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Behavioural sources of repeat *Chlamydia trachomatis* infections: importance of different sex partners

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Abstract

Objective—To examine sources of repeat *Chlamydia trachomatis* infections using behavioural and molecular methods.

Methods—Women with *C trachomatis* had baseline and 4-month follow-up visits consisting of behavioural surveys and genotyping of *C trachomatis*. Frequencies and population-attributable risk percentages (PAR%) were estimated for possible sources of repeat infections including sex partners not known to be treated, new sex partners, and sex partners not known to be monogamous. Women with different genotypes at baseline and follow-up were classified as different partner sources of infection.

Results—The cumulative incidence of repeat infections in the sample (n=183) was 13% (95% CI 8% to 18%). Predictors of repeat infections included younger age and continued sex with a partner not known to be treated. Frequencies of having partners not known to be treated, new partners, or partners not known to be monogamous at follow-up were 21% (95% CI 15% to 27%), 37% (95% CI 30% to 44%) and 33% (95% CI 28% to 41%), respectively. The PAR% for having a partner not known to be treated was 26% (95% CI 3% to 49%) and for having a new sex partner was 21% (95% CI 0% to 50%). Among eight patients with available genotypes at baseline and follow-up, five had different genotypes and were classified as having a different partner source of infection.

Conclusions—Different sex partner sources of repeat *C trachomatis* infections other than untreated sex partners may contribute substantially to the burden of repeat infections.

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Competing interests None.

Patient consent Obtained.

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Contributors LMN and MMP cowrote the study protocol, set up the operational aspects of the study, and oversaw all aspects of data collection. LMN conducted statistical analyses and wrote the first draft of the manuscript. MMP developed and oversaw all laboratory procedures. KAL served as primary liaison with clinic staff and performed data collection (interviews and specimens). ASL conducted and analysed laboratory tests. All authors provided substantive contributions to the final version of the manuscript.

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INTRODUCTION

Chlamydia trachomatis infections comprise a substantial public health burden because of high frequency and negative health effects including pelvic inflammatory disease, ectopic pregnancy and chronic pelvic pain.[1] *C trachomatis* is the most common reportable disease in the USA; the number of cases surpassed one million in 2006.[2] Since then, the number of cases has increased annually by 7.5% in 2007 and 9.2% in 2008.[2] Increases may reflect improved screening, test performance, reporting and/or rising prevalence.[2]

Repeat infections with *C trachomatis* are also common and more closely linked to pelvic inflammatory disease and ectopic pregnancies.[3] A literature review revealed a median proportion of women who are reinfected of 13.9%.[4] The range of estimates (eg, 7–29% within 6 months of initial diagnosis[5–8]) probably reflects various amounts of time for which women were followed. In British Columbia, repeat infections increased from 3 per 100 000 in 1991 to 52 per 100 000 in 2003[9]; although this trend may be due to changing testing patterns, this further suggests the public health importance of repeat *C trachomatis*.

It is important to understand sources of repeat infections to implement effective prevention measures. Repeat infections may result from continued sex with the same source partners for the initial infection who were not adequately treated, new sex partners of the index patient, continued sex with same source partners for the initial infection who acquired a new infection from a different sex partner, or treatment failures. Although clinical trial data showed treatment efficacy for *C trachomatis* to be 97%, more recent data from other studies indicate that treatment effectiveness may be closer to 92%.[7,10,11] However, treatment failures are uncommon and most repeat infections are due to post-treatment behaviours of patients. Studies suggest that repeat infections often result from continued sex with untreated partners[5,12,13]; this observation has prompted innovative partner treatment strategies.[7] However, the relative contribution of different partners, either new partners of the index patient or other sex partners of the source partners, is not well established. Understanding the relative contribution of different possible sources has direct implications for prevention counselling at the time of treatment.

