Correspondence

Authors' reply

Low and Smid's Comment $^{\scriptscriptstyle 1}$ on our Article² analysing chlamydia testing data from England stated: "The model assumes the same screening rate in uninfected and infected people, although people at higher risk of chlamydia infection are more likely to be tested." The authors' use of "screening" interchangeably with "testing" is ambiguous. In our model,23 testing in the absence of symptoms (ie, screening) by those with asymptomatic infection and those who are uninfected is indeed at the same rate. However, the rate of (diagnostic) testing by those with symptomatic infection is much higher. Therefore, the overall rate of testing is higher for those who are infected than for those who are not (figure).

Soldan and colleagues challenge our results² by asserting that nonsymptomatic individuals screened for chlamydia are more likely to be infected than those who are not. There is currently no evidence either for or against this assertion. Various studies have shown that some individual-level behavioural predictors of chlamydia infection also predict testing,^{4,5} but there is currently no evidence to determine whether this relationship can (or cannot) be entirely explained by the mediating effect of symptoms-an effect that is



Figure: Testing rates of individuals with symptomatic infection, asymptomatic infection, and uninfected individuals

All individuals without symptoms are tested (ie, screened) at the same rate, while individuals with symptomatic infection seek (diagnostic) testing at a higher rate. The average testing rate of infected individuals (bold text) is therefore higher than the average testing rate of uninfected individuals (italic text). Current surveillance systems do not record whether patients tested for chlamydia do or do not have symptoms and therefore do not distinguish between diagnostic testing and screening. accounted for in our model. Soldan and colleagues cite two references in support of their assertion. The first does not address screening specifically by non-symptomatic individuals.⁵ The second asserts that variation in positivity in different settings is due to differing proportions of patients being symptomatic, making no mention of variable infection risk in nonsymptomatic patients; furthermore, it states that reasons for variation in positivity by test setting are "assumed" and presents no data on proportions of patients with and without symptoms.

Soldan and colleagues state that we ignored variability in the infection risk of people being tested. In fact, we have considered the matter in detail. We discussed it at length in the original description of the method,³ in which we presented a sensitivity analysis based on the paper they cite,⁵ which found that our prevalence estimates are robust.2,3 In any case, and as we also reported, the risk behaviour of individuals tested is not recorded in the surveillance data so it is not possible to include it in an analysis. Our understanding of chlamydia epidemiology and control is currently limited by the lack of sufficiently detailed data.^{2,3,6}

Soldan and colleagues also state that in our model "the likelihood of being screened... is not variable by time or place except as a result of changing prevalence". In fact, in our geographical analysis considering 1 year² the probability of being screened varies by place and in our temporal analysis considering England as a whole³ it varies with time.

We compared our prevalence-change estimates to changes in positivity for all age-sex groups (appendix). As Soldan and colleagues note, since 2008 the changes in estimated prevalence have largely mirrored changes in positivity. Before 2008, however, positivity fell in all four age-sex groups, while estimated prevalence increased (in men) or was stable (in women). Our estimated changes in prevalence are not simply due to changes in positivity: the scatterplots (appendix) show the absence of correlation.

We propose two studies that could elucidate the controversial relationship between sexual risk, prevalent chlamydia infection, symptoms, and testing. First, data could be collected on symptoms. reason for testing, and risk behaviour in those tested and diagnosed. This approach would yield a highly detailed spatiotemporal dataset: there were 9.5 million recorded testing events (813283 positive) in individuals aged in 15-24 years England in 2012-17. Second, mediation analysis could be used to understand whether the correlation observed between being infected with chlamydia and being tested can-or cannot-be explained entirely by the intermediary effect of symptoms.

We declare no competing interests. The views expressed are those of the authors and not necessarily those of the Department of Health, Medical Research Council, NHS, NIHR, or Public Health England.

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For the 2018 Health Protection Report by Public Health England see https://assets.publishing. service.gov.uk/government/ uploads/system/uploads/ attachment_data/file/713944/ hpr2018_AA-STIs_v5.pdf

See Online for appendix