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# Incidence and Correlates of *Chlamydia trachomatis* Infection in a High Risk Cohort of Kenyan Women

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## Abstract

**BACKGROUND—**In Africa, data on *Chlamydia trachomatis* infection are scarce because reliable diagnosis is costly and not widely available. Our objective was to evaluate the incidence and correlates of *C. trachomatis* infection among high-risk Kenyan women.

**METHODS—**We conducted prospective cohort analyses using data from a cohort of women who reported transactional sex. *C. trachomatis* testing was performed using the Gen-Probe Aptima GC/CT Detection System. We used Andersen-Gill proportional hazards modeling to evaluate correlates of *C. trachomatis*.

**RESULTS—**Between August 2006 and December 2010, 865 women contributed 2011 person-years of observation. Sixty-four women experienced 101 episodes of *C. trachomatis* infection (incidence rate of 5.0/100 person-years). There was a large difference in incidence by age group: those below 25 years had an incidence of 27.6 per 100 person-years (95% CI 16.3 – 46.5), those 25 to 34 years old had an incidence of 8.4 per 100 person-years (95% CI 6.4 – 11.0), and those 35 years old and above had an incidence of 2.6 per 100 person-years (95% CI 1.8 – 3.6). In multivariate analyses, younger age (<25 years and 25–34 years versus ≥35 years; hazard ratio [HR] 8.49 95% CI 4.1–17.7 and HR 2.9 95% CI 1.7–5.0 respectively), depot medroxyprogesterone acetate use (HR 1.8 95% CI 1.1–3.0) and recent *Neisseria gonorrhoeae*

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**AUTHORS' CONTRIBUTIONS**

R.S.M., L.M., J.M.B., B.A.R., G.J-S, E.B., and W.J. conceived the question and designed the study. R.S.M. obtained funding for the study. L.M., R.S.M., J.M.B., B.A.R., E.K., and R. D. participated in collection and interpretation of the data. L.M, J.M.B., and B.A.R. conducted the data analyses. All authors participated in preparation of the manuscript and approved the final draft for submission.

infection (HR 3.3 95% CI 1.5–7.4) were significantly associated with increased risk of acquiring *C. trachomatis* infection.

**CONCLUSIONS—**The high incidence of *C. trachomatis* among younger high-risk women suggests the need for screening as an important public health intervention for this population.

### Keywords

*Chlamydia trachomatis*; incidence; risk factors; women; Africa

## INTRODUCTION

In 1999, the World Health Organization (WHO) estimated the global incidence of *Chlamydia trachomatis* infection to be 92 million cases worldwide, with 16 million (17%) occurring in Africa.[1] However, estimates from resource-constrained settings including most of Africa are imprecise due to lack of surveillance, limited laboratory infrastructure and diagnostic capacity, and the widespread use of syndromic management. The few community-based studies that have measured *C. trachomatis* in sub-Saharan Africa suggest a low prevalence in the general population (1.6–3.2%).[2,3] A cross-sectional study among female adolescents in Uganda estimated *C. trachomatis* prevalence at 4.5%.[4] Prevalence is much higher among high-risk groups when compared with community data. For example, prevalence estimates among female sex workers range from 9% in Nairobi, Kenya[5] to 28.5% in Dakar, Senegal.[6]

Incidence studies are particularly valuable in understanding the risk of a disease. Studies of the incidence of *C. trachomatis* infection in Africa are few.[5,7,8] Among women attending family planning clinics in Zimbabwe and Uganda, the incidences of *C. trachomatis* were 3.6 and 4.1 per 100 person-years respectively.[8] In the past decade, two studies of female sex workers in Kenya have reported incidence rates of 6.5 and 9.0 per 100 person-years respectively.[5,7]

Less than 10% of women infected with *C. trachomatis* develop acute signs and symptoms of infection.[9] The absence of symptoms in the majority of those infected results in a substantial number of cases that are unrecognized, untreated, and persist as a reservoir for ongoing transmission. In addition, immunity to *C. trachomatis* infection is partial and short-lived,[10] so reinfection is common. *Chlamydia trachomatis* is associated with a number of serious reproductive health problems such as pelvic inflammatory disease (PID), tubal infertility, ectopic pregnancy, and chronic pelvic pain.[11] In addition, *C. trachomatis* infection has been associated with increased risk of HIV-1 acquisition.[8,12] These data highlight the importance of screening and treatment for *C. trachomatis* in order to prevent the spread of this infection and related complications.