Although measures of relative risk are commonly used to estimate the strength of associations between risk factors and health outcomes, absolute measures of association based on risk differences are also important. These measures provide estimates of the public health impact of risk factors.[14] Population-attributable risk percentages (PAR%) indicate the proportion of disease in a population that can be attributed to an exposure. Despite the public health importance of this measure, PAR% have not been used to describe the impact of different post-treatment behaviours on risk of repeat *C trachomatis* infection.

Another underused tool in the study of repeat *C trachomatis* is molecular genotyping. *OmpA* genotyping in combination with epidemiological data can be used to infer sources of repeat infections. When mixed infections can be ruled out, detection of different genotypes at baseline and follow-up indicates a different source of infection. With supporting behavioural data, the identification of the same genotype at baseline and follow-up can help discern

potential sources of repeat infections. *OmpA* genotyping is not commonly used, but the few studies that have incorporated this method show the promise of the approach.[111315]

We examined sources of repeat *C trachomatis* infections using an innovative combination of epidemiological measures and molecular data. We examined predictors of repeat *C trachomatis* in a cohort of women, and assessed the relative contributions of three different possible sources of repeat infections by estimating the frequency and PAR% for sex partners not known to be treated, new sex partners, and sex partners not known to be monogamous. We also examined the discordance of genotypes between initial and repeat infections to further describe possible sources of repeat infections.

METHODS

Setting, study participants and study design

Data were collected in a cohort study conducted during 2005–2008. Eligibility criteria included being female, age 15 years or older, and diagnosed with *C trachomatis* by nucleic acid amplification testing at one of two reproductive health centres. Participants were provided with a prescription for or the medication of a single-dose 1 g azithromycin. All participants were instructed to refrain from sexual activity for 7 days, notify partners of the need for treatment, be retested in 3–6 months, and use condoms. Healthcare providers referred eligible patients to study staff. Participants were enrolled at the time of diagnosis, treatment, or at a separately scheduled study visit. Follow-up visits were scheduled 4 months after the baseline visit. All study procedures were conducted in private offices at the health centres. Participation was voluntary, and participants were paid US$30 for each study visit. The human subjects review boards of the participating sites approved this study, and all participants provided written informed consent.

Survey interview procedures and measures

Participants completed structured surveys using audio computer-assisted survey interviewing (A-CASI) technology. The baseline survey ascertained demographic information, sexual histories and behaviours, and information pertaining to the current *C trachomatis* diagnosis. Partner modules were used to ask respondents partnership-specific questions for up to three sex partners in the past three months. The follow-up survey ascertained non-mutually exclusive post-treatment behaviours and changes in partnerships including any sex without a condom, continued sex with a baseline partner not known to be treated, new sex partners, and sex with a partner not known to be monogamous. Partners were classified as not known to be treated if the participant responded ‘no’ or ‘don’t know’ to questions about partners’ treatment. Partners were classified as not known to be monogamous if the participant responded ‘no’ or ‘don’t know’ to questions about his monogamy.

Statistical analysis

To determine predictors of repeat infections, covariates associated in univariate analyses at p value <0.20 using likelihood ratio χ^2 tests were included in the initial multivariate model. Logistic regression was used to estimate adjusted ORs (aOR) and 95% CIs. Manual backward selection was used to eliminate non-significant covariates using the p<0.05

criterion to arrive at the final, most parsimonious multivariate model. To determine the contribution of different possible sources to repeat infections, proportions and 95% CI were estimated for three possible behavioural sources including partners not known to be treated, new partners, and partners not known to be monogamous. PAR% were calculated using the formula $((\text{Risk}_{\text{total population}} - \text{Risk}_{\text{unexposed population}})/\text{Risk}_{\text{total population}})$, where risk was estimated by cumulative incidence to describe the proportion of repeat infections in the total study population attributed to each source.[14] The 95% CIs for PAR% were computed using published formulae.[16]

