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ANTIMICROBIAL ACTIVITY: DIFFERENT MECHANISTIC ELUCIDATION OF ANTIBACTERIAL, ANTIVIRAL, ANTIFUNGAL & ANTIALGAE AGENTS

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ABSTRACT: In recent days, the increasing number of microbes and their increasing resistance power against conventional drugs have led to enormous worldwide mortalities, hence they pose a great threat to human health. The modern era is already going through the threat of COVID-19, also caused by one of those microbes called the virus. In order to get a clear understanding, all the microbes have been classified in certain types. Nowadays, to develop new alternative antimicrobial medicines, scientists must acquire clarity about the responsible functional groups of different conventional drugs with proper mechanistic elucidation on different types of microbes. This information not only clarifies the functionalities and properties responsible for exhibiting antimicrobial effects, but also facilitates the idea of new drug development through proper functional group incorporation or modification. These modifications increase the efficacy of antimicrobial drugs as well as their activity and water solubility. In this review, my focus will majorly be on the four main types of microbes and their possible mechanistic elucidation of commonly used antibiotics and alternative antimicrobial medicines discovered till now.

INTRODUCTION: The term microorganism is too vast to understand if it is not categorized properly. Basically, the term microbe includes four major types: bacteria, viruses, fungi and algae [1]. Most of the microbes challenge modern medical sciences by creating several diseases [2]. General usage of conventional antibiotics is very common to resist their growth [3]. But these antibiotics are very specific to particular microbes. The most challenging situation is sometimes created by excessive usage of conventional drugs, resulting in resistance against common antibiotics.

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Recently, multidrug-resistant pathogenic microorganisms have created a serious problem in medical sciences [4]. They cannot be destroyed by conventional antibiotics and hence cause several diseases and infections to human health [5]. This phenomenon leads to an urgent requirement to prepare several alternative antimicrobial agents. Hence, scientists must acquire clarity about conventional antimicrobial medicines with proper mechanistic elucidation on different microbes.

This information clarifies the functionalities and properties responsible for exhibiting antimicrobial effects and facilitates the idea of new drug development through proper functional group incorporation or modification. In this essay, my focus will be on the four major types of microbes and the possible mechanistic elucidation of commonly used antibiotics and alternative antimicrobial medicines discovered till now.

Antibacterial Mechanism:

What are Bacteria: Bacteria are also termed as germs [6]. They are organisms, which can only be visible under a microscope. They have a wide variety but depending on their ability to retain primary stain color (crystal violet color) and cell wall structure, they can be classified as Gram-positive and Gram-negative bacteria. They are present inside and outside of the human body and can survive in a wide variety of environments.

Mechanistic Elucidation

Bioactive and Biopassive Mechanism [7]: An active killing involves direct interaction between bacterial cell walls or gene to antibacterial agents. These could be bactericidal *i.e.*, with a hundred percent killing efficiency or bacteriostatic, i.e. the lag phase of the bacterial grown curve is enhanced, but complete killing is not possible. When the effect of bacteriostatic agents removes; the log replication phase of those bacteria become reactivated. On the contrary bio-passive killing means reducing the adhesion of bacteria on the surfaces through electrostatic repulsive interaction but do not necessarily kill the bacteria cells. Generally, negatively charged or zwitterionic species have a capacity of electrostatic repulsion of the negatively charged bacterial cell wall and hence inhibit biofilm formation. Recently, biofilm formation efficacy of some selective pathogenic microbes on the easily colonizable surfaces of the medical device increases the risk further, which can be diminished by introduction of polyethylene oxide (PEO) or polyethylene glycol (PEG) brushes as hydrophilic neutral or zwitterionic polymeric functionality to antibacterial agents through repulsion of negatively charged bacterial cells in a biopassive way reducing the adhesion of bacteria on the surfaces but do not necessarily kill the bacteria cells [8, 9]. Another major capacity of synthetic cationic antibacterial scaffolds to disrupt biofilm matrix was already explored by several groups along with dispersion of the membrane integrity of the pathogenic bacteria. This property added a new dimension to amphiphilic cationic biocides to act as a promising weapon in negatively charged biofilm disintegration in a bioactive way. Hence, recently major attention has been paid on antibacterial agents with coupled bioactive and biopassive killing efficacy.

