Articles

Intimate partner violence, relationship power inequity, and incidence of HIV infection in young women in South Africa: a cohort study

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Summary

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Background

Cross-sectional studies have shown that intimate partner violence and gender inequity in relationships are associated with increased prevalence of HIV in women. Yet temporal sequence and causality have been questioned, and few HIV prevention programmes address these issues. We assessed whether intimate partner violence and relationship power inequity increase risk of incident HIV infection in South African women.

Methods

We did a longitudinal analysis of data from a previously published cluster-randomised controlled trial undertaken in the Eastern Cape province of South Africa in 2002–06. 1099 women aged 15–26 years who were HIV negative at baseline and had at least one additional HIV test over 2 years of follow-up were included in the analysis. Gender power equity and intimate partner violence were measured by a sexual relationship power scale and the WHO violence against women instrument, respectively. Incidence rate ratios (IRRs) of HIV acquisition at 2 years were derived from Poisson models, adjusted for study design and herpes simplex virus type 2 infection, and used to calculate population attributable fractions.

Findings

128 women acquired HIV during 2076 person-years of follow-up (incidence 6·2 per 100 person-years). 51 of 325 women with low relationship power equity at baseline acquired HIV (8·5 per 100 person-years) compared with 73 of 704 women with medium or high relationship power equity (5·5 per 100 person-years); adjusted multivariable Poisson model IRR 1·51, 95% CI 1·05–2·17, p=0·027. 45 of 253 women who reported more than one episode of intimate partner violence at baseline acquired HIV (9·6 per 100 person-years) compared with 83 of 846 who reported one or no episodes (5·2 per 100 person-years); adjusted multivariable Poisson model IRR 1·51, 1·04–2·21, p=0·032. The population

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attributable fractions were 13·9% (95% CI 2·0–22·2) for relationship power equity and 11·9% (1·4–19·3) for intimate partner violence.

Interpretation

Relationship power inequity and intimate partner violence increase risk of incident HIV infection in young South African women. Policy, interventions, and programmes for HIV prevention must address both of these risk factors and allocate appropriate resources.

Funding

National Institute of Mental Health and South African Medical Research Council.

Introduction

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For two decades, gender-based violence and gender inequity in relationships have been associated with increased risk of HIV in women.[1, 2, 3] Cessation of violence against women and girls is one of nine priority areas in the UNAIDS Outcome Framework for 2009–11,[4] and many national HIV strategic plans acknowledge the need to address gender issues, albeit with uneven implementation. Although most new HIV infections in high prevalence areas are in women, prevention agendas remain dominated by promotion of male condom use, HIV testing, treatment for sexually transmitted infections (STIs), and more recently male circumcision and antiretroviral treatment. By not focusing on gender issues, these interventions provide little help for vulnerable women.[5, 6, 7] Additionally, research linking gender inequity and gender-based violence to HIV is limited. The absence of longitudinal research on the topic has enabled sceptics to resist prioritising gender in resource allocation. Yet cross-sectional studies, from both low and high prevalence settings, show associations between partner violence and male controlling practices and HIV serostatus in women.[8, 9, 10, 11] These connections arise through multiple pathways (figure), predominantly in settings or populations with a high prevalence of HIV.

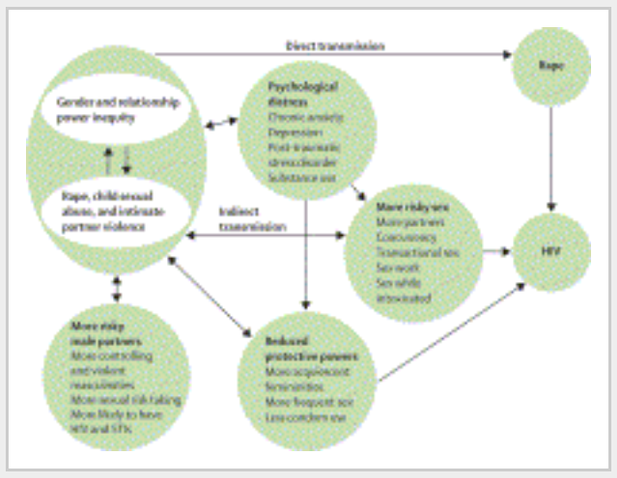

Figure
Pathways through which gender-based violence and gender and relationship power inequity might place women at risk of HIV infection

STIs=sexually transmitted infections.

