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Is 2 a "High Number of Partners"? Modeling, Data, and the Power of Concurrency

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To the Editor

Go and Blower[1] claim 3 necessary conditions for concurrency to drive heterosexual HIV epidemics: (1) "there should be many concurrent partnerships in the population;" (2) "the number of concurrent partners should be fairly high for the average individual;" (3) "the duration over which the partnerships overlap should be fairly long." Also, because "no heterosexual community in which these conditions are met has been identified," they conclude that concurrency is irrelevant to African HIV epidemiology. They provide no citations for their conditions, with good reason: the first 2 are untrue. Condition 1 depends on the definition of "many," but modeling shows that only a relatively small fraction of the population need concurrent partners to generate sizeable heterosexual epidemics. Condition 2 is entirely incorrect.

For example, Goodreau et al.[2] considered behavioral data from 18- to 30-year-old Zimbabweans, the ages where incidence is concentrated; 11% of men and 5% of women had concurrent partners. Mean momentary degree (MMD; i.e., number of ongoing partnerships at any point) was 0.66. Using per-stage transmission probabilities,[3] they showed that this would generate 9% HIV prevalence. If sexual contacts and concurrency were slightly underreported (MMD increased to 0.7, concurrency by 2 percentage points), prevalence rose to 14%. The population is on a threshold, and small underreporting in concurrency (a socially undesirable behavior, for women especially) could generate a realistic epidemic. Among those with concurrent partners, 79% had the minimum possible (2); MMD for those with concurrent partners was 2.28. When modeling the same MMD and durations but without concurrency, the epidemic disappeared. Concurrent partnerships do not need to be very numerous to drive an epidemic.[4]

As explained elsewhere,[5] the compartmental models predominating in HIV epidemiology for a quarter century cannot explicitly include concurrency. To generate heterosexual epidemics, they typically include *mean* lifetime partner counts across the entire population near or above 100,[6–14] and/or that approximately 7% to 26% of the population have lifetime partnerships numbering many hundreds to thousands, [7–9,11–13,15] although no population-based survey contains numbers resembling this. Some authors[16] including Vardavas and Blower[17] justify this by explicitly omitting behavioral data and assuming partnership counts to match observed HIV prevalence. Others have highlighted the behavioral implausibility required by compartmental models[18] and shown that realistic serial monogamy parameters cannot generate observed African HIV epidemics.[19,20] However, Go and Blower accuse network modelers of being "unconstrained by empirical data" and suggest that we simply continue telling all Africans to avoid having many sex partners. This message may be useful to some—for example, women with 5 or more recent partners. However, for those with only 2, but which overlap temporally—and such women are more common and, as network models show, contribute more to HIV spread than common sense suggests—that message will continue to fall on deaf ears.

I implore more researchers to take the effort to understand modeling and closely analyze different models' assumptions. No model is perfect, but in comparison with compartmental models that cannot include all consequences of relational overlap and, instead, assume unrealistic partnership counts—making policy recommendations driven by those assumptions—I am confident that network-based concurrency models will fare well in the evaluation of who is unconstrained by empirical data.

References

1. Go M-H, Blower S. What impact will reducing concurrency have on decreasing the incidence of HIV in heterosexual populations? Sex Transm Dis. 2012; 39:414–415. [PubMed: 22588465]
2. Goodreau SM, Cassels S, Kasprzyk D, et al. Concurrent partnerships, acute infection and HIV epidemic dynamics among young adults in Zimbabwe. AIDS Behav. 2012; 16:312–322. [PubMed: 21190074]
3. Hollingsworth TD, Anderson RM, Fraser C. HIV-1 transmission, by stage of infection. J Infect Dis. 2008; 198:687–693. [PubMed: 18662132]
4. Morris M, Epstein H, Wawer M. Timing is everything: International variations in historical sexual partnership concurrency and HIV prevalence. PLoS One. 2010; 5:e14092. [PubMed: 21124829]
5. Goodreau SM. A decade of modelling research yields considerable evidence for the importance of concurrency: A response to Sawers and Stillwaggon. J Int AIDS Soc. 2011; 14:12. [PubMed: 21406079]
6. Abbas UL, Anderson RM, Mellors JW. Potential impact of antiretroviral therapy on HIV-1 transmission and AIDS mortality in resource-limited settings. J Acquir Immune Defic Syndr. 2006; 41:632–641. [PubMed: 16652038]
7. Abu-Raddad LJ, Longini IM Jr. No HIV stage is dominant in driving the HIV epidemic in sub-Saharan Africa. AIDS. 2008; 22:1055–1061. [PubMed: 18520349]
8. Abu-Raddad LJ, Magaret AS, Celum C, et al. Genital herpes has played a more important role than any other sexually transmitted infection in driving HIV prevalence in Africa. PLoS One. 2008; 3:e2230. [PubMed: 18493617]
9. Baggaley RF, Garnett GP, Ferguson NM. Modelling the impact of antiretroviral use in resource-poor settings. Public Libr Sci. 2006; 3:e124.
10. French K, Riley S, Garnett G. Simulations of the HIV epidemic in sub-Saharan Africa: Sexual transmission versus transmission through unsafe medical injections. Sex Transm Dis. 2006; 33:127–134. [PubMed: 16508523]
11. Garnett GP, Anderson RM. Factors controlling the spread of HIV in heterosexual communities in developing countries: Patterns of mixing between different age and sexual activity classes. Philos Trans R Soc Lond B Biol Sci. 1993; 342:137–159. [PubMed: 7904355]
12. Hallett TB, Singh K, Smith JA, et al. Understanding the impact of male circumcision interventions on the spread of HIV in southern Africa. PLoS One. 2008; 3:e2212. [PubMed: 18493593]
13. Lopman BA, Nyamukapa C, Hallett TB, et al. Role of widows in the heterosexual transmission of HIV in Manicaland, Zimbabwe, 1998–2003. Sex Transm Infect. 2009; 85(suppl 1):i41–i48. [PubMed: 19307340]
14. Walker PT, Hallett TB, White PJ, et al. Interpreting declines in HIV prevalence: Impact of spatial aggregation and migration on expected declines in prevalence. Sex Transm Infect. 2008; 84(suppl 2):ii42–ii48. [PubMed: 18799492]
15. Abu-Raddad LJ, Patnaik P, Kublin JG. Dual infection with HIVand malaria fuels the spread of both diseases in sub-Saharan Africa. Science. 2006; 314:1603–1606. [PubMed: 17158329]
16. Granich RM, Gilks CF, Dye C, et al. Universal voluntary HIV testing with immediate antiretroviral therapy as a strategy for elimination of HIV transmission: A mathematical model. Lancet. 2009; 373:48–57. [PubMed: 19038438]
17. Vardavas R, Blower S. The emergence of HIV transmitted resistance in Botswana: "When will the WHO detection threshold be exceeded? PLoS One. 2007; 2:e152. [PubMed: 17225857]
18. Deuchert E, Brody S. Plausible and implausible parameters for mathematical modeling of nominal heterosexual HIV transmission. Ann Epidemiol. 2007; 17:237–244. [PubMed: 17320790]

19. Delva, W.; Pretorius, C.; Vansteelandt, S., et al. How Explosive Can It Get?. Washington, DC: International AIDS Society; 2012. Serial Monogamy and the Spread of HIV.

20. Leclerc PM, Garenne M. Inconsistencies in age profiles of HIV prevalence: A dynamic model applied to Zambia. Demogr Res. 2007; 16:121–139.