To add to the limited data characterizing the epidemiology of *C. trachomatis* in sub-Saharan Africa, we sought to determine the incidence and correlates of *C. trachomatis* infection among HIV-1-seropositive and seronegative women enrolled in a prospective cohort study of women at high risk for sexually transmitted infections (STIs).

## MATERIALS AND METHODS

We conducted longitudinal follow-up in an open cohort of female sex workers in Mombasa, Kenya. The eligibility criteria to join the cohort are: age 18–50 years, residing in the Mombasa area, self-identifying as exchanging sex for payment in cash or in kind, and able to provide informed consent. The present analysis utilized data collected between August 2006 and December 2010. This study was approved by the ethical review committees at

Kenyatta National Hospital and the University of Washington. All participants provided written informed consent.

## Clinic Procedures

At enrollment and monthly follow-up visits, a study nurse conducted a standardized interview covering demographic data, medical, gynecological, and sexual history. A study physician performed a physical examination including a pelvic speculum examination. Swabs of cervical and vaginal secretions were collected for laboratory diagnosis of STIs. After April 2008, the visit schedule for HIV-1 seropositive women who were not on antiretroviral therapy changed from monthly to every three months, consistent with local clinical standards for this group.

Between August 2006 and April 2008, *C. trachomatis* testing was performed monthly. After April 2008, testing was performed every three months. All participants received free outpatient medical services including treatment of STIs. Diagnosed STIs were treated according to WHO[13] and Kenyan national guidelines. Women received doxycycline 100mg twice daily for seven days if they were diagnosed with *C. trachomatis* infection. Syndromic management was offered during examination visits if indicated. When participants returned to receive their tests results, additional treatment was provided if infections were diagnosed by laboratory testing that had not been treated syndromically at the prior visit.

## Laboratory Procedures

Endocervical samples were tested for the presence of *Neisseria gonorrhoeae* and *C. trachomatis* by transcription mediated amplification (TMA) using the Gen-Probe Aptima GC/CT Detection System (Gen-Probe, San Diego, California, USA). Culture for *N. gonorrhoeae* was performed on modified Thayer-Martin media. Cervical Gram's stained slides were examined microscopically for Gram-negative intracellular diplococci consistent with a diagnosis of *N. gonorrhoeae* infection. Vaginal Gram stained slides were evaluated using Nugent's criteria, with bacterial vaginosis (BV) being defined as a score of 7–10.[14] Nugent scoring was performed by laboratory technicians with over 10 years experience with this technique. Internal quality assurance was performed weekly and external quality assurance was performed semiannually. Saline and potassium hydroxide wet mounts were examined at 40X power for the presence of motile trichomonads, clue cells, *Lactobacillus* morphotypes, and yeast. HIV-1 serostatus was determined by ELISA (Detect HIV1/2, BioChem Immunosystems, Montreal, Canada or PT-HIV 1,2–96, Pishtaz Teb Diagnostics, Tehran, Iran). Positive tests were confirmed using a second ELISA (Recombigen, Cambridge Biotech, Worcester, MA, USA or Vironostika HIV-1 Uniform II AG/AB, bioMerieux, Marcy l'Etoile, France). For the confirmatory tests, the cut-off values used were as suggested by the manufacturers. In addition to the manufacturers' recommendations, we took into consideration a grey zone. This grey zone range was determined by identifying 10% of readings above and 10% of readings below the cut-off. We performed repeat testing for any results that fell within this range.

