# Emergence and spread of the SARS-CoV-2 omicron (BA.1) variant across Africa: an observational study

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## Summary

**Background** In mid-November, 2021, the SARS-CoV-2 omicron variant (B.1.1.529; BA.1 sublineage) was detected in southern Africa, prompting international travel restrictions. We aimed to investigate the spread of omicron BA.1 in Africa.

**Methods** In this observational study, samples from patients infected with SARS-CoV-2 from 27 laboratories in 24 African countries, collected between June 1, 2021 and April 14, 2022, were tested for omicron BA.1 and delta (B.1.617.2) variants using real-time RT-PCR. Samples that tested positive for BA.1 by RT-PCR and were collected before estimated BA.1 emergence according to epidemiological properties were excluded from downstream analyses. The diagnostic precision of the assays was evaluated by high-throughput sequencing of samples from four countries. The observed spread of BA.1 was compared with mobility-based mathematical simulations and entries for SARS-CoV-2 in the Global Initiative on Sharing All Influenza Data (GISAID) genomic database. We estimated the effective reproduction number ($R_t$) at the country level considering the BA.1 fraction and the reported numbers of infections. Phylogeographical analyses were done in a Bayesian framework.

**Findings** Through testing of 13 294 samples from patients infected with SARS-CoV-2, we established that, by November–December, 2021, omicron BA.1 had replaced the delta variant of SARS-CoV-2 in all African subregions, following a south–north gradient, with a median $R_t$ of 2·60 (95% CI 2·46–2·71). This south–north spread, established on the basis of PCR data, was substantiated by phylogeographical reconstructions, ancestral state reconstructions, and GISAID data. PCR-based reconstructions of country-level BA.1 predominance and the availability of BA.1 genomic sequences in GISAID correlated significantly in time ($p=0·0002$, $r=0·78$). The first detections of BA.1 in high-income settings beyond Africa were predicted accurately in time by mobility-based mathematical simulations ($p<0·0001$). Comparing PCR-based reconstructions with mobility-based mathematical simulations suggested that SARS-CoV-2 infections in Africa were under-reported by approximately ten times. Inbound travellers infected with BA.1, departing from five continents, were identified in six African countries by early December, 2021.

**Interpretation** Omicron BA.1 was widespread in Africa when travel bans were implemented, limiting their effectiveness. Combined with genomic surveillance and mobility-based mathematical modelling, PCR-based strategies can inform $R_t$ and the geographical spread of emerging pathogens in a cost-effective and timely manner, and can guide evidence-based, non-pharmaceutical interventions such as travel restrictions or physical distancing.

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## Introduction

By the end of June, 2024, more than 7 million people worldwide had died from COVID-19.[1] The true number of deaths is likely to be under-reported, particularly in regions where diagnostic capacities are low.[2] In 2021, WHO estimated that only 14% of all SARS-CoV-2

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For the French translation of the abstract see **Online** for appendix 1

For the Portugese translation of the abstract see **Online** for appendix 2

For the Spanish translation of the abstract see **Online** for appendix 3

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## Research in context

### Evidence before this study

We searched PubMed for articles published between database inception and Aug 6, 2024, using the search term "SARS-CoV-2 AND BA.1 AND Africa" with no language restrictions. The search identified 86 peer-reviewed articles. 31 articles investigated the protection from new SARS-CoV-2 variants provided by previous vaccination or exposure, which was found to reduce the risk of an infection with the omicron (B.1.1.529) BA.1 variant by 13–49%. Although the attack rate during the BA.1 wave in Africa was higher than that during the delta (B.1.617.2) wave, severe disease was less common, potentially due to increased pre-existing immunity. Studies investigating the spread of BA.1 in Egypt, South Africa, The Gambia, and Zambia showed that BA.1 was detected in different regions of Africa during November and December, 2021. However, the exact routes and the speed of BA.1 spread—which affect the success of containment measures such as travel restrictions—have not been investigated systematically. Additionally, retrospective analyses revealed that the countries where SARS-CoV-2 variants, including BA.1, were first identified made a relatively small contribution to their global spread. By contrast, secondary hubs with extensive mobility connections are of high importance for the rapid spread of new variants.

### Added value of this study

We traced the spread of omicron BA.1 in Africa using PCR-based data from 24 countries generated between Nov 25, 2021 and April 29, 2022 combined with phylogeographical analyses of genomic data present in public databases up to 31 months after BA.1 was first detected and mobility-based simulations. We show that BA.1 was already widespread in Africa when travel restrictions were implemented, explaining why the restrictions failed to contain its spread. The early detection of international travellers infected with BA.1 highlights the importance of long-distance connectivity for the rapid spread of emerging SARS-CoV-2 variants. Moreover, we show that real-time RT-PCR-based variant typing can be used to scale up and speed up the surveillance of new SARS-CoV-2 variants as they emerge in resource-limited settings.

### Implications of all the available evidence

Our results highlight the need for global surveillance systems to guide efficient containment measures for new SARS-CoV-2 variants and other emerging pathogens. We show that real-time RT-PCR-based surveillance is feasible in resource-limited settings and can be a useful complement to high-throughput sequencing of patient-derived specimens and wastewater surveillance. PCR-based methods can be widely applied, at low cost and with very short turnaround times, to inform robust mathematical models that can adequately predict the spread of a newly emerging variant.

infections were being detected in Africa[3] and regional post-mortem data suggested that the real COVID-19 death toll might be underestimated.[4]

Throughout the COVID-19 pandemic, the most pronounced genomic change in SARS-CoV-2 was the emergence of the omicron (B.1.1.529) variant (BA.1 sublineage), which was first reported on Nov 11, 2021, in a patient from South Africa. By mid-December, BA.1 had been reported in 87 countries,[5] becoming the predominant SARS-CoV-2 variant globally by the end of December, 2021.[6] In Africa, the fast spread of BA.1 was probably boosted by low vaccination rates and limited testing and contact tracing.[7]

In response to the emergence of BA.1, by late November, 2021, countries within and outside of Africa had restricted international travel from and to the southern and eastern African countries of Botswana, Eswatini, Lesotho, Mozambique, Namibia, South Africa, Zambia, and Zimbabwe, for 4–6 weeks.[8,9] The direct economic loss caused by these travel restrictions was estimated at US$600 million in South Africa alone.[10] However, despite these measures, BA.1 spread rapidly to all continents,[11] questioning the effectiveness of the travel bans. Here we present the results of an epidemiological study, conducted between Nov 25, 2021 and April 29, 2022 during the BA.1 wave, to elucidate the spatiotemporal spread of BA.1 across Africa, relying on country-level molecular data and analyses that are robust against potential false-positive samples, such as those included in the analyses reported in our previously retracted article investigating the evolution of BA.1.[12] Beyond reconstructing BA.1 spread, we demonstrate technical and computational tools to enhance future preparedness measures, including evidence-based assessments of viral spread in the context of non-pharmaceutical interventions such as border closures.

