The relationship between intimate partner violence and HIV: A model-based evaluation

Simon W. Rigby[*], Leigh F. Johnson

Centre for Infectious Disease Epidemiology and Research, University of Cape Town, Cape Town, South Africa

A B S T R A C T

Background: Many studies have shown that women who have experienced intimate partner violence (IPV) are at a greater risk of HIV, but the factors accounting for this association are unclear, and trials of interventions to reduce IPV have not consistently reduced HIV incidence.

Methods: This study uses an agent-based model, calibrated to South African data sources, to evaluate hypotheses about likely causal pathways linking IPV, HIV, and other confounding factors. Assumptions about associations between IPV and HIV risk behaviours were based on reviews of international literature.

Findings: There is an association between past IPV experience and HIV incidence even when no causal effects are assumed (IRR 1.28, 95% CI 1.23–1.34), because women with a propensity for multiple partners are more likely to have ever been in a relationship with a violent partner. If, in addition, men with a propensity for concurrent relationships are more likely to perpetrate IPV, the IRR increases to 1.42 (95% CI 1.36–1.48), consistent with empirical IRR estimates. Alternative scenarios in which experience of IPV is assumed to cause changes in women's sexual behaviour have little effect on the IRR. An intervention that reduces IPV by 50% could be expected to reduce HIV incidence by at most 1.3%.

Interpretation: Much of the observed association between IPV and HIV is likely to be due to confounding behavioural factors. Although interventions to reduce IPV are important, these interventions alone are unlikely to have a substantial impact on HIV incidence.

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1. Introduction

Intimate partner violence (IPV) is highly prevalent in Southern Africa, and is a major public health and human rights concern. It is estimated that 30% of women in Southern Africa have experienced physical or sexual IPV in their lifetimes (Devries et al., 2013). Southern Africa is also home to 40% of the global population living with HIV, and women are far more likely than men to be infected (Dellar, Dlamini, & Abdool Karim, 2015).

Cross-sectional studies from sub-Saharan Africa have found that past experience of IPV is associated with women's positive HIV status (Dude, 2011; Dunkle et al., 2004; Durevall & Lindskog, 2015a; Kayibanda, Bitera, & Alary, 2012; Shi,

* Corresponding author. Centre for Infectious Disease Epidemiology and Research, Faculty of Health Sciences, University of Cape Town, Anzio Road, Observatory, 7925, South Africa.
E-mail address: rgbsim001@myuct.ac.za (S.W. Rigby).

Abbreviations

CI   Confidence interval
DHS   Demographic and Health Survey
IPV   Intimate partner violence
IQR   Interquartile range
IRR   Incidence rate ratio
OR   Odds ratio
PAF   Population attributable fraction

Kouyoumdjian, & Dushoff, 2013). Cohort studies in South Africa (Jewkes, Dunkle, Nduna, & Shai, 2010) and Uganda (Kouyoumdjian et al., 2013) have also found that past experience of IPV is associated with incident HIV in women.

Consequently, it has been suggested that IPV prevention is a worthwhile strategy for reducing HIV (Abdool Karim & Baxter, 2016; Mathews et al., 2016; UNAIDS, 2011). Several trials in South Africa (Jewkes et al., 2008; Pettifor et al., 2016; Pronyk et al., 2006) and Uganda (Wagman et al., 2015) have significantly reduced IPV, but their findings are inconclusive about the effect on HIV incidence.

Various explanations for the association between IPV and HIV are considered to be plausible. These are summarised by a causal diagram in Fig. 1. Firstly, couples in violent relationships are less likely to use condoms consistently (Were et al., 2011) which may increase the transmission probability of HIV in those relationships. Secondly, it is plausible that women experiencing IPV have a reduced rate of marriage, and an increased rate of relationship dissolution, which may increase their HIV risk. Thirdly, women exposed to IPV may tend to acquire more concurrent partners, perhaps as a result of the harmful psychological effects of IPV (Dunkle & Decker, 2013).

Alternatively, the association between IPV and HIV could be explained by confounding factors. Firstly, women with a high number of lifetime sexual partners are more likely to have experienced violence at some point, because of their exposure to a greater number of potentially violent men. This would explain some of the increased HIV prevalence in survivors of IPV, as well as the association with incident HIV if patterns of multiple partnering persist. Secondly, the association is likely exacerbated by the fact that men with high sexual risk behaviour (who have high chances of being HIV positive) are more likely than other men to be violent (Decker et al., 2009; Jewkes et al., 2006). This association between men's HIV status and their perpetrating IPV would imply that transmission occurs disproportionately more in violent relationships. Thirdly, when sexual
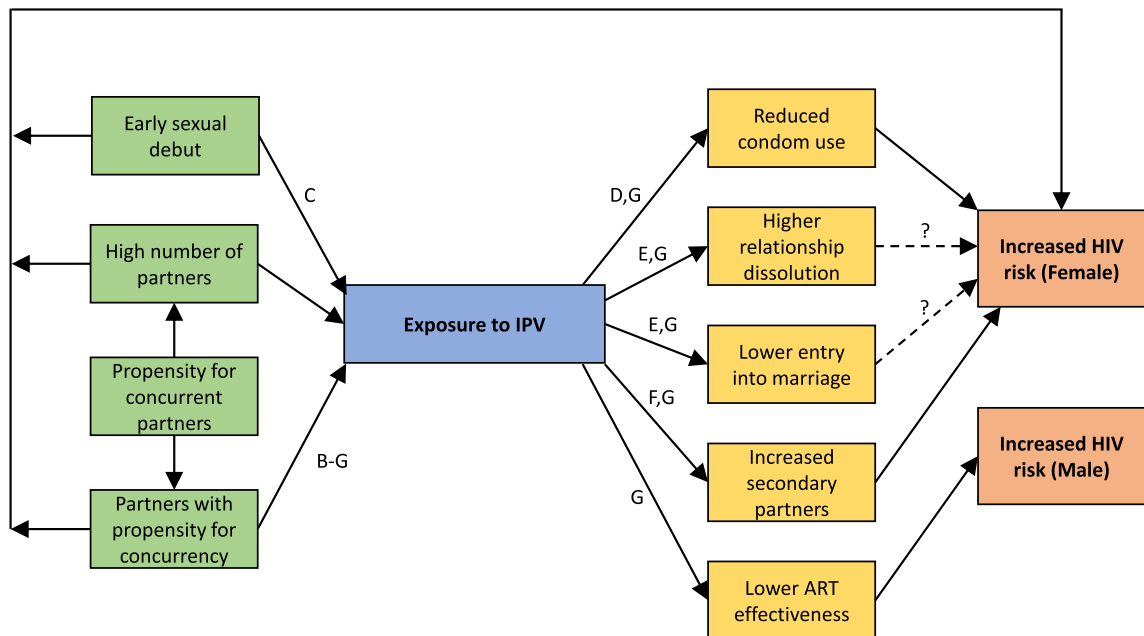

Fig. 1. Causal diagram for the relationship between IPV and HIV. Boxes indicate variables that are potential confounders between IPV and HIV (green) or potential mediators between IPV and HIV (gold). Arrows indicate causal pathways between the variables. The simulated scenarios are denoted by letters A–G, and next to each arrow are listed the scenarios in which that causal pathway applies. Where no letter appears next to a pathway, that pathway applies in all scenarios.

debut occurs at younger ages there is a high probability that it is forced (García-Moreno, Jansen, Ellsberg, Heise, & Watts, 2005), and early sexual debut is associated with HIV infection (Pettifor, Van der Straten, Dunbar, Shiboski, & Padian, 2004).

IPV perpetration could also affect men's HIV risk. HIV-positive women exposed to violence appear to have lower rates of antiretroviral therapy use and adherence, and lower levels of viral suppression when on treatment (Hatcher, Smout, Turan, Christofides, & Stöckl, 2015), which probably increases their male partners' risk of acquiring HIV.

This study uses an agent-based model (ABM) to improve understanding of the association between IPV and HIV, and evaluate the potential impact of IPV prevention on HIV incidence. More precisely, this study aims to answer two questions. The first is: Which causal pathways or confounders play an important role in the relationship between IPV and HIV? The second is: Can interventions prevent HIV incidence by reducing IPV? To answer the first question, we determine by simulation whether confounding factors can explain some or all of the observed association between IPV and HIV. An ABM cannot easily prove or disprove the existence of a causal relationship, but by simulating an assumed causal effect, it may be possible to disprove that it plays an important role. To answer the second question, we simulate and evaluate two intervention scenarios based on the IMAGE study (Pronyk et al., 2006) and the SASA! study (Abramsky et al., 2014). The first of these trials had limited power and did not detect an effect on HIV incidence, while the second did not measure HIV outcomes.

