2007-11-27

Determinants of Sexual Activity and Its Relation to Cervical Cancer Risk among South African Women

Cooper, Diane, Margaret Hoffman, Henri Carrara, Lynn Rosenberg, Judy Kelly, Ilse Stander, Lynnette Denny, Anna-Lise Williamson, Samuel Shapiro. "Determinants of sexual activity and its relation to cervical cancer risk among South African Women" BMC Public Health 7:341. (2007)
https://hdl.handle.net/2144/3427

Boston University
Determinants of sexual activity and its relation to cervical cancer risk among South African Women

Diane Cooper*1, Margaret Hoffman1, Henri Carrara1, Lynn Rosenberg2, Judy Kelly2, Ilse Stander3, Lynnette Denny4, Anna-Lise Williamson5,6 and Samuel Shapiro1

Address: 1Women’s Health Research Unit, School of Public Health and Family Medicine, Faculty of Health Sciences, University of Cape Town, Anzio Road, Observatory, 7925, Cape Town, South Africa, 2Slone Epidemiology Unit, Boston University, Boston, USA, 3Medical Research Council of South Africa, Tygerberg, South Africa, 4Department of Obstetrics and Gynaecology, Faculty of Health Sciences, University of Cape Town, Anzio Road, Observatory, 7925, Cape Town, South Africa, 5Institute of Infectious Disease and Molecular Medicine, Faculty of Health Sciences, University of Cape Town, Anzio Road, Observatory, 7925, Cape Town, South Africa and 6National Health Laboratory Service, South Africa

Email: Diane Cooper* - Diane.Cooper@uct.ac.za; Margaret Hoffman - Margaret.Hoffman@uct.ac.za; Henri Carrara - henri.carrara@gmail.com; Lynn Rosenberg - lrosenberg@slone.bu.edu; Judy Kelly - jkelly@slone.bu.edu; Ilse Stander - ilsestander@absamail.co.za; Lynnette Denny - ldenny@uctgh1.uct.ac.za; Anna-Lise Williamson - Anna-Lise.Williamson@uct.ac.za; Samuel Shapiro - samshap@mweb.co.za

* Corresponding author

Abstract

Background: Invasive cervical cancer is the commonest cause of cancer morbidity and mortality in South African women. This study provides information on adult women’s sexual activity and cervical cancer risk in South Africa.

Methods: The data were derived from a case-control study of hormonal contraceptives and cervical cancer risk. Information on age of sexual debut and number of lifetime sexual partners was collected from 524 incident cases and 1541 hospital controls. Prevalence ratios and adjusted prevalence ratios were utilised to estimate risk in exposures considered common. Crude and adjusted relative risks were estimated where the outcome was uncommon, using multiple logistic regression analysis.

Results: The median age of sexual debut and number of sexual partners was 17 years and 2 respectively. Early sexual debut was associated with lower education, increased number of lifetime partners and alcohol use. Having a greater number of sexual partners was associated with younger sexual debut, being black, single, higher educational levels and alcohol use. The adjusted odds ratio for sexual debut < 16 years and ≥ 4 life-time sexual partners and cervical cancer risk were 1.6 (95% CI 1.2 – 2.2) and 1.7 (95% CI 1.2 – 2.2), respectively.

Conclusion: Lower socio-economic status, alcohol intake, and being single or black, appear to be determinants of increased sexual activity in South African women. Education had an ambiguous effect. As expected, cervical cancer risk is associated with increased sexual activity. Initiatives to encourage later commencement of sex, and limiting the number of sexual partners would have a favourable impact on risk of cancer of the cervix and other sexually transmitted infections.
Background
In developing countries invasive cancer of the cervix is the commonest cause of cancer morbidity and mortality among women [1,2]. The South African age-standardized incidence is estimated to be 30/100,000 per year [2]. High-risk human papillomavirus (HPV) infection is a necessary cause[3]. HPV is sexually transmitted with a strong association existing between sexual activity and cervical cancer risk [4,5]. Only a small proportion of HPV infected women go on to develop squamous intraepithelial lesions or invasive cancer. Hence there must be co-factors to HPV infection that lead to the development of cervical cancer. There is scant information on sexual activity in adult South African women, despite high persistent levels of high risk HPV among older women [6]. In this paper patterns and determinants of sexual activity among adult South African women and their relationship to cancer of the cervix are described.

