Title
Cervical cancer incidence after up to 20 years of observation among women with HIV

Journal
International Journal of Cancer, 141(8)

ISSN
0020-7136

Authors
Massad, L Stewart
Hessol, Nancy A
Darragh, Teresa M
et al.

Publication Date
2017-10-15

DOI
10.1002/ijc.30866

Peer reviewed

IJC
International Journal of Cancer

Cervical cancer incidence after up to 20 years of observation among women with HIV

L. Stewart Massad[1], Nancy A. Hessol[2], Teresa M. Darragh[3], Howard Minkoff[4], Christine Colie[5], Rodney L. Wright[6], Mardge Cohen[7] and Eric C. Seaberg[8]

[1] Department of Obstetrics & Gynecology, Washington University School of Medicine, St. Louis, MO
[2] Department of Clinical Pharmacy, University of California, San Francisco, CA
[3] Departments of Pathology and of Obstetrics, Gynecology and Reproductive Science, University of California, San Francisco, CA
[4] Department of Obstetrics & Gynecology, Maimonides Medical Center, Brooklyn, NY
[5] Department of Obstetrics & Gynecology, Georgetown University, Washington, DC
[6] Department of Obstetrics & Gynecology and Women's Health, Montefiore Medical Center, Bronx, NY
[7] Departments of Medicine/CORE Center, John H. Stroger Hospital, Cook County Bureau of Health Services, Chicago, IL
[8] Department of Epidemiology, Johns Hopkins Bloomberg School of Public Health, Baltimore, MD

To estimate the incidence of invasive cervical cancer (ICC) across up to 21 years of follow-up among women with human immunodeficiency virus (HIV) and to compare it to that among HIV-uninfected women, we reviewed ICC diagnoses from a 20-year multi-site U.S. cohort study of HIV infected and uninfected women who had Pap testing every 6 months. Incidence rates were calculated and compared to those in HIV-negative women. Incidence ratios standardized to age-, sex-, race-, and calendar-year specific population rates were calculated. After a median follow-up of 12.3 years, four ICCs were confirmed in HIV seropositive women, only one in the last 10 years of observation, and none in seronegative women. The ICC incidence rate did not differ significantly by HIV status (HIV seronegative: 0/100,000 person-years *vs*. HIV seropositive: 19.5/100,000 person-years; $p = 0.53$). The standardized incidence ratio for the HIV-infected WIHS participants was 3.31 (95% CI: 0.90, 8.47; $p = 0.07$). Although marginally more common in women without HIV, for those with HIV in a prevention program, ICC does not emerge as a major threat as women age.

Infection with the human immunodeficiency virus-1 (HIV) increases risk for human papillomavirus (HPV) infection, abnormal Pap tests, cervical intraepithelial neoplasia (CIN), and recurrence of CIN after treatment.[1–5] Rates of invasive cervical cancer (ICC) among women with HIV also appear to be higher than those among HIV-negative women.[6–8] In the US HIV Epidemiology Research Study, HIV-infected women had a risk for ICC that was eight-fold higher than US age and race adjusted incidence.[9] Though the increase is less dramatic than that seen for other cancers that define the acquired immune deficiency syndrome (AIDS) or anal cancer in men, elevated risk persists late after AIDS onset.[10]

Longer life expectancy due to effective combination antiretroviral therapy (cART) may allow persistent HPV infections

Key words: cervical cancer, HIV in women, cancer prevention

Abbreviations: AIDS: acquired immune deficiency syndrome; cart: combination antiretroviral therapy; CIN: cervical intraepithelial neoplasia; HIV: human immunodeficiency virus; ICC: invasive cervical cancer; SEER: surveillance epidemiology and end results; SIR: standardized incidence ratio; WIHS: women's interagency HIV study

