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Transmitted Drug Resistance Among People Living With HIV-1 in Henan Province, China, 2025

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Abstract

Background

Transmitted drug resistance (TDR) among newly diagnosed people living with HIV-1 (PLWH) has seen an upward trend globally, and its detection before the initiation of antiretroviral therapy (ART) is vital for treatment efficacy.

Methods

Newly diagnosed PLWH receiving routine genotypic drug resistance testing from January 2025 to December 2025 in Henan Province were included for TDR analysis. The partial pol and the complete integrase gene coding sequences were separately amplified, sequenced, and analyzed.

Results

Of a total of 1,286 patients, partial pol sequences and integrase sequences were obtained from 1,253 patients. Among these, CRF07_BC (46.61%, 584/1,253) was the dominant subtype, followed by CRF01_AE (25.54%, 320/1,253), B (17.40%, 218/1,253), CRF55_01B (4.87%, 61/1,253), and CRF08_BC (2.23%, 28/1,253). TDR was detected in 175 patients (13.97%), and the prevalence of low-level or above drug resistance against nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), and integrase strand transfer inhibitors (INSTIs) was 1.44% (18/1,253), 8.94% (112/1,253), 3.83% (48/1,253), and 0.40% (5/1,253), respectively. The prevalence of TDR for CRF55_01B increased by approximately 10% compared with the previous year, and transmission network analysis showed no evidence of cluster-based transmissions.

Conclusions

The overall transmitted TDR rate is showing an upward trend compared to previous years, with certain subtypes increasing rapidly, warranting special attention.

Background

Human Immunodeficiency Virus type 1 (HIV-1) remains one of the most significant global public health challenges. Since its identification in the early 1980s, HIV-1 has infected over 85 million people worldwide, leading to the Acquired Immunodeficiency Syndrome (AIDS), a condition characterized by progressive immune system collapse, opportunistic infections, and ultimately death if left untreated. Despite decades of intensive research and unprecedented scientific advances, HIV-1 infection is still an incurable disease.[1] This incurability stems from the virus's intrinsic biological nature—specifically, its ability to integrate its genetic material into the host cell genome and establish latent reservoirs in resting

CD4 + T lymphocytes and other long-lived cells.[2] These latent proviruses remain invisible to the immune system and unaffected by current antiretroviral drugs, meaning that once a person is infected, the virus persists for life. Even after years of successful viral suppression, treatment interruption almost invariably leads to rapid viral rebound from these reservoirs.[3,4] Consequently, the primary goal of HIV management has shifted from eradication to long-term viral suppression.

The advent of combination antiretroviral therapy (ART) in the mid-1990s revolutionized HIV care. ART, typically consisting of two or three drugs from different classes such as nucleoside reverse transcriptase inhibitors (NRTIs), non-nucleoside reverse transcriptase inhibitors (NNRTIs), protease inhibitors (PIs), and integrase strand transfer inhibitors (INSTIs), effectively blocks viral replication. By reducing plasma viral load (VL) to undetectable levels, ART restores immune function, prevents disease progression to AIDS, and dramatically reduces the risk of both HIV-related and non-HIV-related morbidity and mortality. Indeed, people living with HIV (PLWH) with early initiation of treatment can expect an increased life expectancy.[5] However, the long-term success of ART is threatened by the emergence and spread of drug-resistant HIV strains.[6] Among the most concerning phenomena in this regard is transmitted drug resistance (TDR)—the presence of drug resistance-associated mutations in the virus of an individual who has never received ART. TDR occurs when a person is initially infected with a viral strain that already carries resistance mutations from a treatment-experienced individual. As ART coverage expands globally, the prevalence of TDR among PLWH has seen an upward trend, transforming TDR from a clinical management issue into a significant public health threat that can undermine first-line treatment regimens from the very start of therapy.[7–9]

While early studies in resource-rich settings reported TDR prevalence of 5–10% in the early 2000s, more recent surveillance from various regions, and low- and middle-income countries in particular, has shown a steady increase.[8,10] The World Health Organization (WHO) has warned that pretreatment drug resistance (PDR), which includes TDR and prior ART exposure, is exceeding 10% for NNRTIs in many African and Asian countries, reaching levels where the efficacy of standard first-line regimens becomes compromised. In China, the reported TDR prevalence was 4.36%, with variations among regions.[9] In Henan Province, systemic TDR surveillance has been initiated since 2018, and it has shown a year-by-year increase, reaching 11.25% in 2024.[8] Routine monitoring and analysis of TDR can be important for the implementation of both clinical and prevention and control measures. To this end, we retrospectively analyzed TDR in Henan Province in 2025 and our results show gradual increase of TDR prevalence and significant increase for that of subtype CRF55_01B.