Methods
Data were derived from a case-control study conducted between January 1998 and September 2001 among women aged < 60 years, resident for at least six months within 150 km of Cape Town in the Western Cape, South Africa. The study’s main purpose was to assess the relationship between hormonal contraception and cervical cancer risk and details of the methods have been described elsewhere [7]. Written informed consent was obtained from participants and data was kept confidential. Ethical approval was obtained from the Research and Ethics Committee at the University of Cape Town and the Institutional Review Board at Boston University.

Women developing cervical cancer and using public health facilities were all treated at two tertiary care hospitals, in Cape Town. Incident cases were women with clinically evident and histologically confirmed invasive cervical epithelial cancer (stages 1b-IVb), diagnosed ≤ 6 months previously, with no previous history of any malignancy, presenting at the oncology clinics of these hospitals. Controls were treated and recruited in nongynecologic, nonobstetric wards in local hospitals, accounting for the wider range of hospitals for controls. Cases and controls were series-matched in a ratio of 1:3 on decade of age, race and geographic residence (urban or rural areas in same catchment geographical areas). Controls were selected to be representative of the same population from which the cases were drawn and were similar in a range of socio-demographic characteristics (see Table 1). Eligible controls were women with primary diagnoses such as trauma or acute infections, that have not been linked in previous studies, either positively or negatively to cervical cancer risk, to contraceptive use (e.g. trauma, appendicitis, disc disease and other major orthopaedic conditions); selective admissions for major surgery for conditions such as inguinal hernia or to sexual practices: women with conditions such as ischemic heart disease or venous thromboembolism, or gynecologic disorders, were not eligible.

There were 535 potential cases and 1668 potential controls. Two cases (0.4%) and 107 controls (6.4%) refused participation: 38 of the latter were recently screened for cervical abnormalities and refused a repeat test after a short interval. Seven controls had stage 1A cervical cancer and were excluded from the study after referral for treatment. Permission was obtained from controls and endocervical scrapings taken for HPV testing for current infection with high risk HPV at the same time. These were assayed for HPV infection using the Hybrid Capture II test for detection of high risk HPV types [7]. HPV specimens were not taken from cases as many were deemed to be too sick at the time (a large proportion were stages 3 and 4 cancer) to engage in invasive specimen collection [7]. The high risk HPV status of controls only was therefore verifiable. White women were initially included in the study, but as only nine white cases and 14 controls were enrolled in the first year, the study was confined to coloured and black women. After exclusions, there were 524 cases and 1541 controls. The mean age of cases and controls was 45 years and 44 years, respectively; 75% of the women were coloured.

Trained nurses administered a standardized questionnaire in a face-to-face interview in the subject's first language. Information was collected on a wide range of variables including demographic data; life-time contraceptive (including history of condom use as a marker of safer sex) and reproductive history (including parity and past history of own and partners' sexually transmitted infections); cervical dysplasia history; PAP smear history; weight; height; smoking (ever smoked; number of years smoked, if ceased smoking, time since smoked and number of cigarettes if current smoker); alcohol consumption; and sexual activity history including recognized key indicators of sexual behaviour [8] age of sexual debut (penetrative sexual intercourse) and number of lifetime sexual partners.

Age at first sex was an ordinal rather than a continuous variable collected three categories (< 16 years; 16–19 years and ≥ 20 years). This split was practically necessary to ensure adequate numbers by variable categories for analysis. Descriptive associations between the two main sexual activity indices that we created (age at first sex < 16 years and 3+ sexual partners) are presented as odd ratios derived from logistic regression analyses in Tables 2 and 3. Case-control methods were employed for data presented in Tables 4 and 5. Prevalence ratios and adjusted prevalence ratios were utilised to estimate risk in Tables 2 and 3 as some of these exposures were considered com-
Distributions of age of sexual debut and number of sexual partners were similar across major diagnostic categories (trauma, acute infection, other conditions) among controls.

Both tables 2 and 3 show socio-economic factors associated with earlier sexual debut and increased number of partners respectively. These are restricted to controls.

