Social Network Analysis of Ebola Virus Disease During the 2014 Outbreak in Sukudu, Sierra Leone

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Background. Transmission by unreported cases has been proposed as a reason for the 2013–2016 Ebola virus (EBOV) epidemic decline in West Africa, but studies that test this hypothesis are lacking. We examined a transmission chain within social networks in Sukudu village to assess spread and transmission burnout.

Methods. Network data were collected in 2 phases: (1) serological and contact information from Ebola cases (n = 48, including unreported); and (2) interviews (n = 148), including Ebola survivors (n = 13), to identify key social interactions. Social links to the transmission chain were used to calculate cumulative incidence proportion as the number of EBOV-infected people in the network divided by total network size.

Results. The sample included 148 participants and 1522 contacts, comprising 10 social networks: 3 had strong links (>50% of cases) to the transmission chain: household sharing (largely kinship), leisure time, and talking about important things (both largely non-kin). Overall cumulative incidence for these networks was 37 of 311 (12%). Unreported cases did not have higher network centrality than reported cases.

Conclusions. Although this study did not find evidence that explained epidemic decline in Sukudu, it excluded potential reasons (eg, unreported cases, herd immunity) and identified 3 social interactions in EBOV transmission.

Keywords. Ebola virus; rural; Sierra Leone; social networks; transmission chains.

Social networks provide an important lens through which to examine disease spread and decline, particularly when transmission occurs through direct contact, as with Ebola virus (EBOV). Furthermore, networks can be feasibly mapped in small communities, offering nuanced approaches to testing hypotheses for epidemic growth and decline [1]. The discovery of paucisymptomatic/asymptomatic EBOV led to suggestions that unreported infections were key to ongoing local transmission [2]. Alternatively, several competing hypotheses were posited as to why the 2013–2016 Ebola virus disease (EVD) epidemic in West Africa rapidly declined [3], including herd immunity, behavior change, and public health interventions [1–5].

At the time of the West Africa epidemic, the role of pauci-/asymptomatic cases to both spread EBOV and to confer herd immunity were relatively unknown. During the postepidemic period, a few studies combined data from transmission chains and serological testing of communities to assess unreported pauci-/asymptomatic EBOV infections and herd immunity in affected communities [2, 5, 6]. These and other studies showed that most community members, specifically those living in quarantined households, were seronegative and likely uninfected [6, 7]. In some of these communities, public health interventions came after substantial transmission occurred, which reduced their impact [5]. Furthermore, the role of local solutions and behavior change was yet to be fully appreciated [8]. An emerging hypothesis is that transmission chains were linked to the social networks of the first households with EVD cases, and that EBOV transmission declined when most individuals were no longer susceptible within these micronetworks.

Outside the Ebola literature, social network analyses have been widely used [9–13] to describe transmission pathways and identify high-risk nexuses. Network analyses have been applied to the West African EVD epidemic as well, but are limited to theoretical, data-free models [14, 15] including agent-based models [16] contact tracing data [17], and mobility that reveal only transmission events, and not the social contacts

underpinning transmission. Likewise, behavior change in response to a local outbreak can have protective effects across a social network by breaking transmission chains and onward spread [18].

In a previous serological study of residents of 4 villages in the Kono District, Sierra Leone, Kelly et al [18] found that 34% of EBOV cases were undetected. To determine how village social interactions and their resultant social structure were related to transmission chains in one of those villages, Sukudu, this work was extended by combining an EBOV transmission chain [5] with social network data. This study analyzed (1) the types of social contacts forming community structure and potential transmission pathways through social behavior; (2) whether transmission cessation within local networks was observed among social contacts of EVD cases; and (3) whether people are likely to have a relationship based on risk characteristics. This article further discusses our findings with regard to integrating network insights into emergency response to future outbreaks.

METHODS

Ethics Statement

The study protocol was approved by the Sierra Leone Ethics and Scientific Review Committee and the University of California, San Francisco and Harvard University institutional review boards. Participants provided written informed consent, with parental assent provided on behalf of minors. The Kono District Ebola Response Center, a government facility responsible for coordinating Ebola-related activities, granted access to the Viral Hemorrhagic Fever database.

Study Design and Participants

This cross-sectional study was conducted in Sukudu, Sierra Leone, a rural community of approximately 900 inhabitants living among 115 households in the Kono District. The community sustained an outbreak of 34 reported EVD cases in late 2014. The index case was a woman living in the district capital. Six family members traveled to care for her, and then returned to Sukudu. Two of the 6 relatives developed EVD, which triggered the 1-month outbreak in Sukudu.

Fieldwork occurred in 2 stages. In 2015–2016, the first stage of data collection mapped a transmission chain that included the reported EVD cases (n = 34) and then tested 221 quarantined people for EBOV immunoglobulin G antibodies to determine their serostatus, using information from a survivors' association, local chiefs, and the Viral Hemorrhagic Fever contact tracing database [5]. Fourteen seropositive contacts who likely had unreported pauci-/asymptomatic EBOV infection or unrecognized EVD were identified, for a total of 48 total cases in Sukudu. The transmission chain was formatted into a network of primary cases and subsequently infected people, where the tie between was the transmission event.

