

Supplementary Materials for:

Generic Patterns in HIV Transmission Dynamics: Insights from a Phenomenological Risk-Stratified Modelling Approach

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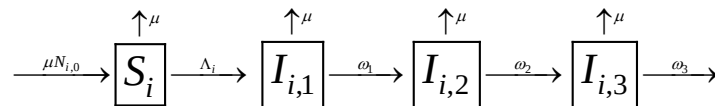
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1. Supplementary Methods

1.1 Equations and description

The compartmental model structure is shown in Figure S1.1. The transmission of HIV is represented by individuals “flowing” from the compartment of susceptible to the compartments of infected, while HIV-related mortality is removed from the compartments of infected. The population is divided into arbitrary number of sexual risk groups, defined with the index i .

Figure S1.1. Compartmental structure of the HIV population level model. S is the susceptible population, the I 's is the infected population in their different stages.



The rates Λ_i are the HIV forces of infection (hazard rates) experienced by each susceptible population S_i . The Λ_i is given by

$$\Lambda_i = \rho_{S_i} \sum_{j=1,2,3,4} \sum_{\beta=1,2,3} t_{I_{j,\beta} \rightarrow S_i} G_{i,j} \frac{\rho_{I_{j,\beta}} I_{j,\beta}}{\rho_{S_j} S_j + \sum_{\beta=1,2,3} \rho_{I_{j,\beta}} I_{j,\beta}} \quad (\text{S1.1})$$

ρ_{X_i} describe the *effective* new sexual partner acquisition rates for each population variable X_i . The parameters $t_{I_{j,\beta} \rightarrow S_i}$ defines the HIV transmission probability per partnership in a partnership between a member of the susceptible population S_i and a member of the HIV infected population $I_{j,\beta}$, using the binomial model

$$t_{I_{j,\beta} \rightarrow S_i} = 1 - \left(1 - p_{I_{j,\beta} \rightarrow S_i}^{HIV} \right)^{n_{I_{j,\beta} \leftrightarrow S_i} \tau_{I_{j,\beta} \leftrightarrow S_i}} \quad (\text{S1.2})$$

It is expressed in terms of HIV transmission probability per coital act per HIV stage in this partnership ($p_{I_{j,\beta} \rightarrow S_i}^{HIV}$), the frequency of coital acts per HIV stage in this partnership ($n_{I_{j,\beta} \leftrightarrow S_i}$), and the duration ($\tau_{I_{j,\beta} \leftrightarrow S_i}$) of this partnership.

The mixing between the risk groups is dictated by the sexual-mixing matrix $G_{i,j}$ that provides the probability that an individual in risk group i would choose a partner in risk group j . It is given by the expression

$$G_{i,j} = e \delta_{i,j} + (1-e) \frac{\rho_{S_j} S_j + \sum_{\beta=1,2,3} \rho_{I_{\beta}(j)} I_{\beta}(j)}{\sum_{k=1,2,3,4} \left(\rho_{S(k)} S(k) + \sum_{\beta=1,2,3} \rho_{I_{\beta}(k)} I_{\beta}(k) \right)}. \quad (\text{S1.3})$$

Here, $\delta_{i,j}$ is the identity matrix and the parameter $e \in [0,1]$ measures the degree of assortativeness in the mixing. At the extreme $e = 0$, the mixing is proportionate while at the other extreme $e = 1$, the mixing is fully assortative as individuals choose partners only from within their own risk group.

1.2 Equilibrium solution

The equilibrium was obtained by setting the left-hand-side of the four differential equations (Eq. (1-4) in the main script) equal to zero. It can be written in the form of a matrix equation namely,

$$Mz = b \quad (S1.4)$$

The matrix z was defined as

$$z_i = \begin{pmatrix} S_i \\ I_{i,1} \\ I_{i,2} \\ I_{i,3} \end{pmatrix} \quad (S1.5)$$

The $4N_{risk} \times 4N_{risk}$ matrix M can be written as

$$M_i = \begin{pmatrix} -(\mu + \Lambda_i) & 0 & 0 & 0 \\ \Lambda_i & -(\mu + \omega_1) & 0 & 0 \\ 0 & \omega_1 & -(\mu + \omega_2) & 0 \\ 0 & 0 & \omega_2 & -(\mu + \omega_3) \end{pmatrix} \quad (S1.6)$$

