



Modes of Action of Some Recently and Previously Discovered and Used Antimicrobial Agents/Drugs and Molecules: An Overview

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Received date: July 11, 2017; Accepted date: July 29, 2017; Published date: August 03, 2017

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Abstract

The need of controlling infectious microbes has increased in the past years due to the increased resistance of the microorganisms towards the antimicrobial agents as a result of the changes in their cellular membrane proteins, ionic channels, and cell surface receptors. Scientists are working their best to offer such an agent that could prove practical to harmful microbes that pose threats to the lives of people. This review focuses on the information about the modes of actions of some antimicrobial agents that are recently discovered, approved or used by scientific laboratories and pharmaceutical companies to make the future studies somewhat easier for the young scientists to make their way through their research works leading to newer drug discoveries with new and improved mechanisms against microbial integrity. Many new drugs have recently been reported to have performed their best against multi-drug resistant *Staphylococcus aureus* and *Pseudomonas*, apart from bacterial control several new antifungal and antiviral agents have also been reported that are known to have performed best at clinical levels.

Keywords: Antimicrobials; Antibacterials; Antiviral; Antifungals; Antimicrobial drugs; Antibiotics

Introduction

Infectious diseases have led to the jeopardy of humanity in some ways by posing threats to their health and even to their valuable lives. Scientists have become busy in digging out the genomic databases and already available microbial libraries to find out the drugs that could lead to overcome the causes of the various fatal diseases to give humanity with the best of their efforts, such drugs that could help them to gain their health again after the attack of some dangerous pathogenic microbes. Many pathogenic microorganisms have been thoroughly studied to bring out a possible cure that could prevent colonization of such microbes in the human body and save the suffering patients from the health hazards. In order to discover new drugs scientists got indulged in finding out and discovering the drugs that could help in overcoming the fatal diseases by destroying the pathogenic microbes. Of all the microorganisms that are known, the major ones that pose threat to humanity are bacteria, fungi and viruses and their control have gained much attention by the scientists of today. Discovery of many drugs in the recent years to overcome various strains of all three types of microorganisms, viruses, bacteria, and

fungi was carried out. Pathogenic microorganisms of today that are known to cause various fatal pathogenic infections are becoming resistant to the already available antimicrobial agents [1]. Discussed here are the mechanisms of action of some of the recently discovered antibacterial, antifungal and antiviral agents/drugs/molecules among which some are available for use in humans and some are under clinical investigations after which they could be made available to use in humans to save the lives of billions of people in those countries that are susceptible to deadliest pathogenic microbe attacks due to the favoring environment of their ecological conditions to such microbes and can only be controlled by the use of such valuable drugs discussed below.

Newly Discovered Antibacterial, Antifungal and Antiviral Drugs

Antibacterial agents/antibiotics

Nowadays, the major problem related to microbial infections is their increasing resistance to some commonly used antibiotics [1]. Microorganisms that were previously controlled by some antibiotics have now become resistant to those drugs by certain changes in their cellular structures and molecules they produce. Antibiotics are the

most widely used drugs that are mainly used for eradicating colonization of infectious microbes, like bacteria. For treatment of pneumonia, tuberculosis, meningitis, and certain surgical infections, antibiotics have become vital role players to cure patients whose immune system gets compromised [2].

They may sometimes prove harmful to the humans due to their uncertain and unlikely damaging side effects; therefore, they have must be taken only on the prescription of a certified medical practitioner. As bacteria belong to the Kingdom Prokarya, so, the difference between prokaryote and eukaryote is extensively exploited to discover the drugs that effect only on the prokaryotic machinery and do not negatively influence the eukaryote.

There are different classes of antibiotics; each class of antibiotics has different modes of action that specifically effect prokaryotic biosynthetic machinery to limit or to kill the bacteria. The figure given below shows different classes of antibiotics that target specific mechanisms (Figure 1).

The prescription of antibiotics must be written keeping in view the following major points that are ultimately related to the human health or wellbeing:

- Posology;
- Side effects;
- Potential of causing resistance in microbes;
- Minimum inhibitory concentration;
- Price of drug;
- Affectivity against disease.