Table 1. Participant and Contacts Demographics From a Single Village in Kona District, Sierra Leone

Characteristic	Participants (n = 148)	Named Contacts (Not Interviewed) (n = 1522)	Cases (n = 48)
Sex			
Male	77	783	29
Female	71	727	19
Unknown	0	3	…
Age, y			
≤20	11	495	11
21–35	55	469	21
36–50	53	340	11
≥51	29	150	5
Unknown	0	59	0
Education			
None	85	15	33
Primary	34	2	15
Secondary or more	29	5	0
Unknown	0	1501	0
Quarantined?			
Yes	106	…	48
No	37	…	0
Unknown	5	1523	0
Job			
High risk	13	417	13
Low risk	135	1106	29
No. of people in household			
≤5	56	0	3
6–10	67	1	6
>10	24	0	4
Unknown	1	1522	35
Median (range)	6 (1–30); IQR, 4	…	…

Data are presented as No. unless otherwise indicated.

Abbreviations: IQR, interquartile range.

In 2019, the second stage was collected: social network data to complement the EBOV transmission chain data. Anyone aged ≥18 years then living in Sukudu was eligible to be interviewed. For recruitment, randomized snowball sampling, a standard sampling approach for ethnographic egocentric network data collection, was used [19]. Using GoogleEarth images, all houses in the geographic area were identified to create a sampling frame. A random number generator was applied to select a household for recruitment. Next, a list of all members of enrolled households was obtained, and this list was randomly sampled to select an eligible participant to interview. After this and each subsequent interview, all named contacts were numbered for the random number generator to recruit participants from the named contacts of the prior participants for the next round of interviews. If the first person selected could not be interviewed, the next person from the contact list was randomly selected. This sampling process was continued until all listed contacts were contacted; at that point, the researchers returned to the enumerated household list to select a new random

eligible participant to interview and conduct snowball sampling from their named contacts, including all surviving Sukudu EVD cases who were included in the EBOV transmission chain.

Measurements

The survey instrument included social network questions, demographic characteristics, and household-member information (Table 1). A name-generator technique was used to collect social network data, wherein participants were asked to name people with whom they have important social interactions [20, 21]. Each name provided by a participant represents a social tie, and a person's social network is the collection of their social ties. Name generators are useful for producing multiple social networks that may be combined, compared, or selected from for further analysis. The name generators are listed in Supplementary Table 1. Several focus groups with community members were held to determine common social interactions in Sukudu to design the 10 name-generator questions included in our survey. Focus groups represented a range of characteristics found among participants in the study, including age, sex, and occupation.

For every person named by participants in each name-generator question, demographic information (age, sex, job) and their relationship to the participant (nuclear family, extended family, friend, acquaintance) were collected. Following Kelly et al [5], this study used occupation type to classify transmission risk (low vs high) and considered high-risk occupations to be those that required spending long hours indoors with close physical contact (eg, housewife, student).

Network Analyses

Representative networks and descriptive statistics for key measures of position and sociability—that is, centrality—for each name-generator were constructed. Individual-level centrality was estimated, including degree (number of ties/person) and betweenness (number of shortest paths between an individual and all other people in that individual's network). People, including interview participants and their named contacts, are represented as nodes, while social interactions between 2 people are represented as edges. A critical data-processing step was entity resolution [22] (see Supplementary Material for full description), which involves identifying people with multiple entries in the dataset, either because they were named as a contact more than once or because they were both a participant and a named contact.

Next, the transmission chain to each network representation from the name-generator questions was added. From these transmission–social union network representations, the number of EBOV-infected people in the network was divided by the total number of people in the network to calculate the cumulative incidence proportion of EBOV infection.

Descriptive and statistical analyses of the networks were conducted, including the types of relationships (eg, nuclear family, extended family, friend) that form social ties. Second, mean degree centrality and betweenness centrality was calculated for reported cases, unreported cases, and susceptible people from the transmission–social union networks to determine whether undetected cases held critical network positions for social influence or transmission potential. Third, exponential random graph models (ERGMs) of the fully connected subcomponents of the social networks were built to test whether personal characteristics that determined a social tie correlated with the risk characteristics elucidated from the transmission chain by Kelly et al [5]. ERGMs were fit with node-level covariates to identify social phenomena, such as homophily (social assortment by like characteristics) and sociality (contact number), according to personal characteristics, including serostatus, sex, job risk level, and household size. Our ERGMs also included network-level attributes that capture structural phenomena, such as triad closure (the gwesp function), dyadic ties (gwdegree), and degree frequency (degree) [23]. All analyses were performed in R version 3.2.4 software (R Foundation for Statistical Computing, Vienna, Austria), using the igraph and statnet packages.

RESULTS

Study Population

The analytical cohort comprised 1670 unique people—148 participants (including 13 survivors from the transmission chain) and 1522 named contacts across all name generators (Table 1). Our sample included similar proportions of men and women overall, and among participants and contacts. Age distributions were similar between participants and named contacts. A significantly greater proportion of contacts than participants had occupations deemed "high-risk" ($\chi^2 = 23.5$, $df = 1$, $P < .001$). Most participants had no formal education (57%). Median household size was 6 (interquartile range, 4), often including extended family. More than 80% of participants reported a household size ≤10 people.

Social Networks From Name Generators, Subcomponents, and Transmission Chain

Among the name generators, several produced structured network subcomponents (Supplementary Table 1, Supplementary Figure 1). The networks—"talking about politics" (Supplementary Figure 1C), "helping solve disputes" (Supplementary Figure 1E), "sharing meals" (Supplementary Figure 1F), "living in same household" (Supplementary Figure 1G), "spending leisure time" (Supplementary Figure 1H), and "talking about important things" (Supplementary Figure 1D)—all produced networks with some chains or cluster structures.

