

London's HIV epidemic was driven by clusters of sexual contacts

The rapid growth of the HIV/AIDS epidemic in London during the late 1990s was driven in part by transmission of the AIDS virus within clusters of sexual contacts, with individuals frequently passing the virus to others within months after becoming infected themselves, according to research published in *PLoS Medicine* by Andrew Leigh Brown and colleagues at the University of Edinburgh and London's Chelsea and Westminster Hospital.

These results indicate that growth of the HIV epidemic among men who have sex with men (MSM) in London was not a slow and steady process, but episodic, with multiple clusters of transmission occurring within a few years during the late 1990s, a time when the number of HIV infections in this population doubled.

Information about sexual contact networks can be used for partner notification so that people who may have been unknowingly involved in transmitting the infection can be identified, treated, and advised about disease prevention. Information on network structures can also be used to develop effective community-based prevention strategies.

Traditionally, epidemiologists have reconstructed sexual contact networks by interviewing people with the infection. Interview data has been of limited use in understanding HIV transmission patterns, however, largely because the time over which an infected individual might transmit HIV is long, and the risk of infection from a single sexual contact is low. A more novel way to analyze the spread of HIV is through phylogenetics, which examines the genetic relatedness of viruses obtained from different individuals.

Because genetic data on HIV is routinely obtained in the viral resistance tests used to choose the best HIV medications for an individual patient, Leigh Brown and colleagues were able to compare the sequences of HIV genes from more than 2,000 patients, mainly men who have sex with men (MSM), attending a large London HIV clinic between 1997 and 2003. Of the sequences analyzed, 402 were found to closely match at least one other viral sequence in this group. Further analysis showed that the patients whose viruses showed matches with others formed six clusters of ten or more individuals, as well as many smaller clusters, based on the genetic relatedness of their HIV viruses.

The researchers then used the date when each sample was collected and, taking into account the rates at which changes occur in the viral genes, determined dated phylogenies (essentially family trees of the virus strains) within the clusters. They found that most of the transmissions within each cluster occurred over periods of 3–4 years during the late 1990s, and that at least one in four transmissions between individuals in the large clusters occurred within 6 months following infection of the transmitting partner, suggesting that transmission of the virus during the early stages of HIV infection is likely to be an important driver of the epidemic.

Whether these results apply more generally to MSM at risk for transmitting or acquiring HIV depends on whether the patients in this study are representative of other populations. If additional studies confirm these results more widely, then this quantitative description of HIV transmission patterns should help in the design of strategies to strengthen HIV prevention among MSM.

In an accompanying Perspective, Christopher Pilcher and colleagues at the University of California San Francisco, who were not involved in the study, discuss the implications of this study for public health and clinical practice.

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