Global and regional molecular epidemiology of HIV-1 during 1990–2024: systematic review, global survey, and analysis of prevalence

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Summary

Background The extensive genetic diversity of HIV presents major challenges to treatment and prevention. We aimed to estimate the global and regional distribution of HIV-1 subtypes and recombinants during 1990–2024.

Methods We conducted a systematic literature review by searching PubMed, Embase, Global Health, and CINAHL for country-specific HIV-1 subtyping data published between Jan 1, 2022, and Jan 22, 2025, and a global survey of the Global HIV Molecular Epidemiology Collaboration for unpublished data collected between 2016 and 2024. We included primary HIV-1 subtyping data with ≥20 samples and known country and years of sample collection during 1990–2024. We excluded publications and survey responses that had no or incomplete subtyping data, were restricted to specific HIV-1 variants, included superinfections, or used secondary data. These data were combined with HIV-1 subtyping data previously collected between 1990 and 2021. Data were aggregated by country for six time periods (1990–99, 2000–04, 2005–09, 2010–14, 2015–19, and 2020–24). Proportions of HIV-1 subtypes, circulating recombinant forms (CRFs), and unique recombinant forms (URFs) were calculated by country and period and were weighted using UNAIDS country estimates of numbers of people living with HIV to estimate regional and global HIV-1 variant proportions. The systematic review is registered with PROSPERO, CRD42017067164.

Findings HIV-1 subtyping data were available for 1 395 222 samples from 154 countries during 1990–2024. In 2020–24, subtype C accounted for 48·7% (95% CI 48·3–49·1) of global HIV-1 infections, followed by subtype A (11·5%; 10·9–12·1), subtype B (10·3%; 10·0–10·5) URFs (5·3%; 4·4–6·3), CRF02_AG (5·1%; 4·5–5·8), CRF01_AE (5·1%; 4·8–5·4), other CRFs (3·9%; 3·3–4·5), subtype G (3·1%; 2·1–4·1), subtype D (3·0%; 2·7–3·3), and CRF07_BC (2·1%; 2·0–2·1). Subtypes F, H, J, K, and L combined accounted for 1·1% of infections and unspecified recombinants for 0·9% (0·7–1·0). HIV-1 variants are differentially distributed across regions, with subtype C dominating in southern Africa; Ethiopia, Eritrea, and Djibouti; and south Asia; subtype A in east Africa and eastern Europe and central Asia; subtype B in North America, Latin America, and western and central Europe; CRF01_AE in southeast Asia; and CRF07_BC in east Asia. Central Africa exhibited the greatest HIV-1 diversity. Global HIV-1 variant distributions were broadly stable during 2000–24, but notable regional changes included increases of HIV-1 recombinants in western and central Europe and of CRF07_BC in east Asia.

Interpretation Global and regional HIV-1 genetic diversity is complex and evolving, affecting the efficacy of diagnostic and viral load assays, emergence of drug resistance, and vaccine development. Continued surveillance of spatiotemporal trends in HIV-1 genetic diversity is essential.

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Introduction

In 2024, an estimated 40·8 million people were living with HIV.[1] The advent of antiretroviral therapy (ART) has been instrumental in improving life expectancy and reducing HIV transmission.[1] The number of AIDS-related deaths fell by 54% to 630 000 from 2010 to 2024, and new HIV infections by 40% to 1·3 million.[1] However, in early 2025, five countries providing over 90% of international funding to combat HIV announced reductions in aid.[2] Funding disruptions could result in as many as 3 million excess AIDS-related deaths and 11 million excess new HIV infections by 2030.[2] Gains made in the global fight against HIV are fragile, and an HIV vaccine, affording long-lasting, universal protection, is probably needed to end the HIV pandemic.[3]

The extensive global genetic diversity of HIV challenges efforts to control the HIV pandemic. Since the virus was first transmitted from chimpanzees to humans in Central Africa in the early 1900s, the pandemic HIV-1 main (M) group has diversified into ten subtypes (A–D, F–H, and J–L).[4] The amino acid difference in the envelope glycoproteins is estimated at 17% (range 4–30%) within

Research in context

Evidence before this study

The extensive global genetic diversity of HIV-1 presents a major challenge to treatment and prevention efforts. Up-to-date knowledge of the distribution of HIV-1 subtypes and recombinants worldwide is crucial for developing HIV vaccines and ensuring viral assays and antiretroviral treatments are effective. However, sources of data on global HIV-1 genetic diversity are scarce. HIV sequence databases containing information on sequenced HIV-1 samples deposited by researchers might not be representative of populations. Global and regional HIV-1 variant distributions have been previously described for 1990 to 2015 and 2010 to 2021, but recent estimates are not available. We aimed to estimate global and regional HIV-1 variant distributions during 1990–2024 using published and unpublished data. We searched PubMed, Embase, Global Health, and CINAHL for studies containing country-specific HIV-1 subtyping data published between Jan 1, 2022, and Jan 22, 2025. Search terms included "HIV", "subtypes", "recombinants", and their synonyms as subject headings and free text words. No exclusions were applied for language or study type. We also conducted a global survey to collect unpublished data for 2016 to 2024 from researchers working on HIV molecular epidemiology worldwide.

Added value of this study

After combining with previously collected data from 1990 to 2021, we assembled a database of nearly 1·4 million subtyped HIV-1 samples from 154 countries collected during 1990–2024. We combined this country-specific HIV-1 subtyping data with UNAIDS estimates of the numbers of people living with HIV in each country to estimate the global and regional distribution of HIV-1 subtypes and recombinants over the past 35 years. To our knowledge, this is the largest and most up-to-date study of the global distribution of HIV-1 variants over time. Our study included all published data available since 1990, supplemented with unpublished data for recent years and for countries with limited published data.

Implications of all the available evidence

We found that the global distribution of HIV-1 variants remained broadly stable over the study period, but regional differences persisted, and changes in regional distributions over time were observed. Changes could reflect differences in the biological properties of HIV-1 variants; coverage and effectiveness of treatment and prevention interventions; and the social, political, economic, and environmental contexts of the populations in which different HIV-1 variants spread. Ongoing global and regional surveillance is essential for effective testing and treatment of HIV. Changing HIV-1 variant distributions might challenge diagnostic and viral load assays, and drug resistance in less common HIV-1 variants remains understudied. Knowledge of regional HIV-1 variant distributions is crucial for the development of an HIV vaccine.

HIV-1 subtypes and at 25% (range 20–36%) between subtypes.[5] Over time, population movement introduced different HIV-1 variants to different parts of the world. For instance, subtype B spread from central Africa to Haiti in the 1960s, to the USA in the 1970s, and then to Europe and Oceania.[6] Along the way, HIV-1 continued to diversify as an error-prone reverse transcriptase and high replication rates introduced mutations, and co-infection with multiple strains resulted in recombinant strains.[7] Recombinants are classified as circulating recombinant forms (CRFs) if found in at least three epidemiologically unrelated people and as unique recombinant forms (URFs) otherwise.[8] A total of 168 CRFs have been identified to date.[9]

An effective HIV vaccine must account for the genetic diversity of HIV-1. For comparison, influenza virus requires an updated variant-specific vaccine annually, although the global diversity of influenza virus sequences in a single year is comparable to the diversity of HIV sequences in a single individual at a single time point (up to 10%).[5] Mosaic HIV vaccines have not yet demonstrated efficacy.[10] Vaccines targeting specific HIV-1 variants and populations, alongside surveillance of circulating HIV-1 variants, are probably needed.[11]

HIV-1 genetic diversity also affects HIV diagnosis and treatment. Recombinant forms can challenge the efficiency of diagnostic and viral load assays.[12] HIV subtypes and recombinants can differ in rates of disease progression and transmission.[13] Selective pressure from antiretroviral drugs can lead to the emergence of drug resistance mutations, which can cause treatment failure.[14]

For effective HIV treatment and vaccine development, understanding the global genetic diversity of HIV is essential. Studies show that the global and regional distribution of HIV-1 variants is continuing to evolve;[15–20] yet, sources of up-to-date data on global HIV genetic diversity are scarce. Sequence databases, including the Los Alamos National Laboratory (LANL) HIV Sequence Database[9] and the Stanford HIV Drug Resistance Database,[21] contain sequencing information from HIV-1 samples often collected by researchers for specific purposes and are probably not representative of populations, limiting their suitability for epidemiological study.[22] We aimed to assemble published and unpublished HIV-1 subtyping data collected worldwide to estimate the global and regional distribution of HIV-1 subtypes and recombinants during 1990–2024.

Methods

Systematic review

We searched PubMed, Embase (Ovid), Global Health (Ovid), and CINAHL (EBSCOHost) for studies containing

HIV subtyping data published between Jan 1, 2022 and Jan 22, 2025 (appendix 1 pp 2–5). Search terms included "HIV", "subtypes", "recombinants", and their synonyms as subject headings and free text words (appendix 1 pp 2–5). No exclusions were applied for language or study type. Search results were screened and duplicate references were removed using Covidence software (Veritas Health Innovation).[23] Screening, full-text review, and data extraction were each conducted by at least two independent reviewers (AK, LG, LS, XL, and LLLW) and discrepancies were resolved through discussion with the senior author (JH). This systematic review was registered with PROSPERO (CRD42017067164) and follows PRISMA guidelines.[24]

Additional sources were manually searched to collect published HIV-1 subtyping data not found through the electronic literature search, including tables of contents of specialist journals (*AIDS*; *Journal of Virology*; *AIDS Research and Human Retroviruses*; *Infection, Genetics and Evolution*; *Clinical Infectious Diseases*; and *Journal of AIDS*) between Jan 1, 2000, and May 31, 2025, references of review articles on HIV genetic diversity, and studies indexed on Scopus which referenced previous related publications.[15–20]

Global survey

A survey was developed to gather unpublished subtyping data for HIV-1 samples collected for surveillance or research purposes between 2016 and 2024 by researchers working on HIV molecular epidemiology worldwide. The survey was piloted with 14 researchers before being distributed to 1071 researchers between Feb 24 and March 3, 2025. Contributing researchers consented to the use of their data for this study and are listed as coauthors. Researchers included members of the Global HIV Molecular Epidemiology Collaboration and corresponding authors of relevant publications found through the current and previous systematic reviews.[16,17,20] The Global HIV Molecular Epidemiology Collaboration (previously the WHO–UNAIDS Network for HIV Isolation and Characterisation) is a network of researchers focused on the surveillance of HIV genetic diversity worldwide.