Molecular genotyping procedures

Women submitted either first-void urine or a self-collected vaginal swab for genotyping. Swabs were added in the second year because of accumulating evidence of patient acceptability and higher sensitivity.[17] Genomic DNA was extracted from urine within 72 h using the QIAamp DNA Mini Kit. From the swabs, we removed 1 ml liquid and added 50 μl SDS-Out (Pierce Biotechnology, Rockford, IL, USA) to precipitate SDS. We then used the QIAamp DNA Mini Kit to extract DNA from the supernatant. *OmpA* DNA sequences were amplified using previously described methods.[18] Women were classified as having a repeat infection if they were positive by PCR. Nested PCR products were sequenced, and DNA sequences were trimmed using EditSeq software from DNAStar. Individual consensus sequences were compared with *ompA* nucleotide sequences from known *C trachomatis* serovars (http://www.ncbi.nih.gov/BLAST/). Genotypes were assigned the letter designation used for serovar grouping. A reverse dot-blot method was used to detect mixed *C trachomatis* infections in samples from participants with genotypes detected at both visits.[19] Among participants with baseline and follow-up genotypes, the proportion and 95% CI with different genotypes was computed. For each of these participants, changes in sex partnerships during the follow-up period were examined. Different genotypes at baseline and follow-up were considered different partner sources of infections other than an untreated sex partner.

RESULTS

Of 323 screened patients, 237 (73%) enrolled and 183 (77%) had a follow-up visit (median time to follow-up = 17.3 weeks). Participants who did not have a follow-up visit were not significantly different from those who did with respect to demographic and sexual history variables ($p>0.05$ for all). Sample characteristics are presented in table 1.

At follow-up, 84% reported continued sexual activity, and 39% reported sex without a condom. Twenty-four participants were positive by PCR for *C trachomatis* for a cumulative incidence of repeat infections of 13.1% (95% CI 8.2% to 18.0%). On the basis of univariate analysis (table 2), the following covariates were considered candidates for inclusion in the multivariate model because of observed p values >0.20: age 15–19 years, age at first intercourse younger than 15 years, new sex partner, and continued sex with a baseline partner not known to be treated. In the final multivariate logistic regression model, age at first intercourse and new sex partner were removed during backward selection due to p values >0.05, and the final model included the following two covariates that remained

significant predictors of repeat infection: age 15–19 years (OR=3.2, 95% CI 1.2 to 8.3; p=0.02) and continued sex with a baseline partner not known to be treated (OR=3.4, 95% CI 1.3 to 8.6; p=0.01). Analyses were also run excluding those who did not report having sex during the follow-up period (n=29); results did not differ substantively (not shown).

At follow-up, the prevalence of having a sex partner not known to be treated was 20.8% (95% CI 14.9% to 26.7%). The prevalence of having a new sex partner was 37.2% (95% CI 30.2% to 44.2%). The prevalence of having a sex partner not known to be monogamous was 33.4% (95% CI 27.6% to 41.3%). The PAR% for having a sex partner not known to be treated was 26.0% (95% CI 3.4% to 49.4%) and for having a new sex partner was 20.6% (95% CI 0% to 50.1%). Because risk for repeat infection was lower (although non-significant) among those reporting having a sex partner not known to be monogamous, the PAR% for this source would be negative and was not estimated.

Sixty participants had sufficient *C trachomatis* DNA for genotyping at baseline (33%). Missing genotypes were due to collection of study specimens after antibiotic treatment. Forty patients were enrolled at time of treatment and 143 were enrolled (and provided specimen) after treatment (median time of 4 days); enrolment at time of treatment was associated with successful baseline genotyping (75% vs 21%, p<0.001). Reverse dot-blots did not detect any mixed infections. Molecular and behavioural data for identification of sources of repeat infections are in table 3 (GenBank accession numbers GQ228430–GQ228445). Five of eight (62.5%, 95% CI 29.0% to 96.1%) participants with available genotypes for baseline and follow-up had different genotypes and were classified as having a different partner source of infection (either a new sex partner or a partner not known to be monogamous). Non-mutually exclusive risk factors for repeat infections were: two reported continued sex with a baseline partner not known to be treated, three reported having a new partner, and one reported sex with a partner not known to be monogamous. One woman with the same genotype at baseline and follow-up did not report having continued sex with an untreated partner, but did report both a new sex partner and continued sex with a partner not known to be monogamous.

