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Transmission probabilities and durations of immunity for three pathogenic group B *Streptococcus* serotypes

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Abstract

Group B *Streptococcus* (GBS) remains a major cause of neonatal sepsis and is an emerging cause of invasive bacterial infections. The 9 known serotypes vary in virulence, and there is little cross-immunity. Key parameters for planning an effective vaccination strategy, such as average length of immunity and transmission probabilities by serotype, are unknown. We simulated GBS spread in a population using a computational model with parameters derived from studies of GBS sexual transmission in a college dormitory. Here we provide estimates of the duration of immunity relative to the transmission probabilities for the 3 GBS serotypes most associated with invasive disease: Ia, III, and V. We also place upper limits on the durations of immunity for serotype Ia (570 days), III (1125 days) and V (260 days). Better transmission estimates are required to establish the epidemiological parameters of GBS infection and determine the best vaccination strategies to prevent GBS disease.

Keywords

vaccines; modeling; *Streptococcus agalactiae*; group B *Streptococcus*

Group B *Streptococcus* (GBS) is a major cause of sepsis and meningitis in neonates, and it has also recently emerged as a leading cause of invasive disease in adults, especially among the elderly and chronically ill. Indeed, there are now more GBS-associated deaths among the elderly than among neonates in the United States [1, 2] and the incidence of GBS disease among nonpregnant adults increased by 32% between 1999 and 2005. While GBS remains susceptible to penicillin and ampicillin, the antibiotics most often used for intrapartum prophylaxis, and to vancomycin, resistance to the second-line antibiotics erythromycin and clindamycin is 32% and 15% respectively nationwide [1]. For this reason the development of new GBS prevention strategies, especially a vaccine, is a research priority [3].

Vaccine development is complicated by the fact that GBS bacteria are divided into nine serotypes based on their expression of capsular polysaccharide antigens and most current vaccine candidates are designed to provoke an immune response to the capsular

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polysaccharide [4, 5, 6, 7, 8], which means that each serotype could require a different vaccine. More recently, researchers have begun to explore the possibility of creating a universal vaccine by targeting other protein antigens whose structures are conserved across GBS serotypes [3, 9, 10, 11, 12, 13].

Assuming a vaccine can be developed, however, we still need to determine an effective vaccination strategy for preventing GBS disease within high-risk groups. Developing such a strategy will require knowledge of the significant GBS transmission pathways and their relative importance. GBS transmission occurs principally via person-to-person close contact, although foodborne transmission may also occur. For example, GBS colonization is associated with sexual activity [14, 15, 16, 17, 18, 19] and fish consumption [14], but not casual contact [20]. Unfortunately, although recent studies have determined average colonization durations for several GBS serotypes through indirect means [20], little is known about other important parameters related to GBS transmission, such as the probability of transmission per sexual contact or average length of immunity following colonization. Furthermore, all parameters may potentially show serotype-dependence, about which little is known. Because colonization status can vary dramatically over periods as short as three weeks [20] and there are currently no direct measurements of the duration of carriage by serotype, traditional statistical methods are insufficient for estimation of these important transmission parameters.

Here we estimate serotype-specific GBS transmission probabilities per sexual contact using stochastic computer modeling of GBS spread in a population of sexually-active individuals. We obtain estimates of the transmission probabilities relative to the average duration of immunity by optimizing the parameters of our model to fit known prevalence data.

Methods

Data collection

Three GBS serotypes (Ia, III, and V) are currently responsible for most invasive disease in the United States [1]. Population prevalences and average durations of colonization for serotypes Ia, III, and V GBS as well as estimates for the “general” case of colonization with any strain of GBS (including but not limited to types Ia, III, and V) were obtained from previously-collected data [16], and can be found in Table 1.

As described elsewhere [16, 20], in January and February of 2001 first-year college students living in a single dormitory at the University of Michigan were invited to participate in the study. Dormitory floors were randomly selected and an advertisement was placed in 738 student mailboxes. A total of 462 students, 216 males and 246 females, completed the enrollment protocol [16]. Participants provided informed consent, a throat and mouth (cheek) culture, self-collected initial-void urine and anal orifice and vaginal specimens, and completed a self-administered questionnaire. All participants who tested positive for group B *Streptococcus* at enrollment and a random sample of those who tested negative were invited to return for four additional visits at 3, 6, 9, and 12 weeks after enrollment. At each follow-up visit, additional specimens and questionnaire responses were collected.

