



Pathways from parental AIDS to child psychological, educational and sexual risk: Developing an empirically-based interactive theoretical model

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ABSTRACT

Increasing evidence demonstrates negative psychological, health, and developmental outcomes for children associated with parental HIV/AIDS illness and death. However, little is known about *how* parental AIDS leads to negative child outcomes. This study used a structural equation modelling approach to develop an empirically-based theoretical model of interactive relationships between parental or primary caregiver AIDS-illness, AIDS-orphanhood and predicted intervening factors associated with children's psychological distress, educational access and sexual health. Cross-sectional data were collected in 2009–2011, from 6002 children aged 10–17 years in three provinces of South Africa using stratified random sampling. Comparison groups included children orphaned by AIDS, orphaned by other causes and non-orphaned, and children whose parents or primary caregivers were unwell with AIDS, unwell with other causes or healthy. Participants reported on psychological symptoms, educational access, and sexual health risks, as well as hypothesized sociodemographic and intervening factors. In order to build an interactive theoretical model of multiple child outcomes, multivariate regression and structural equation models were developed for each individual outcome, and then combined into an overall model. Neither AIDS-orphanhood nor parental AIDS-illness were *directly* associated with psychological distress, educational access, or sexual health. Instead, significant *indirect* effects of AIDS-orphanhood and parental AIDS-illness were obtained on all measured outcomes. Child psychological, educational and sexual health risks share a common set of intervening variables including parental disability, poverty, community violence, stigma, and child abuse that together comprise chain effects. In all models, parental AIDS-illness had stronger effects and more risk pathways than AIDS-orphanhood, especially via poverty and parental disability. AIDS-orphanhood and parental AIDS-illness impact child outcomes through multiple, interlinked pathways. The interactive model developed in this study suggests key areas of focus for interventions with AIDS-affected children.

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Introduction

Individualistic approaches to understanding HIV/AIDS obscure the widespread family ramifications of illness, disease and death. For many children, especially those living in countries with high endemic HIV rates, parental HIV/AIDS is a heavy burden, with an estimated 16 million children orphaned by AIDS by 2012 (UNICEF &

UNAIDS, 2011), and a further 70–90 million living with HIV/AIDS-affected parents or primary caregivers. For the purposes of this paper, 'orphanhood' refers to death of a biological parent, and 'parental AIDS-illness' refers to a parent or primary caregiver.

Increasing evidence, now synthesized in systematic reviews, demonstrates that parental HIV/AIDS is associated with major negative developmental outcomes for children, primarily in psychological health, such as depression, anxiety and stress (Breuer, Myer, Struthers, & Joska, 2011; Sherr & Mueller, 2008; Sherr et al., 2008), educational access, such as enrolment and achievement (Guo, Li, & Sherr, 2012) and sexual health, such as early debut and transactional sex (Operario, Underhill, Chuong, & Cluver, 2011). Recent longitudinal studies suggest that these negative outcomes are

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long-lasting (Cluver, Orkin, Gardner, & Boyes, 2011) and impact children's functioning, economic opportunities, and risks of becoming infected with HIV themselves (Case, Paxson, & Ableidinger, 2002; Operario, Pettifor, Cluver, MacPhail, & Rees, 2007).

Understanding *how* parental HIV/AIDS leads to negative child outcomes is essential both for understanding child development in sub-Saharan Africa, and for informing effective interventions for AIDS-affected children. However, this relationship is far from clear. Studies on parental bereavement and illness more generally (Dowdney, 2008) have shown understandable negative impacts on children's well-being, connected to grief, loss of attachment figures and uncertainty (Forrest, Plumb, Ziebland, & Stein, 2006). But why is this one particular virus – HIV/AIDS – linked to greater negative impacts than other causes of parental illness and death such as cancer or homicide (Cluver et al., 2011)? Within the comparatively recent field of research on children affected by AIDS, a small number of studies have empirically tested variables that intervene between parental AIDS and children's outcomes. These studies have identified important risk pathways, demonstrating that orphanhood is linked to sexual behaviour via reduced educational access (Birdthistle, Floyd, Nyagadza, Gregson, & Glynn, 2009) and via psychological distress (Nyamukapa et al., 2008) and that AIDS-orphanhood is linked to psychological disorder via AIDS-related stigma (Boyes & Cluver, in press; Cluver, Gardner, & Operario, 2008) and poverty (Cluver, Gardner, & Operario, 2009), or both (Cluver & Orkin, 2009).

However, to date, such empirical studies have focused on unitary or at most dual pathways from parental HIV/AIDS to single child outcomes. This contrasts with the more established research field of child development in the developed world, which suggests that it is not single factors, but rather multiple interlinking pathways that predict child outcomes in contexts of high risk (Gershoff, Aber, Raver, & Lennon, 2007; Lober & Farrington, 2000; Rutter et al., 1997). Influential frameworks include Sameroff's transactional theory of the impacts of parental psychopathology (Sameroff, 2000), Cicchetti's ecological/transactional model of the impacts of child maltreatment (Cicchetti & Lynch, 1993), and Rutter's pathways theory which identifies lifetime direct and indirect chain effects of childhood adversity (Rutter, 2005). These theoretical models – whilst having distinctive characteristics – share commonalities. First, all models are based on an ecological framework that conceptualizes the child at the centre of a network of interacting influences at individual, family and community-level (Bronfenbrenner, 1979), and suggests that improvements in one area can buffer against deficits in another. Second, all recognize that risk and protective factors function not as unitary cause–effect models but rather within complex relationships of multiple, interacting factors. Third, all models have been empirically tested using statistical techniques which allow simultaneous analysis of multiple variables. These three attributes inform the model advanced in this paper, but to date no interactive theoretical models have been tested to examine the pathways by which parental HIV/AIDS impacts children's outcomes.