DISCUSSION

Our findings support and extend previous research regarding the high frequency and predictors of repeat *C trachomatis* infections.[5,7,12,20] We estimated a 13% 4-month cumulative incidence of repeat infections, higher rates of repeat infections among younger women, and continued sex with partners not known to be treated as a strong predictor of repeat infections. Our findings also highlight the previously under-recognised importance of different sex partners, of either the patient or her partners, as sources of repeat infections by calculation of PAR% and *ompA* genotyping. While having a sex partner not known to be treated was a significant predictor of repeat infections, this behaviour was reported significantly less often than different partner sources, as indicated by non-overlapping 95% CIs. This raises the overall contribution of new partners and having partners not known to be monogamous to repeat infections despite non-significant associations in the multivariate model. The importance of new sex partners is further reflected in the comparable PAR% for

having new sex partners and having sex partners not known to be treated (21% and 26%, respectively; p>0.05).

Although based on a limited sample size, molecular results provide additional evidence for the role of different partner sources in repeat infections. Of eight women who had genotype data at both study visits, five had a different genotype detected at follow-up from that at baseline, indicating that a majority of repeat infections were from partners other than untreated baseline sex partners. This high proportion of repeat infections attributed to different partners other than the original source supports our behavioural findings of the importance of additional sex partners and is consistent with findings from other studies. A study from the Netherlands, based on molecular data alone, reported a high frequency of different genotypes at repeat infections: three of five participants were infected with a different *C trachomatis* genotype.[15] In a larger and more recent study in the USA, a majority (100/184, 55%) of patients with repeat *C trachomatis* infections and both genotypes available had different genotypes detected at the two episodes.[11] Although the confidence bounds on our estimate of 63% are large, it should be noted that this point estimate is a minimum estimate because the same genotypes can also result from different partner sources. Among three participants with the same genotype at both infections, one woman did not report having a sex partner not known to be treated but did report a new partner and a partner not known to be monogamous, indicating that her infection may have also been due to a different partner. Two participants with repeat infections did not report any possible behavioural sources; this may be due to misreporting of behavioural information or treatment failures. Others have also found repeat infections among individuals who deny exposures.[711]

Our study has implications for the practice of expedited partner treatment (EPT). First, the strength of the association between partners not known to be treated and repeat infections supports use of EPT, which has proven efficacy in clinical trials and is included in US CDC treatment guidelines.[721] However, the high frequency with which repeat infections due to different partners are likely suggests that EPT should be conducted in conjunction with risk-reduction counselling promoting initiation and maintenance of condom use and reductions in number of sex partners. Our findings also have implications for EPT effectiveness research. Different sources of repeat infections may reduce the statistical power of trials designed to evaluate EPT and may partially explain non-significant results from previous research.[2223] Future trials should account for this in the design and may benefit from including genotyping.

It is noteworthy that the combined contribution of partners not known to be treated and new partners accounts for less than 50% (21%+26%) of repeat infections, suggesting that the other source, sex partners' other partners, may account for a substantial burden of repeat infections, although this could not be directly estimated in the present study. Unfortunately, this variable is probably subjected to mis-measurement, as women may not have accurate knowledge of their partners' other partners. Approximately one-third of participants reported not knowing whether their partner was monogamous, and 44% (revealed in post-hoc analyses) reported not talking to all of their partners about having other partners. Difficulty

in measuring this variable makes precise determination of its role in repeat infections challenging.