Eligibility criteria

The same eligibility criteria were applied to publications and survey responses. We included publications and survey responses that contained primary HIV-1 subtyping data for ≥20 samples with known country and years of sample collection during the 1990–2024 period. Any study purpose (eg, genetic diversity and drug resistance), study design (eg, cross-sectional and cohort), infection status (ie, incident, prevalent, and newly diagnosed), and key population (eg, men who have sex with men and heterosexual) were eligible. We excluded publications and survey responses that had no or incomplete subtyping data (eg, provided an aggregated category of some variants), were restricted to specific HIV-1 variants (eg, only non-subtype B included), included superinfections, or used secondary data (eg, review articles or data from the LANL HIV Sequence Database).

See **Online** for appendix 1

Data extraction

Study characteristics; locations and years of sample collection; study methods; participant characteristics; sequencing and subtyping methods; and the number of samples of each HIV-1 subtype, CRF, and URF were extracted. We extracted subtyping assignments as were reported in publications or shared by survey respondents, as we did not have access to the genetic sequences of collected samples. If recombinants were not reported as CRFs or URFs, they were included as unspecified recombinants. In cases where HIV-1 subtyping results were available from sequencing of multiple gene regions, we prioritised subtyping assignments by full-length sequencing. Discordant results were classified as recombinants. No patient identifiable data were retrieved. Data quality was assessed using a modified version of a risk of bias tool for prevalence studies (appendix 1 p 6).[25]

Data processing

The collected data were combined with a database of previously collected published (1990–2021) and unpublished (1990–2015) HIV-1 subtyping data to create a global HIV-1 molecular epidemiology database of subtyped HIV-1 samples collected between 1990 and 2024.[15–17,20] For each country, we manually compared the location and years of sample collection, as well as contributors and HIV-1 variant distributions, for all records to identify and remove duplicate data. In cases of overlapping data, the largest and most complete record was retained.

Data analysis

Each country was assigned to one of 15 geographical regions based on UNAIDS classifications,[1] modified according to HIV-1 variant distributions (appendix 1 p 7). Taiwan and Hong Kong were grouped into China as per UNAIDS. For records that spanned multiple years of sample collection, HIV-1 subtyping data were split equally across all years. Each year-record was assigned to one of six time periods (1990–99, 2000–04, 2005–09, 2010–14, 2015–19, and 2020–24). All data processing and analyses were done using R (version 4.4.1).

UNAIDS annual country-level estimates of the numbers of people living with HIV[26] were used to calculate the mean number of people living with HIV in each country and time period. These means were summed to obtain regional and global numbers. Breadth of coverage was calculated as the number of people living with HIV in countries for which HIV-1 subtyping data were available in a region or globally as a proportion of the total number of people living with HIV in that region or globally. Depth of coverage was calculated as the

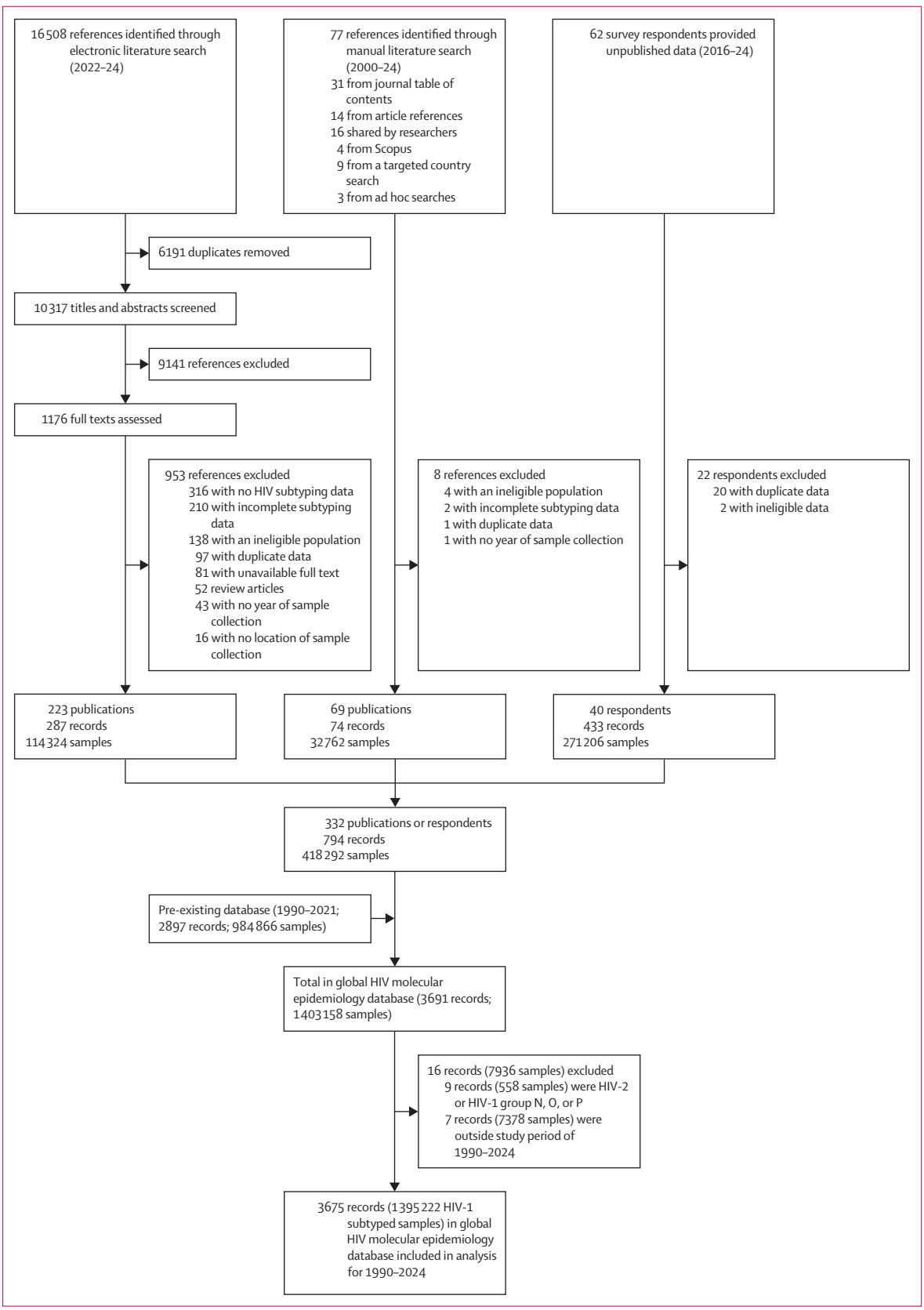

Figure 1: **Flow diagram of data collection and database assembly**

number of subtyped HIV-1 samples in a region or globally as a proportion of the total number of people living with HIV in that region or globally.

To calculate country-level HIV-1 variant proportions, the numbers of samples of each subtype, CRF, and URF were aggregated by each country and period and divided by the total number of samples in each country and period. These country-specific HIV-1 variant proportions were multiplied by the UNAIDS estimates of the total number of people living with HIV in each country to estimate the numbers of people living with HIV with each HIV-1 variant by country and period. To calculate regional weighted HIV-1 variant proportions, the sum of the estimated numbers of people living with HIV with each HIV-1 variant across countries in each region was divided by the total number of people living with HIV residing in countries with HIV-1 subtyping data in that region. Regional variant proportions were multiplied by UNAIDS estimates of the total number of people living with HIV in each region, including countries without available subtyping data, to obtain estimates of the numbers of people living with HIV with each HIV-1 variant in each region. Global HIV-1 variant proportions were calculated as the sum of the estimated numbers of people living with HIV with each HIV-1 variant across regions, divided by the UNAIDS estimates of the global number of people living with HIV. For regional and global proportions, 95% CIs were calculated using the normal approximation to the binomial distribution, with variances estimated using Taylor linearisation for linear combinations.

Three sensitivity analyses on restricted datasets were conducted: including samples from prevalent infections only, as incident or newly diagnosed infections might have different HIV-1 variant distributions; including samples subtyped based on sequencing of the *pol* gene only, as classification of variants can vary based on the genes analysed; and including samples from data sources assessed as low or medium risk of bias only. Three additional sensitivity analyses were conducted on modified datasets: allocating samples to the middle year for records spanning multiple years of collection; weighting each record by its risk of bias score before calculating country-level HIV-1 variant proportions; and bootstrapping at the country-level (resampling countries within regions with replacement) to estimate region-level variant proportions (1000 iterations).

Role of the funding source

The funder of the study had no role in study design, data collection, data analysis, data interpretation, or writing of the report.

Results

The electronic literature search yielded HIV-1 subtyping data from 223 publications, and the manual literature search yielded data from 69 publications (figure 1). Eligible unpublished data were shared by 40 survey respondents. Altogether, published and unpublished HIV-1 subtyping data were collected for 418 292 samples. After combining with previously collected HIV-1 subtyping data and excluding non-HIV-1 group M samples and samples collected outside the study years 1990–2024, we assembled a global HIV-1 molecular epidemiology database of 1 395 222 subtyped HIV-1 samples for the period 1990–2024 (figure 1). A table summarising our database by country, time period, and publication status is provided in appendix 1 (pp 8–11).