**Table 1: comparison of socio-demographic background of cases and controls**

| Characteristics       | Cases (N = 524) | Controls (N = 1541) |
|-----------------------|----------------|--------------------|
| **Mean age (and standard deviation)** |                 |                    |
| 45 years (8.5) N (%)  | 44 years (8.7) N (%) |
| **Residence**         |                 |                    |
| Rural                 | 221 (42)        | 712 (46)           |
| Urban                 | 303 (58)        | 829 (54)           |
| **Marital status**    |                 |                    |
| Marital               |                 |                    |
| Single                | 128 (25)        | 366 (23)           |
| Divorced/separated    | 80 (15)         | 229 (15)           |
| Widowed               | 76 (15)         | 190 (12)           |
| Married               | 239 (46)        | 786 (51)           |
| **Race**              |                 |                    |
| Black                 | 133 (25)        | 386 (25)           |
| Coloured              | 391 (75)        | 1155 (75)          |
| **Years of education**|                 |                    |
| ≤ 4 years             | 129 (25)        | 431 (28)           |
| 5–9 years             | 319 (61)        | 857 (56)           |
| ≥ 10 years            | 76 (15)         | 253 (16)           |

Mon (> 10%). These were estimated using an experimental Proc TPHREG in SAS 9.1.3. In the instance where the outcome is uncommon, as is the case in Tables 4 and 5 for cervical cancer risk, (estimated incidence of 30/100,000) crude and adjusted odd ratios were most likely to be a good estimate of relative risk. We assessed the association of potential cervical cancer risk factors with age at sexual debut using unconditional multiple logistic regression to adjust for confounding and estimated odds ratios for first sex at age ≤ 16; relative to first at a later age. Similar analyses assessed associations between potential risk factors and ≥ 3 lifetime sexual partners. Logistic regression assessed determinants of sexual activity to risk of cervical cancer. We controlled for age, race, marital status, progestogen only contraceptives (IPC) use, combined estrogen/progestogen contraceptive use (COC), years of formal education, life-time smoking and alcohol consumption and life-time pap smear history. Crude and adjusted relative risks (odd ratios) were estimated using multiple logistic regression analysis. The crude and adjusted relative risk estimates were similar and both are presented.

**Results**

**Determinants of sexual activity**

The median age of sexual debut was 17 years and the median number of lifetime sexual partners was 2 for cases and controls. As can be seen in Table 1 showing a comparison of socio-demographic background of cases and controls, women in both groups had similar characteristics: the mean age was 45 and 44 years respectively; urban residence 58% and 54% and having more 5 or more years of schooling. 76% and 72% respectively; the proportion of married women was 46% and 51% respectively and black and coloured women both 25% and 75% respectively.

The strongest risk association was for < 4 years of education, (APR = 4.1, 95% CI 2.6–6.6) relative to ≥ 10 years. Other factors significantly associated with age of sexual debut < 16 years, were ≥ 4 sexual partners relative to 1 (APR = 1.9, 95% CI 1.3–2.7) and current alcohol use relative to nonuse (APR = 1.4, 95% CI 1.1–1.8); and Age, marital status, race, residence, ever-use of hormonal contraceptives, ever had a Pap smear, smoking status and having current high risk HPV were not significantly associated with early sexual debut. As there was very little reported condom use, few women had knowledge of whether reproductive infections they had had were STI’s or not and few women knew whether their partners had had an STI, this had little impact on the analysis.

**Number of sexual partners**

Thirty-nine percent of controls had sex < 16 years. The chi square test for trend, an indicator of changes over time, was 1.33, p = 0.25, showing no evidence of a trend of younger sexual debut over time. Table 2 describes socio-demographic factors associated with earlier age at first intercourse and gives crude and adjusted prevalence ratios (APR) for age at first sexual intercourse < 16 years relative to first sex at age ≥ 17 among the controls.

The strongest risk association was for < 4 years of education, (APR = 4.1, 95% CI 2.6–6.6) relative to ≥ 10 years. Other factors significantly associated with age of sexual debut < 16 years, were ≥ 4 sexual partners relative to 1 (APR = 1.9, 95% CI 1.3–2.7) and current alcohol use relative to nonuse (APR = 1.4, 95% CI 1.1–1.8); and Age, marital status, race, residence, ever-use of hormonal contraceptives, ever had a Pap smear, smoking status and having current high risk HPV were not significantly associated with early sexual debut. As there was very little reported condom use, few women had knowledge of whether reproductive infections they had had were STI’s or not and few women knew whether their partners had had an STI, this had little impact on the analysis.
Sexual activity and cervical cancer risk

Table 4 shows cervical cancer risk, as crude and adjusted odds ratios, in relation to the age at first sexual intercourse.