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Violence is a consequence of gender power inequities, at both a societal and relationship level, and also serves to reproduce power inequities.[12] Qualitative research has shown that the links between HIV/AIDS, gender inequity, and gender-based violence lie in the patriarchal nature of society, and ideals of masculinity that are based on control of women and that celebrate male strength and toughness.[12, 13] These ideals readily translate into risky sexual behaviours, predatory sexual practices, and other acts of violence against women.[2] Additionally, they allow men to have multiple partners and control their sexual encounters. Emerging evidence from South Africa and India shows that men who perpetrate violence are more likely to be HIV infected.[11, 14] Although individual women might resist male power, women largely acquiesce to these practices, especially in developing countries. Violence prevents women from influencing the circumstances of sex, resulting in more frequent sex, and less condom use.[15, 16, 17]

The figure shows rape as a potential source of HIV infection. Yet even in high prevalence settings with injury, a single sexual act has a low risk of HIV transmission;[18] thus, rape results in few HIV cases in

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women. From a population perspective, partner violence and gender inequity have a greater effect on risk of HIV through longacting indirect pathways. These pathways pertain in chronically abusive relationships, with repeated exposure to one individual, and in women who have had previous, but not necessarily continuing, exposure to violence (in childhood or as adults) and controlling practices.

In developing and developed countries, exposure to gender-based violence, including controlling behaviour of a partner, is associated with high-risk sexual behaviour, including multiple and concurrent sexual partnerships, substance use, transactional sex and prostitution, and less frequent condom use.[8, 15, 19, 20, 21, 22, 23, 24] This association partly results from psychological effects, which often last years after the incidents of violence.[25] Women might agree to riskier sex, and be less able to refuse it, when drunk, drugged, dissociating, desperately seeking affection, or otherwise manipulated by controlling partners.[23, 24, 25] Thus, there is a vicious cycle, with abuse enhancing risks of HIV infection and further abuse.

Although research has improved our understanding of potential connections between intimate partner violence, gender inequity, and HIV infection, convincing assertions of causality require epidemiological evidence on temporal sequence, which has not been available.[26] The dataset from the evaluation of the HIV prevention intervention Stepping Stones in rural South Africa[27, 28] presents an opportunity to examine hypotheses that exposure to sexual and physical intimate partner violence and gender power inequity in relationships at baseline predict incident HIV infections over 2 years of follow-up. We did a longitudinal analysis of data from 1099 young South African women from this trial who were HIV negative at baseline and had subsequent HIV test results.

Methods

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Participants

Between 2002 and 2003, male and female volunteers aged 15–26 years were enrolled in a cluster-randomised controlled trial to assess the effect of an HIV prevention programme on incidence of HIV, herpes simplex virus type 2 (HSV2), and sexual behaviour (ClinicalTrials.gov number NCT00332878).[28] 70 locations (clusters) in the Eastern Cape province of South Africa were grouped into seven geographically defined strata. Within each stratum, equal numbers of clusters were randomly allocated to intervention or control. In each cluster, 15–25 male and 15–25 female volunteers were recruited from schools. Volunteers were eligible for enrolment if they were aged 16–23 years, normally resident in the village where they were at school, and mature enough to understand the study and the consent process. All participants gave written informed consent.

Participants in intervention clusters were assigned to receive Stepping Stones, a 50-h participatory intervention on sexual and reproductive health and HIV, delivered over 6–8 weeks. Participants in control clusters received a 3-h intervention on safer sex and HIV, delivered on one occasion. Apart from these interventions, participants in the two groups were not treated differently. Assessments at baseline, 12 months, and 24 months consisted of a face-to-face questionnaire and blood tests for HIV and HSV2. The cohort was maintained by use of details obtained at enrolment, with follow-up undertaken nationwide to trace migrant youth. Further information about all assessments, study recruitment, access, and ethical issues, including support for participants testing HIV positive, is published elsewhere.[27, 28] Ethics approval for the trial was given by the University of Pretoria, Pretoria, South Africa.

We used data obtained in this trial to assess the effects of intimate partner violence and power inequity in relationships on incidence of HIV infection at 2 years of follow-up. For this longitudinal analysis, we excluded women who had HIV infection at baseline, women with missing data, and those who were lost to follow-up at both 12 months and 24 months.