## Methods

### Study design

This observational study included SARS-CoV-2-positive samples obtained from diagnostic testing of patients and travellers between June 1, 2021 and April 14, 2022 from 27 laboratories in 24 countries in Africa: Algeria, Angola, Benin, Botswana, Burkina Faso, Cameroon, Ethiopia, Gabon, Ghana, Guinea, Kenya, Madagascar, Mali, Morocco, Mozambique, Namibia, Niger, Republic of the Congo, Senegal, South Africa, The Gambia, Togo, Uganda, and Zimbabwe. We re-tested diagnostic samples using a SARS-CoV-2 typing PCR test to reconstruct the spread of BA.1 within Africa, made phylogeographical reconstructions using publicly available data on SARS-CoV-2 genomes to validate the robustness of PCR-based genotyping, and explored whether a publicly available simulation tool informed by the PCR results could

forecast the BA.1 spread. This study was approved by the institutional research ethics board (IRB) of Charité-Universitätsmedizin Berlin (EA2/028/22). Ethical approval for re-testing and scientific use was also provided by IRBs in Burkina Faso (Laboratoire National de Référence-Grippes, Ouagadougou; 2020-7-126), Cameroon (Centre Pasteur du Cameroun, Yaoundé; 2020/05/1224/CE/CNERSH/SP), Ghana (Kumasi Centre for Collaborative Research in Tropical Medicine, Kwame Nkrumah University of Science and Technology, Kumasi; CHRPE/AP/566/21), Kenya (Department of Biochemistry, Jomo Kenyatta University of Agriculture and Technology, Nairobi; JKU/2/4/896B), Uganda (Gulu University Multifunctional Laboratories, Gulu; GUREC-093-20 and Makerere University, College of Health Science, Kampala; SBS-2022-130), and Zambia (Tropical Diseases Research Centre, Ndola Teaching Hospital, Ndola; 00003729). In all other countries, IRB approval for re-testing anonymised specimens was not required.

### Sampling and data cleaning

We developed and distributed real-time RT-PCR tests (denoted throughout as PCR) that used hydrolysis probes for SARS-CoV-2 detection and the genotyping of BA.1 (spike 214 GluProGlu insertion) and delta (B.1.617.2; spike deletion 157/158) variants (appendix 4 p 3). Each participating laboratory reported results from the anonymised re-testing of respiratory samples from both local residents and inbound travellers who tested positive for SARS-CoV-2 in previous routine diagnostics.

To be included in the study, samples were required to have a positive SARS-CoV-2 test result by real-time RT-PCR, an unambiguous PCR-based typing result, a known location and date of collection, and a collection date between June 1, 2021, and April 30, 2022. The only analysed variable that was not available for all samples was place of residence. Samples that were incompatible with the epidemiological features of SARS-CoV-2 infection, such as those with implausible serial intervals, were excluded.

The in-silico assessment of the diagnostic performance of the tests for the omicron BA.1 and delta variants was based on the presence and absence of the PCR targets in SARS-CoV-2 genomes available in the Global Initiative on Sharing All Influenza Data (GISAID) genomic database by Jan 18, 2022 (appendix 4 p 3). To assess the clinical diagnostic performance, the PCR-based typing results were compared with high-throughput sequencing (HTS) results, with HTS results considered the benchmark. Complete viral genomes were generated by HTS for a subset of samples from Benin, Botswana, Guinea, and South Africa, according to local sequencing capacities.

Data cleaning and analyses were done in R version 4.2.1. The original country-level PCR data on SARS-CoV-2 detection and sequence data available on GISAID were analysed in parallel for the predominance of BA.1. The exclusion of potentially incorrect genomic sequence entries included all sequences that led to the retraction of a previous article,[12] which focused on evolutionary analyses rather than epidemiological analysis as we have done here. In particular, we excluded six samples from Benin that were suspected to be early BA.1 samples but could not be confirmed by sequencing.[12] To further reduce the effect of potential false-positive BA.1 PCR test results and of potential BA.1 ancestors harbouring the BA.1 marker but not genetically classifiable as BA.1, the dataset was filtered by removing samples that were classified as BA.1 by PCR but had been collected before the first expected BA.1 infection in the corresponding country. To estimate the first expected BA.1 infection for a given country, generalised additive models were calculated for each country to estimate the date on which BA.1 became the dominant SARS-CoV-2 variant in our data (ie, accounting for >50% of SARS-CoV-2 infections). The dates of the first expected true BA.1 infections were back-calculated on the basis of the modelled dates of BA.1 dominance, a doubling time of 3 days,[13] and the population of each country (appendix 4 p 2).

### Statistical analysis

To explore the spread of BA.1, we modelled the proportion of BA.1 samples as a function of days since June 1, 2021, through generalised linear models (GLMs) with different formulas (appendix 4 p 5) using the glm function from the R package stats with binomial likelihood and logit-link function based on binary PCR-based data on BA.1 status. We compared values predicted using GLMs with the PCR-based moving average of the BA.1 fraction calculated using a window size of 21 days to reduce the effect of sampling gaps. On the basis of the mean Akaike information criterion (appendix 4 p 5) and the predicted BA.1 fraction at the beginning of the studied timeframe (appendix 4 pp 14–15), the formula $y \sim x$ was selected for further GLMs. PCR data were grouped by African subregion (according to the African Union[14]) to reduce the effect of regionally heterogeneous sample numbers and distributions over time. For each subregion, a GLM was fitted using the grouped and filtered data in R. BA.1 dominance was defined as more than 50% of SARS-CoV-2-positive samples being classified as BA.1.

To estimate the number of daily infections with the BA.1 and delta variants, the estimated fraction of a specific variant among all SARS-CoV-2 infections in a given country was multiplied by the number of country-specific smoothed new infections per 1 million people, as reported by WHO.[15]

We calculated the median effective reproduction number ($R_t$) on the basis of estimated daily BA.1 infections using the EpiEstim package in R. The serial interval was set to 3·3 days (SD 2·4) on the basis of epidemiological analyses of BA.1.[16]

To evaluate the predictability of the spread of a new SARS-CoV-2 variant, we simulated the global spread of

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See Online for appendix 4

BA.1 using a global epidemic and mobility (GLEAM) model, based on a susceptible–exposed–infectious–recovered model, in the GLEAMviz Simulator version 7.2 (appendix 4 p 16).[17] Multi-run simulations (20 simulations) were started on Nov 11, 2021, corresponding to the collection date of the first identified BA.1 infections, and simulated for 109 days until Feb 28, 2022.[18] Simulations were calculated with a deterministic approach based on literature-derived model parameters and the number of infected individuals was informed by PCR at the start of the simulation. To consider potential under-reporting, additional simulations were calculated with increasing numbers of infected individuals at the start of the simulation.[3] To assess the quality of the selected literature-based model parameters, GLEAM simulations were re-calculated with varying values for attack rate, pre-existing protective immunity, and duration of the infectious phase (appendix 4 pp 9–10).[19]

To compare the BA.1 spread observed from PCR-based typing data with that from HTS data available on GISAID and predictions from GLEAM simulations, GLMs were calculated on the basis of SARS-CoV-2 entries on GISAID as described for PCR-based models. Results from GLMs based on PCR data and GISAID entries were compared with GLEAM simulations by Pearson's correlation.