Relative to those who commenced sexual intercourse at age ≥ 20 years, the multivariate adjusted odds ratio for age at commencement at age < 16 years was 1.6 (95% CI, 1.2–2.2) (p = 0.0006); among coloured and black women the estimates were 1.7 (95% CI, 1.2–2.4) (p = 0.002) and 1.3 (95% CI 0.7–2.4) respectively.

Table 5 shows cervical cancer risk, as crude and adjusted odds ratios, in relation to the number of lifetime partners.
The multivariate adjusted OR increased with increasing number of sexual partners, to 1.7 (95% CI, 1.2 – 2.2) (p = 0.0001) for ≥ 4 lifetime sexual partners relative to 1. The adjusted OR for cervical cancer in women who had ≥ 4 lifetime sexual partners was 2.0 (95% CI 1.4–2.9) (p < 0.0001) among coloured women and 1.2 (95% CI, 0.6–2.3) among black women. When high risk HPV positive controls only were included (16% of the women), the results were unchanged.

Discussion

Determinants of sexual activity

A dearth of information exists on adult women's sexual activity in developing countries. In South Africa cancer of the cervix is a major public health problem and there are
high sustained prevalence levels of the oncogenic types of HPV associated with cervical cancer among older women in the country [6]. Information on sexual activity and its determinants are therefore important.

In this study of South African black and coloured women, as for other recent youth studies, [9,10] the median age of sexual debut was 17 for cases and the controls. In keeping with recent international data, our results show no trend of younger sexual debut over time [8]. In addition, age of sexual debut is similar to that shown in a study bringing together international data, including from developed countries [8].

Mostly strongly associated with early sexual debut were lower education, greater number of lifetime sexual partners and alcohol use. Women with low educational levels may have poorer knowledge and a less control over reproductive health decisions.

**Table 4: Cervical cancer risk in relation to age of sexual debut**

| Age of sexual debut | Cases (N = 524) | Controls (N = 1541) | Crude OR CI (95%) | Adjusted OR* CI (95%) |
|---------------------|----------------|---------------------|-------------------|----------------------|
| All women**         |                |                     |                   |                      |
| ≥ 20 years          | 76 (15)        | 305 (20)            | [1.0]**         | [1.0]**              |
| 16–19 years         | 266 (51)       | 838 (54)            | 1.3 (1.0–1.7)    | 1.1 (0.8–1.5)        |
| < 16 years          | 168 (32)       | 357 (23)            | 1.9 (1.4–2.6)    | 1.6 (1.2–2.2)        |
| Coloured women+     |                |                     |                   |                      |
| ≥ 20 years          | 60 (15)        | 265 (23)            | [1.0]**         | [1.0]**              |
| 16–19 years         | 200 (51)       | 616 (53)            | 1.4 (1.0–2.0)    | 1.2 (0.9–1.7)        |
| < 16 years          | 127 (33)       | 268 (23)            | 2.1 (1.5–3.0)    | 1.7 (1.2–2.4)        |
| Black women++       |                |                     |                   |                      |
| ≥ 20 years          | 16 (12)        | 40 (10)             | [1.0]**         | [1.0]**              |
| 16–19 years         | 66 (50)        | 222 (58)            | 0.7 (0.4–1.4)    | 0.9 (0.5–1.4)        |
| < 16 years          | 41 (31)        | 89 (23)             | 1.2 (0.6–2.3)    | 1.3 (0.7–2.4)        |

* Adjusted for number of sexual partners controlled for age, race, marital status, hormonal contraceptive use, years formal education, residence, ever smoked, ever alcohol consumption and ever having had a Pap smear
** 5 & 8 women with missing or unknown values for cases and controls respectively are not shown
*** Reference category
++ 4 & 6 women with missing or unknown values for cases and controls and 10 & 35 women with missing or unknown values for cases and controls respectively are not shown

