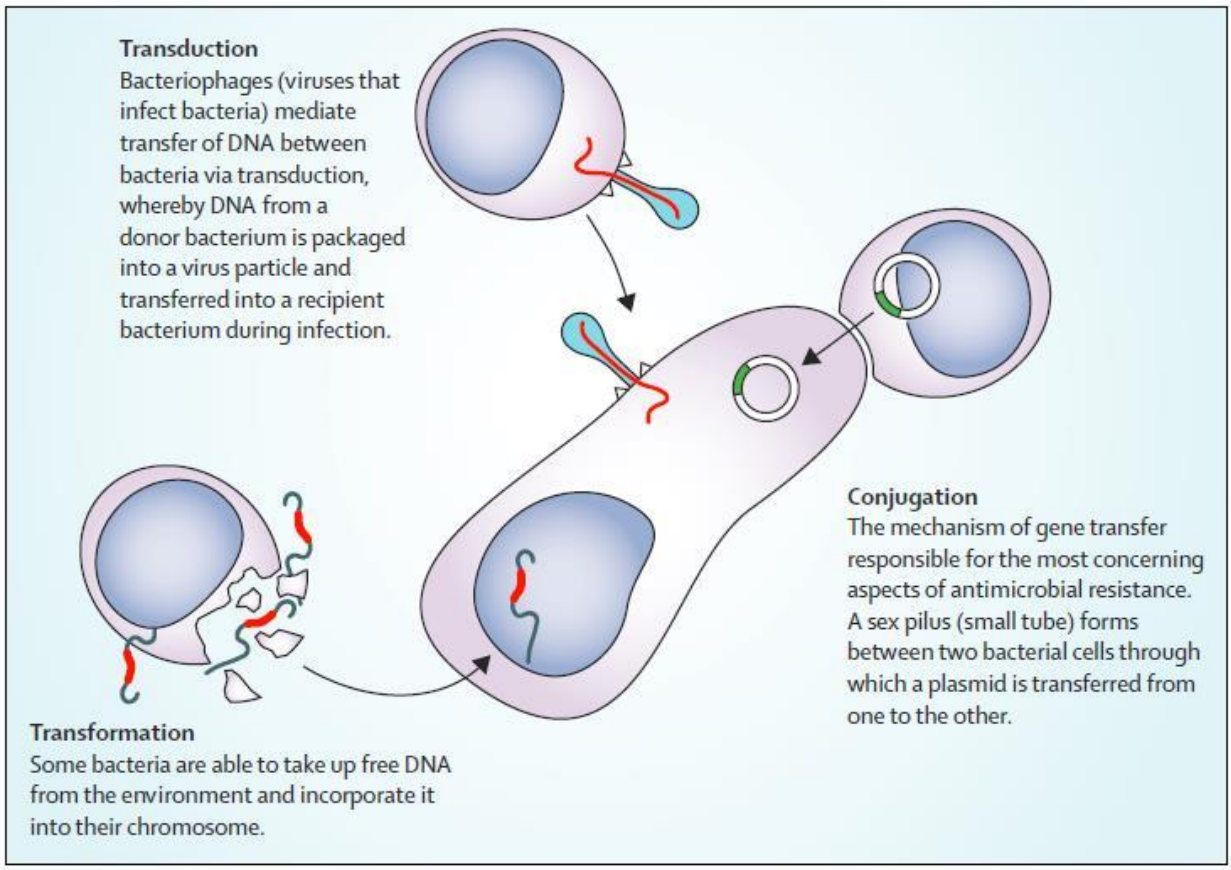
Currently, we have only discussed the surface level of the spread of infections and resistant gene transfer. Different bacteria have different mechanism.

If we have to understand the real-world consequences of these mechanisms, consider these bacteria we encounter most frequently:

Staphylococcus aureus (MRSA): It is located on the skin, nasal passages and surprisingly carried asymptomatically by ~30% of population. Hospitals, gyms, schools and crowded places are common locations. It can be transmitted through direct skin-to-skin contact, contaminated surfaces, respiratory droplets in the air and infected wounds. It develops resistance through the following ways: Target modification (Produces altered PBP2a where penicillin can't bind), β-lactamase production (that destroys Penicillin ring structure), efflux pumps (expels drugs from inside), Biofilm production (a layer that the bacteria produce which prevent the penetration of antibiotics). It causes resistance to all β-lactams, often also resistant macrolides, fluroquinolones, tetracyclines.

Escherichia coli: Located in normal gut flora which are usually harmless and spreads through contaminated food & water. Resistance mechanism: ESBL production (Extended spectrum beta-lactamases destroying Cephalosporins), Efflux pumps (AcrAB-ToIC pump expels Fluroquinolones, Tetracyclines), Porin loss (reduces Carbapenem entry), Plasmid-mediated resistance (Horizontal gene transfer to another species). Its one of the most common causes of treatment failure in UTIs worldwide.

