Proceedings of the 7th International Conference on Materials Chemistry and Environmental Engineering
DOI: 10.54254/2755-2721/2027.39377

Zidovudine Versus Maraviroc in HIV-1 Therapy: A Comparative Review of Development, Chemistry, Clinical Roles, and Access

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Abstract. Zidovudine (AZT) and Maraviroc mark different stages in the development of antiretroviral therapy. AZT emerged from synthetic nucleoside research, was repurposed after anti-HIV activity was identified, and became the first approved antiretroviral drug. Maraviroc was developed through a CCR5-focused medicinal chemistry programme and became the first approved oral drug to block a host co-receptor used for HIV-1 entry. This review directly compares their origins and development, chemical structures and physicochemical characteristics, oral delivery and dosing, molecular mechanisms, clinical applications and evidence, limitations, and economic and access considerations. AZT is a small thymidine analogue that requires intracellular phosphorylation and terminates viral DNA synthesis; it is available orally and intravenously, but marrow and mitochondrial toxicity and thymidine-analogue resistance have displaced it from routine adult first-line treatment. Its current importance is mainly historical and selected, including specific intrapartum and neonatal settings. Maraviroc is a larger non-peptide small molecule that allosterically binds CCR5 at the cell surface. It is orally administered, its dose varies with CYP3A interactions, and it must be combined with other active antiretroviral agents. Its use is restricted to confirmed CCR5-tropic HIV-1; CXCR4-tropic and mixed/dual-tropic virus are not susceptible. Required tropism testing creates additional cost, turnaround-time and feasibility barriers. The comparison shows that the two drugs should not be ranked by trial percentages obtained in different eras and populations. Instead, they illustrate how target selection, toxicity, biomarkers, delivery, affordability and health-system capacity together determine clinical value.

Keywords: HIV-1, zidovudine, maraviroc, antiretroviral therapy, drug access

1. Introduction

1.1. Global burden and significance of HIV infection

HIV remains a major global health problem despite the effectiveness of antiretroviral therapy (ART). The virus primarily infects CD4+ T lymphocytes and progressively damages cellular immunity when untreated. For a single consistent epidemiological year, WHO estimated that 40.8

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million people were living with HIV at the end of 2024, 1.3 million acquired HIV during 2024, and 630,000 died from HIV-related causes; 65% of people living with HIV were in the WHO African Region [1]. Figure 1 summarises these global estimates.

Combination ART can suppress plasma HIV RNA, permit immune recovery, reduce morbidity and mortality, and prevent sexual transmission when suppression is maintained [2, 3]. It does not remove integrated proviral DNA from long-lived cellular reservoirs, so treatment is generally lifelong [4]. This review uses AZT and Maraviroc to examine how two very different molecular designs became clinically useful, and why neither now serves as a universal adult first-line drug.

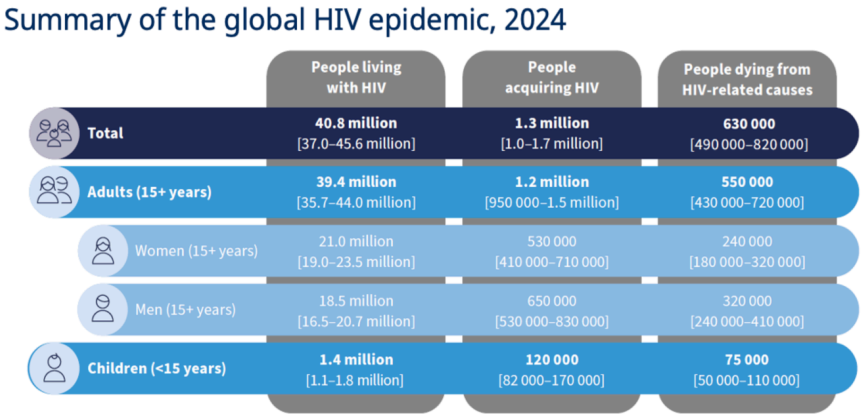


Figure 1. Summary of the global HIV epidemic in 2024. The same 2024 estimates are used in Section 1.1. Source: WHO [1]

1.2. HIV life-cycle context for the comparison

Only two stages of the HIV-1 life cycle are required for the present comparison. Entry begins when viral gp120 binds CD4 and then a co-receptor, usually CCR5 or CXCR4, enabling gp41-mediated membrane fusion. After entry, reverse transcriptase converts viral RNA into DNA, which can then integrate into the host genome [5].

