AIDS Care
Vol. 22, No. 8, August 2010, 927–938

Routledge
Taylor & Francis Group

AIDSIMPACT SPECIAL ISSUE 2010

Sexual risk among orphaned adolescents: is country-level HIV prevalence an important factor?

Laura Robertson[a]*, Simon Gregson[a,b] and Geoff P. Garnett[a]

[a]*Department of Infectious Disease Epidemiology, Imperial College London, St. Mary's Campus, Norfolk Place, London W2 1PG, UK;* [b]*Biomedical Research & Training Institute, Harare, Zimbabwe*

(Received 23 October 2009; final version received 5 March 2010)

Previous studies from sub-Saharan Africa have found that orphans experience increased sexual risk compared to non-orphans. We developed a theoretical framework for the investigation of determinants of HIV risk and used it to generate specific hypotheses regarding the effect of country-level HIV prevalence on the sexual risk experience of orphans. We expected that countries with high HIV prevalence would experience a higher prevalence of orphanhood. We further hypothesised that orphans in countries with high HIV prevalence would experience increased sexual risk, compared to non-orphans, due to pressure on the extended family network, which is primarily responsible for the care of orphans in sub-Saharan Africa, resulting in poorer standards of care and guidance. We used hierarchical logistic regression models to investigate this hypothesis using cross-sectional, Demographic and Health Survey data from 10 sub-Saharan African countries. We found that countries with high HIV prevalence did indeed have higher prevalence of orphanhood. We also found that, amongst female adolescents, maternal and double orphans were significantly more likely to have started sex than non-orphans in countries with high HIV prevalence but were not at increased risk in low HIV prevalence countries. This effect of country-level HIV prevalence on the sexual risk of orphans was not explained by household level factors such as wealth, overcrowding or age of the household head. The same pattern of risk was not observed for male adolescents – male orphans were not more likely to have started sex than non-orphans. This suggests that orphaned adolescent women are an important target group for HIV prevention and that efforts should be made to integrate prevention messages into existing support programmes for orphans and vulnerable children.

Keywords: orphans; HIV; adolescents; sexual behaviour; education

Introduction

Several studies have been published suggesting that orphans are at increased sexual health risk. The majority of these studies used cross-sectional data on female adolescents from southern Africa, particularly South Africa and Zimbabwe, where HIV prevalence is high (Birdthistle, Floyd, Machingura, Mudziwapasi, Gregson, & Glynn, 2008; Birdthistle, Floyd, Nyagadza, Mudziwapasi, Gregson, & Glynn, 2009; Gregson et al., 2005; Kang, Dunbar, Laver, & Padian, 2008). Three studies have used data on male and female adolescents from southern Africa (Nyamukapa et al., 2008; Operario, Pettifor, Cluver, MacPhail, & Rees, 2007; Thurman, Brown, Richter, Maharaj, & Magnani, 2006). These studies found that orphans were at increased risk of HIV infection (Birdthistle et al., 2008, 2009; Gregson et al., 2005; Kang et al., 2008; Operario et al., 2007), STI infection (Birdthistle et al., 2008, 2009), pregnancy (Gregson et al., 2005; Kang et al., 2008), sexual debut (Birdthistle et al., 2008, 2009; Gregson et al., 2005; Kang et al., 2008; Nyamukapa et al., 2008; Operario et al., 2007; Thurman et al., 2006), multiple partners, unprotected sex (Birdthistle et al., 2008; Operario et al., 2007), transactional sex and forced sex (Kang et al., 2008; Thurman et al., 2006). Some studies have found maternal and/or double orphans to be particularly at risk (Birdthistle et al., 2008; Gregson et al., 2005; Kang et al., 2008). The aim of this analysis was to investigate the association between orphanhood and sexual health risk using pooled data from across sub-Saharan Africa. These investigations were guided by the development of a new theoretical framework from which specific hypotheses regarding potential causal pathways were generated and tested.

Methods

Theoretical framework and hypotheses

A number of theoretical frameworks have been developed to aid investigation of the complex risk factors that influence sexual health and HIV risk.

*Corresponding author. Email: l.robertson06@imperial.ac.uk

ISSN 0954-0121 print/ISSN 1360-0451 online

DOI: 10.1080/09540121003758622
http://www.informaworld.com

Boerma and Weir (2005) developed the proximate determinants framework, which integrates epidemiological and demographic approaches. The proximate determinants (e.g., coital frequency, condom use, treatment with ARVs, etc.) provide a link between underlying social/environmental determinants (e.g., socio-economic variables, education) and biological determinants (e.g., duration of infectivity, efficiency of transmission per contact) of HIV risk. This framework has facilitated hypothesis generation regarding causal pathways between risk factors and HIV infection, and has been tested using data on adults from Zimbabwe (Lewis et al., 2007; Lopman et al., 2008). Poundstone, Strathdee, and Celentano (2004) developed a social epidemiological framework using an eco-social approach. They state that ''factors at multiple levels – from the microscopic to the societal'' contribute to the distribution of HIV. Mosley and Chen (2003) developed a conceptual framework for child survival, which has been adapted and tested using data from Zimbabwe (Watts et al., 2007). They suggest that underlying determinants of mortality (and morbidity) exist at three levels – the individual level, the household level and the community level. In our framework (see Figure 1) we have expanded Boerma and Weir (2005)'s proximate determinants framework to make explicit the country/regional, household and individual level, underlying determinants of HIV.

