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Title: Assessing SARS-CoV-2 transmission in African households from the reanalysis of serosurveys

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Authors' Contributions:

LCF and SC designed and planned the study. LCF, SC, BN and BR contributed to the formal analysis. LCF, SC and NH contributed to methodology. All other authors contributed to data collection. LCF performed the data analysis and prepared the figures. LCF and SC wrote the original draft. All authors critically edited the manuscript. LCF and SC had accessed and verified the data reported in the study. SC had final responsibility to submit for publication.

Assessing SARS-CoV-2 transmission in African households from the reanalysis of serosurveys

ABSTRACT

Household transmission studies provided key insights on SARS-CoV-2 transmission in high-income countries but were rarely implemented in Africa. To help fill this gap, we analyzed SARS-CoV-2 seroprevalence studies with a household-based recruitment, focussing on households with ≤7 members, in four Sub-Saharan African cities: Kinshasa (82 households, 370 individuals), Lubumbashi (225 households, 970 individuals), Conakry (149 households, 649 individuals), and Yaoundé (311 households, 1,183 individuals), between late 2020 and mid-2021. Using an extended chain-binomial model accounting for missing serology, we estimated both the probability of community-acquired infection and within-household transmission. The proportion infected in the community rose sharply over time, reaching up to 73% by June 2021. Household transmission varied by location, with secondary attack rates (SAR) ranging from 8.9% to 26.7%, and households accounting for 9% to 28% of infections. Simulations showed that including households with missing serology improved the precision of estimates without introducing bias. SAR estimates were consistent with findings from South Africa and slightly lower than global pooled estimates, mostly from high-income settings, suggesting different transmission dynamics in African contexts. Our approach for handling missing serology can improve transmission estimates accuracy.

INTRODUCTION

Although reported SARS-CoV-2 case numbers were lower in Africa than elsewhere, seroprevalence studies revealed that the virus widely spread in Africa [1–7], with pooled seroprevalence reaching 65.1% (95% Confidence Interval (CI): 56.3-73%) by July-September 2021 [8]. In this context, there remains a need to further understand transmission patterns in Africa, particularly compared with other regions. For example, in high- and upper-middle-income countries, household transmission studies have provided major insights into SARS-CoV-2 transmission [9–15]. In the standard case-ascertained design, once an index case is identified, household members are followed-up to monitor infections and symptoms. However, such studies remain scarce in Africa [3,16,17] due to logistical and cost challenges.

In this paper, we propose a framework to tackle this major data limitation through the secondary analysis of more common cross-sectional serosurveys conducted in Africa to evaluate SARS-CoV-2 seroprevalence [1–7]. In these studies, recruitment was often household-based. Although such information was usually ignored in primary analysis, the measurement of serostatus among individuals in the same household allows reconstruction of household transmission dynamics.

Indeed, modeling approaches are available to estimate from such data the household transmission probability and the probability of community infection [18–20]. Such inference relies on analyzing the clustering of cases within households, with the intuition that if all cases are concentrated in a small number of households, household transmission probability must be high. These models have been successfully applied to influenza [21–23], and SARS-CoV-2 in high-income and upper-middle-income economies such as Switzerland [24], France [25], Costa Rica [26] and South-Africa [27], but not in resource-limited African settings.

Data requirements differ when the objective is to estimate seroprevalence vs to characterize household transmission. For estimating seroprevalence, absent serostatus data for some household members (e.g. due to survey design, refusal, etc.) is not critical. In contrast, for quantifying household transmission, non-participating household members must be treated as missing, as they still contribute to household transmission. This can result in substantial missing serology which may bias parameter estimates.

The aim of our study was therefore twofold. First, we performed secondary analyses of cross-sectional serosurveys to characterize SARS-CoV-2 within-household and community transmission in low-income and lower-middle-income settings in four African cities: Kinshasa and Lubumbashi (Democratic Republic of Congo, DRC), Conakry (Guinea), and Yaoundé (Cameroon). We extended the chain-binomial model of [24], to account for missing serological status among some household members in the surveys. Our analysis was restricted to households with ≤7 members, both for modeling feasibility and for comparability with existing studies. Second, we performed a simulation study to assess under which conditions our model could produce unbiased estimates of household and community transmission. In particular, we examined whether estimates remain unbiased when including households with substantial missing serology, and whether high community transmission could bias household transmission estimates due to saturation effects.

METHODS

Data description

In Kinshasa, Conakry, and Yaoundé, cross-sectional, household-based, age-stratified seroprevalence surveys were conducted under the ARIACOV project [4,5,28] between October 2020 and June 2021 (Table 1). In Kinshasa, surveys took place before and during the rise of the second wave in DRC (Figure 1A). In Conakry, they were carried out before, during, and after Guinea's second wave (Figure 1B). In Yaoundé, they were conducted during the rise and decline of the second wave in Cameroon (Figure 1C).

The fourth study [29] was conducted by Epicentre (Médecins Sans Frontières) in Lubumbashi, DRC's second-largest city, from April 12 to May 18 2021, following the country's second wave (Figure 1D).

In Lubumbashi, samples were tested with an IgG-ELISA (EUROIMMUN IgG) detecting responses against the SARS-CoV-2 spike (SP) protein. In Kinshasa, Conakry, and Yaoundé, a Luminex-based assay detecting antibodies against both SP and nucleocapsid (NC) proteins was used. For consistency, individuals were considered seropositive if their sample reacted against SP (see Appendix S1 for details).

