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Characterising household transmission dynamics of clade Ib mpox in Burundi: a prospective cohort study

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Summary

Background Knowledge of intrahousehold transmission dynamics of clade Ib mpox, especially in recently epidemic African contexts, is scarce. Our study aimed to quantify household transmission patterns of clade Ib mpox in Burundi, with a focus on children.

Methods We conducted a prospective cohort study in two health districts, Bujumbura and Kayanza, in Burundi from Jan 23 to March 20, 2025, enrolling 88 laboratory-confirmed primary mpox cases and 432 of their household contacts. We estimated household secondary attack rates (SARs), serial intervals, and basic reproduction number (R_0), including a sensitivity analysis to assess the effect of potential misclassification of mpox index cases younger than 15 years. The primary outcome was occurrence of a secondary mpox infection within the household, defined as any laboratory-confirmed mpox case identified among contacts during the follow-up period.

Findings Of the 88 households, 18 (20%) experienced secondary transmission, with most primary mpox cases generating a single secondary case. The overall SAR across all households was 6·15% (95% CI 4·02–8·95) and was significantly higher among those younger than 15 years (8·77% [5·44 –13·22]) than among those aged 15 years or older (2·84% [0·92–6·50]). The overall R_0 was 0·30 (95% CI 0·17–0·46), and was significantly higher for those younger than 15 years (0·43 [0·21–0·70) than those aged 15 years or older (0·15 [0·03–0·27]). The sensitivity analysis showed significantly higher estimates (R_0 0·9 [0·71–1·09]; SAR 17% [13·57–21·03]).

Interpretation Intrahousehold transmission of clade 1b mpox in Burundi was limited, and unlikely to sustain a broader community spread. The involvement of children in transmission chains within the household underscores their vulnerability, emphasising the need for accurate household investigation, early detection, and strategies to protect them. Our findings suggest that infection outside the household, with adults serving as a source for initial household introductions, might be a primary driver of the outbreak. The mpox outbreak response should adopt a dual approach combining interventions for household settings, and targeted prevention strategies for adults at risk where community transmission is more probable.

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Introduction

A distinct monkeypox virus lineage, clade Ib, emerged in the Democratic Republic of the Congo in late 2023, and has shown evidence of increased human-to-human transmissibility.[1] As of May 1, 2025, the clade Ib outbreak has spread to 15 countries worldwide, including 11 in Africa.[2] In response to the resurgence and geographical spread of mpox in Africa, the Africa Centres for Disease Control and Prevention (Africa CDC) declared the outbreak a Public Health Emergency of Continental Security,[3] followed by a renewed Public Health Emergency of International Concern declaration by WHO in August, 2024.[4]

Burundi reported its first case of clade Ib mpox on July 25, 2024, epidemiologically linked to the outbreak in the Democratic Republic of the Congo.[5] By May 1, 2025, 3800 confirmed cases of mpox had been recorded across 46 of the country's 49 administrative districts.[2]

Mpox transmission occurs through direct contact with lesions, body fluids, or contaminated materials.[6,7] Although sexual or intimate contact was an important route of transmission during the 2022 global outbreak, other forms of contact also contribute to the transmission of the disease.[8] The unprecedented global and regional spread of monkeypox virus has revealed crucial knowledge gaps concerning mpox transmission dynamics and risk factors, particularly in African settings experiencing endemic transmission or rapidly expanding outbreaks due to clade Ib.

Children have been disproportionately affected by clade Ib in African settings.[9–11] In Burundi, children younger than 15 years accounted for approximately 34% of all confirmed cases, whereas in the Democratic

Research in context

Evidence before this study

We searched PubMed for the term "mpox", "monkeypox", or "Clade Ib" and, successively "Household transmission", "Reproduction number", "Serial interval", and "Secondary attack rate" for articles published in the indexed literature up to June 20, 2025, the date of our search. The search was limited to articles with English abstracts available on PubMed.