We used our theoretical framework to develop specific hypotheses regarding the relationship between orphanhood and sexual debut and HIV infection. We expect that the orphans, particularly maternal and double orphans, will experience increased sexual risk compared to non-orphans. We also expect that in high HIV prevalence countries there will be a greater burden of adult (parental) mortality and morbidity (Bicego, Rutstein, & Johnson, 2003; Hosegood et al., 2007; Monasch & Boerma, 2004). This will put strain on the extended family network that traditionally cares for orphaned children resulting in households being less able to provide adequate care to these children compared to households in countries with low HIV prevalence. Thus orphans living in countries with high HIV prevalence will receive a lower standard of care and guidance, which will put them at even greater risk of starting sex and becoming infected with HIV. We further hypothesise that the effect of country-level HIV prevalence on orphans' sexual risk will be mediated through household level variables that reflect the circumstances experienced by families affected by HIV e.g., female and elderly headed households, overcrowding and reduced socio-economic status. We tested these hypotheses using a data-set compiled from Demographic and Health Surveys (DHS) conducted in sub-Saharan African countries.

Due to its long incubation period there is a lag between HIV incidence and AIDS-related mortality. As a result, HIV prevalence may increase in a region but mortality can take some time to catch up (Bicego et al., 2003). Countries experiencing a mature epidemic may also experience a decline in prevalence due to increasing AIDS-related mortality. These issues are further complicated by the roll-out of anti-retroviral therapy, which will increase HIV prevalence and reduce mortality. It was therefore necessary to test our assumption that parental mortality (orphan prevalence) is higher in countries, from our sample, with higher HIV prevalence.

Recent studies have found that adolescents attending school experience lower sexual risk (Birdthistle et al., 2009; Gregson et al., 2005; Hargreaves et al., 2008; Nyamukapa et al., 2008). However, a study using data on adolescent women from urban Zimbabwe (Birdthistle et al., 2009) found that differences in school participation and completion did not explain the increased sexual risk experienced by orphans i.e., education did not seem to be on the causal pathway between orphan status and sexual risk. Other analyses of this data-set (Birdthistle et al., 2008, 2009) found that married orphans experience reduced sexual risk compared to unmarried orphans. We investigated both these findings in our data-set.

Data sources

DHS are cross-sectional, nationally representative surveys that have collected a large quantity of data from countries in sub-Saharan Africa. They use a stratified, two-stage cluster sampling design – enumeration areas (or sample clusters) are selected from census files and then a sample of households is selected from within each cluster. To ensure robust estimates of key demographic variables at urban/rural and province/regional level, some areas are oversampled in the surveys i.e., the surveys are not self-weighting. We extracted the data of adolescents aged 15–17 years from surveys that performed HIV testing and collected data on orphan status from children up to age 17 years. If a country had more than one survey meeting these criteria, the most recent was selected. Thus we included 10 surveys in our data-set – Democratic Republic of Congo 2007; Cote d'Ivoire 2005; Lesotho 2004; Liberia 2007; Malawi 2004; Rwanda 2005; Swaziland 2006; Tanzania 2003; Zambia 2007, Zimbabwe 2005/06. In some surveys, HIV testing was only carried out on a subsample of those surveyed. Data on country-level HIV

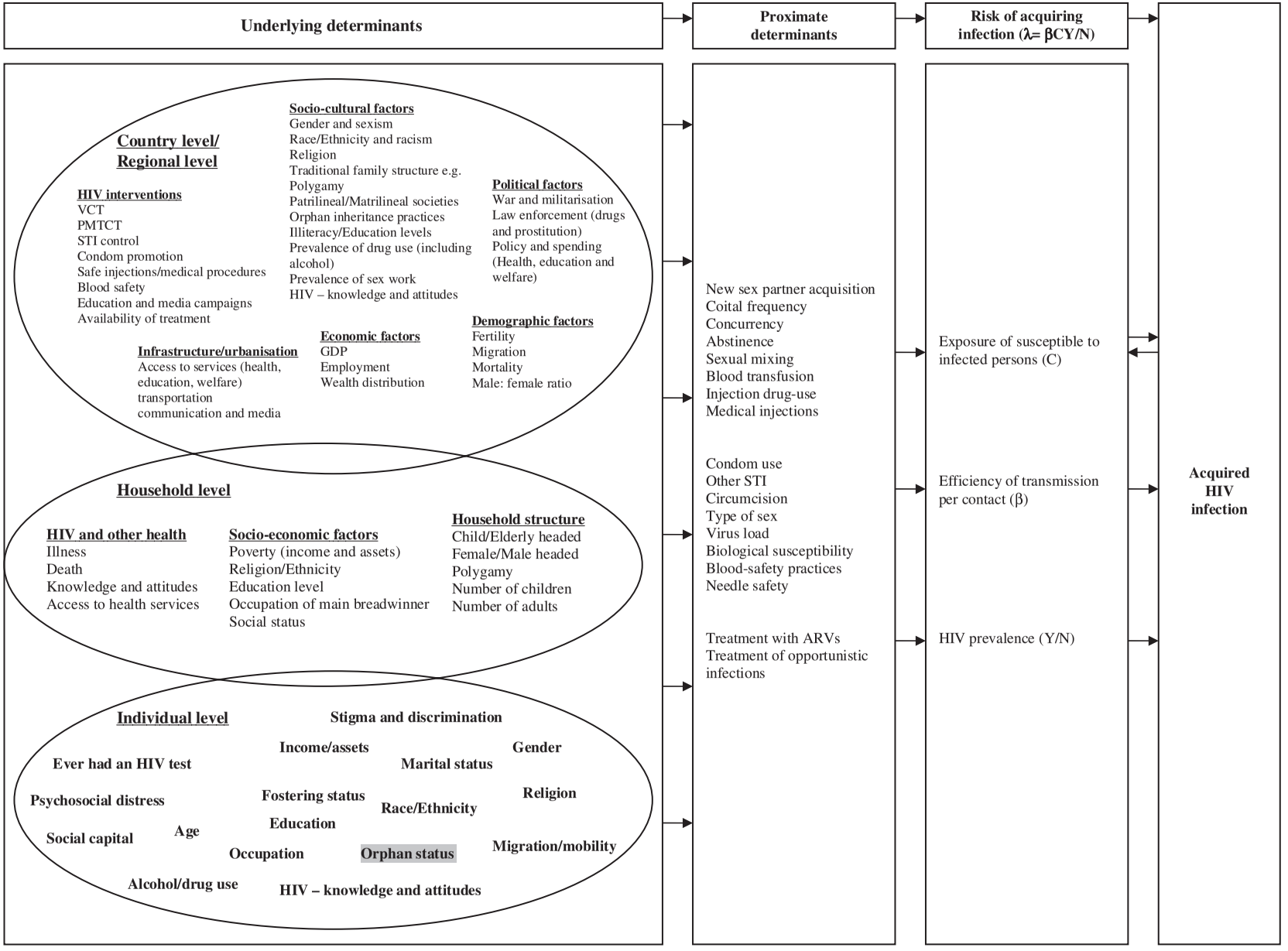

Figure 1. Theoretical framework.