Laboratory methods

HIV serostatus at baseline was assessed by use of two rapid tests.[29] The Determine (Abbott Diagnostics, Johannesburg, South Africa) test was used for screening and samples with positive results were retested with Uni-Gold (Trinity Biotech, Dublin, Ireland). Indeterminate results were clarified by use of an HIV-1 screen ELISA (Genscreen; Bio-Rad, Steenvoorde, France) followed by two

confirmatory ELISAs if the sample was positive for HIV (Vironostika; BioMérieux, Marcy l'Etoile, France and Murex 1.2.0; Murex Biotech, Dartford, UK). Towards the end of the second round of interviews, collection of blood as dried spots was introduced for some participants to ease logistics and improve acceptability. In the third round of interviews, most blood was obtained as dried spots. The samples were tested with a screen and confirmatory ELISA. In this analysis, 745 (68%) of the final HIV outcomes were from dried blood spots, equally distributed among participants who remained HIV negative (n=658, 68%) and those who seroconverted (n=87, 68%).

A glycoprotein G-based HSV2 ELISA was used to test for herpes infection (Kalon; Kalon Biological, Aldershot, UK). A CAPTIA HSV IgG type-specific ELISA was used to resolve discrepant results.

Questionnaire

We recorded age and completed years of schooling. Socioeconomic status was assessed by use of a scale that encompassed household goods ownership, food, and cash scarcity. We asked about numbers of boyfriends, concurrency, condom use, sex or pregnancy, and time since first sexual intercourse.

A sexual relationship power scale (ten items, Cronbach's alpha 0·73) was used to measure gender power equity.[8, 30] A typical item was “When (NAME OF BOYFRIEND) wants me to sleep over he expects me to agree”. Each item was assessed on a 4-point Likert scale and the measure was scored and categorised into tertiles of the measure. For the analysis, the tertile with lowest equity was compared with the middle and higher ones.

We used the WHO violence against women instrument to measure physical partner violence (five items) and sexual partner violence (four items) either over the past year or during the participant's lifetime.[31] By use of the method described by Dunkle and colleagues,[8] we coded physical or sexual violence into more than one episode versus none or only one. Women who disclosed being gang raped, being “forced or persuaded against their will” to have sex by a non-partner, or forced by more than one man to have sex were coded as experiencing rape by a non-partner.

We derived variables for possible time-varying covariates during follow-up. In each case we considered behaviour reported at either the 12 month interview (ie, between baseline and 12 months) or the 24 month interview (if the participant had not seroconverted at 12 months). Concurrency was defined as any reported *khwapheni.* This is an indigenous partner category that is, by definition, concurrent.[8] Condom use and correctness of condom use (ie, without breakage, slipping off, or late use) was assessed for the last sexual intercourse. Partner numbers were categorised by whether two or more were reported. Transactional sex with a casual partner was measured from questions asking about sex motivated by expectations of receiving one of a range of items (as described by Dunkle and colleagues[8]).

Data analysis

Analyses were done with Stata version 10.0. All procedures took into account the study design, and considered the dataset as a cohort with a stratified, two-stage structure with participants clustered within villages. For each participant, we calculated the person-years of exposure as the time from baseline to the last negative HIV result if the person remained negative, or as the total time between any negative tests as well as half the time between the last negative and first positive HIV test results.

Social, demographic, and relationship characteristics, prevalence of HSV2 infection, and violence exposures were summarised as percentages (or means) with 95% CIs, by use of standard methods for estimating CIs from complex multistage sample surveys (Taylor linearisation). Pearson's χ^2 test was used to test associations between categorical variables.