We identified 15 studies published between 1980 and 2025 that reported secondary attack rates (SARs) across various mpox outbreaks in both endemic African countries and global contexts. Nine articles focused on Africa (Democratic Republic of the Congo, Central African Republic, Nigeria, and historical reports from Zaire and Kenya), with household SARs ranging from 3% to 59·5%, reflecting differences in exposure settings, contact intensity, immunity profiles, and probably surveillance or data quality. One of these studies reported a SAR of 2% with data from the Kenya clade Ib outbreak. Estimates of mpox reproduction numbers (R_0 or R_t) have increased during the global outbreak, particularly in Europe and the USA, with most studies focusing on clade II. Of the 50 studies we reviewed, few specifically addressed the transmission potential of clade I. Marziano and colleagues estimated an R_t for clade I in the Democratic Republic of the Congo at 0·82 (95% CI 0·79–0·85) based on 2013–17 surveillance data, with R_t ranging from 1·08 to 1·18 (uncertainty range 0·96–1·27) for clade Ib in South Kivu province. Charniga and colleagues using 2010–19 surveillance data from Tshuapa (Democratic Republic of the Congo), estimated R_t to be 0·85, with an uncertainty range of 0·51–1·25. 16 studies reported the serial interval of mpox, none related to clade Ib. A systematic review and meta-analysis by Brochero and colleagues, based on the 2022–23 global outbreak, estimated a pooled serial interval of 8·30 days (95% CI 6·74–10·23). Additional estimates derived from clade II outbreaks in non-endemic settings were available, including in the USA, UK, the Netherlands, Peru, and Gulf countries, and might not apply to transmission patterns associated with clade Ib.

Added value of this study

Our study provides, to our knowledge, some of the first empirical assessments of household transmission of clade Ib mpox in an African epidemic. We found that household transmission of clade Ib mpox in Burundi is limited, affecting mostly children. Our study supports emerging evidence that extra-household transmission, particularly among adults, probably drives community spread and is potentially a source of household introduction.

Implications of all the available evidence

Public health response should adopt a dual approach combining household-based interventions that prioritise early detection, protection for children, and targeted interventions focused on adults and high-risk groups that aim to reduce transmission through close physical contact, including reducing transmission through sexual contact.

Republic of the Congo, surveillance data indicated that children younger than 5 years represent a proportion as high as 80% of all reported mpox-related deaths.[12] This high burden among children suggests both heightened vulnerability and a potentially important role in transmission dynamics, including within households.

However, knowledge about household transmission related to clade Ib in affected African countries remain relatively scarce. A systematic review by Beer and colleagues[13] estimated that pooled secondary attack rate (SAR) among unvaccinated household contacts of confirmed mpox cases (clade non-specified) ranged from 0% to 11%, with an overall average of 8% during past outbreaks in endemic settings. Marziano and colleagues[14] found a mean estimate for the effective reproduction number (R_t) in Kamituga (eastern Democratic Republic of the Congo) during the clade Ib outbreak ranging from 1·08 to 1·18 using publicly available outbreak data. More recently, Mutuku and colleagues[15] reported a SAR of 2% from the clade Ib outbreak in Kenya.

Given the burden among children during the 2024 clade Ib outbreak and the knowledge gaps surrounding transmission, UNICEF and the National Institute of Public Health (INSP) of Burundi jointly designed and implemented a study to guide the country's mpox response. Our study aimed to quantify the household transmission dynamics of clade Ib mpox in Burundi by estimating key epidemiological parameters, assessing the role of children, and identifying factors associated with mpox transmission within household settings.

Methods

Study design and participants

The study protocol was adapted from the WHO generic protocol for investigating mpox transmission within households and close-contact settings (unpublished). A comprehensive version of the protocol for this study has been previously published.[16]

This prospective cohort study (enhanced surveillance) was done in two purposively selected health districts of Bujumbura (urban) and Kayanza (rural), Burundi, chosen for their high mpox burden and operational feasibility.