prevalence were based on UNAIDS estimates from the year of the survey (UNAIDS, 2009). Data on HIV prevalence were not available for Demographic Republic of Congo so this country was excluded from analyses involving country-level HIV prevalence.

Definitions

The outcome indicators that we used to represent sexual risk were ''ever had sex?'' and HIV status. Our orphan status categories were defined as:

- non-orphan – both parents are alive;
- maternal orphan – mother deceased; father alive;
- paternal orphan – father deceased; mother alive;
- double orphan – both parents deceased.

DHS use principal components analysis to produce household wealth indices for each country's survey, based on data on household assets and utility. These are then used to divide the samples of households into wealth quintiles [described elsewhere (Rutstein & Johnson, 2004)]. Following HIV-related shocks, some aspects of household socio-economic status may be subject to change (e.g., household assets) whereas others will be unaffected (e.g., education level of the household head). We used the country-specific wealth quintiles in our analyses because we required a socio-economic measure that would be susceptible to change following HIV-related shocks. Data on marital status (ever married vs. never married), education status of the adolescent and household head (highest level of education achieved – none, primary or secondary and above), number of people in the household (five or less vs. more than five), migration status (two or more years in current area vs. less than two years) sex and age of household head (60 years or younger vs. over 60 years) and urban/rural region were also used in our analyses. To increase statistical efficiency, in examining the effect of country-level HIV prevalence, we split the countries in our sample into a low HIV prevalence group (less than 5%) and a high prevalence group (5% or higher).

Statistical analysis

We calculated the prevalence of orphanhood amongst 0–17-year olds for each country, applying appropriate sample weights and adjustments for DHS sampling design. We used these to investigate the correlation between country-level HIV prevalence and prevalence of orphanhood. Pearson correlation coefficients (PCCs) were calculated using Microsoft Excel 2007. The rest of this analysis was conducted using Stata 10.0 and was performed separately for male and female adolescents.

Hierarchical (multi-level) statistical modelling is used to analyse nested data where different levels of variability are associated with each level of nesting (Rabe-Hesketh & Skrondal, 2005; Twisk, 2006). There were four levels of hierarchy in our data-set – country, sample cluster, household and individual. Orphan status and our outcome variables were individual level variables so we had to have an individual level in all models. Since we had pooled data from a number of countries and due to the DHS sampling design, there was likely to be variation with respect to the outcomes (and possibly the effect of orphan status on those outcomes) at the country and sample cluster level. For this reason we also considered these levels in our models. Over 90% of households contributed only one adolescent, of each sex, to our data-set. Adding this fourth (household) level to the models, especially given the large data-set and the small number of households with two adolescents, would have made the models difficult, if not impossible, to run. In light of this, we selected one child at random from each household where more than one child was eligible for inclusion in the analysis. This removed the need for household to be included as a level in the models.

Hierarchical, logistic regression models were developed separately for each outcome – ''ever had sex?'' and HIV status. Random intercepts were added to the models at the country level to investigate the amount of variation in the outcome variables at this level. Since it was not possible, in Stata 10.0, to compare a ''naïve'' logistic regression model with a hierarchical logistic regression model using likelihood ratio tests, we assessed the importance of allowing the outcome variables to vary across the countries by comparing the variance of the country-level intercepts with the standard error of these variances. If the variance is more than double the standard error, the variance should be considered important (Twisk, 2006). Random intercepts at the sample cluster level were then added to the models to investigate the variation in the outcome variables at this level. Likelihood ratio tests were used to compare these models to the models with only country-level random intercepts and to assess the importance of including the sample cluster level intercepts in the models.

The hierarchical logistic regression models with random intercepts at the cluster- and country-level were computationally intensive and time consuming to run and, when investigating our hypotheses, some of the models would not run. We therefore simplified our models when investigating our hypotheses. A random sample of one orphan per sample cluster was

selected and a random sample of non-orphans, matched on sample cluster, was selected as a comparison group. This removed the need for sample cluster to be included as a level in the model i.e., the matching procedure was necessary because of the underlying structure of our data-set. The rest of the analysis was conducted on this matched data-set.

Age-adjusted, logistic regression models with a random intercept at the country level were then used to investigate the effects of orphan status on each of the outcomes and the possible confounding of this effect by education status and marital status. We used likelihood ratio tests to investigate possible interactions, with respect to our outcome variables, between orphan status and country-level HIV prevalence, education status and marital status. We also investigated whether the inclusion, in the models, of household level indicators that reflect some of the effects of HIV-related shocks would modify the interaction between orphan status and country-level HIV prevalence. If the evidence of an interaction disappeared it would suggest that the household level variables were on the causal pathway between country-level HIV prevalence and increased sexual risk among orphans (Lewis et al., 2007; Twisk, 2006). Finally, we added a random coefficient (slope) for orphan status at the country level and used likelihood ratio tests to compare these models to models without a random slope. This allowed us to determine whether the effect of orphan status on our outcomes differed by country.