The vector b can be obtained by comparing Equation (S1.6) to Equations (1-4) (from the main script) with the left-hand-side set equals to zero

$$b_i = \begin{pmatrix} -N_{i,0} \\ 0 \\ 0 \\ 0 \end{pmatrix} \quad (S1.7)$$

We derived the expressions for the population variables in terms of the force of infection and other parameters

$$S_i = \frac{N_{i,0}\mu}{\Lambda_i + \mu} \quad (S1.8)$$

$$I_{i,1} = \frac{N_{i,0}\Lambda_i\mu}{(\Lambda_i + \mu)(\mu + \omega_1)} \quad (\text{S1.9})$$

$$I_{i,2} = \frac{N_{i,0}\Lambda_i\mu}{(\Lambda_i + \mu)(\mu + \omega_1)(\mu + \omega_2)} \quad (\text{S1.10})$$

$$I_{i,3} = \frac{N_{i,0}\Lambda_i\mu}{(\Lambda_i + \mu)(\mu + \omega_1)(\mu + \omega_2)(\mu + \omega_3)}. \quad (\text{S1.11})$$

1.3 Key parameter values

Table S1.1. Model assumptions in terms of parameter values.

Assumption	Parameter value	Sources
HIV transmission probability per coital act per stage of infection ($p_{I_\beta(i) \rightarrow S(j)}^{HIV}$):		
Acute stage	0.0107	[1-4]
Latent stage	0.0008	[4,5]
Late stage	0.0042	[1,2,4]
Duration of each of HIV stages ($1/\omega_{I_\beta}$):		
Acute stage	49 days	[1,4,6-9]
Latent stage	9.0 years	[1,4,6-9]
Late stage	2.0 years	[1,4,6-9]
Frequency of coital acts per HIV stage ($n_{I_\beta(i) \leftrightarrow S(j)}$):		
Acute stage	10.6 per month	[4]
Latent stage	11.0 per month	[4]
Late stage	7.1 per month	[4]
Duration of sexual partnerships ($\tau_{I_\beta(i) \leftrightarrow S(j)}$):	6 months	Representative value
Degree of assortativeness (e)	0.3	Representative value
Duration of the sexual lifespan ($1/\mu$)	35 years	[5,10-12]

1.4 Individual based model

Networks have some emerging properties such as the degree to which the nodes in a network have the tendency to cluster together, i.e., the presence of a connection between nodes A and B, and another connection between B and C, indicates that A and C are most likely to be connected too [13,14]. In network topology terms, clusters enclose a high density of triangles ABC in the network. The clustering of the network can be quantified by estimating the local clustering coefficient (C_i), which quantifies how close the neighbors of a node are to be connected to each other and is defined as [13]:

