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Estimated Impact of Screening on Gonorrhea Epidemiology in the United States: Insights From a Mathematical Model

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Abstract

Background: The burden of gonorrhea infections in the United States is high. There are marked disparities by race/ethnicity and sexual orientation. We quantified the impact of screening and treatment on gonorrhea rates in the US population aged 15 to 39 years for the period 2000 to 2015 and estimated the impact that alternative screening strategies might have had over the same period.

Methods: We developed a national-level transmission model that divides the population by race/ethnicity, preferred gender of sex partners, age, gender, and sexual activity level. We compared our fitted model ("base case") to 4 alternative strategies: (i) no screening, (ii) full adherence to current screening guidelines, (iii) annual universal screening, or (iv) enhanced screening in groups with the highest infection burden. Main outcomes were incidence, infections averted, and incidence rate ratios by race/ethnicity. Mean values and 95% credible intervals were calculated from 1000 draws from parameter posterior distributions.

Results: The calibrated model reproduced observed trends in gonorrhea, including disparities in infection burden by race/ethnicity. We estimated that screening for gonorrhea from 2000 to 2015 averted 30% (95% credible intervals, 18–44%) of total infections that would otherwise have occurred. All alternative active screening strategies were estimated to further reduce, but not eliminate, gonorrhea infections relative to the base case, with differential impacts on the subpopulations of interest.

Conclusions: Our model results suggest that screening has reduced gonorrhea incidence in the US population. Additional reductions in infection burden may have been possible over this period with increased screening, but elimination was unlikely.

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The burden of sexually transmitted infections (STI) caused by *Neisseria gonorrhoeae* is high, with almost 400,000 cases reported in the United States in 2015.[1] There are marked disparities in the distribution of infections by geographic region, race/ethnicity, and sexual orientation.[1,2] Two epidemiologically distinct gonorrhea epidemics are occurring in the United States: one is focused in young, black heterosexuals, whereas the other is focused in men who have sex with men (MSM).[2]

Non-Hispanic whites comprise the largest segment of the US population, with black and African Americans and Hispanics forming the largest racial and ethnic minorities, respectively.[3] In 2015, the gonorrhea rate in Hispanics was 1.8 times the rate in non-Hispanic whites, whereas in blacks, the rate was 9.6 times that in non-Hispanic whites.[1] Although national-level estimates of gonorrhea burden in MSM are lacking, regional sentinel surveillance data indicate that reported gonorrhea rates are higher in MSM than in men who have sex with women.[1,2]

Both individual behaviors and broader social determinants likely contribute to elevated gonorrhea incidence in certain population groups.[4,5] Differences in individual behavior (eg, sex partner numbers) are insufficient to explain racial disparities in STIs. Local network factors, such as preferential mixing (assortativity) with partners of the same racial/ethnic group or risk behavior profile may also be important.[6,7] This has implications for infection spread in sexual networks. Imperfect disease surveillance and an incomplete understanding of screening and treatment practices in the population further complicate interpretation of observed infection trends and disparities.[8,9]

Mathematical models are useful for understanding gonorrhea transmission dynamics but capturing intervention effects is challenging. Simple models have suggested that gonorrhea persistence in populations is fragile[10,11]; with incidence sensitive to small changes in treatment and behavior, disease persistence appears untenable in the simplest models, a finding at odds with clinical reality. Adding model complexity to capture population heterogeneity can produce more realistic results.[12–14] A metapopulation modeling approach, which describes the population of interest as multiple interconnected subpopulations, has been proposed when modeling populations with unequally distributed disease burden.[14] Such models account for disease risks associated with subpopulation membership and capture core group dynamics (individuals who contribute disproportionately to gonorrhea transmission in a population), as well as differences in infection trends in subpopulations driven by different types of sexual mixing or health care access.

We developed a metapopulation gonorrhea model that captures epidemiologically important population groups and calibrated this model to multiple data sources for the years 2000 to 2015. Our objectives were to:

- **(i)** Quantify impacts of screening and treatment on both reported and true burden of gonorrhea in the US population as a whole, and in population subgroups over this period;
- **(ii)** Estimate the potential impact of alternate (hypothetical) screening strategies over the same period.

METHODS

Model Overview and Population Structure

We developed a deterministic compartmental model of gonorrhea transmission in the US population aged 15 to 39 years (Fig. 1). Compartments were stratified by age, sex, and sexual activity level. The population was divided into 4 subpopulations defined by race/ethnicity and preferred gender of sex partners: (i) non-Hispanic black heterosexual males and females; (ii) Hispanic heterosexual males and females; (iii) "white and other" heterosexual males and females (encompassing all race/ethnicities except non-Hispanic blacks and Hispanics, including those with unknown race/ethnicity, with white Americans comprising the majority of this group); and (iv) gay, bisexual, and other MSM. Given the data limitations, the MSM population was not stratified by race/ethnicity. We included a "never sexually active" compartment to account for transitions into the sexually active class as individuals aged. Additional details on the population structure are provided in Table 1. Overviews of key model components and data inputs are provided in the subsequent sections, with more complete details provided in the Appendix (see supplemental Appendix, http://links.lww.com/OLQ/A278).

The modeled age categories were 15 to 24 years and 25 to 39 years, with the cut point chosen to align with the current recommendation of routine annual screening for *N. gonorrhoeae* in all sexually active females younger than 25 years.16 For each age-sex-subpopulation stratum, we assigned 10% of the population to a higher sexual activity group, characterized by elevated annual rates of partner acquisition relative to the rest of the subpopulation. Rates of partner change differed by subpopulation (see Appendix for details, http://links.lww.com/OLQ/A278). Sexual partnerships were formed within and across subpopulations. Mixing between MSM and heterosexual subpopulations was assumed to occur via MSM forming sexual partnerships with females.

Gonorrhea Natural History

Gonorrhea natural history in the absence of antimicrobial resistance was modeled using the approach of Garnett et al[11] and is described in detail in the Appendix. Individuals with symptomatic infections were assumed to seek treatment, with a delay between infection onset and receipt of treatment. Asymptomatic cases could be identified and treated via screening. This would include individuals undergoing opportunistic screening and those seeking testing due to perceived risk (eg, partner with identified infection). We also modeled the reporting process, recognizing that not all treated (symptomatic and asymptomatic) cases will be captured in the surveillance data. After treatment or natural recovery, individuals returned to the susceptible state.

Model Fitting

We calibrated parameters describing sexual mixing, gonorrhea natural history, and screening rates using an adaptive Metropolis-Hastings MCMC algorithm implemented in R.[17] This method uses a Bayesian approach to estimate probability distributions for uncertain parameters, given the model and available data. The adaptive procedure optimizes the

proposal distribution by first adapting the size of the covariance matrix to achieve an optimal acceptance rate, and then adapting the shape of the covariance matrix.[18]

Prior parameter distributions were guided by the available data, using point estimates and plausible ranges from the biomedical literature where possible, or expert opinion and assumption when estimates were unavailable (Tables 1 and 2). When information about parameters was scarce (eg, sexual mixing coefficients), we assumed broad priors.