Results

The initial sample contained 9821 women and 6464 men aged 15–17 years. About 30.6% (3002/9799) of women and 26.8% (1731/6457) of men had started sex. About 2.5% (156/6219) of women and 1.4% (77/5546) of men were infected with HIV. Among women there was evidence that the proportion of adolescents that had started sex varied by country (ratio of variance to SE 2.21) and sample cluster ($p < 0.0001$). There was little evidence that the proportion infected with HIV varied by country (ratio of variance to SE 1.85) or sample cluster ($p = 0.106$), although the number of adolescent girls that were infected with HIV was small. Amongst men there was evidence that the proportion of adolescents that had started sex varied by country (ratio of variance to SE 2.06) and sample cluster ($p < 0.0001$). There was little evidence that the proportion infected with HIV varied by country (ratio of variance to SE 1.37), but there was some evidence that it varied by sample cluster ($p = 0.018$).

In our matched female sample, 1767 orphans were initially selected. The sample clusters were small, especially since only one eligible adolescent per household was included in the data-set. The clusters ranged in size from 1 to 15 female adolescents, with 44% containing only one or two adolescents. Thus matching non-orphans could not be found for 325 (18.4%) of the selected orphans. These orphans were dropped and the final sample contained 1442 orphans matched to 1442 non-orphans (total = 2884). Amongst the orphans, 18.4% were maternal orphans, 58.5% were paternal orphans and 23.2% were double orphans. About 28.2% had started sex and 3.3% were infected with HIV. Amongst non-orphans, 25.3% had started sex and 3.0% were infected with HIV.

The age-adjusted model shown in Table 1 indicates that female double orphans were at increased risk of starting sex (OR 1.45; 95% CI 1.08–1.92). This effect was reduced but remained borderline significant in the fully adjusted model (OR 1.34; 95% CI 1.00–1.84). Paternal and maternal orphans did not experience a significantly increased risk of having started sex. None of the orphan types experienced an increased risk of HIV infection, although the size and direction of the effect estimates were comparable to those in the sexual debut models. The number of infected women in the sample was small (56 infections). There was no evidence that the effect of orphan status, on either outcome, varied by country ($p > 0.1$ for all tests).

The effect of orphan status on having started sex was significantly modified by country-level HIV prevalence ($p = 0.045$; see Table 1). In countries with HIV prevalence greater than 5% (Cote d'Ivoire, Lesotho, Malawi, Swaziland, Tanzania, Zambia, Zimbabwe) there was evidence that maternal orphans (OR 1.61; 95% CI 1.13–2.29) and double orphans (OR 1.41; 95% CI 1.02–1.95), though not paternal orphans (OR 1.06; 95% CI 0.82–1.36) were at increased risk of having started sex (see Table 2 and Figure 2). However, in countries with HIV prevalence lower than 5% (Liberia, Rwanda), there was no evidence that orphan status was associated with increased risk of having started sex. The interaction remained significant ($p = 0.036$) after adjusting for household level variables (age and sex of the household head, number of people in the household and wealth quintile), which suggests that these variables were not on the causal pathway between country-level HIV prevalence and increased risk of starting sex amongst female orphans. It was not possible to perform the above analysis for our HIV infection outcome because the number of infections in the low HIV prevalence countries was too small (two infections).

Table 1. The effects of orphan status on sexual risk among female adolescents.

	Effect of orphan status on started sex (non-orphan $N = 1441$[c])					Effect of orphan status on HIV infection (Non-orphan $N = 914$[d])				
	N (Orphans)	OR	95% CI	*p*-value	Test of interaction (*p*-value)	*N* (Orphans)	OR	95% CI	*p*-value	Test of interaction (*p*-value)
Age adjusted models										
Maternal orphan vs. non-orphan	265	1.25	0.91–1.70	0.172		165	1.48	0.63–3.53	0.372	
Paternal orphan vs. non-orphan	843	1.09	0.88–1.35	0.428	–	522	0.81	0.40–1.62	0.547	–
Double orphan vs. non-orphan	334	1.45	1.08–1.92	0.011		202	1.57	0.74–3.35	0.240	
Fully adjusted models[b]										
Maternal orphan vs. non-orphan	234	1.11	0.79–1.57	0.564		138	1.07	0.38–3.03	0.889	
Paternal orphan vs. non-orphan	751	0.99	0.77–1.27	0.919	–	450	0.73	0.33–1.63	0.449	–
Double orphan vs. non-orphan	314	1.34	1.00–1.84	0.052		188	1.13	0.48–2.66	0.780	
Adjusted for age and education level										
Maternal orphan vs. non-orphan	265	1.22	0.89–1.67	0.217		165	1.46	0.61–3.48	0.395	
Paternal orphan vs. non-orphan	843	1.08	0.87–1.34	0.478	0.144	522	0.80	0.40–1.62	0.539	MA[a]
Double orphan vs. non-orphan	334	1.39	1.04–1.85	0.025		202	1.50	0.70–3.21	0.295	
Adjusted for age and marital status										
Maternal orphan vs. non-orphan	–	–	–	–		165	1.50	0.63–3.56	0.362	
Paternal orphan vs. non-orphan	–	–	–	–	–	522	0.81	0.41–1.64	0.564	0.179
Double orphan vs. non-orphan	–	–	–	–		202	1.56	0.73–3.32	0.251	
Adjusted for age and country HIV prevalence										
Maternal orphan vs. non-orphan	234	–	–	–		148	–	–	–	
Paternal orphan vs. non-orphan	774	–	–	–	0.045	498	–	–	–	MA[a]
Double orphan vs. non-orphan	304	–	–	–		188	–	–	–	

[a]Maximisation aborted.

[b]In the "ever has sex?" model we adjusted for age; wealth quintile; education level; migration status; urban/rural region; number of people in the household; age; sex; and education level of the household head. In the HIV infection model we also adjusted for material status.

[c]In the fully adjusted model non-orphan $N = 1291$. In the country HIV prevalence model non-orphan $N = 1311$.

[d]In the fully adjusted model non-orphan $N = 792$. In the country HIV prevalence model non-orphan $N = 855$.