Key commentators have hypothesized that risks for AIDS-affected children interact within a similar ecological framework (Li et al., 2008; Richter, Foster, & Sherr, 2006). An important step towards testing this was the recent paper by Wang et al. (2012), modelling predictors of depression within rural children in China whose parents had been HIV-infected via unhygienic blood collection. This study identified traumatic events and stigma as risk factors, and highlighted the need for further research including children whose parents have contracted HIV through sexual transmission (Wang et al., 2012). Our study addresses this, and includes both AIDS-affected children and comparison groups of non-affected children to allow examination of pathways of increased risk. Such models are important not only because they provide potential

explanatory pathways but also because they may identify entry points for interventions to reduce or avoid such negative impacts.

This study aimed to develop and test an interactive theoretical model of the impacts of parental AIDS illness and death within the sub-Saharan African context, a region experiencing 67% of the global burden of HIV/AIDS (WHO, 2011a). Until now researchers have lacked sufficient data to allow empirical testing of such a model, which requires substantial sample size, inclusion of both AIDS-affected and non-affected comparison groups (Li et al., 2008) and hypothesized risk factors at individual, family, and social levels. This study aims to address these gaps in our knowledge for three groups of child outcomes: psychological disorders, education risks, and sexual health risks. An interactive model has the potential to elucidate important pathways through which parental AIDS and AIDS-orphanhood may impact child development in sub-Saharan Africa. In addition, such a model will allow the identification of factors that, if modified, could potentially ameliorate negative pathways for AIDS-affected children. A structural equation modelling approach was chosen in order to allow simultaneous analysis of multiple predictors, intervening variables and outcomes (Seifer & Sameroff, 1982).

Method

Procedures

A community-based survey was conducted using stratified random sampling of census enumeration or designated tribal areas, in six randomly-selected health districts with over 30% antenatal HIV-prevalence, in three South African provinces. Sites comprised deep rural, dense rural, commercial farming rural, peri-urban, urban and urban-homeland areas. In consecutive door-to-door household sampling, one randomly-selected child aged 10–17 per household was interviewed ($n = 6002$). Voluntary informed consent was obtained from both children and parents or primary caregivers (response rate 97.2%). All interviewers were community health or social workers, trained in working with AIDS-affected families. All questionnaires were translated and back-translated into Xhosa, Zulu, Sotho, Swati and Shangaan and children completed face-to-face interviews lasting 60–70 min in the language of their choice. Confidentiality was maintained, except where participants were at risk of significant harm or requested assistance. Where participants reported abuse, rape or risk of significant harm, immediate referrals were made to child protection and health services. Where prior abuse or rape was no longer occurring, referrals were made to support and counselling services, and to HIV/AIDS testing and treatment services where appropriate. Ethical protocols were approved by Oxford University, the Universities of Cape Town and KwaZulu-Natal, and Provincial Health and Education Departments of the Western Cape, Mpumalanga and KwaZulu-Natal. No participant incentives were given, apart from refreshments and certificates.

Measures

Child risk outcomes

Psychological distress was measured using standardized scales used previously with children in South Africa. Depression was measured with the 10-item Child Depression Inventory Short Form (Kovacs, 1992), highly correlated with the full scale ($r = 0.89$; Kovacs, 1992). Reliability in the current sample was $\alpha = 0.73$. Anxiety was measured using an abbreviated version of the 28-item Revised Children's Manifest Anxiety Scale (Reynolds & Paget, 1983), which has been validated for use in South Africa (Boyes & Cluver, 2012). The 14 highest loading items were identified through

factor analysis in a previous study (Cluver, Orkin, Gardner, & Boyes, 2012) ($\alpha = 0.84$ in the current sample). Posttraumatic stress symptoms were measured using the Child PTSD Checklist (Amaya-Jackson, 1995). This comprises 28 DSM-IV-derived items, and has been validated and frequently used in South Africa (Boyes, Cluver, and Gardner, in press; Cluver, Fincham, & Seedat, 2009; Seedat, Nyamai, Njenga, Vythilingum, & Stein, 2004; Suliman, Kaminer, Seedat, & Stein, 2005) ($\alpha = 0.94$ in the current sample). Suicidality was measured with the MINI International Psychiatric Interview for Children and Adolescents suicidality subscale (Sheehan, Shytle, & Milo, 2004) ($\alpha = 0.80$ in the current sample). Educational access risks were identified in collaboration with the government's National Department of Basic Education and included non-enrolment; extended periods (>1 week) of past-year school non-attendance; 2+ years behind age-appropriate school grade; inability to concentrate in school due to worry about home circumstances (Cluver, Operario, Lane, & Kganakga, 2012). Sexual health risks were measured using a checklist of items from the National Survey of HIV and Risk Behaviour amongst Young South Africans (Pettifor et al., 2003) and the South African Demographic and Health Survey (Department of Health & Medical Research Council, 2007). HIV-risk behaviours included: sexual debut younger than age 15; three or more past-year sexual partners; more than half of sexual acts unprotected in past year; transactional sexual exploitation (sex in exchange for food, shelter, school fees, transport or money); and having a sexual partner more than five years older than the child.