Mathematical modelling is an important tool for analysing the aetiologic pathways and mechanisms that give rise to the distribution of disease in a population (El-Sayed, Scarborough, Seemann, & Galea, 2012; Garnett, 2002). ABMs simulate events in a population of individual agents, at each time step, based on interactions with other agents and a governing set of 'update rules'. ABMs are especially useful when social network structures play a role (El-Sayed et al., 2012; Marshall & Galea, 2014). Through introducing hypothesised causal pathways and confounders, this study demonstrates how ABMs can be used to assess the likely significance of different causal relationships and confounding factors in explaining observed associations. This follows a similar approach to previous studies that have used ABMs to assess the extent to which confounding factors may explain observed associations between exposures and HIV outcomes (Boily & Anderson, 1996; Johnson et al., 2014).

2. Methods

2.1. Model overview

We built on previous research by extending an existing model of HIV in South Africa (Johnson & Geffen, 2016) to include IPV against women. The model, developed in C++, simulates a network of heterosexual relationships over time (beginning in 1985) within a population of individuals (initially 20 000) intended to represent the demographic profile of South Africa.

The model assumes that there are two sexual behaviour risk groups. 'Low-risk' individuals have only monogamous relationships and no engagement in commercial sex. 'High-risk' individuals have a propensity for engaging in concurrent partnerships (up to a maximum of two partners at a time) and may become sex workers (if female) or clients of sex workers (if male).

The model considers three relationship types: marital/cohabiting relationships, short-term relationships, and sex worker-client contacts. A pair can only enter into marriage if they are both unmarried and in a short-term relationship. A degree of assortative selection is assumed, whereby members of the same risk group are more likely to pair than members of different risk groups.

IPV (defined here as sexual or physical violence) is assigned randomly to some marriages and short-term relationships at each time step. It is assumed that IPV can only occur in a partnership if the man has a 'predisposition' for violence. Violent predispositions are an attribute assigned to men with assumed probabilities depending on whether the man is high- or low-risk. These probabilities, given in Table 1, are determined such that the model predicts a 35% prevalence of lifetime IPV exposure among women aged 15–49 (World Health Organisation, 2013).

The random assignment of IPV to partnerships is determined by an incidence rate parameter. That is, at each time step, each partnership that is not yet violent, but in which the man is predisposed to violence, becomes violent with probability $1 - \exp(-\lambda/48)$, where λ is the annual rate applying to that partnership at that time, and 48 is the number of times steps per year. The rate is assumed to differ according to whether it is a short-term relationship, a new marriage (<2 years duration), or an established marriage (>2 years duration). The parameters for modifying the incidence rate in this way are given in Table 1. The rate is also multiplied by the woman's 'susceptibility factor', which is intended to account for additional sources of heterogeneity (such as self-esteem, mental health, childhood abuse, personality, and social network influences). The susceptibility factor is a number between zero and one, randomly assigned to women from a Beta distribution with a mean of 0.4 and a variance of 0.12. It is assumed that once IPV begins in a partnership, it persists for the duration of that partnership.

2.2. Model scenarios

We consider a number of scenarios, each intended to explore one or more possible causal pathways between, or confounders to, the relationship between IPV and HIV. Scenarios A–C include only confounders to the relationship, while Scenarios D–G include some confounders and also some causal pathways. The scenarios are summarised by the causal diagram in Fig. 1.

In Scenario A, no causal pathways are assumed, but the model implicitly takes into account confounding due to women with greater numbers of partners being more exposed to both IPV and HIV.

Table 1
Main parameters and parameter values used in the IPV-HIV model.

Parameter description	Parameter value	Scenarios where applicable	Data source
Ratio of probability of violent predispositions in high-risk men to that in low-risk men	1.00	A	
	1.70	B–G	(Abrahams, Jewkes, Laubscher, & Hoffman, 2006)
Probability of a violent predisposition, high-risk men	0.3650	A	Fitted to prevalence of lifetime IPV exposure (World
	0.3315	C	Health Organisation, 2013)
	0.4620	B,D–G	
Annual rate of IPV incidence: short-term relationships[a]	0.3300	A–G	
Annual rate of IPV incidence: marriages with <2 years duration[a]	0.4950	A–G	Fitted to duration until onset of IPV in relationships (Peterman, Bleck, & Palermo, 2015)
Annual rate of IPV incidence: marriages with >2 years duration[a]	0.2475	A–G	Half of the above value (Kishor & Johnson, 2004)
Probabilities of forced sexual debut	–	C	See Fig. A1, Appendix
Odds ratio for not using a condom, per-sex-act, in violent relationships vs non-violent	1.80	D,G	(Were et al., 2011)
Reduction in rate of marriage in violent short-term relationships	25%	E,G	
Increase in rate of relationship dissolution in violent relationships/marriages	50%	E,G	(DeMaris, 2000)
Increase in rate of acquiring secondary partners among women experiencing IPV	50%	F,G	
Reduction in level of viral suppression among women on ART, if experiencing IPV	36%	G	(Hatcher et al., 2015)

[a] This is the rate that applies if the male partner has a violent predisposition and if the female susceptibility factor is 1 (the maximum).

Scenario B is the same as Scenario A, but we assume that high-risk men are more likely to be violent.

Scenario C is the same as Scenario B, but we assume that sexual debut is more likely to be forced if it occurs at younger ages.

Scenario D is the same as Scenario B, but we allow for reduced condom use in partnerships that are violent.

Scenario E is the same as Scenario B, but we allow for an increased rate of relationship dissolution and a reduced rate of entry into marriage whenever partnerships are violent.

Scenario F is the same as Scenario B, but we assume that high-risk women acquire secondary partners at a greater rate whenever their primary partnership is violent.

Scenario G combines the assumptions from B and D–F, while also assuming less viral suppression in treated HIV-positive women who are experiencing IPV.

Parameter values used in the various scenarios are summarised in Table 1, together with the main sources on which they are based. A more in-depth discussion regarding the selection of parameter values is included in the Appendix (Section 1).

3. Calculation

We assessed the plausibility of each scenario by measuring the effect of IPV on HIV incidence (a longitudinal effect) and prevalence (a cross-sectional effect), using measures similar to previous observational studies.

For the longitudinal effect, we calculated an HIV incidence rate ratio (IRR) for lifetime IPV exposure, and compared it to empirical estimates of the IRR (Jewkes et al., 2010; Kouyoumdjian et al., 2013). More specifically, in each simulation, the HIV incidence rate in the period 2013–2015 was measured for ever-partnered women who were HIV-negative at the start of the period and aged 15–35 at the end of it. The IRR is the ratio of the incidence rates in the exposed (ever experienced IPV prior to 2013) and unexposed groups.

For the cross-sectional effect, we calculated an odds ratio (OR) for the association between current IPV and HIV in married women, and compared it to empirical estimates of the OR (Durevall & Lindskog, 2015a; Harling, Msisha, & Subramanian, 2010). In each simulation, the OR was calculated based on the HIV status of married women aged 15–49 in 2015, comparing those in violent marriages and non-violent marriages.

We also calculated a population attributable fraction (PAF) representing the cumulative fraction of HIV infections attributable to IPV, over the history of the epidemic (1990–2015), for each of Scenarios D–G, by comparing the scenario to a corresponding counterfactual scenario with no IPV.

3.1. Evaluating interventions

To evaluate the effect of IPV prevention on HIV incidence, we introduce a hypothetical intervention intended to imitate the IMAGE programme (Pronyk et al., 2006). In 2015, half of all men predisposed to violence are assumed to become non-violent, with immediate effect on all their current and future relationships.

Because synergies could make it feasible for interventions to reduce both IPV and men's sexual risk taking, we consider a second hypothetical intervention that imitates the SASA! programme (Abramsky et al., 2014). In 2015, IPV is halved (as before) and, additionally, the rate at which high-risk men acquire concurrent partners is assumed to decrease by 40%.

The interventions were assessed by calculating the proportionate reduction in cumulative HIV infections over ten years, from mid-2015 to mid-2025.

