WORLD JOURNAL OF PHARMACEUTICAL RESEARCH
Coden USA: WJPRAP Impact Factor 8.63
Volume 15, Issue 12, 361-377. Review Article ISSN 2277–7105

A BRIEF REVIEW ON DRUGS USED IN TREATMENT OF HIV INFECTION (AIDS)

Abhijit P. Borkar*, Anushree A. Dhole, Shreya A. Dhurandhar, Khemu G. Shedame, Dr. Manisha D. Kitukale

Department of Pharmacology P. Wadhwani College of Pharmacy, Yavatmal.

Article Received on 14 May 2026,
Article Revised on 04 June 2026,
Article Published on 16 June 2026,

https://doi.org/10.5281/zenodo.20695879

***Corresponding Author**

Abhijit P. Borkar

Department of Pharmacology P. Wadhwani College of Pharmacy, Yavatmal.

How to cite this Article: Abhijit P. Borkar*, Anushree A. Dhole, Shreya A. Dhurandhar, Khemu G. Shedame, Dr. Manisha D. Kitukale. (2026). A brief review on drugs used in treatment of HIV infection (AIDS). World Journal of Pharmaceutical Research, 15(12), 361–377.

This work is licensed under Creative Commons Attribution 4.0 International license.

ABSTRACT

Human Immunodeficiency Virus (HIV) continues to pose a crucial challenge for public health across the globe. Although ART therapy has been successful in turning HIV into a treatable chronic condition, current three-drug combinations often lead to side effects, pill burden, and potential drug-drug interactions. Therefore, current scientific studies tend to focus on developing efficient two-drug therapies. The aim of this systematic review of literature is to analyze the efficacy, safety profile, pharmacology, and prospects for Idvynso use (doravirine 100 mg + islatravir 0.25 mg in one tablet) for HIV-1 treatment in adults. This document describes the general epidemiology of HIV and pathogenetic processes associated with HIV infection, as well as analyzes current classification systems used for antiretroviral drugs. Furthermore, the study discusses results of pivotal Phase 3 trials (Trial 051, Trial 052). Pharmacological assessment demonstrates that doravirine operates as a new generation NNRTI that does not produce central nervous system side effects and circumvents mutations experienced historically by the class. Islatravir is a unique NRTTI characterized by its prolonged intracellular half-life and dual activity as both a translocation inhibitor and a delayed chain terminator. Pharmacological findings demonstrate that a reduced calibrated dose of islatravir (0.25 mg) successfully achieves non-inferior virologic suppression when compared to triple-drug combinations (e.g., Biktarvy) and counteracts the historical concerns associated with lymphocyte depletion. Even with its limited metabolism, occasional hypersensitivity, and specific CYP3A drug interactions, Idvynso offers a safe, highly

effective, and extremely valuable tenofovir-free, one-pill treatment solution for the aging population affected by HIV.

KEYWORDS: HIV 1, Antiretroviral Therapy [ART], Idvynso, Doravirine, Islatravir, Two-Drug Regimen, Non-inferiority Clinical Trials (Trial 051/ Trial 052), Nucleoside Reverse Transcriptase, Translocation Inhibitor (NRTTI), Nucleoside Reverse Transcriptase Translocation Inhibitor (NRTTI), Translocation Inhibition, Delayed Chain Termination, Fixed-Dose Combination (FDC), INSTI-Sparing Regimen, Tenofovir-Free Treatment.

INTRODUCTION

- **Brief Information About HIV Disease**

The Human Immunodeficiency Virus (HIV) is one of the biggest public health challenges in modern medicine. Since it was first identified in the early 1980s, HIV has led to millions of deaths around the globe. It also requires lifelong medical care for those who are infected.[1,2] The HIV mainly attacks the human body's immune system and infects certain types of immune cells such as CD4+ T lymphocytes, macrophages, and dendritic cells. With the gradual decrease in these very important immune cells, there is an overall decrease in immunity levels, thereby making the person vulnerable to diseases like AIDS.[3,4]

Over the years, there have been some dramatic changes in the landscape of HIV infections. It used to be a death sentence for the infected individuals; however, today it is a chronic illness, all because of the discovery of ART.[5,6]

Even with all the successes in therapies, the search for perfect treatment protocols is still ongoing. Three drugs treatment regimes, although very successful, have side effects including high drug burden, toxicity over time, and potential drug interactions.[7]

As such, there is an ongoing shift towards a two-drug combination which achieves viral suppression but reduces the overall exposure to drugs. It is within this framework that Idvynso (a combination of doravirine and islatravir) stands out as a novel treatment option.[8,9]

- **Global Epidemiology and Public Health Strain**

While the scaling of anti-retroviral therapies for treating HIV has been successful in managing the horizontal and vertical transmission mechanisms in the past, the current

epidemiology of HIV infection around the globe continues to be an epitome of the stark socio-economic, geographical and demographic divides that exist among different populations. As per the recently developed dynamic models by UNAIDS, there are approximately 39.9 million people who suffer from HIV infection worldwide, and sub-Saharan Africa carries most of the burden associated with this epidemic.[33] The continual prevalence of newly acquired cases within at- risk populations around the world demonstrates a failure on several fronts, including PrEP implementation, structural barriers to good health at the local level, and stigma within society that stops patients from seeking early diagnosis and care.[7]

In addition to this, a significant demographic transformation is altering the nature of healthcare delivery through the development of an extremely complicated second medical problem that arises due to the effectiveness of antiretroviral treatment among HIV-infected people above 50 years old.[3] More than half of the individuals diagnosed with HIV infections globally belong to this age group as a result of the effectiveness of continuous antiretroviral therapy.