HIV-1 subtyping data were mostly collected through systematic literature reviews, especially in earlier time periods (figure 2A). In 2020–24, nearly two-thirds of data were unpublished data collected through the survey (65·1%), probably due to the lag time between sample collection and publication (figure 2A). For each period, samples from western and central Europe and North America comprised about half of all samples (figure 2B). Since 2000, more than 93·9% of samples were genotyped using sequencing and over 76·7% genotyped the *pol* gene fragment (appendix 1 pp 12, 18–21). The most commonly used subtyping tools used were the REGA HIV-1 subtyping tool, the Stanford HIV Drug Resistance Database subtyping tool, and phylogenetic analysis (appendix 1 pp 12, 14–17). Nearly three-quarters (74·5%) of samples were from prevalent HIV-1 infections (appendix 1 p 12). Overall, almost all (94·6%) samples were from data sources assessed as low or medium risk of bias, with more published sources assessed as low risk than unpublished sources (appendix 1 p 13).

HIV-1 subtyping data were available from 154 countries during 1990–2024, covering countries harbouring 88·6–97·6% of the global population of people living with HIV in each period (table 1). Breadth of coverage exceeded 82·8% for most regions and time periods, except for Latin America and southeast Asia in 2020–24, and for the Caribbean, Oceania, and Middle East and north Africa in multiple time periods. However, the Caribbean, Oceania, and Middle East and north Africa comprised the lowest numbers of people living with HIV over time (0·1–1·0% of global infections; table 1).

Subtyped HIV-1 samples represented 0·3–1·1% of the global number of people living with HIV over time (table 1). Depth of coverage was highest in western and central Europe in each period, and in North America, Oceania, and east Asia for periods besides 1990–99 and 2020–24. Depth of coverage was lowest in regions with the highest numbers of people living with HIV, including southern, central, east, and west Africa and south Asia, as well as Ethiopia, Eritrea, and Djibouti (≤0·3% in each period).

In 2020–24, nearly half of global HIV-1 infections were subtype C (48·7% [95% CI 48·3–49·1]) and almost a quarter were recombinants (22·4% [21·3–23·5]; figure 3A; table 2; appendix 1 pp 23–26). Subtype A accounted for

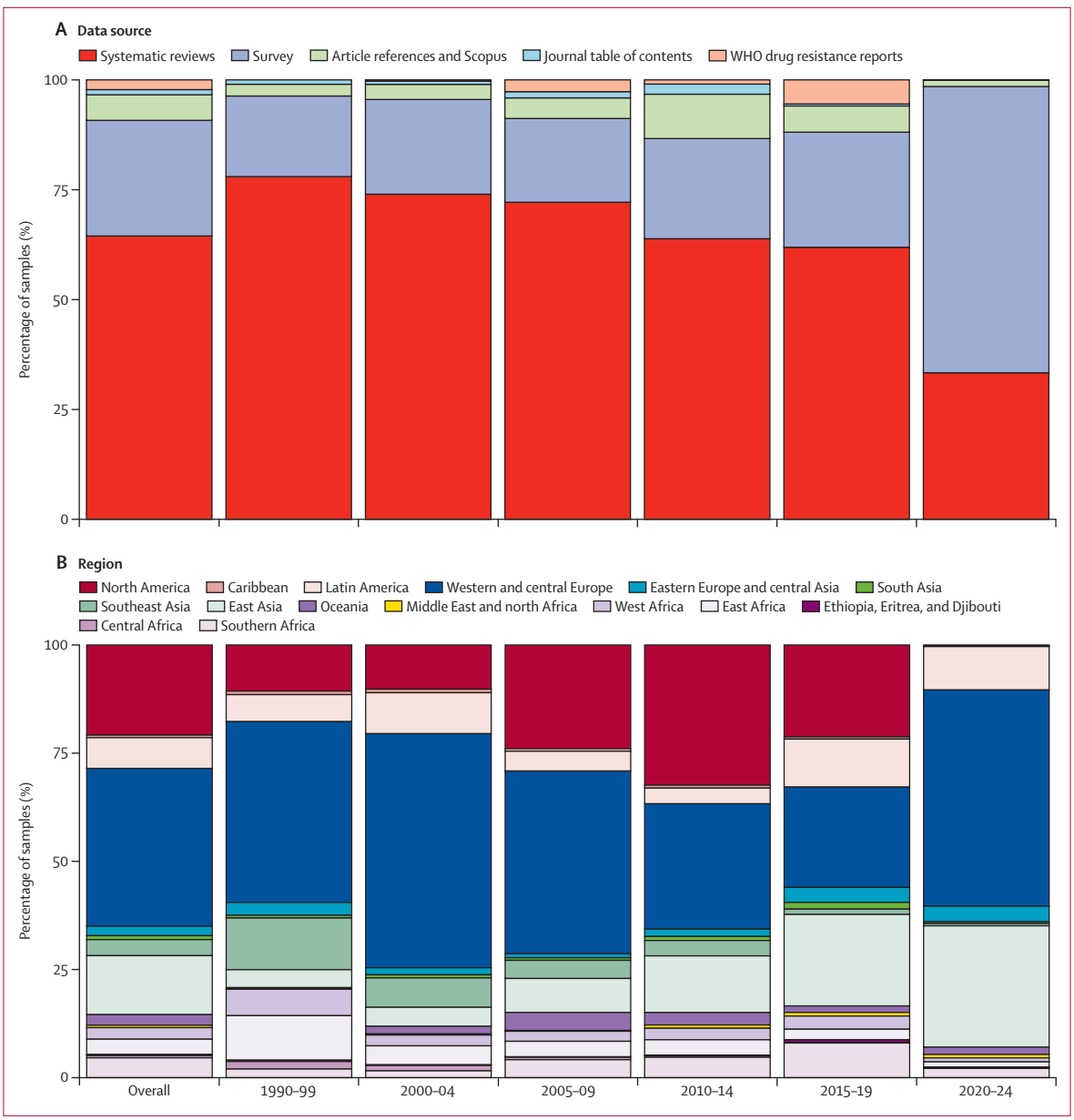

Figure 2: **Distribution of samples in the global HIV-1 molecular epidemiology database, 1990–2024**

11·5% (10·9–12·1) of infections and subtype B for 10·3% (10·0–10·5) of infections, followed by URFs (5·3% [4·3–6·3]), CRF02_AG (5·1% [4·5–5·8]), CRF01_AE (5·1% [4·8–5·4]), other CRFs (3·9% [3·3–4·5]), subtype G (3·1% [2·1–4·1]), subtype D (3·0% [2·7–3·3]), and CRF07_BC (2·1% [2·0–2·1]). Subtypes F, H, J, K, and L combined were responsible for 1·1% (appendix 1 pp 23–26) of infections and unspecified recombinants for 0·9% (0·7–1·0) of infections. Sensitivity analyses limited to prevalent infections, *pol* sequencing, and low or medium risk of bias records, as well as sensitivity analyses allocating records to time periods based on the middle year, weighting records by risk of bias scores, and bootstrapping at the country-level, all showed similar results to the primary analysis (appendix 1 p 22).

Over time, there were limited changes in the global distribution of HIV-1 variants, especially from 2000 onwards (figure 3B; table 2; appendix 1 pp 23–26). Recombinants increased from 20·8% (95% CI 20·3–21·3) in 2000–04 to 24·2% (23·9–24·5) in 2010–14, before reducing to 22·4% (21·3–23·5) by 2020–24. CRF07_BC consistently increased over time, from 0·4% (0·3–0·4) in 2000–04 to 2·1% (2·0–2·1) in 2020–24. Other CRFs increased from 2·2% (2·0–2·4) in 2000–04 to 4·9% (4·7–5·1) in 2010–14, before reducing to 3·9% (3·3–4·5) in 2020–24.