It has been found that inappropriate use of antibiotics leads the modern world to the edge where disease time is increasing, the therapeutic rate is low, and the onset of the disease is rapid, also including increased retention time of patient in the hospital and low immunity against microbes. This is an alarming situation which invokes many pharmaceutical companies to seek an alternate way to cope up with the increasing resistance in bacteria, due to which many guidelines as suggested by the international agencies including FDA and others some of them are given below:

- Limited use of broad spectrum antibiotics;
- Promoting use of narrow spectrum antibiotics;
- Use of alternative ways to control infections instead of antibiotics.

Keeping in view, these points, a new era of research is progressing that focuses on the development of drugs, strategies to limit microbial resistance in various ways, most common are mentioned in Figure 1.

Some of the recently discovered antibacterial drugs include the following: Auranofoin, Polymyxin, Teixobactin, Ceftolozane/tazobactam, Ceftazidime avibactam, Eravacycline.

Cephalosporins: Cefaroline, Ceftobiprole, Glycopeptides.

Newer carbapenems: Doripenem, Imipenem/MK-7655, Brilacidin, MX-2401 (lipopeptide antibiotic), OP0595 (a new diazabicyclooctane), Quinolones, Macrocyclic antibiotics, Pleuromutilin, Glycocyline, Ketolides, Lipopeptides, Iciaprim, Oxazolidinone.

Antifungal agents/drugs

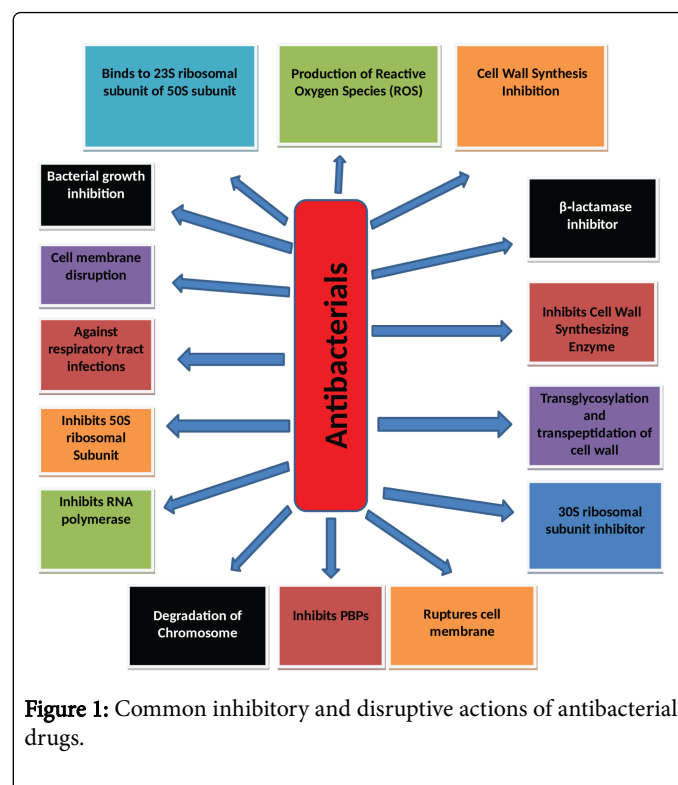
Fungi are eukaryotes, so, antifungal agents are those drugs that exploit the differences between these two eukaryotic cells and hence do

not cause harm to recipients. Antifungal drugs have 3 major targets that limit the growth of fungi, these include:

- Inhibition of cell wall synthesis;
- Cell membrane disruption;
- Inhibition of cell division.

Antifungal agents or the fungal attack-countering drugs play an important role in overcoming damaging effects of fungal diseases, as shown in Figure 2 given below.

Recently used antifungal agents are as follows: 7-hydroxy-6-nitro-2H1-benzopyran-2-one (Cou-NO₂-a coumarin derivative).