To account for clustering of women within villages, random effects (multilevel) models were fitted. Random effects Poisson models were built to test the hypothesis that baseline partner violence and relationship inequity predicted HIV incident infections. Each model included variables for age, study treatment group, stratum, and person-years of exposure. Partner violence, rape, and relationship power equity variables were initially modelled separately. We then fitted models with both a measure of relationship power inequity and partner violence. For the initial models, we modelled

partner violence exposure in women who had had a boyfriend at baseline. We tested for, and found no interactions between variables in the model, including treatment group. We repeated the modelling with adjustment for variables that we determined a priori to be potential confounders. HSV2 seropositivity at baseline was deemed to be a potentially important biological cofactor. We also tested the models for the possibility of confounding by age, education, socioeconomic status, pregnancy, and duration of sexual activity, but found none. We tested the HSV2-adjusted model for the range of possible behavioural covariates during follow-up and found none to be confounders. We tested goodness of fit by use of the Poisson test. We confirmed the findings of associations for each outcome variable by modelling survival time under observation with a Weibull model, with the same set of other variables included. To investigate whether results were robust to missing data, we undertook a sensitivity analysis with inverse probability weighting.

By use of Greenland's method,[32] the population attributable fractions (PAFs) for partner violence and low levels of relationship power equity were calculated with the incidence rates (IRs) from the adjusted model and the formula PAF=([IR−1]/IR)×P_e where P_e was the proportion of the cases that had the exposure. CIs were calculated by use of the same formula, but with the upper and lower confidence limits of the incidence rate ratio (IRR).

Role of the funding source

The National Institute of Mental Health had no role in study design, data collection, data analysis, data interpretation, or writing of the report. The corresponding author had full access to the data, analysed it, and had final responsibility for the decision to submit for publication. The South African Medical Research Council's contribution to the study consisted of discretionary funds and staff time that were entirely under the control of the first author. These made up the difference between available funds from the National Institute of Mental Health and project funding needs.

Results

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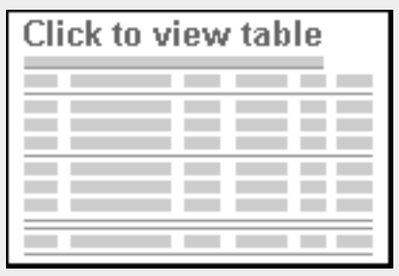
Of the 1415 women who were enrolled into the trial, 316 were excluded from this analysis (159 women had HIV infection at baseline, one had missing data, and 156 were lost to follow-up at both 12 and 24 months). The 1099 women included in this analysis represent 88% of 1256 HIV-negative women in the trial.

Table 1 shows characteristics of women who were followed up and lost to follow-up. HIV-negative women who were lost to follow-up (156 of 1256, 12%) were older, more likely to have had a boyfriend and sex at baseline, and more likely to have low relationship power equity than were those who were followed up.

Table 1

Sociodemographic and behavioural characteristics of HIV-negative women who were followed up and lost to follow-up



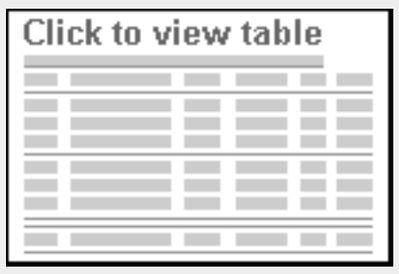
Data are n (%) or mean (95% CI). IPV=intimate partner violence. HSV2=herpes simplex virus type 2.

* n=1097 followed up, n=156 lost to follow-up.
† Only for sexually active participants; n=980 followed up, n=148 lost to follow-up.
‡ Only for sexually active participants; n=965 followed up, n=146 lost to follow-up.
§ Only for partnered women; n=1029 followed up, n=148 lost to follow-up.
¶ n=152 lost to follow-up.

At 12 months and 24 months, 41 (4%) of 976 women and 18 (2%) of 962 women had not had sex,

respectively. Most women were in school (1079 of 1099, 98%), with most being two or more years from school completion (table 2). Generally, the participants' households were poor or very poor, for example 385 (35%) of 1099 often or sometimes went hungry. All participants were unmarried, but 197 (18%) of 1099 were mothers.

Table 2
Sociodemographic and behavioural characteristics of women who did and did not acquire HIV over 2 years



IPV=intimate partner violence. HSV2=herpes simplex virus type 2.

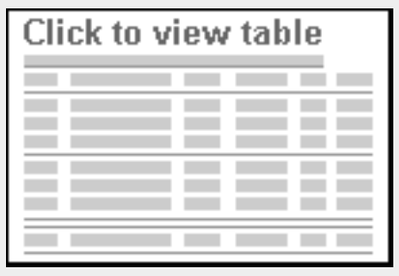
* Data are available for 124 women who seroconverted because one participant had never had a boyfriend, and two had had no recent boyfriend. One participant had missing data on this scale. Data are available for 905 women who did not seroconvert because only these women had partners.