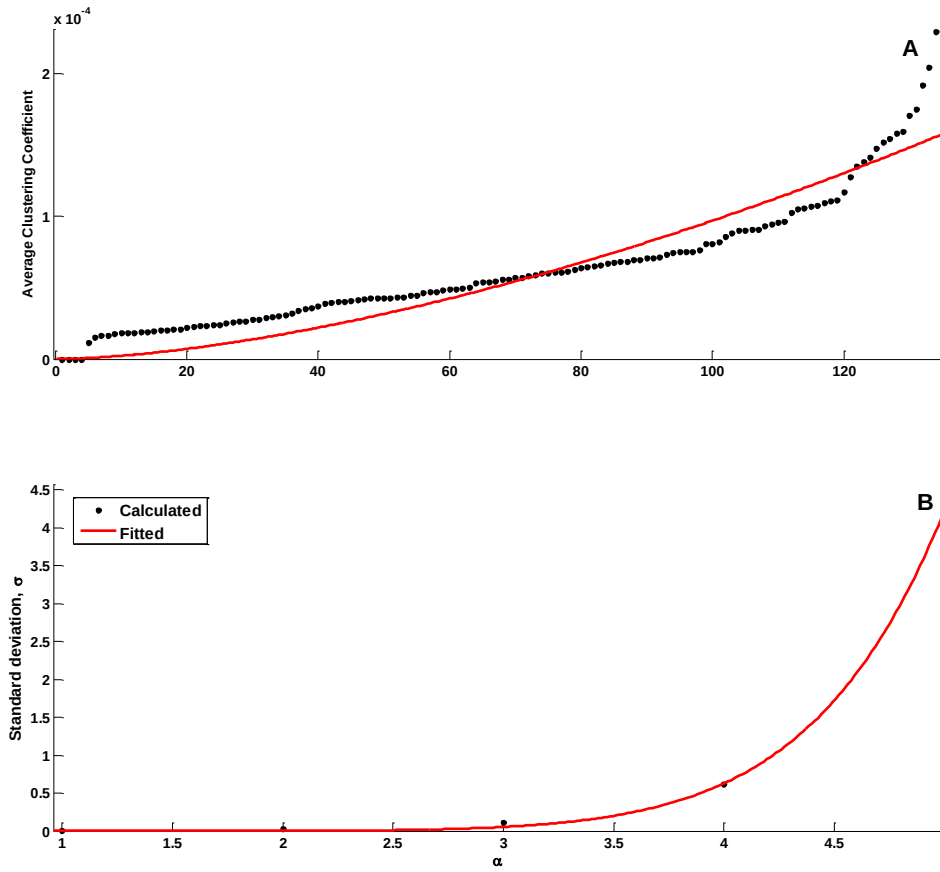
$$C_i = \frac{|\{e_{jk}\}|}{k_i(k_i - 1)}. \quad (\text{S1.12})$$

Where the C_i for a node i is given by the proportion of connections between node (e_{jk}) with its neighborhood and by the entire possible number of connections that exist between them ($k_i(k_i - 1)$); where k_i is the degree of the node i). In epidemiology, this network's attribute can have substantial effect on the spread of an infection and the size of the epidemic. The clustering in a network generate many several paths between individuals which provide many different routes for transmission of the infection, making it easier for the infection to spread and increasing the likelihood for individuals to catch the infection by one route or another, independently of the transmissibility of the infection [13,15]. Motivated by the previous argument, we generated an individual-based network model to explore the diversity of the clustering into the different risk groups. We first generated a fixed number of virtual individuals which were connected with a fixed number of links. To generate the links, the program successively picks

two individuals in a random fashion until the maximum number of connections is reached. This procedure is repeated until all possible topological configurations are generated. Afterwards, we estimated the local clustering coefficient for each individual (n), and estimated the network average clustering coefficient for each network configuration as [13]:

$$C_i = \frac{1}{n} \sum_{i=1}^n C_i . \quad (\text{S1.13})$$

Figure S1.2. Panel A shows the average clustering coefficient distribution for all possible configurations of a network with fixed number of individuals and partnerships. Panel B shows the standard deviation in HIV risk of exposure as a function of the distribution of sexual risk behavior, as parameterized by the parameter α ($\rho_i = Ci^\alpha$).



1.5 Sexual contact structure

The heterogeneity of sexual risk behavior in human populations was mapped by defining different forms of the sexual contact structure and variability within each sexual contact structure. Each sexual contact structure was specified by stratifying the sexually active population into a number of risk groups, a hierarchy of risk behavior across these risk groups starting from lower to higher levels of risk behavior, a specific risk behavior distribution across these risk groups, and a mixing matrix describing how each risk group mixes with the rest of the groups.

The number of individuals initially in a specific risk group i , that is in absence of HIV infection and AIDS mortality, is given by

$$N_0(i) = N_0 p(i), \quad (\text{S1.14})$$

where $p(i)$ is the proportion of the initial population in each risk group i . $p(i)$ was described in different calculations using each of a gamma distribution and a uniform distribution. The uniform distribution was motivated by the concept of dividing the population generically into equal segments, each with a different level of risk behavior. The gamma distribution was motivated by the number of sexual partners degree distribution identified empirically in studies in sub-Saharan Africa, the epicenter of the HIV epidemic [16-19].