Conventional Antibiotics: Generally, conventional antibiotics with quinolone functionality like ciprofloxacin kills bacteria by interfering with bacterial DNA replication and transcription through inhibition of DNA gyrase/ topoisomerase II and DNA topoisomerase IV. This eventually leads to the formation of quinolone-enzyme−DNA complexes and thus the generation of reactive oxygen species (ROS) with subsequent cellular death.

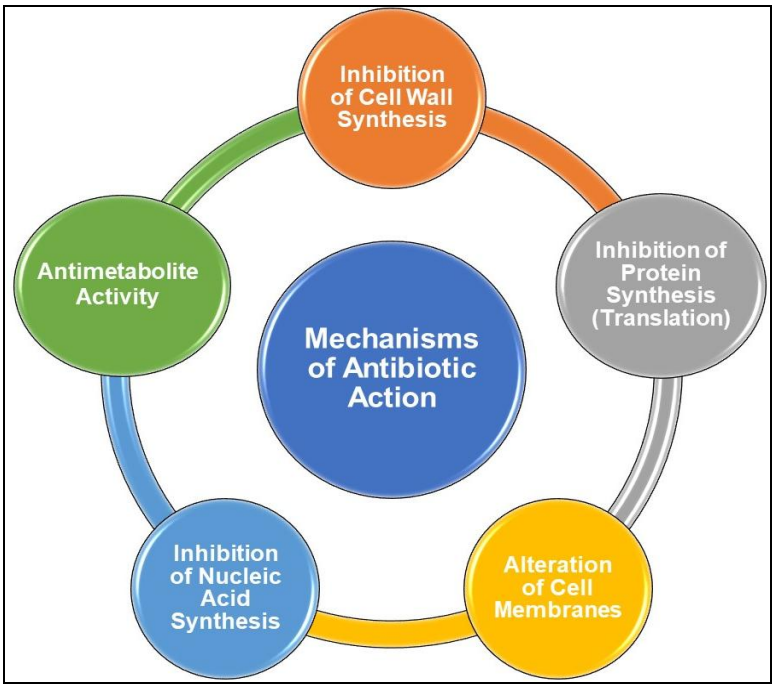

FIG. 1: REPORTED MECHANISM OF ANTIBIOTIC ACTIONS

Antimicrobial Peptides (AMPs): Antimicrobial peptides (AMPs) have recently been considered a promising alternative to conventional antibiotics. Antibiotics preserve bacterial cell morphology, whereas AMPs efficiently show bactericidal properties by physically disrupting the bacterial cytoplasmic membrane instead of targeting mammalian cells. They attack bacterial cell membranes directly, and the disruption is mediated by forming electrostatic interaction between cationic charge of AMPs and anionic charge of phosphate head-groups on the membrane surface which in turn disrupt the membrane by insertion of hydrophobic components into plasma membrane. AMPs selectivity attack microorganism over mammalian cells as zwitterionic phospholipids provide a net neutral charge on the surface of mammalian cells.

For example, several AMPs such as indolicidin, LL-37, human lactoferrin-derived peptide can bind to bacterial endotoxins such as negatively charged hydrophobic lipopolysaccharide (LPS), a major component of outer membrane of Gram-negative bacterial cells [10]. LPS impulsively self-assembles in aqueous solutions leading to aggregation and AMPs, being amphiphilic molecules, are reported to interact with those LPS cumulative accompanied by dissociation resulting an unmanageable and detrimental inflammation.