The study targeted households with at least one confirmed mpox case and at least one child younger than 15 years. A convenience sample of 100 eligible individuals considered as primary cases of mpox was identified from the three mpox treatment centres serving the study districts. Consecutive patients were screened and approached for enrolment on the basis of the following criteria: laboratory-confirmed mpox; residence in one of the two districts; household eligibility; and willingness to

participate [A: is this the full list of eligibility criteria? And do they exactly match your protocol?].

With the consent of the primary case (or their guardian) secured, permission was obtained from the head of household to enrol the household, followed by consent from individual members or guardians (for those aged <15 years).

Household contacts were individuals, related or unrelated, living under the same roof as the primary mpox case, sharing meals, and recognising the authority of a head of household, with at least one child younger than 15 years (including the index case). To be eligible for follow-up, the contact should have been residing in the same household as the primary mpox case during the infectious period (about 21 days, from symptom onset to complete resolution of lesions). Based on Burundi's average household size of five,[17] approximately 500 contacts from the 100 expected households were to be followed for 21 days after enrolment.

The study protocol was reviewed and approved by the National Ethics Committee of Burundi (CNE/34/2024). Written informed consent was obtained from all participants before enrolment. For minors, consent was obtained from a parent or legal guardian. All data were anonymised before analysis to ensure participant confidentiality, and no personally identifiable information was retained in the final dataset.

Procedures

For each primary case, sociodemographic and clinical information, including symptom onset, laboratory confirmation, and exposure history, were collected at enrolment by a health-care provider member of the study team. Data collection for primary cases took place at enrollment. Household-level data included occupancy, dwelling type, number of rooms, water source, sanitation, energy access, and distance to the nearest health facility. Each enrolled household and its contacts were visited every 7 days for a planned total of three visits from the time of enrolment. At each visit, a trained health-care worker examined household contacts for signs and symptoms of mpox (fever and skin rash). In addition to scheduled visits, contacts could notify the study team at any time to report the onset of symptoms suggestive of mpox.

All data were collected using WHO-adapted forms at case and household enrolment, and during each of the three weekly follow-up visits for each contact. Data were entered electronically using KoboCollect.[18] Data collection began on Jan 23 until March 20, 2025, over 7·5 weeks, reflecting staggered case enrolment.

Mpox case confirmation was done at the INSP Laboratory using real-time RT-PCR, following WHO guidance.[19] Contacts developing symptoms compatible with mpox during follow-up were classified as suspected mpox cases, with samples (skin lesions) collected and tested as per national guidelines. Those testing positive were classified as secondary cases. We collected sex data as indicated by the participant (as per the country surveillance guide).

Outcomes

The primary outcome was occurrence of a secondary mpox infection within the household, defined as any individual with laboratory-confirmed mpox identified among contacts during the follow-up period.

Statistical analysis

We summarised the clinical and sociodemographic characteristics of primary and secondary cases, household contacts, and household-level factors.

Overall and age-stratified (aged <15 years and ≥15 years) secondary attack rates (SARs) were calculated as the proportion of contacts who developed mpox within the follow-up period. We assumed no previous immunity from infection or vaccination, because mpox vaccination had not been introduced in Burundi at the time of the study.

Serial interval within the household was estimated as the time from symptom onset in the primary case to symptom onset in the secondary case.

We estimated the overall and age-stratified (age <15 years and ≥15 years) basic reproduction number (R_0) using a chain-binomial model.[20] To quantify uncertainty around R_0 estimates, we ran non-parametric bootstrapping with 1000 resamples to generate the 95% CIs.

For risk factors of household secondary transmission, logistic regression was used to identify individual-level and household-level characteristics associated with secondary transmission. The dependent variable was whether a household contact developed mpox (yes or no). Household-level variables included household crowding (number of residents per living area), and hygiene-related variables (access to water and type of sanitation facility).

For the sensitivity analysis we assessed the robustness of SAR and R_0 to potential misclassification of primary and secondary cases, especially among individuals younger than 15 years. Concerns of misclassification arose from scarce data on the infection source of primary and secondary cases, a high proportion of paediatric primary cases, and possible bias in case detection, where children might have been diagnosed earlier (and therefore identified as primary cases) than adults who delayed care seeking because of factors such as stigma. In our sensitivity analysis, we assumed that all individuals younger than 15 years considered primary cases were actually secondary cases.

