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SHORT REPORT
Low socioeconomic status and risk for infection with Human Herpesvirus 8 among HIV-1 negative, South African black cancer patients

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SUMMARY

Between January 1994 and October 1997, we interviewed 2576 black in-patients with newly diagnosed cancer in Johannesburg and Soweto, South Africa. Blood was tested for HIV-1 and HHV-8 antibodies and the study was restricted to 2191 HIV-1 antibody-negative patients. We examined the relationship between infection with HHV-8 and sociodemographic and behavioural factors using unconditional logistic regression models. Of the 2191 HIV-1 negative patients who did not have Kaposi's sarcoma, 854 (39·1%) were positive for antibodies against the latent nuclear antigen of HHV-8 encoded by orf73 in a immunofluorescence assay. Infection with HHV-8 was independently associated with increasing age (P trend = 0·02). For females, independent risk factors also included working in a paid domestic capacity (OR 1·63, 95% CI 1·09–2·44, $P = 0·02$), defining occupational status as economically non-active unemployed (OR 1·70, 95% CI 1·06–2·72, $P = 0·03$), having a state pension or being on a disability grant (OR 1·49, 95% CI 1·05–2·11, $P = 0·02$), using oral contraceptives (OR 1·43, 95% CI 1·03–1·99, $P = 0·03$) and having a delayed age at menarche (P trend = 0·04). The relationship between these variables and HHV-8 antibody status requires further, prospective study.

INTRODUCTION

Human herpesvirus 8 (HHV-8, also known as Kaposi's sarcoma-associated herpesvirus) is understood to be the necessary, causal agent in the development of Kaposi's sarcoma [1–3]. In sub-Saharan African populations, where the prevalence of HHV-8 is estimated to be between 30 and 60% [4–6], research indicates that most transmission occurs horizontally during childhood [6, 7]. Cross-sectional studies from Central Africa have found that HHV-8 prevalence in children

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Table 1. *Risk factors for HHV-8 antibody positivity*

	Number positive/total (%)[a]	aOR[b] (95% CI)
Sex		
Male[c]	335/857 (39·1)	1·0
Female	519/1334 (38·9)	1·11 (0·92–1·33)
Test for homogeneity[d]		$\chi^2 = 1{\cdot}16$, $P = 0{\cdot}28$
Age (years)		
<35[c]	48/205 (23·4)	1·0
35–44	113/333 (33·9)	1·67 (1·13–2·48)
45–54	190/533 (35·7)	1·82 (1·26–2·63)
55–64	220/539 (40·8)	2·28 (1·58–3·28)
$\geqslant 65$	281/578 (48·6)	3·17 (2·20–4·56)
Test for trend[d]		**$\chi^2 = 48{\cdot}3$, $P < 0{\cdot}0001$**
Education		
None[c]	271/581 (46·6)	1·0
1–5	339/878 (38·6)	0·79 (0·63–0·98)
$\geqslant 6$ years	240/719 (33·4)	0·72 (0·57–0·91)
Test for trend[d]		**$\chi^2 = 7{\cdot}08$, $P = 0{\cdot}008$**
Place of birth		
Rural[c]	516/1245 (41·5)	1·0
Urban	313/866 (36·1)	0·93 (0·77–1·12)
Test for homogeneity[d]		$\chi^2 = 0{\cdot}70$, $P = 0{\cdot}40$
Place of residence		
Urban[c]	660/1698 (38·9)	1·0
Rural	176/435 (40·5)	1·11 (0·89–1·39)
Test for homogeneity[d]		$\chi^2 = 1{\cdot}16$, $P = 0{\cdot}28$
Place of residence		
Gauteng[c]	1032/1681 (61·4)	1·0
Other	205/509 (40·3)	1·14 (0·92–1·40)
Test for homogeneity[d]		$\chi^2 = 0{\cdot}99$, $P = 0{\cdot}32$
Working now		
No[c]	673/1656 (40·6)	1·0
Yes	167/497 (33·6)	0·88 (0·71–1·11)
Test for homogeneity[d]		$\chi^2 = 1{\cdot}17$, $P = 0{\cdot}28$
Pension/Disability		
No[c]	449/1328 (33·8)	1·0
Yes	392/825 (47·5)	1·37 (1·06–1·77)
Test for homogeneity[d]		**$\chi^2 = 5{\cdot}81$, $P = 0{\cdot}02$**
Occupation		
White-collar[c]	89/270 (33·0)	1·0
Non-active/unemployed	99/237 (41·8)	1·71 (1·17–2·52)
Farming	41/97 (42·3)	1·29 (0·79–2·09)
Industry	181/559 (32·4)	0·86 (0·62–1·19)
Domestic	331/755 (43·8)	1·59 (1·17–2·16)
Unspecified	32/88 (36·4)	1·46 (0·87–2·46)
Test for homogeneity[d] (2 D.F.)		**$\chi^2 = 15{\cdot}5$, $P = 0{\cdot}004$**
Smoke cigarettes/pipes		
No[c]	487/1257 (38·7)	1·0
Past	233/583 (40·0)	0·96 (0·76–1·21)
Yes	128/339 (37·8)	0·97 (0·74–1·27)
Test for homogeneity[d] (2 D.F.)		$\chi^2 = 0{\cdot}12$, $P = 0{\cdot}94$
Maize beer		
Never[c]	762/1961 (38·9)	1·0
Less than 1 × week	50/113 (44·3)	1·15 (0·78–1·70)