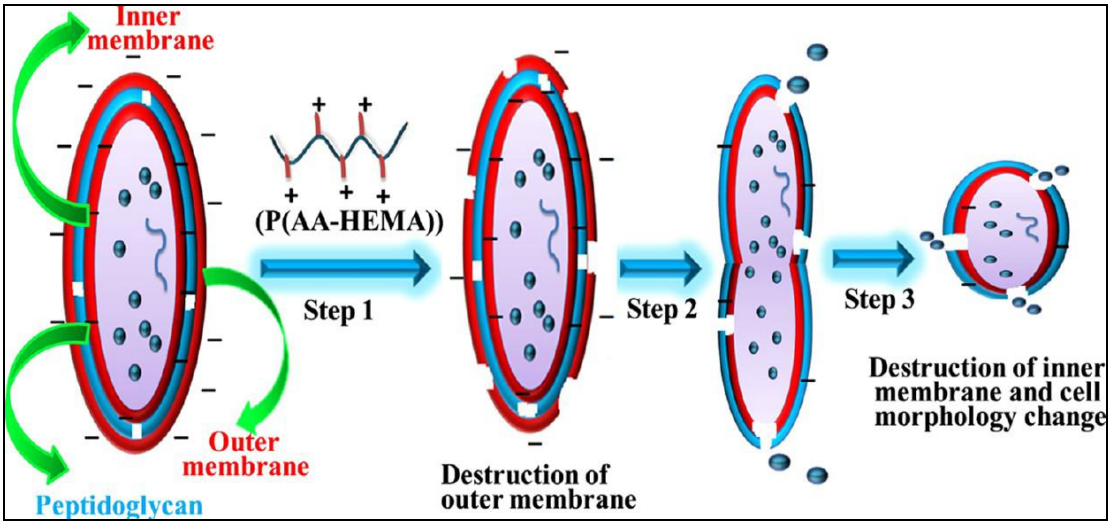

FIG. 2: STEP 1: POSITIVELY CHARGED POLYMER DISRUPTS THE OUTER MEMBRANE OF GRAM-NEGATIVE BACTERIAL CELL WALL THROUGH ELECTROSTATIC INTERACTIONS. STEP 2: POLYMER PENETRATES THE INTERMEDIATE PEPTIDOGLYCAN LAYER AND INTERACTS WITH THE IM THROUGH CLEAVABLE INTERMEDIATE MORPHOLOGICAL VARIATION. STEP 3: TOTAL MORPHOLOGICAL SWITCHING OF BACTERIAL CELL FROM ROD SHAPE TO SPHERICAL SHAPE WITH DESTRUCTION OF INNER CELL MEMBRANE. REPRODUCED WITH PERMISSION FROM REF 7. COPYRIGHT 2017 AMERICAN CHEMICAL SOCIETY

AMP Mimicking Cationic Antibacterial Polymers: An alternative approach to developing new antimicrobial agents utilizing synthetic polymer chemistry has become popular due to the difficulty and cost of large-scale synthesis of AMPs and the rapid degradation of AMPs by protease enzyme inside the human body. Different classes of polymers, such as polyethers, polycarbonates, poly-methacrylates, polynorbornenes, poly-*β*-lactams, *etc*. have been synthesized as AMP mimics [11]. Recently the antimicrobial efficacy of cationic or hydrophobic polymeric substances and their cell penetration have been investigated extensively. While designing antimicrobial polymers, the sufficient cationic charge has to be incorporated into the macromolecule to undergo electrostatic adhesion to the negatively charged microbial cell wall. Further, the introduction of hydrophobic moiety into the polymeric system can lead to disruption of the cellular membrane.

Polymer Antibiotic Conjugate: High costs for new drug development and stringent approval protocols result in fewer trials of new antimicrobial agents per year. An alternative path to shorten the development time for new antimicrobial agents is the formulation and derivatization of existing antibiotics with modified functional groups or

alteration of charge density, solubility, degradability, selectivity, and efficiency. Target specificity of these drugs can be further enhanced by molecular modifications with liposomes, micelles or nanomaterials. Efficient derivatization of antibiotics with polymeric material could be a promising alternative to the new antibiotics class due to the lower toxicity, increased solubility, and prolonged activity originating from polymer antibiotic conjugates (PACs) [12]. Though there are several reports regarding the potential applications of PACs in detoxifying anticancer drugs, such as doxorubicin, in therapeutic antibiotic carriers by using degradable or nondegradable polymer matrices; but little attention has been paid towards exploring permanently bound polymer antibiotic conjugates in the treatment of bacterial infections. Introduction of commonly employed polyethylene glycol to several antibiotics' moieties like Penicillin V, Tobramycin, or Vancomycin can lead to the generation of a series of PACs that could exhibit antimicrobial properties against several microorganisms, including bacteria. Polyacrylates and poly (*N*-isopropyl acrylamide) (PNIPAM) have also been utilized to generate antibacterial PACs. Continuous efforts to enhance the antibacterial efficiency of PACs toward infection-causing bacteria would become mandatory to combat the increasing cases of antidrug resistance in the future world.