Orphanhood/primary caregiver sickness status

The UN definition of orphanhood was used – i.e. loss of one or both biological parents (UNAIDS, 2004). In South Africa, death certificates are unreliable sources of AIDS mortality (WHO, 2011b) and clinical data is rarely available. Cause of parental death was therefore determined using the youth-report Verbal Autopsy method (Lopman et al., 2006), validated in previous studies of adult mortality in South Africa, with sensitivity of 89% and specificity 93% (Kahn, Tollman, Garenne, & Gear, 2000). Determination of AIDS-related parental death required a conservative threshold of three or more AIDS-defining illnesses; e.g. Kaposi's sarcoma or shingles. Where possible, reports were corroborated by teachers, social workers, and surviving parents. It is important to note that many orphaned children live with either a surviving parent, relative or foster carer who may themselves be unwell with HIV/AIDS. For current sickness amongst children's parents or primary caregivers, self-report is also unreliable due to low levels of HIV testing, with an estimated one-third of HIV-positive South Africans aware of their status (Peltzer, Matseke, Mzolo, & Majaja, 2009). Parental AIDS-illness was thus determined using a verbal symptom checklist, parallel to the Verbal Autopsy method, and intended to define Stage 4 AIDS-illness through identification of HIV-related opportunistic infections such as recurrent diarrhoea, oral candidiasis, and jaundice. In this study, determination of parental AIDS-illness required either (i) a conservative threshold of three or more AIDS-defining illnesses or (ii) parent self-identification of symptomatic HIV/AIDS or CD4 count less than 300.

Hypothesized intervening variables

All potential intervening variables were identified by literature review as linked to HIV/AIDS and/or child outcomes. a) *Poverty* was measured using two scales from the South African National Food Consumption Survey (Labadarios et al., 2003) assessing past-week hunger at the child level, as well as past-week insufficient food at the household level, and lack of any employment in the household; b) *Inability to afford school fees or uniform* was assessed using items from the National South African Social Attitudes survey

(Pillay, Roberts, & Rule, 2006); c) *Child abuse* used UNICEF scales for sub-Saharan Africa, with conservative cut-offs for severe abuse (Snider & Dawes, 2006): Physical abuse was weekly or more frequent beating with an object or deliberate harm; Emotional abuse was weekly or more frequent exposure to verbal abuse, threats to evict children from home, or invoking evil spirits against children; Sexual abuse was unwanted genital contact or rape; Exposure to domestic conflict was measured by past-week verbal violence and exposure to domestic violence was measured by past-week physical violence between adults in the household; d) *AIDS-related stigma* was measured using a 10-item Stigma-by-Association Scale, adapted from the HIV Stigma-by-Association Scale (Mason, Berger, Ferrans, Sultzman, & Fendrich, 2010), and validated in South Africa (Boyes, Mason, & Cluver, 2013). Reliability in the current sample was $\alpha = 0.87$; e) *Community violence* was measured using items from the Child Exposure to Community Violence Checklist (Richters & Martinez, 1993), adapted to reflect commonest community traumas for children in South Africa, as identified by national police statistics (SAPS Strategic Management, 2005): assault, robbery and witnessing of stabbings and shootings; f) *Child sickness* used clinical symptom checklists for common childhood causes of morbidity: colds or flu, body pains, worms, rashes, respiratory tract infections, vomiting or diarrhoea and burns or injuries; g) *Parental disability* used the WHO International Classification of Functioning, Disability and Health (WHO, 2003), mobility and self-care scales ($\alpha = 0.95$); h) *Child housework* was measured using the Becker 'Young Carers Tasks and Outcomes Questionnaire', adapted for the context of sub-Saharan Africa ($\alpha = 0.66$) (Becker, 2009); i) *Pregnancy* was asked alongside the sexual risk items, and answers stored in a separate envelope, which children sealed themselves; j) *Number of unwell adults in a household*; k) *number of moves between primary caregivers*; l) *formal/informal housing* and m) *child migration* were measured using items from the Demographic and Health Survey (Department of Health & Medical Research Council, 2007).

Sociodemographic factors of child gender and child age were measured using census items (Statistics South Africa, 2001).

Analysis strategy

Due to the lack of complex models in the literature on AIDS-affected children, a hypothesized model was not proposed. Instead, a sequential model-building process was followed (Kline, 2005), in four steps. First, confirmatory factor analyses were conducted in order to identify latent constructs for all three outcomes – psychological distress, educational risks, and sexual health risks – and for all hypothesised intervening factors identified in the literature. Second, in order to indicate which potential intervening factors to include in each model, a series of multivariate regression analyses, controlling for child age and gender, were conducted. Variables predicted by either AIDS-orphanhood or having an AIDS-unwell parent, which were also associated with a given outcome, were included in models. Third, a model containing all potential pathways was tested for each of three outcomes independently. Non-significant pathways were then dropped and the resulting models re-analysed (Byrne, 2010). Given the large sample size, pathways that were significant but with standardised β weights <0.05 were also dropped from the models (Davis, 1985). This resulted in a final model for each of the three outcomes. Finally, a unified model including psychological, educational, and sexual risk outcomes simultaneously was tested. All models controlled for child gender and age.