Conditions like cardiovascular disease, cognitive impairment, metabolic syndrome, liver fat accumulation, and bone loss happen more frequently and more rapidly in older patients with HIV than in similarly aged patients without HIV.[9] As a result, modern HIV treatment needs to move beyond the concept of achieving viral suppression alone and place greater emphasis on safety, tolerability of medications, and metabolism over a lifetime.

- **Structural and Immunological Architecture of HIV**

The causative agent, known as Human Immunodeficiency Virus, is considered to be a human retrovirus that belongs to the Lentivirus genus, which is characterized by a low reproduction rate and long-term latency period of the virus. The mature virion of HIV virus is a spherical structure enclosed by a lipoprotein complex obtained directly from the membrane of the host cell through budding process. Heteromeric glycoprotein spike, made up of gp120 and gp41 proteins, is embedded on the outer surface of the virus. The outer surface of the HIV virus contains the bullet shaped capsid consisting of the p24 protein.[14]

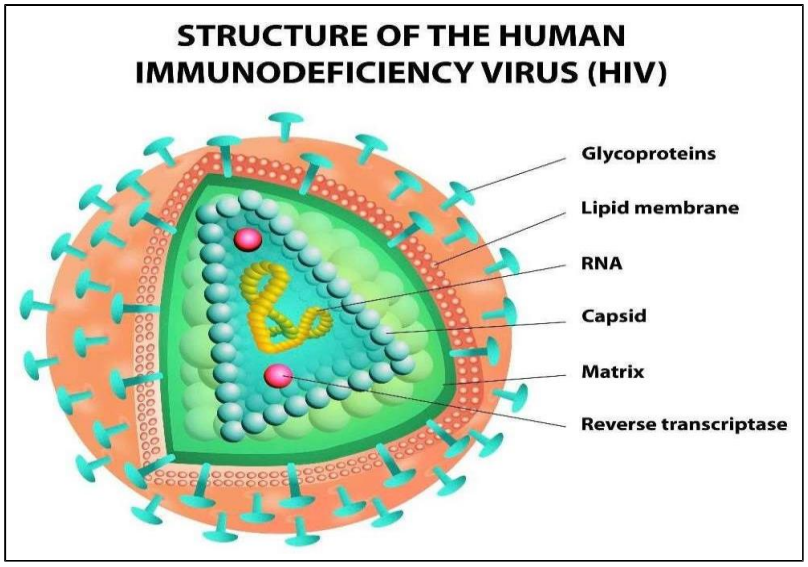

Fig. No. 1: Structure of the Human Immunodeficiency Virus (HIV).

In the inner core region, there are two complementary strands of positive-sense viral single-stranded RNA, which are linked to vital enzymes involved in the replication process, including reverse transcriptase, integrase, and protease. The basis of cellular pathology involves the affinity of the viral envelop gp120 glycoprotein for human CD4 receptor, which has a high expression level in helper T lymphocytes, monocyte-macrophage lineages, and follicular dendritic cells. Following this interaction, conformational changes lead to engagement of a secondary host chemokine coreceptors (either CCR5 or CXCR4), and as a result, the gp41 subunit undergoes another conformational alteration facilitating membrane fusion and releasing the inner core into the cytoplasm.[14]

- **Cause of the Disease**

The Human Immunodeficiency Virus (HIV) is a type of lentivirus, a member of retroviruses. There are two major strains of the HIV infection: HIV-1, which is most aggressive, contagious, and prevalent in the world; and HIV-2, mostly limited to the region of West Africa and possessing relatively lesser aggressiveness.[10,11] It spreads via certain human bodily fluids like blood, semen, vaginal fluids, rectal fluids, and breast milk.[12] This spread happens via physical contact of such fluids either with a mucous membrane or with damaged tissues or direct entry into the bloodstream via infected syringes and needles.[13] The underlying reason behind the pathological symptoms associated with this disease is the viral life cycle. The Human Immunodeficiency Virus attaches itself to the CD4 receptors present on the surface of host cells with the help of viral envelope glycoprotein (gp120); however, the

attachment of the virus and subsequent fusion requires the presence of co-receptors, mainly CCR5 and CXCR4.[14] After the penetration, the viral RNA is converted into double-stranded DNA using reverse transcriptase enzyme of the virus – a highly error-prone procedure that results in mutation of the virus.[15]

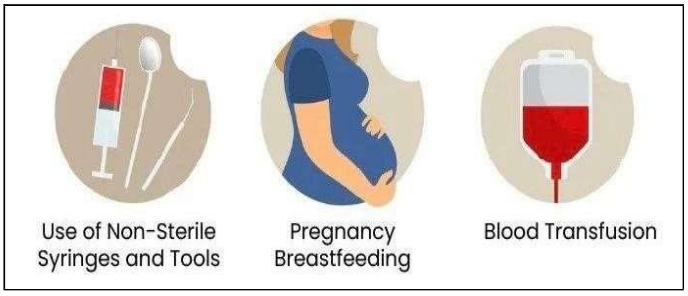

Fig. No. 2: Causes of HIV.