3.2. Statistical analysis

The original model was calibrated to age-specific South African HIV prevalence data. This means that each simulation draws from a set of preselected HIV transmission parameters that give similarly good fits to observed HIV prevalence levels (Johnson & Geffen, 2016). For each scenario, the model was run 100 times, using the 100 best-fitting HIV parameter combinations generated previously. Relevant outputs are summarised by the sample mean and 95% CI, except when they are related to the calibration of the model, in which case the median and IQR are reported.

3.3. Sensitivity analysis

We calculated the IRRs and ORs at various times to determine if the results were sensitive to the year of measurement. We also ran sensitivity analyses on Scenario B to test for robustness (see Appendix, Section 3). These tests included: (1) varying the prevalence of lifetime IPV exposure in women by (a) increasing the rates of IPV incidence and (b) reducing the probabilities that men are violently predisposed, and (2) allowing for different IPV incidence rates according to the age of the perpetrator.

4. Results

In Scenarios A–C, the model fitted South African HIV prevalence quite well (Fig. 2A). In the other scenarios, the deviation in prevalence from scenarios A–C, caused by introducing behavioural effects of IPV, was negligible (see Fig. A5, Appendix, which shows the HIV prevalence over time in each scenario). In all of the scenarios except C, the median age of first experiencing IPV was 22 years, and approximately one third of women who had experienced IPV were victims within the previous 12 months (Fig. 2B).

4.1. The longitudinal association between IPV and HIV incidence

In Scenario A, where no confounders or causal pathways were explicitly assumed, ever-partnered women aged 15–35 were 28% more likely to acquire HIV between 2013 and 2015 if they had previously experienced IPV (IRR 1.28, 95% CI 1.23–1.34) (Fig. 3A). The fact that the association was positive in Scenario A indicates significant confounding by female propensity for multiple partners: women who partner more frequently are more likely to have experienced IPV, and are also more likely to acquire HIV.
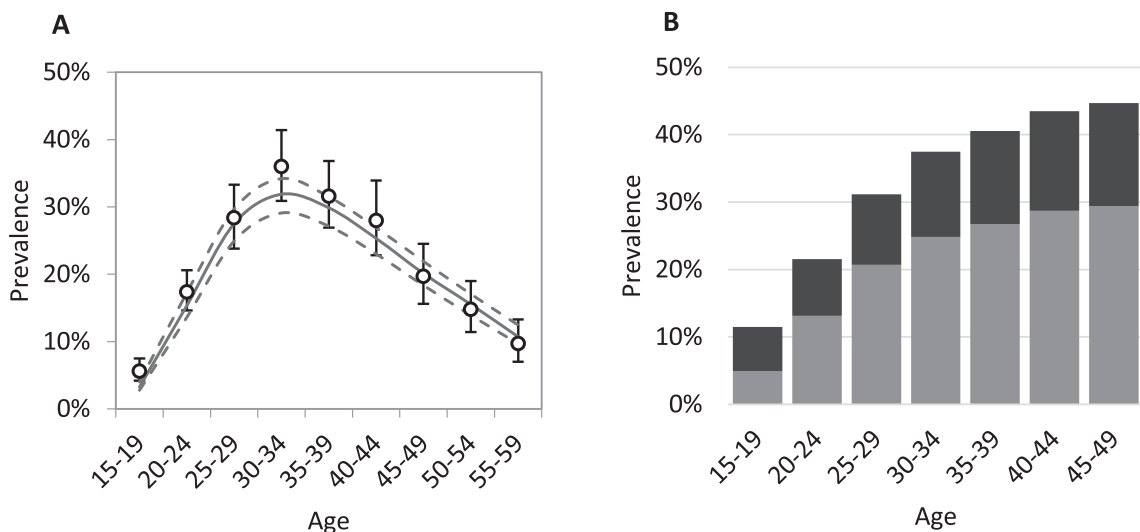

Fig. 2. Prevalence of HIV and IPV by age groups of women, simulated in Scenario B. (A) HIV prevalence among women in 2012 (median and IQR of 100 simulations) is plotted by age and compared to estimates from the 2012 South African National HIV Prevalence, Incidence and Behaviour survey (Shisana et al., 2014). The solid line and dotted lines represent the median and IQR of 100 simulations. Circles and error bars represent the estimate and 95% CIs from the survey. (B) Prevalence of recent (dark grey) and non-recent (light grey) IPV exposure among ever-partnered women in 2015 (mean of 100 simulations) is plotted by age.
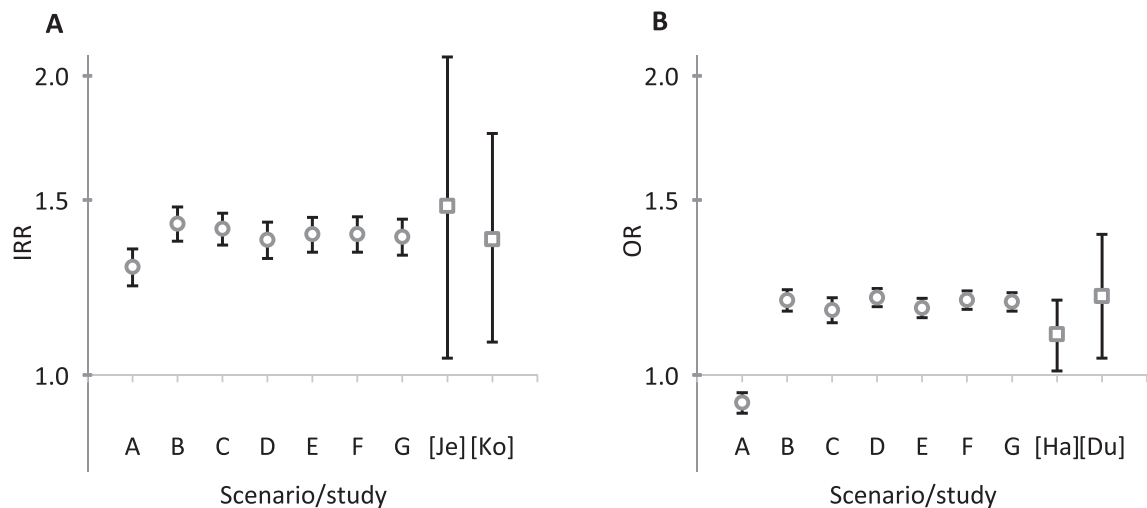
Fig. 3. Two measures of the relative risk of HIV in women exposed to IPV. (A) IRRs for the effect of lifetime IPV exposure on HIV incidence from 2013 to 2015, among ever-partnered women aged 15—35 in 2015, plotted for each simulated scenario and compared to empirical unadjusted IRR estimates. (B) ORs for the association between current IPV and HIV, among married women aged 15—49 in 2015, plotted for each simulated scenario and compared to empirical unadjusted OR estimates. Circles/squares represent the mean of simulated/empirical evidence, respectively, and error bars represent the 95% CI of the mean. The y-axis scale is a logarithmic scale. [Je] = Jewkes et al. (2010); [Ko] = Kouyoumdjian et al. (2013); [Ha] = Harling et al. (2010); [Du] = Durevall and Lindskog (2015a).

In Scenario B, ever-partnered women aged 15—35 were 42% more likely to acquire HIV when previously exposed to IPV (IRR 1.42, 95% CI 1.36—1.48). The IRR increase in Scenario B (relative to A) indicates significant additional confounding due to the association between male concurrency and propensity for IPV perpetration.

The remaining scenarios (C—G) closely resembled B with respect to the association between IPV exposure and HIV incidence (Fig. 3A), suggesting that the additional sources of confounding and hypothesised causal pathways had little effect. The unadjusted IRRs observed in cohort studies by Jewkes et al. (2010) and Kouyoumdjian et al. (2013) are very similar to the IRRs simulated in Scenarios B—G.

4.2. The cross-sectional association between IPV and HIV

Associations between IPV and HIV differed between Scenario A and the other scenarios when considering the cross-sectional effect in married women (Fig. 3B). In Scenario A, married women aged 15—49 had 0.94 (95% CI 0.92—0.96) times the odds of being HIV-positive when they had experienced IPV from their current partner, which is inconsistent with most of the literature. In Scenario B, the OR was 1.19 (95% CI 1.16—1.22), which is very close to the unadjusted OR of 1.20 (95% CI 1.04—1.39) found by Durevall and Lindskog (2015a) in their analysis of sub-Saharan African DHS data. Again, Scenarios C—G were very similar to B in this measure of relative risk.