Analyses were conducted in AMOS 19 using maximum likelihood estimation. As some variables were non-normally distributed, all parameters were estimated using the bootstrapping procedure available in AMOS (using 1000 bootstrapped samples). Error terms

were only allowed to correlate within latent constructs, apart from pregnancy and sexual risk behaviours. Model fit was evaluated using the mean chi-square statistic obtained from the bootstrapped sampling distributions (mean χ^2) and the associated Bollen–Stine statistic (which should be non-significant); however, both χ^2 and the Bollen–Stine statistic are sensitive to sample size. Therefore χ^2 divided by its degrees of freedom (mean χ^2/df) is also reported. The maximum acceptable value of χ^2/df is 3; however, a value of near 1 is considered a sign of good fit (Blunch, 2008). Additionally, RMSEA, SRMR, and CFI are all reported. For RMSEA and SRMR a value of 0.05 or less indicates good fit and a value of 0.08 or less indicates adequate fit. For CFI a value of 0.95 or greater indicates good fit and a value of 0.90 or greater indicates adequate fit (Blunch, 2008).

Results

Descriptive statistics for all socio-demographic variables and all observed scores used to create latent constructs (for both intervening and outcome variables) are summarised in Table 1. Univariate ANOVAs and Chi-square tests were used to determine whether children who were AIDS-orphaned and children with AIDS-unwell parents differed from the general population. Amongst AIDS-orphaned children in this sample, 54% were maternally bereaved, 22% were double orphans (i.e. both maternally and paternally bereaved) and 35% lived with a surviving, AIDS-unwell parent. Amongst other-orphaned children, 32% were maternally bereaved and 12% were double orphans. With regard to the outcomes of

interest in this paper, both AIDS-orphaned children and children with AIDS-unwell parents showed higher levels of all psychological distress outcomes, concentration problems, unprotected sex and transactional sex than other children. AIDS-orphaned children showed more grade delay, and those with AIDS-unwell parents showed higher non-enrolment and school absence, and more early sexual debut and older sexual partners.

There were also gender differences on many of the variables used to identify latent outcome measurements (Table 2), with girls reporting more mental health and concentration problems, transactional sex and older sexual partners, and boys reporting more grade delay, early debut and multiple sexual partners. Risk of all negative outcomes increased with child age. In light of these, gender and age was controlled for in all models.

Measurement models

Confirmatory factor analyses were conducted to identify latent constructs (and confirm the measurement models) for all outcomes and hypothesised interveners included in the structural models. *Outcomes:* The psychological distress latent factor was identified by total scores for depression, suicidality, anxiety and post-traumatic stress. The educational risks latent factor was identified by school non-enrolment, being two or more years behind their expected grade, long-term school absences, and concentration problems at school. The sexual health risks latent factor was identified by sexual debut under 15 years old, unprotected sexual acts, having had three

Table 1
Summary descriptive statistics (means, standard deviations in parentheses, and proportions) for all sociodemographic characteristics and variables used to identify latent constructs in structural models.

	Full sample (n = 5998)	AIDS-orphaned (n = 811)	Not AIDS-orphaned (n = 5187)	p	AIDS-sick parent (n = 1530)	Parent not AIDS-sick (n = 4468)	p
Sociodemographic variables							
Age	13.50 (2.18)	14.13 (2.04)	13.41 (2.19)	<0.001	13.55 (2.14)	13.50 (2.20)	0.432
Gender (% male)	44.4%	43.2%	44.6%	0.450	39.5%	46%	<0.001
Outcome variables							
Depression	1.41 (2.23)	1.70 (2.47)	1.29 (2.13)	<0.001	2.09 (2.73)	1.10 (1.92)	<0.001
Anxiety	3.77 (3.45)	4.37 (3.75)	3.50 (3.36)	<0.001	4.93 (3.61)	3.20 (3.26)	<0.001
PTSD	11.85 (13.22)	15.51 (14.64)	11.16 (12.87)	<0.001	15.88 (16.33)	10.44 (12.44)	<0.001
Suicidal	5%	8.1%	4.6%	<0.001	7.1%	4.3%	<0.001
School non-enrolment	1.8%	1.8%	1.8%	0.972	2.7%	1.5%	0.004
Long term school absence	3.7%	4.6%	3.5%	0.139	4.8%	3.2%	0.004
2+ Years behind grade	10.9%	13.2%	10.6%	0.029	11.4%	10.8%	0.467
Concentration problems	21.1%	24.3%	20.6%	0.016	29.4%	18.2%	<0.001
Sexual debut < 15 years	5.8%	6.5%	5.7%	0.330	7.4%	5.2%	0.002
3+ Sexual partners	3.6%	4.4%	3.4%	0.142	4.2%	3.3%	0.096
Unprotected sex	6.3%	8.5%	6.0%	0.006	8.6%	5.6%	<0.001
Transactional sex	1.6%	2.7%	1.4%	0.006	3.0%	1.1%	<0.001
Sexual partner > 5 years older	1.8%	2.3%	1.8%	0.246	2.9%	1.5%	<0.001
Hypothesized intervening variables							
Hunger (household level)	19.5%	26.2%	18.4%	<0.001	32.5%	15.0%	<0.001
Hunger (individual level)	10.2%	14.0%	9.7%	<0.001	17.2%	7.9%	<0.001
Employment in household	64.9%	58.9%	65.9%	<0.001	62.7%	65.7%	0.034
Can't afford school uniform/fees	17.5%	20.6%	17.0%	0.046	25.7%	14.7%	<0.001
Live in informal housing	23.0%	18.9%	25.9%	<0.001	28.7%	23.6%	<0.001
Physical abuse	14.7%	16.2%	14.5%	0.206	21.0%	12.6%	<0.001
Emotional abuse	9.1%	9.2%	9.1%	0.928	14.1%	7.5%	<0.001
Sexual abuse	3.6%	5.2%	3.4%	0.012	5.4%	3.0%	<0.001
Domestic violence	5.6%	4.8%	5.7%	0.281	8.6%	4.6%	<0.001
Stigma	1.07 (2.46)	1.86 (3.05)	0.87 (2.24)	<0.001	1.91 (3.24)	0.73 (1.96)	<0.001
Community violence (number of events)	0.47 (0.74)	0.43 (0.73)	0.44 (0.71)	0.840	0.58 (0.81)	0.39 (0.68)	<0.001
Child sickness (number of symptoms)	2.01 (1.63)	2.15 (1.75)	1.94 (1.61)	0.001	2.40 (1.64)	1.83 (1.60)	<0.001
Parental disability	2.06 (3.99)	2.53 (4.35)	1.99 (3.96)	<0.001	4.88 (4.94)	1.17 (3.20)	<0.001
Child housework (number of tasks)	17.14 (11.92)	18.35 (12.81)	16.73 (11.70)	<0.001	22.96 (14.26)	15.04 (10.29)	<0.001
Number of sick adults in household	0.49 (0.62)	0.54 (0.65)	0.46 (0.61)	<0.001	0.99 (0.53)	0.30 (0.54)	<0.001
Number of homes child has lived in	1.25 (0.85)	1.37 (0.87)	1.26 (0.82)	<0.001	1.25 (0.87)	1.29 (0.81)	0.135