The EBOV transmission chain resulted in 4 distinct outbreak clusters (Figure 1), the largest of which accounts for more than half (25/48 [52%]) of all transmission events involving Sukudu residents [5]. For 8 of 10 union transmission–social network representations (Table 2), the largest transmission cluster was always incorporated into at least 1 major structural subcomponent, and transmission chain incorporation always increased the number of social ties in the largest subcomponent. Living in the same household, spending leisure time, and talking about important things generated novel and larger components that included >1 of the transmission clusters (Figure 2) and more than half of the transmission events (60%, 71%, and 63%, respectively). After combining the transmission chain into the social networks, the resultant subcomponents were 12%–18% larger than the social-only subcomponents and had infection rates of 22% (living in the same household: 31/143), 23% (spending leisure time: 37/198), and 40% (talking about important things: 32/81). The overall cumulative incidence in these overlapping social networks was 37 of 311 (12%).

Unreported Cases Within the Social Networks and Transmission Chain

This study tested whether unreported cases had higher centrality in the transmission–social union networks. While EBOV cases, in general, had higher degree and betweenness than people who never contracted EBOV (Table 3), unreported cases did not have distinctly higher degree or betweenness than reported cases (Table 3). In fact, the transmission–social union subcomponents show that all unreported cases lie at the ends of transmission chain (observable in Figure 1). Also, because the undetected cases survived the outbreak, they were frequently the critical link between the chain and the social network (Figure 2). Comparing mean betweenness among reported and unreported cases, betweenness was higher in reported cases in the "living in same household" network, lower in reported cases in the "spending leisure time" network, and almost identical in the "talking about important things" network.

Determinants of Social Ties

Among edges with relationship information, 97% of relationships were nuclear (60%) or extended family (37%) within the "living in same household" network (Figure 3A), and households were frequently connected by extended family members who co-resided in multiple households. Among the "spending leisure time" and "talking about important things" networks, 93% and 95% of relationships were non-kin, respectively (Figure 3B and 3C).

There was a tendency for EBOV-seropositive people to have ties in common in each network, but no trend was observed for seronegative people. Sex assortment (ie, same-sex ties) was important only for non-kin social networks, spending leisure time, and talking about important things. Uniform homophily (mean assortment) yielded better model fit than differential homophily (different assortment rates for men and women), indicating that men and women have similar self-assortment tendencies. People's social relationships according to job risk level was different for each network (Table 4 and more details in the Supplementary Material).

DISCUSSION

The cumulative incidence proportion indicates that the rapid decline of EBOV cases in Sukudu was not due to network saturation of susceptible people (eg, herd immunity or personal network burnout). Additionally, the structural positioning of unreported pauci-/asymptomatic cases in both the transmission chain (distal) and the social network (central) suggests that these previously unknown cases were not important for onward transmission. An alternative hypothesis for transmission cessation within these micronetworks is behavioral change.

The networks that best integrated with the transmission chain—living in the same household, spending leisure time, and talking about important things—represent social interactions defined by both high intensity and frequency of potential exposures to EVD cases, which likely increased transmission risk [24]. These personal social interactions served as conduits for relatively high transmission and were associated with contact pathways that stretched beyond common contact tracing targets (household, immediate recent contacts).

The key networks were composed of different relationship types, primarily kin-based household relations and non-kin social contacts. Living in the same household yielded connected networks both within and across households, among nuclear or extended family, demonstrating how household fluidity and kinship may have played a role in EBOV spreading between related households. Other studies reported that much of the rural EVD outbreak occurred in large households, due to both crowded living and family obligations to provide honorable care to the sick [25].

In a study of a rural Sierra Leone community that sustained an EVD outbreak, most transmission occurred within a few large households that had prolonged transmission and high death rates [25]. Even in dense urban locales, household transmission was the most common infection route [26].