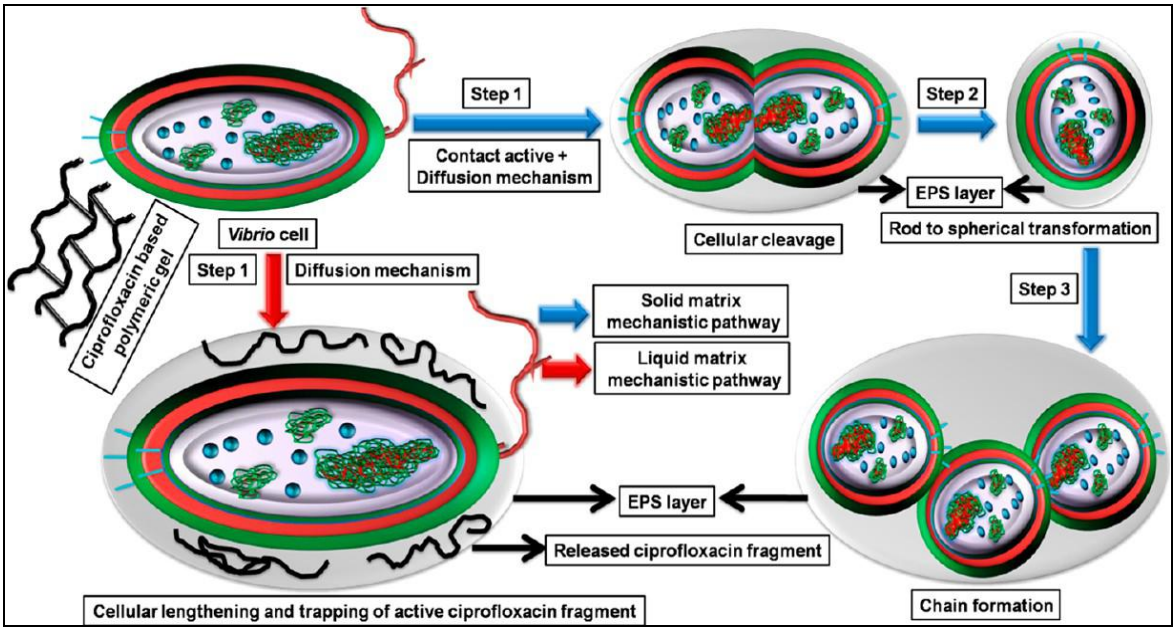

FIG. 3. SCHEMATIC REPRESENTATION OF BACTERICIDAL MECHANISM OF ANTIBIOTIC-BASED GEL (CIPROFLOXACIN-BASED POLYMERIC GEL) AGAINST *VIBRIO CHEMAGURIENSIS* STRAINS IN SOLID AND LIQUID MATRICES. REPRODUCED WITH PERMISSION FROM REF 12. COPYRIGHT 2019 AMERICAN CHEMICAL SOCIETY

Antibacterial Gel: On the other hand, the porous, moist, and robust structure of highly cross-linked three-dimensional (3D) hydrogel matrix may lead to enhanced drug delivery and reduced biofilm formation. The broad-spectrum activity of the gel network originates from its structural integrity. Several hydrogel systems generated from natural or synthetic polymers, including chitosan-iron oxide coated graphene oxide nanocomposite hydrogel, cationic chitosan- or polycarbonate-grafted poly(ethylene glycol) methacrylate-based gels, cationic betaine ester-based gels, ε-poly-L-lysine-based gels, self-assembled gels from amino acids or peptides or through supramolecular interaction were developed with potential biomedical applications especially antibacterial activity. They can kill microorganisms and inhibit biofilm formation through the trap and kill, contact active or release-based pathways. Contact active mechanism is applicable to an amino acid, peptide, or antimicrobial peptides (AMPs) based gels where the drug is not incorporated through a physical or reversible chemical bond. Again, hydrogels that destroy microorganisms by releasing mechanisms and their active released components have also been urbanized by fragmented antibiotics, metal nanoparticles, or AMPs.

Antiviral Mechanism:

What is Virus: Viruses are ultra-microscopic microbes possessing either DNA or RNA as the genetic material responsible for several diseases in flora and fauna. They are structurally composed of a simple protein coat, nucleic acid, viral enzymes and, sometimes, a lipid envelope. They utilize the cellular equipment of the host cell for the replication process, hence are termed as intracellular pathogens. Such features generate difficulties in antiviral drug development with selective toxicity.

Mechanistic Elucidation: The fight between human body system and viruses is a tedious procedure, as both will implement and strategize differently to fight against each other [13]. Antiviral drugs development is a difficult process as mentioned earlier. This method involves many stages such as

- target identification and screening
- lead generation and optimization
- clinical studies and the drug registration

The recent development of antiviral drugs is an urgent requirement, as viral infections have caused millions of mortalities throughout human civilization. Unlike most antibiotics, antiviral drugs could not terminate the actions of their targeted pathogens; rather restrict their development. As the viruses utilize the host's cells for replication, finding a process for designing a safe and effective antiviral drug without damaging the host's cells, is the most complicated task.