## Data Analyses

All women who had more than one visit at which *C. trachomatis* testing was performed were included in the analyses. The outcome was time to *C. trachomatis* infection. The exposures of interest were known or suspected risk factors for *C. trachomatis* infection including age, hormonal contraceptive use (oral contraceptive pills [OCP] or depot medroxyprogesterone acetate [DMPA] versus no hormonal contraception), vaginal microbiota (intermediate vaginal microbiota or bacterial vaginosis versus normal vaginal microbiota), place of work (bar versus night club or home-based/other), educational level, marital status, sexual risk behavior in the past week (unprotected intercourse, number of sex partners), vaginal

washing, presence of other genital tract infections (*Trichomonas vaginalis, Candida albicans, N. gonorrhoeae*), HIV-1 serostatus, and cervical ectopy. These were assessed as predictors of infection using Andersen-Gill proportional hazards models and the Efron method for ties. Participants were included in the analyses beginning in August 2006 (when Gen-Probe *C. trachomatis* screening was initiated), or from the date of enrollment for those who enrolled after August 2006. Data were censored at a participant's last follow-up visit or at the end of the analysis period in December 2010. We first conducted univariate analyses to determine whether individual risk factors were associated with *C. trachomatis* infection. Variables that were associated with *C. trachomatis* (alpha = 0.10) in the univariate analyses were then included in the multivariate model.

As in previous analyses, we estimated that the effect of oral or injectable hormonal contraception would persist for 70 days after discontinuation of use.[15] We assumed that *C. trachomatis* was acquired at the midpoint between the pre-infection visit and the visit at which the infection was detected. Because visits were, on average, every 30 days, we used an exposure interval of 85 (70+15) days for women who changed their method of hormonal contraception. We used a 45 day exposure interval for other STIs and abnormal vaginal microbiota or BV (assuming approximately 30 days for a persistent effect and 15 days incubation period). The lag period included the current visit. Analyses were performed using PASW 18.0 (PASW Inc., Chicago, USA) and STATA 11 (StataCorp, College Station, TX, USA).

## RESULTS

Between August 2006 and December 2010, 865 women had more than one visit where *C. trachomatis* testing was performed. Baseline characteristics for these participants are presented in Table 1. Their median age was 35 years (interquartile range [IQR] 30–40). One hundred and eighty-one women (20.9%) were using DMPA. At baseline, the prevalence of *C. trachomatis* was low (1.9%). With the exception of HIV-1 (N=457; 52.8%), the prevalence of other STIs at baseline was also low.

Participants in this study contributed a total of 2011 person-years of follow-up. The median duration of follow-up was 3.8 years (IQR 2.7–4.1). Sixty-four women experienced 101 episodes of *C. trachomatis* infection, resulting in an incidence of 5.0 per 100 person-years. Twenty women had more than one episode of *C. trachomatis* infection (range: 2–5 episodes). There was a large difference in incidence by age group. Women below 25 years had an incidence rate of 27.6 per 100 person-years (95% CI 16.3 – 46.5), those 25 to 34 years old had an incidence rate of 8.4 per 100 person-years (95% CI 6.4 – 11.0), and women 35 years old and above had an incidence rate of 2.6 per 100 person-years (95% CI 1.8 – 3.6). While *C. trachomatis* infection was associated with a higher likelihood of reporting symptoms (lower abdominal pain and/or vaginal discharge) (OR 1.7 95% CI 1.0–3.0; p=0.05), only a minority *C. trachomatis* episodes were symptomatic (N=16, 15.8%). Of 101 episodes of *C. trachomatis*, 6 (5.9%) included co-infection with *N. gonorrhoeae*.