4.3. Population attributable fractions

Table 2A summarises the PAFs for males, females, and the whole population. In general these were low (at most 2.9% in Scenario G) and in sharp contrast with the PAFs of 11.9% (95% CI 1.4—19.3) and 22% (95% CI 12.5—30.4) reported by Jewkes et al. (2010) and Kouyoumdjian et al. (2013) respectively.

4.4. Evaluation of the interventions

Table 2B summarises the reduction in cumulative HIV incidence attributable to the first intervention (reducing IPV by half in 2015). Even when all of the hypothesised causal pathways were assumed to exist, reducing IPV instantly by half resulted in only a 0.2% (95% CI -1.0%—1.3%) reduction in new HIV infections over ten years. On the other hand, if the intervention was accompanied by a 40% reduction in men's rates of concurrency, there was a 3.4% (95% CI 2.2—4.6) reduction in population cumulative incidence in Scenario B and a 5.5% (95% CI 4.2—6.7) reduction in Scenario G (Table 2C).

4.5. Sensitivity of results

The IPV-HIV associations calculated from the model varied slightly according to the stage of the epidemic (see Fig. A2, Appendix, which shows the IRRs and ORs between 2005 and 2015, and is accompanied by an explanation). However, the associations were not sensitive to the prevalence of lifetime IPV exposure, or the assumption that men's rates of IPV perpetration depend on their age (see Fig. A4, Appendix, which shows the IRRs and ORs in each of the three sensitivity tests).

Table 2
Fractions of HIV infections attributable to IPV or preventable by reducing IPV, according to simulated scenarios.

Scenario	Males	95% CI	Females	95% CI	Combined	95% CI
A Population attributable fraction, 1990—2015						
A-C	0.0%	0.0—0.0	0.0%	0.0—0.0	0.0%	0.0—0.0
D	0.8%	−1.4—2.9	0.5%	−1.7—2.7	0.6%	−1.6—2.8
E	0.7%	−1.3—2.7	1.0%	−0.9—3.0	0.9%	−1.1—2.8
F	1.1%	−0.7—2.8	0.9%	−0.8—2.7	1.0%	−0.8—2.7
G	3.1%	1.1—5.2	2.7%	0.7—4.8	2.9%	0.8—4.9
B Reduction in cumulative HIV incidence, 2015—2025, resulting from a 50% reduction in IPV[a]						
A-C	0.0%	0.0—0.0	0.0%	0.0—0.0	0.0%	0.0—0.0
D	0.0%	−1.4—1.4	−0.5%	−2.0—1.0	−0.2%	−1.6—1.1
E	−0.8%	−2.3—0.6	0.0%	−1.3—1.2	−0.3%	−1.5—0.9
F	−1.2%	−2.5—0.2	−0.2%	−1.5—1.1	−0.6%	−1.8—0.6
G	0.4%	−0.9—1.7	0.0%	−1.3—1.2	0.2%	−1.0—1.3
C Reduction in cumulative HIV incidence, 2015—2025, resulting from a 50% reduction in IPV and a 40% reduction in men's acquisition of concurrent partners						
A-C	2.0%	0.7—3.3	4.4%	3.0—5.7	3.4%	2.2—4.6
D	2.6%	1.1—4.0	4.6%	3.1—6.1	3.8%	2.4—5.2
E	1.6%	0.1—3.1	4.5%	3.1—5.9	3.3%	2.0—4.7
F	2.1%	0.5—3.6	5.2%	4.0—6.3	3.9%	2.7—5.2
G	4.7%	3.4—6.1	6.0%	4.7—7.3	5.5%	4.2—6.7

[a] Due to the stochastic variation inherent in the model, it is possible for the intervention to have a negative impact on HIV incidence in individual simulations (even though we do not expect the true effect to be negative on average). If the confidence interval includes zero, this implies that the mean is not statistically significantly different from zero.

5. Discussion

This analysis suggests that the most plausible explanation for the observed association between IPV and HIV is that men with a propensity for multiple or concurrent partners are substantially more likely to be violent (although exactly how much more likely is subject to some uncertainty). Causal pathways between IPV and HIV, if they exist, make very little difference to the prevalence and incidence of HIV in survivors of IPV.

These findings are consistent with Durevall and Lindskog's (2015b) analysis of married couples data in sub-Saharan Africa: after conditioning women's HIV status on the HIV status of their husbands, they found that IPV experience had little effect on women's risk of HIV infection. The findings may also explain why several trials that reduced IPV failed to achieve significant reductions in HIV incidence (Jewkes et al., 2008; Pettifor et al., 2016; Pronyk et al., 2006), even though observational evidence suggests a strong association between IPV and HIV.

Even with strong causal assumptions in the model, a hypothetical IPV intervention made little impact on projected HIV incidence in the population. This suggests that very few HIV infections would be averted simply by reducing IPV, unless there is a corresponding reduction in the sexual risk behaviours that are concentrated in perpetrators of violence, and to some extent in victims too.

This study resolves several deficiencies in the methodological approaches previously used to investigate the role of IPV in HIV. Traditional epidemiological approaches focus on understanding individual-level risk factors, and do not account for the role of social networks, and interrelatedness between exposures, in determining a person's risk of disease (El-Sayed et al., 2012). Analyses of IPV and HIV, based on this kind of approach, have not adequately controlled for confounding between IPV perpetration and men's sexual concurrency, or between women's frequency of partnering and their experience of IPV (Castor, Cook, Leclerc-Madlala, & Shelton, 2010). Our method advances the field: using an ABM, we showed that confounding with men's concurrency combined with heterogeneous (assortative) sexual mixing is both necessary and sufficient to reproduce the empirical association between IPV and HIV.

The model presented in this study — like every model — does not reflect all aspects of reality. For example, studies have consistently found that violence is more prevalent in cohabiting, non-marital relationships than either formal marriages or short-term relationships (Abramsky et al., 2011; Gass, Stein, Williams, & Seedat, 2011). It is possible that the model could be improved if cohabitation and formal marriage were considered separately. Another limitation is the binary distinction made between men who can be violent and men who cannot. In reality, some men would have a tendency to be more frequently violent, and more severely violent, than others.

At least two other possible causal pathways between HIV and IPV could have been modelled. Firstly, reverse causality could occur if women's disclosure of a positive HIV status triggers violent reactions. This would not explain why past IPV is associated with future HIV infection in women (Jewkes et al., 2010; Kouyoumdjian et al., 2013), although it would probably increase the cross-sectional IPV-HIV association. Secondly, some reviewers (Campbell et al., 2008) and modellers (Watts et al., 2010) have speculated that, due to physical trauma, there is a higher probability of HIV transmission when sex is forced, relative to consensual sex. Had this scenario been tested, one would expect the outcomes to resemble Scenario D (lower condom use). In any case, observed associations with HIV persist when only physical violence is considered, and hence are not generally dependent on there being instances of forced sex (Dunkle & Decker, 2013).

There are a few limitations to the generalisability of these findings, which future research could improve on. Firstly, the assumed patterns of sexual behaviour in South Africa may have had some influence on the results, and the results might not be generalisable to settings with very different sexual behaviour patterns. In particular, the allocation of men and women to sexual risk groups and the assumed assortativeness of sexual mixing may have been influential in the results (Boily & Anderson, 1996). Given the large number of parameters in the model, and the limited HIV prevalence data used in calibration, identifiability is a concern. Other HIV models, applied to other settings, would be well-placed to compare our findings. Secondly, because of the wide confidence intervals around the IRRs and ORs in the studies being compared, and the fact that they were based on different populations at different times, it was not possible to calibrate the model to these observations with any degree of precision. However, despite these limitations in the available data, the model has provided valuable qualitative insights into the role of IPV in HIV epidemiology.

6. Conclusions

Although this paper refutes the idea that eliminating IPV will significantly reduce HIV infections, it does not invalidate the need for IPV interventions. Preventing IPV is important in its own right, and it is well-known that there are other serious mental and physical health consequences of violence (Campbell, 2002). Researchers, donors, and policymakers should not neglect the prevention of IPV as a distinct priority in global health (Mullan, 2014). Furthermore, because of the concentration of sexual risk behaviours in perpetrators and victims of IPV, it may be efficient to implement joint interventions.

The association between men's sexual risk-taking and violence perpetration may emerge from social networks where harmful social norms are influential (Neville, 2015). It appears that these behaviours have a common source in harmful gender norms and notions of "hegemonic masculinity" (Kenyon & Buyze, 2015; Townsend et al., 2011). Reducing risk behaviours in high-risk men, including multiple partnering and concurrency, is a potentially viable HIV prevention strategy, as exemplified by the second simulated intervention. Structural interventions that focus on men's normative behaviours, like SASA! in Uganda (Abramsky et al., 2014), do have the potential to reduce both IPV and HIV in severely affected regions.