The level of sexual risk behavior in each risk group was parameterized by the effective sexual partner acquisition rate (ρ_i) [20,21]. This parameter describes the number of sexual partners an individual in a specified risk group acquires, but also effectively factors effects arising from the topology of sexual networks such as concurrency and clustering within these networks [5,22-24],

and the variability in risk behavior in a population [5,25]. The sexually active population was stratified into $n=100$ or $n=200$ risk groups, with specific risk behavior. To synthesize these different behavioral attributes, we used a generic form for the distribution of ρ_i across the risk groups defined through a power law as

$$\rho_i = Ci^\alpha. \quad (S1.15)$$

Here C and α are the parameters of the distribution, where:

- ρ_i : Represents the relative risk level of group i (e.g., number of sexual partners).
- C : A scaling constant that normalizes the risk distribution.
- i : The index of the risk group (ordered by ascending risk level).
- α : The heterogeneity parameter, which controls the degree of risk stratification.

This equation reflects the heterogeneous nature of risk observed in real-world HIV transmission, where a small subset of high-risk individuals disproportionately drive the epidemic. For higher values of α , risk becomes increasingly concentrated in the highest-risk groups, representing populations such as sex workers, men who have sex with men (MSM), or people who inject drugs (PWID). Conversely, lower α values indicate a more uniform distribution of risk across the population.

This form was motivated based on different theoretical grounds. First, we developed an individual based network model (described above), where we had a fixed number of individuals (nodes) and partnerships (edges or connections) and generated all possible topologies in the sexual network by connecting the individuals randomly. Consequently we calculated the local clustering coefficient, a measure of the level of clustering for each node in the network, and then

we calculated the average clustering coefficient for the whole network [26], for each random network generated. We observed that the average clustering coefficient distribution among all possible network configurations follows a power law, see Figure S1.1. in Supplementary Materials. This provided the first motivation for this form.

A second motivation for a power law arises from analyses of the architecture of complex weighted networks [27,28]. Sexual networks are one form of weighted networks where the frequency of coital acts per partnerships is a measure of the *weight* of the partnership (link). In weighted networks, the strength of a node can be expressed as a metric called *strength*, defined using the total weight of the node links rather than the node degree [27]. Strength is a natural measure of the centrality of an individual in a sexual network, and therefore a measure of the risk of HIV exposure in a sexual network. Statistical analyses of weighted networks indicate that the strength of a node follows a power law behavior $s(k) \sim k^\beta$, where k is the node degree [27,28].

Lastly, a power law is also reasonable on intuitive grounds. An individual with k partnerships can in principle acquire HIV infection directly from k other individuals. Each of these partners however can have in turn a number of partners from which they can acquire the infection and pass it indirectly to this individual. Each of the partners of the partners can also have a number of partners from which they can acquire HIV infection and pass it indirectly to the original individual. This suggests that the risk of exposure to HIV may scale according to some power law with the number of direct partnerships (k). Indeed, in a network of average k partnerships per individual, there are k^2 two links away and roughly k^d nodes d links away [29].

Accordingly, the parameter α in the power law can be thought of as a measure of the average separation in a network or sub-network [29]; a proxy of the size of the “ecology” through which an individual can acquire HIV infection (risk of exposure to HIV infection). It bears notice that

for a random network $d \approx \frac{\log N}{\log k}$ where N is the number of individuals (nodes) in the network [26,29].

Likewise, the power-law structure directly influences the force of infection (Λ_i) through the following mechanisms:

- *Direct Scaling Effects:* At low prevalence, the scaling factor is approximately proportional to $(i\alpha)$, meaning that a higher value of (α) increases the differences between risk groups. However, at higher prevalence levels, saturation effects make this relationship more complex as transmission rates approach their maximum limits.
- *Transmission Amplification:* The power law governs both the acquisition and transmission dynamics within each risk group, creating multiplicative effects on Λ_i . These effects produce the observed exponential-like growth in Λ_i across higher-risk groups. This relationship establishes the connection between the underlying population structure and the emergent epidemic dynamics observed in the model results.

1.6 Power-Law Scaling of the Force of Infection

The risk level ρ_i directly influences the force of infection (Λ_i) experienced by individuals in group i . As α increases, Λ_i scales nonlinearly across groups, leading to superspreading effects where high-risk groups account for the majority of new infections. This dynamic underpins the RMP, which represents the critical threshold where transmission transitions from unsustainable to self-sustaining.