Grant sponsor: UAB-MS WIHS; **Grant number:** U01-AI-103401; **Grant sponsor:** Atlanta WIHS; **Grant number:** U01-AI-103408; **Grant sponsor:** Bronx WIHS; **Grant number:** U01-AI-035004; **Grant sponsor:** Brooklyn WIHS; **Grant number:** U01-AI-031834; **Grant sponsor:** Chicago WIHS; **Grant number:** U01-AI-034993; **Grant sponsor:** Metropolitan Washington WIHS (Seble Kassaye); **Grant number:** U01-AI-034994; **Grant sponsor:** Miami WIHS (Margaret Fischl and Lisa Metsch); **Grant number:** U01-AI-103397; **Grant sponsor:** UNC WIHS (Adaora Adimora); **Grant number:** U01-AI-103390; **Grant sponsor:** Connie Wofsy Women's HIV Study, Northern California; **Grant number:** U01-AI-034989; **Grant sponsor:** WIHS Data Management and Analysis Center; **Grant number:** U01-AI-042590; **Grant sponsor:** Southern California WIHS; **Grant number:** U01-HD-032632, UL1-TR000004, UL1-TR000454; **Grant sponsors:** National Institute of Allergy and Infectious Diseases (NIAID); Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD); the National Cancer Institute (NCI); the National Institute on Drug Abuse (NIDA); the National Institute on Mental Health (NIMH); National Institute of Dental and Craniofacial Research (NIDCR); the National Institute on Alcohol Abuse and Alcoholism (NIAAA); the National Institute on Deafness and other Communication Disorders (NIDCD); NIH Office of Research on Women's Health
DOI: 10.1002/ijc.30866
History: Received 28 Feb 2017; Accepted 21 June 2017; Online 3 July 2017

Correspondence to: Dr. Massad, Division of Gynecologic Oncology, Washington University School of Medicine, P.O. Box 8064, St. Louis, MO 63110, E-mail: massadl@wudosis.wustl.edu; Tel.: 314-362-3181; Fax: 314-362-2893

What's new?

Women infected with HIV face higher risks for carcinogenic human papillomavirus (HPV) infection and pre-cancer. Longer life expectancy due to effective combination antiretroviral therapy may allow persistent HPV infections to progress to cancer. Here, the authors show that HIV infection only minimally raises invasive cervical cancer (ICC) risk when women are enrolled in care that includes intensive screening and protocol-based referral to treatment. Cervical cancer has not emerged as a major cause of morbidity and mortality in such a prevention program, underscoring the importance of regular screening and assiduous treatment of ICC precursors in women with HIV.

to progress to cancer, although Pap screening and precursor treatment may block this. Introduction of cART has led to reductions in other AIDS-defining illnesses, but this may not be true for ICC.[11] In Italy, ICC incidence rose early in the cART era.[12] Decreasing competing mortality from other AIDS-defining diseases among HIV infected women may lead to an increase in cervical cancer incidence.

We have shown that cervical cancer incidence in a heavily screened U.S. study cohort study remains low during the first decade of observation,[13] but risk over longer periods is unknown. Studies showing increased cervical cancer risk have some limitations: case verification in these studies may have been limited, as most studies were registry based and did not confirm cases through central slide review. Also, some studies did not distinguish prevalent cases detected after HIV diagnosis and entry into care from incident cases. Unfortunately, some registry cases actually represent CIN or abnormal cytology miscoded as ICC and so may lead to overestimates of ICC incidence.[14]

To address limitations to prior studies that provide contradictory data on the long term risk of ICC among women with HIV, we set out to reassess our findings in an expanded study with follow-up extending up to 20 years.

Methods

This investigation was part of the Women's Interagency HIV Study (WIHS), a multicenter prospective cohort study of the health US women with and at risk for HIV infection.[15,16] WIHS enrollment began with six study consortia enrolling 2,623 women in 1994–5. Written informed consent was obtained from all participants, with approval from local human subjects committees. Follow-up continues, but this analysis includes only follow-up information obtained before October 1, 2015.

Every 6 months, demographic, behavioral, and health information was updated, including diagnoses of ICC made since the prior visit, as well as a physical examination and conventional Pap smear. HIV status was established by Western blot. Pap tests were interpreted centrally according to the 1991 Bethesda system for classification of cervicovaginal cytology.[17] Study protocol required referral for colposcopy for squamous abnormalities of any grade, including atypia, though decisions about biopsy and diagnostic or therapeutic excision were individualized.

Regional cancer registries were computer matched for all participants and records were reviewed to obtain case histories; women whose histories refuted a cancer diagnosis were considered not to have ICC. Women also were considered not to have ICC when primary source reports listed a diagnosis other than ICC or when central slide review failed to confirm ICC. Slides could not be retrieved for one woman after a fire destroyed records and for one woman treated for ICC outside her study site; as a conservative measure, incidence was calculated including both as cases of ICC.