**Table 5: Cervical cancer risk in relation to the number of lifetime partners**

| Number of lifetime sexual partners | Cases (n = 524) | Controls (n = 1541) | Crude OR CI (95%) | Adjusted OR* CI (95%) |
|-----------------------------------|----------------|---------------------|-------------------|----------------------|
| All women**                       |                |                     |                   |                      |
| 1                                 | 106 (20)       | 417 (27)            | [1.0]**         | [1.0]**              |
| 2                                 | 157 (30)       | 521 (34)            | 1.2 (0.9–1.6)    | 1.1 (0.8–1.5)        |
| 3                                 | 123 (23)       | 310 (20)            | 1.6 (1.2–2.1)    | 1.4 (1.1–1.9)        |
| ≥ 4                               | 133 (25)       | 285 (18)            | 1.8 (1.4–2.5)    | 1.7 (1.2–2.2)        |
| Coloured women+                   |                |                     |                   |                      |
| 1                                 | 94 (24)        | 370 (32)            | [1.0]**         | [1.0]**              |
| 2                                 | 124 (32)       | 427 (37)            | 1.1 (0.8–1.5)    | 1.1 (0.8–1.5)        |
| 3                                 | 92 (24)        | 213 (18)            | 1.7 (1.2–2.4)    | 1.5 (1.1–2.2)        |
| ≥ 4                               | 80 (20)        | 142 (12)            | 2.2 (1.6–3.2)    | 2.0 (1.4–2.9)        |
| Black women++                     |                |                     |                   |                      |
| 1                                 | 12 (9)         | 47 (12)             | [1.0]**         | [1.0]**              |
| 2                                 | 33 (25)        | 94 (24)             | 1.4 (0.7–2.9)    | 1.2 (0.6–2.3)        |
| 3                                 | 31 (23)        | 97 (25)             | 1.3 (0.6–2.7)    | 1.0 (0.5–2.1)        |
| ≥ 4                               | 53 (40)        | 143 (37)            | 1.5 (0.7–2.9)    | 1.2 (0.6–2.3)        |

* Adjusted for age of sexual debut controlled for age, race, marital status, use of hormonal contraceptives, years of formal education, area of residence, ever having smoked, ever having drunk alcohol and ever having had a Pap smear
** 5 & 8 women with missing or unknown values for cases and controls respectively are not shown
*** Reference category
++ 4 & 6 women with missing or unknown values for cases and 10 & 35 women with missing or unknown values for cases and controls respectively are not shown

...
ductive and sexual decision-making [11]. While it may seem paradoxical that an early sexual debut is associated with lower educational levels while having a greater number of partners is associated with the inverse, this is not necessarily the case. Higher education may increase a woman’s financial autonomy, allowing her to have greater independence in choice in intimate relationships and hence increase her number of life-time partners. Early pregnancy limits women’s educational prospects and may in turn limit employment opportunities and create lower perceptions of life chances and decreased incentive to delay childbirth [11,12]. Urban and rural South African studies have found education, employment and earning opportunities significantly reduced risk-taking behavior [13,14]. Less educated young women may face greater constraints engaging in safer sex [15]. Alcohol use could indicate riskier life styles. While smoking and cervical cancer risk has been associated in some studies, [16,17] we found no association, but the median number of cigarettes per day reported by smokers in this study was low.

Factors most strongly associated with having more sexual partners were being single, black, more educated, use of alcohol, and young age at first sex. Being single creates more opportunity for having more partners, as does younger sexual debut. Higher education may increase women’s financial autonomy, affecting number of sexual partners.

Black women’s sexual behaviour may be influence by historical socio-political inequality. Apartheid migratory labour practices and the pass laws that controlled the movement of Black South Africans, severely restricting movement outside of rural areas, created separation of black households, placing women at risk for multiple sexual partners [18,19]. Women’s economic constraints also led to a greater prevalence of women in urban areas engaging in transactional sex [18]. Current infection with high risk HPV was not found to be significantly associated with sexual risk behaviour. While it would be expected that the two variables studies would show an association, it was possible in this study only to ascertain current and not life-time exposure to HPV infection.

**Sexual activity and cervical cancer risk**

In agreement with previous studies conducted internationally early age of sexual debut and a large numbers of sexual partners were associated with increased cervical cancer risk in the present study [21-23]. The ORs were larger for coloured women than for black women. We may have expected a similar association between sexual activity and cervical risk for both groups. However, the number of black women was limited, confidence intervals were wide, and odds ratios in the two groups were compatible with a uniform value. An alternative explanation may be confounding by the sexual activity of the male partners. Because of migratory labour, black women’s male partners may have more partners than coloured women’s male partners, increasing the risk of transmission of HPV infection, and of cervical cancer. We had no data on male partners’ sexual activity. Studies examining male partners’ their sexual behaviour show these to have a greater impact on cervical cancer risk than the women’s own behaviour. [24,25]. Data collected on contraceptive history included condom use. However this was very low among this population of women. Data was also collected on whether women and their partners had ever had a sexually transmitted infection, with no marked effects evident, possibly due to the high life time prevalence of reproductive infections among women. The information on partners’ STI’s was unlikely to be reliable. The study could only obtain endocervical scrapings for HPV from controls and not cases, due to the latter presenting in South Africa with advanced disease [7]. Hence any attenuation of the sexual behavior through inclusion of HPV status in the multivariate models could not be verified.