Taking all GISAID entries from Africa available up to June 19, 2024 that were classified as BA.1 and had at least 95% genome completeness, we conducted phylogeographical analyses in a maximum likelihood framework (using TreeTime version 0.7.6) without a fixed clock rate, after removing molecular clock outliers to ensure good model fit (appendix 4 p 1). In ancestral state reconstructions, we interpreted Bayes factors—a ratio comparing two competing models, eg, one assuming BA.1 emerged in southern Africa and one assuming it did not—as strong evidence if greater than 10 and as decisive evidence if greater than 100.

To compare PCR-based and HTS-based SARS-CoV-2 variant typing in terms of cost and time consumption, we considered commonly applied standard protocols and their required reagents and consumables.

### Role of the funding source

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

## Results

Of 39 laboratories in 34 African countries that were invited to join this study, 27 laboratories in 24 countries agreed to participate. 13 294 samples obtained from patients with laboratory-confirmed SARS-CoV-2 infection from 225 municipalities (figure 1A), sampled between June 1, 2021 and April 14, 2022, were included (table, appendix 4 p 12). HTS-based full-genome sequences were available for 811 (6·1%) of the 13 294 samples. The provided genotyping PCR kits had in-silico specificities of 99·8% for the delta variant and 98·7% for BA.1. Compared with HTS, the kits had a clinical sensitivity of 92% (95% CI 89–95; 293 of 317 samples) and a clinical specificity of 100% (all 494 samples) for the delta variant and a clinical sensitivity of 100% (99–100; 475 of 477 samples) and a clinical specificity of 98% (96–99; 326 of 334 samples) for BA.1 (appendix 4 p 5). For eight samples initially reported by the laboratories as containing BA.1, available HTS-based genomic data showed the absence of the spike 214 GluProGlu insertion targeted by the test, suggesting false-positive results during reporting or contamination of samples with BA.1. The specificity of PCR-based BA.1 detection was substantiated by the absence of the BA.1 marker in 545 additional SARS-CoV-2-positive respiratory samples from Benin, western Africa, collected between Jan 1, and April 30, 2021, before BA.1 emerged.[20]

We analysed the generated PCR data to reconstruct the spread of the omicron variant in Africa. Cleaned data were analysed on the basis of the timepoints when BA.1 became dominant, which is more robust than considering the first BA.1 detections. In most of the included countries, BA.1 had replaced delta as the predominant SARS-CoV-2 variant by the end of December, 2021 (figure 1B, appendix 4 p 17). According to the continent-wide PCR data, BA.1 became the dominant variant (>50% of SARS-CoV-2 variants detected) on Nov 19 (95% CI Nov 16 to Nov 21) in southern Africa, Dec 6 (Dec 5 to Dec 8) in western Africa, Dec 11 (Dec 9 to Dec 14) in central Africa, Dec 15 (Dec 12 to Dec 18) in continental eastern Africa, and Dec 25 (Dec 24 to Dec 26) in northern Africa (figure 1C). These findings suggest that BA.1 spread from south to north and had spread widely in and beyond southern Africa when travel restrictions were put into place within and beyond Africa on or soon after Nov 26, 2021 (figure 1C). This analysis was repeated with different models, with unfiltered PCR data, and based on retrospective analyses of GISAID entries in parallel with cleaned PCR data. All approaches indicated that BA.1 spread from south to north (figure 1D, appendix 4 p 17). Not excluding 24 potentially false-positive PCR results that could not be confirmed by genomic sequencing, three of which were part of the group of eight samples that lacked the targeted spike insertion, had a minimal effect on the predicted BA.1 spread: the estimated dates of BA.1 dominance changed by 1 day for eastern Africa and by 5 days for western Africa, highlighting the robustness of our approach (appendix 4 p 17). The south–north gradient suggested by the PCR data was consistent with the emergence of BA.1 in southern Africa and phylogeographical reconstructions based on available GISAID sequences (figure 2). These phylogeographical reconstructions suggested transitions of BA.1 from South Africa to all African regions, whereas only one backwards transition was reconstructed at high posterior probability: from Ghana to South Africa in late December, 2021. In ancestral state reconstructions, a

South African origin of BA.1 had decisive support (Bayes factor 399·0) and a southern African origin had strong support (Bayes factor 12·8). The delayed introduction of BA.1 into northern Africa could be linked to the geographical distance to southern Africa, little land connectivity between African regions, or very few regional BA.1 infections when travel bans were implemented.[9] Similarly, border closure in Madagascar until late 2021 delayed the introduction of BA.1 (appendix 4 p 6). The median time between the first BA.1 detection and BA.1 predominance in the 22 included countries for which the BA.1 takeover was represented in our data was 9 days (95% CI −3 to 22). This short time indicates that, for some countries, early BA.1 transmission was not represented in the PCR dataset (appendix 4 p 18).

Combining all country-level PCR data from Africa, the $R_t$ of BA.1 was 2·60 (95% CI 2·46–2·71) in the 40 days before BA.1 became the dominant variant and decreased to less than 1 within 29 days after BA.1 became dominant (average of 1·92 across the 40 days before and 40 days after the day of predominance; figure 3A, appendix 4 p 7), probably due to widespread immunity following the rapid spread of BA.1.[21] This interpretation is in line with