Missing serostatus was defined as the absence of serology data, regardless of cause (survey design, refusal, or logistical challenges such as absence on sampling day). While not problematic for estimating seroprevalence in the original studies [4,5,28,29], non-participants still affect household transmission dynamics and must therefore be considered when studying these dynamics. Households were excluded sequentially if: (1) ≥50% of members lacked serology; (2) they had only one member; (3) they had >7 members; and (4) any member lacked age information. Excluded households are summarized in Table S1, and Figure S1 shows household size distribution prior to the exclusion of households of >7 members.

Chain-binomial model with missing serological data

Chain-binomial models [18,22] estimate two key quantities based from household members' serostatus: (i) the probability of community infection, i.e. the probability of being infected outside the household since the start of the epidemic, and (ii) the person-to-person within-household transmission probability, i.e. the per-capita probability that an infected member transmits the infection to a susceptible household contact. We adapted the model of [24], which operates in discrete time steps (generations). Community-acquired infections are assigned to generation 0. Individuals infected by members of a generation are assigned to the subsequent generation. Transmission events within a generation are assumed to be independent, given the infection status of the previous generation. The model also assumes homogeneous mixing within households, that all individuals are initially susceptible, and that serostatus perfectly indicates previous infection.

Unlike traditional chain-binomial models [18], this approach easily accounts for individual covariates like age. For each household size, all possible generation sets consistent with observed serostatus are enumerated, with complexity manageable for small-to-moderate household sizes.

For a household of size n, with serostatus vector x, the probability of observing x is:

$$P(x) = \sum_{\mathbf{g}} \prod_{i=1}^{n} P(s_i|\mathbf{g})$$

where s_i is the serostatus of member $i = 1, \ldots, n$, and $\mathbf{g}$ is a vector of unobserved infection generations.

Each conditional probability $P(s_i|\mathbf{g})$ depends on B_i, the probability that individual i escapes community infection (from outbreak start to sampling time), and Q_{ji}, the probability that i escapes infection from j.

These probabilities act on different time scales: B_i reflects cumulative community exposure, while Q_{ji} captures transmission risk during a household contact's infectious period.

We extended the model to handle missing serology by considering all possible generation sets that can arise from unobserved members, assuming they were equally likely to be seropositive or seronegative. Details and simple examples with and without missing serology are provided in Appendix S2.

Model assumptions for real data analysis

For the four cities, we assumed that $1 - B_i$, the cumulative probability of community infection for individual i, was equal to $1 - exp(-\alpha)$, where α is the community risk depending on the survey and on the age of the individual [30]. Age groups were defined as children (<12 years) and adults (≥12 years), based on observed similarities in COVID-19 outcomes between teenagers and adults [31]. This classification also reflects local context, where adolescents often assume adult-like community roles [32]. We also tested an alternative cutoff at 18 years. Overall, we estimated 4 (=2*2), 6 (=3*2), 4 (=2*2), and 2 (=1*2) community parameters for Kinshasa, Conakry, Yaoundé and Lubumbashi, respectively.

The person-to-person within-household transmission probability was parameterized as $1 - exp(-\beta/(n/4)^\gamma)$, where n is household size, β is the person-to-person risk in households of size 4 (the median across cities), and γ ($\gamma > 0$) controls how infection probability decreases with n: proportional if $\gamma = 1$, faster if $\gamma > 1$ or slower if $\gamma < 1$.

Inference used a Bayesian framework with Hamiltonian Monte Carlo in Rstan, with Uniform(0,10) priors for all parameters except γ, which used a Lognormal(0,1) (see Appendix S2). We report median and 95% Credible Intervals (CrI).

Secondary Attack Rates and proportion of infections within households

The household secondary attack rate (SAR) is a common indicator of household transmission, with several possible definitions [21,33]. In studies with detailed temporal data, the 'classical SAR' is often defined as the proportion of contacts infected shortly after the index case (typically 1 or 2 weeks). In [21], the 'actual SAR' is defined as the person-to-person within-household transmission probability computed in our model. We aimed to compute a SAR close to the 'classical SAR' to match how the SAR is typically estimated in the literature using detailed temporal data,

i.e. limiting community infections but counting tertiary ones. This was done by simulating 1,000 datasets, starting with a single index case per household and no community transmission, using within-household transmission probabilities to generate transmission events. The final proportion of infected contacts (either by the index case or by other household members) was evaluated by household. The overall SAR per country was a weighted average of SARs by household size, with weights based on the percentage of households with 2 to 7 members in each country [34–36].

We estimated the proportion of infections attributable to household transmission by simulating 1,000 datasets using samples from the posterior distributions, counting the number of infections in the community (in generation 0) and within households (in generation ≥ 0 and $< \infty$).

Sensitivity analyses

To assess whether our estimates were robust to the exclusion of households with missing age data, we conducted a sensitivity analysis in which these households were retained, and community transmission was modeled without age stratification.

To evaluate the impact of the prior assumption that individuals with missing serology were seropositive with 50% probability, we conducted a sensitivity analysis using alternative values of 10% and 30%.

Vaccination was only relevant for surveys 2 and 3 in Conakry, where coverage was limited (Table S3). In each of the 37 households where at least one individual had received a vaccine dose, 80% (95% CI: 50-86%) of members were unvaccinated. As a sensitivity analysis, we excluded these households to assess whether their inclusion, despite low vaccination coverage, significantly affected our SAR estimates. This analysis was not intended to evaluate the effect of vaccination on SAR, but rather to confirm that our main results are robust to the presence of limited vaccine-induced seropositivity.