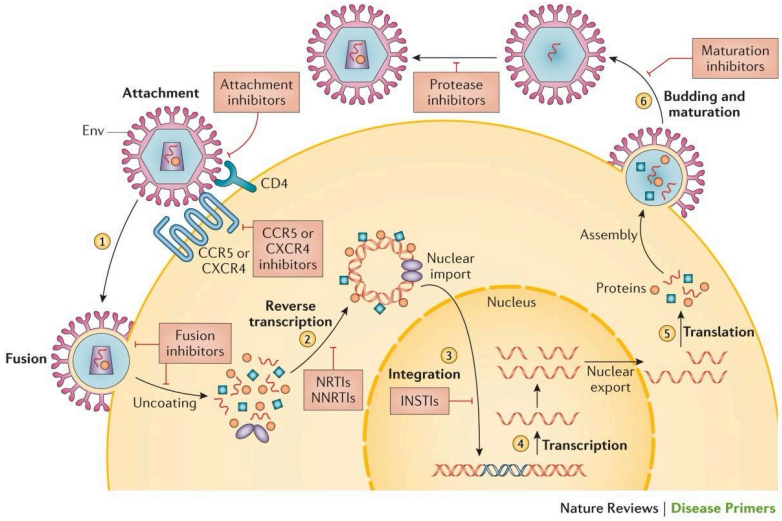


Figure 2. HIV-1 replication cycle and major antiretroviral targets. The comparison in this review focuses on entry and reverse transcription. Adapted from Deeks et al. [5]

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Maraviroc acts before entry by altering CCR5 so that CCR5-tropic HIV-1 cannot productively engage the receptor. AZT acts later, after cellular uptake and phosphorylation, by interrupting reverse transcription. The remaining stages shown in Figure 2 provide context for combination therapy but are not reviewed in detail here.

1.3. ART context relevant to AZT and maraviroc

Modern ART combines active agents to obtain durable suppression and prevent resistance. Current U.S. guidance generally builds initial therapy around a second-generation integrase inhibitor plus an appropriate nucleoside backbone, or another recommended complete regimen; AZT is no longer commonly used in routine adult practice, and Maraviroc is reserved for selected patients with confirmed CCR5-tropic virus [6].

AZT and Maraviroc must therefore be understood as components of combination treatment rather than competing monotherapies. Table 1 limits the ART background to the classes needed to locate the two drugs in contemporary care.

Table 1. ART classes required for the AZT-Maraviroc comparison

Class	Stage/target	Relevance to this review
NRTIs	Reverse transcription	AZT is the historical example; it is now used selectively rather than as a routine adult first-line drug.
CCR5 antagonists	CCR5-dependent entry	Maraviroc is used only for confirmed CCR5-tropic HIV-1 and only with other active agents.
INSTIs	Integration	These agents anchor many current initial regimens and explain the changed clinical position of both comparison drugs.

2. Zidovudine (AZT)

AZT was not designed for the HIV epidemic. Its synthesis was first reported by Horwitz, Chua and Noel in 1964 during research on modified nucleosides, two decades before HIV was identified [7]. Mitsuya et al. [8] subsequently showed that AZT inhibited the infectivity and cytopathic effects of the virus then called HTLV-III/LAV. Early clinical evidence followed rapidly, and AZT became the first antiretroviral approved in the United States in 1987 [9]. This sequence - chemical synthesis, biological repurposing and accelerated clinical development - is central to its historical importance.

2.1. Origin, chemical properties, delivery and mechanism

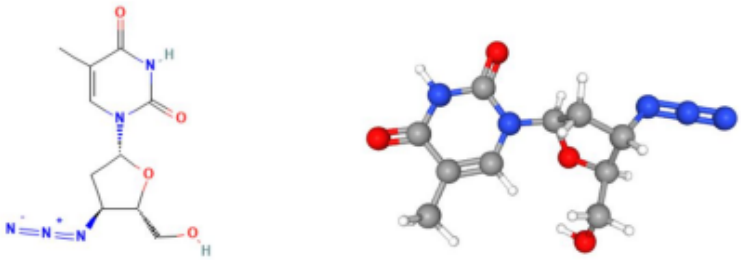


Figure 3. Two-dimensional (left) and three-dimensional (right) representations of AZT, showing the thymidine analogue and 3'-azido substitution. Source: NCBI PubChem CID 35370 [10]

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AZT (C10H13N5O4; molecular weight 267.24 g/mol) is a synthetic thymidine analogue. Its defining modification is replacement of the sugar's 3'-hydroxyl group with an azido group (Figure 3). The molecule is therefore small and nucleoside-like, which supports cellular uptake but also means that it is a prodrug requiring intracellular activation [10]. Current U.S. labelling retains oral and intravenous formulations and specifies 300 mg twice daily for adults, used with other antiretroviral agents [11]. Table 2 summarises the main chemical and delivery properties of AZT.