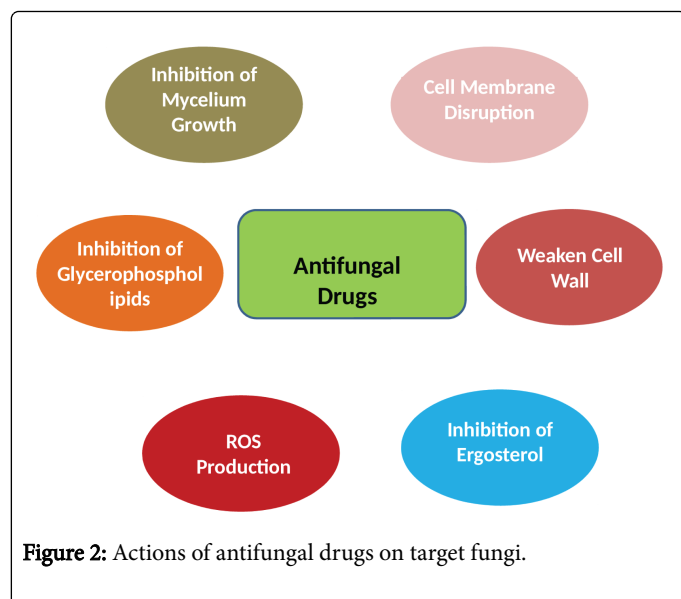


Azoles

- Eflinaconazole (approved in 2014);
- Terconazole;
- Posaconazole;
- Voriconazole;
- Imidazole;
- Isavuconazonium (a triazole-approved in 2015).

Echinocandins

- Caspofungin;
- Anidulafungin;
- Micafungin;
- Allylamines, Thiocarbamates and Morpholines;
- Amphotericin B (a Polyene);
- Fungal cell wall targeting peptides/Molecules;
- Fungal Glycerophospholipids targeting drugs;
- Target to membrane by ROS generation.



Antiviral agents

Antiviral drugs are less in number in comparison to the antibiotics, there are many reasons for it, and some of them are given below:

- These are toxic for humans;
- Needs more potency;
- A few viral diseases are lethal.

Antiviral agents are towards halting or interfering with the viral replication pathways and also overcome viral attacks by interfering with other virus-infected cell metabolic pathways. Some common newly discovered antiviral agents include:

- Nitazoxanide;
- Amiodarone;
- Clomiphene;
- Daclatasvir;
- Peramivir;
- Tenofovir alafenamide;
- Zinc oxide tetrapods;
- Brincidofovir;
- Valomaciclovir (H2G);
- N-methanocarbothymidine;
- Inhibitors of helicase-primase complex;
- Entry inhibitors;
- CD4-gp 120 binding drug.

Modes of Action of Recently Discovered Antibacterial Drugs

Auranofin

In drug-resistant strains of *Staphylococcus aureus* auranofin shows an inhibition of multiple biosynthetic pathways, where this drug at its sub-inhibitory concentration inhibits cell wall and DNA synthesis and at minimal inhibitory concentration it inhibits protein synthesis by down regulating 20% of essential proteins of *S. aureus* [3].

Polymyxin

The major target site polymyxin in gram-negative bacteria is the lipopolysaccharides present in the cell membrane, it kills bacteria by causing lysis of the outer membrane of the bacterial cell [4]. By the disruption of the anchoring lipopolysaccharide A in the outer membrane layer, the outer membrane gets weakened which disrupts the whole cell membrane structure [5], and after entering the cell, it causes the rapid production of reactive oxygen species that are toxic to the cell [4].

Teixobactin

In *S. aureus*, *Bacillus anthracis* and *Clostridium difficile* it causes strong blocking of the peptidoglycan (cell wall) biosynthesis by the accumulation of cell wall solubilizing precursor named as undecaprenyl-N-acetylmuramic acid-pentapeptide *in vitro* and also *in vivo*, also shows efficacy against *Streptococcus pneumoniae* in mice [6].

Ceftolozane/tazobactam (cephalosporin and β -lactamase inhibitor combination)

Combination of cephalosporin ceftolozane with a beta-lactamase inhibitor (tazobactam) in a 2:1 ratio in *Pseudomonas aeruginosa* shows cellular inhibition activities. Ceftolozane causes inhibition of cell wall synthesis by binding to penicillin binding proteins and tazobactam inhibits the β -lactamase enzyme synthesis that is known to provide resistance to penicillin [7].