Mycobacterium tuberculosis (TB): Located airborne (respiratory droplets), anywhere within the proximity of an infected individual, High prevalence in overcrowded, poorly ventilated settings. It has no animal reservoir i.e. strictly human to human. The resistance mechanism: Target mutation (mutations in rpoB gene; Rifampicin resistance), Enzyme modification (Catalase-peroxidase mutation; Isoniazid resistance), Efflux pumps (discussed above), Thick waxy cell wall (natural barrier which is hard to penetrate), Intracellular survival (Hides inside macrophages where many antibiotics can't reach). Survival through the macrophages is an interesting method. We all know that macrophages are supposed to kill bacteria but, in this case, the TB uses it as its own safe house. During phagocytosis, the macrophage recognizes specific receptors such as PRRs, TLRs and mannose receptors. So, it engulfs the bacteria in a membrane-bounded compartment called phagosome. Generally, phagosomes are supposed to combined with lysosome to form phagolysosome. But the phagosome remains immature in TB, never fusing with lysosome. That's why TB is so notoriously difficult to treat, being shielded from both the immune system and antibiotics.



Turning the tide: When humans start winning

Infections caused by antibiotic-resistant organisms are difficult to treat or sometimes outright impossible. But it's not a lost cause. Humans have survived through countless deadly microorganisms which could possibly cause mass extinction and will continue to survive new deadly diseases. No one can completely avoid getting an infection, but we can take necessary measures to reduce the risk and prevent the spread.

Due to the mutation or evolution, traditional antibiotics has become inefficient which prompted researchers to find another way to combat these invisible enemies. Some of these methods are:

New Antibiotic development: Cefiderocol (a siderophore cephalosporin that hijacks bacteria's own iron uptake system to enter cell; approved 2019), Lefamulin (first pleuromutilin antibiotic approved to combat community pneumonia; 2019), Omadacycline (Next generational Tetracycline; 2018), Zoliflodacin (target DNA gyrase different from fluroquinolones; prototype). However, this is a temporary

solution as bacteria will continue to mutate and will develop resistance against these drugs too.

Bacteriophage therapy: Perhaps it is one of the most interesting alternatives to antibiotics. Bacteriophages are our greatest ally against these enemies as bacteriophages are viruses that specifically infect and kill bacteria. It was actually used before antibiotics were discovered; however, much research needs to be done to use it to its maximum potential. We have discussed above that bacteriophages can be used by some bacteria to mutate or evolve so,

In the winter of 2015, a 68-year-old American professor named Tom Patterson lay in a coma at UC San Diego, infected with a strain of Acinetobacter baumannii resistant to every antibiotic available — carbapenems, colistin, tigecycline. Everything had failed. His doctors told his family to prepare for the worst.

His wife, Dr. Steffanie Strathdee — an infectious disease epidemiologist — refused to give up. She contacted four research laboratories asking a single question: Do you have a virus that can kill this specific bacterium?

Two labs found phages that matched his strain. The FDA granted emergency authorisation — the first time in American history intravenous phage therapy was approved for a systemic infection. Within days of treatment, Tom began to recover. In August 2016, he walked out of hospital alive — cured by viruses, not drugs.

What made this case scientifically remarkable was an unexpected twist: when the bacteria began developing resistance to the phages midway through treatment, those same resistance mutations restored the bacteria's sensitivity to antibiotics it had previously defeated. Phage pressure had forced the organism into an evolutionary corner. The team combined phages with antibiotics — and the combination finished the infection completely.

obligately lytic phages are the key. They are genetically modified phages selected naturally kill the bacteria rather than integrating. This eliminates the risk of transduction significantly. Furthermore, unlike antibiotics, they evolve alongside bacteria, potentially evading resistance and they do not affect human cells, normal flora or other organisms.

CRISPR-Based Antimicrobials: In simple words, “Using the bacterial immune system against themselves”. CRISPR-Cas systems can be engineered to target specific DNA sequences including antibiotic resistance genes. It is delivered via bacteriophages

or nanoparticles into the resistant bacteria which will cut the resistance genes of the bacterial DNA and thereby re-sensitizing the bacteria to existing antibiotics. However it is largely experimental and the development is significant, showing promise in laboratory and in animal studies.

In 2015, one-year-old girl, Layla Richards was dying at great Ormond Street hospital, London with leukaemia. Every treatment had failed. As a last option , her doctors used CRISPR technology to modify donor immune cells and infused them into her body.

Within weeks, her cancer was gone. She's back to home.