The periods covered by the data sources used for calibration varied, but overall, the model described gonorrhea transmission between 2000 and 2015. Calibration targets were based on National Health and Nutrition Examination Survey (NHANES) prevalence data,[19,20] national gonorrhea case reports,[21] case characteristics as reported by the STD Surveillance Network,[2] and National Survey of Family Growth sexual behavior data.[15]

Model Outputs and Analysis

To evaluate the impact of gonorrhea screening we compared our fitted model ("base case") to a scenario using the same parameters, but with screening removed (ie, only symptomatic cases treated). We also compared the base case to 3 alternative screening scenarios: (i) full adherence to screening guidelines ("guidelines"),16 (ii) annual screening for all age and racial/ethnic groups ("universal"), or (iii) enhanced screening in groups with highest incidence in the base case ("enhanced") (Table 3). All of these scenarios were applied retrospectively to the period 2000 to 2015.

Key model outputs included: total incident gonorrhea infections over the 16-year period, infections averted relative to the base case, actual and reported incidence rates, true and reported incidence rate ratios by race/ethnic group, proportion of male infections occurring in MSM, and number needed to screen to avert an infection. Further details are provided in the Appendix. We calculated mean values and 95% credible intervals (CrI) based on 1000 draws from the parameter posterior distributions, with intervention effect relative to the base case compared within each parameter set draw.

Sensitivity Analysis

In the main analysis, we allowed for differential reporting of symptomatic cases by race/ethnicity and sex. We repeated the model calibration and analysis assuming a single reporting rate for all symptomatic cases.

RESULTS

Model Calibration

The transmission model reproduced trends in reported cases over time and the observed disparities in the burden of gonorrhea in the US population by age, sex, and race/ethnicity (Fig. 2). It also fit well to other calibration targets (S1 Fig, http://links.lww.com/OLQ/A272).

Fitting the model to both prevalence and reported case data required a large proportion of unreported symptomatic cases in males (S2 Fig, http://links.lww.com/OLQ/A273). The risk of symptomatic reporting, relative to the reporting rate in asymptomatic cases, in nonblack

males (0.09; 95% CrI, 0.04–0.15) was estimated to be lower than in black males (0.62; 95% CrI, 0.40–0.83). Overall, reporting for female symptomatic cases was higher, but a similar trend of higher relative risks of reporting for black females was seen (0.61; 95% CrI, 0.37–0.83 for nonblack females; 0.92; 95% CrI, 0.83–0.97 for black females). Estimates of asymptomatic screening and treatment rates were consistent with current screening guidelines,[16] with lower rates in males and higher rates in MSM and females.

Estimated Gonorrhea Burden Between 2000 and 2015

We estimated that approximately 21 million (95% CrI, 1.710 2.6 10) incident gonorrhea cases occurred over the 16-year period, with a trend of stable or slightly increasing incidence in males and declining incidence in females (Fig. 3). For comparison, 4,931,200 cases were reported nationally over this period.21 Incidence was higher in males than females, with a mean male/female ratio of 1.9 (95% CrI, 1.3–2.7). Infection burden was concentrated in MSM, who comprised 67% (95% CrI, 56–76%) of total infections in males.

In 2015, gonorrhea incidence rate ratios were estimated to be 1.8 (95% CrI, 1.6–2.2) and 2.9 (95% CrI, 2.4–3.3) in black males and females, respectively, relative to rates in the overall population for a given sex (Fig. 3). Among black males, the rate ratio for incidence was smaller than the rate ratio for reported cases (3.9; 95% CrI, 3.4–4.5). By contrast, for black females, the rate ratio for reported cases was not significantly different from that for incidence.

Quantifying the Impact of Screening and Treatment

We compared our base-case model with a counterfactual scenario that assumed a complete absence of screening and treatment of asymptomatic infections. Model comparison suggested that 30% (95% CrI, 18–44%) of total infections were averted by the screening undertaken between 2000 and 2015, with a larger effect observed in females (40% of infections averted; 95% CrI, 27–54%) than males (23%; 95% CrI, 11–37%). However, in MSM, screening was estimated to have averted a negligible number of infections (mean of 0%; 95% CrI, −6% to 8%). In 2015, screening only modestly reduced disparities in incidence in the black population, with most of the effect concentrated in males (incidence rate ratio of 2.5 (95% CrI, 2.0–2.9) without screening and 1.8 (95% CrI, 1.6–2.2) with screening).

Impact of Alternative Screening Approaches

We compared our base-case model estimates to alternative screening approaches that might have been used between 2000 and 2015 (Fig. 4). We estimated that perfect adherence to guidelines (Guidelines) would have averted 51% (95% CrI, 23–75%) of gonorrhea infections. Uncertainty in this scenario was due in part to parameter combinations that markedly reduced transmission in MSM, who experience the most disease.

Annual screening for the entire sexually active population (Universal) was estimated to have a similar effect to following guidelines but was less effective for reducing infection burden in MSM, as screening frequency in this group was reduced compared with guideline recommendations. Enhanced screening (Enhanced) was estimated to most effectively reduce

both overall infection burden and racial disparities in incidence (Fig. 5). Although transmission was significantly reduced, gonorrhea persisted in most simulations, primarily in MSM.

Adherence to screening guidelines (Guidelines) was identified as the most efficient strategy for averting gonorrhea infections, whereas universal screening was the least efficient, requiring over 5 times as many screening tests as with the guidelines to avert a single infection (Fig. 6).

Sensitivity of Model Results to the Assumption of Differential Reporting

Repeating calibration without differential reporting of symptomatic cases by race/ethnicity or sex resulted in a low estimated risk of reporting of symptomatic cases relative to asymptomatic cases (0.27; 95% CrI, 0.16–0.41) (S3 Fig and S4 Fig, http://links.lww.com/OLQ/A274, http://links.lww.com/OLQ/A275). The relative burden of infection in MSM was estimated to be lower than that in the main analysis, and consequently, there was a smaller difference in incidence between males and females (S5 Fig, http://links.lww.com/OLQ/A276). Screening was estimated to have been more impactful, averting 38% (95% CrI, 20–55%) of incident cases over the period, relative to no screening.

With the exception of the guidelines strategy, all alternate screening approaches were expected to be as, or more, effective than was estimated in the main analysis (S6 Fig, http://links.lww.com/OLQ/A277). The enhanced screening approach dramatically reduced the total number of new infections to ~7% of what was estimated with screening at base-case rates. By contrast, adherence to guidelines was as, or less, effective for reducing incidence, compared with our findings with differential reporting.

Without differential reporting, racial/ethnic disparities in males were larger than estimated in the main analysis and estimates of disparities using reported cases were more reflective of true underlying differences in incidence in males (S6 Fig, http://links.lww.com/OLQ/A277). Despite the changes in the estimated impact of the different screening approaches on various population groups without differential reporting, the relative efficiencies of the strategies did not change, with adherence to guidelines remaining the most efficient approach for reducing overall gonorrhea incidence in the population (S6 Fig, http://links.lww.com/OLQ/A277).

DISCUSSION

We developed a dynamic mathematical model that describes gonorrhea transmission in the United States, including observed disparities in infection burden. This novel model provides a platform for estimating the burden of both nonreported and reported gonorrhea infections, as well as the impact of current and alternative screening approaches, in a way that captures population characteristics relevant for prevention.