Ceftazidime/avibactam

Combined antibiotics, ceftazidime-avibactam when used in a fixed ratio of 4 μ g/ml, show good antibacterial activities. Ceftazidime inhibits the cell wall synthesizing enzymes in bacteria. Avibactam inhibits β -lactamases [8]. Avibactam inhibits class A, D and class C β -lactamases in various bacteria [9].

Eravacycline (TP-434)-a fluorocycline

Causes inhibition of protein synthesis in various gram positive and gram negative bacterial cells by binding to the 30S bacterial ribosomal subunit [10]. It has also shown to destroy the biofilms formed by uropathogenic *E.coli* strain *in vitro* [11].

Cephalosporins

Ceftaroline: It has the activity of inhibiting cell wall synthesis by binding to penicillin binding proteins in methicillin-resistant *Staphylococcus aureus* (MRSA), this drug has high affinity for binding to the penicillin binding proteins usually the mecA proteins that aids in the methicillin-resistance of *S. aureus* [12]. Promotes transpeptidase and transglycosidase reactions in the cell walls of Enterobacteriaceae by binding to membrane penicillin binding proteins [13]. It is a pro-drug that is converted to active form in blood by the action of enzymes.

Ceftobiprole

It has strong affinity for penicillin-binding protein 2a (PBP2a) which is present in Methicillin-resistant *Staphylococcus Aureus* (MRSA) and *S. pneumoniae* [14].

Glycopeptides

This class of drug act by trans-glycosylation and trans-peptidation reaction of peptidoglycan biosynthesis. They have additional action on disruption of membrane hence increasing cell permeability and causing rapid bactericidal activity depending on the actual member of drug used [15]. Examples include Oritavancin, Dalbavancin, and Telavancin.

Newer Carbapenems

Doripenem

This drug binds to penicillin binding proteins and thus inhibits cross linking of the peptidoglycan structure, they have high affinity for PBP-2 and PBP-3. They are highly effective against *Pseudomonas aeruginosa*, *E. coli*, and *Klebsiella* species [16].

Imipenem/MK-7655

Combination of carbapenem Imipenem with MK-7655 inhibits bacterial cell wall synthesis by inhibiting β -lactamase enzyme; it is effective *in vitro* against *Pseudomonas aeruginosa* and many other bacteria including carbapenemase-resistant strains [17].

Brilacidin (PMX-30063)

Actively ruptures the bacterial cell membrane structures in gram-negative enteric bacteria [17].

Plazomicin (ACHN-490)

It is a next generation antibacterial drug made by Achaogen Company, it is commonly known as the neoglycoside and shows potential activity against many gram-negative bacteria [17]. It is resistant to enzyme-based inhibition of antibiotics caused by bacterial enzymes and acts by inhibiting protein synthesis in bacteria [18].

MX-2401 (lipopeptidic antibiotic)

When bound to the substrate undecaprenylphosphate it causes the inhibition of lipid I and lipid II which are the precursors of bacterial cell wall biosynthesis and also inhibits biosynthesis of lipid III which is the precursor of teichoic acid [19].

OP0595 (a new diazabicyclooctane)

Acts as β -lactamase inhibitor and enhancer of the β -lactam activity, shows antibiotic activity against *Enterobacteriaceae* by inhibiting penicillin-binding proteins (PBP2a) and also causes the bacteria to attain a spherical morphology thus inhibiting them [20].

Quinolones

Convert bacterial topoisomerases IV and gyrase enzymes into chromosome degrading enzymes which cause the fragmentation of their own chromosome [21].

Macrocyclic antibiotics

Fidaxomicin is a drug that has narrow spectrum of activity, if is effective for *Clostridium difficile* [22]. In contrast to treating the

Clostridium difficile with broad spectrum antibiotic, use of fidaxomicin is very effective in multiple ways, which includes:

- Low effect on intestinal flora;
- Low dose comparing broad spectrum;
- Low chances of reoccurrence of disease;
- Comparable treatment regime.