Note: –, No attempt was made to run the model. For the marital status model this was because all married individuals had started sex and for the HIV prevalence models this was because country-level HIV prevalence could not be on the casual pathway between orphan status and risk of starting and risk of starting sex or HIV infection.

Table 2. Age adjusted effects of orphan status on risk of having started sex amongst female adolescents by country HIV prevalence.

	Started sex			
	N (Orphans)	OR	95% CI	*p*-Value
HIV prevalence $<5\%$[a] (non-orphan $N=332$)				
Maternal orphan vs. non-orphan	53	0.37	0.13–1.10	0.074
Paternal orphan vs. non-orphan	220	0.82	0.48–1.41	0.481
Double orphan vs. non-orphan	59	0.73	0.28–1.91	0.521
HIV prevalence $>5\%$[a] (non-orphan $N=979$)				
Maternal orphan vs. non-orphan	181	1.61	1.13–2.29	0.009
Paternal orphan vs. non-orphan	554	1.06	0.82–1.36	0.658
Double orphan vs. non-orphan	245	1.41	1.02–1.95	0.036

[a]All models are age adjusted.

Adjusting for education status very slightly reduced the size of the effect of maternal and double orphan status on having started sex, although the pattern of effect across the orphan types remained the same and the association with double orphanhood remained significant (see Table 1). Education status was not a significant modifier of the effect of orphan status on having started sex ($p=0.144$). It was not possible to test for an interaction between orphan status and education with respect to risk of HIV infection due to small numbers of infected individuals. Marital status did not confound the relationship between orphanhood and risk of HIV infection and there was no evidence that it was an effect modifier ($p=0.179$).

In our matched male sample, 1248 orphans were initially selected. The sample clusters were small, especially since only one eligible adolescent per household was included in the data-set. The clusters ranged in size from 1 to 11 male adolescents, with around 60% containing only one or two adolescents. Thus matching non-orphans could not be found for 358 (28.7%) of the selected orphans. These were dropped and the final sample contained 890 orphans matched to 890 non-orphans (total = 1780). Amongst the orphans, 19.7% were maternal orphans, 58.8% were paternal orphans and 21.6% were double orphans. About 25.2% had started sex and 1.6% were infected with HIV. Amongst non-orphans, 23.7% had started sex and 1.5% were infected with HIV.

Both the age adjusted and fully adjusted models indicated that there was no association between orphan status and risk of starting sex or HIV infection for male adolescents (see Table 3), although it was not possible to run the fully adjusted model of risk of HIV infection due to the small numbers of HIV-infected cases in the sample (23 infections). There was little evidence that the effect of orphan status, on either outcome, varied by country ($p>0.1$ for all tests, except for the effect of maternal orphan status on risk of starting sex: $p=0.061$). Neither country-level HIV prevalence or education status was a significant modifier of the effect of orphan status on having started sex. Adjusting for education status did not alter the size of the effect of orphan status on risk of starting sex or HIV infection. It was not possible to run tests for interaction for the HIV infection models due to the small number of individuals infected with HIV.

We found a strong, positive correlation between the prevalence of maternal (PCC = 0.89), paternal (PCC = 0.70) and double (PCC = 0.68) orphans in a country and the prevalence of HIV in that country (Figure 2).

Discussion

We developed a theoretical framework for investigating determinants of HIV risk and used it to generate specific hypotheses regarding the effects of orphan status on sexual health risk among adolescents. We hypothesised that in high HIV prevalence countries there would be a greater prevalence of orphanhood, which would increase pressure on the extended family network, which facilitates the care of orphans. Thus, orphans living in countries with high HIV prevalence would receive a lower standard of care and guidance, and therefore increased sexual health risk relative to non-orphans, compared to orphans living in low HIV prevalence countries. In our data-set we found that the prevalence of orphanhood was higher in high HIV prevalence countries – a strong, positive correlation between country-level HIV prevalence and orphan prevalence was observed. We also found that, among female adolescents, maternal and double