Controls in this study were age matched cases of cervical cancer, a disease more common in older women. Hence the study is limited in making inferences about younger women since only a small percent of the study population (6%) was under the age of 30 years.

It is important to consider whether the present findings are due to bias. Refusal rates were low, reducing the possibility of selection bias. Additionally, the distributions of sexual activity were similar across the diagnostic categories in the controls, suggesting that no bias in the selection of controls. Regarding detection bias, most cases of women with cervical cancer would be treated at hospitals monitored by the study, because the study was confined to women with invasive cancer (Stage I B – IV) for whom diagnosis and hospital admission was inevitable. To guard against information bias, standardized interviews were administered to cases and controls in similar settings. However, questions about sexual activity are intrinsically sensitive and sexual activity has many determinants such as culture, religion and socio-economic status that may limit precision. Thus the potential for information bias is acknowledged. Confounding is also a possibility. While we controlled for major risk factors, we could not control and rule out confounding for sexual activity of the male partners.

**Conclusion**

This study provides valuable information on patterns and determinants of sexual activity in a population of South African adult women. This is relevant in understanding the high levels of cervical cancer among women in South Africa and other developing countries and more broadly...
in examining STI acquisition, including HIV that has high prevalence levels among older South African women in their reproductive years [26]. In this study low socio-economic status, alcohol intake, single marital status and being black, most likely related to historical socio-political inequality, appear to be determinants of increased sexual activity. As expected, such activity was also associated with an increased risk of cervical cancer, confirming the findings of studies conducted in other countries. They suggest that initiatives to encourage later commencement of sex, and limit the number of sexual partners would have a favourable impact on risk of cancer of the cervix and other STI's, including HIV. The findings underscore the importance of calls made elsewhere for public health interventions for sexual risk reduction to pay greater attention to the social context in which sexual activity occurs [8].

**Competing interests**
The author(s) declare that they have no competing interests.

**Authors’ contributions**
All authors have been involved either in the conception, design of the study and/or in the analysis and interpretation of data. SS, LR, MH were involved in the conception and design of the study. DC, MH, AW, LD & GD were involved in analysis and design of the study. SS, LR, MH were involved in the conception and/or in the analysis and interpretation of data. The first author drafted the manuscript all authors have been involved in critically revising the article for important intellectual content and have given final approval to the version to be published.

**Acknowledgements**
We are indebted to our clinical colleagues at provincial hospitals, clinics and health centres in the Western Cape for their co-operation and support. We would also like to acknowledge the fieldworkers, Beverley Arendse, Vanessa Daries, Phoebe Gribble and Lungiswa Mankayi and the administrator/research assistant, Eleanor Marks for their contributions. This study was supported by a grant from the National Cancer Institute (USA) – Grant no. R01 CA 73985.

**References**

1. Parkin DM, Pisani P, Ferlay J: Estimates of world wide incidence of 25 major cancers in 1990. Int J Cancer 1999, 80:827-841.

2. Mqoqi M, Kelly P, Sitas F, Musa J: Incidence of histologically diagnosed cancer in South Africa 1998–9. National Cancer Registry of South Africa Johannesburg 2004.

3. Herrero R, Schiffman R, Bratti C, Hildesheim A, Morales J, Alfaro M, Sherman ME, Wacholder S, Chen S, Rodriguez AC, Burk RD: Epidemiology of type-specific HPV infection in Guanacaste. Proceedings of HPV Conference 2002.

4. Herrero R, Britton L, Reeves WC, Brenes MM, Tenorio F, de Britton RC, Gaitan E, Garcia M, Rawls WE: Sexual Behaviour, Venereal Diseases, Hygiene Practices and Invasive Cervical Cancer in a High-Risk Population. Cancer 1990, 65:380-386.

5. Slattery M, Overall JC Jr, Abbott TM, French TK, Robison M, Gardner J: Sexual Activity Contraception, Genital infections and Cervical Cancer: Support for a sexually transmitted disease hypothesis. Am J Epidemiol 1989, 130(2):248-259.

