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Review article

A review on basics of pharmaceutical antiviral drug

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ABSTRACT

Eleven drugs approved by the food and drug administration for the treatment of viral infections (other than those caused by human immunodeficiency virus type-1 {HIV-1} or those complicating such infection) will be reviewed in this article. Antiviral drug design could, in principle, be targated at either viral proteins or cellular proteins. The first approach is likely to yield more specific, less toxic compound, with ta narrow spectrum of antiviral activity and a higher likelihood of virus drug-resistance development.challenges.

Keywords: Antiviral agent, antiviral drug MOA (mechanism of action), Strategies in the design of antiviral drugs.

INTRODUCTION

The definition of antiviral drug are used to treat infections caused by viruses or the antiviral are medication that help your body fight off certain viruses that can cause disease are known as antiviral agent. The viruses are spread rapidly and are well –adapted to changing environmental events. they can infect the human body readily and trigger fatal disease, Recent highlight in the development of new antiviral drugs – Twenty antiviral drugs, that is about half of those that are currently approved, are formally licensed for clinical use in the treatment of human immunodeficiency virus infections (acquired immune deficiency syndrome). The others are used in the treatment of herpesvirus (e.g. herpes simplex virus. varicella zoster virus and cytomegalo virus) hepatitis B virus, hepatitis C virus or influenza virus infections. Recent endeavour have focused on the development of improved antiviral therapies for virus infections that have already proved amenable to antiviral drug treatment, as well as for virus infections for which at present, no antiviral drugs have been approved [1].

Infectious diseases are well known since ancient time to human civillisation. infectious disease are caused due to different microorganism (bacteria, viruses and fungi) viral structure is simple and consists of a protein coat, nucleic acid, viral enzymes and, sometimes, a lipid envelop, unlike the complex structure of fungi, helminthes and protozoa. Additionally, viruses use the host's cellular machinery for replication, hence obligate intracellular pathoges. The current scenario all over the world indicates that continuous emergence of microbial threat at an accelerating pace, mainly due to unprecedented climate change and globalization. Antiviral drugs are a class of medicines particularly used for the treatment of viral infections. Specific antiviral drugs are a class of medicines particularly used for the treatment of viral infections.

DNA Virus

Viruses such as poxviruses, herpes, adenoviruses and papiloma viruses usually contain double-stranded DNA, leaving single-digit DNA,leaving single-digit DNA virus enters the cell centre and leads to new viruses

RNA Virus

RNA viruses include influenza, measles, mumps, colds,p olio, retrovirurses, arena viruses, all considered, single descriptor RNA. viral RNA virus does not enter the cell center.viral RNA is then used to make a DNA copy of the viral RNA, which is organized by the host genome followed by a retroviruses [2].

Steps of viral infections: - the sex steps of viral replication include viral attachment, invasion, uncoating, replication, and release. The steps of virus life cycle highlighting the entry and exit of the virus are described below:-

The virus attaches to a host cell injecting its genetic material into the host cell during attachment and penetration stage.

In the next step, the viral DNA or RNA is itself incorporated into the genetic material of the host cell inducing it to replicate the viral genome. This step involves the uncoating, replication and assembly during the virus life cycle. During release, the host cell releases the newly created viruses, either

through the breakage of the cell,waiting cell death or by budding off through off through the cell membrane.

Figure 1: Women's well-being device.