Several exposures including younger age, use of DMPA, more recent enrollment in the research cohort, having >1 sex partner in the last week, having >1 sexual encounter in the last week, being HIV-1-seropositive, and having recent or concurrent *N. gonorrhoeae* infection were associated with an increased likelihood of acquiring *C. trachomatis* infection in univariate analyses (Table 2). In multivariate analyses, younger age (<25 years and 25–34 years versus ≥35 years; hazard ratio [HR] 8.5 95% CI 4.1–17.7 and HR 2.9 95% CI 1.7–5.0 respectively), DMPA use (HR 1.8 95% CI 1.1–3.0) and recent or concurrent *N. gonorrhoeae* infection (HR 3.3 95% CI 1.5–7.4) remained significantly associated with increased risk of acquiring *C. trachomatis* infection.

Although age was associated with the number of sexual partners and hormonal contraceptive use, there were no statistically significant interactions between age and either of these additional covariates. Condom use was not different among hormonal contraceptive users compared to women not using hormonal contraception, and the interactions between condom use and hormonal contraception were also not statistically significant (data not shown). Thus, only a non-stratified model is presented.

## DISCUSSION

The overall incidence of *C. trachomatis* infection among female sex workers in Mombasa was 5.0/100 person-years, which is similar to incidence estimates from studies conducted among female sex workers in Nairobi between 1998 and 2002 and in Mombasa between 1993 and 2003.[5,7] The incidence of *C. trachomatis* was markedly higher among younger women (18–25 years), suggesting the need for expansion of screening to address this problem among young at-risk women. Other factors associated with *C. trachomatis* infection were use of DMPA and recent or concurrent infection with *N. gonorrhoeae*.

Studies of risk factors for *C. trachomatis* have mostly been conducted in developed countries, and have identified similar risk factors including younger age, higher number of sexual partners, and use of hormonal contraception.[16–20] Recent prospective studies have also highlighted the potential importance of BV as a risk factor for infection with *C. trachomatis.*[21,22] In contrast, we did not find an association between abnormal vaginal microbiota or BV and *C. trachomatis* infection. Further research is needed to explore the association between vaginal microbiota and incident STIs.

Susceptibility to *C. trachomatis* infection may be influenced by a number of biological factors. First, cervical ectopy occurs when the squamocolumnar junction lies outside the endocervix, resulting in exposed columnar cells. This anatomical characteristic has been associated with increased risk for numerous pathogens, including *C. trachomatis.*[23] Cervical ectopy tends to be greatest during adolescence and decreases with age.[23] This age-dependent phenomenon may help to explain the higher incidence of *C. trachomatis* infection in younger women. A second important biological factor is exposure to DMPA, which induces a systemic hypo-estrogenic state associated with decreased vaginal colonization with hydrogen peroxide producing *Lactobacillus* species.[24] This decrease in protective vaginal bacteria may, in turn, increase the risk of *C. trachomatis* infection. In our study, DMPA was associated with a nearly 2-fold increase in risk of *C. trachomatis* infection, adding to data suggesting a possible link between hormonal contraception and acquisition of STIs including HIV-1.[25] We did not find an association between OCP use and *C. trachomatis* infection, possibly due to the relatively small number of women (N=41 [4.7%]) using OCP in this cohort. Condom use among women on hormonal contraception was similar to condom use among women not using any hormonal contraception. Moreover, we have previously noted that not every STI risk is increased in contraceptive users. A study conducted within this same cohort demonstrated that women using DMPA had a significantly decreased risk of bacterial vaginosis (hazard ratio, 0.7; 95% confidence interval, 0.5–0.8) and trichomoniasis (hazard ratio, 0.6; 95% confidence interval, 0.4–1.0).[15] These findings argue against condom use as the mediating factor for *C. trachomatis* infection among hormonal contraceptive users.

Women with a recent or concurrent *N. gonorrhoeae* infection were three times more likely to acquire *C. trachomatis* infection. Prior STI has previously been described as a risk factor for infection by other sexually transmitted pathogens.[26] Although there may be biological interactions, it is likely that this effect is also mediated through exposure to higher risk sexual networks, where a variety of STIs are circulating.