Additional details of the analysis are provided in the appendix (pp 1–2). All analyses were done with R, version 2024.12.1.

Role of the funding source

The sponsor of the study (UNICEF) participated in the study design, data analysis, data interpretation, and writing of the manuscript, but not in data collection, which was done by the INSP of Burundi.

Results

See Online for appendix

Our study consecutively screened and enrolled 92 individuals considered to be primary cases. Three household heads withdrew their consent, and one family relocated from the study area, resulting in a total of 88 primary cases with complete household data on which the analysis was based (figure 1).

Primary cases were generally young, with 53 (60%) of 88 younger than 15 years. 50 (59%) of 85 were female, and they were overall older than male individuals (table 1).

Skin rash was reported among 73 (86%) of 85 primary cases. Rash in the genital area was reported among 27 (79%) of 34 individuals older than 15 years (26 [96%] aged ≥18 years), compared with 15 (29%) of 51 of those younger than 15 years, with no difference in sex. HIV serology was done for 29 (33%) of the 88 primary cases; two (all older than 15 years) tested positive for HIV. Primary cases were identified and enrolled into the study on average 6·9 days (range 0–8) after the reported onset of the disease. Overall, contact with a suspected case was reported for 24 (28%) of 85 primary cases; this proportion was 17 (33%) of 51 for those younger than 15 years and seven (21%) of 34 for those older than 15 years (appendix p 4). Eight (23%) of the 34 primary cases older than 15 years said that a potential source of infection could be sexual intercourse with someone with a skin rash in the genitalia, with 87% of these eight 18 years or older (*vs* none in those aged <15 years).

88 households with 534 residents were identified (mean number of residents per household was 6·0 [SD 2·5]). Most of the households were in areas classified as urban, and 82 (93%) of 88 were located within 5 km of a functional health facility. Households had a median of three (IQR 2–4) areas of living space (table 1).

Of the 534 residents listed in the 88 households, 432 (81%) consented to be enrolled for weekly monitoring by the study team, yielding an average of 4·40 contacts (range 1–13) per primary case.

Median age of household contacts was 17·0 years (IQR 7·0–31·0), with 201 (46%) of 432 younger than 15 years. Female individuals accounted for 253 (59%) of all 432 contacts and were, on average, older than male individuals, with a median age of 18·0 years (8·0–30·0) compared with 15·0 years (7·0–34·0) for male individuals.