- **Signs and Symptoms**

The clinical progression of HIV is generally delineated into three stages, each characterized by distinct signs and symptoms.

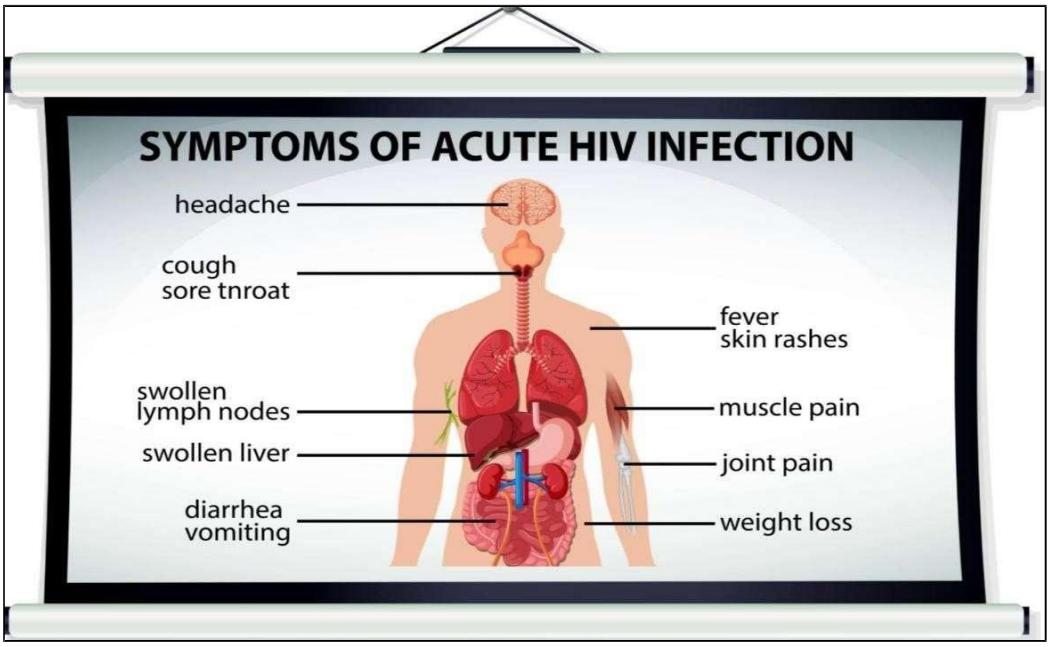

Fig. No. 3: Symptoms of Acute HIV Infection.

- **Acute HIV Infection**

ARS usually appears after about two to four weeks from the time of infection with symptoms similar to influenza or mononucleosis.[17] The most common signs of this condition include fever, swollen lymph glands, sore throat, a reddish rash, muscle pain, fatigue, and night

sweats.[18] At this stage, the amount of virus that may be present in the bloodstream is very high, making the infected person highly infectious, as well as low numbers of CD4+ cells.[18]

- **Clinical Latency (Chronic HIV Infection)**

After passing through the initial acute stage of infection, HIV develops into a stage of clinical latency, otherwise referred to as asymptomatic HIV infection. The virus replication occurs at low levels during this time.[19] There may be no specific signs and symptoms shown by patients for several years, even more than ten years in some instances if left untreated.[2] Nevertheless, there is a constant attack on the immune system by the virus. Late in this stage, with increasing viral loads and reducing CD4 count, symptoms may include mild body symptoms or infections like thrush and shingles.[2]

- **Acquired Immunodeficiency Syndrome (AIDS)**

HIV infection, if not treated, will lead to AIDS, which is characterized by a CD4+ count below 200 cells/mm³ or the presence of certain opportunistic illnesses.[20] These signs are grave and mainly occur because of opportunistic infections and cancers.[21] They include wasting or weight loss syndrome, persistent diarrhea, fever, tiredness, and neurological problems (HIV- associated neurocognitive disorders).[21] Opportunistic diseases like PCP, Kaposi's sarcoma, MAC, and CMV retinitis will now endanger patients' lives.[22]

- **Diagnosis**

It is crucial to identify HIV at an early stage for ART initiation and prevention of transmission. A great deal of improvement has been achieved since early generations of antibody tests until now when combination testing methods have been developed.[23] According to CDC recommendations, the diagnostic algorithm comprises four tests including the fourth- generation HIV-1/2 antigen/antibody combination immunoassay.[24] This test can detect HIV- 1 and HIV-2 antibodies and HIV-1 p24 antigen, which provides more opportunities for detecting the virus at an early stage comparing to antibody-only tests.[25] In case if the combination test result is positive, a supplementary differentiation HIV-1/HIV-2 antibody immunoassay is ordered.[26] It helps to diagnose HIV as well as to identify viral type. If the differentiation test is indeterminate or negative because the acute phase of disease makes only the antigen be detected and not enough antibodies, an HIV-1 NAT should be taken into account. This test can detect the presence of viral RNA directly in the blood, which confirms the acute infection.[27] Besides, point-of-care rapid tests are common despite being

recommended to undergo laboratory confirmatory testing if positive.[26]