The strengths of our study include the large sample size and longitudinal follow-up that enabled us to assess the correlates of *C. trachomatis* infection. In addition, we used the Gen-Probe Aptima GC/CT Detection System, which has excellent sensitivity (94.2%) and specificity (97.6%) for detection of *C. trachomatis* on endocervical swabs.[27] This study also had limitations. Sexual risk behavior was self-reported, making these data subject to recall and social desirability bias. The questions on sexual risk behavior were limited to the past one week to mitigate recall bias. In addition, we have recently demonstrated that within this cohort, self-reported behaviors are associated with biological outcomes including STIs and sperm in genital secretions.[28] Nonetheless, some misreporting of sexual risk behaviors should be anticipated. Secondly, twenty women experienced more than one episode of *C. trachomatis* infection. We did not perform molecular testing to distinguish between treatment failure and re-infection. Future studies should consider molecular characterization to improve our understanding of *C. trachomatis* re-infection or persistence.

Findings from this study add to a sparse body of literature on the incidence and risk factors for *C. trachomatis* in Africa. Data from this study suggest that the risk of *C. trachomatis* among high-risk women under 25 years old could be substantial. Studies on the incidence of *C. trachomatis* among young women in sub-Saharan Africa should be prioritized in view of potentially severe sequelae including PID, tubal infertility, and increased HIV-1 susceptibility. Tubal infertility is especially of concern in Africa, where prevalences of infertility as high as 27% have been reported,[29] and motherhood may be closely associated with a woman's status in the community.[30] Studies from West Africa have shown that *C. trachomatis* antibodies were more likely to be detected among infertile women compared with fertile women.[31,32] Development of inexpensive point-of-care tests that can be used in resource-limited settings would enhance the diagnosis and early management of this largely silent epidemic.

## CONCLUSION

We found a high incidence of *C. trachomatis* infection among high risk women less than 25 years old, suggesting the need for screening as an important public health intervention for younger high-risk women. In addition, data from general population women are urgently needed to gain a greater understanding of the extent to which the epidemic crosses over into the general population.

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## Table 1

Baseline Characteristics of the 865 participants

| Characteristic | Median (IQR) or Number (percent) |
|---|---|
| **Demographics** | |
| Age (years) | 35 (30–40) |
| Education (years) | 8 (7–10) |
| Ever married[a] | 559 (64.6) |
| Work place | |
| Bar/Restaurant | 626 (72.4) |
| Night club | 123 (14.2) |
| Home based/other[b] | 116 (13.4) |
| **Gynecological history** | |
| Parity | 2 (1–3) |
| Hormonal contraceptive use | |
| OCP | 41 (4.7) |
| DMPA | 181 (20.9) |
| **Sexual risk behavior reported in the past week** | |
| Unprotected intercourse | 195 (22.5) |
| >1 sex partner[c] | 186 (31.0) |
| >1 sex encounter[c] | 379 (63.1) |
| **Clinical conditions** | |
| Vaginal discharge | 80 (9.3) |
| Abdominal pain | 66 (7.6) |
| Vulval itch | 94 (10.9) |
| Presence of GUD | 23 (2.7) |
| Cervical ectopy | 48 (5.5) |
| HIV-1 seropositive | 457 (52.8) |
| **Laboratory diagnosis of genital tract conditions** | |
| Candidiasis | 111 (12.8) |
| Bacterial vaginosis | 313 (36.2) |
| *Trichomonas vaginalis* | 36 (4.2) |
| *Neisseria gonorrhoeae* | 22 (2.5) |
| *Chlamydia trachomatis* | 16 (1.9) |
| **Reported vaginal washing** | 834 (96.4) |
| Water only | 359 (43.1) |
| Soap/Other[d] | 475 (56.9) |

[a] Included 15 currently married and 544 widowed or divorced women

[b] 22 (2.5%) women were home based and 94 (10.9%) reported “other” as their place of work

[c] Analyzed in the subgroup of 601 women who reported any sexual activity in the past week

[d] 451 (54.1%) women reported using soap, 18 (2.1%) reported using antiseptic, 4 (0.5%) reported using detergent, and 2 (0.2%) reported using “other” substances for vaginal washing