Kinship was not the only relationship that carried transmission risk during the Sukudu outbreak. The "spending leisure time" and "talking about important things" networks were primarily comprised of non-kin friends and few relatives. Ajelli et al constructed a transmission chain from the Pujehun District, Sierra Leone, and found that nearly 18% of infections happened between non-kin friends [27]. In Sukudu, although family ties may have been important for EBOV transmission between related households, non-family ties could have been important for transmission to new households/families that sustained micro-outbreaks and rapid transmission.
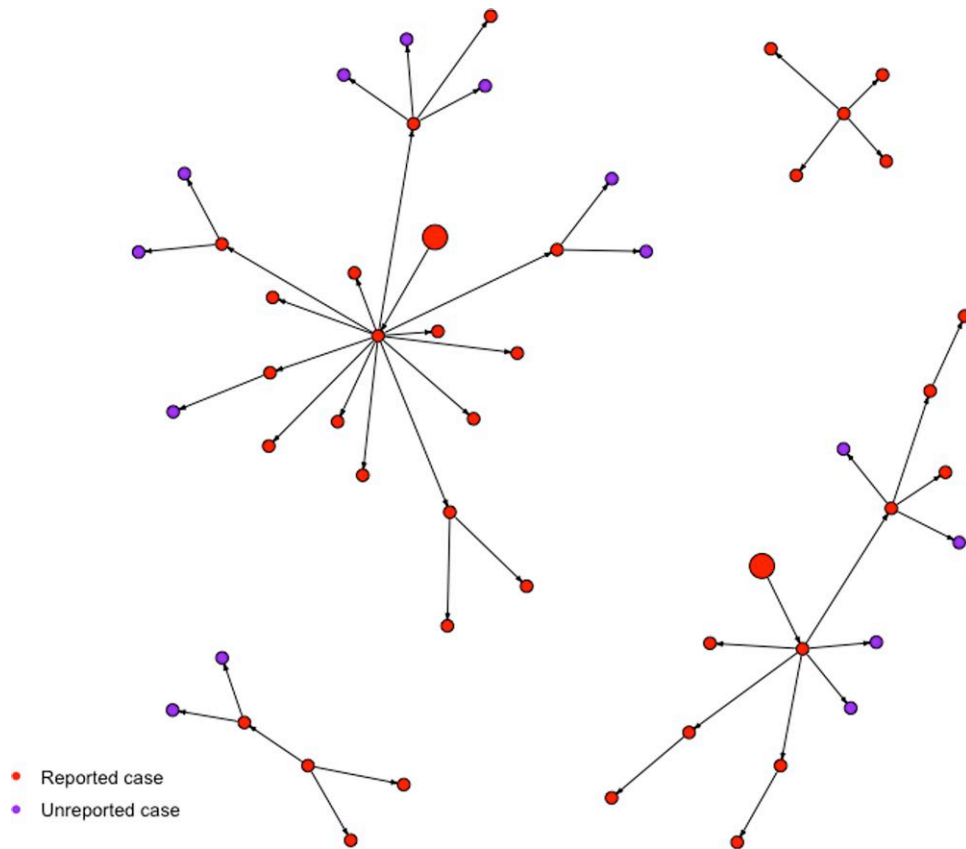
Figure 1. Ebola transmission chain representing the cases and transmission pathways for the Sukudu outbreak. The 2 larger nodes represent non-Sukudu residents who transmitted Ebola virus to a Sukudu resident, kicking off an outbreak in the village.

Role of Undetected Cases in Village-wide EBOV Spread

Our results do not support the hypothesis that undetected cases drove within-village transmission of Ebola virus by serving as social bridges within their community networks. This may be because village physical connectivity was high at baseline, and while bridging is an important mechanism for social capital in rural communities [28], it might not be as meaningful for pathogen diffusion. Individual community members with high betweenness centrality are only meaningful when there are strong subcommunities that do not reach each other without a linking person. However, betweenness could be an important mechanism of intervillage transmission, whereby individual community members who travel to other villages or towns play an important role in virus importation and initiating new outbreaks. Further work on mobility and intervillage connectivity could help reveal the importance of social bridging for EBOV importation into small rural communities.

Moreover, in addition to not occupying key network positions for transmission to unexposed network components, undetected cases also did not seem to have been a major contributor to transmission. This is evident not only from their peripheral position in the transmission chain (Figure 1), but

also from the fact that transmission cessation happened within 1 month and most village residents remained seronegative. Our combined transmission–social union networks are 60%–78% susceptible, reflecting the proportion of the community that could have been exposed but never seroconverted.

No further cases and evidence of no prior immunity suggests that transmission may have declined due to behavior change. Although Chowell and colleagues proposed a model that indicated declining transmission due to behavior change in Liberia [29], people's ability to adopt protective behavior amidst vast structural constraints was frequently underobserved. Most coverage of social behavior focused on entrenched high-risk behaviors, such as burial traditions, but it is important to highlight when and how community members recognized imminent exposure potential and altered their social interactions, thereby breaking risky network links. In fact, some studies reported that early pandemic transmission more frequently resulted from funerals and nosocomial infection [30], whereas late pandemic transmission more frequently occurred in households [31], indicating that some behavior change successfully reduced transmission in certain high-risk conditions (burial) while increasing the relative risk of others (household). This

Table 2. Ebola Virus Transmission Chain Incorporation Into Social Networks

Name Generator	Social Network Nodes[a]	Social Network Edges[b]	Social Network + Transmission Chain Nodes	Social Network + Transmission Chain Edges	Transmission Edges Incorporated	Infected Nodes/ Total Nodes in Network
"… live in the same household as you."	51	78	143	182	29/48 (60%)	31/143 (22%)
"… you usually spend your leisure time."	100	257	198	482	34/48 (71%)	37/198 (23%)
"… you usually share meals."	35	34	98	97	25/48 (52%)	26/98 (27%)
"… you have a sexual relationship with."			No subgraph			
"… would accompany you if you needed to go to the hospital."	21	20	62	61	25/48 (52%)	26/62 (42%)
"… you can talk about important things with."	20	19	81	80	30/48 (63%)	32/81 (40%)
"… you can talk about politics with."	62	61	118	118	25/48 (52%)	26/118 (22%)
"… could help you solve a disagreement."	43	42	93	92	25/48 (52%)	26/93 (28%)
"… you could borrow money from if you needed."	16	15	64	63	25/48 (52%)	26/64 (41%)
"… you have a strained or bad relationship with."			No subgraph			

Data are shown as No. or No. (%). Bold text indicates >50% incorporation of the transmission chain into the social network.

[a]Node: any person, participant, or named contact who appears in the network.

[b]Edge: Tie between 2 people that represents a contact.

Different Characteristics Predict Tie Formation Among Our 3 Social Networks

not only adds another tool to disease-fighting toolkits in underserved communities, it also recognizes the capacity for a community to self-protect and problem-solve.