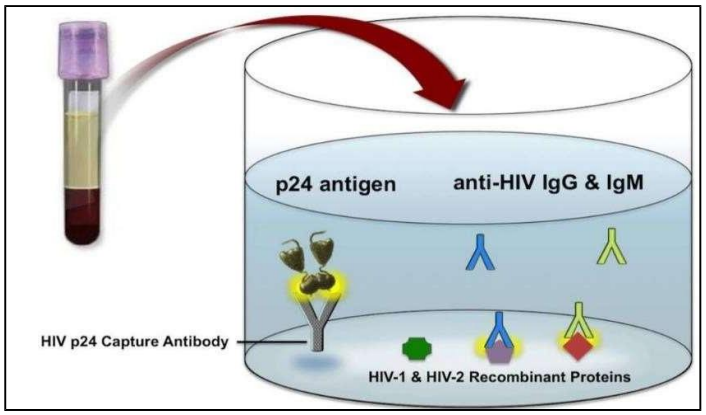

Fig. No. 4: Diagnosis Test.

TREATMENT

The treatment for HIV in modern times hinges on antiretroviral therapy (ART) that entails taking a combination of antiretroviral medication regularly throughout one's life.[5] The main objectives of ART are to ensure viral suppression to zero levels of plasma HIV-1 RNA below 50 copies/mL, preserve or improve immunological functions (reflected by an increased number of CD4+ T-lymphocytes), and reduce drug side effects.[7,20] When the HIV virus is successfully suppressed, clinical progression to AIDS is halted, and the risk of sexual transmission ceases; this is the concept of Undetectable = Untransmittable (U=U).[12]

- **Classification of HIV Drugs**

Antiretroviral drugs are strictly categorized into distinct pharmacodynamic classes based on the specific phase of the HIV replication cycle they inhibit:

Drug Class	Biochemical Mechanism of Action	Common Clinical Examples
Nucleoside/Nucleotide Reverse Transcriptase Inhibitors (NRTIs)	Function as structural analogues of natural deoxynucleotides. They get activated by intracellular phosphorylation to form triphosphates; compete with natural substrates; and become incorporated into the expanding DNA strand of viruses. Since they do not have a 3'- hydroxyl group, chain termination becomes immediate.[5,29]	Emtricitabine (FTC) Lamivudine (3TC) Tenofovir Alafenamide (TAF) Abacavir (ABC)
Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs)	The binding must be done allosterically and specifically to a hydrophobic site near the catalytic region of the reverse transcriptase enzyme. The result is a change in conformation that alters the structure of the enzyme, limiting its ability to	Doravirine (DOR) Rilpivirine (RPV) Efavirenz (EFV) Etravirine (ETR)

	catalyze and terminating transcription.[6]	
Integrase Strand Transfer Inhibitors (INSTIs)	The viral integrase enzyme plays a vital role in integrating viral DNA into the host's genome. This class of inhibitors binds to catalytic core divalent cations (either Mg2+ or Mn2+) and prevents the strand transfer required for the integration process.[31]	Bictegravir (BIC) Dolutegravir (DTG) Cabotegravir (CAB) Raltegravir (RAL)
Protease Inhibitors (PIs)	Direct Competitive Inhibitors of the Active Site of the HIV-1 Protease Enzyme. They block the process of cleavage of the gag-pol polyprotein precursors, thus preventing the maturation of the virus into infectious forms.[30]	Darunavir (DRV) Atazanavir (ATV) Ritonavir (RTV booster)
Nucleoside Reverse Transcriptase Translocation Inhibitors (NRTTIs)	A new generation drug whose mechanism of action involves multiple steps. After phosphorylation within the cell, it integrates itself into the virus's DNA chain just like NRTIs do, but thanks to its special chemical groups, it stops translocation of the reverse transcriptase enzyme, thus stopping further nucleotide matching[3]	Islatravir (ISL)
Entry, Fusion, & Co receptor Antagonists	Inhibit binding or membrane fusion. CCRC5 receptor antagonists work by binding host receptors to hinder the attachment of the virus, while fusion inhibitors act on gp41 of the virus.[14]	Maraviroc (CCR5 antagonist) Enfuvirtide (gp41 inhibitor) Ibalizumab (post attachment mAb)

Reason for choosing Doravirine–Islatravir

1. Novel Combination Therapy

Doravirine and Islatravir Both drug are among the latest innovations in HIV therapy. They were designed to increase treatment efficacy while reducing the risk of adverse effects and simplifying the administration process.

2. Effective Against HIV

Both medicines work at various phases during the HIV lifecycle and thus aid in better suppression of the virus and its multiplication.

Doravirine belongs to the category of Non-Nucleoside Reverse Transcriptase Inhibitors (NNRTIs).

Istravir is an example of Nucleoside Reverse Transcriptase Translocation Inhibitors (NRTTIs).

3. Reduced Drug Resistance

This particular combination was chosen for testing since it might lower the risk of the emergence of resistance to HIV drugs compared to previous methods.

4. Better Patient Compliance

It was created to be taken orally, just once per day by patients, making adherence to the prescribed regimen much easier.

5. Fewer Side Effects

As opposed to certain other HIV medications, Doravirine demonstrated fewer neurological and metabolic side effects. This made it more comfortable for patients.

6. Long-Acting Potential of Islatravir

Islatravir gained attention because of its long half-life, which may allow less frequent dosing in future HIV treatment.