Simulation studies

To assess model fit, we simulated 1,000 datasets with parameters drawn from the posterior distribution and compared the observed and predicted values of seroprevalence by household size and age group. Simulation procedure is detailed in Appendix S3.

We also used simulations to explore how the inference method's performance was affected by the amount of missing serological data (parametrized by the maximum percentage of missing serological data per household, i.e. the threshold above which a household is excluded from analysis, determining the number of participating households) and the values of model parameters. Given that the Lubumbashi dataset had the largest amount of missing serological data (Figure S2), we based our simulations on its structure, replicating its household demographics and patterns of serological data availability.

Simulations assumed a probability of community infection independent of age. We explored values of α (the community transmission risk), β (the household transmission risk in a household of size 4), equal to 0.12, 0.25 and 0.50, with $\gamma = 1.5$. We explored scenarios where the maximum acceptable percentage of missing serological data per household was 0%, 30%, and 50%. For estimating community and household risks (per city), the full dataset included 225 households (Table 1). Applying these thresholds resulted in the inclusion of 73 households (0% missing data), 115 households (≤30% missing data), or all 225 households (≤50% missing data). For estimating γ, since it was assumed to be common across surveys in the real datasets, we assessed the impact of missing data by resampling the 225 households to approximately match the total household count across surveys (760 households; see Table 1). Applying the same missing data thresholds to the resampled set led to the inclusion of either 400, 530, or 760 households, respectively.

We simulated 200 datasets per scenario and ran our estimation algorithm. For each run, we computed the estimation error (difference between the posterior median and the true parameter value). The distribution of these errors was then examined, a distribution centered around zero indicating no systematic bias.

RESULTS

Datasets

The final datasets included 1) 82 households (370 individuals) in Kinshasa; 2) 149 households (649 individuals) in Conakry; 3) 311 households (1,183 individuals) in Yaoundé; and 4) 225 households (970 individuals) in Lubumbashi (Table 1, Figure 1 A-D). In each survey, 16-27% of individuals were children (<12 years old). The proportion of women ranged between 47-62%. The median household size was 4 across all cities and surveys, as well as in most surveys (Figure S3).

The proportion of households with more than 30% missing serology ranged between 1-31%, except in Lubumbashi where it reached 46% (Figure S2). In Yaoundé and Lubumbashi, the proportion of households with incomplete serological data increased with household size (Figure S4). This trend was less apparent in Kinshasa and more variable across surveys in Conakry. Individuals with missing serology were excluded from seroprevalence calculations.

SARS-CoV-2 seroprevalence

In each city, the overall seroprevalence increased over time. In Kinshasa, it rose from 18% (95% CI: 11-26%) to 29% (95% CI: 23-35%) between late October and late December 2020. In Conakry, it almost doubled from 29% (95% CI: 21-37%) in December 2020 to 66% (95% CI: 60-72%) in March-April 2021, reaching 71% (95% CI: 65-77%) by June 2021. In Yaoundé, it increased from 47% (95% CI: 41-53%) in late January - early February 2021, to 68% (95% CI: 65-72%) by May 2021. In Lubumbashi, it was 39% (95% CI: 35-43%) in April-May 2021. For each survey, mean

seroprevalence was higher among adults than among children and increased for both groups over time (Figure 1 E-H). In the four cities, seroprevalence remained relatively stable across household sizes (Figure 1 I-L).

Estimated transmission probabilities

Estimated cumulative probabilities of community infection increased over successive waves, particularly among adults (Figure 2). The steepest increase occurred in Conakry, where adult estimates rose from 24.8% (95% CrI: 16-35.1%) to 71.8% (95% CrI: 62.6-79%) between the first and second surveys. For children, probabilities were generally lower and more uncertain, showing slight, non-significant increases due to overlapping CrI.

The person-to-person within-household transmission probability in a household of size 4 varied across cities, ranging from 7.6% (95% CrI: 1.9-14%) in Kinshasa and 8.4% (95% CrI: 1.6-16.9%) in Conakry to 16.3% (95% CrI: 10.9-21.6%) in Lubumbashi and 19% (95% CrI: 14-23.9%) in Yaoundé (Figure 3A). This probability decreased with household size to a power determined by parameter γ, whose common estimate across cities was 1.38 (95% CrI: 0.88-1.85). Consequently, the median person-to-person within-household transmission probability decreased from 18-42% in households of size 2 to 4-9% in households of size 7 (Figure 3B). Figure S5 shows the posterior distributions of the parameters.

The SARs by household size were only slightly higher than the person-to-person within-household transmission probabilities (Figure S6). The overall SAR was 8.9% (95% CrI: 1.5-19.3%) for Kinshasa, 9.2% (95% CrI: 1.4-20.5%) for Conakry, 21% (95% CrI: 12.6-29.5%) for Lubumbashi and 26.7% (95% CrI: 17.9-35.7%) for Yaoundé.

The estimated proportion of infections attributable to household transmission was 14.3% (95% CrI: 3.2-32%) in Kinshasa, 8.7% (95% CrI: 1-20.2%) in Conakry, 20.8% (95% CrI: 15.8-27.8%) in Yaoundé and 27.7% (95% CrI: 18.8-35.2%) in Lubumbashi.

