ORIGINAL ARTICLE

Modelling the effect of *Chlamydia trachomatis* testing on the prevalence of infection in England: what impact can we expect from the National Chlamydia Screening Programme?

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ABSTRACT

Objective To estimate the impact of increased testing of asymptomatic young people for chlamydia on infection prevalence in England.

Methods An age and sex-stratified deterministic model including three sexual activity groups and the effect of notifying partners of treated individuals was used to describe chlamydia transmission in England. Model predictions were fitted to national chlamydia prevalence data for the year 2000 to estimate unknown parameters, including the transmission rate per partnership.

Results Prevalence was assumed to be at equilibrium in 2000 and found to be 4.5% among 16–24-year-olds. Estimates suggest that population prevalence was 28% lower in 2010/2011 than it would have been had no testing increases occurred. This estimate was insensitive to baseline prevalence but depended on the assumed reinforcement risk and effective notification rate of partners of treated individuals. Annual reductions of 2.4% and 1.4% in prevalence after 2010/2011 were predicted if testing rates thereafter increased to 45% of 16–24-year-olds or remained unchanged at 35%, respectively.

Conclusions Based on available data, the modelling suggests that the current level of chlamydia testing in England is reducing prevalence among 16–24-year-olds. Up-to-date data on population prevalence are needed to confirm this. Investigating the impact of improving partner notification and treatment requires further work.

INTRODUCTION

Genital chlamydia infection is a common sexually transmitted infection, associated with long-term gynaecological sequelae (eg, pelvic inflammatory disease, ectopic pregnancy and tubal factor infertility in women and epididymitis in men) and some neonatal complications. These sequelae are preventable by identifying asymptomatic infections and treating with cheap and effective antimicrobials. This, together with evidence from pilot studies, motivated the creation of the National Chlamydia Screening Programme (NCSP) in England in 2003. The NCSP aims to prevent and control chlamydia through early detection and treatment of asymptomatic infection; to reduce onward transmission to sexual partners; to prevent the consequences of untreated infection; and to ensure all sexually active men and women aged younger than 25 years are aware of chlamydia and its effects and have access to services providing testing, prevention and treatment. Previous modelling work has suggested that, given certain assumptions and simplifications, screening can reduce the prevalence of chlamydia.

The impact of any screening programme on population prevalence is best determined by comparing prevalence data collected before and after the programme starts, ideally within the context of a randomised control trial so that the effects of secular trends can be minimised. The absence of such data complicates the evaluation of the NCSP. In practice, the prevalence of infection in tested individuals (herein referred to as test positivity) differs from that in the general population (herein referred to as prevalence) due to biases towards risk and variations in the characteristics of those tested. Consequently, changes in the test positivity may not reflect changes in prevalence. In the absence of empirical data on changes in prevalence, and to complement such data when they become available, mathematical modelling can be used to simulate the effect of actual chlamydia testing practice and explore how different factors affect the success of the programme.

We present a mathematical model that describes sexual behaviour and chlamydia transmission in England. We use this model to investigate how increased testing for chlamydia in England may have already affected prevalence, and the impact achievable in the future.

METHODS

Overview of the model

We used an age-structured deterministic compartmental model to describe the transmission dynamics of chlamydia in England. The general structure and sexual behaviour basis to our model comes from work by Choi et al on modelling the transmission dynamics of human papillomavirus. The model was used to estimate the infection-specific parameters for chlamydia and adapted to include asymptomatic testing for chlamydia.
(screening and partner notification) as well as the testing and treatment of symptomatic chlamydia infection.

**General structure of the model**

Figure 1 shows the structure of the model. The model population was stratified into those susceptible to chlamydia infection, those infected with chlamydia and those being treated. Infected individuals were assumed to become susceptible to infection after an average duration of 12 months if undiagnosed or not effectively treated (natural resolution) or after a fixed period of treatment lasting 1 month. Individuals were treated if diagnosed through testing, either via screening or self-referral.

Once recovered from infection, individuals were assumed to be indistinguishable from other susceptible individuals, so that neither treatment nor natural resolution confers immunity except for the period while an individual is removed from the population while being treated. We assumed that a proportion of diagnosed individuals did not complete treatment due to non-compliance. For further details of the model structure, see online appendix A.