	Global	North America	Caribbean	Latin America	Western and central Europe	Eastern Europe and central Asia	South Asia	Southeast Asia	East Asia	Oceania	Middle East and north Africa	West Africa	East Africa	Ethiopia, Eritrea, and Djibouti	Central Africa	Southern Africa
Number of people living with HIV*																
1990–99	19 333 593	672 319 (3·5%)	194 396 (1·0%)	542 512 (2·8%)	360 226 (1·9%)	175 501 (0·9%)	2 516 284 (13·0%)	936 401 (4·8%)	279 009 (1·4%)	23 967 (0·1%)	38 679 (0·2%)	2 549 734 (13·2%)	3 736 795 (19·3%)	833 361 (4·3%)	1 008 056 (5·2%)	5 466 354 (28·3%)
2000–04	28 887 669	811 466 (2·8%)	257 681 (0·9%)	937 195 (3·2%)	505 416 (1·7%)	460 019 (1·6%)	3 594 516 (12·4%)	1 447 858 (5·0%)	792 368 (2·7%)	44 966 (0·2%)	89 424 (0·3%)	3 942 910 (13·6%)	4 363 243 (15·1%)	881 019 (3·0%)	1 272 474 (4·4%)	9 487 114 (32·8%)
2005–09	30 637 858	942 026 (3·1%)	261 785 (0·9%)	1 166 457 (3·8%)	633 368 (2·1%)	692 271 (2·3%)	3 094 029 (10·1%)	1 630 702 (5·3%)	1 047 633 (3·4%)	64 211 (0·2%)	135 743 (0·4%)	4 044 911 (13·2%)	4 277 217 (14·0%)	729 526 (2·4%)	1 291 977 (4·2%)	10 626 002 (34·7%)
2010–14	33 587 837	1 081 446 (3·2%)	269 692 (0·8%)	1 473 431 (4·4%)	751 585 (2·2%)	1 095 760 (3·3%)	2 834 888 (8·4%)	1 808 660 (5·4%)	1 205 346 (3·6%)	86 017 (0·3%)	163 700 (0·5%)	4 109 244 (12·2%)	4 503 032 (13·4%)	686 009 (2·0%)	1 309 759 (3·9%)	12 209 268 (36·4%)
2015–19	37 089 025	1 207 162 (3·3%)	284 497 (0·8%)	1 886 624 (5·1%)	868 303 (2·3%)	1 571 456 (4·2%)	2 788 350 (7·5%)	1 942 827 (5·2%)	1 395 594 (3·8%)	110 663 (0·3%)	187 980 (0·5%)	4 087 182 (11·0%)	4 897 323 (13·2%)	661 260 (1·8%)	1 343 904 (3·6%)	13 855 900 (37·4%)
2020–24	39 906 326	1 302 465 (3·3%)	314 590 (0·8%)	2 338 168 (5·9%)	988 228 (2·5%)	1 988 139 (5·0%)	2 919 280 (7·3%)	2 054 598 (5·1%)	1 586 470 (4·0%)	143 529 (0·4%)	235 576 (0·6%)	4 022 021 (10·1%)	5 142 356 (12·9%)	633 849 (1·6%)	1 418 607 (3·6%)	14 818 451 (37·1%)
Number of countries with HIV-1 subtyping data																
1990–99	99	2	5	13	27	9	3	7	3	1	4	10	3	1	6	5
2000–04	111	2	6	14	31	8	3	6	3	3	5	11	5	2	5	7
2005–09	117	2	7	14	30	6	3	8	4	2	4	13	6	2	7	9
2010–14	115	2	7	11	30	7	2	9	4	2	7	13	6	1	6	8
2015–19	117	2	7	14	26	10	5	6	4	2	9	11	5	2	5	9
2020–24	69	2	2	6	18	3	2	3	3	2	4	7	4	1	5	7
Number of samples																
Overall	1 395 222	290 747 (20·8%)	8233 (0·6%)	99 022 (7·1%)	508 851 (36·5%)	30 399 (2·2%)	12 764 (0·9%)	51 789 (3·7%)	190 113 (13·6%)	34 270 (2·5%)	7712 (0·6%)	37 228 (2·7%)	49 209 (3·5%)	3663 (0·3%)	7275 (0·5%)	63 947 (4·6%)
1990–99	72 869	7763 (10·7%)	599 (0·8%)	4542 (6·2%)	30 450 (41·8%)	2166 (3·0%)	458 (0·6%)	8707 (11·9%)	3016 (4·1%)	157 (0·2%)	112 (0·2%)	4455 (6·1%)	7492 (10·3%)	236 (0·3%)	1262 (1·7%)	1453 (2·0%)
2000–04	176 663	18 031 (10·2%)	1471 (0·8%)	16 717 (9·5%)	95 564 (54·1%)	2874 (1·6%)	1296 (0·7%)	11 976 (6·8%)	7762 (4·4%)	3119 (1·8%)	451 (0·3%)	4408 (2·5%)	7740 (4·4%)	285 (0·2%)	2185 (1·2%)	2784 (1·6%)
2005–09	322 925	77 349 (24·0%)	1947 (0·6%)	14 779 (4·6%)	136 328 (42·2%)	2983 (0·9%)	1958 (0·6%)	13 543 (4·2%)	25 377 (7·9%)	13 401 (4·1%)	620 (0·2%)	7497 (2·3%)	11 562 (3·6%)	424 (0·1%)	1765 (0·5%)	13 390 (4·1%)
2010–14	360 137	116 707 (32·4%)	2392 (0·7%)	13 081 (3·6%)	104 291 (29·0%)	6154 (1·7%)	3378 (0·9%)	12 761 (3·5%)	47 143 (13·1%)	10 408 (2·9%)	2696 (0·7%)	9573 (2·7%)	12 747 (3·5%)	529 (0·1%)	1329 (0·4%)	16 949 (4·7%)
2015–19	332 182	70 538 (21·2%)	1711 (0·5%)	36 814 (11·1%)	77 026 (23·2%)	11 524 (3·5%)	5148 (1·5%)	4107 (1·2%)	70 230 (21·1%)	4990 (1·5%)	2790 (0·8%)	10 107 (3·0%)	8115 (2·4%)	2121 (0·6%)	369 (0·1%)	26 593 (8·0%)
2020–24	130 447	358 (0·3%)	112 (0·1%)	13 089 (10·0%)	65 192 (50·0%)	4699 (3·6%)	527 (0·4%)	695 (0·5%)	36 585 (28·0%)	2194 (1·7%)	1043 (0·8%)	1188 (0·9%)	1554 (1·2%)	69 (0·1%)	365 (0·3%)	2777 (2·1%)
Breadth of coverage†																
1990–99	91·0%	100·0%	4·0%	91·9%	98·5%	99·0%	99·8%	99·2%	98·4%	42·6%	32·3%	89·2%	89·5%	96·1%	92·6%	87·8%
2000–04	94·9%	100·0%	55·6%	93·1%	98·9%	95·5%	99·0%	91·6%	99·1%	99·9%	44·9%	92·5%	96·7%	96·9%	85·7%	95·8%
2005–09	97·6%	100·0%	91·4%	92·4%	98·2%	91·0%	98·5%	99·5%	99·2%	27·6%	53·3%	95·4%	96·1%	97·1%	100·0%	100·0%
2010–14	96·2%	100·0%	47·9%	83·2%	97·4%	90·9%	97·9%	99·9%	99·1%	96·3%	56·8%	95·2%	98·9%	95·5%	96·1%	97·6%
2015–19	95·1%	100·0%	91·7%	86·9%	96·7%	95·7%	99·7%	94·7%	99·1%	95·7%	63·7%	83·6%	92·8%	98·3%	83·1%	100·0%
2020–24	88·6%	100·0%	27·3%	73·0%	82·8%	89·0%	97·7%	44·7%	99·0%	23·5%	20·9%	85·1%	90·5%	96·6%	84·5%	97·0%

(Table 1 continues on next page)

(Continued from previous page)

	Global	North America	Caribbean	Latin America	Western and central Europe	Eastern Europe and central Asia	South Asia	Southeast Asia	East Asia	Oceania	Middle East and north Africa	West Africa	East Africa	Ethiopia, Eritrea, and Djibouti	Central Africa	Southern Africa
Depth of coverage‡																
1990–99	0·377%	1·155%	0·308%	0·837%	8·453%	1·234%	0·018%	0·930%	1·081%	0·657%	0·289%	0·175%	0·200%	0·028%	0·125%	0·027%
2000–04	0·612%	2·222%	0·571%	1·784%	18·908%	0·625%	0·036%	0·827%	0·980%	6·937%	0·504%	0·112%	0·177%	0·032%	0·172%	0·029%
2005–09	1·054%	8·211%	0·744%	1·267%	21·524%	0·431%	0·063%	0·831%	2·422%	20·870%	0·457%	0·185%	0·270%	0·058%	0·137%	0·126%
2010–14	1·072%	10·792%	0·887%	0·888%	13·876%	0·562%	0·119%	0·706%	3·911%	12·100%	1·647%	0·233%	0·283%	0·077%	0·101%	0·139%
2015–19	0·896%	5·843%	0·601%	1·951%	8·871%	0·733%	0·185%	0·211%	5·032%	4·509%	1·484%	0·247%	0·166%	0·321%	0·027%	0·192%
2020–24	0·327%	0·028%	0·036%	0·560%	6·597%	0·236%	0·018%	0·034%	2·306%	1·529%	0·443%	0·030%	0·030%	0·011%	0·026%	0·019%

*Average numbers of people living with HIV calculated using UNAIDS annual country-level estimates. †Calculated as the number of people living with HIV in countries for which HIV-1 subtyping data were available in each region or globally divided by the total number of people living with HIV in that region or globally. ‡Calculated as the number of subtyped HIV-1 samples in each region or globally divided by the total number of people living with HIV in that region or globally.

Table 1: **Global and regional HIV-1 subtyping data collection, 1990–2024**

The proportions of HIV-1 subtypes and recombinants varied greatly between regions (figure 3C; figure 4; table 2; appendix 1 pp 23–26). Central Africa consistently had the greatest genetic diversity of HIV-1. From 2000–04 to 2020–24, subtype A decreased (32·8% [95% CI 30·6–35·0] to 16·5% [11·9–21·2]), whereas subtype C (9·7% [8·6–10·9] to 17·1% (12·0–22·2]) and CRF02_AG (5·7% [4·4–6·9] to 14·9% [11·1–18·7]) increased. Subtypes D, F, G, and H each accounted for between 3·0% and 10·8% of infections over time. Recombinants peaked at 48·6% (46·3–50·9) in 2005–09, driven mostly by increases in other CRFs and URFs, before reducing to about a third of all infections by 2020–24 (33·8% [28·3–39·2]).

In 2020–24, west Africa had the highest proportion of URFs (23·7% [95% CI 14·2–33·3]) of any region and period. CRF02_AG accounted for about half of infections between 2000–04 and 2015–19 and a third in 2020–24 (33·4% [27·5–39·3]). Approximately one-fifth of infections were subtype G in each period (25·0% [15·3–34·7]) in 2020–24) and, since 2000, about 5% were subtype A (5·8% in 2020–24 [1·0–10·6]).

Subtype A accounted for about half of infections in east Africa in 2020–24 (44·7% [95% CI 42·1–47·4]), similar to previous periods. Also similar to previous periods, subtype D comprised 20·5% (18·6–22·3) of infections in 2020–24, subtype C comprised 19·0% (16·9–21·1) of infections, and recombinants comprised 14·9% (12·9–16·9%) of infections. URFs (7·9% [6·3–9·5]) and other CRFs (5·2% [4·3–6·1]) were the key recombinants in 2020–24.

Subtype C infections dominated in southern Africa (99·5% [95% CI 99·2–99·8]); Ethiopia, Eritrea, and Djibouti (97·1% [93·1–100·0]); and south Asia (87·4% [85·3–89·6]) in 2020–24, consistent with previous periods. In south Asia, 3·1% (2·0–4·3) of infections in 2020–24 were subtype A, and CRF02_AG increased from 0·0% (0·0–0·0) in 2000–04 to 6·1% (5·2–7·1) in 2020–24.

Most infections in the Middle East and north Africa in 2020–24 were subtype A (56·3% [95% CI 45·3–67·3]), followed by other CRFs (31·8% [20·8–42·8]), predominantly CRF35_AD (data not shown). Subtype B decreased from 45·2% (38·1–52·3) in 2000–04 to 8·1% (7·4–8·8) in 2020–24.