Our ERGM results demonstrate that certain risk factors for EBOV exposure identified by Kelly et al [5] were also predictive of social tie formation in Sukudu, but tie-formation patterns differed between the social networks. This study offers insights into behavioral variance, including mixing heterogeneities according to age, job risk level, and household size. Rizzo et al [14] argued that a more realistic account of social mixing that captures contact heterogeneities is crucial because assuming random mixing and equal likelihood of mixing with any other person (ie, a set of differential equations with a single contact parameter) will usually lead to an overestimation of the effective reproductive number (R_t) and epidemic size. Our findings not only have potential to contribute to more precise estimates of R_t and epidemic size but also identify how individual characteristics (sex, job, but not age) associated with EBOV exposures influence aggregate social ties and affect overall epidemic dynamics in small, highly connected communities.

Implications for Interventions in Future EBOV Outbreaks

Our study of social behavior in Sukudu offers 2 important insights for designing EVD contact tracing and vaccination programs for small closely connected communities. First, small rural communities are tightly knit and are not easily subdivided into distinct communities with local brokers between them. A highly transmissible virus can spread across a village through strong social ties; thus, interventions based on geographic proximity alone (eg, quarantining neighboring households) may not stop community spread. Second, despite strong social centrality, social ties are not random, and tight connections, or cliques, form at the household and personal levels. These
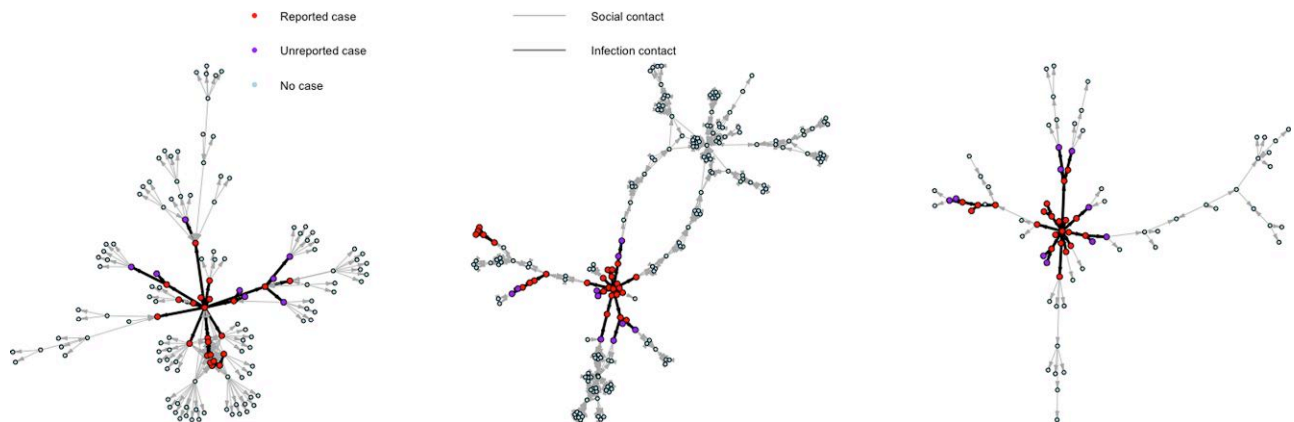

Figure 2. Largest subcomponents with embedded Ebola virus transmission chain from "live in same household" (*left*), "spend leisure time" (*middle*), and "talk about important things" (*right*) networks. Black edges represent transmission events.

Table 3. Social Network Position Centrality of Reported and Unreported Cases Sukudu, Kono District, Sierra Leone

Centrality measure	Total Network	Reported Cases	Unreported Cases	No Case (Ref)
"Please name people who live in your household."				
Nodes	143	23	8	112
Edges	182	…	…	…
Mean degree	2.55 (1–19)	6.52 (1–17)[a],***	3.88 (1–8)*	1.63 (1–19)
Mean betweenness	7.03 (0–327)	34.39 (0–327)***	24.75 (0–63)[a],***	0.14 (0–12)
"… you usually spend your leisure time."				
Nodes	198	27	10	161
Edges	482	…	…	…
Mean degree	4.87 (1–32)	2.85 (1–14)***	2.70 (1–8)***	5.34 (1–32)
Mean betweenness	14.09 (0–469)	14.7 (0–119)[a],***	21.2 (0–73)[a],**	13.55 (0–469)
"… you can talk about important things with."				
Nodes	81	22	10	49
Edges	80	…	…	…
Mean degree	1.98 (1–14)	2.86 (1–14)**	2·20 (1–4)	1.53 (1–6)
Mean betweenness	1.20 (0–56)	3.41 (0–56)[a],***	3.3 (0–15)[a],*	1.57 (0–15)

[a]After log-transformation of outcome variable.

*$P < .05$; **$P < .01$; ***$P < .001$.

cliques are not necessarily tight enough to generate separate subcommunities within a village, but these cliques can generate distinct groups within the network that are socially close but not neighbors. As a result, virus can spread rapidly within cliques and then more slowly between them.