**Demography**

The model population comprised 43 million heterosexuals aged 12–77 years (ie, similar to that in England in the year 2006), stratified into 780 monthly cohorts equally split between men and women. New individuals were added to the youngest cohort every time step (1 month) so the population remained stable over time. We assumed no migration in or out of the population and that deaths occurred according to age- and gender-specific rates for the UK population in 2006.5

**Sexual behaviour and chlamydia transmission**

The model population was stratified into three sexual activity groups: low, medium and high. These groups corresponded, respectively, to the lowest 80th, the next 15th and the highest 5th percentile for the number of new sexual partners acquired each year, as in a previously published model of sexual behaviour.7 Using three activity groups allows incorporation of some of the important variation in sexual behaviours and networking, without adding too much complexity to the model structure and parameter sets. The groups vary in absolute size but are of equal importance in terms of sexually transmitted infection transmission. The number of new partners per year by gender and age group were derived from the National Survey of Sexual Attitudes and Lifestyles (NATSAL)10 conducted in 2000 (see table A1 in online appendix A). The force of infection depended on age, gender and sexual activity group, together with the transmission rate per partnership.

The sexual mixing matrix was compiled from the partner change rates in NATSAL. The number of partnerships between individuals from different gender and age groups was balanced using published methods.11 Two parameters, the assortativity of mixing by age and by activity group, determined the mixing matrix. Assortativity of mixing by age was 0.48, meaning that 48% of sexual partnerships were with individuals of the same age group and the remainder were proportionate across all other age groups, as estimated by Choi et al.7 Assortativity of mixing by activity group was assumed to be 0.5, meaning that 50% of partnerships were between individuals in the same activity group and the remainder were randomly selected from any activity group. Values of 0 and 0.9 were also considered in sensitivity analyses (see online appendix A).

The rates of chlamydia transmission between individuals are poorly understood. Most of the available data are for concordance of chlamydia infection, which is likely to exceed the partnership transmission rate and is likely to be biased by differences in reinfection rates, treatment-seeking behaviour, duration of partnerships and the levels of sexual activity of participants. For example, one study suggested that the transmission rate per partnership was 30%–40%,12 whereas two studies suggested that it exceeds 50% (ie, about 49%–52%15 (authors’ calculation) and 68%14 (partnership concordance)).

We assumed a fixed transmission rate per partnership (β) for partnerships involving only low-activity individuals. This rate was scaled by a factor (α) to obtain the transmission rate associated with partnerships involving either two medium-activity individuals or a high- and a medium-activity individual; it was scaled by α2 to obtain the transmission rate per partnership between two high-activity individuals. This scaling reflects the fact that two individuals from the high-activity group may have fewer sexual acts in their partnership than two individuals from the low-activity group, thereby reducing their transmission rate per partnership. Parameters α and β were estimated by fitting model predictions of the age- and activity-group-specific infection prevalence to the observed NATSAL data (see below). Alternative assumptions about the transmission rates in partnerships involving from different activity groups worsened the fit of model predictions (see online appendix A).

Assumptions of the proportion of symptomatic infected individuals who seek treatment vary between models, for example, between 0% and 75% for men and 5% and 30% for women.4 15 We therefore assumed that this proportion lies between 10% and 50%, and assumed for simplicity that it was identical for everyone. In the base case, 95% of those receiving treatment were assumed to successfully complete treatment, consistent with data reported to the NCSP.16 Values of 90% and 100% for those successfully completing treatment were also considered in sensitivity analyses.

The model ran for 100 years to allow model prevalence to reach equilibrium. Using maximum likelihood, we estimated the two unknown parameters relating to transmission (the transmission probability per partnership and α, which describes the amount of contact between the different activity groups) by fitting the model prediction of the equilibrium proportion infected in each gender, age and activity group to chlamydia prevalence derived from the NATSAL survey (see online appendix A for further details). The proportion of newly infected individuals who seek treatment varied between 10% and 50%, and we selected the value that maximised the

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**Figure 1** Diagram showing the general structure of the model. Ageing and death are not represented. It is assumed that all individuals with new symptomatic infections immediately seek treatment. It is assumed that following treatment failure, a previously symptomatic infection will be asymptomatic.
likelihood of model predictions for the data. Confidence limits on each parameter were calculated using profile likelihood. Table 1 provides details of the key parameters used in the model, while details of all model parameters are provided in online appendix A.