128 women acquired HIV during 2076 person-years of follow-up (HIV incidence 6·2 per 100 person-years). Participants' characteristics by HIV serostatus at 24 months are shown in table 2. There were no significant differences in age, education, or socioeconomic status between women who did and did not acquire HIV. Compared with women who did not acquire HIV, women who seroconverted were more likely to have had sex and a pregnancy at baseline; they had been sexually active for longer, and had more concurrent relationships, both at baseline and during follow-up. A higher proportion of women who acquired HIV during the study had reported violence and high gender power inequity at baseline than had women who did not acquire the infection. Although 517 (54%) of 965 sexually active women had used condoms, women who seroconverted were more likely to have used condoms in the year before the baseline survey and to report having done so correctly at last sexual intercourse before their final HIV test than were women who did not seroconvert. Prevalence of HSV2 infection at baseline was higher in women who acquired HIV than in women who did not acquire the infection. 25 women who were still HIV negative at 12 months acquired HSV2 infection, but this did not increase risk of later HIV seroconversion.

At baseline, women in the lowest power equity category were significantly more likely to have experienced more than one episode of physical or sexual intimate partner violence than were women in the higher category (94 of 325, 29%, *vs* 154 of 704, 22%; p=0·014). Women who had reported violence at baseline were more likely to have HSV2 infection at baseline than were women who had not reported violence (78 of 253, 30·8%, *vs* 167 of 846, 19·7%; p=0·0002).

The incidence of HIV and IRR derived from the adjusted multivariable Poisson models are shown in table 3. Women in relationships with low gender equity at baseline had a much higher incidence of HIV than did women in the middle or highest relationship equity category. HIV incidence was higher in women who had reported more than one episode of physical or sexual intimate partner violence at baseline than in women who reported one or no episodes of violence. Women who had been raped by a non-partner had a similar incidence of HIV to those who had not. Table 4 shows two Poisson models with exposure to violence and relationship power equity as independent variables. Both variables had an independent association with HIV incidence, after adjustment for age, study design, and HSV2 infection at baseline. Behavioural variables were not confounders (table 5).

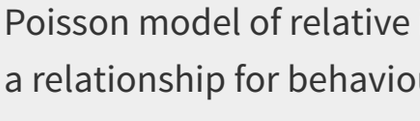
Table 3
Incidence and relative incidence of HIV infection, by exposure to forms of violence and inequity



IRR=incidence rate ratio. HSV2=herpes simplex virus type 2. All Poisson models adjusted for age, treatment, stratum, and person-years of exposure.

* Models additionally adjusted for HSV2 infection at baseline.
† Data available for 124 women who seroconverted because one had never had a boyfriend, and two had had no recent boyfriend. One had missing data on this scale.
‡ Model for ever-partnered women.

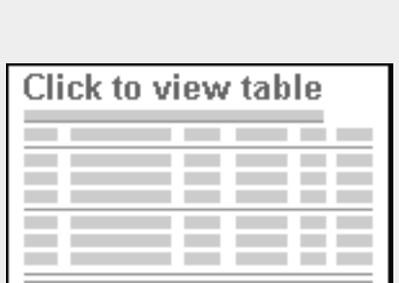
Table 4
Relative HIV incidence with exposure to both partner violence and relationship inequity



IRR=incidence rate ratio. HSV2=herpes simplex virus type 2. IRRs adjusted for age, treatment, stratum, and person-years of exposure.

* Additionally adjusted for HSV2 infection at baseline.

Table 5
Effects of behavioural variables on relative incidence of HIV



IRR=incidence rate ratio. HSV2=herpes simplex virus type 2. Effects of adjusting the multivariable Poisson model of relative incidence of HIV of exposure to partner violence and gender inequity in a relationship for behavioural variables in addition to HSV2 infection at baseline.

The population attributable fractions (table 6) show that 13·9% of incident HIV infections could be avoided if gender equity in heterosexual relationships was enhanced so that no women were in relationships with low power. Similarly, for violence, 11·9% of new HIV infections could be prevented if women did not experience more than one episode of physical or sexual partner violence.