We demonstrated that screening likely reduced gonorrhea incidence in the population over the years 2000 to 2015, although this impact was primarily seen in heterosexual men and women. Alternate screening strategies could have further reduced gonorrhea burden in the population, but with differential impacts on the subpopulations of interest. In particular,

adhering to guidelines had the potential to reduce gonorrhea transmission in MSM, whereas universal and enhanced screening approaches were more effective at reducing racial/ethnic disparities. As we compared gonorrhea trends under our best estimates of actual screening coverage to what might have been achieved in an ideal world, it is notable that even the most intensive screening strategy was not expected to eliminate gonorrhea transmission. This is an important finding that contrasts with previously published models.[10,11] Our results suggest that a shift in programmatic focus from elimination to other outcomes (eg, improved case management, reduction in repeat infections, or reducing disparities) may be appropriate.[22]

Although our aim was to estimate gonorrhea burden and disparities, and identify programmatic alternatives, the exercise of model calibration to available data also provided important insights into heterogeneities in both gonorrhea epidemiology and surveillance in the United States. The calibrated model estimates a low probability of reporting of symptomatic infections that varies by race/ethnicity and gender. This low level of reporting may appear inconsistent with improvements in electronic and automated reporting systems that have been implemented in many jurisdictions.[23,24] Reasons for this finding could include individuals not seeking treatment, self-treatment[25] or cases receiving presumptive treatment without laboratory testing. Our calibration results suggested a higher reporting probability in black Americans than the rest of the population, which could reflect differential health care access and utilization patterns by race/ethnicity,[4,26–29] which in turn may impact reporting.[8] The implication of differential case reporting by race/ethnicity is that underlying relative disparities in gonorrhea incidence may be exaggerated in the reported case data. When we repeated our model calibration without allowing for differential reporting of symptomatic cases, we did observe some differences in the estimated impact of different screening strategies, with the major divergence relating to the estimated impact of adhering to current screening guidelines. In the absence of differential reporting, adherence to guidelines was estimated to enhance relative disparities but possibly reduce absolute disparities in gonorrhea burden.

Like any mathematical model, ours has limitations. We had nonoverlapping time series for several of the data sources used for model fitting and limited data on changes in screening and reporting over time. The model also had a large number of parameters, some of which were informed by limited data or relied on expert opinion or assumption. Although the calibration approach allowed us to account for the uncertainty associated with our data sources and input parameters and to propagate that uncertainty in our model estimates, issues of parameter identifiability were a concern. However, we did not use the model fitting process to attempt to infer the true values of individual parameters; rather we used this approach to identify combinations of parameters that reproduced trends in the data. Our model was able to replicate the surveillance data with reasonable fidelity, but did not capture the increase in male cases aged 25 to 39 years that has been observed since 2011.[1] Given this limitation, and the overall challenges associated with fitting the model to multiple data sources, we focused our analysis on the period between 2000 and 2015, rather than forecasting future trends. Of necessity our model included a number of simplifying assumptions: we did not model different anatomical sites of infection or screening. Our approach to modeling sexual behavior resulted in varying levels of partner change in different subpopulations, such that mapping of modeled interventions onto current screening

guidelines was an approximation. An alternate modeling approach, such as an agent-based model, would be better able to apply screening to individuals with specific risk factors, at the expense of added model complexity. Our model does not account for emergence of antimicrobialresistant gonorrhea,[30] which could attenuate the impact of any screening program. Because this was a retrospective analysis, we reasoned that the impact of resistant strains on transmission was likely to be relatively minor. Nonetheless, our finding that screening has the potential to reduce gonorrhea incidence must be interpreted with caution, because modeling studies have demonstrated that increased treatment of gonorrhea has the potential to increase the spread of resistance (31 s, 32 s). Given that screening for asymptomatic cases may play a key role for limiting the spread of antimicrobial resistant strains, the inclusion of resistance in models projecting future trends will be critical.

Using a mathematical model calibrated to multiple data sources, we have shown that screening has likely reduced the gonorrhea burden in the US population and can be used strategically to further control infection spread. It is important to note that we find that gonorrhea was likely to have persisted in United States over the period modeled, regardless of the screening strategy used.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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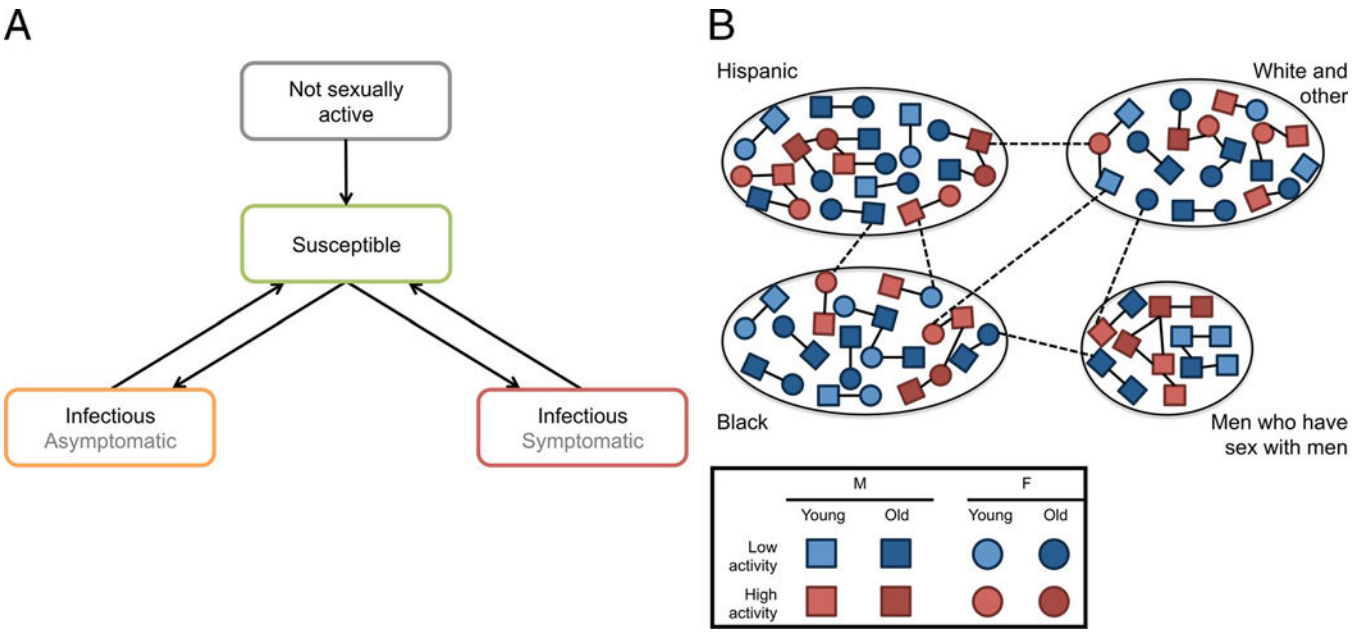

Figure 1.