Table 2. Selected chemical and delivery properties of AZT

Property	AZT
Chemical class	Synthetic thymidine nucleoside analogue; NRTI
Formula / molecular weight	C10H13N5O4 / 267.24 g/mol
Key structural feature	3'-azido group replacing the 3'-hydroxyl group
Activation	Three sequential intracellular phosphorylation steps to AZT triphosphate
Delivery	Oral capsules/tablets/solution; intravenous formulation is also available
Labelled adult oral dose	300 mg twice daily with other antiretroviral agents
Main elimination	Hepatic glucuronidation; glucuronide metabolite is renally excreted

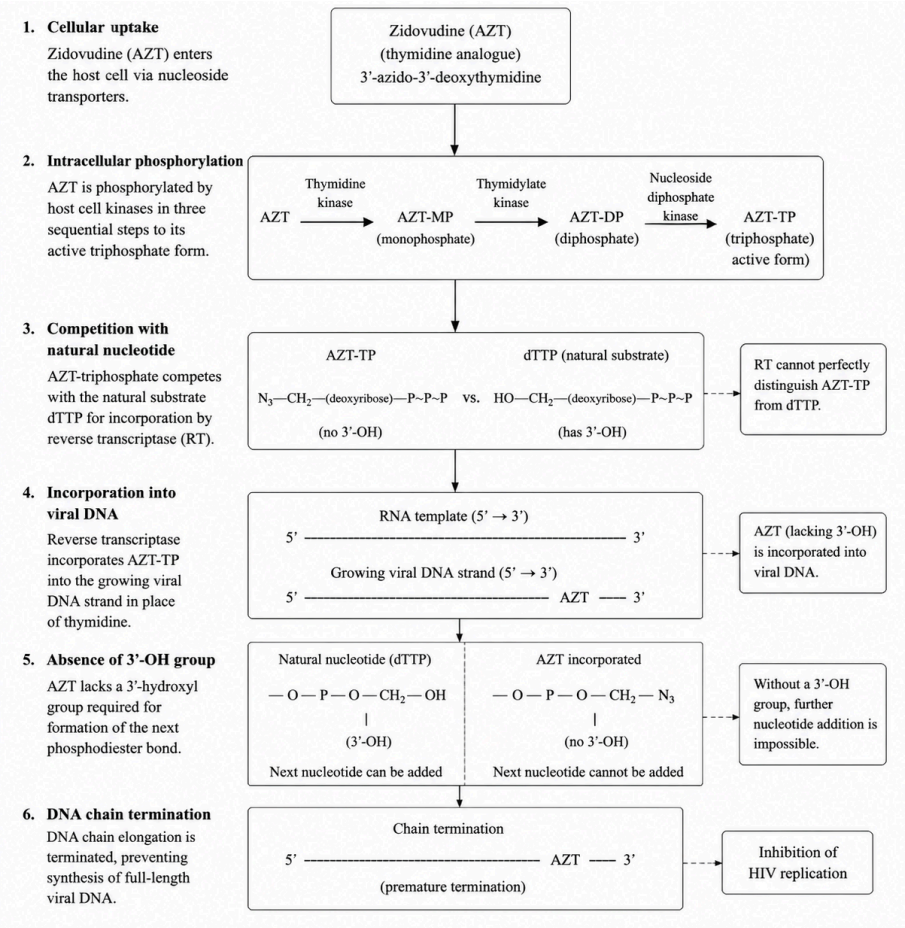


Figure 4. AZT activation and chain-termination mechanism. Created by the author based on Mitsuya et al. [8] and Furman et al. [12]

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After uptake, host kinases convert AZT to mono-, di- and finally triphosphate forms. AZT triphosphate competes with natural deoxythymidine triphosphate for incorporation by HIV reverse transcriptase. Because the incorporated analogue lacks a functional 3'-hydroxyl group, the next phosphodiester bond cannot form and viral DNA elongation terminates [8, 12]. This condensed mechanism is shown once in Figure 4 rather than repeated throughout the review.