Subtype B dominated in North America (86·4% [95% CI 82·6–90·1]) and Latin America (79·1% [76·9–81·2]) in 2020–24. In North America, recombinants increased from 2·1% (1·9–2·3) in 2000–04 to 11·4% (7·9–15·0) in 2020–24, mainly due to URFs (5·6% [3·0–8·2]) and CRF02_AG (5·0% [2·6–7·5]). In Latin America, subtype C increased from 4·6% (4·3–4·8) in 2000–04 to 6·9% (5·4–8·4) in 2020–24, whereas subtype F ranged from 2·6% (2·4–2·8) to 5·6% (4·2–6·9) of infections over time. URFs increased from 3·7% (3·4–3·9) in 2000–04 to 8·5% (8·2–8·9) in 2015–19, before reducing to 4·4% (3·4–5·5) in 2020–24.

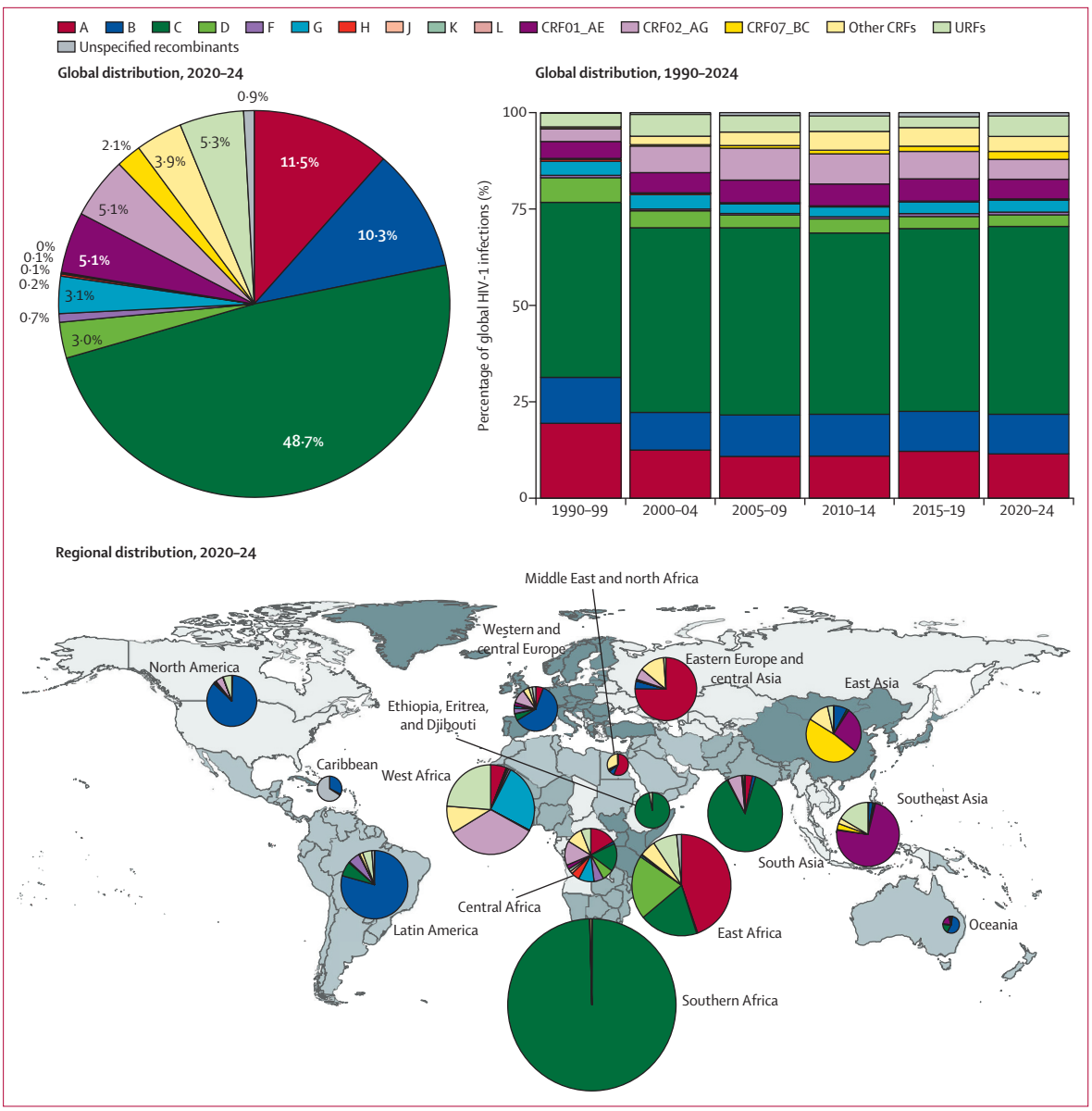

Figure 3: **Global distribution of HIV-1 variants, 1990–2024**
Distributions represent HIV-1 subtypes, CRFs, and URFs as a percentage of global or regional HIV-1 infections. The surface area of each pie chart on the world map corresponds to the relative number of people living with HIV in each region. Countries forming a region are shaded (appendix 1 p 7). Other CRFs refers to all CRFs except CRF01_AE, CRF02_AG, and CRF07_BC. CRFs=circulating recombinant forms. URFs=unique recombinant forms.

The Caribbean was dominated by subtype B before 2020 (>84%) but had more unspecified recombinants (66·1% [95% CI 53·3–79·1]) than subtype B (32·1% [19·5–44·8]) in 2020–24. In Oceania, subtype B comprised 54·9% (52·8–57·0) of infections in 2020–24, although subtype C dominated in half of the periods.

Subtype B accounted for 60·7% (95% CI 58·1–63·3) of infections in western and central Europe in 2020–24, decreasing from 70·2% (69·8–70·7) in 2000–04. Subtype A increased from 3·7% (3·6–3·9) in 2000–04 to 5·7% (5·0–6·3) in 2020–24, and recombinants more than doubled, from 10·8% (10·4–11·2) in 2000–04 to 22·7% (20·3–25·0) in 2020–24 due to increasing CRF02_AG, other CRFs, CRF01_AE, and URFs.

Subtype A was responsible for over three-quarters of infections in eastern Europe and central Asia in 2020–24 (75·2% [95% CI 74·0–76·4]), consistent with previous periods. Recombinants increased in the region, from 2·7% (2·0–3·4) in 2000–04 to 20·0% (18·9–21·1) in 2020–24, driven by other CRFs, including CRF63_02A1 (data not shown) and CRF02_AG.

Southeast Asia was dominated by CRF01_AE, comprising 73·2% (95% CI 68·2–78·1) of infections in 2020–24. From 2000–04 to 2020–24, there were increases