In our model, Λ_i , which determines the rate at which individuals in risk group i acquire HIV, follows a power-law relationship with risk behavior (ρ_i^α). This is expressed mathematically as:

$$\Lambda_i \propto \rho_i^\alpha \quad (\text{S1.16})$$

where:

- ρ_i represents the risk level of individuals in group i , such as the partner acquisition rate or contact frequency.
- α is the heterogeneity exponent, which determines the degree of transmission amplification across risk groups.

This relationship reflects the nonlinear concentration of transmission in high-risk groups, where small increases in risk behavior result in disproportionately large increases in infection risk.

When $\alpha > 1$, the force of infection scales super-linearly, meaning that transmission is primarily driven by a small subset of high-risk individuals. Conversely, lower values of α (approx $\alpha \approx 1$) indicate more evenly distributed transmission dynamics across the population.

Relevance of Power-Law Structures for HIV Transmission Modeling

Power-law distributions are widely used in epidemiological modeling to describe heterogeneous contact patterns in infectious disease transmission. Empirical studies have demonstrated that sexual contact networks, particularly in HIV epidemics, do not follow normal or homogeneous mixing assumptions but instead exhibit heavy-tailed distributions where a small proportion of individuals account for a disproportionately high number of interactions. This characteristic,

known as superspreading, is well-documented in key populations such as sex workers, men who have sex with men (MSM), and people who inject drugs (PWID).

By incorporating power-law scaling into the force of infection, our model captures:

1. Extreme heterogeneity in risk behavior, reflecting real-world disparities in HIV transmission.
2. The role of superspreaders in epidemic persistence, where a minority of individuals sustain transmission.
3. The threshold dynamics that determine epidemic transitions, which interact with the RMP to shape intervention strategies.

The use of a power-law structure provides a more realistic framework for epidemic modeling, ensuring that risk stratification accurately reflects observed transmission patterns. This allows for better-targeted interventions, particularly in high-prevalence settings where transmission is concentrated in specific subpopulations.

Our model results show that the power-law scaling of risk levels ($\rho_i = Ci^\alpha$) generates key epidemic dynamics across different prevalence regimes. At low prevalence ($\sim 0.2\%$), higher α values concentrate transmission in extreme high-risk groups ($i > 90$), while at high prevalence ($\sim 20\%$), saturation effects reduce the impact of stratification, leading to broader transmission across risk levels. This scaling behavior aligns with real-world observations of superspreading dynamics in key populations and emphasizes the importance of targeting high-risk groups to disrupt epidemic persistence.

2. Supplementary results

2.1 definition of Average risk behavior

The distribution of risk behavior is governed by the function ρ_i defined in Equation (6) in the main script. The average risk behavior is defined as

$$\rho_{avg} = \frac{\sum_{i=1}^{N_{risk}} \rho_i N_i}{\sum_{i=1}^{N_{risk}} N_i} \quad (S2.1)$$

N_i is the total population per risk group and is defined as

$$N_i = S_i + \sum_{\beta=1,2,3} I_{i,\beta} . \quad (S2.2)$$

2.2 Incidence rate distribution fitting

Table S2.1. Power law model fit for the incidence rate at six different distributions of risk behavior. The table shows the values of the parameters to fit the incidence rate with the power law function ($f(i) = ai^b$) for six different distribution of sexual risk behavior as parameterized by α along with the adjusted R^2 of each fit.

α	a	b	Adjusted R-square
0	0.03172	0	0.8883
1	0.001591	1	1
2	4.76×10^{-5}	2	1
3	1.52×10^{-6}	3	1
4	4.91×10^{-8}	4	1
5	1.86×10^{-9}	5	1

2.3 Logistic function fitting

The shape of the prevalence per risk group y_i as a function of i for the nonlinear case in Figure 4 in the manuscript is very suggestive of a logistic model. This statement is motivated by the three distinct regions in the calculation. To investigate this conjecture, we fitted a logistic model for y_i as function of i of the form

$$y(i) = \frac{L}{1 + Ce^{-ri}}. \quad (\text{S2.3})$$

A graphical representation of the fit for four nonlinear distributions of the risk behavior ($\alpha = 2, 3, 4, 5$) is shown in Figure S2.1. The logistic model parameters L , C and r as well as the inflection points are given in Table S2.2.