Notes: Significant p values are bolded. p Values associated with univariate ANOVAs or chi square tests.

Table 2

Summary descriptive statistics (means, standard deviations in parentheses, and proportions) as a function of child gender and age for all variables used to identify latent outcomes in structural models.

	Male	Female	<i>p</i>	Younger children	Older children	<i>p</i>
Depression	1.18 (1.99)	1.47 (2.32)	<0.001	1.10 (1.87)	1.59 (2.44)	<0.001
Anxiety	3.33 (3.23)	3.86 (3.57)	<0.001	3.34 (3.30)	3.91 (3.54)	<0.001
PTSD	10.76 (12.53)	12.55 (12.68)	<0.001	10.11 (12.21)	13.43 (13.96)	<0.001
Suicidal	3.2%	6.5%	<0.001	2.6%	7.6%	<0.001
School non-enrolment	1.7%	1.9%	0.584	0.6%	3.1%	<0.001
School non-attendance	3.8%	3.5%	0.598	1.9%	5.5%	<0.001
2+ Years behind grade	14.0%	8.1%	<0.001	8.6%	13.4%	<0.001
Concentration problems	19.7%	22.1%	0.023	19.0%	23.1%	<0.001
Sexual debut < 15 years	7.4%	4.5%	<0.001	1.9%	9.8%	<0.001
3+ Sexual partners	5.6%	1.9%	<0.001	0.6%	6.6%	<0.001
Unprotected sex	6.2%	6.4%	0.727	1.5%	11.3%	<0.001
Transactional sex	1.0%	2.0%	0.001	0.1%	3.1%	<0.001
Sexual partner > 5 years older	0.9%	2.5%	<0.001	0.4%	3.3%	<0.001

Notes: Significant *p* values are bolded. *p* Values associated with univariate ANOVAs or chi square tests. Age categories based on median split (median age = 13).

or more sexual partners in the past year, having a sexual partner more than five years older, and having transactional sex. *Interveners*: The child abuse latent factor was identified by exposure to severe physical, emotional, and sexual abuse, as well as domestic conflict and domestic violence in the household. The latent factor for poverty was identified by child-level and household-level food insecurity and household unemployment. All other latent factors were identified by the full sets of items in the relevant scales. Inability to afford school fees or uniform, and pregnancy were retained as observed variables. Overall, fit statistics for all measurement models were adequate to good (Table 3).

Structural models

Individual structural models were analysed for each outcome independently (psychological distress, education risks, sexual health risks), shown in Fig. 1. All hypothesized intervening variables were entered into models and those not providing significant pathways were removed. Associations with standardised $\beta > 0.10$ are shown as bold arrows. The associations and coefficients among the predictors and interveners were noted to be closely similar across the three separate models. Therefore, a final model testing relationships between the three outcomes simultaneously was conducted. This model is presented in Fig. 2, and accounted for 85% of the variance in psychological distress, 15% of the variance in educational risks and 16% of the variance in sexual health risks respectively. All models controlled for child age and gender.

The fit of the final model was: mean $\chi^2(831) = 1191.14$; Bollen–Stine $p = 0.001$; mean $\chi^2/df = 1.43$; RMSEA 0.031; SRMR 0.036; CFI 0.944; TLI 0.936). With the exception of the Bollen–Stine statistic (which was anticipated to be significant due to large sample size) all fit statistics were either in the good or acceptable range. There were no direct effects of either AIDS-orphanhood or parental AIDS-illness on any of the three outcomes. However, the total indirect effect of parental AIDS-illness was significant for all outcomes of

psychological distress, educational risks and sexual health risks: $\beta = 0.28$ ($p < 0.002$), $\beta = 0.07$ ($p < 0.001$), and $\beta = 0.07$ ($p < 0.002$) respectively. The total indirect effects of AIDS-orphanhood onto the three outcomes were also significant: $\beta = 0.06$ ($p < 0.001$), $\beta = 0.02$ ($p < 0.001$), and $\beta = 0.01$ ($p < 0.001$) respectively. All regression coefficients are adjusted for gender and age. Female gender was significantly linked to more stigma, abuse, and pregnancy, and male gender to more community violence exposure. Older age was significantly associated with more sexual risk, psychological distress, and community violence. For simplicity of presentation, gender and age are omitted from Fig. 2.