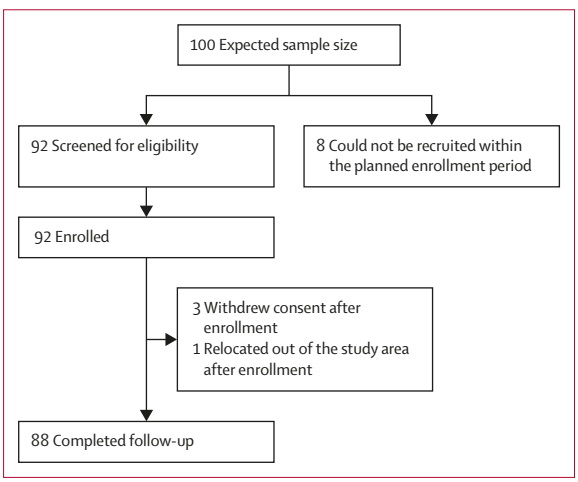

Figure 1: **Index cases and households enrolment flowchart**

	Value
Households (n=88)	
Number of living spaces	3·17 (1·37)
Within 5 km of a functional health facility	93% (82/88)
Access to pipe water	65% (57/88)
Number of residents per household	6·13 (2·63)
Primary cases (n=88)	
Age, years	
Overall (n=88)	13·6 (12·3)
Female (n=50)	15·4 (13·2)
Male (n=35)	10·9 (10·6)
Age <15 years	60% (53/88)
Sex	
Female	59% (50/85)
Male	41% (35/85)
Skin rash	86% (73/85)
Genital rash	
Age ≥15 years	79% (27/34)*
Age <15 years	29 % (15/51)
HIV serology (positive and tested cases only)	7% (2/29)
Days from onset to enrolment (median [range])	6·9 (0–8)
Reported sexual contact as exposure	
Age ≥15 years	23·5% (8/34)†
Age <15 years	0%
Household contacts (n=432)	
Age in years	
Overall (n=431)	17·0 (7·1–31·0)
Female (n=253)	18·0 (8·0–30·0)
Male (n=178)	15·0 (7·0–34·0)
Age <15 years	46·0% (201/432)
Sex	
Female	59·0% (253/432)
Male	41% (179/432)
Contacts enrolled per household (mean [range])	4·43 (2–13)
Completed at least two follow-up visits	93% (401/432)
Completed all three follow-up visits	71% (306/432)

Data are mean (SD), mean (IQR), or % (n/N), unless otherwise specified. Results were rounded up to the nearest integer. *96% [26/27] for individuals aged 18 years and older. †87·5% [7/8] for individuals aged 18 years and older.

Table 1: **General characteristics of households, primary cases of mpox, and their household contacts**

All 432 household contacts enrolled for monitoring had a cumulative number of 1189 visits, with all contacts having at least one visit; 401 (93%) of 432 had two visits and 306 (71%) completed the planned three visits. Contacts were enrolled, on average, within 7·24 days (range 1·0–19·0) from the day of onset of the skin rash in the primary case.

During follow-up, 25 secondary cases were identified across 18 of the 88 households (table 2, 3), representing an overall household-level secondary transmission rate of 20·45%, with household-specific secondary transmission rates varying between 0% and 70%. 21 (84%) of the secondary cases were detected within their first 2 weeks of monitoring and four (16%) in week 3.

The median age of secondary cases was 4·7 (IQR 2·92–12·65), and 20 (80%) of 25 were younger than 15 years. Secondary cases were slightly younger than primary cases, although this difference was not statistically significant (Wilcoxon rank sum test *w* 1242; p=0·20).

The difference in sex distribution between primary and secondary cases was not statistically significant (X^2 Yate's continuity correction 0·01; p=0·91; table 2, 3; appendix p 4).

Among the 18 primary cases who generated secondary cases (secondary case generators), 14 (79%) generated only one secondary case, whereas four (22%) generated more than one secondary case (four, three, two, and two secondary cases per primary case).

Secondary case generators had a median age of 7·81 years (IQR 4·38–17·80). They were younger than those who did not generate secondary cases (median age 8·99 years [3·11–24·36]). These differences were, however, not statistically significant (Wilcoxon rank sum test 612; p=0·59; table 2, 3).

Female individuals accounted for XX (67%) [A: complete absolute number] of the 18 primary cases that generated secondary cases, compared with XX (59%) among the 70 that did not cause secondary cases. This difference was not statistically significant (χ^2 0·12, df 1; p=0·72; table 2, 3; appendix p 5).

Of all the 426 household contacts in the study, 25 secondary cases were generated from 18 primary cases, yielding an mean SAR across all contacts in the 88 households of 6·15% (95% CI 4·02–8·95). Among primary cases younger than 15 years, the SAR was 8·77% (5·44–13·22), versus 2·84% (0·92–6·50) among those aged 15 years or older (p<0·005; [A: where should we cite table 4 now that it has been removed from here?] figure 3; appendix p 6).

The estimated R_0 across all households was 0·30 (95% CI 0·16–0·46; table 3). When stratified by age, for primary cases younger than 15 years, R_0 was 0·43 (0·21–0·70), and for those aged 15 years or older, R_0 was 0·15 (0·03–0·27).

The median time interval between primary and secondary cases (estimated using the date of onset of skin rash), was 13·50 days (IQR 10– 7), with 96% of secondary cases having a serial interval of 22 days or less (appendix p 7).

