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Individual-based models versus deterministic models: which are better for simulating sexually transmitted infections?

Leigh Johnson - actuary and epidemiologist working at the Centre for Infectious Disease Epidemiology and Research, at the University of Cape Town.

Mathematical models are often used to gain theoretical insights into the epidemiology of sexually transmitted infections (STIs) and to inform policy around the prevention and treatment of STIs. Yet these models differ greatly in the assumptions they make, and can sometimes produce vastly different estimates of the likely impact of STI control programmes (1,2). We therefore need to ask ourselves which modelling approaches are most realistic – and how much bias we might be introducing with certain simplifying assumptions.

This article summarises a recent paper that attempted to do this by comparing two broad modelling approaches (3). The first model was a deterministic, frequency-dependent model. The second was an individual-based, network model. Deterministic models calculate expected numbers of outcomes in cohorts of individuals, while individual-based models usually allow for stochastic variation between individuals, both in characteristics and outcomes. Frequency-dependent models assume that an individual's risk of STI acquisition depends on the STI prevalence in the population (without linking individuals to specific partners), while network models link individuals in sexual partnerships, and assume that the individual's risk of STI acquisition depends only on whether the partner has the STI of interest. The frequency-dependent assumption is a simplifying assumption, and the objective of this study was therefore not to assess which approach is more realistic (we already know network models are more realistic), but rather to quantify how much bias might arise due to the frequency-dependent assumption.

We did this by standardizing the two models so that they were as similar as possible in their assumptions (which were based on South African data sources) about demography and sexual behaviour. We then simulated a range of STIs, again making sure that STI assumptions were the same in the two models as far as possible, but allowing the STI transmission probability parameters and assumptions about the course of untreated STIs to vary in the process of fitting the models to South African STI prevalence data. Although the two models produced similar estimates of STI prevalence after completing this calibration exercise, the transmission parameters and natural history parameters required in order to achieve this consistency with observed prevalence levels were quite different. The frequency-dependent model generally estimated much lower probabilities of transmission per sex act and also estimated higher levels of immunity. This suggests that inferences about STI transmission and natural history could be substantially biased if they are based on frequency-dependent models.

We also compared the two models in terms of the predicted impact of hypothetical changes in sexual behaviour. Reductions in commercial sex and concurrent sexual partnerships were estimated to have a dramatically greater impact in the network model than in the frequency-dependent model. However, reductions in unprotected sex in spousal relationships were predicted to have a greater impact in the frequency-dependent model than in the network model. Overall, the results suggest that frequency-dependent models under-estimate the importance of behaviours that increase the connections between individuals (commercial sex and concurrent partnerships) in sustaining STI transmission, while over-estimating the importance of behaviours that intensify the transmission risk between connected individuals (e.g. unprotected sex in spousal relationships). It was also found that the extent of the bias differed between STIs, with the bias being relatively modest for long-term chronic STIs (e.g. HIV and herpes) but substantial for short-term curable STIs (e.g. gonorrhoea and syphilis).

Overall, the results suggest that frequency-dependent models need to be treated with caution, particularly when assessing the potential impact of changes in sexual behaviour, and especially when considering short-term curable STIs. Network models are much better suited to answering questions about the dynamics of STIs, particularly in individuals with high numbers of sexual partners. However, these results are specific to the South African context and are specific to a particular set of behavioural assumptions. The generalizability of these results needs to be explored with other models, in other settings.

It is also important to note that although the majority of deterministic STI models are frequency-dependent, and although the majority of individual-based STI models are network models, there are exceptions. These results should therefore

not be interpreted as showing that individual-based models are always better than deterministic models. However, the deterministic models that do link individuals in partnerships (so-called 'pair models') generally do not allow for concurrency, which is a serious limitation. The vast majority of deterministic models are therefore not well suited to understanding the sexual dynamics that drive and sustain STIs in human populations.

Leigh Johnson *- actuary and epidemiologist working at the Centre for Infectious Disease Epidemiology and Research, at the University of Cape Town. His interests are in mathematical modelling of HIV and other STIs, as well as in monitoring and evaluation of HIV prevention and treatment programmes, and assessing demographic impacts of HIV.* *Leigh.Johnson@uct.ac.za*

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