The final model shows that the pathways from parental HIV/AIDS illness and death to child developmental outcomes are not direct, but proceed via multiple interveners. The following are noteworthy. Firstly, AIDS-orphanhood and parental AIDS-sickness are directly associated with increased AIDS-related stigma, which in turn raises likelihood of child abuse in families, leading to child psychological distress. Secondly, parental AIDS-sickness (strongly) and AIDS-orphanhood (less strongly) are associated with increased poverty. This in turn has strong onward associations with stigma; and also with increased abuse, inability to afford school fees or uniform, and community violence. These factors interlink in different ways to increased risk of psychological distress, education risks and sexual health risks. Thirdly, the child outcomes are linked among themselves and with increased levels of pregnancy. In particular, psychological distress functions not only as an outcome but also as an intervening variable to educational and sexual health risks.

Discussion

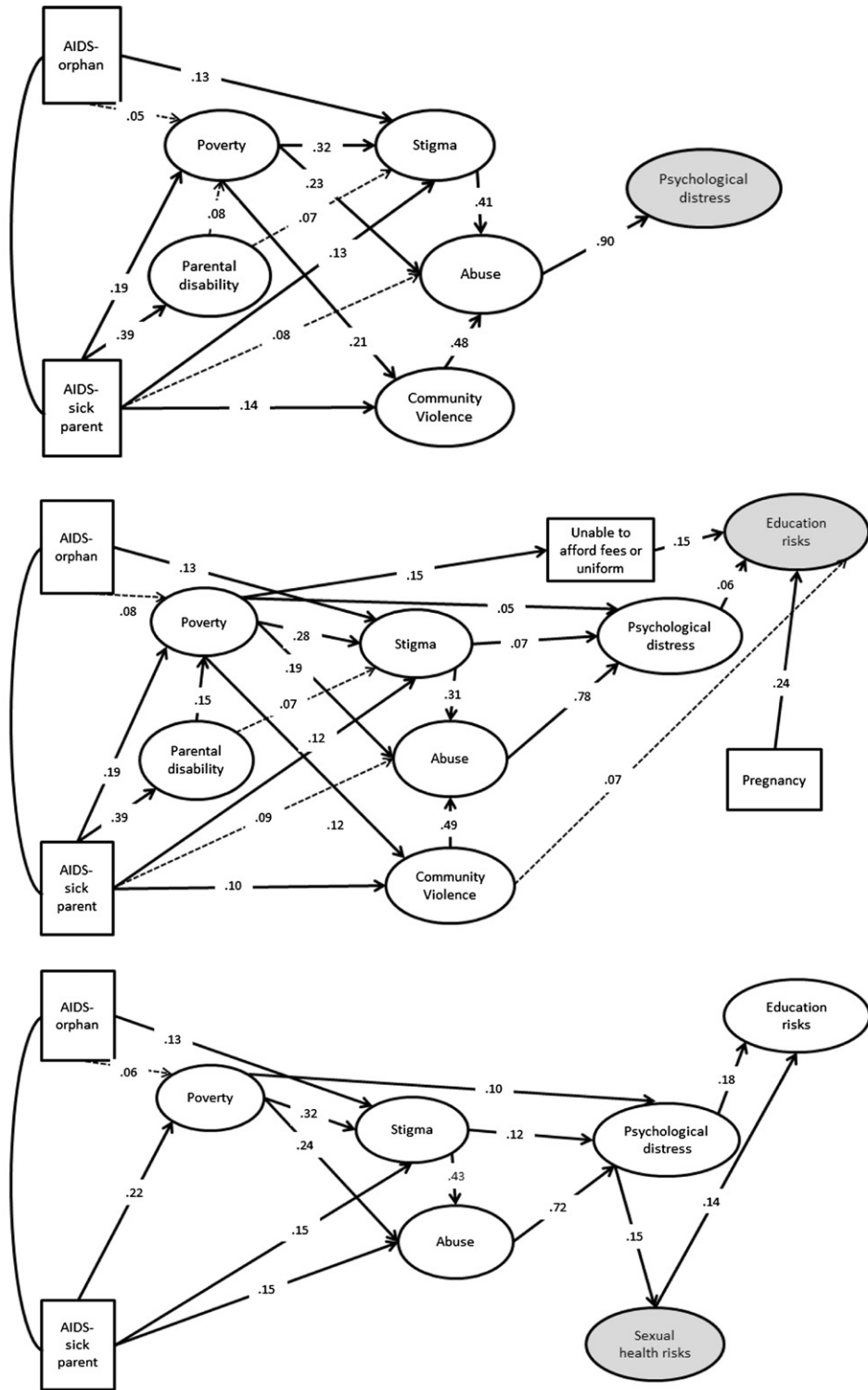
The empirical model developed in this study supports an interactive theory of multiple pathways between parental AIDS-illness, AIDS-orphanhood and negative child outcomes. This is consistent with the interactive/transactional frameworks for other child risks which have been demonstrated in the developed world literature. Models showed no direct relationship from AIDS-orphanhood or parental AIDS-sickness to child psychological distress, educational or sexual health risks. Instead, they showed that parental AIDS illness and death impact on child outcomes *via* a set of factors: parental disability, poverty, community violence, HIV/AIDS-related stigma, and child abuse. These effects show a remarkable consistency of pattern across the different child outcomes, with factors forming interlinked pathways.