2.2. Clinical evidence, current role and limitations

AZT provided early evidence that pharmacological suppression of HIV could improve clinical outcomes, but monotherapy produced incomplete and temporary control and selected resistance. Its most durable public-health contribution came from Pediatric AIDS Clinical Trials Group protocol 076. In that trial, an antepartum, intrapartum and neonatal AZT regimen reduced estimated maternal-infant transmission from 25.5% with placebo to 8.3% with AZT [13]. Figure 5 illustrates the transmission estimates in ACTG 076.

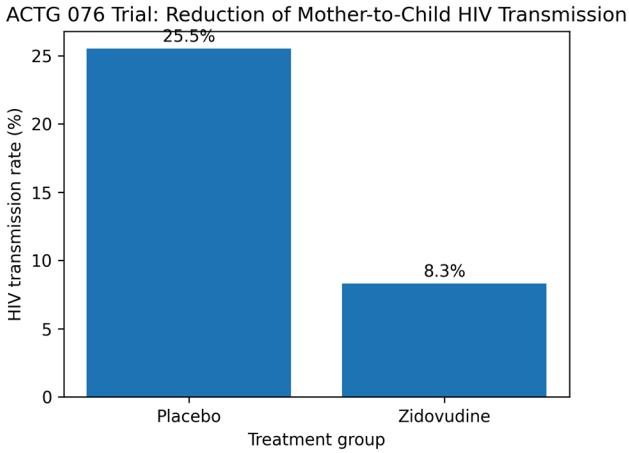


Figure 5. Estimated mother-to-child HIV transmission in ACTG 076. These historical trial percentages describe a perinatal prophylaxis comparison and are not comparable with Maraviroc virological endpoints. Created from Connor et al. [13]

AZT is not a routine first-line drug for most adults or adolescents. Current HHS guidance notes that it is no longer commonly used in adult practice, reflecting the better tolerability, convenience and resistance profiles of newer regimens [6]. It nevertheless remains available for selected circumstances. In perinatal care, intravenous AZT may be used during labour according to maternal viral load and adherence considerations, and AZT remains frequently used in neonatal prophylaxis, with drug number and duration determined by transmission risk and gestational age [14].

The principal limitations are bone-marrow suppression, especially anaemia and neutropenia, and longer-term mitochondrial toxicities including myopathy and lactic acidosis. Resistance commonly involves thymidine analogue mutations in reverse transcriptase that enhance excision of the incorporated analogue [15-17]. These disadvantages explain why AZT's current value is specialised rather than a continuation of its former first-line status.

3. Maraviroc

Maraviroc arose from a deliberate host-targeted discovery programme. A high-throughput screen of Pfizer's compound collection identified an imidazopyridine CCR5 ligand; successive medicinal-

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chemistry optimisation produced the potent, orally bioavailable clinical candidate UK-427,857, later named Maraviroc [18]. The drug was approved in the United States in 2007, establishing CCR5 blockade as a clinically viable antiretroviral strategy.

3.1. Origin, chemical properties, delivery and mechanism

Maraviroc (C29H41F2N5O; molecular weight 513.67 g/mol) is a non-peptide small molecule, not a protein. Its triazole, azabicycloalkane, phenyl and difluorocyclohexane carboxamide features support selective interaction with the transmembrane pocket of the host CCR5 receptor [19, 20]. The chemical structure is shown in Figure 6, and the main chemical and delivery properties are summarised in Table 3.

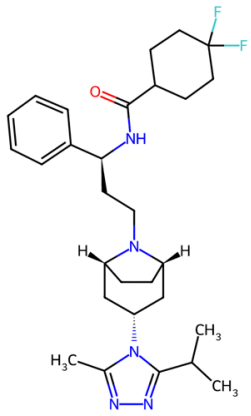


Figure 6. Chemical structure of Maraviroc, a non-peptide small-molecule CCR5 antagonist. Source: NCBI PubChem CID 3002977 [19]