This study has several limitations. First, the main limitation is the small number of specimens available for genotyping at baseline, which resulted in few specimens to compare with repeat infections. This precludes firm conclusions about sources of repeat infections, and larger studies are necessary to confirm our findings. Although our genotype data appear to be missing at random, limiting the potential for bias, we cannot rule out this possibility. Second, data collected by interview may be inaccurate because of participants' inability or unwillingness to provide valid responses. However, in our prospective study, it is not likely that self-reported errors are biased because survey data were collected before determination of repeat infection. Use of A-CASI has been shown to reduce over-reporting of socially desirable responses in STI research.[24] Third, limited study resources precluded us from conducting a test-of-cure after treatment; thus, it is possible that treatment failures occurred. However, the overall impact of this on our study findings is likely to be small, as previous studies have shown that the vast majority (>90%) of repeat infections are due to post-treatment behaviours.[711] Finally, our multivariate model did not show an effect of condom use on preventing repeat infections despite demonstrated condom effectiveness in preventing *C trachomatis* infections; this is probably due to our inability to measure exposure to an infected sex partner accurately.[2526]

Our results suggest that repeat *C trachomatis* infections may be due to different sex partners other than the source of the original infection, either new partners or partners' other partners, to a larger extent than previously recognised. These findings are consistent with the sexual network literature about the importance of STI prevalence in a network in ongoing transmission.[27] Although our findings support the need for effective partner treatment strategies, reducing repeat infections will also require strengthened and renewed risk-reduction counselling programmes to reduce numbers of sex partners and/or increase condom use among those at risk, especially young women. Such efforts are necessary, but unlikely to be effective for all patients; thus retesting of all female patients 3 months after treatment as recommended by treatment guidelines[28] remains an important strategy to improve the health of women and reduce the burden of infection in the community.

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Key messages

- Having a new sex partner is the most common post-treatment behavioural risk factor for a repeat *C trachomatis* infection.
- Frequencies, population-attributable risks and genotyping suggest that different sex partners (new partners and/or partners' partners) may contribute substantially to repeat infections.
- Expedited partner treatment should be accompanied by counselling to initiate and maintain condom use with all sex partners and reduce number of sex partners.

Table 1

Sample characteristics of women diagnosed with *C trachomatis* and followed for repeat infection (n=183)

Characteristic	Value
Baseline demographic and sexual risk history variables	
Age (years)	
15–19	86 (47.0%)
≥20	97 (53.0%)
Race	
African-American	102 (55.7%)
Latina	43 (23.5%)
White	27 (14.8%)
Other	11 (6.0%)
Age at first intercourse (years)	15.5±2.1
Lifetime number of sex partners	
1–3	54 (29.5%)
4–7	68 (37.2%)
≥8	61 (33.3%)
Sex partners in past 3 months (before baseline)	
≤1	121 (66.1%)
≥2	62 (33.9%)
Previous lifetime STI diagnosis	
Yes	78 (42.6%)
No	105 (57.4%)
STI-related symptoms or concern at time of diagnosis	
Yes	74 (40.4%)
No	109 (59.6%)
Post-diagnosis behaviours and repeat diagnoses	
Sexual activity during follow-up period	
Yes	154 (84.2%)
No	29 (15.8)
Any sex without a condom during follow-up period	
Yes	72 (39.3%)
No	111 (60.7%)
Continued sex with a baseline partner not known to be treated	
Yes	38 (20.8%)
No	145 (79.2%)
New sex partner during follow-up period	
Yes	68 (37.2%)
No	115 (62.8%)
Sex with a partner not known to be monogamous	
Yes	63 (33.4%)
No	120 (66.6%)

Values are number (%) or mean±SD.

STI, sexually transmitted infection.