Competing interests

The authors have no conflicts of interest to declare.

Funding

Funded by the South African National AIDS Council.

Ethics

The study did not require approval from an ethics committee, since no human subjects were involved.

Acknowledgements

Appendix

1. Choice of parameter values

We comprehensively searched databases (PubMed and Google Scholar) and reference lists to find sources on which to base our parameters. Where there was uncertainty we tended to overestimate, rather than underestimate, the extent of the causal relationship between intimate partner violence (IPV) and HIV.

1.1 Prevalence of lifetime IPV exposure

We assumed that the prevalence of lifetime IPV exposure was 35%. This number is approximately equal to the World Health Organisation[1] estimate for Africa (36.6%, 95% CI 32.7–40.5), and slightly exceeds the estimate produced by Devries et al.[2] for Southern Africa (29.7%, 95% CI 24.3–35.0).

The margin of 5% on the second estimate[2] is justified, firstly, because surveys are more likely to underestimate violence than to overestimate it.[2,3] Secondly, surveys consistently find that prevalence of IPV exposure in the past year (recent prevalence) is around half the prevalence of lifetime IPV.[3] This finding could not be reproduced in the model, and it is plausible that recall bias causes survey respondents to under-report violence that occurred more than a year ago (thus warranting an upward adjustment in the assumed prevalence of lifetime IPV).

We did not directly specify the prevalence of lifetime IPV, but rather fitted it by manipulating the assumed proportions of men with violent predispositions and the rates of IPV incidence (described below). We accepted the fit if the median prevalence of lifetime IPV was within 0.25 percentage points of the target (35.0%).

The modelled prevalence of lifetime IPV was calculated in 2024, which is the earliest date at which all women aged 15–49 had complete sexual histories (because the simulations begin in 1985 and the earliest possible age of sexual debut is assumed to be ten years).

1.2 Ratio of probability of violent predispositions in high-risk men to that in low-risk men

The few studies that have measured the association between men's sexual risk-taking and perpetration of IPV (Table A1) have reported odds ratios (ORs) as a measure of association. For ease of intuition, we decided to parameterise the association between men's sexual risk groups and violent predispositions in terms of a risk ratio rather than an OR. In Scenario A the risk ratio is assumed to be 1.0 (the null assumption) and in Scenarios B–G, the risk ratio is assumed to be 1.7. This is equivalent to an OR of 2.30 in Scenarios B, D–G and an OR of 2.05 in Scenario C (since in Scenario C there was a slightly lower overall probability of violent predispositions).

Table A1
Summary of studies measuring the correlation between men's sexual risk behaviours and perpetration of IPV.

Study	Country	N	Risk factor	Risk factor prevalence (%)	Outcome	Bivariate OR (95% CI)
Martin et al.[4]	India	6632	Extramarital sex ever	4	Physical IPV only	2.7 (1.8–4.2)
					Sexual IPV only	4.3 (3.0–6.2)
					Both physical and sexual IPV	6.2 (4.0–9.7)
Abrahams et al.[5]	South Africa	1378	>1 current partner	15.7	Past 10 years physical IPV	3.0 (2.2–4.1)
Hembling and Andrinopoulos[6]	Guatemala	4733	Past-year infidelity	5.5	Past-year physical/sexual IPV	3.0 (1.7–5.3)
			Lifetime sex worker patronage	26.3	Lifetime physical/sexual IPV	1.9 (1.6–2.4)
Decker et al.[7]	United States	1585	Lifetime history of concurrent partnerships	48.1	Lifetime physical/sexual IPV	3.9 (3.1–4.9)[a]
Raj et al.[8]	United States	235	Sex with other women, past 3 months	43.1	Past-year physical/sexual IPV	2.0 (1.2–9.3)
Dunkle et al.[9]	South Africa	1275	Concurrent or once-off partner ever	22.9	Physical IPV only (lifetime)	1.5 (1.1–2.1)
				3.6	Sexual IPV only (lifetime)	4.0 (1.6–10.1)
				5.3	Both physical and sexual IPV only (lifetime)	10.6 (3.1–36.1)
Jewkes et al.[10]	South Africa	1370	Transactional sex with a non-primary partner ever	16.6	Ever raped a partner	2.1 (1.3–3.3)[a]
Mthembu et al.[11]	South Africa	975	Casual sexual partner currently	50.9	Lifetime physical IPV	1.67 (1.28–2.17)

[a] Multivariate models; OR = odds ratio.

1.3 Rates of IPV incidence by relationship duration

Very few studies have measured the time until onset of IPV, either in terms of age, time since sexual debut, or time since the beginning of the union. This information is, however, important for determining the incidence rate of IPV in different types of partnerships.

In a study by Kishor and Johnson,[12] three out of four DHSs showed that, across all marital durations, IPV was at least twice as likely to have begun in the first two years of marriage than in any subsequent two-year interval. To reflect this, we assumed that the incidence rate of IPV halves after two years of marriage.

We were not aware of any data on the time until onset of IPV (or the association between IPV and relationship duration) in short-term (non-cohabiting) relationships. The prevalence of IPV in short-term relationships was also difficult to ascertain from the literature, although evidence suggests it is lower than the prevalence in cohabiting/married couples.[13] We thus assumed that the rate of incidence in short-term relationships was two-thirds of the rate in new marriages.

We set the incidence rate parameter values by making a judgement with reference to the reasonableness of the model as a whole, including the proportion of men with violent predispositions (since this was inversely related to the incidence rates of IPV when the prevalence of IPV was fixed).

In partnerships where the man has a violent predisposition and the woman's susceptibility factor is 1 (the maximum), the incidence rates were assumed to be 0.3300 (short-term relationships), 0.4950 (marriages of less than two years duration) and 0.2475 (marriages of greater than two years duration).

We then checked the model's predicted time until onset of IPV against results from a study by Peterman et al.[14] The study showed that, in sub-Saharan Africa, IPV began in the first year of union (marriage or cohabitation) for 37% of violent unions, and in the first three years for 67% of violent unions. In the model, these numbers were 34% and 66% respectively.

In another check, the median time between sexual debut and onset of IPV was 4.6 years (according to the model) which approximately matches the median of 5 years reported by Dunkle et al.[15] in a South African study.

1.4 Assumptions about the probability of forced female sexual debut

Studies have estimated very high probabilities that female sexual debut is forced, especially at younger ages.[3,15–17] The purpose of Scenario C is to determine whether the IPV-HIV association changes when there is confounding by age of sexual debut (which observational studies rarely control for). In this scenario, we assumed a probability of forced female sexual debut for each integer age from 10 on (Fig. A1); at sexual debut, this assumption replaces the usual process of assigning IPV.

When we ran this scenario, we reduced the fraction of men with violent predispositions (from 0.4620 to 0.3315 for high-risk men − and from 0.2718 to 0.1950 for low-risk men) so that the model predicted a 35% prevalence of lifetime IPV exposure (consistent with Scenarios A, B and D−G).
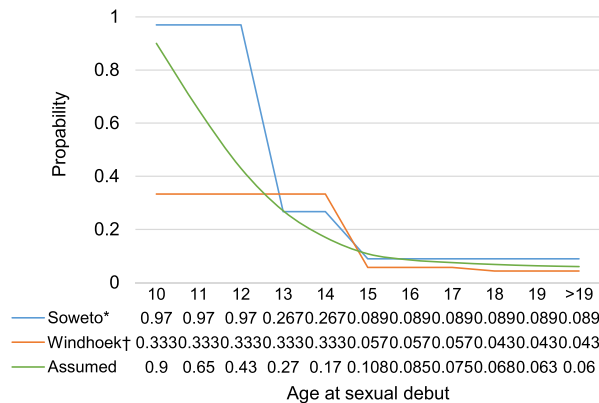

	10	11	12	13	14	15	16	17	18	19	>19
Soweto*	0.97	0.97	0.97	0.267	0.267	0.089	0.089	0.089	0.089	0.089	0.089
Windhoek†	0.333	0.333	0.333	0.333	0.333	0.057	0.057	0.057	0.043	0.043	0.043
Assumed	0.9	0.65	0.43	0.27	0.17	0.108	0.085	0.075	0.068	0.063	0.06

Age at sexual debut

Fig. A1. Probabilities of forced female sexual debut. *Dunkle et al.[15]; †García-Moreno et al.[3].