	HIV-1 subtypes										CRFs				URFs	Unspecified recombinants	Total CRFs*	Total recombinants
	A	B	C	D	F	G	H	J	K	L	CRF01_AE	CRF02_AG	CRF07_BC	Other				
Global																		
1990–99	19·4%	11·9%	45·4%	6·4%	0·6%	3·6%	0·5%	0·2%	0·1%	0·0%	4·4%	3·2%	0·1%	0·4%	3·5%	0·2%	8·2%	11·9%
2000–04	12·5%	9·8%	47·9%	4·4%	0·5%	3·8%	0·2%	0·1%	0·0%	0·0%	5·2%	6·9%	0·4%	2·2%	5·7%	0·4%	14·7%	20·8%
2005–09	10·8%	10·7%	48·6%	3·3%	0·4%	2·5%	0·2%	0·1%	0·0%	0·0%	5·9%	8·3%	0·7%	3·5%	4·3%	0·7%	18·3%	23·3%
2010–14	10·9%	10·8%	47·0%	3·7%	0·6%	2·6%	0·1%	0·1%	0·1%	0·0%	5·7%	7·8%	0·9%	4·9%	4·0%	0·9%	19·3%	24·2%
2015–19	12·2%	10·3%	47·4%	3·1%	0·8%	3·0%	0·2%	0·0%	0·0%	0·0%	5·8%	7·1%	1·4%	4·8%	2·8%	1·1%	19·1%	22·9%
2020–24	11·5%	10·3%	48·7%	3·0%	0·7%	3·1%	0·2%	0·1%	0·1%	0·0%	5·1%	5·1%	2·1%	3·9%	5·3%	0·9%	16·2%	22·4%
North America																		
1990–99	1·3%	96·1%	0·9%	0·1%	0·1%	0·1%	0·0%	0·0%	0·0%	0·0%	0·5%	0·4%	0·0%	0·2%	0·3%	0·0%	1·1%	1·4%
2000–04	0·7%	95·0%	1·6%	0·2%	0·3%	0·2%	0·0%	0·0%	0·0%	0·0%	0·4%	0·7%	0·0%	0·1%	0·9%	0·0%	1·2%	2·1%
2005–09	0·7%	94·8%	1·7%	0·2%	0·1%	0·3%	0·0%	0·0%	0·0%	0·0%	0·5%	0·9%	0·0%	0·3%	0·2%	0·3%	1·8%	2·2%
2010–14	0·7%	94·8%	1·7%	0·2%	0·1%	0·4%	0·0%	0·0%	0·0%	0·0%	0·5%	0·9%	0·1%	0·4%	0·1%	0·2%	1·9%	2·2%
2015–19	0·6%	96·0%	1·6%	0·1%	0·1%	0·2%	0·0%	0·0%	0·0%	0·0%	0·4%	0·5%	0·0%	0·2%	0·2%	0·0%	1·2%	1·4%
2020–24	0·6%	86·4%	0·9%	0·1%	0·1%	0·5%	0·0%	0·0%	0·0%	0·0%	0·6%	5·0%	0·0%	0·2%	5·6%	0·0%	5·8%	11·4%
Caribbean																		
1990–99	0·0%	89·0%	0·6%	5·6%	0·2%	1·4%	0·2%	0·2%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	2·8%	0·0%	0·0%	2·8%
2000–04	0·0%	96·9%	0·3%	0·0%	0·0%	0·2%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	1·3%	1·0%	0·0%	1·4%	2·4%
2005–09	0·2%	96·0%	0·4%	0·1%	0·0%	0·3%	0·1%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	2·2%	0·6%	0·0%	2·2%	2·9%
2010–14	0·5%	88·1%	1·0%	0·3%	0·0%	1·0%	0·2%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	6·6%	2·0%	0·0%	6·8%	8·8%
2015–19	0·1%	84·1%	0·4%	1·1%	0·0%	0·2%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	4·1%	1·5%	8·6%	4·1%	14·1%
2020–24	0·0%	32·1%	1·7%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	66·1%	0·1%	66·2%
Latin America																		
1990–99	0·0%	87·4%	2·2%	0·1%	7·3%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·8%	2·0%	0·2%	0·8%	3·0%
2000–04	0·0%	84·8%	4·6%	0·1%	4·3%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	1·5%	3·7%	1·1%	1·5%	6·3%
2005–09	0·0%	81·4%	4·9%	0·1%	2·6%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	1·6%	5·8%	3·4%	1·7%	10·8%
2010–14	0·1%	78·5%	5·4%	0·1%	4·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·2%	1·0%	2·3%	6·9%	1·3%	3·5%	11·7%
2015–19	0·0%	76·9%	6·9%	0·1%	5·4%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·2%	0·0%	1·0%	8·5%	0·8%	1·3%	10·6%
2020–24	0·2%	79·1%	6·9%	0·5%	5·6%	0·2%	0·0%	0·0%	0·0%	0·0%	0·1%	0·2%	0·0%	1·5%	4·4%	1·2%	1·9%	7·5%
Western and central Europe																		
1990–99	6·4%	75·8%	3·3%	1·0%	3·7%	3·5%	0·2%	0·1%	0·0%	0·0%	0·8%	2·3%	0·0%	0·9%	1·2%	0·2%	4·0%	6·1%
2000–04	3·7%	70·2%	6·8%	1·4%	3·7%	3·2%	0·1%	0·1%	0·0%	0·0%	1·2%	4·9%	0·0%	1·8%	1·5%	1·1%	8·0%	10·8%
2005–09	3·7%	64·7%	6·6%	1·7%	3·6%	3·1%	0·0%	0·0%	0·0%	0·0%	1·7%	7·6%	0·1%	2·8%	2·9%	3·4%	12·2%	16·4%
2010–14	3·9%	62·7%	6·2%	0·6%	4·5%	2·8%	0·1%	0·0%	0·0%	0·0%	1·7%	9·4%	0·1%	3·7%	2·4%	1·3%	14·9%	19·2%
2015–19	4·9%	51·5%	16·0%	0·5%	5·2%	2·3%	0·1%	0·0%	0·0%	0·0%	1·7%	10·1%	0·1%	4·1%	2·0%	0·8%	16·0%	19·4%
2020–24	5·7%	60·7%	5·0%	0·3%	3·6%	2·0%	0·0%	0·0%	0·0%	0·0%	2·5%	10·4%	0·1%	4·5%	2·5%	1·2%	17·5%	22·7%
Eastern Europe and central Asia																		
1990–99	65·8%	19·5%	2·9%	1·4%	0·0%	4·5%	1·0%	0·0%	0·0%	0·0%	0·1%	0·4%	0·0%	3·6%	0·7%	0·1%	4·1%	4·9%
2000–04	80·0%	15·0%	1·2%	0·5%	0·0%	0·5%	0·0%	0·0%	0·0%	0·0%	0·1%	0·2%	0·0%	1·8%	0·6%	0·0%	2·1%	2·7%
2005–09	86·3%	8·1%	0·1%	0·0%	0·1%	0·6%	0·0%	0·0%	0·0%	0·0%	0·1%	1·6%	0·0%	2·7%	0·5%	0·0%	4·3%	4·8%
2010–14	82·2%	8·8%	0·7%	0·0%	0·0%	0·7%	0·0%	0·0%	0·0%	0·0%	0·1%	1·7%	0·0%	3·5%	2·2%	0·1%	5·3%	7·6%
2015–19	80·7%	5·4%	0·2%	0·0%	0·0%	0·5%	0·0%	0·0%	0·0%	0·0%	0·1%	2·5%	0·0%	8·2%	2·2%	0·2%	10·8%	13·1%
2020–24	75·2%	4·0%	0·1%	0·1%	0·1%	0·6%	0·0%	0·0%	0·0%	0·0%	0·1%	5·8%	0·0%	12·9%	1·2%	0·0%	18·8%	20·0%
South Asia																		
1990–99	0·8%	9·4%	88·8%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·9%	0·0%	0·0%	0·9%
2000–04	1·2%	0·4%	97·7%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·2%	0·0%	0·0%	0·0%	0·3%	0·0%	0·0%	0·6%
2005–09	1·9%	0·9%	95·4%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·2%	0·0%	0·1%	1·3%	0·0%	0·4%	1·7%
2010–14	3·9%	2·5%	87·7%	0·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·1%	0·3%	0·0%	0·1%	4·3%	1·1%	0·5%	5·9%
2015–19	4·8%	0·5%	90·6%	0·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·3%	1·4%	0·0%	0·5%	1·6%	0·2%	2·2%	4·0%

(Table 2 continues on next page)

	HIV-1 subtypes										CRFs				URFs	Unspecified recombinants	Total CRFs*	Total recombinants
	A	B	C	D	F	G	H	J	K	L	CRF01_AE	CRF02_AG	CRF07_BC	Other				
(Continued from previous page)																		
2020–24	3·1%	1·5%	87·4%	0·1%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·1%	6·1%	0·0%	0·6%	0·9%	0·1%	6·8%	7·7%
Southeast Asia																		
1990–99	0·1%	18·9%	0·9%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	77·5%	0·0%	0·0%	0·0%	2·4%	0·2%	77·5%	80·1%
2000–04	0·2%	9·6%	2·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	83·8%	0·1%	0·0%	0·4%	3·5%	0·3%	84·3%	88·1%
2005–09	0·2%	5·1%	1·6%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	85·3%	0·4%	0·0%	0·9%	5·5%	1·0%	86·6%	93·0%
2010–14	0·1%	11·9%	0·8%	0·1%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	72·0%	0·7%	0·2%	6·5%	7·0%	0·6%	79·5%	87·0%
2015–19	1·4%	7·2%	4·9%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	80·4%	0·4%	0·3%	0·2%	3·3%	1·8%	81·3%	86·5%
2020–24	0·4%	2·1%	0·9%	0·1%	0·1%	0·4%	0·0%	0·0%	0·0%	0·0%	73·2%	0·3%	3·1%	2·8%	16·7%	0·0%	79·4%	96·1%
East Asia																		
1990–99	0·6%	46·6%	3·5%	0·1%	0·1%	0·4%	0·0%	0·0%	0·0%	0·0%	31·6%	0·4%	8·6%	4·8%	1·8%	1·2%	45·5%	48·6%
2000–04	0·5%	38·1%	2·2%	0·1%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	27·2%	0·4%	13·1%	13·1%	4·8%	0·5%	53·7%	59·1%
2005–09	0·1%	34·3%	2·0%	0·1%	0·0%	0·2%	0·0%	0·0%	0·0%	0·0%	31·8%	0·4%	20·2%	7·2%	3·2%	0·6%	59·5%	63·3%
2010–14	0·1%	20·7%	1·6%	0·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	39·9%	0·2%	24·5%	8·9%	3·5%	0·5%	73·5%	77·5%
2015–19	0·1%	8·6%	0·9%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	36·6%	0·2%	35·1%	13·8%	3·8%	0·7%	85·7%	90·2%
2020–24	0·3%	7·7%	1·3%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	26·3%	0·1%	48·1%	12·6%	3·5%	0·1%	87·1%	90·7%
Oceania																		
1990–99	2·2%	80·7%	8·2%	0·0%	0·0%	0·9%	0·0%	0·0%	0·0%	0·0%	8·0%	0·0%	0·0%	0·0%	0·0%	0·0%	8·0%	8·0%
2000–04	0·2%	31·7%	64·8%	0·1%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	2·8%	0·2%	0·0%	0·1%	0·1%	0·0%	3·1%	3·1%
2005–09	0·7%	82·2%	6·1%	0·2%	0·1%	0·2%	0·0%	0·0%	0·0%	0·0%	8·7%	0·7%	0·0%	0·2%	1·0%	0·0%	9·6%	10·6%
2010–14	0·2%	22·0%	74·2%	0·1%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	2·7%	0·3%	0·0%	0·1%	0·3%	0·0%	3·1%	3·4%
2015–19	0·7%	16·6%	76·6%	0·1%	0·2%	0·1%	0·0%	0·0%	0·0%	0·0%	4·7%	0·2%	0·1%	0·2%	0·5%	0·0%	5·3%	5·8%
2020–24	2·8%	54·9%	18·1%	0·1%	0·3%	0·8%	0·0%	0·0%	0·0%	0·0%	18·0%	2·0%	0·0%	0·4%	2·5%	0·0%	20·4%	22·9%
Middle East and north Africa																		
1990–99	7·2%	10·6%	26·9%	44·8%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	1·4%	0·0%	0·0%	8·9%	0·0%	1·4%	10·3%
2000–04	41·0%	45·2%	0·8%	0·5%	0·3%	0·5%	0·0%	0·0%	0·0%	0·0%	0·3%	6·7%	0·0%	1·7%	3·0%	0·0%	8·7%	11·7%
2005–09	32·3%	39·3%	0·4%	0·0%	0·2%	1·1%	0·0%	0·0%	0·0%	0·0%	1·7%	5·8%	0·0%	3·4%	0·3%	15·5%	10·9%	26·6%
2010–14	2·8%	23·2%	1·6%	0·4%	0·1%	0·3%	0·0%	0·0%	0·0%	0·0%	0·4%	8·5%	0·1%	61·8%	0·8%	0·0%	70·8%	71·6%
2015–19	4·7%	23·8%	5·6%	0·9%	0·0%	1·0%	0·0%	0·0%	0·0%	0·0%	0·8%	21·8%	0·0%	38·3%	3·0%	0·0%	60·9%	63·9%
2020–24	56·3%	8·1%	0·4%	0·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·6%	2·4%	0·0%	31·8%	0·2%	0·0%	34·8%	35·1%
West Africa																		
1990–99	48·4%	0·1%	0·7%	1·0%	0·9%	22·1%	0·1%	0·2%	0·0%	0·0%	0·2%	22·7%	0·0%	0·5%	3·2%	0·0%	23·4%	26·5%
2000–04	6·9%	0·1%	0·9%	0·9%	0·6%	22·2%	0·1%	0·1%	0·0%	0·0%	1·0%	47·2%	0·0%	7·3%	12·6%	0·0%	55·5%	68·0%
2005–09	4·0%	0·3%	0·8%	1·4%	0·7%	15·0%	0·1%	0·1%	0·0%	0·0%	0·2%	57·3%	0·0%	10·5%	9·4%	0·2%	68·0%	77·6%
2010–14	3·6%	0·3%	0·7%	1·0%	0·6%	16·7%	0·1%	0·0%	0·0%	0·0%	0·6%	57·8%	0·0%	7·4%	10·3%	1·0%	65·8%	77·1%
2015–19	5·9%	1·6%	0·4%	0·8%	0·6%	21·9%	0·0%	0·0%	0·0%	0·0%	0·1%	55·8%	0·0%	6·7%	5·0%	1·2%	62·6%	68·8%
2020–24	5·8%	0·1%	0·3%	0·6%	0·8%	25·0%	0·0%	0·0%	0·0%	0·0%	0·2%	33·4%	0·0%	9·9%	23·7%	0·0%	43·6%	67·3%
East Africa																		
1990–99	48·8%	0·0%	10·6%	28·5%	0·1%	0·6%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·5%	10·2%	0·6%	0·6%	11·4%
2000–04	41·3%	0·0%	21·2%	17·1%	0·1%	0·6%	0·0%	0·0%	0·0%	0·0%	0·0%	0·2%	0·0%	0·8%	16·7%	1·9%	1·0%	19·6%
2005–09	50·7%	0·2%	15·6%	19·0%	0·0%	0·4%	0·0%	0·0%	0·0%	0·0%	0·3%	0·2%	0·0%	2·9%	8·3%	2·3%	3·4%	14·0%
2010–14	48·3%	0·3%	16·0%	21·4%	0·0%	0·7%	0·0%	0·2%	0·0%	0·0%	0·7%	0·1%	0·0%	5·4%	5·0%	1·9%	6·2%	13·1%
2015–19	48·5%	0·9%	17·1%	21·2%	0·0%	1·0%	0·0%	0·0%	0·1%	0·0%	0·3%	0·2%	0·0%	2·9%	4·0%	3·7%	3·5%	11·2%
2020–24	44·7%	0·3%	19·0%	20·5%	0·0%	0·6%	0·0%	0·0%	0·0%	0·0%	0·1%	0·2%	0·0%	5·2%	7·9%	1·5%	5·5%	14·9%
Ethiopia, Eritrea, and Djibouti																		
1990–99	0·4%	0·4%	88·6%	0·4%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	10·2%	0·0%	0·0%	10·2%
2000–04	1·3%	0·0%	95·3%	0·5%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·3%	0·0%	0·0%	2·7%	0·0%	0·3%	2·9%
2005–09	0·2%	0·2%	97·9%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	1·1%	0·0%	0·0%	0·1%	0·5%	1·1%	1·7%
2010–14	0·4%	0·2%	98·7%	0·2%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	0·0%	0·4%	0·0%	0·1%	0·5%

