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Does intimate partner violence contribute to HIV incidence?

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Intimate partner violence (IPV) is a worldwide epidemic, not delimited by geographic or socioeconomic boundaries (1, 2). The size, drivers, and dynamics of this epidemic are uncertain, but there is no denying that IPV occurs on a frightening scale in sub-Saharan Africa. In the past, IPV was called domestic violence. The more precise terminology recognises that IPV is different from other forms of violence in the home (for instance, against children or elders). It also recognises that violence occurs in all kinds of intimate partnerships, not only those that are marital or cohabiting.

Physical and sexual violence are the most well-studied forms of IPV from an epidemiological point of view. Both of these have serious implications for other aspects of physical and mental health. In particular, women who have experienced IPV are more likely to be HIV-positive. This has been observed and replicated in enough cross-sectional and cohort studies (3, 4) for us to have a reasonably good idea of the strength of the association (that is, the unadjusted or bivariate association). However, it is one thing to observe that IPV is correlated with HIV, and even that IPV is a *predictor* of future HIV infection, but quite another thing to understand the underlying reasons.

Looking for an explanation

In a modelling study (5), Leigh Johnson and I investigated some closely related questions about HIV and IPV in South Africa. What are the plausible reasons for the observed association between IPV and HIV? Does IPV cause HIV infection? If we can reduce IPV, is it realistic to expect a reduction in HIV incidence? This project was a branch of a much more extensive programme seeking to model the social and structural drivers of HIV and test out intervention strategies (6).

An agent-based model (ABM), also called an individual-based model, simulates random interactions within a varied population of individuals. ABMs are well-suited to investigating the social drivers of HIV. They are more accurate than some other modelling approaches when assessing the role of high-risk population subgroups, and their risky behaviours, in the spread of sexually transmitted infections (7). While ABMs can be challenging to build and run, they have the advantage of being very intuitive because they mimic the interpersonal networking processes that occur in real life. It is also easy to imitate a cross-sectional or a cohort study, and extract data from the simulations that can be compared to findings from those studies.

We used a pre-existing model (8) that incorporates the natural history of HIV in South Africa and simulates the virus within a representative population of individuals. The individuals in the model age with time and engage in behaviours like forming and dissolving intimate partnerships, marrying, having sex with and without condoms, bearing children, having commercial sex, and taking concurrent partners.

In the first stage of the study, we augmented that list of behaviours by including the perpetration of violence by men against their female intimate partners. This involved setting the rates at which different kinds of relationships become violent and allowing the rates to vary according to the individuals involved. Parameters were adjusted until the model predicted a prevalence of IPV that was consistent with the best available statistical estimates. At this stage, IPV was not allowed to affect the dynamics of the network – it was like a dye added to a water system, which traces the flow but does not interact with the system.

Discovering some confounding factors

At this first stage, it was surprising to find that IPV was a correlate and a predictor of HIV. With a baseline assumption that men's violent tendencies were distributed independently of their propensity for risky sexual behaviours (namely, frequent and concurrent partnering and engagement in sex work) the IPV-HIV association was visible but not quite as strong as observed in the real world. We then moved away from the assumption that violent tendencies are distributed independently of risky sexual behaviours because we know with some confidence that high-risk men are more likely to be perpetrators of violence (see (5), Table A1). Building this assumption into the model increased the strength of the association between HIV and IPV so that it matched real-world observations.

The insight gained from this result is that the empirical situation might be explained entirely by two sources of confounding. Firstly, some women are more exposed to both IPV and HIV simply as a result of having more partners. Secondly, men who are prone to sexual risk-taking are disproportionately prone to violence. It follows from

their risk-taking that those men are more likely to be HIV-positive, and it makes sense that we find elevated HIV incidence and prevalence in survivors of IPV.

The end result is that it is *not necessarily* true that IPV has any causal relationship with HIV. It is worth emphasising that this result is contrary to what we expected at the outset because it is quite widely-believed that there are causal pathways leading from IPV to HIV infections. The kinds of confounding that we discovered are difficult to detect in observational studies, because it is necessary to collect data on couples, but there is at least one study (9) that did so and reached a similar conclusion.

Evaluating causal mechanisms

In the second stage of the study, we modelled some hypothesised causal pathways separately and in combination, to see if they might yield another explanation for the association between IPV and HIV. At this stage, IPV was allowed to interact with the network.
Based on our literature review, there is some evidence that IPV disempowers women to the extent that they cannot negotiate about condoms with their partners. It is also likely that IPV disrupts and breaks up relationships, discourages marriage, and (possibly) encourages concurrent partnering. There is also evidence that HIV-positive women in violent relationships are less adherent to antiretroviral therapy, which could affect perpetrators and cause some feedback effects.

The behavioural changes (in condom use, break-up rates and treatment adherence) that were assumed to follow from IPV had a negligible effect on HIV outcomes in the model. This was consistent when we imitated cohort and cross-sectional studies, set up counterfactuals (what would have happened if there were never any IPV), and simulated interventions (what would happen if IPV were suddenly halved).

The result is that while it *may be* true that IPV has a causal relationship with HIV, the effect size is probably tiny. It is therefore unlikely that IPV prevention strategies would reduce HIV incidence, even if they succeeded in reducing IPV. There are some putative causal mechanisms that we did not simulate; we mention this limitation in the article (5), together with some caveats that are typical of agent-based modelling.

Considering the implications

There are some noteworthy trends in HIV control: testing and diagnosis, treatment as prevention, pre-exposure prophylaxis, and behaviour change for primary prevention. On the other hand, the science behind primary prevention of IPV still has a long way to go, despite some promising developments in both theory and practice (10).

Some of the largest randomised controlled trials on IPV prevention have derived momentum from the belief that therein lies a way to prevent HIV. The message that we hope to convey is that IPV is an epidemic and a human rights issue worthy of more attention, independently of its perceived role as an antecedent risk factor for HIV.

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