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Table 1 (*cont.*)

	Number positive/total (%)[a]	aOR[b] (95% CI)
More than 1× week	17/51 (33·3)	0·68 (0·38–1·24)
Most days	20/54 (37·0)	0·82 (0·46–1·45)
Test for trend[d]		$\chi^2=0·77$, $P=0·38$
Sorghum beer		
Never[c]	693/1766 (39·2)	1·0
Less than 1× week	116/316 (36·7)	0·76 (0·59–0·99)
Most days	13/34 (38·2)	0·80 (0·40–1·63)
Test for trend[d]		$\chi^2=2·16$, $P=0·14$
Commercial beer		
Never[c]	508/1342 (37·9)	1·0
Less than 1× week	48/133 (36·1)	0·88 (0·60–1·28)
More than 1× week	81/179 (45·3)	1·23 (0·89–1·71)
Most days	211/524 (40·3)	1·07 (0·85–1·35)
Test for trend[d]		$\chi^2=0·72$, $P=0·40$
Marital status		
Single[c]	175/527 (33·2)	1·0
Married	458/1161 (39·5)	0·97 (0·75–1·23)
Widowed	162/344 (47·1)	1·10 (0·78–1·49)
Separated/Divorced	57/152 (37·5)	0·93 (0·63–1·39)
Test for homogeneity[d] (3 D.F.)		$\chi^2=0·80$, $P=0·85$
No. of lifetime sexual partners		
0–2[c]	166/454 (36·6)	1·0
3–4	423/1051 (40·3)	1·22 (0·97–1·54)
⩾5	202/508 (39·8)	1·29 (0·98–1·70)
Test for trend[d]		**$\chi^2=3·53$, $P=0·06$**
Parity		
0–2[c]	140/399 (35·1)	1·0
3–4	174/436 (39·9)	1·14 (0·86–1·52)
⩾5	176/400 (44·0)	1·23 (0·91–1·66)
Test for trend[d]		$\chi^2=1·80$, $P=0·18$
Age when periods began (years)		
10–13[c]	39/138 (28·3)	1·0
14	81/217 (37·3)	1·58 (0·99–2·52)
15	166/409 (40·6)	1·66 (1·08–2·54)
⩾16	227/554 (41·0)	1·65 (1·09–2·49)
Test for trend[d]		**$\chi^2=3·61$, $P=0·06$**
Age when periods ended (years)		
⩽45[c]	94/252 (37·3)	1·0
46–49	100/245 (40·8)	0·97 (0·65–1·43)
⩾50	198/451 (43·9)	1·01 (0·70–1·46)
Test for trend[d]		$\chi^2=0·31$, $P=0·57$
Oral contraceptives		
No[c]	424/1102 (38·5)	1·0
Yes	86/215 (40·0)	1·31 (0·96–1·80)
Test for homogeneity[d]		**$\chi^2=2·84$, $P=0·09$**

Bold values are significant at $P<0·10$.

[a] Data were not available for all patients for all variables.

[b] Adjusted for age category <35, 35–44, 45–54, 55–64, or ⩾65 years and sex.

[c] These patients served as the reference group.

[d] All values were calculated with the χ^2 test with 1 D.F. unless otherwise noted.