Materials and methods

Study population

Newly diagnosed PLWH from Jan 2025 to December 2025 without history of ART use prior to the date of sample collection were included. Demographic data and medical records including VL, CD4 + T cell

count, and transmission route were collected. The study was conducted in accordance with the Declaration of Helsinki and all participants gave their written informed consent before sample collection.

Genotypic drug resistance testing

Genotypic drug resistance testing was performed as described previously, with viral RNA extracted from plasma samples, followed by reverse transcription-PCR amplification of the partial pol gene and the full-length integrase gene.[11] The resulting sequences were submitted to the Stanford HIV-1 drug resistance database (http://hivdb.stanford.edu/) for DRM identification and susceptibility interpretation. This online system operates on a penalty-score based algorithm: each recognized DRM contributes a predefined score, and the total cumulative score for a given sequence determines the predicted resistance level for each antiretroviral drug. The resistance categories are defined as: susceptible (0–9), potential low-level (10–14), low-level (15–29), intermediate (30–59), and high-level (≥ 60). For the purpose of this analysis, only individual DRMs with a penalty score of 15 or higher were counted as major resistance mutations.

Subtyping and phylogenetic analysis

HIV subtype was firstly analyzed using REGA HIV-1 Subtyping Tool (http://dbpartners.stanford.edu:8080/RegaSubtyping/stanford-hiv/typingtool/) based on the partial *pol* region and subsequently confirmed by phylogenetic analysis using Molecular Evolutionary Genetic Analysis (MEGA) software (version X) based on neighbor-joining method and Tamura-Nei model with 1000 bootstrap replicates.

Transmission network analysis

Transmission clusters were determined as described previously.[12] Partial *pol* gene sequences of CRF55_01B were aligned using MEGA software (version XI). The aligned sequences were used to calculate genetic distances between sequence pairs based on the Tamura-Nei 93 model incorporated in HyPhy 2.2.4. A genetic distance of ≤ 1.5% between the sequence pairs indicated that they were transmission partners.[13,14] The transmission network was constructed using Cytoscape software (version 3.5.1) for visualization. The network was laid out using the Prefuse Force Directed Layout algorithm to reduce line crossing and optimize visual clarity, with nodes uniformly represented as ellipses for standardized presentation.

Statistical analysis

Statistical analysis was carried out using the SPSS statistics program (version 20) for Windows (SPSS, Inc., US). Continuous variables were expressed as mean ± standard deviation if normal or a median with its interquantile range (1st quartile–3rd quartile). Categorical variables were expressed as numbers or

percentages. The differences between or among groups were analyzed using Student's t test or Chi-square test, and a $P < 0.05$ was considered significant.

Results

Demographic and clinical characteristics

1,253 PLWH with partial *pol* and/or integrase genes successfully sequenced were included in this study. The demographic and clinical characteristics are as follows: the median age was 42 years (range: 6–77 years; IQR: 29–56). There was a predominance of male patients, who accounted for 84.20% (1,055/1,253). The majority of participants (98.56%, 1,235/1,253) were of Han ethnicity. Most participants (95.53%) were residents of Henan Province, while the remaining 4.47% came from neighboring provinces. Nearly half (47.96%) of the participants were married. Workers constituted the largest group (81.48%, 1,021/1,253), followed by non-workers (12.45%, 156/1,253) and students (5.75%, 72/1,253). Heterosexual contact (HSX) was the most common transmission category (54.35%, 681/1,253), followed by men who have sex with men (MSM; 43.42%, 544/1,253) and others/unknown (2.23%, 28/1,253). The median baseline CD4 + T cell count was 191 cells/mm³ (IQR: 57.5–322.75), and the median baseline viral load (VL) was 81,604 copies/mL (IQR: 18,832–317,176). Complete demographic details of the participants are presented in Table 1.

Co-circulation of multiple subtypes

Of the 1,253 patients with successful *pol* sequencing, the results showed that CRF07_BC (46.61%, 584/1,253) was the most prevalent, followed by CRF01_AE (25.54%, 320/1,253), subtype B (17.40%, 218/1,253), CRF55_01B (4.87%, 61/1,253), CRF08_BC (2.23%, 28/1,253), C (1.28%, 16/1,253), and others (2.08%, 26/1,253) (Table 2).