Mode of action of this antibiotic is that it affects bacterial enzyme that make RNA called RNA polymerase [23].

Pleuromutilin

It is a novel antibiotic and is first member of this class; it is used topically for skin and soft tissue infections which are resistant to most antibiotics previously available. However it is ineffective against gram-negative organism [24]. Its mode of action is inhibition of protein synthesis by acting on 50S ribosomal subunit of bacteria [25].

Glycocycline

This class is chemical derivative of aminocycline. It has been designed to overcome two major mechanisms of tetracycline resistance mediated by acquired efflux pump and by ribosomal protection. It acts by binding to 30S subunit of ribosome.

Ketolides

This new class is designed particularly for treatment of respiratory tract infection that has acquired macrolides resistance [25], e.g., Telithromycin.

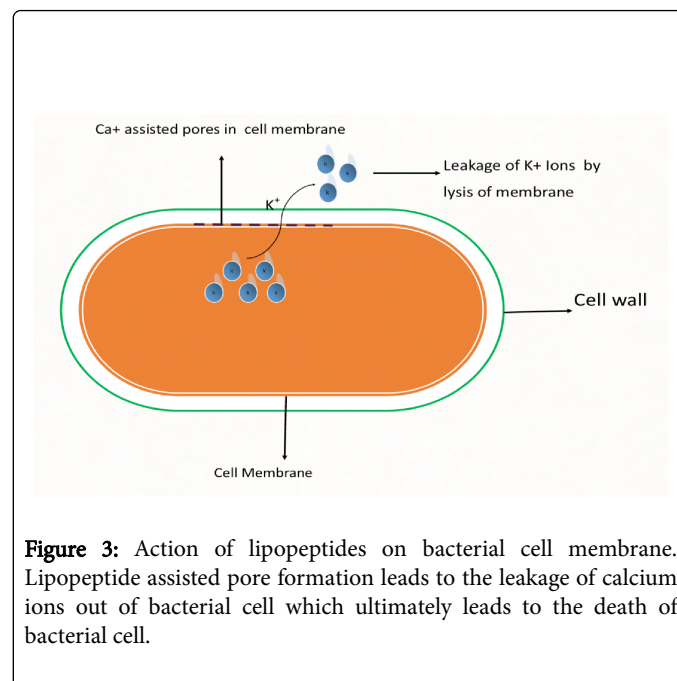


Figure 3: Action of lipopeptides on bacterial cell membrane. Lipopeptide assisted pore formation leads to the leakage of calcium ions out of bacterial cell which ultimately leads to the death of bacterial cell.

Lipopeptides

Its mode of action is insertion of lipophilic tail in to cell membrane of gram-positive bacteria without entering in to bacterial cytoplasm, the channels are formed from which intracellular potassium is lost

(Figure 3) disrupting bacterial cell membrane potential and hence death of bacteria [26], e.g., Daptomycin.

Iciaprim

Dihydrofolate reductase is an enzyme that is required by the bacteria for their active growth. There are few classes of drugs that inhibit this enzyme; due to which bacterial growth becomes limited [27].

Oxazolidinones

This new class of antibiotic acts by preventing the initiation of translation of protein by binding to 23S portion of 50S ribosomal subunit [28], e.g., Torezolid and Radezolid.

All the discussion above was the recent review of newer antibiotics, their modes of action and their benefits over traditional antibiotics. As research is an ongoing process, so there are a lot of fields which can be targeted for the improvement in antibiotics, some of them are given below:

- Target to bacterial proteins (e.g. Platensimycin [29]);
- Target to virulence factors for rapid clearance [30];
- Modulation of host response pathways [31];
- Therapeutic use of bacteriophage;
- Combination of antibiotics [32];
- Combination of antibiotic and bio enhancers [33].

Modes of Action of Recently Discovered Antifungal Drugs:

7-Hydroxy-6-nitro-2H1-benzopyran-2-one (Cou-NO₂-a coumarin derivative)

Inhibits the growth of mycelium and conidia in the species of *Aspergillus* and also inhibits cell wall synthesis and enhances the activity of azoles when used in combination [34].