Data on the prevalence of chlamydia infection

Age- and gender-specific prevalence of chlamydia infection was obtained from the NATSAL 2000 survey. Three thousand five hundred and twenty-nine individuals provided a urine sample that was tested for chlamydia by ligase chain reaction. We excluded data from those whose test result was inconclusive or who provided no information on the number of new sexual partners in the previous year. To better represent the general population, the NATSAL data were weighted for differential selection probabilities into the survey and for the probability of providing a urine sample.

Testing and partner notification

Additional testing of 16–24-year-olds was introduced in year 2000/2001 once model prevalence had reached equilibrium. The levels of testing differed between men and women and were based on reported data for all testing settings: genitourinary medicine (GUM) clinics (from S1 and S2 codes), non-GUM sites, and from 2005/2004 onward, tests performed as part of the NCSP (table 2). Observed test positivity was used to determine the number of infected individuals identified by testing. We assume all settings used tests with sensitivity and specificity of 100%, and the same proportion of symptomatic individuals in each setting.

The testing coverage from 2010/2011 was assumed to either remain unchanged or was increased linearly to reach 45% by 2013/2014. For both scenarios, we assumed that twice as many women were tested than men, and the test positivity was 1.77 times population prevalence in each age and gender group, based on positivity and model prevalence for 2010/2011. Other values were considered but did not qualitatively alter results. Infected individuals were taken from the activity groups in proportion to the relative size of that activity group in the entire infected population. More information about the testing and positivity data is given in online appendix B.

Testing of partners was included in a relatively simplistic way (see online appendix A), and in the base case, we assumed that all partners of 20% infected persons were traced. This is equivalent to tracing an average of 0.5 partners per infected person, which is consistent with the NCSP goals (tracing 0.4 and 0.6 partners per case in rural areas and cities, respectively), though there are qualitative differences between the individuals identified by our model and in practice (see online appendix A for a discussion of this). A base case 40% of cases who do not provide contacts were assumed to be reinfected by an untreated partner, which is consistent with studies that found that 42% of cases in general practice who had not had all their contacts traced were reinected within 1 year. We assumed, as a simplification, no increased risk of reinfection for individuals who provided full details of their partners. We also explored the impact of increased partner notification (40%, approximately one partner per case, and 60%, approximately 1.6 partners per case) and different levels of reinfection by untreated partners (20% and 60%).

Uncertainty and sensitivity analyses

Using rejection sampling we were able to obtain plausible bounds on the impact of testing; the best-fitting parameter estimates were used to generate 100 sets of plausible parameter values consistent with the available data. This process was subject to the same constraints as used during fitting. Parameter combinations leading to the infection prevalence in the low-activity group exceeding that in the high-activity group were rejected.

For each of the estimated parameters, profile likelihood was used to generate 95% confidence intervals. We ran the model for combinations of parameters in these intervals to test the sensitivity of the model predictions of the impact of screening to these parameters. Because the best-fitting parameters involve low transmission rates among highest risk individuals, we also considered an additional scenario where the parameter was calculated to ensure that along with the best-fitting value of the transmission rate, the transmission probability in the highest group was 10%. This lead to a baseline prevalence of around 35% and so this is not discussed further.

We also explored the effect of fitting the model for untreated asymptomatic chlamydia infection lasting 6 months, consistent with ranges observed.

We also considered scenarios with different levels of partner notification and reinfection of individuals by untreated partners (see online appendix A) and explored the sensitivity of the results to different assumptions about positivity for the years and venues in which no data were available (see online appendix B).