(Table 2 continues on next page)

	HIV-1 subtypes										CRFs				URFs	Unspecified recombinants	Total CRFs*	Total recombinants
	A	B	C	D	F	G	H	J	K	L	CRF01_AE	CRF02_AG	CRF07_BC	Other				
(Continued from previous page)																		
2015–19	0·4%	0·2%	98·5%	0·2%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	0·1%	0·3%	0·0%	0·3%	0·6%
2020–24	0·0%	0·0%	97·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	2·9%	0·0%	0·0%	2·9%
Central Africa																		
1990–99	48·3%	0·3%	5·1%	8·4%	4·2%	8·1%	9·7%	2·2%	0·9%	0·0%	2·3%	3·3%	0·0%	1·9%	5·3%	0·0%	7·5%	12·8%
2000–04	32·8%	0·2%	9·7%	10·8%	3·0%	10·0%	5·1%	2·4%	0·9%	0·0%	2·2%	5·7%	0·0%	4·1%	11·4%	1·7%	12·0%	25·0%
2005–09	15·4%	0·5%	13·2%	5·7%	3·3%	6·7%	5·0%	1·4%	0·2%	0·0%	2·0%	7·6%	0·0%	15·3%	22·9%	0·8%	24·9%	48·6%
2010–14	18·1%	1·0%	12·9%	6·7%	4·8%	6·4%	3·2%	1·8%	1·2%	0·0%	2·5%	7·3%	0·0%	14·7%	16·2%	3·1%	24·5%	43·9%
2015–19	21·3%	0·3%	15·1%	3·7%	8·1%	9·5%	4·1%	0·8%	0·0%	0·0%	0·6%	8·0%	0·0%	10·0%	16·5%	1·9%	18·6%	37·0%
2020–24	16·5%	1·4%	17·1%	7·0%	6·0%	9·7%	5·2%	1·6%	1·8%	0·0%	2·6%	14·9%	0·0%	10·2%	6·1%	0·0%	27·7%	33·8%
Southern Africa																		
1990–99	0·6%	2·4%	96·4%	0·4%	0·0%	0·2%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	0·0%	0·1%
2000–04	6·6%	0·6%	86·7%	3·5%	0·0%	0·4%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	1·2%	0·9%	0·0%	1·2%	2·1%
2005–09	0·5%	1·2%	95·7%	0·4%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	1·5%	0·3%	0·1%	1·6%	1·9%
2010–14	0·4%	0·2%	93·8%	1·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	3·7%	0·3%	0·4%	3·8%	4·4%
2015–19	0·8%	0·0%	92·8%	0·1%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·1%	0·0%	5·4%	0·2%	0·3%	5·5%	6·1%
2020–24	0·1%	0·0%	99·5%	0·0%	0·0%	0·1%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·0%	0·3%	0·0%	0·0%	0·3%	0·3%

Distributions represent HIV-1 subtypes, CRFs, and URFs as a percentage of global or regional HIV-1 infections. 95% CIs are provided in appendix 1 (pp 23–26). CRF=circulating recombinant form. URF=unique recombinant form. *Other CRFs refer to all CRFs except CRF01_AE, CRF02_AG, and CRF07_BC.

Table 2: **Global and regional distribution of HIV-1 variants, 1990–2024**

in URFs (3·5% [2·3–4·6] to 16·7% [12·0–21·4]), CRF07_BC (0·0% [0·0–0·0] to 3·1% [2·2–4·0]), and other CRFs (0·4% [0·3–0·6] to 2·8% [1·9–3·7]).

In east Asia in 2020–24, about half of infections were CRF07_BC (48·1% [95% CI 47·6–48·6]), nearly triple since 2000–04 (13·1% [12·2–14·0]). Over a quarter of infections were CRF01_AE (26·3% [25·8–26·7]), followed by other CRFs (12·6% [12·3–13·0]) including CRF08_BC and CRF55_01B (data not shown). Subtype B decreased from 38·1% (36·7–39·4) in 2000–04 to 7·7% (7·4–8·0) in 2020–24.

Discussion

To the best of our knowledge, this analysis of nearly 1·4 million HIV-1 samples from 154 countries provides the largest and most up-to-date global synthesis of HIV-1 molecular epidemiology over the past 35 years. Overall, the global distribution of HIV-1 subtypes and recombinants remained broadly stable between 2000 and 2024, with subtype C accounting for nearly half of infections and subtypes A and B for about a tenth of infections each, consistent with previous studies.[17,20] Marked regional heterogeneity persisted, with distinct HIV-1 variants dominating across regions and the greatest genetic diversity observed in central Africa. Important regional shifts in HIV-1 variant distributions were identified over time, particularly due to increasing recombinants in western and central Europe and east Asia.

In 2020–24, over a third of HIV infections in western and central Europe were attributable to non-subtype B variants, a region historically dominated by subtype B. This change was mainly driven by increasing recombinants, including CRF02_AG and other CRFs, and is distinct from North America, where subtype B continues to contribute over 86% of infections. In previous analyses, the high and increasing proportion of recombinants in western and central Europe had not been apparent, as western and central Europe and North America were combined into one region.[17,18,20] Non-subtype B infections in Europe have previously been associated with immigration from Africa and eastern Europe.[27–29] For instance, CRF02_AG, which dominates in west Africa, accounted for higher proportions of infections among people with HIV who immigrated to Spain,[29] Portugal,[28] and Belgium,[27] compared with individuals native to those countries. Subtype A, which is prevalent in eastern Europe and central Asia, also increased in western and central Europe in recent time periods. Recombinants, especially CRF02_AG and CRF63_02A1, have increased in eastern Europe and central Asia since 2000.[18] The ongoing influx of war-displaced refugees from eastern Europe might continue to challenge subtype B dominance in western and central Europe.[30]

Recombinants increased in other regions as well, including in east Asia. The increase of CRF07_BC in east Asia drove the global increase in CRF07_BC. The spread of CRF07_BC in east Asia probably reflects dispersion along drug-trafficking routes in China, which initially involved people who inject drugs and men who have sex