Azoles

All azoles whether newly or previously discovered have the same mode of action in fungal cells. They interfere with the ergosterol biosynthesis by inhibiting the activity of 14 α -lanosterol demethylase which is involved in the conversion of lanosterol to ergosterol [35].

This ergosterol inhibition results in the depletion of the cellular membrane ergosterol component and also interferes with the fungal cell growth and cell division [36]. e.g., Eflinaconazole (approved in 2014), Terconazole, Posaconazole, Voriconazole, Imidazole, Isavuconazonium (a triazole-approved in 2015).

Echinocandins

They target the cell wall components of fungi and inhibit β -1, 3 glucan synthase enzyme which helps in the synthesis of β -glucan, an important fungal cell wall component, this results in the stress on fungal cell wall and thus it becomes weak towards certain osmotic pressures [36], e.g., Caspofungin, Anidulafungin, and Micafungin.

Allylamines, Thiocarbamates and Morpholines

Allylamines (e.g., terbinafine) and thiocarbamates (e.g., tolnaftate) inhibit biosynthesis of ergosterol through inhibition of ERG1 gene and morpholines (e.g., fenpropimorph and amorolfine) inhibit ergosterol biosynthesis by inhibiting ERG2 and ERG24 genes [35].

Amphotericin B (a Polyene)

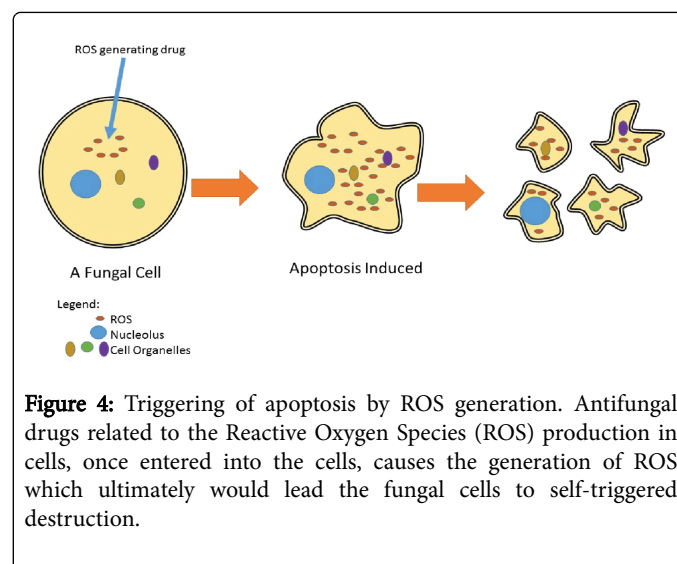
Bind to ergosterols and disrupts fungal cell membrane by making pores in the cell membrane which causes the loss of cell membrane morphology, ionic balance and leads to the cell death [37].

Fungal cell wall targeting peptides/molecules

There must be some distinguishing feature in fungal cell so that only fungal cell wall is destroyed, it is mannoproteins. Mannoproteins are a diverse group of proteins that have structural function, cell adhesion proteins, enzymes involved in cell wall synthesis. Any molecule that is capable of targeting these mannoproteins, indirectly targets fungal cell integrity. Such molecules include plant defensins, like NaD1 [38], the histatins, which are histidine rich salivary polypeptides [39] are found to be damaging to cell wall of fungus.

Fungal glycerophospholipids targeting drugs

Glycerophospholipids (GPL) is a class of phospholipids associated with cell membrane; GPL varies with mammalian and fungal cell wall. Due to differences in content of GPLs, fungal membranes are more negative and mammalian membranes are neutral electrostatically. This difference can well be exploited. Electroactive compounds like cationic Antimicrobial Peptides (AMPs) and many Antifungal Proteins (AFPs) are found to be disruptive to cellular membrane, causing leakage of ions and other molecules [40]. Exact mechanism of action of creation of pores varies by different studies [41-43].