Sensitivity analyses

In sensitivity analyses, including households with missing age data did not substantially affect demographics nor transmission estimates (Figures S7-S8 vs. Figure S3 and Figure 1, Table S2). Redefining adults as ≥18 instead of ≥12 years also had little effect, though age-group differences were less pronounced (Table S4). Lower prior probabilities for the seropositivity of individuals with missing serostatus produced largely stable estimates with overlapping CrIs (Table S5), though household parameters shifted somewhat when the prior probability was 10%, particularly in Yaoundé's second survey. Excluding Conakry households with vaccinated members likewise had no substantial impact (Table S6).

Model adequacy

The model showed a good fit to seroprevalence by household size (Figure S9) and age group (Figure S10) replicated the distribution of infections by household size well (Table S7), with most observed values falling within expected ranges, except for a few instances in Lubumbashi (households of size 3 and 4) and in Kinshasa (households of size 4 and 6).

Simulation studies

In the simulation study, we found that bias on the community and household risks, α and β, was negligible across all scenarios. It was at most 2% (in the scenario where all households with missing serology are excluded, i.e. with only 73 households analysed) (Figure 4A-B). Including households with missing serological data did not introduce bias in the estimates, but reduced uncertainty, as it increased the number of participating households. Across scenarios, the standard deviation of the community risk estimate was at most 8%, 7% and 4% when the proportion of missing serological data in a household was constrained to be ≤0% (73 households), ≤30% (115 households), and ≤50% (225 households), respectively. Similarly, the standard deviation of the household risk estimate was at most 10%, 9% and 6%, respectively.

Similarly, for power coefficient γ on household size, uncertainty decreased as more households were included, even in the presence of missing data (Figure 4C). As expected, larger uncertainty was observed for smaller household risk β. Bias was almost null when β was ≥25%. When β was 12%, bias was at most -18% when including 400 households without missing data, and -10% when including 760 households with up to 50% missing data.

DISCUSSION

Our study examined SARS-CoV-2 transmission in four African cities from one low-income and two lower-middle-income countries: Kinshasa and Lubumbashi (DRC), Conakry (Guinea), and Yaoundé (Cameroon). Through secondary analysis of household-based cross-sectional seroprevalence surveys, we estimated community and household transmission risks and provided SAR estimates for each city, gaining key insights into SARS-CoV-2 transmission in African cities and demonstrating that such risks can be quantified using simple seroprevalence data when recruitment occurs by household.

SARS-CoV-2 SAR estimates vary widely, between 0.5-78% for studies published up to June 2022 [33]. This variability may arise from multiple factors, including cultural and behavioral factors, differences in interventions, emergence of variants over time, and variations in study designs. The co-existence of many SAR definitions [21,33] can also contribute to this observed heterogeneity.

Heterogeneity in SAR estimates can occur even within a single study over time, as shown in a detailed study in South Africa, where SAR increased from 9% (95% CI: 5-14%) in the country's first wave, to 32% (95% CI: 28-37%) in the third [37]. Using less detailed data, we estimated SARs for four African cities in low- and lower-middle-income countries, consistent with those estimated for South Africa. In Kinshasa and Conakry, the point estimate of the SAR was around 9%, comparable to South Africa's first wave (~9%). In Yaoundé and Lubumbashi, SAR were 20-30%, aligning with South Africa's Beta-driven second wave (17-29%). These estimates are also slightly lower than global pooled estimates, mostly based on studies from high-income countries, pre-VOC (variants of concern) (19.5%, 95% CI: 15.9-23.1%), and during VOC co-circulation with ancestral strains (28.2%, 95% CI: 24.8-31.7%) [33].

Few studies have reported SARs for Africa, particularly in low- and low-middle-income settings. In 2020, estimates ranged from low in Rwanda (2.93%, 95% CI: 1.84-4.60%) [38] and Zambia (6.9%, 95% CI: 5.6-8.1%) [39], to higher in Madagascar (38.8%, 95% CI: 19.5-58.2%) [16], and exceptionally high in Egypt (89.8%, 95% CI: 82.2-94.3%) [40]. A 2021 study in Kenya [41] showed large uncertainty (17%, 95% CI: 1-43%). The wide range and wide uncertainty highlight the importance of obtaining more refined estimates of SARS-CoV-2 SAR in low- and low-middle-income African countries. Previous African studies mostly relied on PCR-confirmed infections and time-constrained exposure windows to reduce misclassification of community infections (Table S8). By contrast, our approach used only cross-sectional serology and a simulation-based framework that explicitly excludes community-acquired infections. This allowed standardized SAR estimation across sites, even without time-stamped data, complementing prior methods and expanding their geographic scope to other resource-limited settings.

We found person-to-person within-household transmission probabilities decreased as household size increased, to the power of γ (Figure 3B). This decline might reflect family-ties, as observed in France [25], where the relationship between transmission probability and household size was not statistically significant once family ties were considered. The estimated γ~1.4 suggests a more rapid decline with household size than typically observed in high-income countries (~1 or lower [42–44]), although the CrI included 1, indicating uncertainty about the magnitude of this decline. This steeper decline might reflect behavioral factors specific to resource-limited settings or the presence of subunits, such as multiple families living together. Our estimate aligns with a study in Costa Rica, a high-income economy, where SARs decreased with household size to the power of 1.7 [26]. Seroprevalence by survey was relatively stable across household sizes (Figure S9), which can be explained by a high community transmission probability and person-to-person within-household transmission probabilities decreasing steeply with household size.