Outline of model structure. A, The natural history of gonorrhea is described by the following states: not sexually active, susceptible, and infectious. Infectious individuals may have either symptomatic or asymptomatic infection. Transition rates between health states are defined in Tables 1 and 2. Individuals enter the model via the not sexually active or susceptible states and can exit by all states. B, The model is stratified by subpopulation, sex, age, and sexual activity group. The young and old age groups include individuals aged 15 to 24 years and 25 to 39 years, respectively. The lower and higher activity groups are defined based on relative rates of partner change. The black, Hispanic, and "white and other" (non-Hispanic nonblack) subpopulations describe heterosexual partnerships between males and females. Sexual mixing between gay, bisexual, and other MSM and the other subpopulations is assumed to occur via sexual contact between MSM and females in the heterosexual subpopulations.

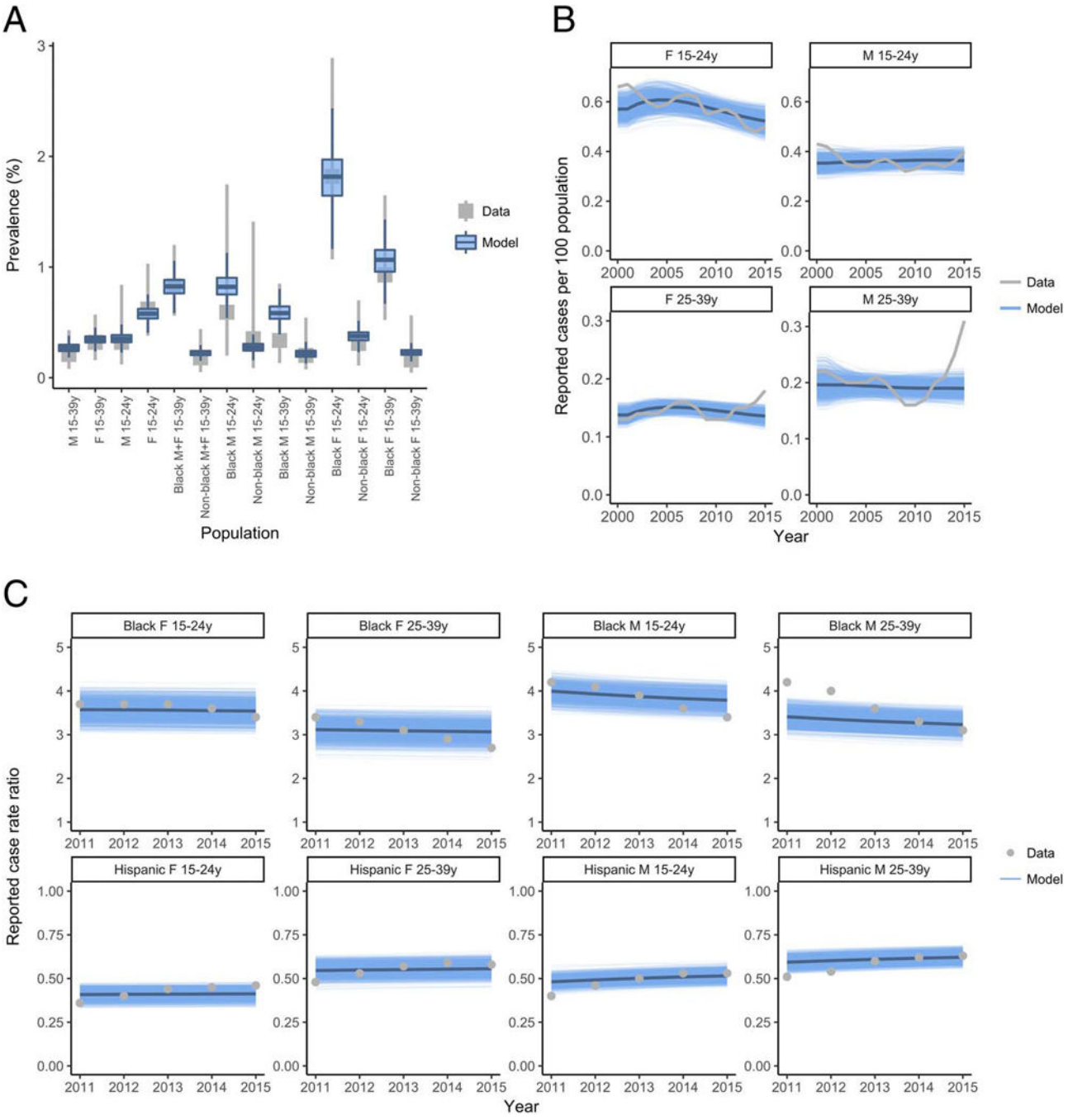

Figure 2.

Model fits to national estimates of gonorrhea prevalence and reported cases. Comparison of model outputs (blue) to data (gray) for different calibration targets. A, Average gonorrhea prevalence for the indicated population groups, as estimated from NHANES (1999–2008). Boxes represent the mean and lines represent the 95% confidence intervals for the NHANES data. For the model estimations, the lower, middle, and upper hinges of the box correspond to the 25th, 50th and 75th percentiles, with the whiskers extending to the largest and smallest values up to 1.5 times the interquartile range. B, Reported gonorrhea cases by age

group and sex, 2000 to 2015. C, Reported case rate ratios by age group, sex, and race/ethnicity, as estimated from national surveillance data. Note that rate ratios for the “white and other” population were not used as calibration targets and so are not shown here. Model outputs from 1000 model simulations are shown. F and M indicate female and male, respectively. For panels B and C, the darker blue line indicates the mean value.