Table 3. Selected chemical and delivery properties of Maraviroc

Property	Maraviroc
Chemical class	Non-peptide small-molecule CCR5 antagonist
Formula / molecular weight	C29H41F2N5O / 513.67 g/mol
Primary target	Host-cell CCR5 co-receptor
Activation	No intracellular phosphorylation required
Delivery	Oral tablets or solution; taken twice daily
Adult dosing principle	150, 300 or 600 mg twice daily according to interacting medicines
Main metabolism	CYP3A-mediated; clinically important inhibitor/inducer interactions
Virological prerequisite	Only CCR5-tropic HIV-1; pre-treatment tropism testing required

Maraviroc binds allosterically within CCR5 rather than occupying the external gp120-binding surface. This stabilises a receptor conformation that HIV-1 gp120 cannot use productively, so gp41-mediated fusion and entry are blocked [18, 20]. The target is the host receptor CCR5; the drug itself is a small molecule. It has no meaningful activity against CXCR4-tropic HIV-1 or mixed/dual-tropic populations. Figure 7 illustrates CCR5 blockade by Maraviroc.

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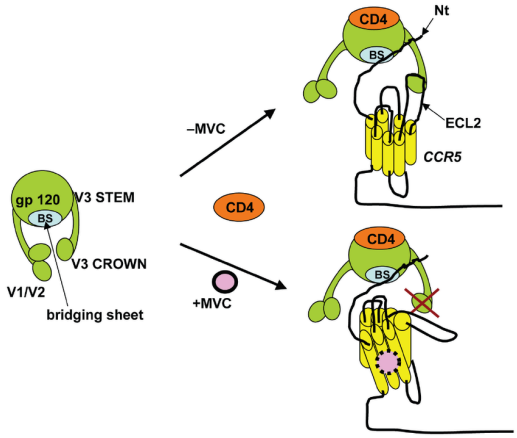


Figure 7. Maraviroc binds within CCR5 and prevents productive gp120-CCR5 engagement, thereby blocking entry of CCR5-tropic HIV-1. Adapted from Latinovic et al. [21] under CC BY 2.0

3.2. Clinical evidence, patient selection and limitations

Maraviroc must be prescribed with other active antiretroviral agents; it is not a monotherapy. The MOTIVATE-1 and MOTIVATE-2 trials enrolled treatment-experienced adults with CCR5-tropic HIV-1 and compared Maraviroc plus optimised background therapy with placebo plus optimised background therapy. At week 24, HIV-1 RNA below 50 copies/mL was achieved by 45.3% of the twice-daily Maraviroc group and 23.0% of the placebo group, with greater CD4+ T-cell recovery in the Maraviroc group [22]. These results establish add-on efficacy in a selected population, not efficacy as a single agent. Figure 8 presents the week-24 virological response.

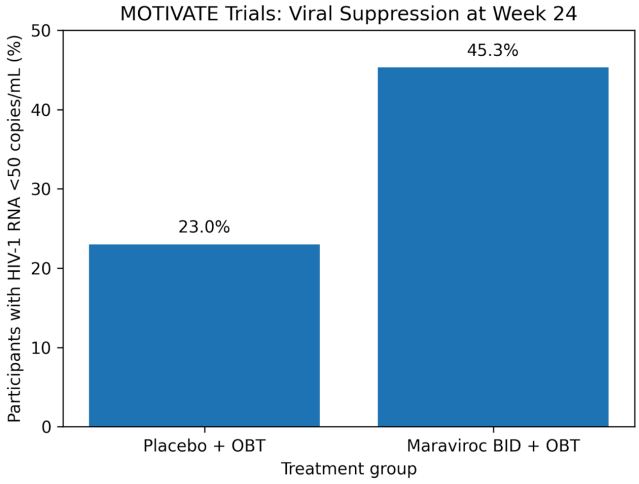


Figure 8. Week-24 virological response in the pooled MOTIVATE studies. Both groups received optimised background therapy; the comparison is Maraviroc versus placebo added to that regimen. Created from Gulick et al. [22]

Clinical selection begins with a sufficiently sensitive co-receptor tropism assay. Maraviroc is appropriate only when CCR5-tropic HIV-1 is confirmed; it should not be used for CXCR4-tropic or mixed/dual-tropic virus [6, 23]. Failure can reflect outgrowth of previously undetected CXCR4-using variants or envelope changes that allow use of inhibitor-bound CCR5 [24].