<table>
<thead>
<tr>
<th>Parameter</th>
<th>Value</th>
<th>Method of selection</th>
</tr>
</thead>
<tbody>
<tr>
<td>Population at equilibrium</td>
<td>43 million</td>
<td>Fixed</td>
</tr>
<tr>
<td>Age of sexual debut (years)</td>
<td>14</td>
<td></td>
</tr>
<tr>
<td>Assortativity of mixing by age</td>
<td>0.48</td>
<td></td>
</tr>
<tr>
<td>Mean number of new partners per year (entire population)</td>
<td>0.26</td>
<td></td>
</tr>
<tr>
<td>a0, the proportion of the population in the high sexual activity group</td>
<td>0.05</td>
<td></td>
</tr>
<tr>
<td>a1, the proportion of the population in the medium sexual activity group</td>
<td>0.15</td>
<td></td>
</tr>
<tr>
<td>a2, the proportion of the population in the low sexual activity group</td>
<td>0.8</td>
<td></td>
</tr>
<tr>
<td>β, transmission rate per partnership, low-activity couples</td>
<td>0.239 (0.236 to 0.247)</td>
<td>Estimated by fitting model predictions to data</td>
</tr>
<tr>
<td>α, scaling factor for transmission rates for partnerships between individuals in different activity groups (see main text)</td>
<td>0.167 (0.163 to 0.179)</td>
<td></td>
</tr>
<tr>
<td>ϕ, percentage of newly infected individuals who seek treatment (constrained to lie between 10% and 50%, see main text)</td>
<td>15% (10% to 50%)</td>
<td>Fixed (varied in sensitivity analysis)</td>
</tr>
<tr>
<td>D, average duration of asymptomatic infection</td>
<td>12 months (6 months)</td>
<td></td>
</tr>
<tr>
<td>Assortativity of mixing by activity group</td>
<td>0.5 (0 and 0.9)</td>
<td></td>
</tr>
<tr>
<td>Efficacy of treatment (see main text)</td>
<td>95% (90%, 100%)</td>
<td></td>
</tr>
</tbody>
</table>

For estimated parameters (α and β), the values in parentheses are 95% CIs for that parameter generated by profile likelihood. For fixed parameters, the parentheses contain the alternative values used in the sensitivity analysis.
16 in NATSAL and a prevalence of 4.5% in women aged 18–24 years by 2013/2014. Alternatively, if testing is increased to 45% of 16–24-year-olds by 2010/2011, prevalence falls to a median of 2.9% (IQR 2.7% to 3.5%) in men aged 18–24 years compared with 3.0% (95% CI 1.7% to 5.0%) in women aged 18–24 years. Our best-fitting estimates are at the upper end of plausible ranges for the NATSAL observations.

Online appendix A provides further technical details about the model fitting estimates are at the upper end of plausible ranges for the NATSAL observations.

### Results

#### Fitting of the model

Table 1 shows the parameter values that produced the best-fitting model predictions to the NATSAL data using maximum likelihood. This resulted when 15% of infected individuals were assumed to seek treatment and when the transmission rate per partnership (β) involving two low-activity persons was 24%. Of all scenarios considered for the average duration of untreated asymptomatic infections, treatment efficacy and assortativity of mixing by activity group, the best fit is achieved for an average duration of 12 months, treatment efficacy of 95%, assortativity of mixing by risk group equal to 0.5 and when transmission rates are dominated by low-activity partners (see online appendix A, table A4). In this case, the model predicts a prevalence of 4.5% in men aged 16–24 years compared with 2.7% (95% CI 1.2% to 5.8%) in men aged 18–24 years observed in NATSAL and a prevalence of 4.5% in women aged 16–24 years compared with 3.0% (95% CI 1.7% to 5.0%) in women aged 18–24 years. Our best-fitting estimates are at the upper end of plausible ranges for the NATSAL observations.

Online appendix A provides further technical details about the parameter estimates and compares the best-fitting model predictions of chlamydia prevalence by age group, gender and activity group to the observed data.

#### The impact of interventions

Figure 2 shows the effect of testing on the prevalence of chlamydia infection among 16–24-year-olds between 2000/2001 and 2013/2014. In the base case, the prevalence fell from a median of 4.8% at equilibrium in 2000 (IQR 4.4%–5.1%) to a median of 4.4% (IQR 4.0%–4.7%) in 2002/2003 before the start of NCSP testing activity. By 2010/2011, prevalence falls to a median of 3.5% (IQR 3.1%–3.8%), an absolute decrease in prevalence of around 1.5%. This is a 20% reduction since the introduction of the NCSP and a 28% reduction since baseline (2000).