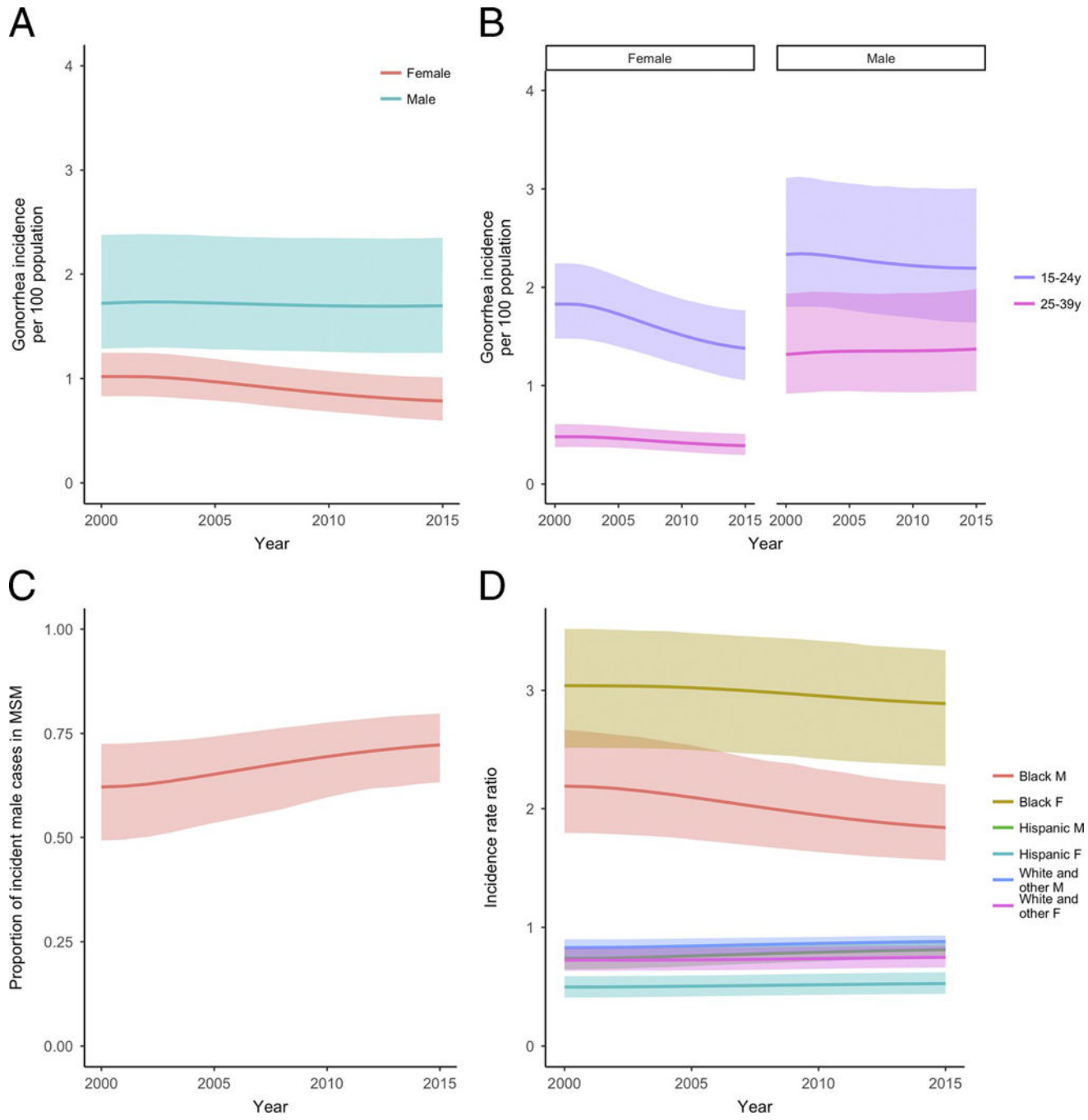

Figure 3.

Model-estimated trends in gonorrhea incidence. A, Gonorrhea incidence in males and females over time. B, Gonorrhea incidence over time by age group and sex. C, Proportion of male gonorrhea cases occurring in MSM over time. D, Trends in incidence risk by race/ethnicity and sex, relative to overall incidence for a given sex. Solid lines represent the mean, and shaded areas the 95% CrI of 1000 simulations.

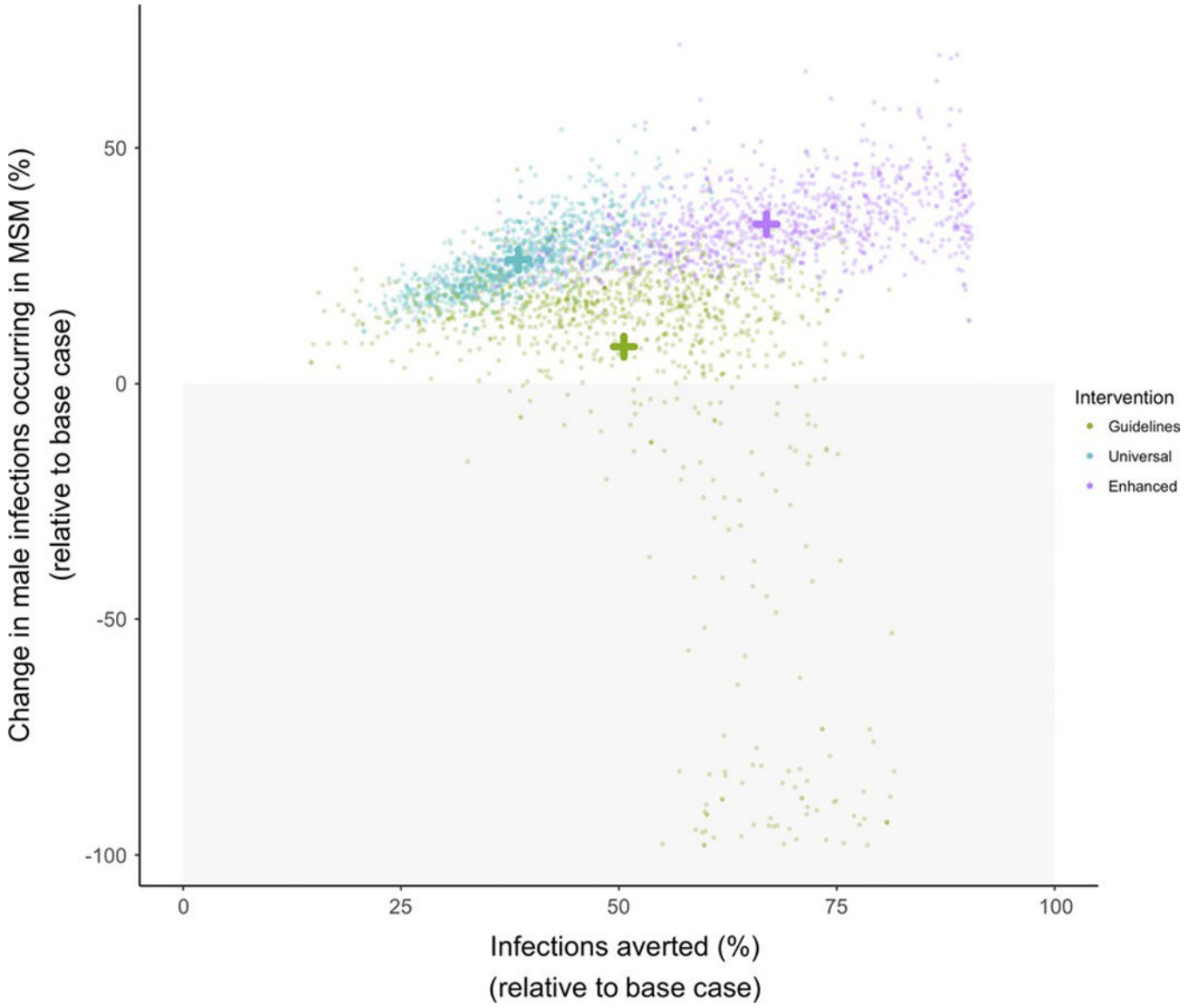

Figure 4.

Infections averted and change in burden in MSM with different screening strategies. Infections averted and the change in percentage of incident male infections occurring in MSM were calculated relative to estimates under the base-case scenario. Each point represents a single simulation result and crosses indicate the mean values for each screening strategy. Results are for 1000 simulations with parameters drawn from posterior distributions.The gray shaded area highlights simulations where the burden of infections in MSM was reduced with the alternate screening approach relative to what was observed under base-case conditions.