We assumed that sexual debut could only be forced if the partner had a violent predisposition, so it was necessary to convert the assumed probabilities (which were unconditional on the partner) to probabilities that were conditioned on the partner's having a violent predisposition.

To keep the mathematics relatively simple, we did not adjust the probabilities of forced sexual debut for variation in women's susceptibility. The following paragraphs explain the calculations for converting the assumed probabilities to conditional probabilities:

An initial run of a full set of 100 simulations was used to estimate the probabilities $\pi_{H,x}$ that female sexual debut at age x occurs with a high-risk man, and the probabilities $\pi_{L,x} = 1 - \pi_{H,x}$ that female sexual debut at age x occurs with a low-risk man, according the model.

The following equation, which we could then easily evaluate, gives the probability that sexual debut at age x is with a violently predisposed partner:

$$Pr[\text{1st sexual partner has violent predisp.}] = \pi_{H,x}*\text{Proportion of high-risk men who have violent predisp.}$$
$$+ \pi_{L,x}*\text{Proportion of low-risk men who have violent predisp}$$

We let θ_x be the assumed probability that sexual debut at age x is forced, then in accordance with probability theory:

$$\theta_x = Pr[\text{forced sexual debut}] = Pr[\text{1st sexual partner has violent predisp.}] * Pr[\text{forced sex} \mid \text{1st sexual partner has violent predisp.}]$$

The following equation then gives the probability of forced sexual debut given that the male partner has a violent predisposition (these are the values we needed to input into the model):

$$Pr[\text{forced sex} \mid \text{first sexual partner has violent predisp.}] = \theta_x / Pr[\text{1st sexual partner has violent predisp.}]$$

The above equation holds provided $\theta_x \leq Pr[1st\ sexual\ partner\ has\ violent\ predisp.]$. At younger ages, when the assumed probability of forced sex is high, it is possible that $\theta_x > Pr[1st\ sexual\ partner\ has\ violent\ predisp.]$. In this case, we assumed that sexual debut at age x was always forced whenever the encounter was with a man who had a violent predisposition.

1.5 Effect of IPV on condom usage

It is generally accepted in the literature that women experiencing violence have limited ability to negotiate condom use.[18–26]

Kacanek et al.[27] analysed the effect of IPV on adherence to condom use in a (secondary) analysis of a randomised controlled trial of 4505 women aged 18–49 in Johannesburg, Durban and Harare. They found that IPV exposure resulted in an adjusted OR of 1.66 (95% CI 1.39–1.98) of using condoms inconsistently in the past three months. Persistent IPV (reported at both baseline and follow-up) had an even stronger effect (adjusted OR 2.69, 95% CI 1.45–4.98).

Townsend et al.[28] found that reported IPV perpetration resulted in increased odds (adjusted OR 1.80, 95% CI 1.06–3.06) of high-risk South African men using condoms inconsistently in the past year.

Were et al.[29] also found that in Southern and East African serodiscordant couples, IPV was associated with having had unprotected sex between quarterly follow-up interviews (adjusted OR 1.86, p < 0.001).

In Scenarios D and G, we assumed that the per-sex-act odds of not using a condom were 1.8 times higher in violent relationships compared to non-violent. This number is similar to findings from the studies mentioned above, but one should note that translating an OR for inconsistent condom usage into a per-sex-act OR is bound to overestimate the true effect (thus exaggerating the extent of the causal relationship).

1.6 Reduction in rate of marriage in violent short-term relationships

A longitudinal study[30] of 411 couples in the US found that male violence had no significant effect on transitions from cohabitation into formal marriage. We were not aware of any studies measuring the effect of IPV on transitions from casual/short-term relationships into marriage/cohabitation (large longitudinal studies would be required to measure this).

We assumed in Scenarios E and G that current exposure to IPV resulted in a 25% decrease in rates of marriage (bearing in mind that the increased rate of relationship dissolution would even further decrease the relative risk that a violent couple marries).

1.7 Increase in rate of relationship dissolution in violent relationships/marriages

In a study of 3508 married or cohabiting couples in the US, with 5–7 years of follow-up, DeMaris[31] estimated a hazard ratio of 1.47 (95% CI 1.04–2.07) for the hazard of union disruption when the man was physically violent.

We thus assumed in Scenarios E and G that the rate of relationship dissolution in violent relationships was 1.5 times higher than in relationships which were otherwise identical but non-violent.

1.8 Increase in rate of acquiring secondary partners among women experiencing IPV

The effect of IPV on female infidelity has not been investigated by any studies we know of, but there has been speculation that this might be a factor explaining the association between IPV and HIV.[17,25,32,33] We therefore made an arbitrary assumption, in Scenarios F and G, that high-risk women currently exposed to IPV acquired secondary partners at a rate 1.5 times greater than women not exposed to IPV.

1.9 Reduction in level of viral suppression among women experiencing IPV

HIV-positive adults have substantially reduced risk of transmitting HIV to their sexual partners if they receive antiretroviral treatment (ART), although this is very dependent on the extent of viral suppression while on ART.[34,35] Hatcher et al.[36] found that HIV-positive women who had experienced IPV had lower rates of ART use and adherence, and lower levels of viral suppression if they were on ART. In particular, their meta-analysis found that among HIV-positive women on ART, the odds of viral load suppression were significantly lower among survivors of IPV (OR 0.64, 95% CI 0.46–0.90).

We thus assumed in scenario G that treated women currently experiencing IPV had 36% less reduction in infectivity compared to treated women who were not currently experiencing IPV.

2. Measures of relative risk

In the intervals 2003–2005, 2008–2010, and 2013–2015, we counted HIV infections in ever-partnered women who were HIV-negative at the start of the intervals, and aged 15–35 at the end, and calculated IRRs for the effect of lifetime IPV exposure on HIV incidence. Similarly, in the middle of 2005, 2010, and 2015, we calculated cross-sectional ORs for the association between current IPV exposure and HIV in married women aged 15–49. Table A2 summarises these results for each of Scenarios A–G. The results in 2015 are the same as those shown in Fig. 3 of the main text.

Table A2
Two measures of the relative risk of HIV in women exposed to IPV.

	IRRs for lifetime IPV (95% CI)			ORs for IPV in marriages (95% CI)		
	2005	2010	2015	2005	2010	2015
Scenario A	1.17 (1.13–1.21)	1.22 (1.17–1.27)	1.28 (1.23–1.34)	0.85 (0.83–0.87)	0.88 (0.86–0.90)	0.94 (0.92–0.96)
Scenario B	1.29 (1.25–1.33)	1.34 (1.29–1.39)	1.42 (1.36–1.48)	1.10 (1.07–1.13)	1.13 (1.11–1.16)	1.19 (1.16–1.22)
Scenario C	1.30 (1.26–1.34)	1.35 (1.30–1.39)	1.40 (1.35–1.46)	1.08 (1.06–1.11)	1.10 (1.07–1.13)	1.16 (1.13–1.20)
Scenario D	1.30 (1.26–1.33)	1.33 (1.28–1.38)	1.37 (1.31–1.43)	1.09 (1.06–1.12)	1.13 (1.10–1.15)	1.20 (1.17–1.22)
Scenario E	1.29 (1.25–1.33)	1.34 (1.30–1.39)	1.39 (1.33–1.44)	1.07 (1.04–1.09)	1.10 (1.08–1.13)	1.17 (1.14–1.19)
Scenario F	1.28 (1.23–1.32)	1.32 (1.27–1.36)	1.39 (1.33–1.44)	1.11 (1.08–1.13)	1.12 (1.09–1.14)	1.19 (1.17–1.22)
Scenario G	1.29 (1.25–1.33)	1.36 (1.31–1.41)	1.38 (1.32–1.43)	1.08 (1.05–1.11)	1.11 (1.09–1.14)	1.19 (1.16–1.21)

IRR = incidence rate ratio; OR = odds ratio.

Table A3 summarises the results from the four observational studies that are shown in Fig. 3 of the main text.