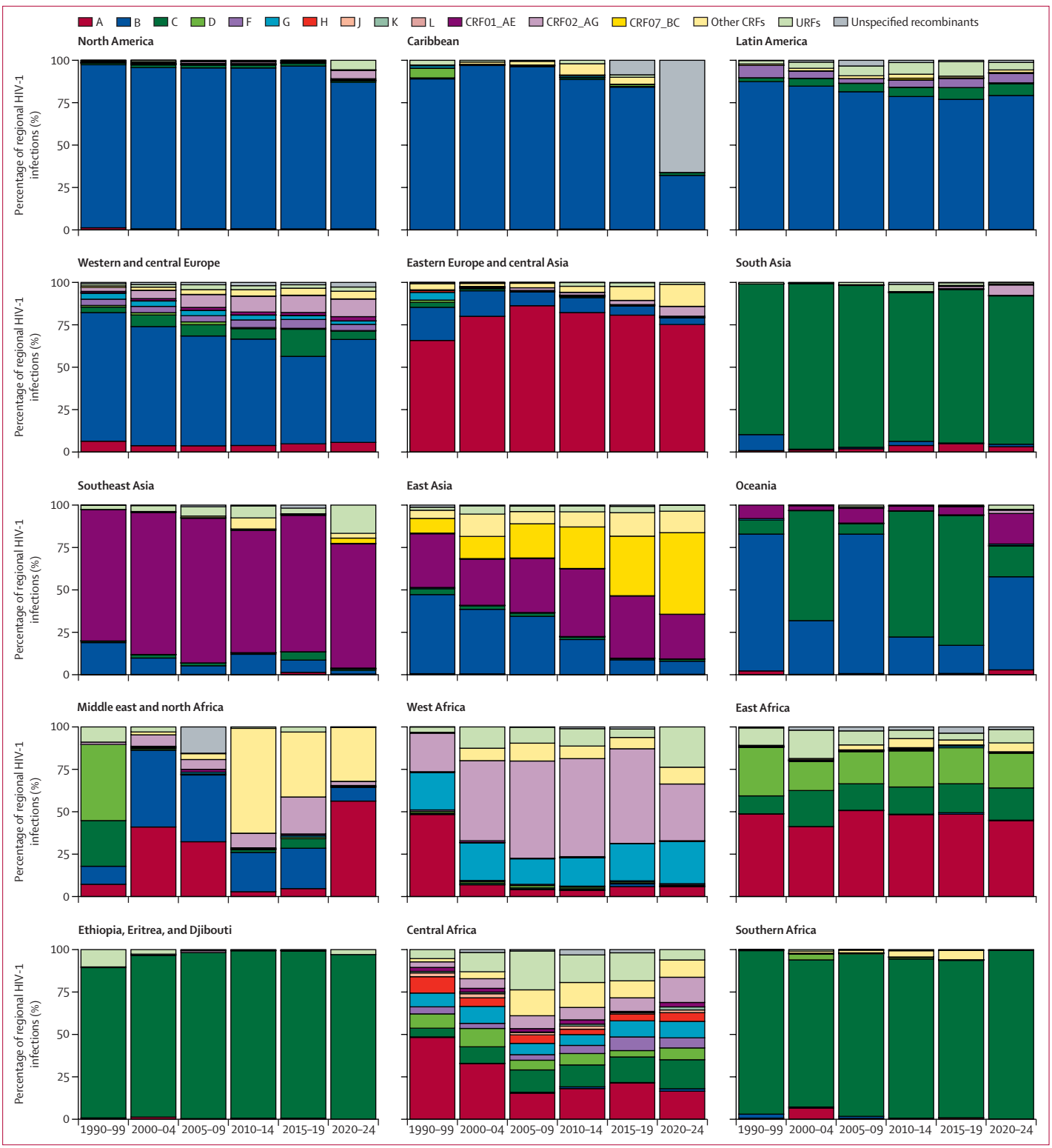

Figure 4: **Regional distribution of HIV-1 variants, 1990–2024**
Distributions represent HIV-1 subtypes, CRFs, and URFs as a percentage of regional HIV-1 infections. Other CRFs refers to all CRFs except CRF01_AE, CRF02_AG, and CRF07_BC. CRFs=circulating recombinant forms. URFs=unique recombinant forms.

with men, before spreading to the wider population.[31] Higher rates of new infections and limited access to care among key populations, including people who inject drugs[1,32] and a lower pathogenicity of CRF07_BC compared with other variants,[33] probably enabled CRF07_BC to spread. Emigration from parts of east Asia has the potential to further affect global patterns of HIV-1 diversity.

This study has several strengths. Nearly 1·4 million samples provided a representative dataset for estimating global and regional HIV-1 subtype, CRF, and URF proportions. Our database unified HIV-1 subtyping data from both published and unpublished sources. We included all eligible published data available since 1990. Unpublished data in particular included information for recent years and for countries with limited published HIV subtyping data. We achieved high coverage (>90%) for most regions and periods and globally.

This study also has some limitations. Published data are prone to publication bias and delays, and survey data are prone to response bias. Overall, data were less abundant for the 1990–99 and 2020–24 periods than other periods. We divided HIV-1 subtyping data spanning multiple years of sample collection evenly across years, which might have concealed changes over time. An alternative method of allocating samples to the middle year as a part of our sensitivity analyses showed no perceptible differences in global variant proportions. Global and regional HIV-1 variant distributions reflect the underlying country-level data, and subtyped HIV samples are not always nationally representative, requiring cautious interpretation. Depth of coverage was low in regions with the highest numbers of people living with HIV, including southern, central, east, and west Africa and south Asia. Although breadth of coverage was high overall, differential availability of data across countries and periods might have distorted HIV-1 variant distributions. For instance, there were no data from Haiti in 2020–24, leading to a potentially misleading reduction in subtype B in the Caribbean in 2020–24. Low coverage in Oceania and Middle East and north Africa led to fluctuations in HIV-1 variant patterns. However, Oceania, Middle East and north Africa, and the Caribbean have the smallest numbers of people living with HIV and hence have little influence on global proportions. Nevertheless, efforts should be made to improve surveillance in these regions.

Heterogeneity in available data, due to differing study designs, study populations, and subtyping methods, as well as potential misclassification of HIV-1 variants, could have oversampled or undersampled some variants and affected our estimates of HIV-1 variant proportions. Weighting of proportions by country-level UNAIDS estimates does not correct for differences in sampling within countries. Countries in some regions, such as western and central Europe, might have more resources to routinely monitor and detect recombinants than others. Overall, recombinants are probably underestimated in this analysis because most samples were subtyped using *pol* sequencing only, which can miss recombinants, as the rest of the genome is not assessed.[7,17] Whole genome sequencing is important for understanding the true prevalence and complexity of circulating HIV recombinants. We avoided drawing comparisons with 1990–99, as subtyping methods changed after 2000 (appendix 1 p 12). For instance, in parts of west Africa, many earlier samples were mistakenly classified as subtype A based on *env* subtyping alone, before recognition of CRF02_AG led to their correct classification from 2000 onwards.[34] As a part of our sensitivity analyses, we weighted each record in our database by its risk of bias score, which summarises population representativeness, sampling frame quality, sampling selection method, sample size, and subtyping method into a single score, and found similar results to the primary analysis (appendix 1 p 22). We could not stratify HIV-1 variant distributions by sex, gender, race, or ethnicity as these data were largely unavailable. Finally, subtyping could only be performed on samples from people living with HIV with detectable viral loads, including newly diagnosed people and those experiencing treatment failure, who might differ from individuals with suppressed viral load.

Knowledge of the global and regional distribution of HIV-1 variants is instrumental for effective HIV treatment and prevention. Diagnostic and viral load assays might be challenged by an increasing prevalence of previously uncommon HIV-1 subtypes and recombinants.[12,35–37] Drug resistance in less common HIV-1 subtypes and recombinants remains understudied, although they can contain novel mutations that affect treatment response,[14] and the same mutation can cause differing responses to treatment depending on the HIV-1 variant.[38] Infection with HIV-1 variants prone to treatment-resistant mutations can challenge the efficacy of long-acting ART or pre-exposure prophylaxis.[39] For instance, trial data has shown an increased occurrence of virological failure with cabotegravir–rilpivirine long-acting treatment in people with HIV-1 infected with subtype A6.[40] To date, the only HIV vaccine trial to show modest protection was the phase 3 RV144 trial in Thailand based on subtype B and CRF01_AE.[41] Several mRNA-based HIV-1 vaccines are under development, including recent phase I candidates that induced neutralising antibody responses in approximately 80% of participants.[42] The flexibility of mRNA vaccines in addressing genetic variation could help account for the extensive genetic diversity of HIV-1.[43] A comprehensive understanding of the HIV-1 variant distributions and the populations and geographical areas to target are crucial for developing variant-specific HIV vaccines.[11]

This study shows that, although the global HIV-1 variant distribution might be stabilising, regional patterns are complex and changing over time. Changes in HIV-1 variant distributions reflect the differences between new HIV infections and deaths due to HIV/AIDS, which are influenced by the biological properties of HIV-1 variants;

the social, political, economic, and environmental contexts of the populations in which they spread; and interventions for treatment and prevention. Ongoing surveillance and further research are required to examine and predict spatial and temporal trends in HIV-1 genetic diversity as well as the factors driving these trends.

Global HIV Molecular Epidemiology Collaboration

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Contributors

AK conducted the electronic and manual literature searches. AK, LG, and JH designed and conducted the global survey. AK, LG, LS, XL, LLLW, and ANM screened electronic literature search results, extracted data from eligible publications, and conducted risk of bias assessments. AK processed, analysed, and interpreted the data; made the figures and tables; and wrote the first draft of the manuscript. PJG supported the database development. KA assisted with the statistical analyses. JH conceived, designed, and coordinated the study; provided supervision; wrote the study protocol; guided data analysis, interpretation, and visualisation; and wrote the manuscript. AK and JH verified the data underlying the study. All authors had full access to all the data in the study and accept responsibility to submit for publication.

Declaration of interests

JH is a member of the writing group of the British HIV Association guidelines on the management of HIV in pregnancy and the postpartum period 2025. All other authors declare no competing interests.

Data sharing

References of published HIV subtyping data used in this study are listed in appendix 2. Unpublished HIV subtyping data (appendix 1 pp 8–11) can be made available upon request to the corresponding author, at the discretion of the relevant contributing member of the Global HIV Molecular Epidemiology Collaboration. Country-level HIV prevalence estimates are available on the UNAIDS AIDSinfo website. We provide the code used for our analysis in the following GitHub repository: https://github.com/ayishakhalid/HIV_MOL_EPI_1990–2024.

See **Online** for appendix 2

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