Multiple phylogenetic studies of HIV in sub-Saharan Africa have shown that mobility-driven transmission frequently occurs: many communities export and import strains. Mobility-driven transmission can result in source–sink dynamics: one community can sustain a micro-epidemic in another community in which transmission is too low to be self-sustaining. In epidemiology, the basic reproduction number ($R_0$) is used to specify the sustainability threshold. $R_0$ represents the average number of secondary infections generated by one infected individual in a community in which everyone is susceptible. If $R_0$ is greater than 1, transmission is high enough to sustain an epidemic; if $R_0$ is less than 1, it is not. Here, we discuss the conditions that are needed (in terms of $R_0$) for source–sink transmission dynamics to occur in generalised HIV epidemics in sub-Saharan Africa, present an example of where these conditions could occur (ie, Namibia), and discuss the necessity of considering mobility-driven transmission when designing control strategies. Additionally, we discuss the need for a new generation of HIV transmission models that are more realistic than the current models. The new models should reflect not only geographical variation in epidemiology and demography, but also the spatial–temporal complexity of population-level movement patterns.

**Introduction**

The HIV pandemic is concentrated in sub-Saharan Africa, where around 25·5 million people are living with HIV. In countries in this region, HIV epidemics are driven by heterosexual transmission; populations are predominantly rural and highly mobile. When populations are mobile, transmission networks can be very complex. Individuals can become infected in their own communities by other residents, in their own communities by visitors from other communities, or during visits to other communities. Multiple phylogenetic studies of HIV in sub-Saharan Africa have shown that communities export and import strains, demonstrating that mobility-driven transmission frequently occurs. 

This type of transmission could potentially result in source–sink dynamics—ie, a community sustaining a micro-epidemic in another community in which transmission is too low to be self-sustaining. In epidemiology, the basic reproduction number ($R_0$) is used to specify the sustainability threshold. $R_0$ is defined as the average number of secondary infections generated by one infected individual (over their lifetime) in a community in which all individuals are susceptible. If $R_0$ is greater than 1, transmission is high enough to sustain an epidemic; if $R_0$ is less than 1, it is not.

The term source–sink dynamics was first used in the 1980s in the context of an ecological theory to explain how a species can persist in places where the population growth rate is negative. The theory was described by quantitatively defining demographical sources and sinks. The environment was assumed to consist of patches that differed in their quality to support population growth, and the species to be capable of dispersing. In a patch that functions as a source, the birth rate is greater than the death rate; in a patch that functions as a sink, the death rate is greater than the birth rate. By framing the theory in mathematical terms, it was shown that, under particular conditions, a source could maintain the persistence of the species in a demographical sink. The conditions were shown to depend upon four factors: the population growth rate in the source, the population growth rate in the sink, the relative size of the source and sink, and the dispersal rate. The theory has been used to understand the geographical range of various species, from butterflies to giraffes.

In 2004, Smith and colleagues introduced the concept of source–sink dynamics to the field of modelling the transmission dynamics of infectious diseases. They used the concept to understand, and to predict, the regional spread of hospital-acquired infections. In their model, a source is defined as a hospital that can sustain transmission of the infection, and a sink is a hospital that cannot. Over the past decade, mathematical models that include source–sink transmission dynamics have been used to understand the spatial epidemiology of malaria, polio, and plague. These multi-patch models are often referred to as metapopulation models.

**The classic model**

In the classic model of HIV transmission dynamics, only a single community is considered; the equations that specify this model are given in the appendix (p 1). Standard methods can be used to analyse the model and derive an expression for $R_0$:

$$R_0 = \frac{\beta C}{\mu + \sigma}$$

In this formula, $\beta$ is the average probability of transmission per partnership, $C$ the average rate of acquisition of sex partners, $\mu$ the per capita background death rate, and $\sigma$ the per capita AIDS-related death rate. If $R_0$ is greater than 1, the level of sexual activity within the community is high enough to sustain transmission, hence a micro-epidemic (ie, a community-level epidemic)
can occur.6 If $R_0$ is smaller than 1, the level of sexual activity within the community is too low to sustain transmission, hence a micro-epidemic cannot occur.

**The mobility model**
The classic model of the sexual transmission of HIV can straightforwardly be extended to include two connected communities that are linked by mobility; the equations that specify this new model (that we subsequently refer to as the mobility model) are given in the appendix (p 1). The $R_0$ for the mobility model (that we subsequently refer to as the mobility-linked $R_0$) was derived using standard methods8 (appendix p 2). The mobility-linked $R_0$ is defined as the average number of secondary HIV infections that one infected individual generates (over their lifetime) in a susceptible population with two linked communities.