Table 2

Predictors of repeat C trachomatis infection

Characteristic	Repeat (n=24)	No repeat (n=159)	Unadjusted OR (95% CI)*	Adjusted OR (95% CI)*†
Baseline demographic and sexual risk history variables				
Age				
15–19	17 (19.8%)	69 (80.2%)	3.16 (1.24 to 8.06)	3.19 (1.23 to 8.26)
≥20	7 (7.2%)	90 (92.8%)	1.0	1.0
Race				
White	3 (11.1%)	24 (88.9%)	1.0	NI
African-American	14 (13.7%)	88 (86.3%)	1.27 (0.34 to 4.79)	
Latina	5 (11.6%)	38 (88.4%)	1.05 (0.23 to 4.81)	
Other	2 (18.2%)	9 (81.2%)	1.78 (0.25 to 12.45)	
Age at first intercourse				
<15	17 (16.5%)	86 (83.5%)	2.06 (0.81 to 5.24)	NI
≥15	7 (8.8%)	73 (91.2%)	1.0	
Lifetime number of sex partners				
1–3	6 (11.1%)	48 (88.9%)	1.0	NI
4–7	8 (11.8%)	60 (88.2%)	1.07 (0.35 to 3.28)	
≥8	10 (16.4%)	51 (83.6%)	1.57 (0.53 to 4.65)	
Sex partners in past 3 months				
≤1	15 (12.4%)	106 (87.6%)	1.0	NI
≥2	9 (14.5%)	53 (85.5%)	1.20 (0.49 to 2.92)	
Previous STI diagnosis				
Yes	15 (14.3%)	90 (85.7%)	1.28 (0.53 to 3.10)	NI
No	9 (11.5%)	69 (88.5%)	1.0	
Reason for clinic visit at time of current diagnosis				
STI-related symptoms or concern	12 (16.2%)	62 (83.8%)	1.56 (0.66 to 3.70)	NI
Other	12 (11.0%)	97 (89.0%)	1.0	
Post-diagnosis behaviours and repeat diagnoses				
Sexual activity during follow-up period				
Yes	22 (14.3%)	132 (85.7%)	2.25 (0.50 to 10.14)	NI
No	2 (6.9%)	27 (93.1%)	1.0	
Any sex without a condom during follow-up period				
Yes	12 (16.7%)	60 (83.3%)	1.65 (0.70 to 3.91)	NI
No	12 (10.8%)	99 (89.2%)	1.0	
New sex partner during follow-up period				
Yes	12 (17.6%)	56 (82.4%)	1.84 (0.78 to 4.36)	NI
No	12 (10.4%)	103 (89.6%)	1.0	
Continued sex with a baseline partner not known to be treated				
Yes	10 (26.3%)	28 (73.7%)	3.34 (1.35 to 8.29)	3.37 (1.33 to 8.57)
No	14 (9.7%)	131 (90.3%)	1.0	1.0

Characteristic	Repeat (n=24)	No repeat (n=159)	Unadjusted OR (95% CI)*	Adjusted OR (95% CI)*†
Sex with a partner not known to be monogamous				
Yes	7 (11.1%)	56 (88.9%)	1.0	NI
No	17 (14.2%)	103 (85.8%)	1.32 (0.52 to 3.38)	

*From logistic regression models.

†Estimates adjusted for other covariates in the model.

NI, not included in final multivariate model because of p>0.05; STI, sexually transmitted infection.

Table 3

C trachomatis genotypes at baseline and follow-up and post-treatment behaviours among women diagnosed with repeat infections

Patient	Baseline and follow-up genotypes	Sex partner not known to be treated	New sex partner	Sex partner not known to be monogamous
Same genotypes				
SS2	E–E	Yes	Yes	Yes
SS5	Ia–Ia	No	Yes	Yes
SS8	E–E	No	No	No
Different genotypes				
SS1	J–K	No	Yes	No
SS3	J–E	No	Yes	No
SS4	E–J	No	No	No
SS6	D–E	Yes	No	Yes
SS7	I–E	Yes	Yes	No