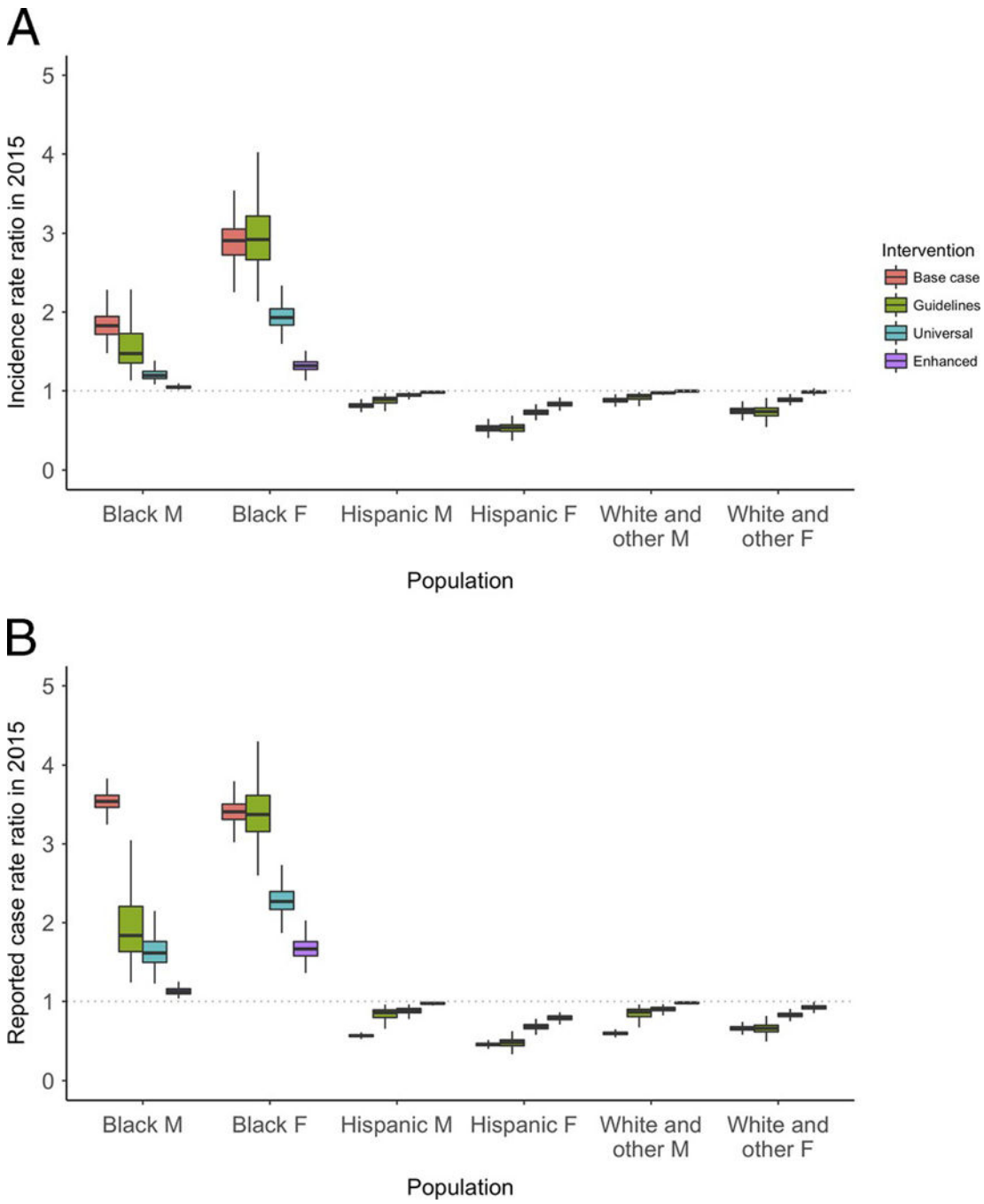

Figure 5.

Infection burden in subpopulations with different screening strategies. The risk of being an (A) incident or (B) reported case for a given sex and racial/ethnic group is shown relative to the overall sex-specific rate for the different screening approaches (described in [Table 3]). The dotted line indicates a rate ratio of 1 (no differential burden by race/ethnicity). Results are for 1000 simulations with parameters drawn from posterior distributions.

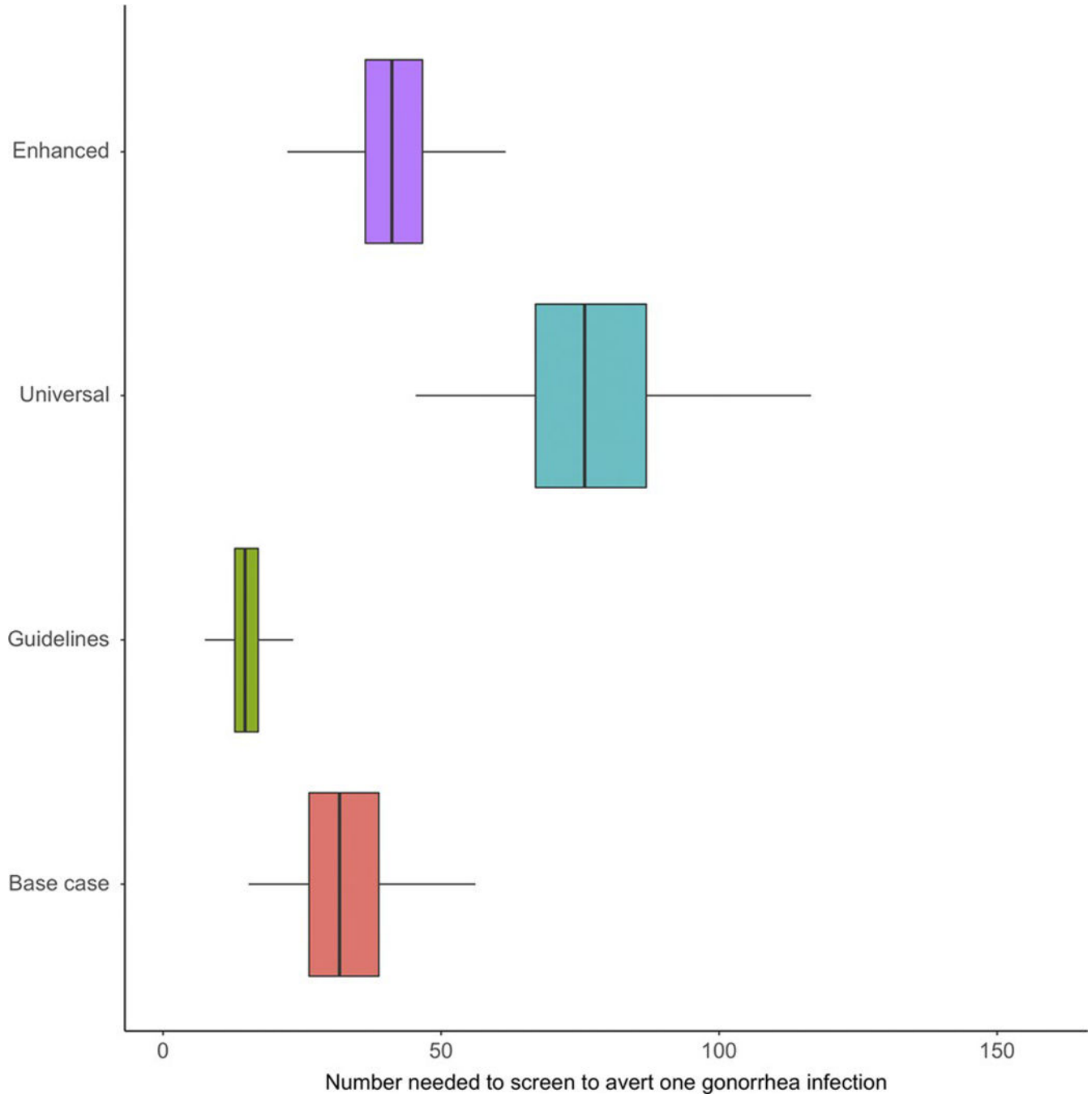

Figure 6.