The study by Jewkes et al.[32] only published results that considered exposure to more than one episode of sexual or physical IPV ever and its effect on HIV incidence. In order to compare this study to the model, it was preferable to have an IRR that considered lifetime exposure to any sexual or physical IPV. All participants in the study were tested for HIV 12 months and 24 months after enrolment, unless they were lost to follow-up. Loss to follow-up was not significantly different in the group exposed to more than one episode of physical or sexual IPV compared to the unexposed group. It was not possible (from the publication) to tell which period of follow-up infections occurred in, but if we assume that incident cases were followed up for one year on average (before infection) and non-incident cases for two years on average, then the incidence rates of HIV in study participants exposed and unexposed to any IPV at baseline are as follows:

	Exposed to IPV	Unexposed to IPV	Total
Incident cases	60	68	128
Person-years	774	1296	2070
Incidence rate	0.078	0.052	

The estimated IRR is 1.48 (95% CI 1.04–2.09) when lifetime exposure to any sexual or physical IPV is considered (confidence interval calculated using the approximate Poisson method.)[37]

Table A3
Observed associations between IPV and HIV.

	Location; years	Participants	Unadjusted association
Cohort studies			
Jewkes et al.[32]	Eastern Cape, South Africa; 2002–2006	1099 women aged 15–26	IRR = 1.48 (95% CI 1.04–2.09)
Kouyoumdjian et al.[38]	Rakai, Uganda; 2000–2009	10 252 women aged 15–49	IRR = 1.37 (95% CI 1.08–1.75)
Cross-sectional studies			
Harling et al.[39]	Dominican Republic, Haiti, India, Kenya, Liberia, Malawi, Mali, Rwanda, Zambia, Zimbabwe; 2003–2007	60 114 women aged 15–49	Pooled OR = 1.10 (95% CI 1.01–1.19)
Durevall and Lindskog[33]	Burkina Faso, Côte d'Ivoire, Gabon, Kenya, Liberia, Malawi, Mali, Rwanda, Zambia, Zimbabwe; 2004–2012	40 247 married women aged 15–49	Pooled OR = 1.20 (95% CI 1.04–1.39)

IRR = incidence rate ratio; OR = odds ratio.

3. Sensitivity analyses

3.1 Time-dependence in the relative risk of HIV in women exposed to IPV

The first sensitivity analysis did not require running different simulations: we simply explored whether the associations between IPV and HIV (in terms of IRRs and ORs) were dependent on the time at which they were measured.

Both the IRRs and the ORs showed an increasing trend between 2005 and 2015 (Fig. A2). However, the general result — that the hypothesised causal pathways had little effect on measures of relative risk — was not sensitive to the time at which the associations were measured.
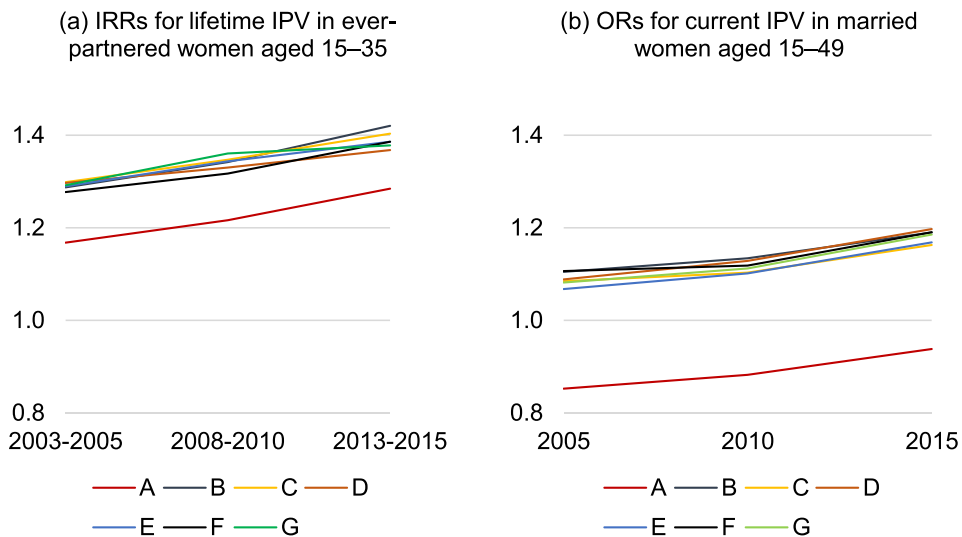
Fig. A2. Temporal trends in associations between HIV and IPV.

One possible reason for the observed trend is that throughout most of the 2000s, incidence and prevalence of HIV in women was declining more rapidly at younger ages than at older ages.[40,41] Since lifetime IPV is associated with older age, this could lead to an upward trend in the association between IPV and HIV.

Another reason is that the infection rate differential between high- and low-risk individuals changes according to the stage of the epidemic. Following two major HIV interventions — increases in condom use, in the mid-1990s, and increases in access to antiretroviral therapy, in the mid-2000s — high-risk individuals made up an increasing proportion of total new infections, due to a reversal of the saturation effects that occurred in the earlier stages of the epidemic.[42] This means that past IPV experience (which is more prevalent in women in the high risk group) becomes more strongly associated with HIV incidence.

We explored this second explanation by running the simulations again without modelling increases in condom use and antiretroviral therapy. We observed that the trend in the strength of the HIV-IPV association between 2005 and 2015 reversed (with IRRs and ORs decreasing over that period) (results not shown). This is the pattern that would be expected if saturation effects continued to cause a decline in the fraction of new HIV infections in high-risk individuals.

3.2 Tests for robustness

We also ran three additional scenarios where we varied certain parameters in Scenario B, each time running 100 simulations using the same HIV transmission parameters as before, to determine whether the model was robust to those changes.[43]

Sensitivity analyses 1a and 1b were intended to test the model robustness against changes in the assumed prevalence of lifetime IPV, which is uncertain in South African studies.[15,44–48] We changed this prevalence by:

1a. Increasing the rates of IPV incidence so that the model predicted a 45% prevalence of lifetime IPV exposure (Table A4).
1b. Decreasing the proportion of men who have violent predispositions so that the model predicted a 25% prevalence of lifetime IPV exposure (Table A4).

Table A4
Parameters used for sensitivity analyses 1a and 1b.

	Scenarios B, D–G	Sensitivity analysis 1a	Sensitivity analysis 1b
Ratio of probability of violent predispositions in high-risk men to that in low-risk men	1.70	1.70	1.70
Probability of a violent predisposition, high-risk men	0.4620	0.4620	0.2770
Annual rate of IPV incidence: short-term relationships[a]	0.3300	0.6100	0.3300
Annual rate of IPV incidence: marriages with <2 years duration[a]	0.4950	0.9150	0.4950
Annual rate of IPV incidence: marriages with >2 years duration[a]	0.2475	0.4575	0.2475

[a] This is the rate that applies if the male partner has a violent predisposition and if the female susceptibility factor is 1.

In sensitivity analysis 2, we explored the possibility that our results may have been distorted by the simplifying assumption that IPV is perpetrated by men at the same rate regardless of their age.

In multivariate models based on sub-Saharan African DHS data, Durevall and Lindskog[49] found a significant negative association between women's IPV experience and their husbands' ages, and Johnson et al.[50] explain why IPV perpetration is generally thought to peak in young adulthood: approximately 19 for males in the US, according to their study of adolescents.

We multiplied the incidence rates of IPV by an assumed adjustment factor of $\max\{1.1-0.8(\ln x - \ln 19)^2, 0\}$, where x is the centre of the man's five-year age bracket (Fig. A3). This formula had the convenient feature of allowing us to keep all other parameters the same and still maintain a 35% prevalence of lifetime IPV.
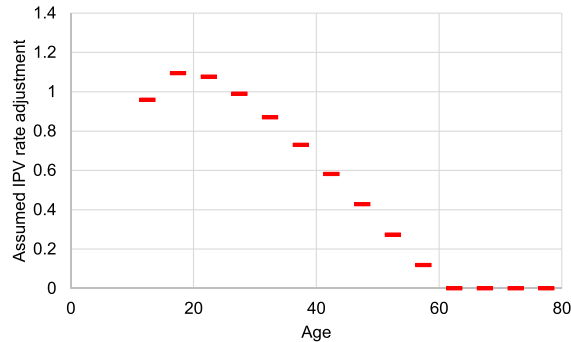

Fig. A3. Assumed multiplicative adjustment to the incidence rate of IPV according to men's ages.

Fig. A4(a) shows that there were slight differences in the results for the IRRs when parameter values were changed, but in general the magnitude of changes was small and the direction inconsistent (except for sensitivity analysis 1a, which consistently resulted in slightly higher IRRs). Similarly, there were only slight differences in the ORs (Fig. A4(b)) with a higher prevalence of lifetime IPV generally implying a higher OR.