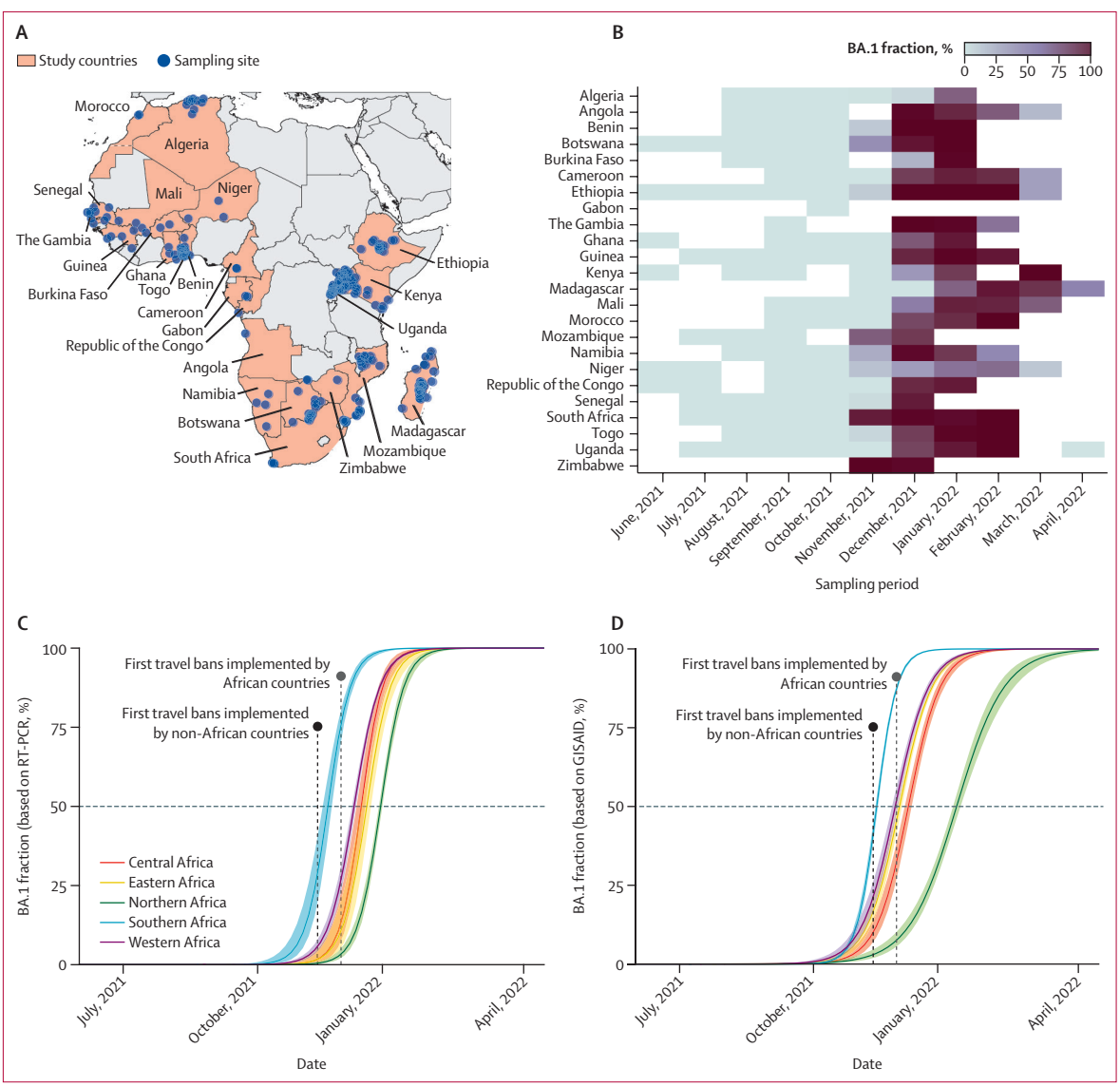


***Figure 1:*** **Study location and epidemiology of BA.1 in Africa**
(A) Countries represented in the study and the geographical distribution of sampling sites. (B) Proportion of samples testing positive for the omicron (B.1.1.529) BA.1 marker in each country over time, after excluding 24 potentially contaminated samples. (C) Modelled increase in the BA.1 fraction of all SARS-CoV-2 infections in each African region, based on PCR testing data. The curves for central and eastern Africa overlap. (D) Modelled increase in the BA.1 fraction of all SARS-CoV-2 infections in each African region, based on GISAID entries. In C and D, the heavy lines represent the estimated BA.1 fraction and the shaded areas indicate the 95% CI; samples collected more than 10 days after the plateau of the BA.1 fraction was reached were excluded for each region. GISAID=Global Initiative on Sharing All Influenza Data.

| | Total number of samples | Delta (B.1.617.2) variant | BA.1 (B.1.1.529.1) variant | Other variant | SARS-CoV-2 negative | Variant unclear | Samples included in analyses* | Collection date range | Number of infections reported | Percentage of reported infections included |
|---|---|---|---|---|---|---|---|---|---|---|
| Algeria | 901 | 733 | 166 | 2 | 0 | 0 | 901 | Aug 18, 2021–Jan 30, 2022 | 62 111 | 1·5% |
| Angola | 671 | 57 | 471 | 80 | 63 | 0 | 608 | Aug 24, 2021–March 31, 2022 | 53 093 | 1·1% |
| Benin | 1762 | 269 | 1234 | 167 | 91 | 1 | 1670 | Aug 2, 2021–Jan 27, 2022 | 18 056 | 9·2% |
| Botswana | 231 | 38 | 169 | 24 | 0 | 0 | 231 | June 12, 2021–Jan 16, 2022 | 178 198 | 0·1% |
| Burkina Faso | 197 | 40 | 146 | 11 | 0 | 0 | 197 | Aug 21, 2021–Jan 28, 2022 | 6916 | 2·8% |
| Cameroon | 708 | 335 | 220 | 153 | 0 | 0 | 708 | Sept 1, 2021–March 25, 2022 | 36 119 | 2·0% |
| Ethiopia | 158 | 54 | 12 | 68 | 24 | 0 | 134 | June 1, 2021–March 7, 2022 | 197 466 | 0·1% |
| Gabon | 298 | 189 | 0 | 109 | 0 | 0 | 298 | Oct 1–20, 2021 | 5370 | 5·5% |
| Ghana | 542 | 60 | 145 | 73 | 54 | 210 | 278 | June 12, 2021–Jan 21, 2022 | 60 447 | 0·5% |
| Guinea | 357 | 157 | 188 | 12 | 0 | 0 | 357 | July 6, 2021–Feb 17, 2022 | 12 483 | 2·9% |
| Kenya | 238 | 27 | 15 | 22 | 101 | 73 | 64 | June 2, 2021–March 25, 2022 | 152 266 | <0·1% |
| Madagascar | 1091 | 680 | 130 | 26 | 255 | 0 | 836 | Nov 11, 2021–April 11, 2022 | 20 417 | 4·1% |
| Mali | 1137 | 187 | 648 | 103 | 198 | 1 | 938 | Aug 2, 2021–March 2, 2022 | 15 804 | 5·9% |
| Morocco | 994 | 551 | 440 | 3 | 0 | 0 | 994 | Sept 1, 2021–Feb 22, 2022 | 298 636 | 0·3% |
| Mozambique | 210 | 64 | 68 | 45 | 30 | 3 | 177 | July 8–Dec 11, 2021 | 70 292 | 0·3% |
| Namibia | 486 | 31 | 288 | 75 | 92 | 0 | 394 | Aug 30, 2021–Feb 23, 2022 | 32 344 | 1·2% |
| Niger | 733 | 46 | 366 | 321 | 0 | 0 | 733 | June 27, 2021–March 17, 2022 | 3310 | 22·1% |
| Republic of the Congo | 95 | 7 | 37 | 51 | 0 | 0 | 95 | June 2, 2021–Jan 12, 2022 | 10 760 | 0·9% |
| Senegal | 1348 | 410 | 826 | 39 | 72 | 1 | 1275 | July 19–Dec 31, 2021 | 22 959 | 5·6% |
| South Africa | 412 | 162 | 239 | 11 | 0 | 0 | 412 | July 1, 2021–Feb 8, 2022 | 1 633 456 | <0·1% |
| The Gambia | 187 | 7 | 174 | 6 | 0 | 0 | 187 | Sept 1, 2021–Feb 22, 2022 | 2226 | 8·4% |
| Togo | 1014 | 480 | 533 | 0 | 0 | 1 | 1013 | Aug 1, 2021–Feb 22, 2022 | 20 957 | 4·8% |
| Uganda | 823 | 408 | 189 | 115 | 10 | 101 | 712 | July 9, 2021–April 14, 2022 | 78 454 | 0·9% |
| Zimbabwe | 96 | 0 | 80 | 2 | 13 | 1 | 82 | Nov 24–Dec 13, 2021 | 33 466 | 0·2% |

*Samples with missing information on collection date, those that were negative for SARS-CoV-2 on re-testing or had unclear results regarding variant, those without unique sample IDs, and those collected before June 1, 2021 (as BA.1 was unlikely to be circulating at that time) or after April 30, 2022, were not included in the analyses. The 24 samples excluded from epidemiological analyses are included in the descriptive analyses presented here. Samples with missing age or sex data were included in the analyses as both variables were not considered essential.