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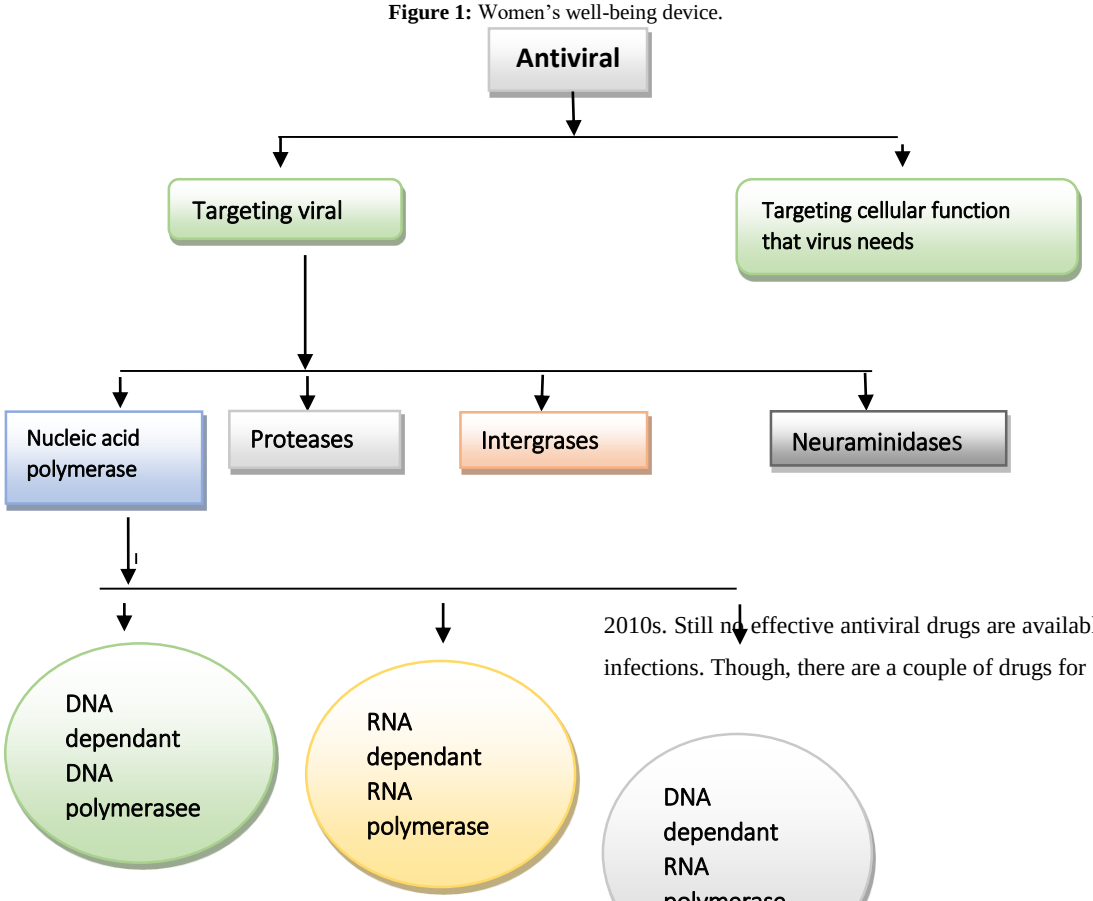


Classification of Antiviral drugs
Anti-herpes virus drugs
e.g. Idoxuridine, acyclovir
Anti-influenza virus
e.g. Amantadine, rimantadine
Anti-Hepatitis virus drug
e.g.Lamivudine,rabavirin
Nucleoside reverse transcriptase inhibitors
e.g. zidovudine, lamivudine
Non-nucleoside reverse transcriptase inhibitors
e.g. Delaviridine
Protease inhibitors
e.g. Ritonavir

The mechanism of action of antiviral drugs

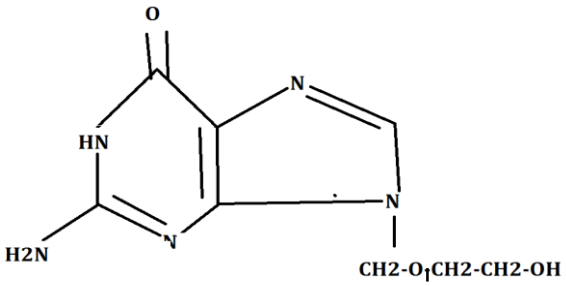
Antiviral drugs are a class of medicines particularly used for the treatment of viral infections. Drugs that combat viral infections are called antiviral drugs. Viruses are among the major pathogenic agents that cause number of serious diseases in humans, animals and plants. Viruses cause many diseases in humans, from self-resolving diseases to acute fatal diseases. Developing strategies for the antiviral drugs are focused on two different approaches. Targeting the viruses themselves or the host cell factors. Antiviral drugs that directly target the viruses include the inhibitors of virus attachment, inhibitors of the virus entry, uncoating inhibitors, polymerase inhibitors, protease inhibitors, inhibitors of nucleoside and nucleotide reverse transcriptase and the inhibitors, protease inhibitors of integrate. The inhibitors of protease (ritonavir, atazanavir and darunavir) viral DNA polymerase (acyclovir, tenofovir, valganciclovir and valacyclovir) and of integrate (rategravir) are listed among the Top200 Drugs by sales during 2010s. Still no effective antiviral drugs are available for many viral infections. Though, there are a couple of drugs for

Herpes viruses, many for influenza and some new antiviral drugs for treating hepatitis C infection and HIV. Action mechanism of antiviral drugs consists of its transformation to triphosphate following the viral DNA synthesis inhibition. An analysis of the action mechanism of know antiviral drugs concluded that they can increase the cell's resistance to a virus (interferons), suppress the virus adsorption in the cell or its diffusion into the cell and its deproteinisation process in the cell (amantadine) along with antimetabolites that causes the inhibition of nucleic acids synthesis. This review will address currently used antiviral drugs, mechanism of action and antiviral agents reported against COVID-19 [3].