A close study of individual relationships and how they aggregate into community networks can also guide emergency vaccine rollout and social support [32, 33], especially when vaccine supplies are limited and vaccine refusal is present. Instead of traditional ring vaccination programs [34, 35] that prioritize immediate contacts of exposed or infected individuals in a clustered manner, social networks may inform novel approaches for interventions aimed at breaking transmission chains that can reach between clusters and protect people before exposure. The high mortality rate of EVD and its relatively longer serial interval compared with respiratory viral pathogens such as severe acute respiratory syndrome coronavirus 2 mean that effective vaccine rollout will reduce death as well as transmission.

In addition to long-term reparative approaches to epidemic prevention [36–38], our findings underscore the suitability of a network approach for rapid interventions in future EBOV outbreaks. Because transmission requires close contact, apparently with a conspicuously symptomatic person, people likely know who their infectious contact was, unlike with asymptomatically transmissible respiratory viruses or slow-acting infections like
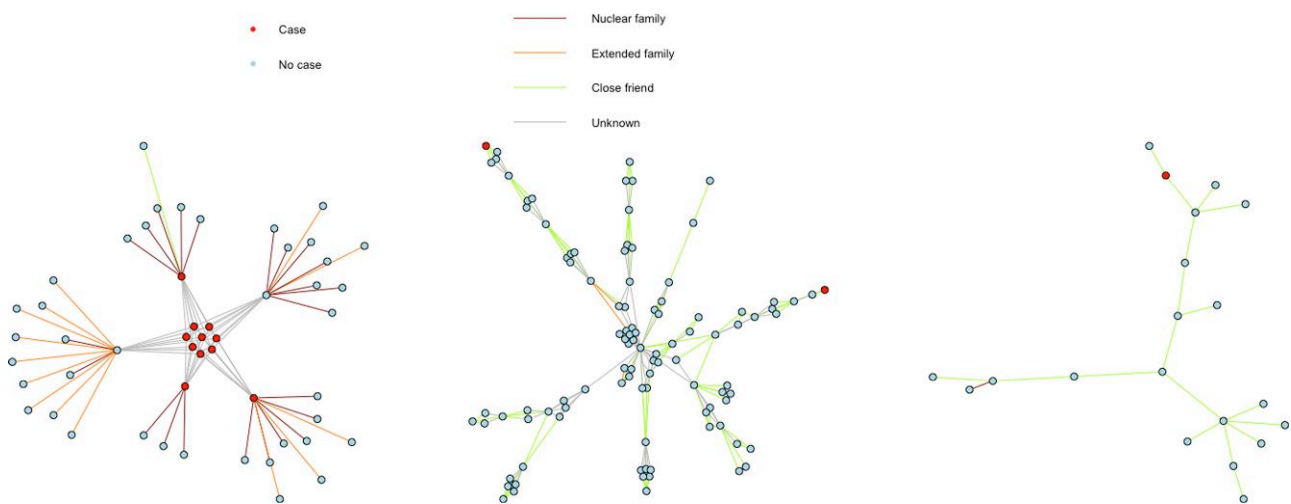

Figure 3. Connected subcomponents for 3 social networks: "live in same household" (*left*), "spend leisure time" (*middle*), and "talking about important things" (*right*). Edges are colored by relationship type. Household edges are mostly family (nuclear and extended), whereas spending leisure time and talking about important things are mostly nonfamily close relationships.

tuberculosis. Furthermore, because EBOV transmission frequently occurs during commonplace personal interactions—as opposed to the sensitive nature of sexual transmission—gathering reliable social contact information in an emergency timeframe is feasible.

Limitations

This study has several limitations. First, data used to construct the social network were collected after the EBOV outbreak and from a single village. Surviving reported cases were interviewed and, although some had left Sukudu, most remained within the community and their personal social networks likely did not change significantly after the outbreak. Second, some people in our relational dataset have similar identifying information, including name, age, and sex, which could have resulted in misclassification during the entity resolution process; the difference between conservatively and liberally resolved name lists was 5.3%. Third, certain data for named contacts who were not interviewed were unable to be collected, such as education level or household location. Also, the relationships of noninterviewed people who had ties to each other (eg, kin vs non-kin) were unknown. These missing data limited our ability to conduct additional analyses. Furthermore, while the most relevant and apparent social interactions were captured, there may have been epidemiologically relevant, close relationships that people do not speak about openly [39]. Although sexual contacts were asked about, it is possible that people underreported sexual partners. Fourth, ERGMs cannot always fit the degree distribution or edgewise shared ties for sampled data from small closely connected communities, even though they are still the best option for relational datasets. Nonetheless, they are still the best models for relational datasets, and iterative parameter fitting indicated that suboptimal structural fit did not affect nodal covariates. Finally, serology for previous infection can underestimate actual disease incidence, especially in people who had mild infections and faster viral clearance, due to attenuated antibody production [40, 41]; however, even a relatively large underestimate would not substantively impact our conclusions.

CONCLUSIONS

Although this study did not find evidence that explained epidemic decline in the rural village of Sukudu, the findings were able to exclude potential reasons (eg, unreported cases) for epidemic decline within micro-networks. In small, highly connected populations of rural villages, chains of personal social interaction between kin-based household relations and non-kin social contacts yield structures that influence EBOV exposure, facilitate EBOV transmission between personal networks, and offer clues to how behavior change can lead to outbreak decline. Our work presents a social map of how relationships in a small rural community could provide a contact pathway for rapid local EBOV spread while also offering insights into how these pathways can be leveraged to build an effective outbreak response that includes rapid quarantine and vaccination rollout in exposed communities, coupled with upstream structural analyses to provide synergy for epidemic containment.