MECHANISM OF ACTION

- **Comprehensive Pharmacological Explanation of Selected Agents Doravirine (DOR / MK-1439)**

Doravirine is an advanced generation pyridinone drug that acts on the NNRTI mechanism; it has been engineered with a high level of specificity to counteract problems inherent in first generation NNRTIs.[5] It has ecellent pharmacokinetics with half-life of about 15 hours, allowing daily administration in a single tablet regimen. Doravirine metabolizes through oxidative reactions mainly in the liver using enzymes of the CYP3A4 system.[10] Unlike first-generation NNRTIs like efavirenz, doravirine does not bind to central nervous system structures leading to neuropsychiatric side effects and it has a neutral effect on lipids with no rise of total cholesterol and LDL levels.[6] Its mechanism involves binding to allosteric sites near the active site of the HIV-1 reverse transcriptase, causing conformational changes in the enzyme, limiting its motion and catalytic activity hence preventing transcription of the single stranded RNA virus to double stranded DNA.[18] Importantly, this drug has special chemical structure that enables it to maintain complete pharmacological potency despite NNRTI resistance mutations (K103N, Y181C, G190A).[3]

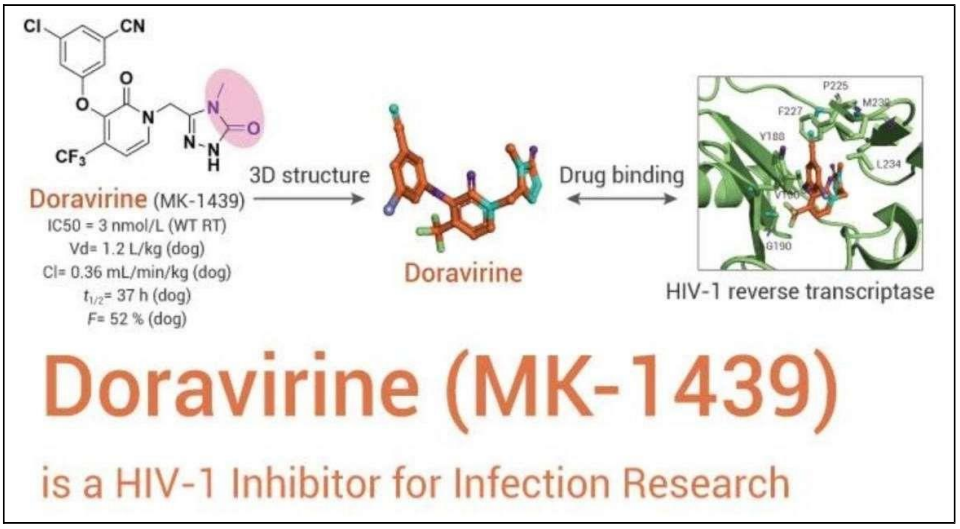

Fig. No. 5: Doravirine (MK-1439).

- **Islatravir (ISL / MK-8591)**

Islatravir is the first NRTTI.[8] Once taken up by the targeted CD4+ T-cell, the drug undergoes conversion to its active triphosphate form, islatravir triphosphate, through phosphorylation by host cellular kinases.[25] The active drug has a remarkably long intracellular half-life of over 100 hours, making it highly potent at low daily therapeutic concentrations, currently optimized to 0.25 mg/day.[29] The drug works on viral replication by means of a unique dual mode of action. Firstly, it acts as a Translocation Inhibitor because the drug has a unique 4'-ethynyl structural feature that fixes the reverse transcriptase enzyme to the growing DNA chain.[7] This prevents translocation and DNA assembly. Furthermore, it serves as Delayed Chain Terminator because if the enzyme ensures the process of translocation and incorporates another nucleotide, the islatravir compound present within the system alters the spatial orientation of the DNA chain that results in non-matching and thus stops the replication process.[9] This multiple step method forms an incredibly high barrier for the virus to be resistant to.[20] In addition to that, the optimized dose of 0.25 mg ensures complete potency against the virus without dropping the CD4+ T-lymphocytes and total lymphocytes.[19]