Figure S2.1. The distribution of HIV prevalence at different distributions of sexual risk behavior with 200 risk groups. The HIV prevalence as a function of the risk groups along with the fitted logistic function. Panel A) to D) shows the fitting at different distributions of sexual risk behavior, as parameterized by α ($\alpha = 2, 3, 4, 5$).

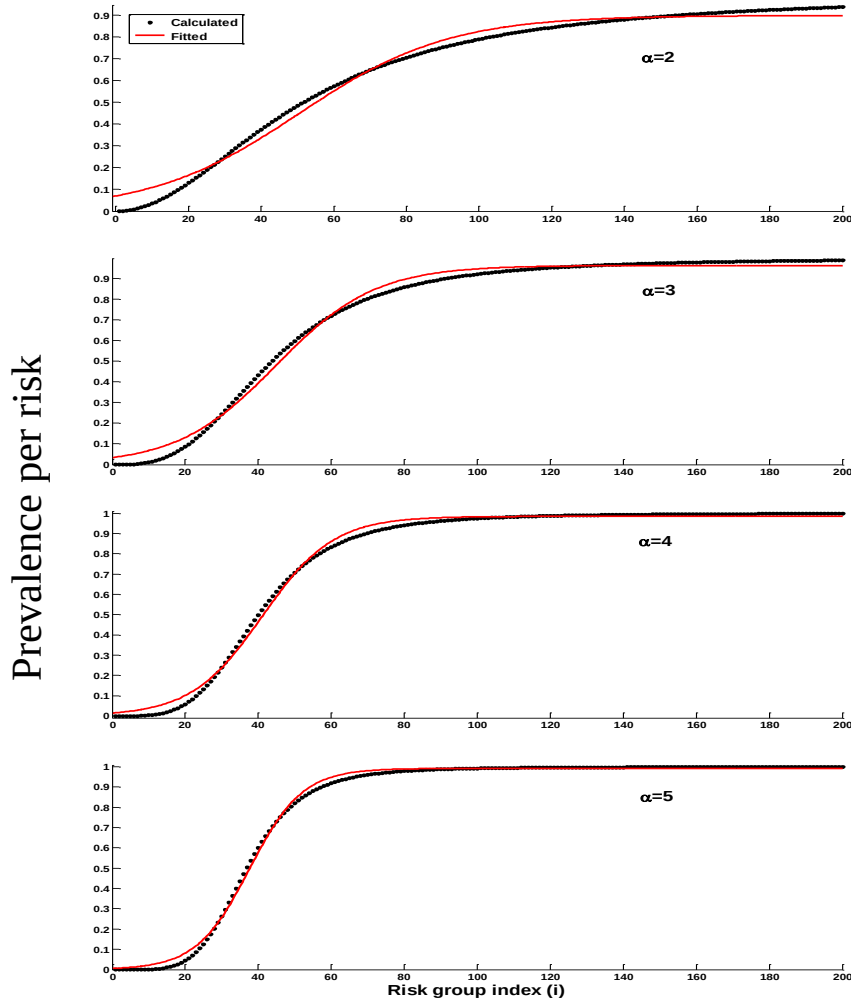


Table S2.2: HIV prevalence fitted with logistic functions at different distributions of risk behavior for 20% prevalence with 200 risk groups. The coefficients for the best fitting of the distribution of HIV prevalence to logistic function at different distributions of risk behavior along with the inflection point (point of dynamical transition) and the point where $R_0 = 1$ (risk group and corresponding prevalence).

Alpha	L	C	r	Adjusted R-square	Inflection point	$R_0 = 1$
2	0.8982	11.78	0.04882	0.9887	0.4491	(37,0.4321)
3	0.9641	27.73	0.07384	0.9932	0.4925	(45,0.4877)
4	0.9844	68.97	0.1029	0.9965	0.4922	(43,0.4982)
5	0.9922	176.7	0.1376	0.9981	0.4961	(40,0.5050)

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