***Table:*** **Summary of molecular results and sample collection**

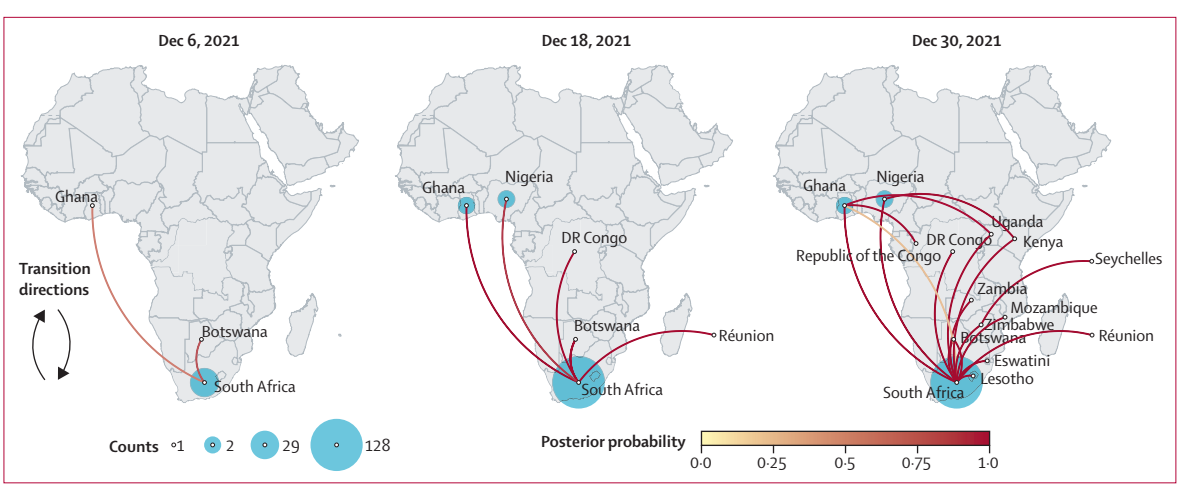


***Figure 2:*** **Phylogeographical analyses of the spread of BA.1 in Africa**

BA.1 sequences from Africa available on GISAID and two BA.1 sequences from Benin generated in this study were filtered by at least 95% genome coverage. Of 1550 genomes, 1456 were included in the analysis after removing outliers (appendix 4 p 22). Removed outliers included two Benin-derived sequences generated in this study. Transitions and reward counts (counts of transitions from or to a distinct place) are shown for three timepoints. The first timepoint was chosen when two transitions were completed; the second timepoint was 12 days after the first and the third timepoint was 24 days after the first. Only completed transitions are shown. For details of the methods, see appendix 4 (p 1). GISAID=Global Initiative on Sharing All Influenza Data.

the steep increase in reported infections, probably corresponding to the short duration of the BA.1 wave in Africa (appendix 4 p 18).[11,21] Excluding five countries that reported SARS-CoV-2 infections inconsistently and excluding travellers from the PCR dataset had nearly no effect on $R_t$, showing the robustness of our approach (figure 3B). Retrospective analyses of more than 67 800 SARS-CoV-2 genomes available in GISAID by mid-2023 revealed that, within 1 week of the first report of omicron BA.1 to WHO on Nov 24, 2021, it had spread to all African regions except northern Africa (figure 4, appendix 4 p 19). Genomic sequence-based and PCR-based data were therefore consistent with the early occurrence of BA.1 across Africa.

The rapid spread of SARS-CoV-2 is known to be facilitated by human mobility, including both short-distance and long-distance travel.[22] The importance of long-distance travel for the spread of BA.1 was consistent with its detection among inbound travellers in our study. From the ten countries for which information on the testing of travellers was available, we included samples from 2789 travellers. By Nov 24, 2021—when BA.1 was first reported to WHO—inbound travellers departing from Burkina Faso and Nigeria had already tested positive for the variant in Togo and one inbound traveller from Mauritania had tested positive in Senegal. 2 weeks later, inbound travellers tested positive for the variant in Algeria and Niger. The travellers testing positive for BA.1 before Dec 8, 2021 had departed from diverse locations on five continents (appendix 4 p 8), highlighting the rapid global spread of BA.1 and suggesting that the variant emerged several weeks before it was first detected.

To evaluate the predictability of BA.1 spread, mobility-based mathematical simulations based on a susceptible–exposed–infectious–recovered model were calculated using GLEAMviz, which has been used previously to study the global dispersion of SARS-CoV-2.[22] The overall best accordance between the estimated number of BA.1 infections (based on PCR data) and the simulated number of BA.1 infections was observed in a simulation that assumed 10 times more infected individuals than the model informed by reported cases and the determined BA.1 fraction at the start of the simulation and literature-derived model parameters (appendix 4 p 20), suggesting substantial under-reporting of SARS-CoV-2 infections in line with WHO estimates.[3] In this simulation, the emergence of BA.1 (one BA.1 infection in 100 000 inhabitants) was first predicted in southern Africa (by Nov 30, 2021). By comparison, BA.1 emergence was delayed by 26 days in eastern Africa, 35 days in central Africa, and roughly 2 months in western and northern Africa (appendix 4 p 10). The modelled south–north gradient concurred with our continent-wide PCR data (figure 1C). Conversely, the simulation deviated from PCR-based data and GISAID data regarding the emergence of BA.1 in eastern Africa