6. Allan BR, Marais DJ, Denny L, Hoffman M, Shapiro S, Williamson AL: The agreement between cervical abnormalities identified by cytology and detection of high-risk types of human papillomavirus. S Afr Med J 2006, 96(11):1186-1190.

7. Shapiro S, Rosenberg L, Hoffman M, Kelly J, Cooper D, Carrara H, Denny LE, du Toit G, Allan BR, Stander IA, Williamson A-L: Risk of invasive cancer of the cervix in relation to use of injectable progestogen contraceptives and combined estrogen/progestogen contraception. Cancer Causes and Control 2003, 14:485-495.

8. Wellings K, Collumbien M, Slaymaker E, Singh S, Hodges Z, Patel D, Bajos N: Sexual behaviour in context: a global perspective. The Lancet 2006, 368(9548):1706-1728.

9. Department of Health in South Africa: South African Demographic and Health Survey. Pretoria 2001.

10. Petoefor A, Rees H, Stevens A: HIV & Sexual Behaviour Among Young South Africans: A National Survey of 15–24 Year Olds. Johannesburg: University of the Witswatersrand; 2004.

11. Mamadani M, Garner P, Harpham T, Campbell O: Review article. Fertility and contraceptive use in poor urban areas of developing countries. Health Policy Plan 1993, 14:199-209.

12. Klugman B: Balancing means and ends: population policy in South Africa. Reprod Health Matters 1993, 1:44-57.

13. Eaton L, Fisher AJ, Aaro L: Unsafe sexual behaviour in South African youth. Soc Sci Med 2003, 56:149-165.

14. Kaufman CE, Clark S, Manzini, May J: Communities, opportunities and adolescents' sexual behaviour in KwaZulu Natal, South Africa. Stud Fam Plan 2004, 35:261-274.

15. Jewkes R, Levin JB, Penn-Kekana L: Gender inequalities, intimate partner violence and HIV preventive practices: findings of a South African cross-sectional study. Soc Sci Med 2003, 56:125-134.

16. Roth LK, Taylor HS: Risks of smoking to reproductive health: Assessment of women's knowledge. Am J Obstet Gynecol 2001, 184(5):935-939.

17. Zivlajevic B, Vlajinac H, Adanja B, Zivlajevic V, Kocev N: Smoking as a risk factor for cervical cancer. Neoplasma 2001, 48(4):254-256.

18. Ramphelhe M: The dynamics of gender politics in the hostels of Cape Town – Another legacy of the South African Migrant Labour system. J South Afr Stud 1989, 15(3):393-414.

19. Murray C: Migration and changing family structure in the rural periphery of Southern Africa. J South Afr Stud 1980, 6(2):139-156.

20. Caldwell J, Caldwell P, Quiggin P: The social context of AIDS in Subsaharan Africa. Popul Dev Rev 1989, 15:185-234.

21. Biwas LH, Manna B, Maiti PK, Sengupta S: Sexual Risk Factors for Cervical Cancer among Rural Indian Women: A Case-Control Study. Int J Epidemiol 1997, 26(3):491-495.

22. Williams MA, Keny Pr, Maji JK, Thomas DB: Risk Factors for Invasive Cervical Cancer in Kenyan Women. Int J Epidemiol 1999, 23(5):906-912.

23. Stone KM, Zaidi A, Rosero-Bixby L, Oberle MW, Reynolds G, Larsen S, Nahmias J, Lee FK, Schachter J, Guinan ME: Sexual Behaviour, Sexually Transmitted Diseases, and Risk of Cervical Cancer. Epidemiology 1995, 6:409-414.

24. Britton LA, Reeves WC, Brenes MM, Herrero R, Gaitan E, Tenorio F, de Britton RC, Garcia M, Rawls WE: The male factor in the etiology of cervical cancer among sexually monogamous women. Int J Cancer 1989, 44:199-203.

25. Agarwal SS, Sehgal A, Sardana S, Kumar A, Luthra UK: The role of male behaviour in cervical carcinogenesis among women with one life-time sexual partner. Cancer 1993, 72(5):1666-9.

26. Department of Health, Republic of South Africa: Summary Report. National HIV and Syphilis Antenatal Seroprevalence Survey in South Africa 2005. Pretoria 2006.

**Pre-publication history**
The pre-publication history for this paper can be accessed here:

http://www.biomedcentral.com/1471-2458/7/341/prepub