The structure of the mobility model reflects results from phylogenetic studies of HIV in sub-Saharan Africa that have shown that viral introductions frequently occur in communities that are linked by individuals moving between them.2–4 In addition, the structure reflects the results from multiple epidemiological studies that have shown that individuals spend a lot of time outside their home communities, and acquire HIV infection when away from home.16–18

In the mobility model, residents of each community spend a certain proportion of their time in the other community. Therefore, sexual transmission can occur via three pathways. Firstly, an individual can acquire HIV in their home community from another resident of their home community (localised transmission). Secondly, an individual can acquire HIV in their home community from a visitor from another community (visitor transmission). Thirdly, an individual can acquire HIV in a community that they visit (external-community transmission). Viral introductions can occur because of visitor transmission or external-community transmission.

Each community can function as a source or a sink. A community is a source if, when unlinked, the frequency and patterns of sexual contacts within the community are enough to sustain transmission in that community—ie, $R_0$ in the unlinked source is greater than 1. A community is a sink if, when unlinked, the sexual contacts within the community cannot sustain transmission in that community—ie, $R_0$ in the unlinked sink is less than 1. The mathematical expression for the $R_0$ for the source or the sink, when unlinked, is the same as for the classic model.

The mathematical expression for the mobility-linked $R_0$ is complex (appendix p 2), because it reflects three possible transmission pathways that occur within each community or between the two communities A and B. Namely, localised transmission can occur in community A, localised transmission can occur in community B, visitor transmission can occur if a resident of A (who is a person living with HIV) visits community B, visitor transmission can occur if a resident of B (who is a person living with HIV) visits community A, external-community transmission can occur if a resident of A acquires HIV infection when visiting community B, and external-community transmission can occur if a resident of B acquires infection when visiting community A.

If the mobility-linked $R_0$ is greater than 1, the overall level of sexual transmission is high enough to sustain micro-epidemics in both communities. If the mobility-linked $R_0$ is less than 1, the overall level of sexual transmission is too low to sustain a micro-epidemic in either community. In this case, due to mobility, the source is no longer able to sustain a micro-epidemic in its own community because its residents spend some of their time in the sink community.

**Conditions for sources and sinks**
We propose that two conditions are needed for source–sink transmission dynamics to occur in generalised HIV epidemics in sub-Saharan Africa: substantial geographical heterogeneity in HIV prevalence and a highly mobile population that connects high-prevalence and low-prevalence areas.

Multiple geospatial mapping studies have shown that considerable geographical variation in HIV prevalence exists among countries in sub-Saharan Africa.20–24 These studies have used georeferenced HIV-testing data, collected using demographic and health surveys,25 to construct epidemic surface prevalence maps.21 An epidemic surface prevalence map for Namibia (figure 1A) was constructed using HIV-testing data from 9309 individuals (15–64 years) who participated in the 2013 Namibian Demographic and Health Survey.28 The average HIV prevalence in Namibia is 14%, but the epidemic surface prevalence map shows that it varies geographically from 0% to 32%. Considerable variation in prevalence exists within some of the 13 regions of Namibia. However, at a smaller scale (ie, within constituencies), very little variation exists. On a temporal scale, HIV prevalence in Namibia has remained stable for several years, because rates of HIV incidence and AIDS-related mortality are fairly low.

It is well known that population mobility is high throughout sub-Saharan Africa.20–24 However, to determine whether individuals move between high-prevalence and low-prevalence areas, it is necessary to identify population-level movement patterns. These patterns can be identified by analysing large datasets of call detail records collected from mobile phones. The availability of this new type of data has transformed the study of infectious diseases.25 Datasets of call detail records have been used to identify population-level movement patterns in several African countries, including Kenya,26 Senegal,21,23 the Ivory Coast,24 and Namibia.29 The dataset of call detail records for Namibia was collected between October, 2010, and September, 2011, and is based on 9 billion calls or texts from 1·19 million unique SIM cards. Data were collected from around 90% of cell phone subscribers in Namibia. Population-level movement patterns in
Namibia have been shown to be fairly similar from year to year, over 4 years.34 Ruktanonchai and colleagues11 used these Namibian data to construct a mobility network (figure 1B), which they applied to study the transmission dynamics of malaria. 96 of the 106 constituencies in Namibia are included in the network; the remaining ten do not contain cell phone towers. The data show that residents of Namibia spent around 22% (IQR 18–26%) of time, over a 12-month time span, outside their home constituency. A visual comparison of the mobility network (figure 1B) with the epidemic surface prevalence map (figure 1A) suggests that the movement patterns in Namibia could result in some strong linkages between constituencies in which the prevalence of HIV is high and constituencies in which the prevalence is low.