Prevalence of TDRMs

Among the 1,253 patients, TDRMs (drug-resistance mutations associated with a penalty score of 15 or higher) that confer low- or higher-level resistance to any drug class were detected in 220 (17.56%). When stratified by drug class, the prevalence of TDRMs was 5.59% (70/1,253) for NRTIs, 8.94% (112/1,253) for NNRTIs, 3.83% (48/1,253) for PIs, and 0.32% (4/1,253) for INSTIs. The most frequent TDRMs were S68G (55, 4.39%) for NRTIs, K103S/K/N (54, 4.31%) for NNRTIs, Q58E (37, 2.95%) for PIs, and G163K (2, 0.16%) for INSTIs (Table 3).

Prevalence of actual drug resistance (low-Level or higher based on total penalty score)

When translating these TDRMs into predicted drug susceptibility using the Stanford HIVdb algorithm, the proportions of patients showing low-level or higher resistance to at least one drug within each class

were as follows: 1.44% (18/1,253) for NRTIs, 8.94% (112/1,253) for NNRTIs, 3.83% (48/1,253) for PIs, and 0.40% (5/1,253) for INSTIs. Of the 5 INSTI-resistant cases, 4 had individual TDRMs with a penalty score ≥ 15, while 1 case had two accessory mutations (T97A and D232N) that each scored < 15 but together conferred low-level resistance to EVG and RAL. This case is counted in the actual drug resistance but not in the TDRM count. When stratified by subtype, the highest prevalence of drug resistance (low-level or above) was observed in CRF55_01B (26.23%, 16/61), followed by subtype B (19.27%, 42/218), CRF08_BC (17.86%, 5/28), CRF07_BC (12.16%, 71/584), and CRF01_AE (10.94%, 35/320) (Table 2). High-level resistance was detected for NRTIs and NNRTIs, but not for PIs or INSTIs. Specifically, high-level resistance was most frequently observed for emtricitabine (FTC) and lamivudine (3TC) (9 cases each) among NRTIs, and for nevirapine (NVP) (72 cases) among NNRTIs (Fig. 1). Chi-square test showed significant associations of drug resistance with age ($P = 0.021$), ethnicity ($P = 0.002$), education level ($P = 0.005$), and HIV subtype ($P = 0.007$), but no associations with marriage status, transmission route, occupation, baseline CD4 T cell count or baseline HIV VL ($P > 0.05$).

Transmission network characteristics of CRF55_01B

As CRF55_01B showed the highest prevalence of TDR, we performed genetic association network analysis to determine if there was evidence of large-scale cluster-based transmission. Based on the genetic distance threshold of ≤ 1.5%, we constructed a network of 61 CRF55_01B sequences, in which nodes represent individual viral sequences and edges represent potential direct genetic links (genetic distance ≤ 1.5%) (Fig. 2). The network revealed a highly fragmented structure. Only a subset of sequences formed small connected components, including the largest cluster of 6 sequences, two clusters of 3 sequences each, and one cluster of 2 sequences. Most sequences (47/61, 77.05%) did not form any detectable genetic links, indicating a lack of large-scale cluster-based transmission.

Discussion

Genotypic drug resistance testing is freely provided for all newly diagnosed PLWH in Henan Province since 2018. In this study, 1,253 newly diagnosed PLWH, accounting for approximately one-quarter of total annual new cases in Henan Provinces, were included. The overall prevalence of TDRMs was 17.56%, with an associated low-level or higher drug resistance rate of 13.97%. The prevalence of TDRM is higher than nation-wide levels, which are reported to be within the range of 4.36% to 11.4%.[9] Globally, it is also higher than those of most countries, such as South Korea (3.5%),[15] the United Kingdom (7.5%),[16] and four African countries (11%)[17], but lower than those of other countries, such as the United States (18.9)[18] and Germany (17.8%).[19] TDRMs and associated TDR were observed in 5.59% and 1.44% of NRTIs, 8.94% and 8.94% of NNRTIs, 3.83% and 3.83% of PIs, and 0.32% and 0.40% of INSTIs. The prevalence of TDR against NNRTIs, PIs and INSTIs increased when compared with those in 2024 while it decreased for NRTIs. This result is superficially contradictory to the widely used 3TC/dolutegravir (DTG) and BIC/FTC/TAF single tablet regimens, as well as high prevalence of resistance to 3TC in treatment-experienced individuals. The possible explanation for this might be due to the inferior fitness introduced by the M184V/I mutations, commonly seen in 3TC-resistant treatment-experienced individuals.[20] The

virus carrying this mutation in newly diagnosed PLWH might be out-competed by fitter viruses following several round of subsequent replications.