We excluded 222 women who reported having had a hysterectomy prior to enrollment, 7 with cervical cancer prior to study entry, and 99 with no follow-up. Thus, the study cohort consisted of 2,295 women.

ICC incidence rates were computed as the number of observed incident ICC over the total person-years of observed follow-up, expressed per 100,000 person-years. The follow-up time available for any woman was the number of years from the enrollment visit until diagnosis of ICC, death from causes other than ICC, loss to follow-up, incident hysterectomy, or the censoring date of September 30, 2015, whichever occurred first. The observed ICC incidence rates for HIV seropositive and seronegative women were estimated assuming the Poisson distribution and compared statistically using exact Poisson regression.

Comparison to the US population was undertaken by determining the number of ICCs expected based on age, sex, race, and calendar year specific rates for women between 1994–2013, which were obtained from the 1973–2013 Surveillance Epidemiology and End Results database (SEER),[18] and then computing standardized incidence ratios (SIRs,[19]) and exact 95% confidence intervals (CIs,[20]). SEER data from 2013 were used to determine the expected number of ICCs in WIHS for 2013–2015. All analyses were performed using SAS 9.4 (SAS Institute, Cary, NC) or LogXact 10.0 (Cytel Corporation, Cambridge, MA). All statistical tests were two-sided, and statistical significance was inferred from p values <0.05.

Results

For the 2,295 women studied, mean follow-up time was 11.8 years (median 12.3 years, interquartile range [IQR] 4.4–20.1 years). Median follow-up among among HIV seronegative women was 17.4 years (IQR 6.9–20.5 years) and among women with HIV was 10.6 years (IQR 3.9–19.0 years), somewhat truncated by early mortality prior to introduction of cART. The longest observation time was 21.4 years, and 25.5% of included women were followed for at least 20 years.

Characteristics of the 1807 HIV seropositive and 488 HIV seronegative women in the study cohort are shown in Table 1.

Table 1. Baseline characteristics of women at risk for invasive cervical cancer in the Women's Interagency HIV Study ($N = 2,504$)

	HIV positive		HIV negative	
Characteristic	***N***	**%**	***N***	**%**
All	1,807	100.0	488	100.0
Age (years) $p < 0.001$				
<30	382	21.1	158	32.4
30–39	919	50.9	209	42.8
40–49	450	24.9	107	21.9
50+	56	3.1	14	2.9
Median (interquartile range)	35.9 (30.8, 40.6)		34.2 (27.6, 40.0)	
Race/ethnicity $p = 0.08$				
Non-Hispanic African-American	975	54.0	257	52.7
Non-Hispanic Caucasian	341	18.9	74	15.2
Hispanic	139	24.5	139	28.5
Other	18	2.7	18	3.7
Follow-up time (years)				
Median (interquartile range)	10.6 (3.9, 19.0)		17.4 (6.9, 20.5)	

Median age at enrollment was 35.9 years for seropositive and 34.2 years for seronegative women.

No cases of ICC were observed among 488 HIV seronegative women during 6,615 person-years of observation.

Four cases of ICC were observed and confirmed among 1,807 women with HIV women during 20,561 person-years of observation, an incidence rate of 19.5 cases/100,000 person-years ($p = 0.53$ compared to HIV seronegative women. Three have been previously reported.[12,13]

One additional case was identified since our last report. This woman had multifocal cervical, vulvar, and vaginal intraepithelial neoplasia was treated with multiple modalities prior to diagnosis of a Stage IA2 squamous cell carcinoma at 40 years of age. She was treated with radical hysterectomy; pathologic assessment showed only residual carcinoma *in situ*. She required additional therapies for preinvasive disease and was diagnosed with recurrence *vs.* a new invasive squamous carcinoma of the vagina in 2011. This was managed with chemoradiotherapy, and the patient remained in remission at last follow-up in May 2016.

Table 2 shows ICC incidence by HIV serostatus, age, and race/ethnicity. The overall ICC incidence rate was 14.7/100,000 person-years (95% C.I. 4.0, 37.7). We did not identify significant differences in ICC incidence in subgroups, including by HIV serostatus (not shown).

On the basis SEER data, we expected a total of 1.21 ICCs among women with HIV 0.37 among HIV seronegative women. The corresponding SIRs 2343 3.31 (95% C.I. 0.90, 8.47, $p = 0.07$) among women with HIV and 0.0 (95% C.I. 0.0, 10.10, $p = 1.0$) among HIV seronegative women. The SIR for the entire WIHS cohort was 2.54 (95% CI: 0.69, 6.50; $p = 0.15$).