Our estimated community transmission probabilities, with medians ranging from 15% in October 2020 to 73% in June 2021, reflected high seroprevalence levels and were higher for individuals over 11 years old. Given the young demographic profile of the

African population, our findings are consistent with other studies showing young adults had the highest risk of community infection [24,25,45]. In sub-Saharan African cities, <5% of residents are ≥65 years old [46,47]. Thus, distinctions between working-age and retired populations, relevant in high-income settings like Geneva [24] (20% of ≥ 65 years old), are less relevant in our study settings. Instead, the most meaningful contrast in transmission dynamics may lie between children and adolescents/adults, a distinction also reflected in our results showing clearer differences when adolescents were grouped with adults. Finally, the median estimate for the proportion of infections attributable to household transmission ranged between 9-28%, which is comparable to estimates from Geneva (22.5%) and France (25.5%) in 2020 [24,25].

Serology reflects cumulative infections, as IgG antibodies typically remain detectable for several months following infection. Both the EUROIMMUN IgG-ELISA (used in Lubumbashi) and the Luminex-based assay (used in Kinshasa, Conakry, and Yaoundé) target spike antibodies and have been shown to detect responses for at least 4-6 months post-infection [48,49]. Thus, serology, and therefore our estimated transmission probabilities, reflect infections acquired during and prior to survey periods, potentially extending back to mid-2020.

To assess whether our model could produce unbiased estimates under the real-world limitations of the serological surveys, we performed a simulation study. Specifically, we evaluated whether estimates remain robust when including households with substantial missing serology, and whether high community transmission could bias household transmission estimates due to saturation effects. The results (Figure 4) showed that our extension of the chain binomial model [24] which effectively incorporates such data limitations, produces reliable estimates even in the presence of high community risk and missing household serostatus. This suggests that future household transmission studies do not necessarily need to exclude households solely on the basis of missing information for some household members, as such households may still provide valuable insights into household transmission dynamics. This is particularly relevant in resource-limited settings, where studies may be more constrained in terms of sample size or data completeness, and excluding households with missing data could significantly reduce statistical power.

Our study was restricted to African households with ≤7 members for computational reasons. Indeed, the chain-binomial framework involves a combinatorial likelihood calculation that becomes infeasible as the number of infections (and hence household size) increases, especially in the presence of missing data. This is a well-known methodological constraint of existing models. Since a substantial proportion of African households are larger than 7 members, this means that our study cannot be considered representative of all African households. In future research, it will therefore be important to develop models allowing the study of transmission in larger African households to complement our analysis. That being said, investigating transmission in this well-defined subpopulation of African households with ≤7 members remains

scientifically relevant and informative. First, it helps maintain comparability with studies from high-income countries, where households are typically smaller. Since the SAR declines with household size [42–44], we need to compare transmission in households of similar sizes to dissociate the impact of context (e.g. European vs African) from that of household size. Second, in our study, households with ≤7 members accounted for over 70% of eligible households (54-75% of individuals) in most settings, and 58% of households (30% of individuals) in Conakry (Table S1), where larger households were more common, particularly in Surveys 2 and 3 (Figure S1). Our subpopulation hence covers a substantial proportion of the population that is important to characterize. Third, our estimates for households with ≤7 members across four large African cities help fill an important gap as the literature on SARS-CoV-2 transmission in African households remains limited. Future modelling of transmission in larger households will open up interesting research questions. For example, very large households are likely structured into multiple sub-units, and it will therefore be important to explore if transmission within these sub-units resembles that within smaller households.

Our study has other limitations. First, we defined serostatus based on reactions to the SP protein to ensure comparability across cities. However, alternative definitions could be possible and impact seroprevalence and transmission parameters estimates. In particular, our study may underestimate transmission rates if important seroreversion was present in the data. In addition, because our definition of seropositivity is based solely on SP, we cannot in principle distinguish between infection- and vaccine-induced seroresponses. However, only a small number of individuals were vaccinated and we found that removing associated households from analysis did not change our results.

Second, we had to assign priors for missing serology. We chose a baseline 50% prior to avoid imposing population-level assumptions at the individual level. Sensitivity analysis showed that the prior can influence some parameter estimates, particularly when set to 10%, but most estimates remained stable. Given the observed seroprevalence range (18-71%), the 50% prior was reasonable for our setting, though in lower-prevalence contexts, using observed seroprevalence as a prior might be preferable.

Third, we made several simplifying assumptions. Particularly, the within-household homogeneous mixing assumption may itself not hold and could bias within-household transmission studies [50,51]. While not entirely realistic, the assumption of no reinfections is reasonable in the context of our study, given the relatively short survey durations (the time between the first and last survey was at most 7 months), and the fact that reinfections with pre-Omicron variants were rare within a few months of primary infection [52]. Additionally, although the surveys spanned different epidemic waves across cities, we assumed constant transmission parameters within each city. Non-pharmaceutical interventions such as mask mandates, school closures, and restrictions on gatherings were officially in place during the different survey periods; however, evidence suggests that adherence and enforcement were limited in sub-

Saharan Africa [53]. In practice, transmission likely varied due to shifts in circulating variants and population behavior, as seen in South Africa where SAR estimates ranged from 9% to 32% across waves [32]. We explored wave-specific analyses but lacked the statistical power to robustly estimate parameters by wave.

Finally, more detailed data are needed to confirm our findings. Particularly, case-ascertained studies conducted in African settings could offer deeper insights into the specific characteristics of household transmission in these contexts. In the absence of such studies, serological surveys including a larger number of households might offer additional or more precise insights.