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and adolescents reaches approximately 30–50% depending on the age group tested [6, 7]. However, in these studies, the prevalence of HHV-8 continues to increase almost linearly with age throughout adulthood, albeit gradually. Some of this may be due to a cohort effect, with people born earlier in the century in less hygienic conditions having a higher prevalence than those born later. Alternatively, as HHV-8 is present most commonly and in greatest concentrations in saliva [8] it is possible that infection continues during adulthood [4, 9]. To explain this increase with age in adulthood, we have presented evidence [4], as have other researchers [9–11] that HHV-8 may be transmitted through heterosexual intercourse in sub-Saharan African adult populations. Our previous research has also indicated that low socioeconomic status may facilitate transmission due to factors related to interpersonal contact although the precise means of transmission in the low socioeconomic status environment have yet to be elucidated [4]. In this Short Report, we present additional evidence of HHV-8 transmission through exposure to a low socioeconomic status environment.

METHODS

We conducted a cross-sectional study of risk factors for infection with HHV-8 as described elsewhere [4] between January 1994 and October 1997 at three Johannesburg hospitals (Chris Hani-Baragwanath, Hillbrow and Johannesburg). Trained nurses interviewed 2576 black in-patients with cancer using a questionnaire in the language of the patient (most commonly Zulu or Sotho). Specific questions were asked about behavioural and sociodemographic characteristics including age, sex, birthplace, current residence, years of education, occupation and reproductive and lifetime sexual history. The subjects included in this study were restricted to 2191 HIV-1 antibody-negative patients without Kaposi's sarcoma. Further details on the study participants can be found in ref. [4].

The serum samples were shipped by air on dry ice to the Institute of Cancer Research in London for HHV-8 testing. Details of the testing procedure are described elsewhere [4]. Briefly, a B-cell lymphoma (primary effusion lymphoma) cell line, BCP-1, infected with HHV-8 but not Epstein–Barr virus (EBV) was used for an indirect immunofluorescence assay to detect HHV-8 IgG antibodies. All assays were examined by a single observer [12, 13]. Slides were screened by ultraviolet microscopy for the latent nuclear antigen of HHV-8 encoded by orf73 [12, 14–18]. Serum samples that were positive for HHV-8 antibody by the immunofluorescence assay were scored as low (median titres were 1:200), medium (1:51200) or high (1:204800) according to the intensity of the fluorescent signal. These scores correlated well with intensity of fluorescence as measured by fluorescence-activated cell sorter (FACS) analysis described elsewhere [4].

We initially examined the relationship between HHV-8 antibody status and answers to all the questions from our questionnaire including age, sex, education, place of birth, place of residence, parity, number of lifetime sexual partners, history of contraceptive use for women, frequency and type of alcohol consumption, use and frequency of tobacco and other lifestyle variables such as fuel used for cooking and heating and building materials used in house construction. Odds ratios were calculated by unconditional logistic regression adjusting for age groups (<35, 35–44, 45–54, 55–64, or $\geqslant 65$ years), sex and other factors were as indicated using STATA version 7 [19]. All P values are two-sided. Note that the number of cases and controls in the tables do not always add up to the total because of missing values. Goodness of fit was assessed by the Hosmer–Lemeshow test [20].

RESULTS

Of the 2191 samples, 854 (39·1%) were positive for HHV-8 antibodies according to the immunofluorescence assay. In bivariate analysis, increasing risk for HHV-8 antibody positivity was associated with increasing age with the greatest risk for those $\geqslant 65$ years old (OR 3·17, 95% CI 2·20–4·56, P trend $< 0{\cdot}0001$; Table 1). As reported previously in Sitas et al. [4], increasing years of education is protective against HHV-8 antibody positivity with those with $\geqslant 6$ years education having a 33·4% seroprevalence compared with 46·6% of those having no education (OR for $\geqslant 6$ years $= 0{\cdot}72$, 95% CI 0·57–0·91, P trend $= 0{\cdot}008$). The number of lifetime sexual partners was marginally significant for HHV-8 antibody positivity with those having more than or equal to five lifetime sexual partners having an increased risk (OR 1·29, 95% CI 0·98–1·70, P trend $= 0{\cdot}06$). In addition, individuals who were on a state pension or a disability grant had an increased

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Table 2. *Summary of risk factors for HHV-8 antibody positivity*