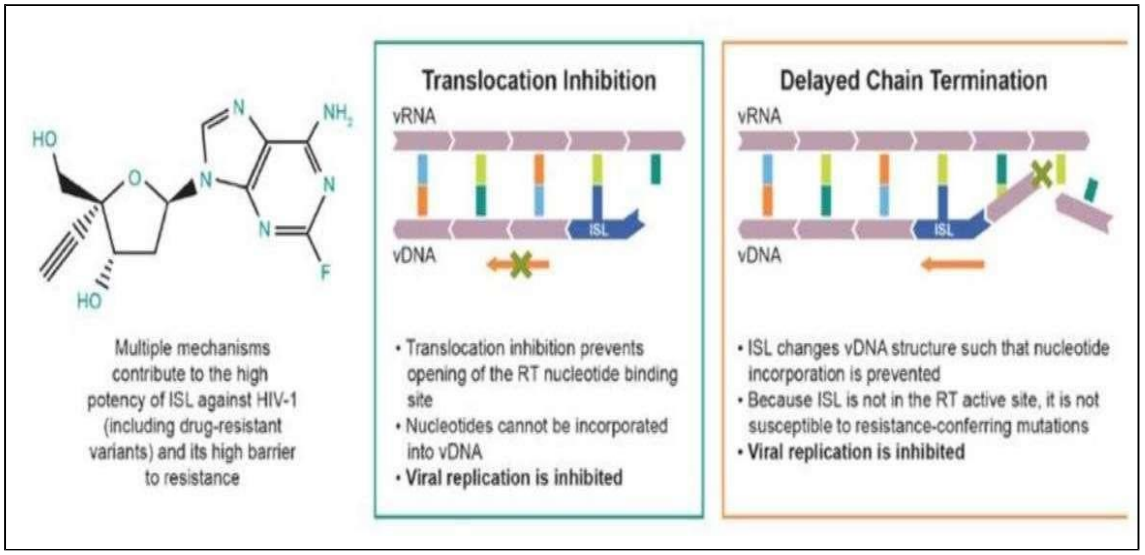

Fig. No. 6: Mechanism Action of Islatravir (ISL).

- **Efficacy of Idvynso**

The clinical effectiveness of Idvynso as evidenced by the results from Week 48 from the two pivotal Phase 3 clinical trials proves excellent efficacy as a switch treatment for virologically suppressed patients.[2] For instance, Trial 052, as a head-to-head comparator trial versus the current standard of care of Biktarvy (BIC/FTC/TAF), shows non-inferiority of Idvynso. The study showed that just 1% of patients who were on the Idvynso regimen had a viral rebound (HIV-1 RNA ≥ 50 copies/mL), and this was no different from the 1% in the group still using Biktarvy.[4] Additionally, 92% of the Idvynso regimen managed to maintain viral suppression while in 94% in the Biktarvy cohort, thus proving non-inferiority.[5] Trial 051 also showed evidence of non-inferiority. On switching patients from several baseline ART regimens to the Idvynso, just 1% of patients failed virologically compared to 5% in the group still using their baseline regimens.[6] In addition, 96% of the Idvynso group maintained viral suppression, proving the antiviral potency of Idvynso, a combination of a novel-generation NNRTI plus NRTTI.[7]

- **Safety and Tolerability**

Safety is an equally important issue when considering its use in the population, especially since the previous clinical hold placed on islatravir.[15] The lowered dose of islatravir (0.25 mg) resolved the previous issues with regard to the depletion of the CD4+ cell and total lymphocytes counts.[23] The adverse reactions seen during the study of Idvynso were found to be mild to moderate in severity.[10] During clinical trials, the adverse reactions (reported by >1% of patients) were as follows: diarrhea, dizziness, fatigue, abdominal bloating, headache,

and slight gain in weight.[1]

- **Precautions and Adverse Warnings**

Although the drug is safe, there are some important precautions which need to be considered while using Idvynso. After-market experience with doravirine-based therapies has documented cases of Stevens-Johnson syndrome (SJS) and toxic epidermal necrolysis (TEN).[3] Moreover, Drug Rash with Eosinophilia and Systemic Symptoms (DRESS) syndrome was observed with Idvynso.[1] If a progressive, severe rash occurs along with mucosal or organ involvement, treatment should be stopped immediately.[5] In addition, doravirine is mainly processed through the metabolic pathway cytochrome P450 3A (CYP3A).[16] Concomitant administration of Idvynso with any potent CYP3A enzyme inducer (e.g., rifampin, carbamazepine, St. John's wort) is highly contraindicated because these enzymes can significantly reduce the levels of doravirine, thus resulting in decreased efficacy of therapy and drug resistance.[17] Besides, concomitant use of lamivudine (3TC) and emtricitabine (FTC) with Idvynso is also contraindicated.[18] This is caused by similar mechanisms involved in intracellular metabolism of islatravir and other.

- **Advantages of the Drug**

Two-Drug Regimen: It lowers the overall number of pills and chemicals taken when compared to traditional three or four drugs regimen; this is extremely important for elderly people suffering from HIV with polypharmacy issues.[11]

INSTI- and Tenofovir-Free: It offers an essential choice for patients who suffer from toxicity, weight gain, and intolerance to Integrase Strand Transfer Inhibitors or tenofovir-based therapy.[11]

Once-Daily Treatment: The medication is given in a single dose once per day, regardless of food intake.[12]

High Resistance to Mutations: Unique modes of action of both medications prevent any potential mutation.[13]

- **Disadvantages of the Drug**

1. Limited Indication: The drug is indicated only for use as a maintenance therapy among individuals who are already virologically suppressed (less than 50 copies of HIV-1 RNA/mL). It cannot be administered to naïve individuals or those that have a record of virological failure.[14]

2. Possibility of Severe Hypersensitivity Reactions: The risk of developing DRESS/SJS/TEN is high, although it occurs rarely.[11]
3. Strictly Controlled Interactions: The strict limitations imposed by interactions with CYP3A inducers as well as certain other antiretrovirals make it difficult to use in patients with conditions that need other medications (e.g., rifampin in cases of tuberculosis).[17]

- **Prevention and Treatment Plan**

Although Idvynso may not be considered a prophylactic vaccine but a drug for the suppression of the virus, it is effective in sexually preventing the transmission of HIV (Undetectable = Untransmittable).[18] In relation to the treatment regime of Idvynso, confirmation of suitability based on the virologic suppression of the virus and lack of prior doravirine resistance should be considered.[19] The dose administered would be 1 tablet per day containing 100 mg doravirine and 0.25 mg islatravir.[20] The assessment of liver enzymes, kidney function, and CD4/lymphocytes should be regularly carried out following standard guidelines for HIV patients.[21] If rifabutin needs to be taken in cases of other infections, then the regimen should be changed to allow for an additional 100 mg of doravirine tablets after taking Idvynso.[22]