In conclusion, the secondary analysis of household-stratified seroprevalence data found SARs in households with ≤7 members in low- and lower-middle-income African cities comparable to those in South Africa and slightly below global pooled estimates, relying mostly on studies in high-income settings. These results highlight the insights provided by our approach, even with high community transmission and substantial missing serological data.

FIGURE CAPTIONS

Figure 1. Epidemic dynamics and seroprevalence over time, by age group and household size. A-D: Timing of the surveys and number of households included per survey. In grey, daily number of reported cases per million people in the country (7-day rolling average). Source: World Health Organization (2024); Population based on various sources (2024) – with major processing by Our World in Data. E-H: seroprevalence per age group and survey. I-L: seroprevalence per household size and survey. Mean and 95% CI.

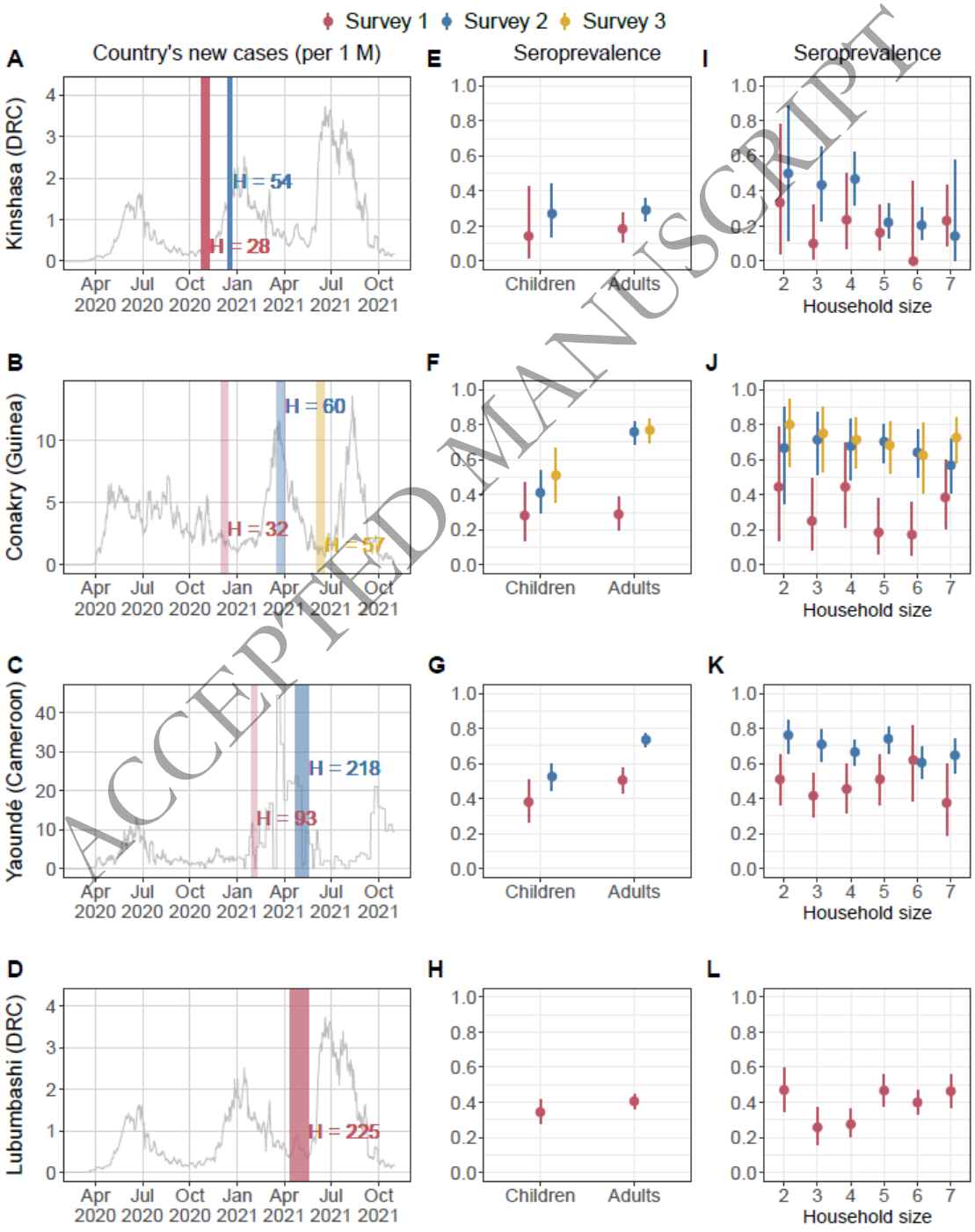


Figure 2. Estimated probability of community infection by survey and age in each city. Each panel corresponds to a specific city, showing the posterior median and 95% CrI of the probability of community infection (left axis) for each survey and age group. The right axis (in grey) represents the cumulative number of reported cases per million people in the country. Source: World Health Organization (2024); Population based on various sources (2024) – with major processing by Our World in Data.