Of the 462 participants in the initial survey, 299 were invited to participate in the follow-up study (149 men and 150 women), of whom 257 (86%) completed the first follow-up and 82%, 80%, and 79% completed the second, third, and fourth follow-ups respectively [16]. Follow-up rates were virtually identical by gender.

GBS was isolated using selective media containing both gentamicin and nalidixic acid, and suspect isolates were confirmed serologically using DNA dot-blot hybridization or anti-

fluorescein-AP antibody. Nontypeable isolates were probed for the presence of the GBS 16S rRNA gene to verify that chromosomal DNA was present. Incidence of GBS colonization was defined as a culture-positive specimen at any site following a negative specimen at all sites at the previous 3-week sampling interval. Average duration of GBS carriage by serotype was estimated using the serotype-specific prevalence and incidence.

In addition, data from the questionnaire were used to construct population distributions of relationship lengths, “gap” lengths (the interval, in days, between the end of one relationship and the beginning of the next), and rates of sex acts, which were used to inform the computational model.

SIRS model

We use a SIRS epidemiological model, which divides individuals into three classes: susceptible (S), infected (I), and recovered/immune (R) [21, 22]. Typically when this model is used for a sexually-transmitted disease, the rates at which individuals move between these classes are governed by three parameters: the probability per sex act that a susceptible individual will become infected (β), the rate at which infected individuals recover (γ), and the rate at which recovered/immune individuals lose their immunity (δ). Our model is slightly more complicated because we must differentiate two of our parameter estimates (β and γ) by sex. This is because women have an extra colonization site, the vaginal cavity, which results in higher GBS prevalences and longer durations of carriage [20]. We denote our sex-specific parameters by β_f for male-to-female transmission and β_m for female-to-male transmission, and γ_m and γ_f for male and female recovery rates, respectively. We assume that δ is the same for men and women, and we neglect same-sex partnerships.

Normally such a model would be solved using differential equations. However, this method is inadequate in the present case because of the broad distributions of rates of sex, partnership lengths, and gap lengths observed in our sample population. We therefore constructed a stochastic model that incorporates variation in these parameters according to the observed distributions. We use this model to estimate the values of β_m and β_f relative to δ .

Model details

Our model simulates a population of 1400 individuals (700 men and 700 women) for a total of 200,000 days. This is a much longer duration than that of the original study, but it is statistically equivalent to performing many studies one after another and averaging the results.

Each simulated individual is labeled with his/her disease state: susceptible, infected, or recovered/immune. Individuals are also labeled with their partnership status: either in a partnership or not. For those in partnerships the identity of the partner is recorded. Each new partnership is assigned a duration and a rate of sex acts drawn at random from the empirical distributions of these quantities. The partnership dissolves when its duration is exhausted and its participants are relabeled as unpartnered. Individuals not in partnerships are assigned a “gap length” drawn from the appropriate distribution and form new partnerships when that gap length is exhausted.

In detail, for each simulated day in our model, the following events occur:

1. For each susceptible individual in a partnership with an infected individual, the susceptible individual becomes infected with probability $c\beta_m$ for males or $c\beta_f$ for females, where c is the rate of sex acts in the partnership.

2. Susceptible individuals, either in partnerships or not, become spontaneously infected with a low probability λ . In our simulations $\lambda = 10^{-5}$. This spontaneous infection can be thought of as infection via some outside source, such as food or environmental contamination. A small but nonzero value of λ is necessary because random fluctuations occasionally cause the infection to die out, and if this occurs the rest of the simulation becomes useless. A low rate of spontaneous infection allows the infection to be restarted in such cases so the simulation can continue.
3. Each infected individual recovers with probability γ_m for males or γ_f for females. Each recovered individual becomes susceptible again with probability δ .
4. Partnerships that have exhausted their assigned duration dissolve and their participants reenter the pool of unpartnered individuals.
5. Individuals who have been unpartnered for a number of days equal to their assigned gap lengths become eligible for new partnerships. These are formed at random between male/female pairs of eligible individuals. Since the number of eligible males and females may not be exactly equal it is possible for some individuals to be unpaired at the end of this process, in which case they remain single until the next day, when they have another chance to find a new partner.