If testing is sustained for 3 years at 2010/2011 levels, prevalence is predicted to fall to 3.3% (IQR 3.0%–3.6%) by 2013/2014. Alternatively, if testing is increased to 45% of 16–24-year-olds by 2013/2014, the prevalence would fall to 3.2% (IQR 2.9%–3.5%). On average, the scenarios of sustained and increased testing are predicted to lead to reductions in prevalence of 1.4% and 2.4% per year, respectively.

#### Uncertainty and sensitivity analyses

Considering different combinations of β and α from the confidence intervals given in table 1 showed that when β and α were smaller than optimal, the baseline prevalence was lower and the impact of the increased testing was greater.

### Table 2

<table>
<thead>
<tr>
<th>Year</th>
<th>Male</th>
<th>Female</th>
<th>Male</th>
<th>Female</th>
<th>Male</th>
<th>Female</th>
<th>Male</th>
<th>Female</th>
<th>Male</th>
<th>Female</th>
<th>Male</th>
<th>Female</th>
</tr>
</thead>
<tbody>
<tr>
<td>2000/1</td>
<td>3.29%</td>
<td>4.82%</td>
<td>17.81%</td>
<td>22.06%</td>
<td>2.673700</td>
<td>2.624300</td>
<td>87.948</td>
<td>126.463</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2001/2</td>
<td>3.93%</td>
<td>6.83%</td>
<td>16.40%</td>
<td>17.83%</td>
<td>2.732000</td>
<td>2.673900</td>
<td>107.328</td>
<td>182.579</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2002/3</td>
<td>4.52%</td>
<td>8.79%</td>
<td>16.41%</td>
<td>16.27%</td>
<td>2.800200</td>
<td>2.716900</td>
<td>126.708</td>
<td>236.696</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2003/4</td>
<td>5.16%</td>
<td>11.40%</td>
<td>16.13%</td>
<td>14.47%</td>
<td>2.872000</td>
<td>2.775700</td>
<td>148.065</td>
<td>316.410</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2004/5</td>
<td>5.93%</td>
<td>14.37%</td>
<td>16.04%</td>
<td>13.46%</td>
<td>2.963900</td>
<td>2.836800</td>
<td>175.770</td>
<td>407.663</td>
<td></td>
<td></td>
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</tr>
<tr>
<td>2005/6</td>
<td>6.76%</td>
<td>17.04%</td>
<td>15.27%</td>
<td>12.29%</td>
<td>3.027700</td>
<td>2.899100</td>
<td>204.777</td>
<td>494.114</td>
<td></td>
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<tr>
<td>2006/7</td>
<td>7.59%</td>
<td>19.40%</td>
<td>14.77%</td>
<td>11.42%</td>
<td>3.088100</td>
<td>2.940700</td>
<td>234.526</td>
<td>570.508</td>
<td></td>
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<td></td>
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<tr>
<td>2007/8</td>
<td>10.08%</td>
<td>24.87%</td>
<td>12.92%</td>
<td>10.53%</td>
<td>3.167800</td>
<td>2.987500</td>
<td>319.289</td>
<td>742.917</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2008/9</td>
<td>15.31%</td>
<td>34.97%</td>
<td>10.35%</td>
<td>9.22%</td>
<td>3.196300</td>
<td>3.030100</td>
<td>489.278</td>
<td>1059.753</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2009/10</td>
<td>21.72%</td>
<td>42.51%</td>
<td>7.79%</td>
<td>7.62%</td>
<td>3.195400</td>
<td>3.042000</td>
<td>694.058</td>
<td>1293.088</td>
<td></td>
<td></td>
<td></td>
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<tr>
<td>2010/11</td>
<td>23.90%</td>
<td>45.55%</td>
<td>6.64%</td>
<td>6.74%</td>
<td>3.219300</td>
<td>3.037900</td>
<td>769.374</td>
<td>1383.852</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2011/12</td>
<td>26.64%</td>
<td>49.65%</td>
<td>1.77%</td>
<td>x population prevalence</td>
<td>24.21%</td>
<td>42.51%</td>
<td>7.79%</td>
<td>7.62%</td>
<td>3.219300</td>
<td>3.037900</td>
<td>769.374</td>
<td>1383.852</td>
</tr>
<tr>
<td>2012/13</td>
<td>29.39%</td>
<td>53.76%</td>
<td>6.64%</td>
<td>6.74%</td>
<td>3.219300</td>
<td>3.037900</td>
<td>769.374</td>
<td>1383.852</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2013/14</td>
<td>32.14%</td>
<td>57.68%</td>
<td>6.64%</td>
<td>6.74%</td>
<td>3.219300</td>
<td>3.037900</td>
<td>769.374</td>
<td>1383.852</td>
<td></td>
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</tbody>
</table>