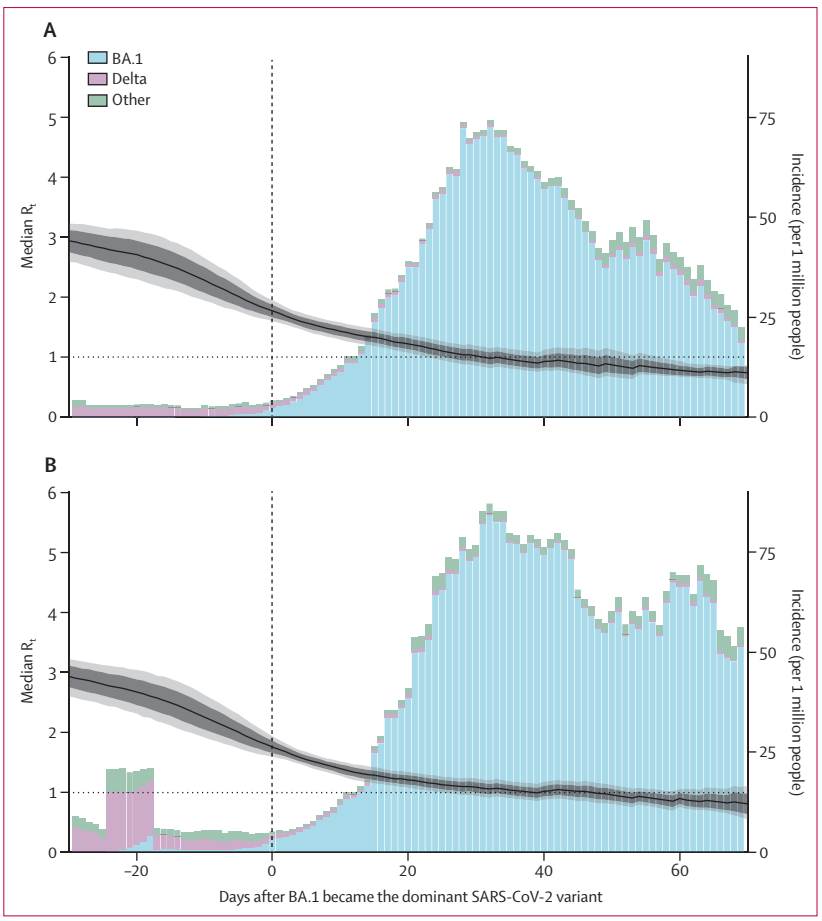


***Figure 3:*** **Speed of BA.1 spread**
(A) Estimated incidence and smoothed $R_t$ of BA.1 over time, using data from 18 African countries that continuously reported cases during the study period. (B) Estimated incidence and smoothed $R_t$ of BA.1 over time, using data from 18 African countries excluding infections recorded in travellers (appendix 4 p 23). The black lines represent the median $R_t$, the dark grey shading indicates the 75% CI, and the light grey shading indicates the 95% CI. $R_t$=effective reproduction number.

(later in PCR and GISAID data than in model data) and in western Africa (earlier in PCR and GISAID data than in model data; figure 5A, B; appendix 4 p 6). These discrepancies could be a consequence of mobility restrictions affecting eastern Africa more than western Africa and the early introduction of BA.1 into western Africa by long-distance travel, as suggested by our PCR data and phylogeographical reconstructions (figure 2, appendix 4 p 19). Overall, the simulated BA.1 spread was slower than the spread according to case data. The relatively slower simulated spread could be due to underestimation of the number of BA.1 infections at the start of the simulation, which is in line with early BA.1 detection outside of southern Africa in both our molecular data and GISAID entries. The GLEAMviz simulation that assumes a ten-times greater number of infected individuals at the start of the simulation was significantly correlated with both PCR-based estimated infections ($p=0·016$, $r=0·57$) and with GISAID entries ($p=0·0010$, $r=0·51$; figure 5C–E). Moreover, the simulated

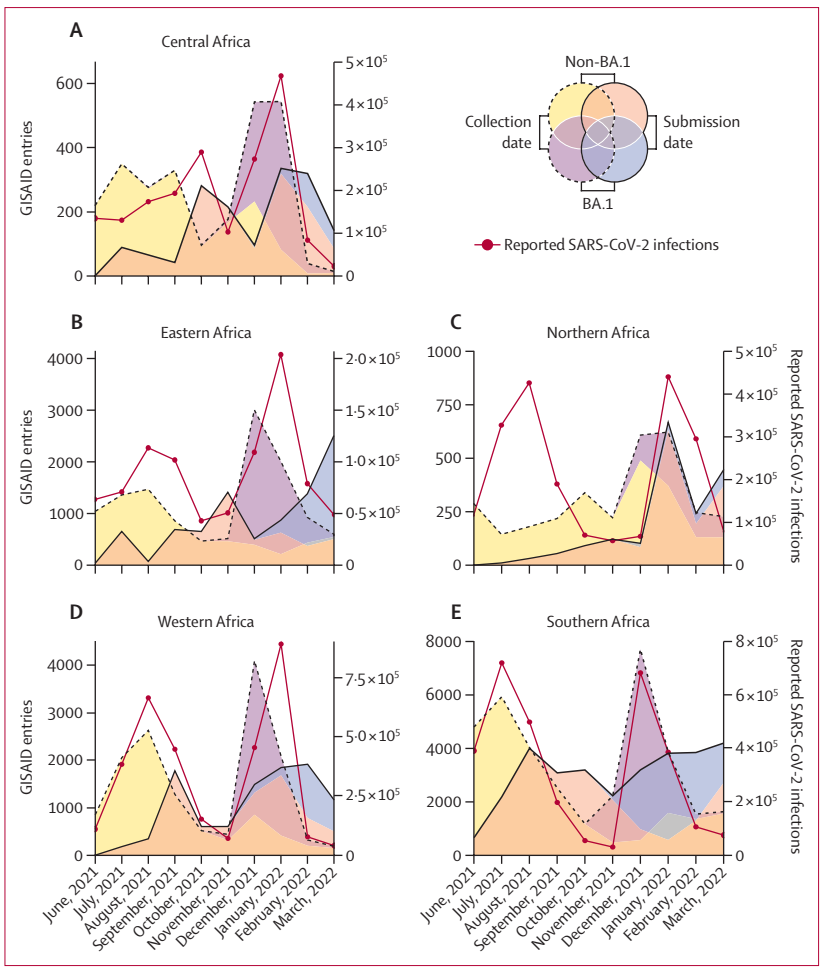


***Figure 4:*** **SARS-CoV-2 sequencing and genome submissions**
BA.1 and non-BA.1 sequences submitted to GISAID, by collection and submission date, from central Africa (A), eastern Africa (B), northern Africa (C), western Africa (D), and southern Africa (E). GISAID=Global Initiative on Sharing All Influenza Data.

BA.1 introduction date in countries outside of Africa was significantly earlier in countries that reported BA.1 infections by mid-December, 2021—when first estimates of global BA.1 spread had become available[23]—than in countries that did not report early BA.1 infections (Kruskal–Wallis test, p<0·0001; appendix 4 p 21).

Surveillance of SARS-CoV-2 variants is commonly done by HTS-based full-genome generation of selected samples. However, this approach is relatively costly and time-consuming. Considering standard procedures and products, genotyping a single sample by HTS—which also requires bioinformatics—was approximately six times more expensive ($49·86) and three times more time-consuming (5·6 min) than PCR-based typing ($7·82, 1·9 min), which requires only laboratory personnel but no bioinformaticians (appendix 4 p 11). A time-based analysis of 67 821 GISAID entries showed that the median time between sample collection and submission of the sequence to GISAID ranged between 38 days and 132 days for the African regions, irrespective of whether the entries deposited were BA.1 or non-BA.1 variants (appendix 4 p 21). The comparative, time-stamped analysis showed that there were no delays in the reporting of BA.1 sequences by African countries or supranational organisations, which could hypothetically have resulted from a fear of unilaterally imposed travel restrictions.