Discussion

Most women with HIV will be diagnosed with HPV, and almost 15% will develop high grade CIN,[1–4] but when enrolled in an organized program of Pap testing and treatment of precursors, ICC risk in women living with long-term HIV infections remains low. In fact, risk is so low that despite >20,000 person-years of observation during follow-up extending often over two decades, ICC incidence among HIV seropositive women in WIHS remained statistically indistinguishable from both HIV seronegative comparison women in our study. Compared to the US population generally, as measured by adjusted SEER incidence, ICC risk was three-fold greater but did not reach statistical signficance. Competing risks in our cohort are much greater, including other AIDS-related causes of death, liver and cardiovascular disease, non-AIDS related cancers and accident, suicide and homicide.[21] While vigilance is needed and multiple procedures including hysterectomy may be required, ICC is a preventable complication of HIV infection. Women who do develop cancer often have missed opportunities for screening and precursor treatment, as we and others have previously reported,[13,22] or repeated episodes of slowly progressive cervical disease, as in the case reported here. Vigilance and repeated therapy may be required to reduce ICC rates among women with HIV.

Despite the lack of statistical significance, we believe that the roughly three-fold increased risk of ICC associated with HIV infection observed in our cohort is real, given consistent increased incidence rates found in other studies. The increased risk of ICC that we observed in HIV seropositive WIHS women is lower than that reported in other recent North American studies.[8,23] This is likely due to several factors. All WIHS participants had Pap testing performed at 6-

Table 2. Incidence of invasive cervical cancer (ICC) by selected characteristics

Characteristic	Number ICCs	Person-Years	Incidence rate per 100,000 PYs[1] (exact 95% CI)	Exact *p* value
All	4	27,175	14.7 (4.0, 37.7)	
HIV status				0.58
Negative	0	6,615	0.0 (0.0, 57.8)	
Positive	4	20,561	19.5 (5.3, 49.8)	
Age (years)				0.56
<30	0	1,855	0.0 (0.0, 198.9)	
30–39	1	8,174	12.2 (0.3, 68.2)	
40–49	3	10,781	27.8 (5.7, 81.3)	
50+	0	6,365	0.0 (0.0, 58.0)	
Race/ethnicity				1.0
Non-Hispanic African-American	2	14,647	13.7 (1.7, 49.3)	
Non-Hispanic Caucasian	1	4,888	20.5 (0.5, 114.0)	
Hispanic	1	6,788	14.7 (0.4, 82.1)	
Other	0	852	0.0 (0.0, 433.0)	

[1]Person-years.

month intervals, with protocol-defined referral for colposcopy for any cytologic abnormality and with organized mechanisms in place to maximize compliance with referral for treatment. Women outside supportive cancer prevention systems may face a higher ICC risk. The WIHS has identified substantial increased risks for other cancers, including non-Hodgkin's lymphoma, Kaposi's sarcoma, and cancers of the anus and oral cavity/pharynx, further support for the concept that low ICC rates in WIHS result from screening and treatment of precursors that has aborted a higher risk for ICC, rather than representing inadequate case ascertainment.

Other factors may have contributed to our lower observed ICC risk compared to prior studies. A French cohort study of persons with HIV showed that cervical cancer risk declined over >15 years of observation,[24] and antiretroviral therapy reduces HPV expression and abnormal cervical cytology,[25] and since most of our observation period was during the cART era, treatment might have reduced ICC risk compared to that existing prior to cART introduction. Registry-based studies do not include report and slide review for case confirmation; in previous analyses of the incidence of ICC in WIHS we found that several reported ICC cases were actually intraepithelial lesions or abnormal Pap tests, not cancers.[13,14] We excluded ICC cases identified within 6 months of enrollment as prevalent lesions in order to better estimate cancer incidence. Finally, cART appears to reduce cervical disease, and careful monitoring of cART use in our observed cohort may have impacted cervical disease progression.[26] A recent Danish cohort study found that women with HIV who were adherent to the national organized cervical cancer screening program did not face an elevated risk of ICC compared to seronegative controls,[22] supporting our results.