That same molecular precision is now being pointed at antibiotic resistance. Scientists at the broad institute (MIT/Harvard) have engineered CRISPR packages delivered inside bacteriophages directly into drug-resistant bacteria. Once inside, the genetic scissors locate the resistance gene — and cut it out. The bacterium survives. Its resistance does not. It becomes sensitive again to antibiotics it had previously defeated.

Every other solution attack bacterium from the outside. CRISPR goes inside the DNA and edits the problem at its source.

The clinical pipeline is still early — but the proof of concept is real, published, and already building toward human trials.

Vaccines: “Why treat the disease where we can prevent it?”. One of the best ways to combat antibiotic resistance is to prevent the bacteria spread infection altogether. Every bacterial infection prevented is an antibiotic course avoided. Everybody knows about vaccines and in general terms, it teaches our immune system to recognize and destroy a pathogen before it ever causes disease. Examples of such vaccines are: Pneumococcal vaccine has dramatically reduced resistant S. pneumoniae infections in vaccinated populations.

There are also several ways which are in development such as antimicrobial peptides, monoclonal antibodies or Faecal Microbiota Transplant. But with the current development in medical sciences and technologies, humans will continue to discover and invent new methods to combat diseases.

Antibiotic Stewardship: What you can do?

For doctors, improving antibiotic prescribing practices and use is critical to effectively treat infections and protect patient from unnecessary antibiotic use and combat antibiotic resistance. The correct amount of antibiotic is not simply, “the prescribed

dose", it is the dose that achieves the right concentration, at the right site of infection, for the right duration, to kill or inhibit the bacteria without causing toxicity.

It generally depends upon:

Time dependent killing- How long the antibiotic concentration stays above the MIC.

Concentration dependent killing- How high the peak concentration rises above MIC

AUC-dependent killing- Total drug exposure over time.

As a patient, weight, renal function, hepatic function, age, pregnancy, immunosuppression are some of the most important factors that can affect correct dose practice. Site of infection, severity of sepsis and bacterial load are also important to monitor the correct dose.

For general readers, your own responsibility is significant.

Don't demand antibiotics for viral infections. Always consult with the doctors. They might know something that you don't. For general people, you might think common cold = antibiotics. But if it is caused by a viral infection (Rhinovirus which has almost 200 subtypes, COVID-19 which also cause fever). Taking antibiotics for a viral infection has no consequence but will create resistance. Our body has the capability to fight the infections. Dependence on antibiotics for every minor inconvenience will cause further complications.

Complete the full course. Stopping early when you start to feel better is one of the most dangerous habits. Feeling better means only the weakest bacteria are dead but the strongest are alive. Stopping the course will pave the way for the most resistant survivors to mutate and multiply.

Do not self-medicate. Wrong drug, wrong, dose and wrong duration will accelerate resistance. A drug that worked the last time may not work this time.

Never share the antibiotics. Your prescription is tailored for you which is influenced by your weight, your infection and the bacteria inside. Sharing creates problem of self-medication in other people.

Dispose of the antibiotics properly. Simply discarding the antibiotics in environment such as water supply will create low-level exposure for environmental bacteria which is exactly the ideal condition that breeds resistance. Who knows that the resistant bacteria in the sewer might end up on your plate.

Conclusion

We started this article with a contaminated Petri dish.

A forgotten culture plate, a curious scientist, and an accidental observation that changed everything. Within two decades of Fleming's discovery, penicillin had saved millions of lives — soldiers on battlefields, mothers after childbirth, children with meningitis who would otherwise not have survived the week. For the first time in human history, doctors had a reliable answer to bacterial infection.

That answer is now being slowly taken away from us. And the uncomfortable truth is that we are the ones taking it.

Not maliciously. Not carelessly, in most cases. But through accumulated decisions — a prescription written without a culture, a course of antibiotics abandoned halfway, a broad-spectrum drug used where a narrow one would have done the job — each one small, each one reasonable in isolation, and each one contributing to a pressure that bacteria respond to the only way evolution allows them to. They adapt. They survive. They pass the survival instructions to their neighbours.

The clinical picture today is genuinely sobering.

In India, where this matters most to the readers of this article, the ICMR's own surveillance data shows that carbapenem resistance — resistance to the drugs we reach for when everything else has failed — is rising steadily in our tertiary hospitals. Nearly half of all Staphylococcus aureus isolates in Indian hospitals are now methicillin-resistant. NDM-1, the carbapenem-destroying enzyme that carries New Delhi's name, has spread from a single patient in 2007 to healthcare systems on six continents. The IHME projects 1.2 million AMR-related deaths in India by 2030 — not a distant estimate, but a projection built on trends that are already in motion.