Table 4. Network and Node Attributes of Interactions in Exponential Random Graph Model

Covariate	Live in Same Household Coefficient (SE)	Sharing Leisure Time Coefficient (SE)	Talking About Important Things Coefficient (SE)
Network attributes			
Edges	−4.8*** (0.6)	−8.0*** (0.3)	−3.4*** (0.3)
GWESP	0.5*** (0.1)	3.4*** (0.2)	…
Degree	−4.0*** (1.0)	…	2.2*** (0.3)
gwdegree	4.9*** (1.0)	…	…
Node attributes			
Ego-alter nodes[a]			
UH	…	−0.3*** (0.1)	−0.9*** (0.2)
DH, ego	−3.7*** (0.8)	…	…
DH, alter	−3.8*** (0.3)	…	…
Serological status	…	…	…
DH, positive	1.9*** (0.2)	1.6*** (0.2)	1.5*** (0.3)
DH, negative	−0.6*** (0.2)	0.8*** (0.1)	0.4 (0.3)
Sex			
UH	…	0.6*** (0.1)	0.8*** (0.2)
Job			
DH, high risk	…	0.9*** (0.3)	…
DH, low risk	1.3** (0.6)	0.2* (0.1)	…
Heterophily, high risk/low risk	1.1** (0.5)	…	…
Household			
UH	1.1** (0.5)	…	…
AIC	1200.0	3342.0	645.4
BIC	1293.9	3405.0	681.9

Abbreviations: AIC, Akaike information criteria; BIC, Bayesian information criteria; DH, differential homophily; gwdegree, geometrically weighted degree distribution; GWESP, geometrically weighted edgewise shared partnership; SE, standard error; UH, uniform homophily.

[a]This is a control variable to account for named contacts having very low degree because, due to not being interviewed, their contacts are largely missing.

*P <0.05; **P <0.01; ***P < .001.

Supplementary Data

Notes

References

1. Blackley DJ, Lindblade KA, Kateh F, et al. Rapid intervention to reduce Ebola transmission in a remote village—Gbarpolu County, Liberia, 2014. MMWR Morb Mortal Wkly Rep **2015**; 64:175–8.

2. Richardson ET, Kelly JD, Barrie MB, et al. Minimally symptomatic infection in an Ebola 'hotspot': a cross-sectional serosurvey. PLoS Negl Trop Dis **2016**; 10: e0005087.

3. Coltart CEM, Lindsey B, Ghinai I, Johnson AM, Heymann DL. The Ebola outbreak, 2013–2016: old lessons for new epidemics. Philos Trans R Soc B Biol Sci **2017**; 372:20160297.

4. International Ebola Response Team; Agua-Agum J, Ariyarajah A, Aylward B, et al. Exposure patterns driving Ebola transmission in West Africa: a retrospective observational study. PLoS Med **2016**; 13:e1002170.

5. Kelly JD, Barrie MB, Mesman AW, et al. Anatomy of a hotspot: chain and seroepidemiology of Ebola virus transmission, Sukudu, Sierra Leone, 2015–16. J Infect Dis **2018**; 217:1214–21.

6. Glynn JR, Bower H, Johnson S, et al. Asymptomatic infection and unrecognised Ebola virus disease in Ebola-affected households in Sierra Leone: a cross-sectional study using a new non-invasive assay for antibodies to Ebola virus. Lancet Infect Dis **2017**; 17:645–53.

7. Diallo MSK, Rabilloud M, Ayouba A, et al. Prevalence of infection among asymptomatic and paucisymptomatic contact persons exposed to Ebola virus in Guinea: a retrospective, cross-sectional observational study. Lancet Infect Dis **2019**; 19: 308–16.

8. Richards P. Ebola: how a people's science helped end an epidemic. London: Zed Books; **2016**.

9. Gardy JL, Johnston JC, Sui SJH, et al. Whole-genome sequencing and social-network analysis of a tuberculosis outbreak. N Engl J Med **2011**; 364:730–9.

10. De P. Sexual network analysis of a gonorrhoea outbreak. Sex Transm Infect **2004**; 80:280–5.

11. Firestone SM, Christley RM, Ward MP, Dhand NK. Adding the spatial dimension to the social network analysis of an epidemic: investigation of the 2007 outbreak of equine influenza in Australia. Prev Vet Med **2012**; 106:123–35.

12. D'Angelo-Scott H, Cutler J, Friedman D, Hendriks A, Jolly A. Social network investigation of a syphilis outbreak in Ottawa, Ontario. Can J Infect Dis Med Microbiol **2015**; 26:268–72.

13. Helleringer S, Kohler H-P. Sexual network structure and the spread of HIV in Africa: evidence from Likoma Island, Malawi. AIDS **2007**; 21:2323–32.

14. Rizzo A, Pedalino B, Porfiri M. A network model for Ebola spreading. J Theor Biol **2016**; 394:212–22.

15. Hébert-Dufresne L, Young J-G, Bedson J, et al. The network epidemiology of an Ebola epidemic. arXiv [Preprint]. Posted online 16 November 2021. https://doi.org/10.48550/arXiv.2111.08686

16. Merler S, Ajelli M, Fumanelli L, et al. Spatiotemporal spread of the 2014 outbreak of Ebola virus disease in Liberia and the effectiveness of non-pharmaceutical interventions: a computational modelling analysis. Lancet Infect Dis **2015**; 15: 204–11.