Acyclovir Drug

It is a synthetic analogue of the purine nucleoside, guanosine. It is prodrug

Structure



Acyclovir is a synthetic guano sine analogue used for treating herpes simplex virus (HSV) and varicella zoster virus (VZV) infections. Intravenous (IV) acyclovir provides excellent tissue and fluid penetration, including the cerebrospinal fluid, whereas oral acyclovir provides modest bioavailability of 15% to 30%. Bioavailability is improved with the use of Val acyclovir, the valyl ester formulation of acyclovir. Acyclovir is excreted by glomerular filtration and tubular secretion.

Herpes viruses have varying degrees of susceptibility to acyclovir, with HSV type 1 (HSV-1) being most susceptible, followed by HSV type 2 (HSV-2) and VZV, and to a lesser extent Epstein-Barr virus (EBV). High acyclovir concentrations may also inhibit CMV in vitro, but acyclovir is not recommended clinically for CMV treatment, acyclovir is not active against human herpes virus (HHV) 6, 7 AND 8.

Acyclovir is approved for the treatment of primary and recurrent genital HSV infection (table 1). Topical acyclovir may be used to treat genital herpes, but the oral formulation is generally recommended, IV acyclovir is used for severe cases. Suppressive therapy with oral acyclovir is also indicated to reduce the incidence of recurrent genital herpes.

Oral acyclovir is modestly efficacious against or labial herpes. In immunocompetent individuals, or labial herpes is often self-limited, and antiviral treatment is generally not recommended. However, oral acyclovir may be indicated for severe cases, for those with recurrent or labial herpes, and in those who are immunocompromised [4].

Intravenous acyclovir is the first-line treatment for HSV encephalitis and should be started as soon as the disease is suspected clinically. Magnetic resonance imaging of the brain typically demonstrates temporal lobe involvement, and diagnosis is confirmed by detection of HSV DNA in the CSF. Major studies have evaluate the efficacy of 10 days of acyclovir treatment for HSV encephalitis; however, the recommended duration of treatment in the clinical setting is 2 to 3 weeks because shorter durations have been associated with relapse. The treatment duration may be further prolonged in immunocompromised patients.

Acyclovir is also approved by the US food and Drug administration (FDA) for the treatment of VZV, however, young immunocompetent patients with zoster may not require treatment if the lesions are localized and have been present for more than 72 hours. Intravenous acyclovir is recommended for patients with disseminated zoster disease or visceral involvement. Acyclovir treatment of zoster reduces duration of viral shedding, formation of new lesions, and short- and long term neuralgia. Therapy should be started early, but even delayed initiation of acyclovir may still be beneficial in immunocompromised patients. Short-course prednisone may be added as an adjunct to acyclovir treatment of zoster to improve quality of life, especially in elderly patients.

DNA polymerase, thereby hating viral DNA synthesis. The major clinical indication for cidofovir is the treatment of CMV retinitis in HIV-infected patients (Table) cidofovir is also used as rescue therapy for immunocompromised patients with CMV disease resistant or unresponsive to Ganciclovir. Because activation of cidofovir does not rely on viral kinases, it retains activity against CMV with the UL97 mutation and HSV with the TK mutation. Resistance to cidofovir occurs when the virus develops mutation in the DNA polymerase gene (i.e, *CMV-UL54 gene Mutation*). Cidofovir has also been used off-label for various illness, such as acyclovir-resistant HSV disease, condylar acuminate, BK virus-associated hemorrhagic cystitis, JC virus-associated progressive multifocal leukoencephalopathy and other infections due to double stranded DNA viruses.

Advances in Antiviral drug design

The purpose of the series on advances in antiviral drug design is to regularly review the “state of the art” on emerging new developments in the antiviral drug research field, thereby spanning the conceptual design and chemical synthesis of new antiviral compounds, their structure-activity relationship, mechanism and target of action, pharmacological behavior, antiviral activity spectrum, and therapeutic potential for clinical use. Volume 2 begins with a description of the antiviral potential of antisense oligonucleotides by J.Temsamani and S.Agrawal. According to the aims of the antisense technology, these oligonucleotides should be targeted at specific viral antisense technology, these oligonucleotides should be targeted at specific viral mRNA sequences so that translation to the virus specified proteins is blocked [5].