**An example**

To see whether source–sink transmission dynamics could occur in sub-Saharan Africa, we used Namibia as an example, and calculated values of the mobility-linked $R_0$ for two communities connected by mobility. To make these calculations, we firstly assumed that one community was a transmission source and the other community a transmission sink. Secondly, we assumed that an infected resident in the sink community (when the sink is not connected to the source) could only cause (on average) between 0·50 and 0·95 secondary infections over their lifetime. We then did an uncertainty analysis35–37 to calculate the potential values, given these assumptions, for a mobility-linked $R_0$ in Namibia. We used Latin hypercube sampling and specified parameter ranges to reflect the geographical variation in HIV prevalence (figure 1A), and the variation in time spent by individuals outside their home community, which was based on the mobile phone data; ranges are given in the appendix (p 2).

To do the uncertainty analysis, we calculated 10000 values of the mobility-linked $R_0$ (figure 2A). The median value is 1·08 (IQR 1·01–1·15) and around 80% of all values are greater than 1, which signifies that many communities in Namibia are capable of functioning as a transmission source and sustaining an HIV micro-epidemic in a transmission sink. However, this analysis does not prove that transmission sinks exist in Namibia.

We used the results of the uncertainty analysis to identify the possible epidemiological outcomes of linking a source to a sink (figure 2B). To do the analysis, we plotted the 10000 values of the mobility-linked $R_0$ in the form of a response hypersurface;38 the mobility-linked $R_0$ is the response variable. Above the threshold (figure 2B), the mobility-linked $R_0$ is greater than 1 and the source can sustain a micro-epidemic in the sink. Below the threshold, the mobility-linked $R_0$ is less than 1 and sustainability is not possible in either the source or the sink. Some sources can sustain micro-epidemics in sinks in which transmission is very low (figure 2A). For example, a source with an $R_0$ (when it is unlinked) of around 1·25 (which is equivalent to an HIV prevalence of around 20%) can sustain a micro-epidemic in a sink in which a resident (on average) generates only 0·50 secondary infections.

Whether a source is able to sustain a micro-epidemic in a sink also depends upon the relative size of the source in comparison to the sink; size is defined as the number

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**Figure 1:** HIV prevalence map and mobility network for Namibia

(A) The epidemic surface prevalence map shows HIV prevalence (% indicated by the colour scale) in individuals aged 15–64 years. The map was constructed using an adaptive bandwidth kernel density estimation method26 to smooth and spatially interpolate the HIV-testing data from the Namibia Demographic and Health Survey; the R programming package prevR was used for implementation.27 Black lines mark region boundaries. (B) Mobility network constructed from the dataset of call detail records.11 Nodes (green dots) represent constituencies and blue lines show the mobility links between pairs of constituencies. The thickness of the blue lines indicates the fraction of time spent, over a year, by the average resident of one constituency in the linked constituency.
of individuals living in the community. The larger the size of the source in comparison to the sink, the easier it is for the source to sustain a micro-epidemic in the sink (figure 2C). Whether a source can sustain a micro-epidemic in a sink also depends upon the time spent in each community: the more time residents of the source spend in the sink, the harder it is for the source to sustain a micro-epidemic in the sink.

Data quality
Modelling is never based on perfect data. All published HIV (and HIV–tuberculosis) models have problems with data quality, representativeness, and granularity. For example, data on sexual behaviour are notoriously inaccurate and unreliable, and there are no complete (or even partially complete) datasets for specifying countrywide sexual-mixing patterns. For tuberculosis, even the basic transmission probabilities and other biological parameters that reflect the natural history of the disease are unknown, and so-called guesses are used. Because data are never perfect, modellers use uncertainty analyses when making predictions. In our modelling, we used an uncertainty analysis to predict the outcome of linking a transmission source to a transmission sink, this application enabled us to predict outcomes, even though the specific values of the mobility and epidemiological parameters are unknown. This analysis also enabled us to show that source–sink transmission dynamics can occur under a wide range of conditions.