	Primary cases (n=88)	Secondary cases (n=25)
Mean age, years	13·55 (12·3)	10·50 (12·75)
Median age, years	8·5 (3·23–21·59)	4·7 (2·92–12·65)
Age <15 years	60% (53/88)	80% (20/25)
Sex		
Female	59% (50/85)	56% (14/25)
Male	41% (35/85)	44% (11/25)

Data are mean (SD), mean (IQR), or % (n/N). There were no statistically significant differences in demographic and transmission characteristics between primary and secondary cases or between primary cases that generated a secondary case and primary cases that did not. NA=not applicable.

Table 2: **Demographic characteristics of primary and secondary cases of clade Ib mpox**

	Generated a secondary case	Did not generate a secondary case
Proportion of primary cases	20% (18/88)	79% (70/88)
Generated only one secondary case	14 (84% [14/18])	NA
Mean age, years	11·5 (9·59)	15·0 (13·0)
Median age, years	7·81 (4·38–17·80)	8·99 (3·11–24·36)
Age <15 years	72% (13/18)	56% (39/70)
Sex		
Female	67% (12/18)	59% (41/70)
Male	33% (4/186)	41% (29/70)

Table 3: **Selected demographic characteristics of primary cases who generated and did not generate secondary cases of clade-Ib mpox**

	Value
Proportion of households with secondary transmission	20% (18/88)
Serial intervals, days (median [IQR])	13·50 (10–17)
Secondary attack rate (% [95% CI])	
Overall	6·15% (4·02–8·95)
Age <15 years	8·77% (5·44–13·22)
Age ≥15 years	2·84% (0·92–6·50)
Basic reproduction number (value [95% CI])	
Overall	0·30 (0·17– 0·46)
Age <15 years	0·43 (0·21–0·70)
Age ≥15 years	0·15 (0·03– 0·27)
Estimated transmission probability (p [95% CI])	
Overall	0·06 (0·04–0·08)
Age <15 years	0·09 (0·05–0·12)
Age ≥15 years	0·02 (0·01–0·06)

Table 4: **Secondary transmission and transmission dynamics of clade Ib mpox**

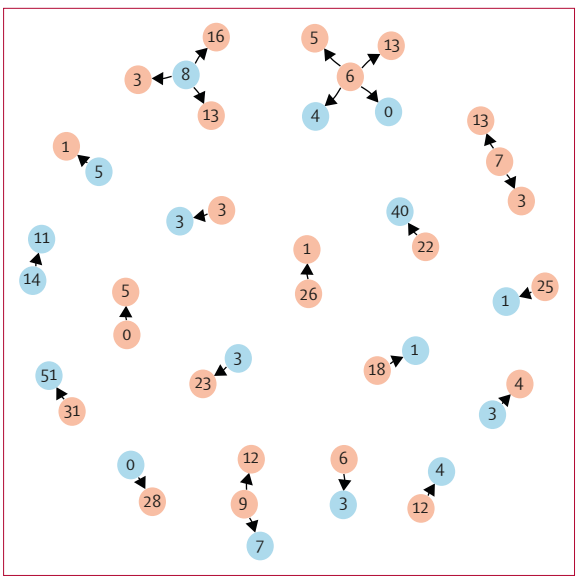

Figure 2: **mpox transmission trees by households**
Numbers in the circle indicate age (in years) of infector and infected **[A: we cannot use this type of language to refer to individuals; could you clarify what you mean here?]** individuals. Arrows from infector to infected person. Red indicates female individuals and light blue indicates male individuals.

All so-called superspreaders (people with primary mpox who generated more than one secondary case) were children aged 6 years, 7 years, 8 years, and 9 years (median age 7·81 [IQR 7·3–8·27]), younger than non-superspreaders (median age 9·53 years [7·3–8·27]); this difference was not statistically significant. Similarly, there was no significant association between sex and likelihood of superspreading (appendix p 5).

We did not find significant differences in household characteristics (amount of living space, number of residents, residents per living area, source of water, or urban or rural setting) between households with and without secondary transmission (appendix p 3).