The population estimates and the imputed aggregate number of tests are also given. For 2011/12, below the solid line, we show the scenario in which testing increases linearly to 45% of individuals aged 16–24 years by 2013/2014. We also consider a scenario in which coverage is fixed at 2010/2011 levels for the following 3 years.
Assuming shorter (6 month) duration of asymptomatic infection led to smaller reductions in absolute prevalence. For example, the median equilibrium prevalence is predicted to be 5.2% (IQR 4.6%–5.8%), decreasing to 4.2% (IQR 3.8%–4.8%) by 2010/2011 (online appendix A, figure A3).

Increasing partner notification leads to a relative reduction in prevalence of around 6% and decreasing the risk of reinfection by untreated partners reduces prevalence by a similar order of magnitude (see online appendix A).

An assumption of constant or less than linear positivity for years without data had little effect on the outcome (online appendix B).

DISCUSSION

Our model describes the transmission of chlamydia infection among heterosexuals in England and the impact of increases in chlamydia screening and diagnoses that have occurred since 2000. The model incorporates uncertainty in many of the parameters required to model chlamydia transmission and intervention using rejection sampling. We can therefore estimate the probable range of reduction in chlamydia prevalence, conditional on our model structure and assumptions about the extent of testing, sexual behaviour and prevalence of chlamydia before screening was introduced.

We have used published data on testing and positivity as observed since 2000 imputing missing data points where necessary. We used the trend in GUM between 2000/2004 and 2010/2011 obtained by linear regression to predict the values of missing data points, while other possible values for these data points did not significantly impact on model predictions (see online appendix B). Using these data means that the model is more reflective of the reality of the screening programme. However, uptake and positivity within the screening programme have varied both temporally and geographically. For example, in 2007/2008 between 2.3% and 7.8% of those eligible were tested for chlamydia, depending on the strategic health authorities encompassed by the programme. Also, sexual behaviour and the prevalence of chlamydia infection before the introduction of screening may vary between different parts of the country (eg, between urban and rural areas). Furthermore, our broad assumption that the conduct of the screening programme and the compliance of young people with testing and treatment was according to recommendations could be doubted, for example, if substantial numbers of infected individuals did not comply with treatment and did not return to the susceptible state when expected. Our findings are for national averages of these parameters and cannot be applied to smaller subregions of the country.

Whether our results are more likely to overestimate or underestimate the impact of screening is debatable. By using data for tests rather than individuals, we implicitly assumed that individuals were tested only once in a given setting and in only one setting in a given year and that current testing behaviour is independent of past testing and test results. In practice, some individuals will have been tested several times so we may have overestimated the number of individuals tested and by extension the effect of increased testing. However, in allocating infected individuals to activity groups randomly, we may have overestimated and/or underestimated the impact of increased testing, particularly if more individuals from higher activity groups have been tested by the programme than we have assumed here. While individuals with higher partner change rates are at increased risk of reinfection compared with low-activity individuals (a potential source of overestimation), identifying and treating them will prevent further transmission of infection (a potential source of underestimation).

Both model structure and parameter choice could have a significant impact on the outcomes of models. Kretzschmar et al compared three individual-based models5 15 22 for the transmission and control of chlamydia and concluded that though the models were broadly consistent with one another, there were significant differences in their predictions for the outcome of a screening programme. While these differences are partly explained by assumptions made concerning levels of treatment seeking, sexual contact structure and transmission probabilities, the complexity of individual-based models means that a definitive explanation of the effect of model assumptions is often difficult. Our model has a slightly lower baseline prevalence for 16–44-year-olds (2.0% for best-fitting parameters) than those models considered by Kretzschmar et al, while the predicted effect of 10 years of screening lies between that of Kretzschmar et al15 and Low et al, albeit with a testing scenario that used observed data.