Target to membrane by ROS generation

Reactive Oxygen Species (ROS) are lethal for cell in multiple pathways that includes triggering of apoptosis (Figure 4) [44], or oxidation of membrane lipids [45,46]. The molecular entities that

generate ROS are of great importance for killing fungal cells, one of such molecule includes tyrocidines [47], and plant defensin PvD1 [48].

Modes of Action of Recently Discovered Antiviral Drugs

Nitazoxanide

In influenza viruses it induces the post-transcriptional blockage of viral hemagglutinin maturation [49].

It also causes host fibroblast cells to initiate α and β interferon production in peripheral blood mononuclear cells stimulated by influenza viruses [50]. All these processes lead to the promotion of antiviral activity of this drug [51].

Amiodarone

It is an anti-arrhythmic drug [52]. This drug is known to target and block calcium, sodium and potassium channels and α and β -adrenergic receptors of host cells and thus blocks Ebola virus entry into the healthy cells [53].

Clomiphene

A cholesterol transporter named as Neimann-Pick C1 acts as an intracellular receptor molecule. Recently an ovarian stimulant drug called clomiphene has been reported to inhibit the infection caused by Ebola virus by altering the function of Neimann-Pick C1 involved in Ebola virus entry into the host [54].

Daclatasvir (Daklinza)

It alters the replication of HCV by down-regulating phosphorylation of NS5A proteins (NS5A proteins are key role players in HCV replication enhancement) [55]. It also has activity of blocking viral RNA production and secretion of virions [56].

Peramivir (BCX-1812, RWJ-270201)

This drug has such a structural chemistry which helps it to bind with more affinity to neuraminidases [57].

Binding of this drug to neuraminidases inhibits influenza A and Influenza B viruses from coming out of the infected cells and entering into the healthy cells thus acting as the neuraminidase inhibitor [58].

Tenofovir alafenamide

This drug is a pro-drug that enters the HIV infected cells and causes the release of an active metabolite called Tenofovir Diphosphate; this metabolite causes the active inhibition of HIV reverse transcriptase enzyme thus halting HIV replication process [59].

Zinc oxide tetrapods

These are the micro or nano-level crystalline structures made up of Zinc Oxide and are moulded to specified shape (tetrapods) using special techniques [60].

In an experiment, it has been found that this zinc tetrapod's exhibit antiviral activity by reducing spread of viral infection and decreasing the production of viral proteins that are necessary for secondary

infection [61]. This experiment has been performed on coreal culture infected with herpes simplex virus 1 that causes keratitis [62].

Brincidofovir

Brincidofovir is structurally an acyclic nucleoside phosphonate having oral bioavailability [63].

After absorption and metabolism it yields high level of cidofovir diphosphate in cell due to which enhanced antiviral activity has been seen against variety of double stranded DNA viruses [64].

Valomaciclovir (H2G)

Valomaciclovir is acyclic guanosine analog. It can be easily metabolized to monophosphate, diphosphate and triphosphate derivatives in the cells infected with viruses especially, as its concentration is found to be very low in non-infected cells.

Its antiviral activity is due to the formation of high concentration of relatively stable H2G triphosphate that it is potent inhibitor of viral DNA polymerase [65].

N-methanocarbathymidine

It is a nucleoside analog that targets viral DNA polymerase. It has antiviral activity against variety of viruses including Epstein Barr Virus (EBV), Herpes Simplex Virus (HSV) and orthopoxvirus etc. [66].

Inhibitors of helicase-primase complex

Most of the antiviral agents described above act by blocking viral DNA polymerase, however a new strategy has been tested to target the helicase-primase complex which is composed of heteromeric protein subunit complex that includes helicase, primase enzymes and ancillary proteins [67], e.g., Pritelivir [68] and Amenamevir [69].

Entry inhibitors

This class of antivirals represents new generation for treatment of viral infections especially Human Immunodeficiency Virus (HIV).

These agents are of importance as resistance of viruses against transcriptase and protease inhibitors is currently rising.

CD4-gp 120 binding drug

The first step of virus entry in cell includes binding of HIV envelope glycoprotein120 (gp120) to CD4 (T-cell) on the target cell surface [70]. Many molecules have been found having different structures and different mechanisms of action that initiate CD4-gp120 binding, hence the entry of virus is avoided in the cell.