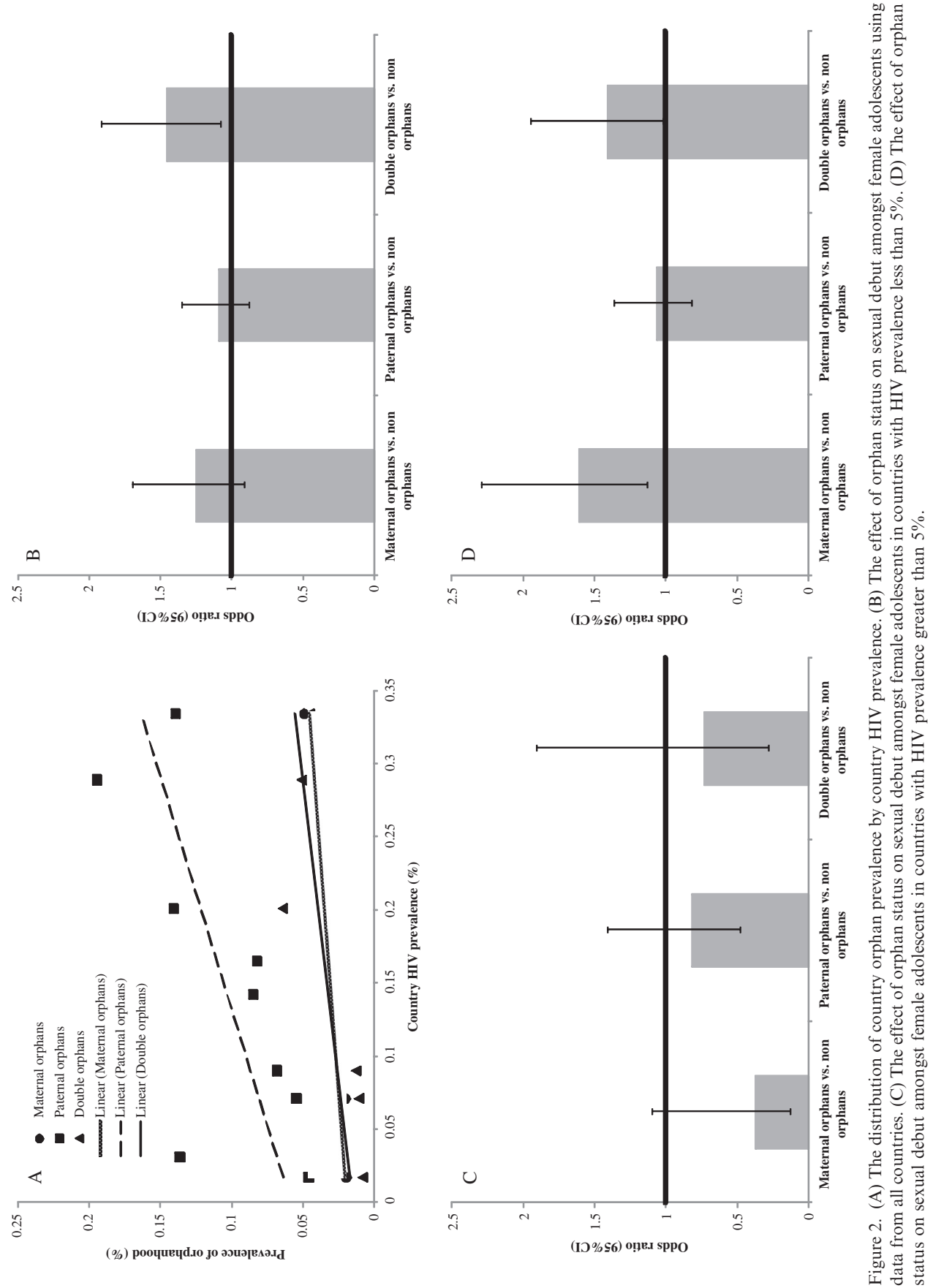

Figure 2. (A) The distribution of country orphan prevalence by country HIV prevalence. (B) The effect of orphan status on sexual debut amongst female adolescents using data from all countries. (C) The effect of orphan status on sexual debut amongst female adolescents in countries with HIV prevalence less than 5%. (D) The effect of orphan status on sexual debut amongst female adolescents in countries with HIV prevalence greater than 5%.

Table 3. The effects of orphan status on sexual risk among male adolescents.

	Effect of orphan status on started sex (non-orphan $N=889$)[c]					Effect of orphan status on HIV infection (non-orphan $N=758$)[d]				
	N (Orphans)	OR	95% CI	p-Value	Test of interaction (p-value)	N (Orphans)	OR	95% CI	p-Value	Test of interaction (p-value)
Age adjusted models										
Maternal orphan vs. non-orphan	175	0.99	0.66–1.48	0.966		150	1.43	0.39–5.24	0.590	
Paternal orphan vs. non-orphan	523	1.19	0.92–1.54	0.189	–	458	0.60	0.19–1.92	0.391	
Double orphan vs. non-orphan	192	0.97	0.66–1.44	0.890		164	1.95	0.66–5.76	0.228	
Fully adjusted models[b]										
Maternal orphan vs. non-orphan	141	1.02	0.66–1.59	0.922		117				
Paternal orphan vs. non-orphan	451	1.15	0.84–1.57	0.381	–	380		MA[a]		–
Double orphan vs. non-orphan	171	0.99	0.65–1.52	0.969		127				
Adjusted for age and education level										
Maternal orphan vs. non-orphan	175	0.99	0.66–1.47	0.947		150	1.43	0.39–5.26	0.590	
Paternal orphan vs. non-orphan	523	1.19	0.92–1.54	0.195	0.814	458	0.61	0.19–1.93	0.397	MA[b]
Double orphan vs. non-orphan	192	0.96	0.65–1.42	0.843		164	1.92	0.65–5.72	0.241	
Adjusted for age and marital status										
Maternal orphan vs. non-orphan	–	–	–	–		150				
Paternal orphan vs. non-orphan	–	–	–	–	–	458		MA[a]		MA[a]
Double orphan vs. non-orphan	–	–	–	–		164				
Adjusted for age and country HIV prevalence										
Maternal orphan vs. non-orphan	133	–	–	–		119	–	–	–	
Paternal orphan vs. non-orphan	429	–	–	–	0.309	397	–	–	–	MA[a]
Double orphan vs. non-orphan	150	–	–	–		145	–	–	–	

[a]Maximisation aborted.

[b]In the "ever has sex?" model we adjusted for age; wealth quintile; education level; migration status; urban/rural region; number of people in the household; age; sex; and education level of the household head. In the HIV infection model we also adjusted for material status.

[c]In the fully adjusted model non-orphan $N=776$. In the country HIV prevalence model non-orphan $N=712$.

[d]In the fully adjusted model non-orphan $N=672$. In the country HIV prevalence model non-orphan $N=613$.

Note: –, No attempt was made to run the model. For the marital status model this was because all married individuals had started sex and for the HIV prevalence models this was because country-level HIV prevalence could not be on the casual pathway between orphan status and risk of starting and risk of starting sex or HIV infection.

orphans experienced an increased sexual risk relative to non-orphans in high HIV prevalence countries but not in low HIV prevalence countries. These findings were consistent with our initial hypotheses and with previous studies using data from Zimbabwe (Birdthistle et al., 2008, 2009; Gregson et al., 2005; Kang et al., 2008; Nyamukapa et al., 2008) and South Africa (Operario et al., 2007; Thurman et al., 2006) – both countries with high HIV prevalence – and from across sub-Saharan Africa (Bicego et al., 2003; Hosegood et al., 2007; Monasch & Boerma, 2004).

We also hypothesised that the effect of country-level HIV prevalence on orphans' sexual risk would be mediated through household level variables that reflected the circumstances experienced by families affected by HIV. However, when the household level variables were added to the female adolescent model, the interaction between orphan status and country-level HIV prevalence, with respect to having started sex, remained significant. This suggests that high HIV prevalence was not affecting female orphans' sexual risk through these household level indicators. It may be that these indicators do not adequately reflect household level changes brought about by HIV-related shocks. For example, the relationship of an adolescent to his/her primary caregivers, which was not available in our data-set, may better reflect a households' ability or willingness to invest in an orphaned child.