Number needed to screen to avert a single gonorrhea infection with different screening strategies. For each screening strategy, the number needed to screen was calculated as the total number of tests performed between 2000 and 2015 divided by the number of gonorrhea infections averted (relative to no screening and treatment of asymptomatic cases). Screening strategy details are provided in Table 3. Results are for 1000 simulations with parameters drawn from posterior distributions.

TABLE 1.

Population Structure, Sexual Behavior, and Mixing Parameters

Parameters	Details	Symbol*	Prior Distribution†	Value (Mean and 95% CrI)	Source
Total population size	Aged 15–39 y	N	Fixed	10^8	(3)
Average time in model (y)		1/μ	Fixed	25	Assumption
Proportion of males in each subpopulation		pop_{ij}			(3, 33 s)
	Black		Fixed	0.13	
	Hispanic		Fixed	0.21	
	MSM		Fixed	0.039	
	White and other		Fixed	0.63	
Proportion of females in each subpopulation		pop_{ij}			(3)
	Black		Fixed	0.14	
	Hispanic		Fixed	0.21	
	White and other		Fixed	0.65	
Proportion ever had sex, male		$P_{S,ijkl}$			(15)
	Black, 15–24 y		Fixed	0.78	
	Hispanic, 15–24 y		Fixed	0.68	
	White and other, 15–24 y		Fixed	0.64	
	MSM, 15–24 y		Fixed	0.67 (male average)	
	Black, 25–39 y		Fixed	0.99	
	Hispanic, 25–39 y		Fixed	0.97	
	White and other, 25–39 y		Fixed	0.96	
	MSM, 25–39 y		Fixed	0.96 (male average)	
Proportion ever had sex, female		$P_{S,ijkl}$		(15)	
	All, 15–24 y		Fixed	0.66	
	All, 25–39 y		Fixed	0.98	
Proportion of population in each sexual activity group					Assumption
	Low		Fixed	0.90	
	High		Fixed	0.10	
Minimum rate of partner acquisition (per y)		$c_{min,jl}$		Assumption	
	Male, 15–24 y		Gamma (5, 5)	1 (0.32–2.0)	
	Male, 25–39 y		Gamma (5, 5)	1 (0.32–2.0)	
	Female, 15–24 y		Gamma (5, 5)	1 (0.32–2.0)	
	Female, 25–39 y		Gamma (5, 5)	1 (0.32–2.0)	
	MSM, 15–24 y		Gamma (5, 5)	1 (0.32–2.0)	
	MSM, 25–39 y		Gamma (5, 5)	1 (0.32–2.0)	
Relative rate of partner acquisition, males 15–24 y		rp_{ijkl}			(15, 31 s); assumption

Parameters	Details	Symbol*	Prior Distribution†	Value (Mean and 95% CrI)	Source
	Black, low		Gamma (2.2, 0.6)	3.7 (0.5–10.0)	
	Black, high		Normal (32.5, 8.9)	32.5 (15.0–50.0)	
	Hispanic, low		Fixed	1	
	Hispanic, high		Gamma (4.3, 0.6)	7.0 (2.0–15.0)	
	White and other, low		Gamma (2.2, 0.6)	3.7 (0.5–10.0)	
	White and other, high		Normal (27.5, 11.5)	27.5 (5.0–50.0)	
	MSM, low		Fixed	1	
	MSM, high		Normal (45, 15.3)	45.0 (15.0–75.0)	
Relative rate of partner acquisition, males 25–39 y		rp_{ijkl}			(15, 31 s); assumption
	Black, low		Gamma (3.4, 1.6)	2.2 (0.5–5.0)	
	Black, high		Normal (45, 15.3)	45.0 (15.0–75.0)	
	Hispanic, low		Fixed	1	
	Hispanic, high		Gamma (5.3,0.4)	14.9 (5.0, 30.0)	
	White and other, low		Gamma (3.4, 1.6)	2.2 (0.5–5.0)	
	White and other, high		Normal (45, 15.3)	45.0 (15.0–75.0)	
	MSM, low		Fixed	1	
	MSM, high		Normal (45.0, 15.3)	45.0 (15.0–75.0)	
Relative rate of partner acquisition, females 15–24 y		rp_{ijkl}			(15, 31 s); assumption
	Black, low		Gamma (2.2, 0.6)	3.7 (0.5–10.0)	
	Black, high		Gamma (1.9, 0.1)	17.7 (2.0–50.0)	
	Hispanic, low		Fixed	1	
	Hispanic, high		Gamma (4.3, 0.6)	7.0 (2.0, 15.0)	
	White and other, low		Gamma (2.2, 0.6)	3.7 (0.5–10.0)	
	White and other, high		Gamma (5.3, 0.4)	14.9 (5.0–30.0)	
			Prior	Value (Mean and	
Relative rate of partner acquisition, females 25–39 y		rp_{ijkl}			(15, 31 s); assumption
	Black, low		Gamma (3.4, 1.6)	2.2 (0.5–5.0)	
	Black, high		Gamma (8.5, 0.8)	11.3 (5.0–20.0)	
	Hispanic, low		Fixed	1	
	Hispanic, high		Gamma (5.3,0.4)	14.9 (5.0, 30.0)	
	White and other, low		Gamma (3.4,1.6)	2.2 (0.5–5.0)	
	White and other, high		Gamma (5.3,0.4)	14.9 (5.0–30.0)	
Assortativity of mixing between sexual activity groups		$\varepsilon_{1,i}$			Assumption
	Black		Beta (1.1, 1.1)	0.5 (0.032–0.97)	
	Hispanic		Beta (1.1, 1.1)	0.5 (0.032–0.97)	
	White and other		Beta (1.1, 1.1)	0.5 (0.032–0.97)	
	MSM		Beta (1.1, 1.1)	0.5 (0.032–0.97)	

Parameters	Details	Symbol*	Prior Distribution†	Value (Mean and 95% CrI)	Source
Assortativity of mixing between age groups		$\varepsilon_{2,ijl}$			(15); assumption
	Male 15–24 y and Female 25–39 y		Beta (9, 2.7)	0.77 (0.5–0.95)	
	Male 25–39 y and Female 15–24 y		Beta (6.1, 2.3)	0.72 (0.4–0.95)	
	MSM		Beta (8.0, 3.8)	0.68 (0.40–0.90)	
Proportion of contacts occurring within subpopulation		$\varepsilon_{3,ij}$			(15, 34 s)
	Black male		Beta (172.7, 52.4)	0.77 (0.71–0.82)	
	Hispanic male		Beta (183.7, 72.6)	0.72 (0.66–0.77)	
	White and other male		Beta (547.2, 70.3)	0.89 (0.86–0.91)	
	MSM		Beta (47.5, 2.5)	0.95 (0.88–0.99)	
	Black female		Beta (217.0, 28.8)	0.88 (0.84–0.92)	
	Hispanic female		Beta (99.1, 59.1)	0.63 (0.55–0.70)	
	White and other female		Beta (437.1, 70.4)	0.86 (0.83–0.89)	

*Subscripts *i, j, k,* and *l* indicate subpopulation, sex, sexual activity group, and age group, respectively.