## Discussion

We provide strong evidence that BA.1 spread rapidly across Africa and beyond before travel restrictions, which are most efficient at low case numbers,[24] were implemented for African countries. The PCR data show a south–north gradient of BA.1 spread in Africa that was substantiated by GISAID entries and phylogeographical reconstructions. Our results indicate that BA.1 emerged and spread before its first detection, in line with retrospective analyses of BA.1 genomes from England[18] and phylogenetic analyses reconstructing the most recent common ancestor of BA.1 to early October, 2021.[5] The reconstructed $R_t$ of BA.1 according to our PCR data was lower than an average $R_t$ of 3·4 for BA.1 reconstructed across several countries in Africa, the Americas, Asia, and Europe.[25] The relatively lower $R_t$ in our study could be a consequence of under-reporting, particularly in times of high infection numbers, which would also explain the observed decrease of $R_t$ when infection numbers increased and when using different serial interval estimations, which vary between 3·0 days and 5·5 days in the literature.[16] Notably, in our $R_t$ analyses, the consideration of constant and systematic under-reporting had little effect on $R_t$ calculations (appendix 4 p 24).

Comparing our observational results with other data enables conclusions on preparedness measures to be derived. The agreement between our PCR data generated during the BA.1 outbreak and retrospectively analysed GISAID entries shows that PCR tests can efficiently contribute to the surveillance of emerging SARS-CoV-2 variants. HTS requires access to technical infrastructure that is often unavailable, resulting in delays between sample collection and the availability of sequencing results, whereas PCR testing is a complementary technique that offers faster testing using widely available equipment.[26] The correlation between the simulated and observed BA.1 introductions also shows the overall usefulness of mobility-based simulations for predicting the global spread of an emerging SARS-CoV-2 variant, particularly for identifying countries at the highest risk of importing the variant.

After the emergence of any new SARS-CoV-2 variant, understanding its virulence, speed of transmission, ability to evade pre-existing immunity, and spatial distribution is crucial. For all four factors, the ability to identify a new variant is pivotal. Sequencing 0·5% of all infections within 21 days after sample collection has been

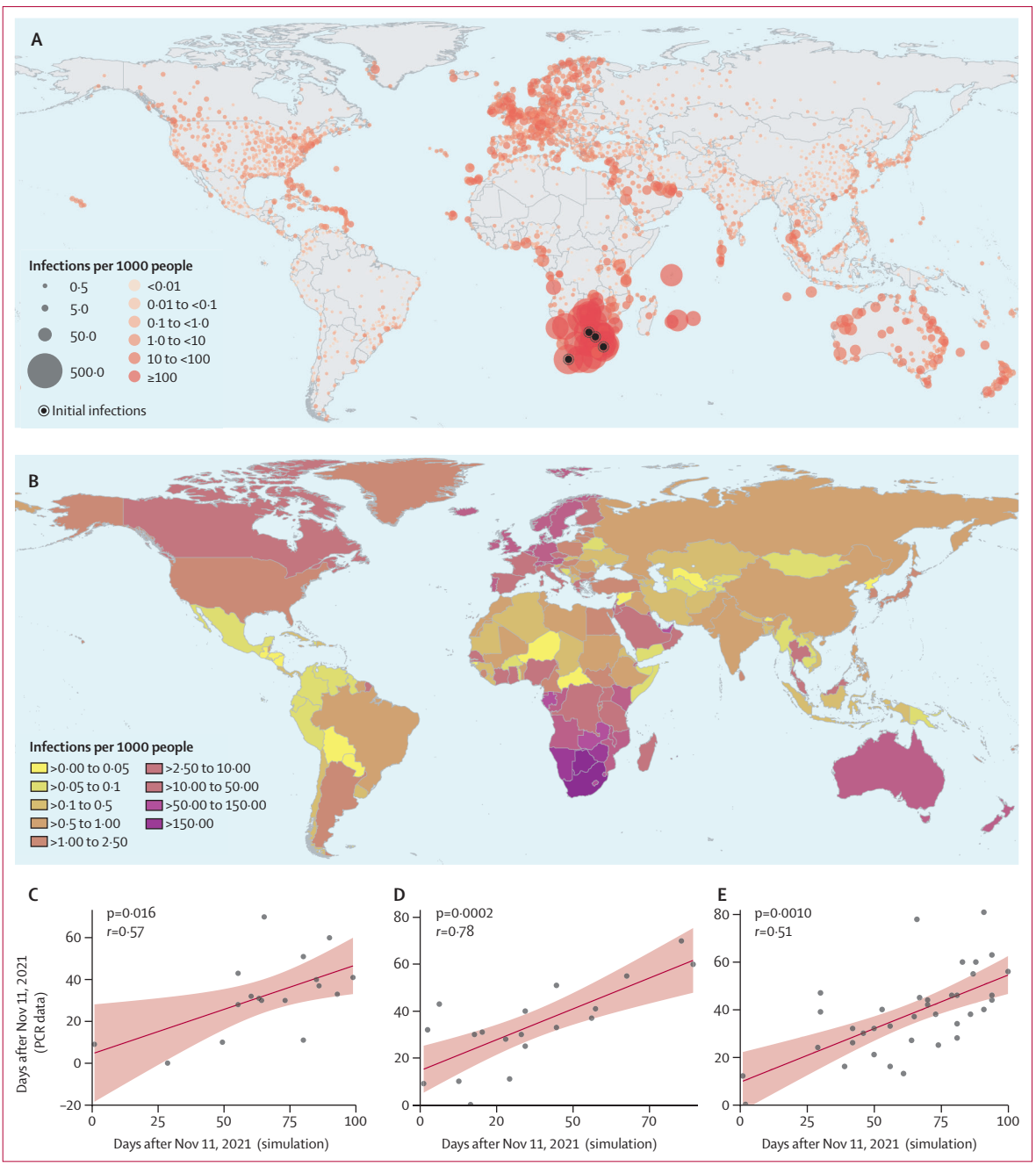


***Figure 5:*** **Simulated global spread of BA.1 and correlation with molecular testing**
(A) Simulated number of BA.1 infections by Feb 28, 2022 on the municipality level. The defined initial infections are shown by black dots. (B) Simulated number of BA.1 infections by Feb 28, 2022 on the country level. For simulation setups for A and B, see appendix 4 (p 16). (C) Correlation between days after Nov 11, 2021 (when BA.1 became the dominant SARS-CoV-2 variant) in models based on our PCR data and when one BA.1 infection was simulated in 5000 inhabitants using GLEAMviz. (D) Correlation between days after Nov 11, 2021 (when BA.1 became the dominant variant) in models based on our PCR data and when at least 20 BA.1 sequences were deposited in GISAID (collection date). (E) Correlation between days after Nov 11, 2021 when at least 20 BA.1 sequences were deposited in GISAID (collection date), and when 1 BA.1 infection was simulated in 5000 inhabitants using GLEAMviz. Only countries for which BA.1 dominance was estimated to occur in November, 2021, or later were considered in C and D to reduce the effect of non-representative data. All values in C–E are on the country level. All correlations in C–E were calculated by Pearson's correlation. GISAID=Global Initiative on Sharing All Influenza Data.

estimated to provide a good chance of efficiently detecting new SARS-CoV-2 variants.[26] However, establishing efficient genomic surveillance infrastructures is a major economical and organisational challenge. Despite globally increasing sequencing capacities, only 5% of low-income and middle-income countries have reached this

benchmark of sequencing 0·5% of infections, and some African countries still rely on external capacities for genome sequencing and submission to online databases.[26] Moreover, SARS-CoV-2 infections are probably widely under-reported in Africa,[3] causing sampling biases that could also affect genomic surveillance. To allow for interventions that are efficient in resource-limited settings,[27,28] strengthening and harmonising surveillance systems on a supranational level—including external quality assessments[29,30] and strategic sampling and data sharing frameworks—is therefore crucial.[31] We show that real-time RT-PCR tests specifically designed for genotyping have the potential to strengthen the surveillance of newly identified SARS-CoV-2 variants before genomic sequencing can be scaled up. Notably, molecular typing assays such as the one we used might be most valuable immediately after the emergence of a variant, because tested markers could emerge convergently in lineages other than the target lineage[20] and because the risk of laboratory contamination increases after substantial amplification for the sequencing of a new variant has been done,[20] particularly when testing for one genomic marker only. The application of RT-PCR tests for genotyping therefore requires parallel randomised confirmatory testing by HTS in case a marker is lost or acquired by other lineages.