There may be some other aspect of the socio-economic and cultural environment that was impacting negatively on orphans in countries with high HIV prevalence. Nyamukapa et al. (2008) found evidence – using national data from Zimbabwe – supporting a causal pathway in which orphans suffer from psychosocial distress, which in turn leads to increased sexual risk behavior. Data from South Africa have indicated that the association between orphanhood and poor psychological health is mediated through poverty and stigma associated with AIDS orphans (Cluver, Gardner, & Operario, 2008, 2009). It is possible that high levels of stigma around orphanhood in countries with high HIV prevalence could lead to psychosocial distress and increased sexual risk amongst adolescents. This is an interesting area for future investigation.

We did not find that the effects of orphanhood on sexual risk or HIV infection varied significantly at the country level, despite our finding that there was a difference in effect depending on country-level HIV prevalence. This may be because there is a threshold effect whereby a significant effect modification only occurs after a country reaches a sufficiently high prevalence i.e., above 5%. Our sample of countries was skewed towards those with high prevalence i.e., the majority of countries in our sample had a prevalence above 5%. Thus it may not have been possible to detect significant variation in the effect of orphan status across the countries in our small sample when this was modeled as a random coefficient at the country level. It is also possible that there was some other difference between the high and low prevalence countries (e.g., in predominant religion, urbanisation, etc.) that produced our observed effect.

There was no evidence in our data-set that orphaned male adolescents experienced increased sexual risk. This was surprising as analyses of data from South Africa and Zimbabwe suggest that adolescent, male orphans are at increased risk of having started sex (Nyamukapa et al., 2008; Operario et al., 2007; Thurman et al., 2006). This suggests that more work is needed to investigate the effects of orphanhood on male adolescents using data from a variety of contexts, especially since the majority of existing studies have focused on young women. It was also surprising that the increased risk of sexual debut amongst female double orphans did not seem to translate into an increased risk of HIV infection, especially given that having started sex was strongly associated with HIV infection amongst female adolescents in our large, unmatched data-set (results not shown). However, there were only a few infected women in our smaller, matched sample, which may have masked the effect of orphan status on this outcome.

Our results regarding the role of education in the causal pathway between orphanhood and sexual risk were consistent with findings from previous studies – differences in education status did not explain the increased risk among female orphans (Birdthistle et al., 2009). Adolescent educational achievement could also be an indicator of household investment in children suggesting that lower investment of resources in orphaned children may not explain their increased sexual risk. We did not find marital status to be a significant effect modifier of the relationship between orphan status and sexual risk for adolescents of either sex. A study of female adolescents in urban Zimbabwe found that married orphans were protected against the increased risk of HIV and/or HSV-2 experienced by unmarried orphans (Birdthistle et al., 2008, 2009). It is likely that the effects of marriage would vary depending on the cultural context and further investigation into the role of marriage in determining sexual risk among orphans is required.

This study has some limitations. The statistical models were complex and this made the analysis difficult to run and, when numbers of HIV-infected individuals were small, power to detect associations

may have been reduced. In order to simplify our models we selected a matched sample. This forced us to exclude orphans from the analysis if we were unable to find a matching non-orphan in their area. This process for excluding orphans is non-random – e.g., excluded orphans may be more likely to come from areas with fewer children – and this could have introduced bias to our sample. However, less than 20% of the female sample and less than 30% in the male sample were dropped. Thus we hope the extent of the bias is small.

The data we used were cross-sectional and information was not available on age at first sex or age of orphaning. It was therefore impossible to determine the temporal arrangement of events – e.g., it is not clear whether children were orphaned before or after beginning sex or becoming infected with HIV (e.g., through vertical transmission). There may also be different effects on sexual risk depending on the time that has elapsed since a parental death – Birdthistle et al. (2008) found that paternal orphans suffered increased sexual risk if their father died before they reached 12 years of age, but not if their father died after they reached this age.

Parental survival data is a relatively crude way of describing the varied experience of orphaned children – some may have living parents taking care of them or sending them money while others may be fostered within the extended family. Non-orphaned children may be at risk due to absent parents. Further work investigating the effects of fostering and other child-care practices on sexual risk behaviour is required. Qualitative work is also needed to explore the details of orphaned children's experiences that are not captured in quantitative data. There may be biases due to under-estimation of orphan prevalence due to foster parents being misreported as biological parents (Robertson et al., 2008; Timaeus, 1991). However, it is likely that such children are being cared for within the extended family and may themselves be unaware of their orphan status (Robertson et al., 2008). Thus they may not represent an important risk group with respect to targeting of orphan-related interventions. Finally, our crude estimate of country-level HIV prevalence may mask heterogeneity at the sample cluster (or regional) level with respect to the effects of parental mortality on adolescent sexual risk behaviour. It was not possible to investigate this here because we do not have reliable estimates of HIV prevalence at the sample cluster level.

Despite these limitations, we have used hierarchical statistical modelling to investigate hypothesised causal relationships relating to HIV risk in pooled data from several sub-Saharan African countries and plausible results have been obtained. This methodology could therefore be used to further validate our theoretical framework through the generation and testing of new hypotheses – e.g., regarding the effects of migration, psychosocial distress or wealth distribution on HIV risk. Furthermore, analyses involving longitudinal data could provide additional temporal information and thus would allow more robust conclusions to be drawn regarding causal pathways. Our results suggest that adolescent orphans represent an important target group for HIV prevention and that efforts should be made to integrate prevention messages into existing support programmes for orphans and vulnerable children.

References

Bicego, G., Rutstein, S., & Johnson, K. (2003). Dimensions of the emerging orphan crisis in sub-Saharan Africa. *Social Science & Medicine*, *56*(6), 1235–1247.