The circulating subtypes determined in this study in descending order were CRF07_BC, CRF01_AE, subtype B, and CRF55_01B, consistent with our previous studies and other studies in China, but different with those in other countries, signifying regional patterns and restricted circulation.[21,22] When stratified by subtype, TDR prevalence increased for all four major subtypes (namely CRF07_BC, CRF01_AE, subtype B, and CRF55_01B) compared with the previous year's figures.[8] In particular, TDR prevalence for CRF55_01B reaches 26.23%, approximately ten percentage points higher than that of last year. Although this subtype exhibited the highest level of drug resistance among all subtypes, transmission network analysis revealed that only limited number of CRF55_01B isolates formed clusters, with the largest comprising only six sequences. This fragmented pattern argues against an ongoing outbreak driven by a single resistant lineage. Most sequences existed as isolated nodes, with only a small proportion forming small clusters including one cluster of six sequences, two clusters of three sequences, and one cluster of two sequences. No large-scale transmission clusters were observed. Alternatively, the relatively small number of CRF55_01B cases and their sparse clustering suggest that while this subtype is prone to accumulating resistance, it does not readily establish self-sustaining transmission networks. Continued monitoring of CRF55_01B and targeted intervention among high-risk populations are needed to prevent future cluster formation.

The 3TC/DTG or BIC/FTC/TAF single-tablet regimens are being increasingly used as first-line or switch therapy due to its favorable safety profile, high genetic barrier, and reduced drug burden. Although the prevalence of TDRMs against NRTI was 5.59% (70/1,253), high-level resistance to 3TC or FTC was observed in only 9 cases (0.72%), and the overall prevalence of low- or higher-level resistance to any NRTI was just 1.44% (18/1,253). This low background of 3TC or FTC resistance suggests that both drugs would retain activity in the vast majority of patients. Furthermore, no high-level resistance to INSTIs was observed. Our results provide solid data supporting the potential efficacy of DTG- or BIC-based regiments in this cohort of PLWH in Henan Province, China.

Our study has several limitations. First, for some patients from other cities, the blood sampling time for VL monitoring and genotypic drug resistance testing may differ. Thus, the VL value and TDR profiles are not strictly correlated and which subsequently affects the correlation analysis results. Second, the sequencing is still based on Sanger sequencing and low-frequency variants may not be readily detected. Third, only about one-quarter of the annual newly diagnosed PLWH in Henan Province received genotypic drug resistance testing, the results presented in this study may have not reflected the true TDR profiles.

In summary, the yearly updated data among newly diagnosed PLWH in Henan Province indicate gradual increase of overall TDR prevalence, supporting the necessity of continued genotypic drug resistance testing before treatment initiation. However, the large proportion of PLWH without genotypic drug resistance results before their initiation of ART under the increasingly widely used highly effective INSTI-

based regimens warrants further evaluation of these two groups to provide better strategies for drug resistance monitoring and treatment regimens selection.

Abbreviations

HIV-1
Human Immunodeficiency Virus type 1
PLWH
people living with HIV
ART
antiretroviral therapy
TDR
transmitted drug resistance
NRTI
nucleoside reverse transcriptase inhibitor
NNRTI
non-nucleoside reverse transcriptase inhibitor
PI
protease inhibitor
INSTI
integrase strand transfer inhibitor
TDRM
transmitted drug resistance mutation
VL
viral load
HSX
heterosexual contact
MSM
men who have sex with men.

Declarations

Ethics approval and consent to participate

This study was approved by the Institutional Ethics Committee of The Sixth People's Hospital of Zhengzhou, China (approval numbers: IEC-KY-2024-39). Written informed consent was obtained from all participants before blood sample collection.

Consent for publication

Not applicable.

Competing interests

The authors declare no competing financial interest.

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Author Contribution

Yuqi Huo and Qingxia Zhao conceived and designed the study. Jinjin Liu and Shuguang Wei, performed experiments. Xuan Yang, Mingjie Hou, Xin Deng, Xiaohua Zhang, and Yuanyuan Chen collected clinical samples. Jinjin Liu and Chaofeng Li collected the clinical information. Jinjin Liu analyzed the data. Jinjin Liu prepared the manuscript. Yuqi Huo revised the manuscript.

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Data Availability

The datasets generated and/or analyzed during the current study are available from the corresponding author on reasonable request.