†Gamma distributions are described by shape (α) and rate (β) parameters; beta distributions are described by shape parameters (α and β).

TABLE 2.

Gonorrhea Natural History, Screening, and Treatment Parameters

Parameters	Details	Symbol*	Prior Distribution†	Value (Mean and 95% CrI)	Source
Probability of transmission		β_{ji}			(10, 11, 35 s-39 s)
	Female to male		Beta (4.5, 4.5)	0.50 (0.20–0.80)	
	Male to female		Beta (34.6, 8.9)	0.80 (0.66–0.90)	
	Male to male		Beta (4.5, 4.5)	0.50 (0.20–0.80)	
Average duration of symptomatic infection, d		$1/\gamma_{ij}$			(2)
	Male		Gamma (3.2, 0.36)	9.0 (2.0–20.9)	
	Female		Gamma (3.2, 0.36)	9.0 (2.0–20.9)	
	MSM		Gamma (3.2, 0.36)	9.0 (2.0–20.9)	
Average duration of asymptomatic infection, d		$1/\delta_{ij}$			(11, 36 s, 39 s, 40s)
	Male		Normal (227.5, 70.2)	227.5 (90–365)	
	Female		Normal (227.5, 70.2)	227.5 (90–365)	
	MSM		Normal (227.5, 70.2)	227.5 (90–365)	
Probability of symptomatic infection		σ_{ij}			(10, 19, 36 s, 41 s-43 s); assumption for MSM
	Male		Beta (5.7, 1.6)	0.8 (0.45–0.98)	
	Female		Beta (9.2, 13.6)	0.40 (0.22–0.61)	
	MSM		Beta (8.0,3.8)	0.70 (0.40–0.90)	
Annual asymptomatic screen and treat rate, low sexual activity group‡		ψijl	Bezier curve		Self-reported testing for chlamydia (females) or any STI (males) as reported in[15]; assumption
	Black male, 15–24 y		Start: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
			End: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
	Nonblack male, 15–24 y		Start: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
			End: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
	MSM, 15–24 y		Start: Beta(5.7, 10.2)	0.36 (0.15–0.60)	
			End: Beta (7.0, 9.9)	0.42 (0.20–0.65)	
	Black male, 25–39 y		Start: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
			End: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
	Nonblack male, 25–39 y		Start: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
			End: Beta (2.6, 22.3)	0.11 (0.02–0.25)	
	MSM, 25–39 y		Start: Beta(5.7, 10.2)	0.36 (0.15–0.60)	
			End: Beta (7.0, 9.9)	0.42 (0.20–0.65)	
	Black female, 15–24 y		Start: Beta (12.6, 24.3)	0.34 (0.20–0.50)	
			End: Beta (24.7, 33.8)	0.42 (0.30–0.55)	
	Nonblack female, 15–24 y		Start: Beta (12.6, 24.3)	0.34 (0.20–0.50)	

Parameters	Details	Symbol*	Prior Distribution†	Value (Mean and 95% CrI)	Source
			End: Beta (24.7, 33.8)	0.42 (0.30–0.55)	
	Black female, 25–39 y		Start: Beta (6.7, 22.0)	0.23 (0.10–0.40)	
			End: Beta (7.9,17.4)	0.31 (0.15–0.50)	
	Nonblack female,		Start: Beta (6.7, 22.0)	0.23 (0.10–0.40)	
	25–39 y		End: Beta (7.9,17.4)	0.31 (0.15–0.50)	
Relative risk of screening, by subpopulation and sex‡		rr_pop_{ij}			Estimated from self-reported testing for chlamydia (females) or any STI (males) as reported in[15]; assumption
	Non-Hispanic male or female		Fixed	1	
	Hispanic male		Gamma, (8.5, 7.5)	1.1(0.5–2.0)	
	Hispanic female		Gamma (8.5, 7.5)	1.1(0.5–2.0)	
Relative risk of screening, by sexual activity group‡		rr_ac_k			Assumption
	Lower sexual activity group		Fixed	1	
	Higher sexual activity group		Gamma (8.5, 7.5)	1.1(0.5–2.0)	
Probability asymptomatic case is reported if treated		Π_a	Bezier curve		Assumption
	Model start (2000)		Beta (90.1, 25,5)	0.78 (0.7–0.85)	
	Model end (2015)		Beta (116.1, 12.1)	0.91 (0.85–0.95)	
Relative risk case is reported if symptomatic		$rr_{symp,ij}$			Assumption
	Black male		Beta (1.1,1.1)	0.5 (0.03–0.97)	
	Nonblack male		Beta (1.1,1.1)	0.5 (0.03–0.97)	
	Female		Beta (1.1,1.1)	0.5 (0.03–0.97)	
	Nonblack female		Beta (1.1,1.1)	0.5 (0.03–0.97)	
Annual increase in transmission probability	MSM	c_{rr}	Beta (1.1,36.9)	0.03 (0.001, 0.1)	Assumption
Proportion of cases in MSM captured in NHANES prevalence estimates	MSM	pNHANES	Beta (1.1,1,1)	0.50 (0.03–0.97)	Assumption

*Subscripts *i, j, k,* and *l* indicate subpopulation, sex, sexual activity group, and age group, respectively.

†Gamma distributions are described by shape (α) and rate (β) parameters; beta distributions are described by shape parameters (α and β); normal distributions are described by mean (μ) and standard deviation (σ).

‡Annual screening rates (φ_{ijkl}) calculated as: $\psi_{ijl} \times rr\_pop_{ij} \times rr\_ac_k$.

TABLE 3.

Screening Scenarios

Scenarios	Details
Base case	Screening follows trends for 2000–2015 as estimated by model fitting
No screening	Treatment of symptomatic cases only
Guidelines	Screen all sexually active females aged <25 y every 12 mo
	Screen all sexually active higher activity females aged 225 y every 12 mo
	Screen all sexually active lower/higher activity MSM every 12/3 mo*
	No screening in non-MSM males
Universal	Screen the entire sexually active population annually
Enhanced	Screen lower/higher activity black males every 12/6 mo Screen lower/higher activity Hispanic and 'white and other' males never/annually Screen lower/higher activity MSM every 6/3 mo Screen lower/higher activity black females every 12/3 mo Screen lower/higher activity Hispanic and "white and other" females every 12/6 months
	Screen lower/higher activity black males every 12/6 mo
	Screen lower/higher activity Hispanic and 'white and other' males never/annually
	Screen lower/higher activity MSM every 6/3 mo
	Screen lower/higher activity black females every 12/3 mo
	Screen lower/higher activity Hispanic and "white and other" females every 12/6 months

*Shown are the screening intervals for lower (every 12 months) and higher (every 3 months) activity MSM; the same notation is used elsewhere in this table.