Birdthistle, I., Floyd, S., Nyagadza, A., Mudziwapasi, N., Gregson, S., & Glynn, J.R. (2009). Is education the link between orphanhood and HIV/HSV-2 risk among female adolescents in urban Zimbabwe? *Social Science & Medicine*, *68*(10), 1810–1818.

Birdthistle, I.J., Floyd, S., Machingura, A., Mudziwapasi, N., Gregson, S., & Glynn, J.R. (2008). From affected to infected? Orphanhood and HIV risk among female adolescents in urban Zimbabwe. *AIDS*, *22*(6), 759–766.

Boerma, J.T., & Weir, S.S. (2005). Integrating demographic and epidemiological approaches to research on HIV/AIDS: The proximate-determinants framework. *The Journal of Infectious Disease*, *191*(Suppl. 1), S61–S67.

Cluver, L., Gardner, F., & Operario, D. (2009). Poverty and psychological health among AIDS-orphaned children in Cape Town, South Africa. *AIDS Care*, *21*(6), 732–741.

Cluver, L.D., Gardner, F., & Operario, D. (2008). Effects of stigma on the mental health of adolescents orphaned by AIDS. *Journal of Adolescent Health*, *42*(4), 410–417.

Gregson, S., Nyamukapa, C.A., Garnett, G.P., Wambe, M., Lewis, J.J., Mason, P.R., … Anderson, R.M. (2005). HIV infection and reproductive health in teenage women orphaned and made vulnerable by AIDS in Zimbabwe. *AIDS Care*, *17*(7), 785–794.

Hargreaves, J.R., Morison, L.A., Kim, J.C., Bonell, C.P., Porter, J.D., Watts, C., … Pronyk, P.M. (2008). The association between school attendance, HIV infection and sexual behaviour among young people in rural South Africa. *Journal of Epidemiology & Community Health*, *62*(2), 113–119.

Hosegood, V., Floyd, S., Marston, M., Hill, C., McGrath, N., Isingo, R., … Zaba, B. (2007). The effects of high HIV prevalence on orphanhood and living arrangements of children in Malawi, Tanzania, and South Africa. *Population Studies*, *61*(3), 327–336.

Kang, M., Dunbar, M., Laver, S., & Padian, N. (2008). Maternal versus paternal orphans and HIV/STI risk

among adolescent girls in Zimbabwe. *AIDS Care*, *20*(2), 214–217.

Lewis, J.J., Donnelly, C.A., Mare, P., Mupambireyi, Z., Garnett, G.P., & Gregson, S. (2007). Evaluating the proximate determinants framework for HIV infection in rural Zimbabwe. *Sexual Transmission Infection*, *83*(Suppl. 1), i61–i69.

Lopman, B., Nyamukapa, C., Mushati, P., Mupambireyi, Z., Mason, P., Garnett, G.P., & Gregson, S. (2008). HIV incidence in 3 years of follow-up of a Zimbabwe cohort – 1998–2000 to 2001–03: Contributions of proximate and underlying determinants to transmission. *International Journal of Epidemiology*, *37*(1), 88–105.

Monasch, R., & Boerma, J.T. (2004). Orphanhood and childcare patterns in sub-Saharan Africa: An analysis of national surveys from 40 countries. *Aids*, *18*(Suppl. 2), S55–S65.

Mosley, W.H., & Chen, L.C. (2003). An analytical framework for the study of child survival in developing countries. 1984. *Bulletin of the World Health Organization*, *81*(2), 140–145.

Nyamukapa, C.A., Gregson, S., Lopman, B., Saito, S., Watts, H.J., Monasch, R., & Jukes, M.C. (2008). HIV-associated orphanhood and children's psychosocial distress: Theoretical framework tested with data from Zimbabwe. *American Journal of Public Health*, *98*(1), 133–141.

Operario, D., Pettifor, A., Cluver, L., MacPhail, C., & Rees, H. (2007). Prevalence of parental death among young people in South Africa and risk for HIV infection. *Journal of Acquired Immune Deficiency Syndromes*, *44*(1), 93–98.

Poundstone, K.E., Strathdee, S.A., & Celentano, D.D. (2004). The social epidemiology of human immunodeficiency virus/acquired immunodeficiency syndrome. *Epidemiological Reviews*, *26*, 22–35.

Rabe-Hesketh, S., & Skrondal, A. (2005). *Multilevel and longitudinal modeling using stata*. College Station, TX: Stata.

Robertson, L., Gregson, S., Madanhire, C., Walker, N., Mushati, P., Garnett, G., & Nyamukapa, C. (2008). Discrepancies between UN models and DHS survey estimates of maternal orphan prevalence: Insights from analyses of survey data from Zimbabwe. *Sexual Transmission Infection, 84*(Suppl. 1), i57–i62.

Rutstein, S., & Johnson, K. (2004). *The DHS wealth index*. Calverton, MD: ORC Macro.

Thurman, T.R., Brown, L., Richter, L., Maharaj, P., & Magnani, R. (2006). Sexual risk behavior among South African adolescents: Is orphan status a factor? *AIDS and Behavior*, *10*(6), 627–635.

Timaeus, I.M. (1991). Measurement of adult mortality in less developed countries: A comparative review. *Population Index*, *57*(4), 552–568.

Twisk, J.W.R. (2006). *Applied multilevel analysis*. Cambridge: Cambridge University Press.

UNAIDS (2009). *AIDS epidemic update: Report archive*. Retrieved from http://www.unaids.org/en/KnowledgeCentre/HIVData/EpiUpdate/EpiUpdArchive/Default.asp

Watts, H., Gregson, S., Saito, S., Lopman, B., Beasley, M., & Monasch, R. (2007). Poorer health and nutritional outcomes in orphans and vulnerable young children not explained by greater exposure to extreme poverty in Zimbabwe. *Tropical Medicine & International Health*, *12*(5), 584–593.