	Females (OR)[a,b]	*P* value	Males (OR)[a,c]	*P* value
Age category (years)				
<35[d]	1·0		1·0	
35–44	1·93 (1·17–3·19)	**0·01**	2·01 (0·81–4·97)	0·13
45–54	2·08 (1·28–3·36)	**0·003**	1·98 (0·86–4·59)	0·11
55–64	2·24 (1·33–3·77)	**0·003**	2·32 (1·00–5·39)	**0·05**
⩾65	2·13 (1·18–3·84)	**0·01**	3·83 (1·58–9·26)	**0·003**
Test for trend[e]	$\chi^2=5{\cdot}34$, $P=$**0·02**		$\chi^2=9{\cdot}18$, $P=$**0·02**	
Education				
None[d]	1·0		1·0	
1–5	0·83 (0·61–1·11)	0·21	0·76 (0·53–1·08)	0·12
⩾6 years	0·87 (0·62–1·22)	0·41	0·70 (0·45–1·08)	0·11
Test for trend[e]	$\chi^2=0{\cdot}79$, $P=0{\cdot}37$		$\chi^2=2{\cdot}53$, $P=0{\cdot}11$	
Pension/Disability				
No[d]	1·0		1·0	
Yes	1·49 (1·05–2·11)	**0·02**	1·13 (0·74–1·73)	0·56
Test for homogeneity[e]	$\chi^2=4{\cdot}76$, $P=$**0·03**		$\chi^2=0{\cdot}37$, $P=0{\cdot}54$	
Occupation				
White collar[d]	1·0		1·0	
Non-active	1·70 (1·06–2·72)	**0·03**	3·26 (0·96–11·05)	0·06
Farming	1·43 (0·63–3·24)	0·39	1·09 (0·56–2·15)	0·79
Industry	0·88 (0·54–1·42)	0·60	0·83 (0·52–1·33)	0·44
Domestic	1·63 (1·09–2·44)	**0·02**	1·31 (0·68–2·49)	0·42
Unspecified	1·46 (0·73–2·90)	0·28	1·71 (0·71–4·12)	0·23
Test for homogeneity[e] (5 D.F.)	$\chi^2=13{\cdot}6$, $P=$**0·02**		$\chi^2=5{\cdot}66$, $P=0{\cdot}34$	
No. of lifetime sexual partners				
0–2[d]	1·0		1·0	
3–4	1·24 (0·94–1·63)	0·13	1·20 (0·73–1·98)	0·47
⩾5	1·31 (0·91–1·90)	0·15	1·31 (0·78–2·20)	0·31
Test for trend[e]	$\chi^2=2{\cdot}62$, $P=0{\cdot}10$		$\chi^2=0{\cdot}46$, $P=0{\cdot}50$	
Oral contraceptive use				
No[d]	1·0			
Yes	1·43 (1·03–1·99)	**0·03**		
Test for homogeneity[e]	$\chi^2=4{\cdot}67$, $P=$**0·03**			
Age at menarche (years)				
10–13[d]	1·0			
14	1·64 (1·01–2·65)	**0·04**		
15	1·66 (1·07–2·57)	**0·02**		
⩾16	1·69 (1·10–2·59)	**0·02**		
Test for trend[e]	$\chi^2=4{\cdot}08$, $P=$**0·04**			

Bold values are significant at $P\leqslant 0{\cdot}05$.

[a] In the multivariate model, all bivariate statistically significant variables at $P=0{\cdot}1$ were included. Each variable was adjusted for all the other variables in the table.

[b] The Homer–Lemeshow goodness-of-fit-statistic = 10·57 with 8 D.F. ($P=0{\cdot}23$).

[c] The Homer–Lemeshow goodness-of-fit-statistic = 5·54 with 8 D.F. ($P=0{\cdot}70$).

[d] These patients served as the reference group.

[e] All values were calculated with the χ^2 test with 1 D.F. unless noted otherwise.

risk of infection (OR 1·37, 95% CI 1·06–1·77), and there was also an increased risk of infection among persons who defined their occupational status as not working/non-economically active (OR 1·71, 95% CI 1·17–2·52) or were employed in domestic capacities (OR 1·59, 95% CI 1·17–2·16) (χ^2 heterogeneity = 15·5, $P=0{\cdot}004$; Table 1).

We did not find any association between the use of snuff ($P=0{\cdot}27$), the smoking of cigarettes ($P=0{\cdot}94$) or the consumption of commercial beers ($P=0{\cdot}40$),

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commercial spirits ($P=0{\cdot}15$), sorghum beer ($P=0{\cdot}38$), maize beer ($P=0{\cdot}38$), or wine ($P=0{\cdot}15$) and HHV-8 antibody positivity (Table 1). Girls reaching menarche at a later age also had a greater risk of being HHV-8 seropositive although the P trend was only marginally significant (age at menarche $\geqslant 16$ years, OR 1·65, 95% CI 1·09–2·49, P trend $=0{\cdot}06$). We found no association with martial status (χ^2 heterogeneity $=0{\cdot}80$, $P=0{\cdot}85$) or parity (P trend $=0{\cdot}18$) and HHV-8 antibody positivity.