Parental AIDS illness and AIDS-orphanhood showed stronger impacts on the more proximal outcome of psychological distress than on the more distal outcomes of educational and sexual health risks, underlining the importance of psychological health as an

Table 3

Summary of fit statistics for the measurement models.

Measurement model	Mean χ^2	Bollen–Stine	Mean χ^2/df	RMSEA	SRMR	CFI	TLI
Psychological distress	3.07	0.032	1.54	0.028	0.008	0.998	0.993
Educational risks	8.30	0.020	4.12	0.021	0.008	0.998	0.993
Sexual risk	11.38	0.437	11.38	0.032	0.007	0.994	0.999
Abuse	9.62	0.001	2.41	0.017	0.061	0.912	0.966
Poverty	2.12	0.001	1.06	0.034	0.013	0.989	0.996
Stigma	20.19	0.399	4.04	0.023	0.006	0.997	0.999
Parental disability	24.99	0.200	5.00	0.032	0.006	0.997	0.999
Community violence	2.81	0.001	1.41	0.053	0.021	0.925	0.975



	mean χ^2 (679)	Bollen-Stine	mean χ^2/df	RMSEA	SRMR	CFI	TLI
Psychological distress	881.84	p=.001	1.30	.028	.027	.960	.934
Educational risks	887.68	p=.001	1.36	.031	.039	.951	.945
Sexual health risks	640.06	p=.001	1.57	.032	.044	.939	.930

Fig. 1. Final estimates (and fit statistics) for the outcomes when modelled independently.

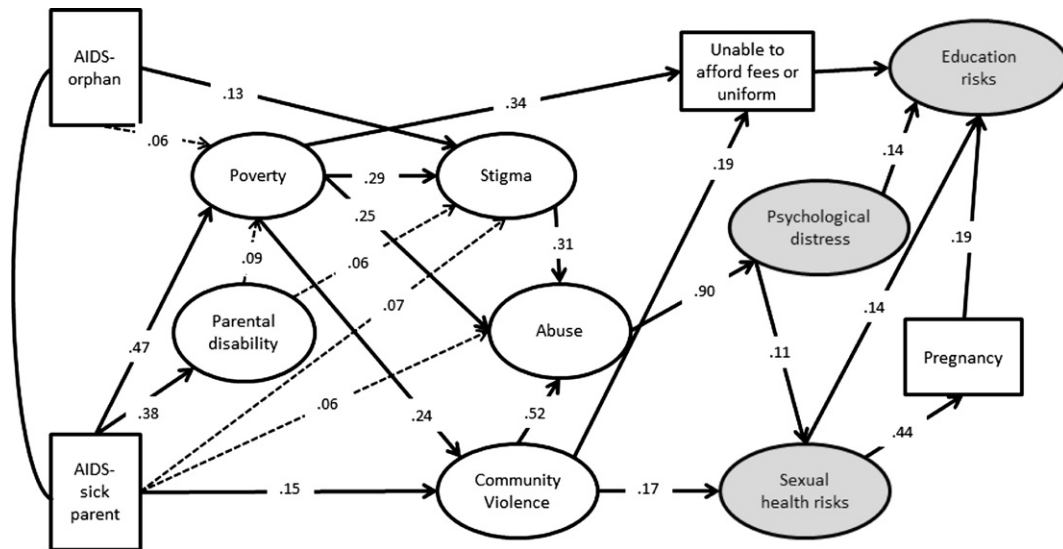


Fig. 2. Final estimates for the outcomes when modelled simultaneously.

intervening factor in other child outcomes. It is also noted that, in all models, parental AIDS-illness had stronger effects and more risk pathways than AIDS-orphanhood, especially via poverty and parental disability. This supports evidence of the severe economic shocks experienced in households where adults are not only unable to work, but also require high levels of medical care (Richter et al., 2009). No known studies have investigated links between familial AIDS and increased levels of child abuse and child exposure to community violence, but empirically-tested theories from the developed world suggest that child physical and emotional abuse are linked to parenting under circumstances of extreme stress, and that child sexual abuse and exposure to community violence may be related to reduced parental capacity to supervise children (Belsky, 1993). It is possible that such situations may be exacerbated by the stress and stigma of AIDS, and the challenges of childcare in circumstances of disability and death.

Given the high levels of overlap between parental AIDS-illness and AIDS-orphanhood, the stronger effects associated with parental AIDS-illness suggests that many of the observed risks associated with AIDS-orphanhood in the research literature are likely to have started during the stage of parental illness. Richter et al. (2006) highlight that 'the impact of chronic parental illness on children is one of the most poorly understood and neglected difficulties faced by AIDS-affected children'. Further research is clearly needed to examine associations between AIDS-orphanhood and parental AIDS-illness and concomitant child outcomes.

In addition, these findings demonstrate that parental AIDS has differential impacts on children by gender and age. In particular, female gender and older child age were associated with increased psychological and sexual risks, with close linkages via AIDS-related stigma, abuse and pregnancy. These findings support the importance of programming that targets pathways of vulnerability for adolescent girls, who have been identified as a 'key population' in the UNAIDS Investment Framework (UNAIDS, 2011). In addition, the linkages shown in this study underline the heightened vulnerability of girls living in AIDS-affected families, and future research and programming could valuably focus on risk pathways for AIDS-affected adolescent girls.