**Table 2**

Univariate and Multivariate Analyses of the Correlates of *C. trachomatis* Infection

| Characteristic | Chlamydia infections/Person-years | Incidence/100 person-years | Univariate Analysis | | Multivariate Analysis | |
|---|---|---|---|---|---|---|
| | | | HR (95% CI) | p value | HR (95% CI) | p value |
| **Age (years)** | | | | | | |
| <25 | 14/51 | 27.6 | 10.1 (4.8–21.1) | <0.001 | 8.5 (4.1–17.7) | <0.001 |
| 25 to 34 | 53/630 | 8.4 | 3.2 (1.9–5.5) | <0.001 | 2.9 (1.7–5.0) | 0.001 |
| ≥ 35 | 34/1331 | 2.6 | 1.0 | | 1.0 | |
| **Hormonal contraceptive method** | | | | | | |
| None | 67/1515 | 4.4 | 1.0 | | 1.0 | |
| OCP | 1/78 | 1.3 | 0.3 (0.0–1.8) | 0.2 | 0.2 (0.0–1.7) | 0.2 |
| DMPA | 33/386 | 8.5 | 1.9 (1.1–3.2) | 0.02 | 1.8 (1.1–3.0) | 0.03 |
| **Abnormal vaginal microbiota** | | | | | | |
| Nugent score 0–3 | 47/998 | 4.7 | 1.0 | | | |
| Nugent score 4–6 | 23/382 | 6.0 | 1.2 (0.6–2.2) | 0.6 | | |
| Nugent score 7–10 | 31/631 | 4.9 | 1.1 (0.6–1.9) | 0.8 | | |
| **Education (> 8 years)** | 41/730 | 5.6 | 1.2 (0.7–2.0) | 0.5 | | |
| **Marital status** | | | | | | |
| Never married | 43/694 | 6.2 | 1.0 | | | |
| Ever married | 58/1318 | 4.4 | 0.7 (0.4–1.2) | 0.2 | | |
| **Work place** | | | | | | |
| Bar/Restaurant | 73/1471 | 5.0 | 1.0 | | | |
| Night club | 18/280 | 6.4 | 1.3 (0.7–2.5) | 0.4 | | |
| Home based/Other | 10/261 | 3.8 | 0.8 (0.3–2.0) | 0.6 | | |
| **Sexual risk behavior** | | | | | | |
| Unprotected intercourse | 26/378 | 6.9 | 1.5 (0.9–2.5) | 0.1 | | |
| >1 sex partner | 24/297 | 8.1 | 1.7 (1.1–2.8) | 0.02 | 1.3 (0.8–2.0) | 0.4 |
| **HIV-1 positive** | 47/1224 | 3.8 | 0.6 (0.3–1.0) | 0.04 | 0.7 (0.4–1.1) | 0.1 |
| **Presence of genital tract conditions** | | | | | | |
| Candidiasis | 19/316 | 6.0 | 1.2 (0.7–2.3) | 0.5 | | |
| Trichomoniasis | 4/86 | 4.7 | 0.9 (0.3–2.4) | 0.8 | | |
| *Neisseria gonorrhoeae* | 8/45 | 17.9 | 3.8 (1.7–8.4) | 0.001 | 3.3 (1.5–7.4) | 0.004 |

| Characteristic | Chlamydia infections/Person-years | Incidence/100 person-years | Univariate Analysis | | Multivariate Analysis | |
|---|---|---|---|---|---|---|
| | | | HR (95% CI) | p value | HR (95% CI) | p value |
| **Reported vaginal washing** | | | | | | |
| None | 4/126 | 3.2 | 1.0 | | | |
| Water | 56/1082 | 5.2 | 1.4 (0.5–4.1) | 0.5 | | |
| Soap/Other | 41/802 | 5.1 | 1.4 (0.5–4.1) | 0.6 | | |
| **Cervical ectopy** | 5/182 | 2.7 | 0.6 (0.2–1.6) | 0.3 | | |