Since we examined approximately 50 potential risk factors in relation to HHV-8 antibody positivity, some associations may have occurred by chance. In our final analysis, only those factors that were significant at the 10% level were considered in a multivariate model (Table 2). Risk factors that were significant for HHV-8 antibody positivity included older age and socioeconomic variables. For both males and females, individuals infected with HHV-8 were more likely to be older ($\geqslant 65$ years for females OR 2·13, 95% CI 1·18–3·84 and males OR 3·83, 95% CI 1·58–9·26, P trend for males and females $=0{\cdot}02$). After stratifying risk factors on sex, since some of the reproductive risk factors are female-specific, six factors remained independently associated with antibody positivity. For females, risk factors included the following: working in a paid domestic capacity (OR 1·63, 95% CI 1·09–2·44, $P=0{\cdot}02$), being non-active (OR 1·70, 95% CI 1·06–2·72, $P=0{\cdot}03$), having a state pension or being on disability (OR 1·49, 95% CI 1·05–2·11, $P=0{\cdot}02$) using oral contraceptives (OR 1·43, 95% CI 1·03–1·99, $P=0{\cdot}03$) and having a delayed age at menarche (P trend for increasing age $=0{\cdot}04$). The associations we found between age and increased risk for antibody positivity concur with results from other studies [21–23].

DISCUSSION

Working as a paid domestic worker, defining occupational status as economically non-active/unemployed and being on a state pension/disability all increased the risk of acquiring HHV-8 infection as did a delayed age at menarche for women. These variables are markers of low socioeconomic status with delayed menarche suggesting a history of undernutrition and low socioeconomic status during childhood [24]. Roberts notes that the chronically poor in South Africa are more likely to receive state pensions, which are given to elderly individuals with no other source of income [25]. Other markers of low socioeconomic status (like birth or residence in a rural area) were not associated with HHV-8 infection. Our data on the relationship between low socioeconomic status and HHV-8 antibody positivity concurs with an earlier study that found an association between HHV-8 antibody positivity and markers of low socioeconomic status in an urban Zambian population [23]. As HHV-8 is more commonly found in saliva and in greater concentrations than other bodily fluids [8], it is possible that exposure occurs throughout an individual's lifetime in addition to childhood in endemic regions, although the pathways of transmission are yet to be determined. It is not clear why these associations were significant only for females and not males. One possible explanation is the close contact between women and young children, with those children infected with HHV-8 shedding the virus in their saliva in endemic areas. Future studies should examine components of the low socioeconomic status environment and sexual risk factors that may place individuals at heightened risk for infection with HHV-8.

One limitation of this study was our inability to assess potential exposures to HHV-8 in childhood independent of exposures to HHV-8 in adulthood. As our study was cross-sectional in nature and our population consisted exclusively of adults with the majority of our sample over the age of 35 years, it was not possible to isolate risk factors in adulthood or childhood risk factors for HHV-8. Some of our questions concerning educational level, age of menarche and place of birth were retrospectively oriented and geared toward assessing childhood exposures. However, we suggest that prospective longitudinal methods are needed to clearly delineate childhood and adult risk factors for HHV-8 infection.

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REFERENCES

1. Chang Y, Cesarman E, Pessin MS, et al. Identification of Herpesvirus-like DNA sequences in AIDS-associated Kaposi's sarcoma. Science 1994; **266**: 1865–1869.