Closing the door on flavivirus: entry as a target for antiviral drug design

With the emergence and rapid spread of west Nile virus in the United States since 1999, and the 50-100 million infections per year caused by dengue virus globally, the treat of flavivirus as re-emerging human pathogens has become a reality. To support the effort that are currently being pursued to develop effective vaccines against these viruses, researchers are also actively pursuing the development of small molecule compounds that target viruses, researchers are also actively pursuing the development of small molecule compounds that target various aspects of the virus life cycle. Recent advances in the structural characterization of the flavivirus have provided a strong foundation towards these efforts. These studies have provided the pseudo atomic structures of visions from several members of the genus as well as atomic resolution structure of several viral proteins. Mostly importantly, these studies have highlight specific structural rearrangement that occur within the vision that are necessary for the virus to complete its life cycle. These arrangements occur when the virus must transition from immature, to mature, to fusion-active states and rely heavily on the conformational flexibility of the envelop protein that forms the outer

glycoprotein shell of the virus. Analysis of these conformational changes can suggest promising target6s for structure based antiviral design for instance, by targeting the flexibility of the E protein, it might be possible to inhibit required arrangement of this presents a structural perspective of the flavivirus life cycle and focuses on the role of the E protein as an opportune target for structure-based antiviral drug design [6].

Rational design of polymerase inhibitors as antiviral drug

Almost all viruses have polymerase which are suitable target for antiviral drugs. The development of selective polymerase inhibitors started with screening of compound in virus-infected cell cultures and the mechanism of action was investigated once and inhibitor had been found. Especially nucleoside analogs were screened as their triphosphates were potential substrates for polymerase. However, the stepwise phosphorylation by cellular, and sometimes viral, kinases to the active triphosphates prevented a truly rational design of polymerases inhibitors. Nucleotide analogs offers a type of compounds which could be designed in a more rational way than nucleoside analogs since the first, most selective, phosphorylation step is eliminated in the path to the active inhibitors. The development of pyrophosphate analogs made rational design possible since these compound act directly on the viral enzyme, but the room for structural variation was limited.

An overview of functional nanoparticles as novel emerging antiviral therapeutic agents.

Research on highly effective antiviral drugs is essential for preventing the spread of infections and reducing losses. Recently, many functional nanoparticles have been shown to possess remarkable antiviral ability, such as quantum dots, gold and silver nanoparticles, Nan clusters, carbon dots, grapheme oxide, silicon materials polymers and dendrites. Despite their difference in antiviral mechanism and inhibition efficacy, these functional nanoparticles-based structures have unique features as potential antiviral candidate. In the topical review, we highlight the antiviral efficacy and mechanism of these nanoparticles. Specifically, we introduce various methods for analyzing the veridical activity of functional nanoparticle and the latest advances in antiviral functional nanoparticles [7].

Figure 1: Suggested Antiviral Drugs for the Treatment of Herpes virus infections

virus	Clinical disease	Drug name (route)	Recommended dosage	comments
Herpes simplex viruses 1 and 2	Mucocutaneous disease	Acyclovir(IV)	5mg/kg IV every *h	IV therapy is preferred for severe and disseminated disease
		Acyclovir (oral)	400 mg orally 3 times daily 800 mg orally twice daily 200 mg orally 5 times daily	Localized disease and genital herpes
		Valacyclovir (oral)	1g orally twice daily 500orally twice daily	First episode of genital herpes
Varicella zoster virus	Varicella zoster	Acyclovir (IV)	10mg-kg IV every 8	Risk of crystalline nephropathy
		Acyclovir (Oral)	600-800 mg orally 5 times daily 1g orally every 6h 1g orally 3 times every 12h	Less preferred than Val acyclovir because of poor bioavailability
		Valacyclovir (Oral)	1g orally 3 times daily	Preferred orally therapy for mild or localized disease
CMV	CMV disease in transplant recepients	Ganciclovir (IV)	5mg /kg IV every 12 h	IV therapy is preferred for severe CMV disease ,gastrointestinal disease, pneumonia, and encephalitis transition to oral val Ganciclovir on clinical and virological improvement Duration of therapy is guided by CMV surveillance using PCR to pp65 antigenemia

CONCLUSION

Antiviral are medications that help your body fight off certain viruses that can cause disease. Antiviral drugs are also preventive. They can protect you from getting viral infections or spreading a virus to others. An analysis of the mechanism of action of existing and used viral drugs permits the conclusion to be made that they can increase resistance of the cell or its diffusion into the cell, and the process of its "deproteinization "in the cell.

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