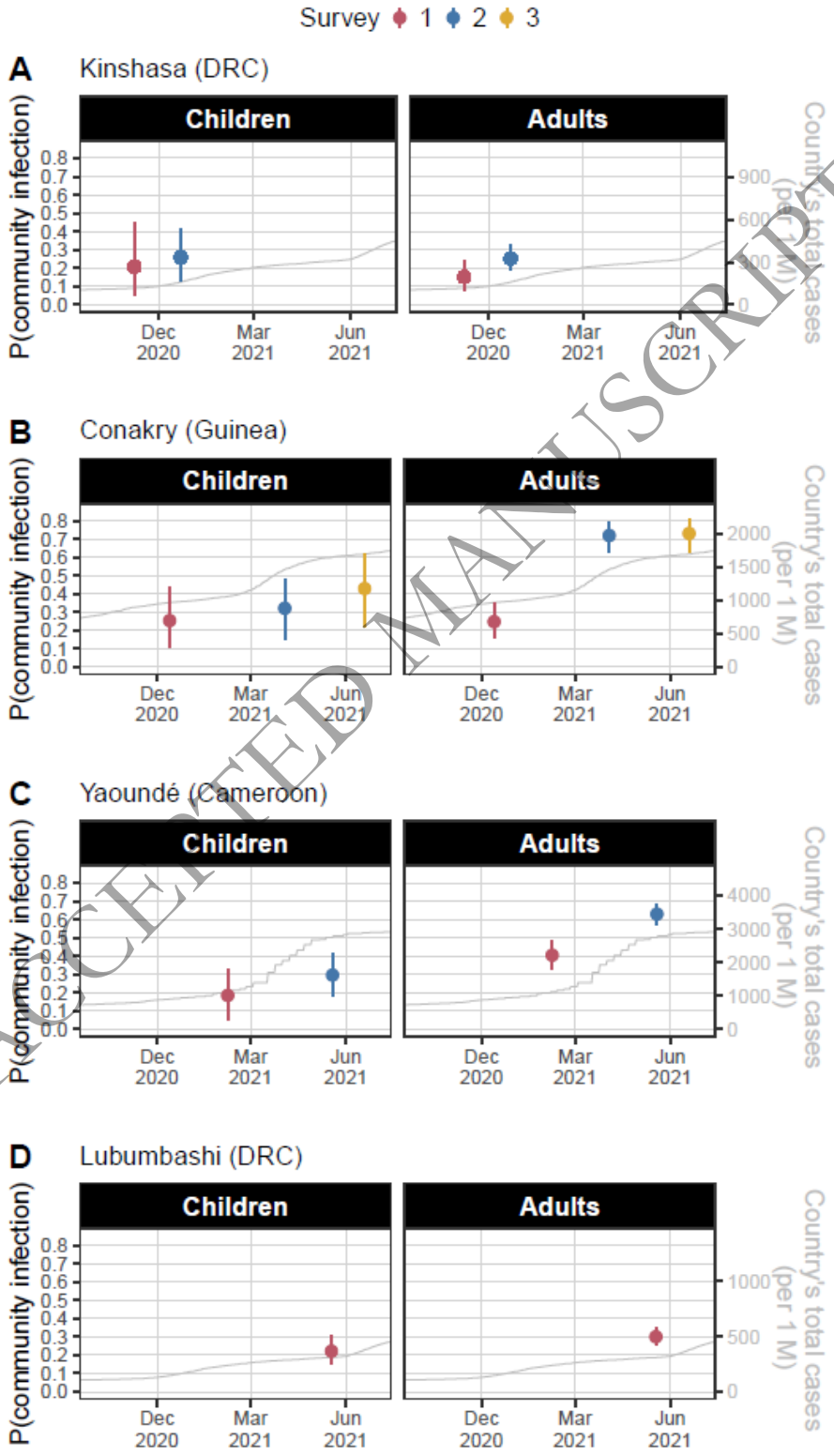


Figure 3. Estimated person-to-person within-household transmission probabilities. A: Per city in households of size 4. B: Per city by household size. Posterior median and 95% CrI.