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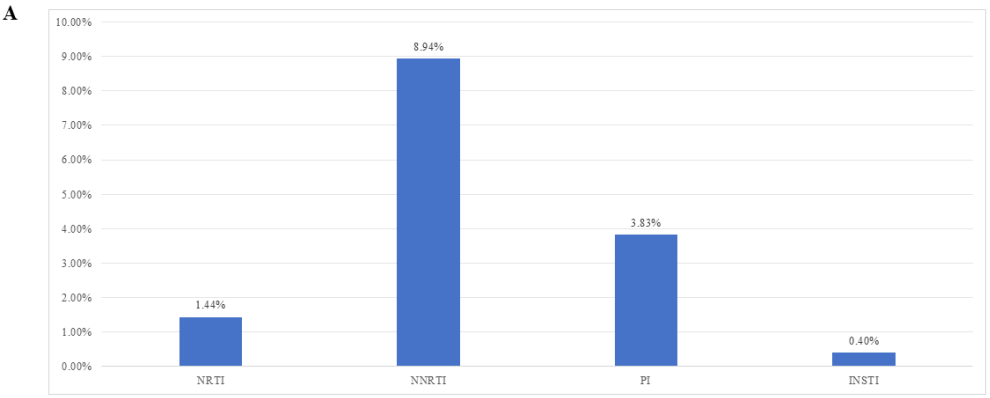
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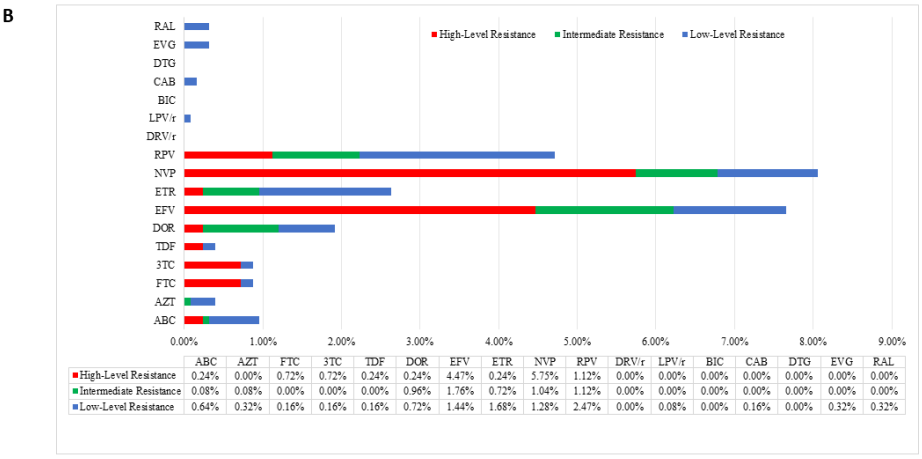
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Tables

Table 1 to 3 are available in the Supplementary Files section.

Figures





	ABC	AZT	FTC	3TC	TDF	DOR	EFV	ETR	NVP	RPV	DRV/r	LPV/r	BIC	CAB	DTG	EVG	RAL
High-Level Resistance	0.24%	0.00%	0.72%	0.72%	0.24%	0.24%	4.47%	0.24%	5.75%	1.12%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
Intermediate Resistance	0.08%	0.08%	0.00%	0.00%	0.00%	0.96%	1.76%	0.72%	1.04%	1.12%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%	0.00%
Low-Level Resistance	0.64%	0.32%	0.16%	0.16%	0.16%	0.72%	1.44%	1.68%	1.28%	2.47%	0.00%	0.08%	0.00%	0.16%	0.00%	0.32%	0.32%

Figure 1. Predicted antiretroviral drug resistance profiles among newly diagnosed PLWH in Henan Province, 2025.

(A) Prevalence of resistance by drug class (NNRTI, NRTI, PI, and INSTI).

(B) Prevalence of resistance to individual antiretroviral drugs: ABC, abacavir; AZT, zidovudine; FTC, emtricitabine; 3TC, lamivudine; TDF, tenofovir; DOR, doravirine; EFV, efavirenz; ETR, etravirine ; NVP, nevirapine; RPV, rilpivirine; DRV/r, darunavir/r; LPV/r, lopinavir/r; BIC, bictegravir; CAB, cabotegravir; DTG, dolutegravir; EVG, elvitegravir; RAL, raltegravir.

Figure 1

See image above for figure legend.

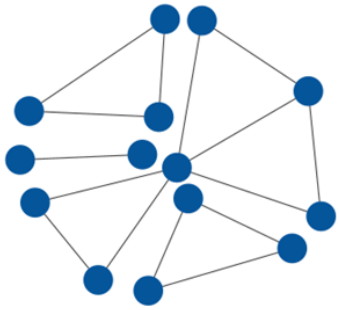


Figure 2. Transmission Network Analysis of HIV-1 CRF55_01B Strains in Henan Province, 2025. Nodes represent individual viral sequences; edges represent genetic distance ≤1.5%.

Figure 2

See image above for figure legend.

Supplementary Files

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- Tables.pptx