2. Whitby D, Howard MR, Tenant-Flowers M, et al. Detection of Kaposi's sarcoma associated herpesvirus in peripheral blood of HIV-infected individuals and progression to Kaposi's sarcoma. Lancet 1995; **346**: 799–802.
3. Gao SJ, Kingsley L, Li M, et al. KSHV antibodies among Americans, Italians and Ugandans with and without Kaposi's sacoma. Nat Med 1996; **2**: 925–928.
4. Sitas F, Carrara H, Beral V, et al. Antibodies against human herpesvirus 8 in black South African patients with cancer. N Engl J Med 1999; **340**: 1863–1871.
5. Olsen SJ, Chang Y, Moore PS, Biggar RJ, Melbye M. Increasing Kaposi's sarcoma-associated herpesvirus seroprevalence with age in a highly Kaposi's sarcoma endemic region, Zambia in 1985. AIDS 1998; **12**: 1921–1925.
6. Mayama S, Cuevas LE, Sheldon J, et al. Prevalence and transmission of Kaposi's sarcoma-associated herpesvirus (human herpesvirus 8) in Ugandan children and adolescents. Int J Cancer 1998; **77**: 543–548.
7. Gessain A, Mauclere P, van Beveren M, et al. Human herpesvirus 8 primary infection occurs during childhood in Cameroon, Central Africa. Int J Cancer 1999; **81**: 189–192.
8. Pauk J, Huang ML, Brodie SJ, et al. Mucosal shedding of human herpesvirus 8 in men. N Engl J Med 2000; **343**: 1369–1377.
9. Baeten JM, Chohan BH, Lavrey L, et al. Correlates of human herpesvirus 8 seropositivity among heterosexual men in Kenya. AIDS 2002; **16**: 2073–2078.
10. Rezza G, Tchangmena O, Andreoni M, et al. Prevalence and Risk Factors for Human Herpesvirus 8 Infection in Northern Cameroon. Sex Transm Dis 2000; **27**: 159–164.
11. Eltom MA, Mbulaiteye SM, Dada AJ, et al. Transmission of human herpesvirus 8 by sexual activity among adults in Lagos, Nigeria. AIDS 2002; **16**: 2473–2478.
12. Gao SJ, Kingsley L, Li M, et al. KSHV antibodies among Americans, Italians and Ugandans with and without Kaposi's sarcoma. Nat Med 1996; **2**: 925–928.
13. Newton R, Ziegler J, Bourboulia D, et al. (Uganda Kaposi's Sarcoma Study Group). Establishing a KSHV + cell line (BCP-1) from peripheral blood and characterizing its growth in Nod/SCID mice. Blood 1998; **91**: 1671–1679.
14. Simpson GR, Schulz TF, Whitby D, et al. Prevalence of Kaposi's sarcoma associated herpesvirus infection measured by antibodies to recombinant capsid protein and latent immunofluorescence antigen. Lancet 1996; **348**: 1133–1138.
15. Kedes DH, Operskalski E, Busch M, Kohn R, Flood J, Ganem D. The seroepidemiology of human herpesvirus 8 (Kaposi's sarcoma-associated herpesvirus): distribution of infection in KS risk groups and evidence for sexual transmission. Nat Med 1996; **2**: 918–924 [Erratum, Nat Med 1996; **2**: 1041].
16. Rainbow L, Platt GM, Simpson GR, et al. The 222 to 234-kilodalton latent nuclear protein (LNA) of Kaposi's sarcoma-associated herpesvirus (human herpesvirus 8) is encoded by orf73 and is a component of the latency-associated nuclear antigen. J Virol 1997; **71**: 5915–5921.
17. Kellam P, Boshoff C, Whitby D, Matthews S, Weiss RA, Talbot SJ. Identification of a major latent nuclear antigen, LNA-1, in the human herpesvirus 8 genome. J Hum Virol 1997; **1**: 19–29.
18. Boshoff C, Gao SJ, Healy LE, et al. Establishing a KSHV + cell line (BCP-1) from peripheral blood and characterizing its growth in Nod/SCID mice. Blood 1998; **91**: 1671–1679.
19. StataCorp. Stata software, version 7. College Park, TX: Stata Corporation, 2001.
20. Lemeshow S, Hosmer Jr. DW. A review of goodness of fit statistics for use in the development of logistic regression models. Am J Epidemiol 1982; **115**: 92–106.
21. Newton R, Ziegler J, Bourboulia D, et al. The seroepidemiology of Kaposi's-sarcoma-associated herpesvirus (KSHV/HHV-8) in adults with cancer in Uganda. Int J Cancer 2002; **103**: 226–232.
22. Plancoulaine S, Abel L, van Beveren M, Gessain A. High titers of anti-human herpesvirus 8 antibodies in elderly males in an endemic population. J Nat Cancer Inst 2002; **94**: 133–135.
23. Olsen SJ, Chang Y, Moore PS, Biggar RJ, Melbye M. Increasing Kaposi's sarcoma-associated herpesvirus seroprevalence with age in a highly Kaposi's sarcoma endemic region, Zambia, in 1985. AIDS 1998; **12**: 1921–1925.
24. Pawloski LR. Growth and development of adolescent girls from the Segou Region of Mali (West Africa). Am J Phys Anthropol 2002; **117**: 364–372.
25. Roberts B. Chronic and transitory poverty in post-apartheid South Africa: evidence from KwaZulu-Natal. J Poverty 2001; **5**: 1–28.