In America, the CDC documented over 35,000 deaths from antibiotic-resistant infections in a single year — and that was before the pandemic. During COVID-19, hospital-onset resistant infections rose by 20%, driven by overwhelmed wards, stretched infection control teams, and antibiotics given to patients with viral pneumonia just in case. The just-in-case prescriptions of today are the untreatable infections of tomorrow.

Globally, the WHO's GLASS 2025 report — drawing on 23 million infections from 104 countries — found resistance rising in over 40% of all monitored antibiotic

combinations between 2018 and 2023. The South-East Asian and Eastern Mediterranean regions, which include India, carry the highest burden of all — 1 in 3 infections resistant, compared to 1 in 10 in Europe.

But here is what we want you to hold onto after finishing this article.

The same Lancet analysis that projects 39 million deaths from AMR by 2050 also calculates that 92 million of those deaths are preventable — not with drugs that don't yet exist, but with interventions that are available right now. Better diagnostics. Rational prescribing. Completed antibiotic courses. Stronger surveillance. Infection prevention that actually gets funded and followed.

Tom Patterson walked out of a San Diego hospital in 2016 because one scientist refused to accept that nothing more could be done. Layla Richards is alive because a team of doctors in London was willing to try something that had never been attempted in a human patient. These stories did not happen because the science was ready. They happened because people within medicine decided the situation was too serious to accept passively.

That decision — to take this seriously, to prescribe thoughtfully, to culture before treating, to de-escalate when the results come back — is not a decision that belongs to future researchers or policy makers alone.

It belongs to every doctor in every ward.

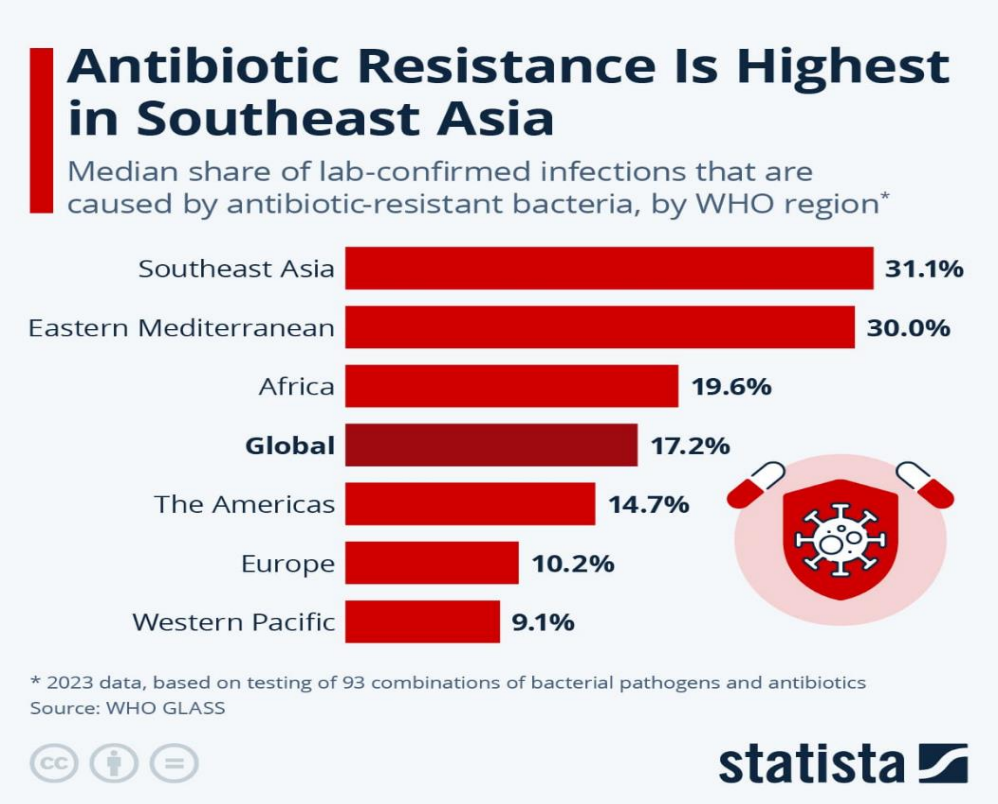
Including the ones reading this article right now, still in medical school, who will write their first antibiotic prescription sooner than they think.

Fleming ended his Nobel lecture in 1945 with a single line that has never stopped being true.

"The microbe is educated to resist penicillin and a host of penicillin-fast organisms is bred out which can be passed to other individuals and perhaps from there to others until they reach someone who gets a septicaemia or a pneumonia which penicillin cannot save."

He was describing 1945. He was describing 2026. The question is whether he will still be describing 2046.

That answer belongs to you.



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