While our model shows that partner notification is important, the constraints of our model structure mean that our results are not as sensitive to this factor as previous models. The models of Kretzschmar et al15 and Turner et al2 were individual-based and captured information concerning contact structure required for the modelling of partner notification. Kretzschmar et al15 found that doubling partner notification (from 25% to 50% of partners) leads to a 50% additional fall in prevalence (without screening, both outcomes are compared to equilibrium without contact tracing), a far greater impact than that seen in our model. Immunity following natural resolution of infection was not assumed, unlike in some models. Incorporating differences in immunity conferred by treatment over natural resolution would result in different predictions of the impact of the NCSP to those presented here. The sensitivity of our results to uncertainties in the natural history such as these is a subject for future modelling work.

Model predictions were fitted to data from one point in time from a survey conducted 10 years ago (NATSAL 2000). The resulting baseline prevalence in our predictions is at the upper end of 95% CI ranges for those observations, which is partly attributable to the data. For example, no infected individuals are observed among the high-activity individuals in the lowest age group (16–19 years), but data for the entire age range suggest large ORs for prevalence in high-activity individuals (5+ new partners in the previous year). As a result, the model (using sexual behaviour parameters also derived from NATSAL) predicts a higher prevalence for high-activity individuals compared with low-activity individuals in the younger age groups, despite this not being observed in the data. The reliability of the predictions could be improved given data for the prevalence of chlamydia in England from representative population-based sampling at multiple time points. It will be possible to update assumptions about sexual behaviour using data from the latest national survey of sexual behaviour (NATSAL3), which commenced in 2010.

The model predicts that prevalence falls almost linearly despite increasing coverage. The near linear decline since 2000 is a result of screening numbers increasing in GUM prior to the introduction of the NCSP and also because positivity has fallen as testing has increased. As prevalence falls, maintaining the same level of testing coverage will produce diminishing subsequent falls in prevalence—especially in the case where positivity falls. This means that larger increases in coverage are then needed to sustain the steady falls in prevalence. This is one of
the things that makes our model different, and we believe that it is an improvement in model-simulation of reality over previously published models. These have assumed equal risk of positivity among all tests (during the time frames considered) and have shown falls in prevalence to be directly associated with increases in coverage, whereas our model shows that the reality is likely to be more complicated.

The projections considered for testing coverage beyond 2010/2011 demonstrate the benefits of continuing to increase coverage above current levels. However, in assuming that the gender ratio of those tested remains constant, our projection for increasing testing to 45% by 2015/2014 involves screening almost 60% of women, which may be unfeasible. Progress in controlling chlamydia in persons aged <5 years may be better monitored using the annual diagnostic rate among the resident 15–24-year-old population rather than with the testing coverage. The diagnostic rate reflects both coverage and positivity of testing, the two key variables relating to screening activity that we have modelled. Our projections for 35% coverage and 45% coverage, with positivity higher than prevalence as described, equate to diagnostic rates of 2100 per 100,000 and 2700 per 100,000, respectively.

In conclusion, our model has explored the impact of screening in the population of England. Though the conclusions of the model depend on the assumptions made, the data used and though further data are still required, all scenarios indicate that increased testing has led to a decrease in prevalence and that continuing to increase levels of coverage, and diagnoses, will likely improve the rate at which prevalence decreases. Rate of partner notification likely has an important effect, and future work will develop an individual-based model in order to explore the effect of increased partner notification and other issues such as repeated testing and reinfection.

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Contributors All authors contributed to determining research questions and the model design. MDD, YHC and EV conducted the mathematical modelling. MDD and EV adapted code written by YHC to model chlamydia transmission and control. MDD wrote the first draft of the manuscript. All authors provided substantive contributions to the final version of the manuscript.

Competing interests None.

References


Key messages

- We used an age- and sex-stratified deterministic model describing chlamydia transmission in England.
- The model estimated the effects of increased testing and proportions testing positive for chlamydia between 2000/2001 and 2010/2011, along with scenario projections to 2013/2014.
- Based on available data, the modelling suggests that the current level of chlamydia testing in England is reducing prevalence among 16–24-year-olds.
- Up-to-date data on population prevalence are needed in order to validate the model, and the investigation of partner notification requires further work.

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