Major complications caused by the genetic variation of virus while developing antiviral drugs and vaccines. The same challenge is facing the modern era while developing COVID-19 vaccine. As a result, very few antiviral drugs are recognized globally till now.

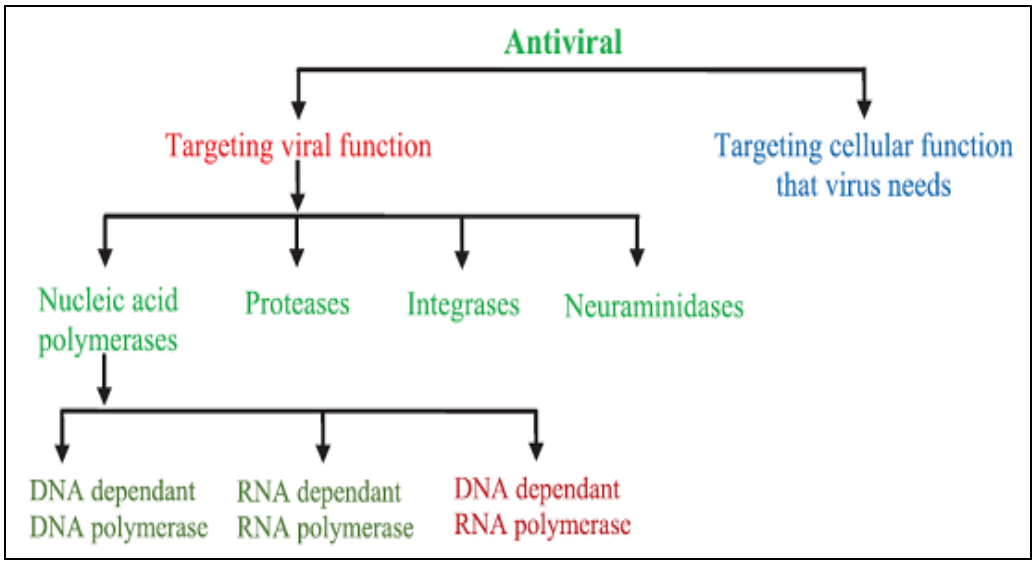

FIG. 4: COMMON INHIBITORY ACTIONS OF ANTIVIRAL DRUGS. REPRODUCED WITH PERMISSION FROM REF 13. COPYRIGHT 2021 PUBMED (SAGE JOURNAL)

Table 1. Mechanism of action of antiviral drugs used for the treatment of COVID-19.

Group	Drugs	Mechanism of action
Viral RNA polymerase inhibitors	Remdesivir (GS-5734)	RdRp inhibitor, prodrug, analogue of adenosine nucleotide
	Favipiravir	RdRp inhibitor, prodrug, analogue of guanosine nucleotide
Viral protein synthesis inhibitors	Ritonavir/Lopinavir	Inhibitor of protease
Inhibitors of viral entry	Hydroxychloroquine Chloroquine	Increase in endosomal pH needed for the virus/cell fusion. Interfere with cellular receptor glycosilation of SARS CoV (ACE-2)
Immunomodulators	Nitazoxanide	Interfere with host regulated pathways of virus replication, amplification of type I IFN pathways and cytoplasmic RNA sensing
	Ivermectin	Inhibition of importin I heterodimer to inhibit the nuclear import of host and viral proteins

FIG. 5: REPRODUCED WITH PERMISSION FROM REF 13. COPYRIGHT 2021 PUBMED (SAGE JOURNAL)

Antifungal Mechanism:

What are Fungi: Fungi has a complicated structure compared to virus. Fungal cell membranes are composed of unique sterol, ergosterol, which replaces cholesterol found in mammalian cell membranes.

Mechanistic Elucidation: An antifungal agent is a drug that selectively disintegrates fungal pathogens from a host cell with negligible toxicity to the host-body system [14, 15].