We conclude that our measures of relative risk are relatively insensitive to the assumed prevalence of IPV in the population, as well as an assumption of age-heterogeneity in men's rates of IPV perpetration.
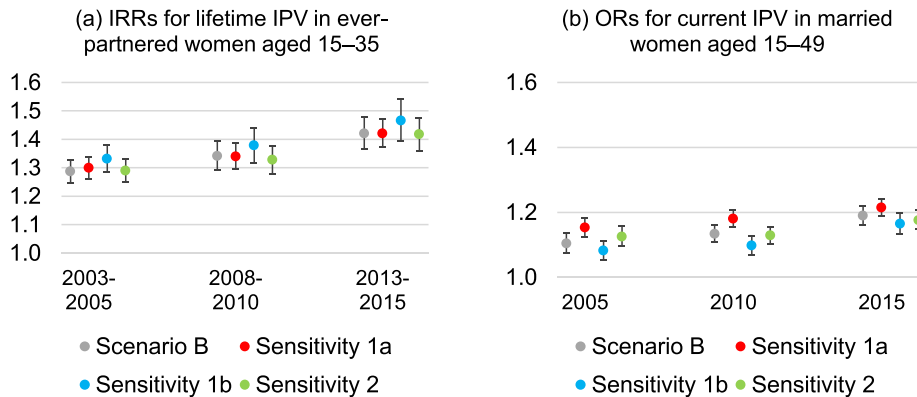

Fig. A4. Relative risk results from the sensitivity analyses.

4. Effects of assumed causal pathways on modelled HIV prevalence

Fig. A5 plots HIV prevalence over time from 1990 to 2024 in each of Scenarios D–G (where sexual behaviours changed in women who experienced IPV), compared to the prevalence in Scenarios A–C (where behaviours and HIV prevalence were identical). The HIV prevalence in Scenarios D–G is similar to that in Scenarios A-C, which indicates that the assumed behaviour changes do not negatively affect the model calibration to the HIV prevalence data (see Fig. 2a in the main text).
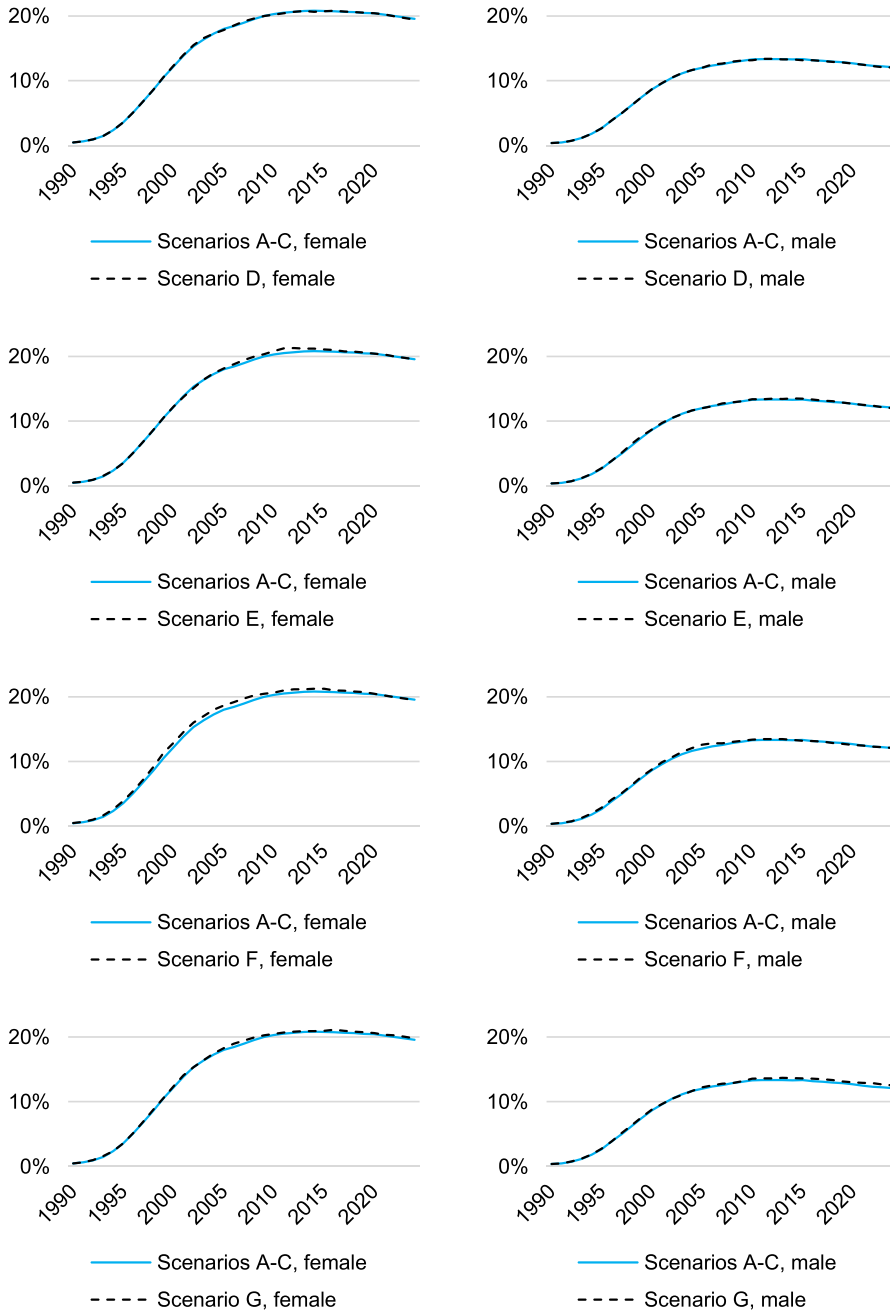
Fig. A5. Simulated HIV prevalence over time in the population aged 15–49 (median).

5. Other statistical results from the model

Table A5 shows the modelled prevalence of IPV exposure among ever-partnered women aged 15–49 (with various definitions of exposure) as well as the proportion of ever-partnered men who are predisposed to violence and, among those, the proportion who have actually perpetrated violence at least once.

Table A5
Modelled prevalence (%) of IPV exposure and perpetration in 2015 (median and IQR).

Scenario	Women				Men	
	Lifetime IPV[a]	IPV in past 12 months	IPV in marriage/cohabitation	IPV in ST relationships	Proportion with violent predispositions[b]	Proportion who have perpetrated IPV
A	32.9 (32.6–33.4)	12.3 (12.1–12.5)	17.4 (17.0–17.9)	2.2 (2.1–2.3)	36.5 (36.1–36.8)	23.9 (23.4–24.2)
B	32.8 (32.4–33.4)	11.7 (11.4–11.9)	15.4 (14.9–15.8)	2.4 (2.3–2.6)	33.7 (33.2–34.0)	22.9 (22.5–23.3)
C	32.9 (32.4–33.4)	8.9 (8.7–9.1)	11.2 (10.8–11.5)	1.9 (1.8–2.1)	24.2 (23.9–24.5)	17.8 (17.5–18.1)
D	32.9 (32.3–33.4)	11.7 (11.4–12.0)	15.4 (15.0–15.8)	2.5 (2.3–2.6)	33.6 (33.2–34.1)	22.9 (22.5–23.3)
E	33.1 (32.7–33.6)	10.9 (10.6–11.1)	14.4 (14.1–14.8)	1.7 (1.6–1.8)	33.6 (33.3–33.9)	22.8 (22.6–23.2)
F	32.9 (32.4–33.4)	11.8 (11.5–12.0)	15.6 (15.0–15.9)	2.4 (2.2–2.6)	33.6 (33.2–34.0)	22.9 (22.5–23.2)
G	33.1 (32.7–33.6)	10.9 (10.6–11.2)	14.4 (14.0–14.9)	1.7 (1.6–1.9)	33.6 (33.3–34.0)	22.9 (22.6–23.3)

[a] The models are fitted such that the prevalence of lifetime IPV is 35% in year 2024; in 2015 the prevalence is slightly less because there is some left censoring − women who were sexually active at the start of the simulations in 1985 could theoretically have been exposed to IPV before that and not subsequently.

[b] In Scenarios B–G, the proportion of adult men with violent predispositions is slightly lower than the proportion initially assigned with violent predispositions. This is because men with violent predispositions, being more likely to be high-risk, experience higher AIDS-related mortality compared to non-violent men.

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