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Tropism testing is also a practical limitation. It adds laboratory cost, may require a reference laboratory, delays regimen selection, and can be difficult to implement where specialised molecular testing is unavailable. A U.S. practice study illustrated both substantial assay cost and turnaround time, although its historical dollar values should not be generalised to current systems [25]. Additional limitations include twice-daily dosing, CYP3A-mediated interactions that can change the dose threefold, postural hypotension, and rare hepatotoxicity or hypersensitivity. Maraviroc therefore remains a precision option for selected patients rather than a standard initial drug for everyone.

4. Direct comparisons of zidovudine and maraviroc

The most informative comparison is not a repetition of their mechanisms, but an integrated assessment of where each drug came from, what kind of molecule it is, how it is delivered, who can receive it, what evidence supports its use, and what health-system resources it requires. Table 4 and Figure 9 summarise these distinctions.

Table 4. Integrated comparison of AZT and Maraviroc

Criterion	Zidovudine (AZT)	Maraviroc
Origin and development	Synthesised in 1964; anti-HIV activity reported in 1985; first U.S.-approved antiretroviral in 1987.	Derived from a Pfizer CCR5 high-throughput screen and medicinal-chemistry optimisation; U.S. approval in 2007.
Chemical design	Small thymidine nucleoside analogue; C10H13N5O4; 267.24 g/mol.	Larger non-peptide small molecule; C29H41F2N5O; 513.67 g/mol.
Delivery and dosing	Oral and IV formulations; labelled adult oral dose 300 mg twice daily with other ART.	Oral tablets/solution; 150, 300 or 600 mg twice daily according to CYP3A interactions, with other ART.
Target and stage	Intracellular AZT triphosphate inhibits HIV reverse transcriptase and terminates DNA elongation.	Binds host-cell CCR5 at the membrane and prevents entry of susceptible HIV-1.
Activation / metabolism	Requires three phosphorylation steps; mainly hepatic glucuronidation.	No activation; CYP3A metabolism creates important inhibitor/inducer interactions.
Patient selection	No tropism restriction; avoid or monitor carefully with anaemia, marrow suppression or relevant resistance.	Confirmed CCR5-tropic HIV-1 only; mandatory tropism testing; ineffective for CXCR4 or mixed/dual tropism.
Clinical evidence	Foundational early treatment evidence and ACTG 076 perinatal prevention evidence.	MOTIVATE add-on evidence in treatment-experienced, CCR5-tropic HIV-1 with optimised background ART.
Current role	Not commonly used in routine adult care; selected use, including specific intrapartum and neonatal settings.	Selected precision option, often when resistance or intolerance limits other fully active agents.
Principal limitations	Anaemia, neutropenia, mitochondrial toxicity, twice-daily dosing and thymidine-analogue resistance.	Restricted tropism, testing burden, CYP3A interactions, twice-daily dosing and virological escape.
Economics and access	Low-cost generic in U.S. benchmarks; ZDV-containing products remain on the 2025 WHO Essential Medicines List.	Higher U.S. generic benchmark plus the cost/turnaround of tropism testing; specialised access may limit feasibility.

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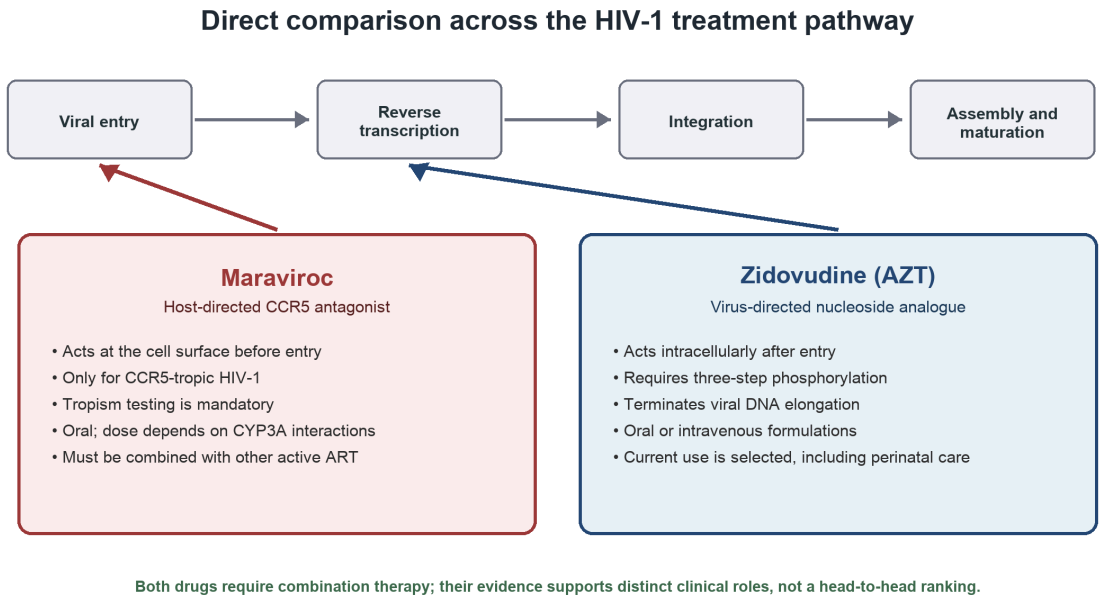