Next-generation transmission models
We believe that there is a need for a new generation of HIV (and HIV–tuberculosis) transmission models that are more realistic than the current models. The underlying structure of HIV (and HIV–tuberculosis) transmission models has remained the same for decades. Models have increased in complexity, but only in terms of modelling sexual behaviour. These complex models can give a misleading sense of certainty. They are often more imprecise than simpler models because they are based on multiple assumptions (many of which are unverifiable), and often require data for parameterisation that do not exist. Current HIV transmission models are based on the assumptions that individuals are immobile,
that only localised transmission can occur, and that viral introductions are not possible. Clearly, these assumptions are unrealistic; they go against all of the evidence shown by the multiple phylogenetic studies and epidemiological studies that have been done in sub-Saharan Africa. Consequently, current HIV (and HIV–tuberculosis) models do not provide an adequate representation of generalised HIV epidemics in sub-Saharan Africa.

We believe that it is essential to develop a new generation of HIV transmission models that allow for countryside spatial mixing. These models should include mobility-driven transmission and allow for the occurrence of source–sink dynamics. They should be data intensive and reflect not only geographical variation in prevalence, but also geospatial heterogeneity in demography and the spatial–temporal complexity of population-level movement patterns. The mobility model, presented in this Viewpoint, could be used as a foundation for building more realistic models than the currently used models. These more realistic models would result in more accurate predictions and enable the design of more cost-effective control strategies for HIV epidemics in sub-Saharan Africa.

Real-world implications

The results from phylogenetic and epidemiological studies of HIV in sub-Saharan Africa have shown that strain exportation and importation occurs, and that mobility-driven transmission is important. However, to date, no phylogenetic or epidemiological study of HIV in sub-Saharan Africa has shown that source–sink dynamics exist. We propose that, in sub-Saharan Africa, source–sink dynamics might be occurring in areas where urban centres are tightly connected, by mobility-linked transmission, to rural communities. In this case, urban centres would function as transmission sources and rural communities as transmission sinks. The necessary conditions for source–sink dynamics exist in many places in sub-Saharan Africa: HIV prevalence in urban centres is often substantially greater than in surrounding rural areas, and there is frequent urban–rural and rural–urban travel. However, the mobility networks that link communities are highly complex and therefore transmission sources and sinks will be hard to identify. We suggest that the methods that have been developed to identify sources and sinks for malaria transmission in sub-Saharan Africa should be used to identify sources and sinks (if they exist) within generalised HIV epidemics in sub-Saharan Africa.

To design cost-effective HIV control strategies for sub-Saharan Africa, it is essential to understand the transmission dynamics of generalised epidemics. The current global health policy for controlling HIV recommends the use of geographical and behavioural targeting, particularly of individuals who are highly sexually active and live in high-prevalence areas that are defined as hot spots. Results derived from mathematical models have shown that this type of targeting would be the most cost-effective strategy for controlling HIV. However, we believe that the cost-effectiveness of targeting hot spots should be re-evaluated using more realistic transmission models that consist of multiple communities connected by a mobility network; such models will have multiple hot spots. We predict that hot spot targeting will be cost-effective for reducing transmission in some of the hot spots, but not in others. For example, treatment as prevention could be cost-effective in hot spots in which incidence is mainly due to localised transmission, but not in hot spots in which a high proportion of new infections are due to mobility-driven transmission. We recommend that, to identify cost-effective strategies, both localised transmission and mobility-driven transmission need to be taken into consideration.

Designing and implementing new HIV prevention strategies that consider mobility will be very challenging. One such approach, which is a focus of our research, is to integrate mobility and prevalence data to construct country-level risk networks that are gender specific. These networks can be used to determine, for each community in the country, whether it exports or imports risk. A community exports risk if its residents have the potential to infect residents of other communities; a community imports risk if its residents can potentially be infected by residents of other communities. The risk networks could be used to design and implement prevention strategies. The communities that are the most important exporters of risk, and those that are the most vulnerable to the importation of risk, are the communities that should be targeted. The communities that are the most important exporters of risk might not be those with the highest prevalence of HIV. High levels of mobility have been shown to be a substantial obstacle to the global attempt to eliminate malaria, and might also be a substantial obstacle to the global attempt to eliminate HIV.

Contributors

SB developed the concept, drafted the Viewpoint, and interpreted results; JTO and KS programmed the model, generated figures, and interpreted results. EV calculated the mobility estimates from the network, and interpreted results. JTO, EV, and LP contributed to writing. All authors read and approved the final manuscript.

Declaration of interests

We declare no competing interests.

References


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