- **Drug Interactions (CYP3A)**

Doravirine is mainly metabolized through CYP3A metabolism pathway. The use of Idvynso together with potent CYP3A enzyme inducers such as rifampin, carbamazepine, and St. John's wort should be avoided at all costs since it may significantly reduce the concentration of doravirine in the body, making it lose its effectiveness and cause resistance in the virus**.**

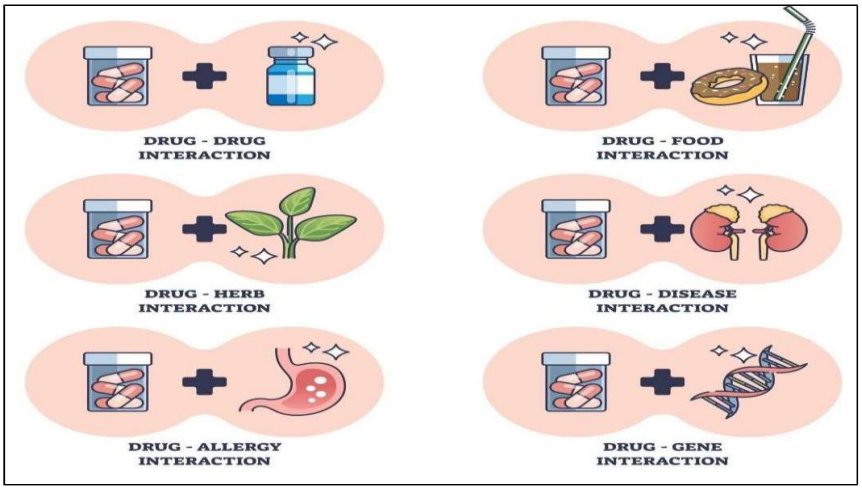

Fig No. 7: Drug Interaction.

- **Contraindicated Co-administration**

The use of Idvynso should be avoided when used alongside lamivudine (3TC) or emtricitabine (FTC), considering that both drugs use a common pathway to function with islatravir.[27]

RESULT

The combined use of Doravirine and Islatravir was found to be effective in controlling HIV infection. Studies showed a noticeable decrease in viral load and an increase in CD4 cell levels, reflecting improvement in immune system function. The treatment was generally well accepted by patients and produced comparatively fewer side effects. Its simple once-daily dosing schedule may also help improve treatment adherence and support long-term management of HIV disease.

CONCLUSION

Optimization in terms of HIV therapeutics has led to great developments over the past four decades. The introduction of Idvynso can be considered a groundbreaking moment in this regard.

Through the combination of the powerful, but extremely tolerable NNRTI doravirine with the novel NRTTI islatravir, whose dual-action mechanism makes it unique, the two-drug therapy was achieved in a highly efficient manner.

The impressive set of data collected during Trials 051 and 052 proves that Idvynso is comparable in its effects with proven three-drug combinations such as Biktarvy. Of note, the adjustment of the islatravir dosage to 0.25 mg solved the problem of possible lymphocyte depletion.

The only drawback of the drug may be the need for careful monitoring of rare adverse effects such as DRESS and SJS as well as potential drug interactions with CYP3A drugs. However, its benefits significantly outweigh the risks. Firstly, this drug is a valuable option for tenofovir- free INSTI alternatives among the expanding adult population living with HIV.

In light of the growing difficulty patients are having regarding polypharmacy and the development of side effects from drug toxicity, being able to suppress viruses using less medications becomes even more crucial. Not only does Idvynso increase drug options

available, but it confirms today's philosophy in treatment, which includes eradicating viruses and ensuring overall health.

REFERENCES

1. UNAIDS. (2024). Global HIV & AIDS statistics — Fact sheet. Geneva: Joint United Nations Programme on HIV/AIDS.
2. Fauci, A. S. HIV and AIDS: 20 years of science. Nature Medicine, 2003; 9(7): 839-843.
3. Beyrer, C., Baral, S. D., Collins, C., et al. The global response to HIV in the context of COVID-19: An opportunity for sustainable health systems. The Lancet HIV, 2021; 8(11): e711- e721.
4. Deeks, S. G., Overbaugh, J., Phillips, A., & Buchbinder, S. HIV infection. Nature Reviews Disease Primers, 2015; 1(1): 1-22.
5. Pau, A. K., & George, J. M. Antiretroviral therapy: current drugs. Infectious Disease Clinics, 2014; 28(3): 371-402.
6. Arts, E. J., & Hazuda, D. J. HIV-1 antiretroviral drug therapy. Cold Spring Harbor Perspectives in Medicine, 2012; 2(4): a007161.
7. Günthard, H. F., Saag, M. S., Benson, C. A., et al. Antiretroviral drugs for treatment and prevention of HIV infection in adults: 2016 recommendations of the International Antiviral Society-USA panel. JAMA, 2016; 316(2): 191-210.
8. Merck & Co. (2026). FDA Approves Merck's Once-Daily IDVYNSO (doravirine/islatravir). Merck Press Release, April 21, 2026.
9. U.S. Food and Drug Administration. (2026). FDA Approves Idvynso for the Treatment of HIV-1 Infection in Adults. FDA Approval Letters.
10. Sharp, P. M., & Hahn, B. H. Origins of HIV and the AIDS pandemic. Cold Spring Harbor Perspectives in Medicine, 2011; 1(1): a006841.
11. Marx, P. A., Alcabes, P. G., & Drucker, E. Serial human passage of simian immunodeficiency virus by unsterile injections and the emergence of epidemic human immunodeficiency virus in Africa. Philosophical Transactions of the Royal Society of London. Series B: Biological Sciences, 2004; 359(1447): 911-926.
12. Cohen, M. S., Chen, Y. Q., McCauley, M., et al. Prevention of HIV-1 infection with early antiretroviral therapy. New England Journal of Medicine, 2011; 365(6): 493-505.
13. Shao, Y., & Williamson, C. The HIV-1 epidemic: low- to middle-income countries. Cold Spring Harbor Perspectives in Medicine, 2012; 2(3): a007187.