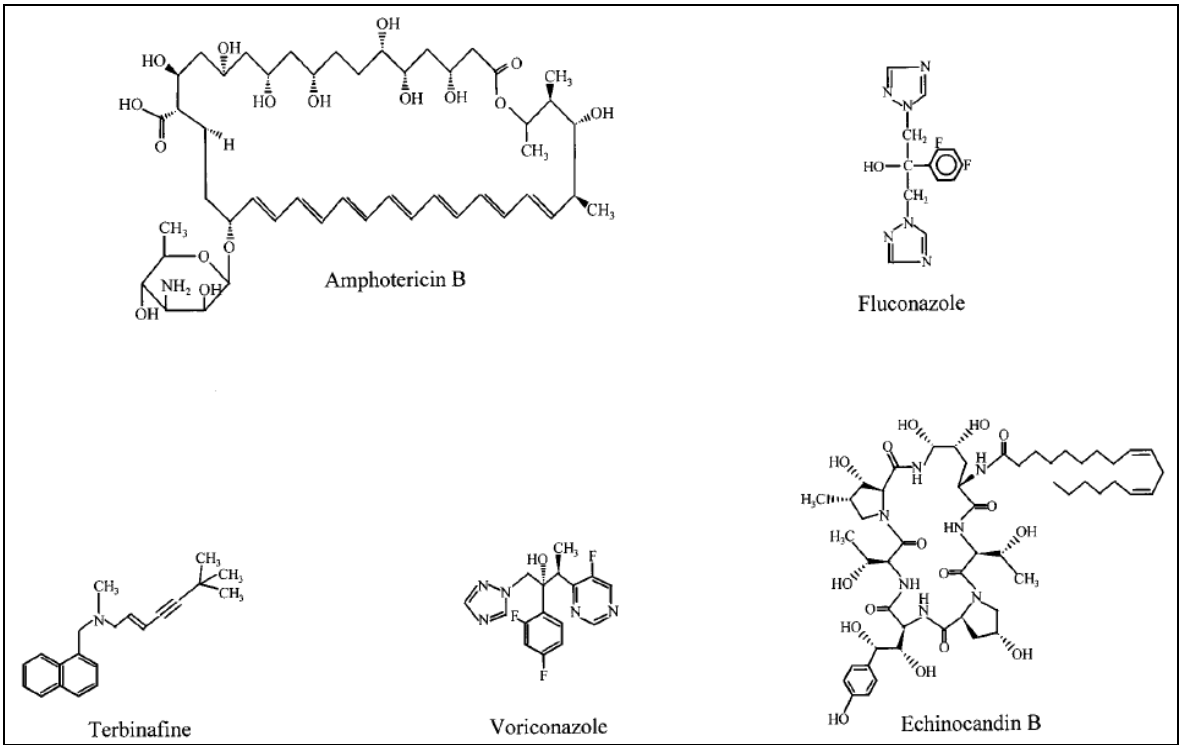

FIG. 6: STRUCTURES OF REPRESENTATIVE ANTIFUNGAL AGENTS. REPRODUCED WITH PERMISSION FROM REF 15. COPYRIGHT 1999 PUBMED

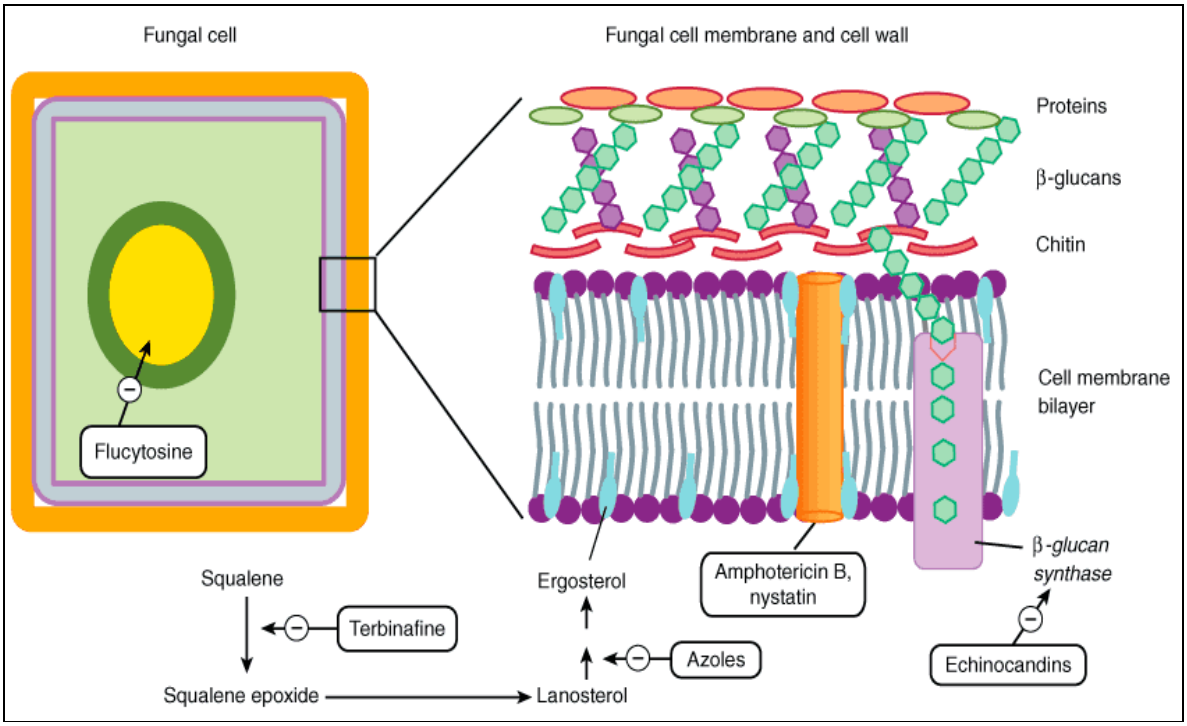

FIG. 7: MECHANISTIC ELUCIDATION OF ANTIFUNGAL AGENTS (SCHEMATIC PRESENTATION)

TABLE 1: MODE OF ACTION AND MECHANISM OF RESISTANCE FOR COMMON ANTI-FUNGAL AGENTS (REPRODUCED WITH PERMISSION FROM REF. 16. COPYRIGHT 2014 ELSEVIER)

Antifungals	Mode of action	Mechanism of resistance
Polyenes Amphotericin B, Nystatin	Fungicidal	Reduction of erogoterol concentration ablating drug-target binding. Alteration in POL gene family
Azoles Fluconazole, Itraconazole, Ketaconazole, Posaconazole, Voriconazole, Miconazole	Fungistatic	Efflux of drug by multi-drug transporters ABC gene family. Amino acid substitution to Ergl IP affecting drug-target binding. Over expression of Ergl Ip minimizing effect of drug. Change in toxic-sterol concentration due to multation in Erg3 LLELES
Echinocandins Caspofungin, MIcafungin, Anidulafungin	Fungi static or fungicidal	Mutation in Fks I and Fks 2 binding units
Allylamines Terbinafine, Naftifine	fungstatic	Interference from multidrug transporters
Pyrimidine analoges 5-Flucytosine	Fungicidal	Mutation in cytosine permease and deaminase

TABLE 2: PREDICTED MECHANISM OF COMMON ANTIFUNGAL DRUGS [15]

Types of antifungal antibiotics	Properties	Predicted mechanism
A. Polyene antibiotics Amphotericin B B. Nystatin	• possess a macrocyclic ring • one side of which has a several conjugated double bonds and is highly lipophilic • the other side is hydrophilic with many OH groups. • In some cases, a polar amino sugar and a carboxylic acid group at one end. • Soluble in water and unstable in aqueous medium. • Amphotericin B is a naturally occurring polyene macrolide antibiotic produced by Streptomyces nodosus. • Nystatin is obtained from noursei and is similar to Amphotericin B in antifungal action and other properties.	