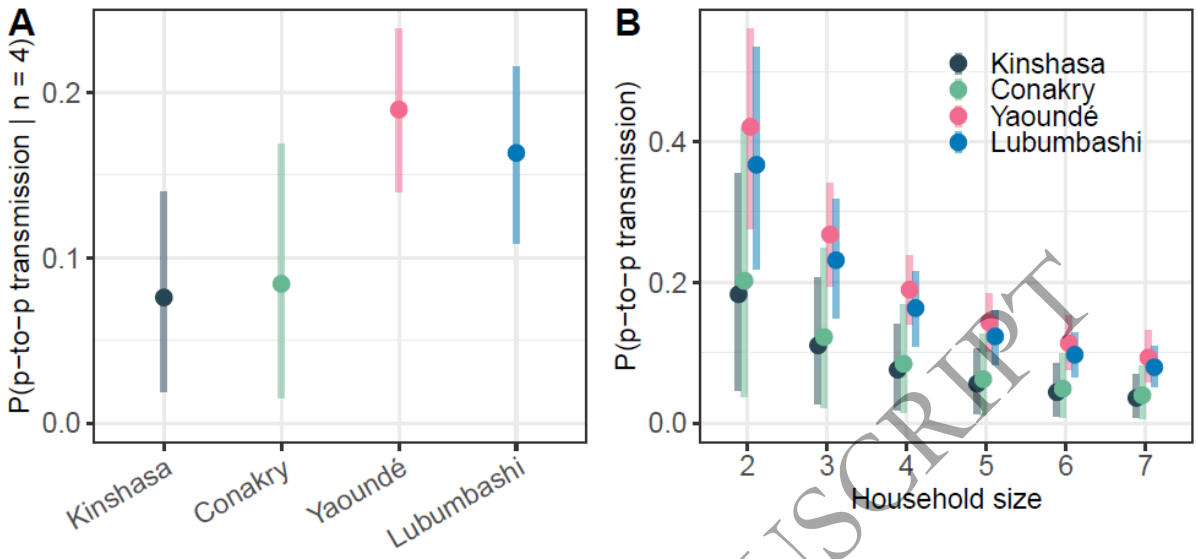
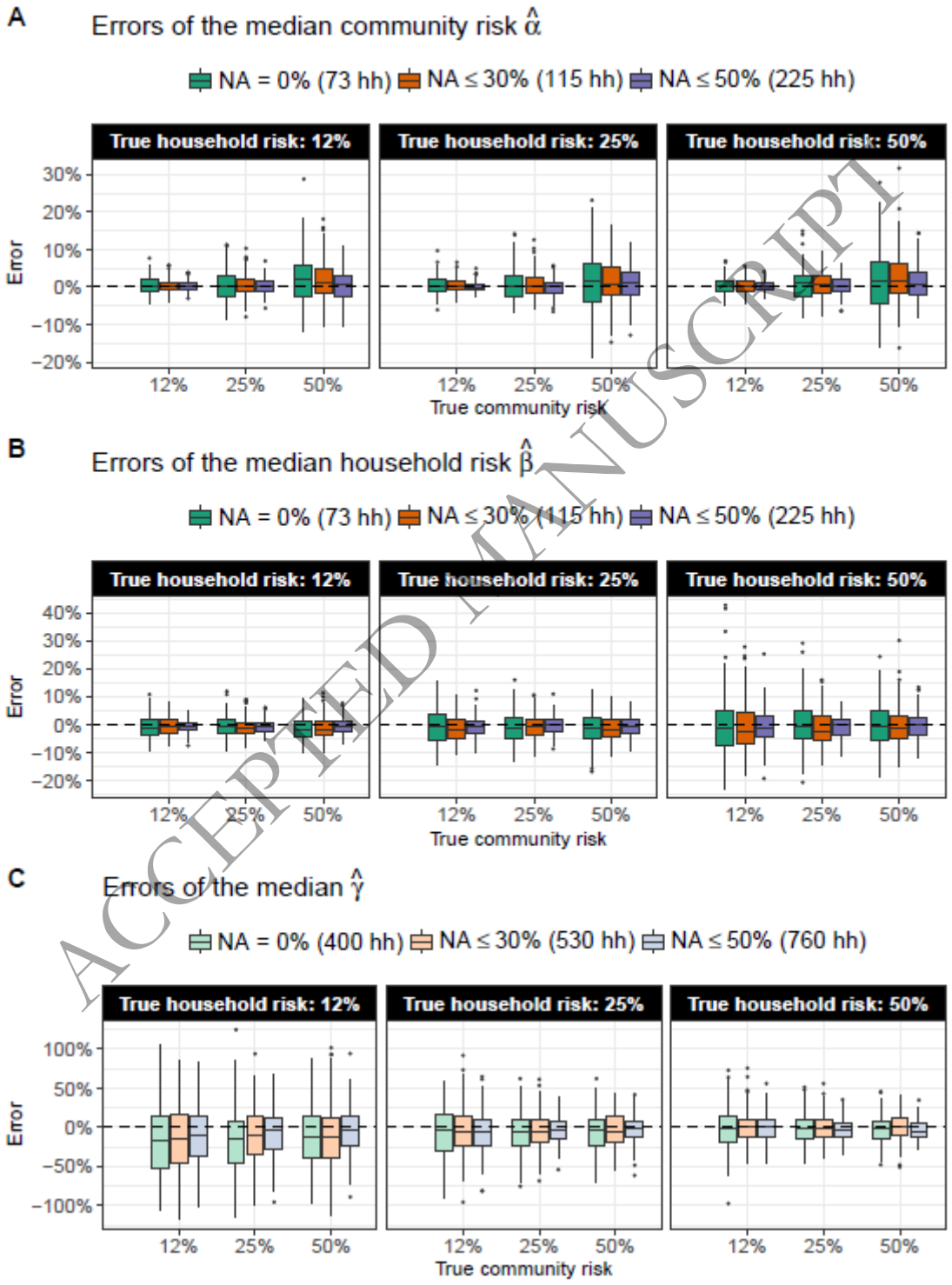


Figure 4. Simulation study results for estimating community risk α, household risk β, and γ with the posterior medians. Simulations use probability of community infection $1 - exp(-\alpha)$ and person-to-person within-household transmission probability $1 - exp(-\beta/(n/4)^{\gamma})$ with $\gamma = 1.5$. See main text for details.



TABLES

Table 1. Location and timing of surveys, and number of participating households and individuals by age

City	Survey	Start	End	Number of households	Number of individuals		
					Children	Adults	Total
Kinshasa (DRC) [28]	1	Oct 22, 2020	Nov 8, 2020	28	20	101	121
	2	Dec 12, 2020	Dec 22, 2020	54	41	208	249
	All			**82**	61	309	**370**
Conakry (Guinea) [4]	1	Dec 1, 2020	Dec 13, 2020	32	33	106	139
	2	Mar 19, 2021	Apr 3, 2021	60	72	202	274
	3	Jun 4, 2021	Jun 19, 2021	57	48	188	236
	All			**149**	153	496	**649**
Yaoundé (Cameroon) [5]	1	Jan 27, 2021	Feb 7, 2021	93	88	232	320
	2	Apr 24, 2021	May 19, 2021	218	197	666	863
	All			**311**	285	898	**1,183**
Lubumbashi (DRC) [29]	1	Apr 12, 2021	May 18, 2021	225	269	701	970
	All			**225**	269	701	**970**

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