While broadly reassuring, our results underscore the importance of regular screening and assiduous treatment of ICC precursors in women with HIV. Cytologic abnormalities and precancer are common,[2–4] compliance with colposcopy is suboptimal and WIHS has developed formal procedures for recall,[27] significant knowledge gaps relevant to cervical cancer patients are refractory to simple educational interventions,[28] and treatment often fails to eradicate HPV-related disease.[29] To achieve similar results outside study settings, clinicians caring for women living with HIV must apply current cervical screening protocols diligently and establish clinical pathways to colposcopy, therapeutic excision, and post-treatment surveillance, while educating women about the hope that these interventions will reduce ICC risk.

Acknowledgement

Data in this manuscript were collected by the Women's Interagency HIV Study (WIHS). The contents of this publication are solely the responsibility of the authors and do not represent the official views of the National Institutes of Health (NIH). WIHS (Principal Investigators): UAB-MS WIHS (Michael Saag, Mirjam-Colette Kempf, and Deborah Konkle-Parker), U01-AI-103401; Atlanta WIHS (Ighovwerha Ofotokun and Gina Wingood), U01-AI-103408; Bronx WIHS (Kathryn Anastos), U01-AI-035004; Brooklyn WIHS (Howard Minkoff and Deborah Gustafson), U01-AI-031834; Chicago WIHS (Mardge Cohen and Audrey French), U01-AI-034993; Metropolitan Washington WIHS (Seble Kassaye), U01-AI-034994; Miami WIHS (Margaret Fischl and Lisa Metsch), U01-AI-103397; UNC WIHS (Adaora Adimora), U01-AI-103390; Connie Wofsy Women's HIV Study, Northern California (Ruth Greenblatt, Bradley Aouizerat, and Phyllis Tien), U01-AI-034989; WIHS Data Management and Analysis Center (Stephen Gange and Elizabeth Golub), U01-AI-042590; Southern California WIHS (Alexandra Levine and Marek Nowicki), U01-HD-032632 (WIHS I – WIHS IV). The WIHS is funded primarily by the National Institute of Allergy and Infectious Diseases (NIAID), with additional co-funding from the Eunice Kennedy Shriver National Institute of Child Health and Human Development (NICHD), the National Cancer Institute (NCI), the National Institute

on Drug Abuse (NIDA), and the National Institute on Mental Health (NIMH). Targeted supplemental funding for specific projects is also provided by the National Institute of Dental and Craniofacial Research (NIDCR), the National Institute on Alcohol Abuse and Alcoholism (NIAAA), the National Institute on Deafness and other Communication Disorders (NIDCD), and the NIH Office of Research on Women's Health. WIHS data collection is also supported by UL1-TR000004 (UCSF CTSA) and UL1-TR000454 (Atlanta CTSA).