• Amphotericin B have high affinity for ergosterol present in fungal cell membrane. • It then gets combined with the membrane and inserted into the membrane, and several molecules orient themselves in such a way as to form the micropore. • The hydrophilic side forms the interior of the pore through which ions, amino acids and other substances leave. • The cell permeability is markedly increased and due to this the cell death occurs. • It binds to fungal cell membrane (Ergosterol) and forms pores. • This alters permeability & transport and as a result, cell death occurs.
Antimetabolite A. Flucytosine Azoles	• It is a pyrimidine antimetabolite and inactive as such	• They inhibit C-14 α-demethylase (a cytochrome P450 [CYP450] enzyme), thereby blocking the demethylation of lanosterol to ergosterol, the principal sterol of fungal membranes. • This inhibition disrupts membrane structure and function, which then inhibits fungal cell growth
Heterocyclic benzofuran A. Griseofulvin	• The azole antifungal drugs act by inhibiting the synthesis of the sterol components of the fungal membrane. • Azoles are predominantly fungistatic.	• It interferes with mitosis-multinucleated and stunned fungal hyphae result from its action. • It causes abnormal metaphase configuration, however doesn't cause metaphase arrest, rather the daughter nuclei fail to move apart or move only a short distance. • It does not inhibit polymerization of tubulin but somehow disorients the microtubules.
Echinocandins	• It was one of the early antibiotics extracted from *Penicillium griseofulvum*. • It is active against most dermatophytes but not against Candida and other fungi causing mycosis. • It is fungistatic in nature	• Echinocandins interfere with the synthesis of the fungal cell wall. • It inhibits 1,3- beta glucan synthase, an enzyme important in fungal cell wall synthesis and subsequently inhibit the synthesis of beta glucan in the fungal cell wall. • Disruption of the fungal cell wall leads to cellular osmotic instability and cell death.
Topical antifungals A. Ciclopirox	• It is Glucan synthesis inhibitor.	• Ciclopirox is active against *Trichophyton rubrum, Trichophyton mentagrophytes*, and