14. Deeks, S. G., Lewin, S. R., & Havlir, D. V. The end of AIDS: HIV infection as a chronic disease. The Lancet, 2023; 402(10407): 1083-1097.
15. Guaraldi, G., Palella, F. J., & Rockwood, K. Clinical management of aging people living with HIV: Frailty and modern antiretroviral therapy choices. Journal of Infectious Diseases, 2022; 226(3): S295-S304.
16. Siliciano, J. M., & Siliciano, R. F. A long-term latent reservoir for HIV-1: discovery and clinical implications. Journal of Antimicrobial Chemotherapy, 2004; 54(1): 6-9.
17. Molina, J. M., Squires, K., Sax, P. E., et al. Doravirine 100 mg combined with low-dose Islatravir 0.25 mg once daily in virologically suppressed adults: Week 96 results from phase 3 randomized clinical trials. Journal of Acquired Immune Deficiency Syndromes, 2025; 98(3): 241-250.
18. Schacker, T., Collier, A. C., Hughes, J., Shea, T., & Corey, L. Clinical and epidemiologic features of primary HIV infection. Annals of Internal Medicine, 125(4): 257-264.
19. McArthur, J. C., Hoover, D. R., Bacellar, H., et al. Dementia in AIDS patients: incidence and risk factors. Multicenter AIDS Cohort Study. Neurology, 2003; 43(11): 2245-2255.
20. Brooks, J. T., Kaplan, J. E., Holmes, K. K., et al. HIV-associated opportunistic infections—going, going, but not gone: the continued need for prevention and treatment guidelines. Clinical Infectious Diseases, 2009; 48(5): 609-611.
21. Branson, B. M., Owen, S. M., Wesolowski, L. G., et al. (2014). Laboratory testing for the diagnosis of HIV infection: updated recommendations. Centers for Disease Control and Prevention.
22. Centers for Disease Control and Prevention. (2014). Recommended laboratory HIV testing algorithm for serum or plasma specimens. Atlanta, GA: US Department of Health and Human Services.
23. Alexander, T. S. Human immunodeficiency virus diagnostic testing: 30 years of evolution. Clinical and Vaccine Immunology, 2016; 23(4): 249-253.
24. Geibel, S., Hossain, M., & Pulerwitz, J. (2016). HIV testing and counseling algorithms. World Health Organization.
25. Fiscus, S. A., Pilcher, C. D., Miller, W. C., et al. Rapid, real-time detection of acute HIV infection in patients in Africa. Journal of Infectious Diseases, 2006; 194(4): 488-495.
26. Broder, S. The development of antiretroviral therapy and its impact on the HIV- 1/AIDS pandemic. Antiviral Research, 2010; 85(1): 1-18.
27. Palella Jr, F. J., Delaney, K. M., Moorman, A. C., et al. Declining morbidity and mortality

among patients with advanced human immunodeficiency virus infection. New England Journal of Medicine, 338(13): 853-860.

28. Blankson, J. N. Integrase inhibitors in HIV clinical practice. Johns Hopkins Advanced Studies in Medicine, 2014; 14(1): 6-12.
29. Bourgi, K., Rebeiro, P. F., Turner, M., et al. Greater weight gain in treatment-naive persons starting dolutegravir based antiretroviral therapy. Clinical Infectious Diseases, 2020; 70(7): 1267-1274.
30. Sax, P. E., Erlandson, K. M., Lake, J. E., et al. Weight gain following initiation of antiretroviral therapy: risk factors in randomized comparative clinical trials. Clinical Infectious Diseases, 2020; 71(6): 1379-1389.
31. Cahn, P., Madero, J. S., Arribas, J. R., et al. Dolutegravir plus lamivudine versus dolutegravir plus tenofovir disoproxil fumarate and emtricitabine in antiretroviral-naive adults with HIV-1 infection (GEMINI-1 and GEMINI-2): week 48 results from two multicentre, double-blind, randomised, non-inferiority, phase 3 trials. The Lancet, 2019; 393(10167): 143-155.
32. Squires, K. E., Ruane, P. J., DeJesus, E., et al. Doravirine (MK-1439), a novel nonnucleoside reverse transcriptase inhibitor. Antimicrobial Agents and Chemotherapy, 2018; 62(10): e00862.