Table 6
Population attributable fractions

Population attributable fractions calculated by use of incidence rates for the Poisson model shown in table 4 adjusted for herpes simplex virus type 2 infection at baseline.

A sensitivity analysis to investigate whether results were robust to missing data suggested that the potential effect of missing data was negligible (data not shown).

Discussion

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This study shows that in rural South Africa, women who experienced intimate partner violence and had high gender inequity in relationships had increased incidence of HIV infection. Risk of incident HIV infection was not associated with rape by a non-partner. Our results substantiate previous findings from cross-sectional studies.[8] The population attributable fractions reported here show the importance of effectively addressing the HIV epidemic through programmes and interventions that address violence and gender inequity in relationships.

This study provides strong temporal evidence to support a causal association between intimate partner violence or relationship inequity and new HIV infection. The relation between these variables is plausible and coherent, and research from several settings has shown consistency and supports the strength of association. There is no experimental evidence to suggest that reducing women's exposure to violence reduces HIV incidence. Replicating this association in the context of trials to assess effective interventions should be a priority. There are some promising interventions that seek to empower women and change ideals of masculinity.[7, 28] In addition to Stepping Stones, potentially effective interventions from around the world include Sexto Sentido from Nicaragua,[33] Program H from Brazil,[34] the Intervention with Microfinance for AIDS and Gender Equity (IMAGE) programme from South Africa,[35] and Better Life Options for Boys from India.[36] Research to assess and refine these and other such programmes is urgently needed.[37]

Incidence of HIV in our cohort (6%) was similar to that estimated nationally in women aged 15–24 years (6·5%).[38] The results of two-way associations between HIV seroconversion and baseline sexual risk behaviour and HSV2 infection, and between seroconversion and concurrency during follow-up were as expected. The exception here was condom use, both frequency of use at baseline and during follow-up. Although possibly explained by measurement error, the consistency across several condom use indicators suggests another explanation. Since condom use is predominantly determined by male partners, it is more likely that men initiate use after a diagnosis of HIV or STI, but then become inconsistent in condom use.

Elimination of confounding variables, without over-adjusting the models,[26] is difficult to achieve in observational epidemiology, especially with few new infections. We tested a range of possible confounders and found that none substantially altered the effect sizes of interest. Having tested the models extensively for confounding and interactions, we are confident that our models were appropriately constructed and fit the data well.

This study has strengths and limitations. The cohort consisted of volunteers, and although retention was high, there was some loss to follow-up and bias in the characteristics of those who were lost. We tested for robustness to missing data and our results suggested that the potential effect was small. We did not adjust for changes in exposure category during follow-up, in view of the short period of observation and uncertainty about temporality of infection and violence. We cannot exclude effects of exposure change on the findings, but expect that any effects would bias findings towards the null hypothesis. Follow-up data were interval censored and the model included the standard method of dealing with uncertainty about the time of HIV seroconversion in such data. We cannot know when seroconversion occurred and so cannot accurately estimate the errors in such an assumption, although we do not think that these would be biased by exposure to violence or gender inequity. We tried to ensure that this recognised and unavoidable problem[39] did not result in spurious findings by confirming the results through two different approaches to model building. New HIV infections might have been slightly under-ascertained because of the period of study. Finally, the Stepping Stones intervention might have introduced biases in this analysis, but we adjusted for study treatment group and checked for interactions. There was no effect of treatment group on incidence of HIV in women.[28]

Exposure to physical and sexual intimate partner violence and low relationship power equity increase incidence of HIV in young women in rural areas of South Africa, and account for a substantial proportion of HIV infections. The association is likely to be causal because it accords with

cross-sectional evidence from countries around the world. Organisations driving HIV prevention agendas for women, particularly UNAIDS and WHO, need to ensure that policies, programmes, and interventions to build gender equity and prevent partner violence are developed and widely implemented. Donors and researchers must invest efforts and resources in developing and testing new interventions.

Contributors

RKJ designed the Stepping Stones study and was the principal investigator for the trial. RKJ analysed the data for this study and wrote the report. KD contributed to the design of this study, assisted with data management, and assisted in the interpretation of the data and drafting of the report. MN contributed to the design of the study and project management. NS contributed to study design and implementation. All authors saw and approved the final version of the report.

Conflicts of interest

We declare that we have no conflicts of interest.

Acknowledgments

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