17. Kiskowski MA. A three-scale network model for the early growth dynamics of 2014 West Africa Ebola epidemic. PLoS Curr **2014**; 6. doi: 10.1371/currents.outbreaks.c6efe8274dc55274f05cbcb62bbe6070

18. Kelly JD, Frankfurter RG, Tavs JM, et al. Association of lower exposure risk with paucisymptomatic/asymptomatic infection, less severe disease, and unrecognized Ebola virus disease: a seroepidemiological study. Open Forum Infect Dis **2022**; 9: ofac052.

19. Handcock MS, Gile KJ. Modeling social networks from sampled data. Ann Appl Stat **2010**; 4:5–25.

20. Burt RS, Meltzer DO, Seid M, et al. What's in a name generator? Choosing the right name generators for social network surveys in healthcare quality and safety research. BMJ Qual Saf **2012**; 21:992–1000.

21. Marin A, Hampton KN. Simplifying the personal network name generator: alternatives to traditional multiple and single name generators. Field Methods **2007**; 19:163–93.

22. Kouki P, Pujara J, Marcum C, Koehly L, Getoor L. Collective entity resolution in familial networks. In: 2017 IEEE International Conference on Data Mining, New Orleans, LA. **2017**:227–236. Available at: http://ieeexplore.ieee.org/document/8215495/. Accessed December 23, 2020.

23. Hunter DR, Handcock MS, Butts CT, Goodreau SM, Morris M. Ergm: a package to fit, simulate and diagnose exponential-family models for networks. J Stat Softw **2008**; 24:nihpa54860.

24. World Health Organization (WHO). Contact tracing during an outbreak of Ebola virus disease. Geneva, Switzerland: WHO; **2014**.

25. Caleo G, Duncombe J, Jephcott F, et al. The factors affecting household transmission dynamics and community compliance with Ebola control measures: a mixed-methods study in a rural village in Sierra Leone. BMC Public Health **2018**; 18:248.

26. Reichler MR, Bangura J, Bruden D, et al. Household transmission of Ebola virus: risks and preventive factors, Freetown, Sierra Leone, 2015. J Infect Dis **2018**; 218: 757–67.

27. Ajelli M, Parlamento S, Bome D, et al. The 2014 Ebola virus disease outbreak in Pujehun, Sierra Leone: epidemiology and impact of interventions. BMC Med **2015**; 13(1):281.

28. Cassidy L, Barnes GD. Understanding household connectivity and resilience in marginal rural communities through social network analysis in the village of Habu, Botswana. Ecol Soc **2012**; 17:art11.

29. Chowell G, Simonsen L, Viboud C, Kuang Y. Is West Africa approaching a catastrophic phase or is the 2014 Ebola epidemic slowing down? Different models yield different answers for Liberia. PLoS Curr **2014**; 6:ecurrents.outbreaks.b4690859d91684da963dc40e00f3da81.

30. Faye O, Boëlle P-Y, Heleze E, et al. Chains of transmission and control of Ebola virus disease in Conakry, Guinea, in 2014: an observational study. Lancet Infect Dis **2015**; 15:320–6.

31. Robert A, Edmunds WJ, Watson CH, et al. Determinants of transmission risk during the late stage of the West African Ebola epidemic. Am J Epidemiol **2019**; 188:1319–27.

32. Kelly JD, Richardson ET, Drasher M, et al. Food insecurity as a risk factor for outcomes related to Ebola virus disease in Kono district, Sierra Leone: a cross-sectional study. Am J Trop Med Hyg **2018**; 98:1484–8.

33. Richardson ET, Kelly JD, Sesay O, et al. The symbolic violence of 'outbreak': a mixed-methods, quasi-experimental impact evaluation of social protection on Ebola survivor wellbeing. Soc Sci Med **2017**; 195:77–82.

34. Merler S, Ajelli M, Fumanelli L, et al. Containing Ebola at the source with ring vaccination. PLoS Negl Trop Dis **2016**; 10:e0005093.

35. Kucharski AJ, Eggo RM, Watson CH, Camacho A, Funk S, Edmunds WJ. Effectiveness of ring vaccination as control strategy for Ebola virus disease. Emerg Infect Dis **2016**; 22:105–8.

36. Richardson ET, Fallah MP. The genesis of the Ebola virus outbreak in West Africa. Lancet Infect Dis **2019**; 19:348–9.

37. Richardson ET. Epidemic illusions: on a coloniality of global public health. Cambridge, MA: MIT Press; **2020**.

38. Frankfurter R, Kardas-Nelson M, Benton A, et al. Indirect rule redux: the political economy of diamond mining and its relation to the Ebola outbreak in Kono District, Sierra Leone. Rev Afr Polit Econ **2018**; 45:522–40.

39. Richards P, Amara J, Ferme MC, et al. Social pathways for Ebola virus disease in rural Sierra Leone, and some implications for containment. PLoS Negl Trop Dis **2015**; 9:e0003567.

40. Huttner A, Agnandji ST, Combescure C, et al. Determinants of antibody persistence across doses and continents after single-dose rVSV-ZEBOV vaccination for Ebola virus disease: an observational cohort study. Lancet Infect Dis **2018**; 18: 738–48.

41. Diallo MSK, Ayouba A, Keita AK, et al. Temporal evolution of the humoral antibody response after Ebola virus disease in Guinea: a 60-month observational prospective cohort study. Lancet Microbe **2021**; 2:e676-–e684.