References

1. Palefsky JM, Minkoff H, Kalish LA, et al. Cervicovaginal human papillomavirus infection in human immunodeficiency virus-1 (HIV)-positive and high-risk HIV-negative women. *J Natl Cancer Inst* 1999;91:226–36.
2. Massad LS, Seaberg EC, Wright RL, et al. Squamous cervical lesions in women with human immunodeficiency virus: long-term follow-up. *Obstet Gynecol* 2008;111:1388–93.
3. Strickler HD, Burk RD, Fazzari M, et al. Natural history and possible reactivation of human papillomavirus in human immunodeficiency virus (HIV) positive women. *J Natl Cancer Inst* 2005; 97:577–86.
4. Massad LS, Xie X, D'souza G, et al. Incidence of cervical precancers among HIV-seropositive women. *Am J Obstet Gynecol* 2015;212:606.e1–8.
5. Massad LS, Fazzari MJ, Anastos K, et al. Outcomes after treatment of cervical intraepithelial neoplasia among women with HIV. *J Low Genit Tract Dis* 2007;11:90–7.
6. Robbins HA, Pfeiffer RM, Shiels MS, et al. Excess cancers among HIV-infected people in the United States. *J Natl Cancer Inst* 2015;107:dju503. doi: 10.1093/jnci/dju503
7. Chaturvedi AK, Madeleine MM, Biggar RJ, et al. Risk of human papillomavirus-associated cancers among persons with AIDS. *J Natl Cancer Inst* 2009;101:1120–30.
8. Abraham AG, D'souza G, Jing Y, et al. North American AIDS cohort collaboration on research and design of IeDEA. Invasive cervical cancer risk among HIV-infected women: a North American multicohort collaboration prospective study. *J Acquir Immune Defic Syndr* 2013;62:405–13.
9. Phelps RM, Smith DK, Heilig CM, HER Study Group, et al. Cancer incidence in women with or at risk for HIV. *Int J Cancer* 2001;94:753–7.
10. Simard EP, Pfeiffer RM, Engels EA. Spectrum of cancer risk late after AIDS onset in the United States. *Arch Intern Med* 2010;170:1337–45.
11. Torre D, Speranza F, Martegani R. Impact of highly active antiretroviral therapy on organ-specific manifestations of HIV-1 infection. *HIV Med* 2005;6:66–78.
12. Dorrucci M, Suligoi B, Serraino D, Italian HIV-Seroconversion Study, et al. Incidence of invasive cervical cancer in a cohort of HIV-seropositive women before and after the introduction of highly active antiretroviral therapy. *J Acquir Immune Defic Syndr* 2001; 26:377–80.
13. Massad LS, Seaberg EC, Watts DH, et al. Long-term incidence of cervical cancer in women with human immunodeficiency virus. *Cancer* 2009;115: 524–30.
14. Massad LS, Seaberg EC, Watts DH, et al. Low incidence of invasive cervical cancer among HIV infected US women in a prevention program. *AIDS.* 2004;18:109–13.
15. Barkan SE, Melnick SL, Preston-Martin S, et al. The women's interagency HIV study. *Epidemiology* 1998;9:117–25.
16. Bacon M, von Wyl V, Alden C, et al. The women's interagency HIV study: an observational cohort brings clinical sciences to the bench. *Clin Diag Lab Immunol* 2005;12:1013–9.
17. Kurman RJ, Solomon D. The Bethesda system for reporting cervical/vaginal cytologic diagnoses. New York (NY): Springer-Verlag, 1994.
18. Surveillance, Epidemiology, and End Results (SEER) Program ((www.seer.cancer.gov) Research Data (1973–2013), National Cancer Institute, DCCPS, Surveillance Research Program, Surveillance Systems Branch, released April 2016, based on the November 2015 submission. Available at: https://seer.cancer.gov/data. Accessed 11 July 2017.
19. Inskip H. Standardization methods. In: Gail MH, Benichou J, eds. Encyclopedia of epidemiologic methods. New York: Wiley, 2000. 871–884.
20. Newman SC. Biostatistical methods in epidemiology. New York, Wiley, 2001.
21. French AL, Gawel SH, Hershow R, et al. Trends in mortality and causes of death among women with HIV in the United States: a 10-year study. *J Acquir Immune Defic Syndr* 2009;51:399–406.
22. Thorsteinsson K, Ladelund S, Jensen-Fangel S, et al. Incidence of cervical dysplasia and cervical cancer in women living with HIV in Denmark: comparison with the general population. *HIV Med* 2016;17:7–17.
23. Patel P, Hanson DL, Sullivan PS, et al. Incidence of types of cancer among HIV-infected persons compared with the general population in the United States, 1992–2003. *Ann Intern Med* 2008; 148:728–36.
24. Hleyhel M, Belot A, Bouvier AM, et al. Risk of AIDS-defining cancers among HIV-1-infected patients in France between 1992 and 2009: results from the FHDH-ANRS CO4 cohort. *Clin Infect Dis* 2013;57:1638–47.
25. Minkoff H, Zhong Y, Burk RD, et al. Influence of adherent and effective antiretroviral therapy use on human papillomavirus infection and squamous intraepithelial lesions in human immunodeficiency virus-positive women. *J Infect Dis* 2010;201:681–90.
26. Minkoff H, Ahdieh L, Massad LS, et al. The effect of highly active antiretroviral therapy on cervical cytologic changes associated with oncogenic HPV among HIV-infected women. *AIDS* 2001;15:2157–64.
27. Cejtin H, Komanoff E, Massad LS, et al. Adherence to colposcopy among women with HIV infection. *J Acquir Immun Deficiency Syndromes Hum Retrovirol* 1999;22:247–52.
28. Massad LS, Evans CT, Wilson TE, et al. Changes in knowledge of cervical cancer prevention and human papillomavirus among women with human immunodeficiency virus. *Gynecol Oncol* 2010;117:70–6.
29. Massad LS, Fazzari MJ, Anastos K, et al. Outcomes after treatment of cervical intraepithelial neoplasia among women with human immunodeficiency virus. *J Lower Genital Tract Dis* 2007;11:90–7.