Figure 9. Direct comparison of target stage, activation, delivery and patient-selection requirements. Created by the author from the sources reviewed

4.1. Integrated clinical interpretation

AZT's chemical resemblance to a natural nucleoside allows it to enter the nucleotide pathway, but this advantage creates dependence on host phosphorylation and contributes to off-target toxicity. Maraviroc needs no activation and acts before intracellular replication, but targeting a host co-receptor narrows its virological spectrum and makes a biomarker assay essential. Thus, a simpler mechanism does not necessarily produce simpler clinical delivery.

The trial percentages in ACTG 076 and MOTIVATE should not be placed in a single efficacy ranking. ACTG 076 measured maternal-infant transmission under a historical prophylaxis regimen, whereas MOTIVATE measured virological response to an add-on drug in treatment-experienced adults with confirmed CCR5-tropic virus. Different eras, populations, background regimens and endpoints prevent a valid direct numerical claim of superiority.

4.2. Economics, access and feasibility

Economic comparisons are country-, payer-, formulation- and date-specific. As one transparent U.S. benchmark, the 2026 HHS guideline tables (using price data current to April 2025) list a monthly wholesale acquisition cost of USD 36-54 for 60 generic 300-mg AZT tablets, compared with USD 700 for 60 generic 150- or 300-mg Maraviroc tablets. These are list-price benchmarks, not patient out-of-pocket costs, and they exclude rebates, insurance arrangements, clinical monitoring and laboratory testing [6].

Access differs beyond tablet price. AZT has multiple generic formulations, oral and IV delivery, and ZDV-containing products remain included in the 2025 WHO Model List of Essential Medicines [26]. Maraviroc also has generic products in some markets, but its use depends on timely tropism testing and capacity to manage interactions. Where molecular testing is unavailable or unaffordable, an otherwise active drug may be clinically infeasible. Conversely, low acquisition cost does not restore AZT to first-line status when toxicity and regimen quality make newer options preferable.

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5. Future outlook

The histories of AZT and Maraviroc point to two practical directions rather than a broad catalogue of future HIV technologies. First, the limitations of AZT favour safer, simpler and longer-acting combinations that preserve complete viral suppression without repeating the toxicity and resistance problems of early monotherapy. Second, Maraviroc shows both the promise and the implementation burden of biomarker-guided, host-directed treatment: future targeted interventions must be paired with affordable diagnostics and must remain active against viral escape pathways.

CCR5-directed gene editing and reservoir-focused strategies extend the host-targeting principle, but they are not substitutes for current ART and remain investigational [27]. The central lesson from both drugs is therefore translational: molecular innovation becomes useful only when it can be combined safely, delivered reliably, selected accurately and accessed equitably.

6. Conclusion

AZT and Maraviroc represent distinct milestones in HIV pharmacology. AZT transformed a previously synthesised nucleoside analogue into the first approved antiretroviral and established reverse-transcriptase inhibition as a workable therapeutic principle. Maraviroc emerged from targeted CCR5 discovery and demonstrated that a host entry factor could be blocked by an oral small molecule.

Their present clinical value is narrower than their historical significance. AZT is not a routine adult first-line drug but remains relevant in selected situations, particularly specific intrapartum and neonatal care. Maraviroc is only for confirmed CCR5-tropic HIV-1, must be combined with other active ART, and carries testing, interaction and access requirements that limit routine use.

A fair comparison therefore integrates chemistry, delivery, evidence, toxicity, patient selection and economics rather than treating results from unrelated trials as a head-to-head contest. Together, the two drugs show how HIV therapy evolved from urgent viral-enzyme inhibition toward more precise but more infrastructure-dependent treatment.

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