To our knowledge, this is the first study to test multiple pathways from parental HIV/AIDS and AIDS-orphanhood to child outcomes. Because of the lack of prior tested models, this research is limited to empirical model-building. Future research is now needed to evaluate

this model using confirmatory techniques, and preferably in countries additional to South Africa. It is to be noted that the current study has several potential limitations. Firstly, causality cannot be determined using cross-sectional data. However, the imputation of causal order on plausible theoretical grounds is well preceded in structural equation modelling (Davis, 1985), and for most of the risk pathways, reverse causality was extremely unlikely (for example, child psychological distress does not cause parental death by AIDS). But for some of these pathways it is possible that causality was bidirectional, for example the linkages between lack of educational access and child sexual risks. In particular, the causal or cyclical relationship between poverty and family AIDS needs to be better understood. Whilst there is strong evidence that AIDS and AIDS-related deaths deepen household poverty (Bachmann & Booyesen, 2003), there is also evidence that poverty is a driver of HIV-infection, and accelerates progression to illness and death. Thus, following Rutter's longitudinal theoretical models of risk, 'poverty' could appear both as a causal factor in parental AIDS-illness and death, and subsequently, as a link to further risks. Further longitudinal research and depth qualitative work will be important in fully understanding the pathways that link risks of parental infection, and child outcomes in the context of parental illness and death.

Secondly, the randomly selected communities in this study did not include any prisons or children's institutions, nor did they include street-children, who usually live in inner-city areas: It is important to note that these children may show even greater vulnerabilities, and further research should examine whether similar pathways exist for AIDS-affected children in such settings. Thirdly, all analyses controlled for child gender and age. Further research could examine differentiated models by child gender and age, and risk pathways for subgroups such as adolescent girls or younger children. Fourthly, the study did not measure whether children were themselves infected with HIV. Only a very small proportion of this sample would have been perinatally HIV-infected, due to birth dates 4–14 years before the beginning of the antiretroviral rollout in South Africa and low rates of survival in the pre-antiretroviral period (Newell et al., 2004), but others could have been infected during childhood. Future research on pathways of risk and resilience for HIV-positive children would be of great value.

Fifthly, this study focused on the potential negative effects for children related to parental AIDS-death or symptomatic AIDS-illness. The study did not test for impacts of parental or primary caregiver

HIV-positive status, which may be pre-symptomatic, or asymptomatic due to good treatment access and adherence. It is likely, therefore, that AIDS-unwell parents in this study are either amongst the 48% of ART-eligible South Africans not accessing treatment (Johnson, 2012), those not adhering to treatment, or those who have experienced treatment failure, and the particular vulnerabilities of this group should be considered. Lastly, this study only includes pathways of risk. Whilst many of these risks can be conceptualised in the reverse as protective factors (i.e. wealth, functional parenting, psychological well-being), it will be of great importance for future theoretical research to identify potential protective pathways, or pathways of resilience, for AIDS-affected children (Betancourt, Meyers-Ohki, Charrow & Hansen, 2012; Skovdal, Ogotu, Aoro, & Campbell, 2010).

Findings of this study can provide a valuable contribution in the development of HIV-sensitive interventions for children. A central aim of all ecological theories of child risk is to inform programming: by intervening to improve modifiable risk factors, there is potential to buffer the negative impacts of less-modifiable factors in a child's life (Bronfenbrenner, 1979). This has implications for programmes aiming to ameliorate the effects of parental HIV/AIDS. Whilst some risk factors such as stigma require further intervention development and research if we are to address them (Sengupta, Banks, Jonas, Miles, & Smith, 2011), many of the risk factors identified in our model have good evidence for successful preventive or responsive interventions. There is strong evidence for effectiveness of income transfers to reduce poverty in families affected by illness and death (Adato & Bassett, 2008; Arnold, Conway, & Greenslade, 2011), and for provision of free schooling, uniforms and transport to increase education access for the poor (Lewin & Sabates, 2012). In addition, two recent World Bank studies of cash transfers to families in Malawi (Baird, Garfein, McIntosh, & Ozler, 2012) and youth in Tanzania (de Walque et al., 2012) have demonstrated reductions in age of sexual partners, HIV and STI incidence amongst participating youth. With accompanying caution regarding ethical and programmatic issues related to conditional and unconditional cash transfers (Lutz and Small, UNDP <http://www.youtube.com/watch?v=W-QueBSEigcw>) increasingly policy frameworks such as UNICEF's HIV-sensitive social protection recognise the potential for effective poverty reduction to impact youth HIV risks.

In addition to poverty reduction, there is now scientific consensus on the capacity of anti-retroviral medication to reduce parental HIV/AIDS-related disability (Ena & Pasquau, 2003), and the importance of access to clinical and home-based palliative care for people living with chronic HIV/AIDS illness. Multiple studies show reductions in community violence associated with social development programmes, and emerging evidence suggests positive impacts of alcohol regulation and firearm restrictions (WHO, 2010). Lastly, systematic reviews in the developed world, and a very small but increasing body of evidence in the developing world demonstrate effectiveness of home visiting and parenting programmes in reducing and preventing child abuse (Knerr, Gardner, & Cluver, in press; Mikton & Butchart, 2009). For such interventions to mitigate the impacts of AIDS on children, it is essential that they adequately reach those who are AIDS-orphaned and those living with AIDS-unwell parents or primary caregivers.

This detailed study clearly elaborates the complexity of pathways of both effect and potential child outcomes. It validates the need for empirically-tested theoretical models to underpin tracking and interventions for children and it points out a set of clear factors where interventions are forcefully indicated and where there is a sound evidence base from other areas of efficacy. Although HIV may be beyond imminent reach of cure, its negative outcome spiral can clearly be tracked, and in doing so can identify interventions and diversions which may enhance child outcomes in the face of illness and death.

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