In the primary analysis, household transmission was identified in 18 households, with 433 susceptible household contacts, yielding a transmission probability of 0·06 (95% CI 0·04–0·08) and an R_0 of 0·3 (95% CI 0·16–0·46). Unlikely direction of transmission (babies transmitting to adults with no clear source of contamination for the babies) between primary and secondary cases (figure 2) suggested possible misclassification or undetected adult sources. To account for this, a sensitivity analysis reclassified all primary cases younger than 15 years as secondary cases, and assumed a theoretical adult primary case. This increased the number of households with secondary transmission to 55, resulting in a three-fold increase in both transmission probability (0·18 [0·14–0·23]) and R_0 (0·9 [0·71–1·09]; appendix p 5).

Discussion

As of May 1, 2025, Burundi had reported nearly 4000 confirmed clade Ib mpox cases, including one death. Children younger than 15 years accounted for 39% of cases, highlighting their vulnerability, and suggesting a potential role in transmission.

We observed limited intrahousehold transmission of clade Ib mpox. Only 20·5% of households experienced secondary transmission, and most transmission chains ended after a single secondary case. These findings are consistent with a modelling study of the clade I outbreak in the USA, suggesting that sustained transmission chains within households are uncommon.[21] Our estimated SAR is consistent with those reported by Beer and colleagues,[13] from a systematic review where SARs ranged from 0% to 11%.

We found no significant associations between secondary transmission and household-level characteristics such as crowding, access to water, or urban versus rural settings. This finding suggests that close interpersonal contact, rather than environmental or structural household factors, is the dominant driver of transmission within households.

R_0 in our primary analysis was 0·30, lower than the threshold for sustained transmission. Our R_0 estimates differ from those of Marziano and colleagues,[14] who reported higher R_t values (1·08–1·18) based on household outbreaks of unspecified clade in Sudan, the Central African Republic, and a hospital outbreak in the Democratic Republic of the Congo. Differences in clade, setting, population structure, estimand, and methodology probably explain this divergence. By contrast, our findings align more closely with those of Charniga and colleagues,[22] who estimated an R_t of 0·82 for clade I mpox (subclade not specified) in Tshuapa Province, Democratic Republic of the Congo, where clade Ia predominates and with findings from other clade IIb outbreaks in non-endemic regions where SARs were similarly low. For example, Wendorf and collagues[23] found a SAR of 4·7% for clade IIb mpox among paediatric household contacts in CA, USA, whereas a US Center for Disease Control model predicted limited household transmission of clade I mpox (SAR ≤15%).[21] R_0 from the sensitivity analysis was three times higher than the baseline estimate, highlighting the potential underestimation of household transmission due to misclassification of mpox cases in individuals younger than 15 years. However, even under this scenario, R_0 remained lower than 1, reinforcing that intrahousehold transmission alone is unlikely to sustain large or prolonged outbreaks.

A key finding of our study is the central role of children in household transmission. Although those aged 15 years or older represented 61% of national cases, they accounted for only 20% of household secondary cases in our cohort. These findings support the household as a primary setting for child-to-child transmission.

The low levels of household transmission observed might also reflect the effect of strengthened outbreak response measures during the study period. Data collection coincided with a phase of declining incidence (weeks 4–14 of 2025), following a peak in late 2024. Community-wide sensitisation campaigns, including in schools, might have contributed to improved symptom recognition and earlier care seeking, whereas enhanced surveillance might have improved timely detection and isolation of suspected cases, thereby reducing opportunities for intrahousehold spread. Although we did not measure individual household behaviours directly, these broader public health efforts probably had a role in mitigating household transmission.