Our findings have three main implications regarding the preparedness for new SARS-CoV-2 variants in resource-limited settings. First, molecular test capacities—including HTS-based and PCR-based surveillance—should be upscaled to enable the early detection of new variants. Wastewater testing could be included to provide cost-efficient information on variants, even in settings with limited sewage systems.[32] Second, after the emergence of a new variant, real-time RT-PCR assays for variant typing can be rapidly designed, validated, produced, and distributed across a region. Third, on the basis of the typing results, mobility-based simulations can be applied to predict the countries at the highest risk of importing the new variant by land or air travel. The identification of these countries is essential to reduce the risk of dispersion from secondary hubs, best exemplified by the global spread of BA.1 predominantly from high-income settings outside of Africa with intense flight connectivity, such as the USA.[33] Importantly, such mobility-based simulations are dependent on regularly updated mobility information and could require continuous funding from supranational actors. Complementary surveillance strategies combined with modelling-based spread predictions could therefore enable timely implementation of evidence-based measures, such as travel restrictions[22,34]—including a decision against their implementation should wide spread of the variant have already occurred.

Our study is limited by heterogeneous sampling in time and space and by the lack of SARS-CoV-2 genomic data from all individuals who tested positive for BA.1 by PCR between June 1, 2021 and April 30, 2022. However, the PCR test was extensively validated by HTS in four countries and geographically widespread testing substantiates the robustness of our findings. As this study was limited to samples from Africa, we cannot exclude a BA.1 origin outside of Africa. However, the wide and early spread of BA.1 in southern Africa observed in our study, the first detection of BA.1 in southern Africa,[5] and the available literature support the emergence of this variant in Africa.[35] Country-specific parameters, such as travel restrictions, could not be included adequately in our mobility-based models, potentially limiting their precision. Similarly, we did not optimise model parameters but applied a deterministic model approach to predict the spread of BA.1, as extensive knowledge on SARS-CoV-2 was available. The application of our approach to an entirely new pathogen would require comprehensive optimisation of model parameters and computationally demanding simulations. In preparation for future outbreaks, inclusion of traveller-associated demographic and socioeconomic data and non-pharmaceutical measures, such as contact restrictions, into programmes or open-source databases could enable rapid assessments of the effect of potential flight restrictions and could aid their precise implementation.

In conclusion, our results highlight that travel restrictions were implemented too late to be effective in substantially reducing the spread of BA.1. We show how PCR-based variant typing enables assessment of the spatiotemporal spread of a new SARS-CoV-2 variant rapidly and economically on a continent-wide scale. Such PCR tests and mobility-based models can contribute to containment strategies for future SARS-CoV-2 variants and other emerging pathogens.

**Contributors**

CF and JFD conceptualised the study. CF and JFD developed the methods. CF and AF curated the data. CF, TGM, AY, and JFD accessed and validated the data. CF, AF, and JFD conducted formal analyses. TGM, AY, NA, EA, PAd, PAf, JA, SFA, LA, YA, MAB, AB, RB, ALMB, FB, MC, PC, RMC, JC, GC, AC, UD'A, XdL, JFMdM, FD, ND, YD, LD, PD, RE, AE, OF, TF, AH, PV-I, NI, RJ, SJ, BK, JK, LK, OK, VL, AL, OL, SEL-D, JBL-D, EI, HL, JL, SMa, IM, BM, PAM, JMa, LM, JMw, NN, CAN, MON, EN, RN, JN, SGN, EOO, AO, JBO, MO, IOD, KKP, ROP, WP, VR, FS, SS, AASa, AASy, PAT-N, ZT, FOT, TBT, PR, NT, SMo, FC, and WC contributed to sample collection, sample storage, and supply of consumables. CF, TGM, AY, AM-S, NA, EA, PAd, PAf, JA, SFA, LA, YA, MAB, AB, RB, ALMB, FB, MC, PC, RMC, JC, GC, AC, UD'A, XdL, JFMdM, FD, ND, YD, LD, PD, RE, AE, OF, TF, AH, PV-I, NI, RJ, SJ, BK, JK, LK, OK, VL, AL, OL, SEL-D, JBL-D, EI, HL, JL, TL, SMa, IM, BM, PAM, JMa, LM, JMw, NN, CAN, MON, EN, RN, JN, SGN, EOO, AO, JBO, MO, IOD, KKP, ROP, WP, VR, FS, SS, AASa, AASy, PAT-N, ZT, FOT, TBT, FC , PR, NT, WC, SMo, and SG conducted and analysed the results of experiments, particularly PCR tests. CF and JFD wrote the original draft of the manuscript, which was reviewed and edited by CF, TGM, AY, and JFD. CF prepared the figures. TGM, RB, JFMdM, CD, IOD, KKP, ROP, AASy, SMo, and JFD acquired funding. AK and JFD were responsible for project administration. JFD and CF supervised the project. All authors had full access to all the data in the study and had final responsibility for the decision to submit for publication. Contributing authors are listed alphabetically.

### Equitable partnership declaration

The authors of this paper have submitted an equitable partnership declaration (appendix 5 p 2). This statement allows researchers to describe how their work engages with researchers, communities, and environments in the countries of study. This statement is part of *The Lancet Global Health*'s broader goal to decolonise global health.

### Declaration of interests

OL is the former owner of TIB Molbiol, the company that produced the kits provided to African partner laboratories in this study. The kits are not commercially available. All other authors declare no competing interests.

### Data sharing

Scripts for data analyses and data are available at GitHub (https://github.com/CarloFischer88/Analyse-the-BA.1-spread-in-Africa.git). Phylogenetic analyses of BA.1 sequences from Africa (appendix 4 p 19) and phylogeographical analyses (figure 2, appendix 4 p 22) are based on 1544 sequences available on GISAID at https://doi.org/10.55876/gis8.240819dq. The performance of the real-time RT-PCR assays was validated against SARS-CoV-2 genomes generated by HTS (https://github.com/CarloFischer88/Analyse-the-BA.1-spread-in-Africa.git). Those genomes that were submitted to GISAID (from Botswana, Guinea, and South Africa) are available at https://doi.org/10.55876/gis8.240201eb. Mapped genomic reads for SARS-CoV-2 genomes generated from samples from Benin are available via the European Nucleotide Archive (project accession number PRJEB64297).

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See **Online** for appendix 5

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