These agents includes PRO-524, TNX-355, BMS-806 that act by mimicking CD4 receptor by its tetravalent soluble recombinant protein structure, competing HIV gp120 for CD4 receptor with monoclonal antibody, binding to gp120 and hence block its conformational change after CD4 binding respectively [38,71-73].

A table given below shows some of the other antivirals arranged categorically that are under clinical trials among which, some of the antivirals have been approved for use while others are still under ongoing trials for their safety (Table 1).

Antivirals	Against	Mode of Action	References
Clevudine (Thymidine nucleoside analogue)	Hepatitis-B Virus (HBV) and Epstein-Barr Virus (EBV)	Inhibitor of DNA dependent DNA polymerase activity of HBV	[74]
MIV-210 (Dideoxyguanosine nucleoside analogue)	HIV and HBV	Inhibitor of DNA dependent DNA polymerase	[74,75]
CMX157 (Prodrug of tenofovir)	HIV and HBV	Inhibits viral DNA polymerase activity	[74]
Heteroaryldihydropyrimidines (includes Bay 41-4109 and GLS4)	HBV	Viral replication inhibition and interferes with the proper viral protein coat formation	[74,76,77]
REP 9 AC (amphipathic DNA polymer)	HBV, HCV, Herpes simplex virus, choriomeningitis virus	Prevents the release of Hepatitis B surface Antigen from the hepatocytes	[74,78]
ARC-520 (RNA interference based antiviral-an siRNA)	HBV	Suppression of HBV RNA and DNA (by intervening at the site of DNA transcription)	[74]
Letermovir	Cytomegalovirus Infection	Cuts DNA at various sites and thus these cut fragments cannot be easily packed into the capsid and thus viral assembly and replication halts	[79]
Fluoxetine	Enterovirus D68	Uncertain	[80]

Table 1: Shows some of the approved and some under trial antivirals.

Conclusion

All the antibacterial, antifungal and antiviral agents discussed above, the major ones are those that are used to control the multi-drug resistant bacteria (*Staphylococcus aureus*), pathogenic fungi and enveloped and complex viruses like Influenza viruses, Herpes Simplex Virus (HSV), HIV, and phages. The need to discuss the above mentioned recently discovered and used antimicrobial agents is to give the young scientists with the best of the newer strategies when working with such antimicrobial agents to consider to the use of these agents with their mechanism of actions near at hand by the help of this effort. The threat to the human lives has increased in the past decade due to the increased microbial mutations which lead to certainly unimaginable changes in their cellular morphology which attracted the scientists of today to gather at one table and get involved in overcoming such hurdles in the microbial control. The major microbes that are a threat to humans are bacteria and viruses that claim the lives of billions of people every year worldwide. Viruses being the rapidly dividing particles and with higher mutation rates are somewhat difficult to control after infection but still, their replication could be halted by the use of some above-mentioned drugs. Bacteria with resistance to certain drugs have now been characterized and their control strategies have gained much attention from the pharmaceutical companies and scientists. Several drugs mentioned above have proved their actions against multi-drug resistant *Staphylococcus aureus* and others like *Pseudomonas aeruginosa* etc. Among fungi, the most concerned are the ones that cause systemic infections and for such fungi, the most beneficial antifungal agents are the ones that inhibit the enzymes involved in cell wall formation and those that aid disruption of cell wall components. With these studies underway, the need to discover newer and improved compounds could be urged in the minds of future scientists to make some effort to make noble discoveries in the near future that could be helpful against all the three types of microbes, bacteria, viruses and fungi (potentially, a 3 in 1 drug).

Acknowledgement

We hereby thank our Professor Dr. Romana Tabassum for assigning us the title for the paper writing. We are also thankful to our research institute “National Institute for Biotechnology and Genetic Engineering (NIBGE)” for providing us the opportunity to get guidance from such enthusiastic and supportive Professors. Although there are no funding agencies involved in supporting our work, this paper is the collective effort of all the authors.

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