Our findings suggest that household transmission alone is unlikely to sustain large or protracted outbreaks in Burundi. The early acceleration of the epidemic was likely driven by non-household transmission routes, particularly among adults through intimate or sexual contacts. Although individuals aged 15 years or older represented 61% of all confirmed cases nationally as of May 2 2025, they accounted for only 20% of secondary cases in our study, suggesting that adults are more often infected outside the household and serve as primary cases within it. The initial localisation of mpox lesions in the anogenital area is suggestive of transmission through sexual practice:[24] genital lesions were present in 79% of primary cases aged 15 years or older, compared with less than 30% in those younger than 15 years. Similar findings were reported during clade Ib outbreaks in Democratic Republic of the Congo, where genital rash was common, and sometimes the only symptom in adult cases linked to sexual contact.[25] In Kenya, Mutuku and colleagues[15] found that 62·5% of adult clade Ib cases likely resulted from sexual transmission, compared with just 10·4% among non-sexual household contacts, reinforcing the role that extrahousehold transmission, particularly among adults, has in sustaining the outbreak.

Overall, our findings emphasise the need for mpox response to distinguish household transmission, primarily affecting children and non-sexual contacts, from community transmission, which appears to be largely driven by sexual transmission among adults who might introduce the virus into households. Tailored interventions should prioritise early detection and care within households while addressing adult-to-adult transmission in the community. However, implementing effective household-level control measures remain challenging in endemic African settings such as Burundi, where stigma, limited capacity for isolation, and delayed care seeking might hinder timely detection and response.

Although our findings offer valuable insights into clade Ib mpox household transmission in Burundi, several limitations should be noted.

Participants' reluctance or inability to report exposures because of concerns over privacy or stigma, particularly if infection was acquired outside the household, limited our ability to assess the role of sexual transmission and might have introduced underreporting or misclassification bias. Furthermore, households might have been more inclined to seek care or enrol when children were affected, whereas adult cases, particularly those with mild symptoms, might have gone undetected because of stigma. Children were overrepresented in our cohort compared with the national profile, possibly reflecting those biases.

Although blood samples were collected for serology to assess asymptomatic infections among household contacts, their analysis was not done at manuscript submission because of unforeseen constraints. While this might have led to an underestimation of SAR, the public health implications of asymptomatic infection are probably limited given that mpox primary transmission through close contact with symptomatic individuals [A: word missing?].

We did not capture behavioural factors such as contact intensity, sleeping arrangements, children playing, individual hygiene practices, and early isolation of suspected or confirmed cases, all of which could influence transmission risk in the household.

Finally, the relatively small number of secondary cases (n=25) limited the statistical power of our analyses and introduced uncertainties around some estimates.

In conclusion, this study quantifies the dynamics of clade Ib mpox transmission within households in Burundi and, to our knowledge, is one of the first done during an active mpox public health emergency in an African setting. We found that, despite the high proportion of children affected by mpox in Burundi, intrahousehold transmission remained limited in scope. Our findings suggest that household settings contribute substantially to paediatric infections while adult infections are more likely to be driven by other transmission routes, such as sexual contact, that might serve as the primary source of mpox introduction into the household. These dynamics reinforce the need for public health preparedness and response that reflect those distinct transmission patterns.

Contributors

All authors contributed to the conception and design of the study. RK, FN, and LN curated and verified the data. RK conducted the formal analysis. FN independently verified the data and analysis. MN, DN, RNi, and JNy served as project administrators. FN, JNg, EK, and RNk supervised field implementation and data collection. RK, FN, and DN drafted or edited the manuscript. HB, BK, MS, and FB led the acquisition of the financial support for the project leading to this publication. RK was responsible for the decision to submit the manuscript. All authors approved the final version of the manuscript and agreed to be accountable for all aspects of the work.

Declarations of interest

We declare no competing interests. The statements in this publication are the views of the authors and do not necessarily reflect the policies or the views of UNICEF.

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Data sharing

Because of national data protection regulations, the full dataset is not publicly available. However, access to a limited version of the dataset can be granted upon reasonable request and with appropriate justification. Interested researchers should direct their inquiries to the National Institute of Public Health, Ministry of Public Health, Burundi. A minimum dataset supporting the findings of this study will be provided in the analysis code following publication of the manuscript (https://github.com/raoulkam/Mpox-hh-transmission-Burundi). Additional related study documents (study protocol, data collection forms, statistical analysis plan, and informed consent forms) can be requested from the corresponding author.

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