B. Tolnaftate		*Malassezia furfur.* • *Tolnaftate* is active against *Epidermophyton*, *Microsporum*, and *Malassezia furfur*
Terbinafine	• Ciclopirox inhibits the transport of essential elements in the fungal cell, disrupting the synthesis of DNA, RNA and protein. • Tolnaftate distorts the hyphae and stunts mycelia growth in susceptible fungi. It is synthetic allylamine and orally active.	• It interferes with ergosterol biosynthesis by inhibiting the fungal enzyme squalene epoxidase rather than interacting with the P450 system. • Acting as a structural analogue of squalene, terbinafine causes the accumulation of this unsaturated hydrocarbon, and a decrease in ergosterol in the fungal cell membrane. • The accumulation of toxic amounts of squalene result in the death of the fungal cell

Antialgae Mechanism:

What are Algae: Algae is a well-known group of microorganisms exhibiting the ability to produce their own food through the process of photosynthesis and creates a huge problem in aquaculture, marine and paint industry through extensive biofilm formation via exopolysaccharide (EPS) generation through the transcription of specific genes at the second step of biofouling, which is one of the distinct features of biofilm formation. Therefore, new antimicrobial agents resisting algae growth inhibiting the action of those specific genes responsible for EPS formation are urgent needs.

Mechanistic Elucidation: Prominent algacidal activity of biogenic compounds *i.e.,* the active metabolites produced by macro- and micro-algae or cyanobacteria (blue-green algae) belonging to the groups of fatty acids, lipopeptides, amides, alkaloids, terpenoids, lactones, pyrroles and steroid is already well established by effective prevention of biofouling. Again, alkaloid-type natural products extracted from plant materials and cyclic hydroxamic acids derived from 1,4-benzoxazin- 3-one structures can also exhibit a high potential to antialgal activity. But these substances have several drawbacks like the difficulty of synthesis, purification and lack of water solubility for natural products and severe nonspecific toxicity problem for chemical substances which led to the generation of new water-soluble, biocompatible algaecides bearing antialgal functionalities, which can be easily synthesized commercially.

Phenolic compounds or peptide structures play a significant role in antialgal activity, though the actual mechanism of biofouling prevention by those functionalities is not well understood yet. But from these reports, the H-bonding of phenolic –OH group can be predicted as a significant criterion to exhibit antialgal activity by restricting the biofilm formation of algae.

The fouling process starts from the moment of quick accumulation of dissolved organic matter and molecules such as polysaccharides and protein fragments. Single-cell diatoms then sense the surface and start settling on it, forming a microbial film. After initial attachment, the cells begin to grow and start forming colonies.

During this period, major changes are noticed through the rapid formation of the EPS layer. The phenolic –OH or amide group may extensively bind to the EPS containing a larger number of hydroxyl group through H-bonding, destroying the EPS layer; hence biofilm formation and excessive growth of algae is prohibited.

CONCLUSION: In conclusion, antimicrobial activity is a broader terminology as it includes a wide range of microbes like bacteria, viruses, fungi, or algae. They have different cell wall compositions and cell ingredients. Hence the modern utilization of commercially available antimicrobial agents and conventional drugs are very selective and provide different mechanistic elucidations. This article mainly portrayed a brief overview and categorization of antimicrobial agents based on my research experiences and ongoing scientific research. The main focus of the current review was to create awareness of different antimicrobial mechanisms as this information is widely required to combat multidrug-resistant microbes or newly evolved viruses with rapid gene mutation capacity like COVID-19. This field has a very high probability of future evolution as many researchers are working on developing novel antimicrobial agents.

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