Parameter Search

The values of γ_m and γ_f can be calculated directly from the survey data but the values of β_m , β_f and δ are unknown. We performed simulations for a variety of values of the latter three parameters, finding for each value of δ those values of β_m and β_f that gave disease prevalences as close as possible to the observed prevalences in Table 1. The resulting parameter estimates show some statistical fluctuation because of the stochastic nature of the simulations, and the figures we report are averages over these fluctuating values.

The end result is not a pair of single values for the transmission probabilities β_m and β_f but a curve of values as a function of δ , the rate of loss of immunity. For some values of δ the corresponding value of β_m or β_f falls outside the allowed range for a probability of zero to one, and these values of δ are thus not possible.

Results

The population distributions of relationship lengths, gap lengths, and rates of sex acts are shown in Fig. 1. Gap lengths were clustered around a mean of 63.9 days (s.d. = 290.1 days). As observed in other studies [23], most individuals ending a partnership found new partners within the infectious period of most sexually-transmitted diseases, meaning that transmission from the first partner to the second was possible.

Partnership lengths had a mean of 319 days, and the rates of sex acts had a mean of 0.59 per day for individuals in partnerships. The spikes visible at one and two sex acts per day in Fig. 1 reflect the large number of single-day partnerships in which an integer number of sex acts occurred.

Values of the transmission probabilities β_m and β_f were obtained for a series of δ values for each serotype, as well as for the general case of no serotype specificity. Results are shown in Figure 2 as a function of $1/\delta$, which is the mean time for which recovered individuals remain immune. As we might expect, the longer individuals remain immune the higher the transmission probabilities needed to yield the correct male and female prevalences. Since the transmission probabilities β_f and β_m cannot be greater than one, however, our relation between β and δ also gives an upper limit on the possible values of $1/\delta$, the average duration

of immunity. For the case of colonization with any GBS serotype this upper limit is about 125 days. For serotype Ia the corresponding figure is 570 days; for serotype III it is 1125 days; and for serotype V it is 260 days.

Note that the limit is considerably higher for each separate serotype than for all serotypes combined. Which limit is most appropriate for actual GBS infections depends crucially on the level of cross-immunity between GBS serotypes. If serotypes confer complete cross-immunity to one another, so that catching and recovering from any serotype leaves an individual immune to all of them, then the calculations and figures for “all serotypes” are the most correct: in this case GBS can essentially be treated as a single monolithic pathogen and it is safe to ignore serotype distinctions. Conversely, if there is no cross-immunity at all between serotypes, so that each can be treated as an entirely separate infection, then the calculations for individual serotypes shown in Fig. 2b–d are most accurate.

In practice, the truth probably lies somewhere between these two extremes, with GBS serotypes conferring partial but not complete cross-immunity to one another. On balance, however, the situation seems to be closer to the scenario in which there is no cross-immunity. As mentioned in the introduction, separate vaccines have been necessary to combat different GBS serotypes, indicating that in practice the body develops little cross-immunity. For the purposes of determining transmission parameters, therefore, the results of the individual serotype models appear more relevant.

Discussion

The course of bacterial infection follows a familiar cycle: a susceptible individual is infected for a certain length of time, then recovers, retaining immunity to the infecting strain for some time, then loses that immunity and becomes susceptible once more. This is the essence of the SIRS model used here. Through repeated simulations of the infection process, we can find sets of values for the model parameters that match the observed course and prevalence of GBS infection. Several parameter values are already known, but two are not—the probability β per sex act of infection and the rate of loss of immunity δ among recovered individuals—and it is on these that our analysis focuses. In effect, our calculations give us a relationship between β and δ , as depicted in Fig. 2, such that if one were known we would be able to calculate the other.

Neither of these two quantities is currently known with great accuracy, but even without such information our results can still provide useful insight. The curves in Fig. 2 each contain a long horizontal portion followed by a sharp rise. (Further detail about the epidemiological reasons for the shape of the curves can be found in the Appendix.) Depending on which portion of the curve the true values of β and δ fall, either (a) β is quite small, or (b) β is larger but the duration of immunity is very close to the upper limit discussed above. Distinguishing between these two scenarios should require only a crude measurement of β or δ . If, for instance, we were to discover that the value of $1/\delta$ (the duration of immunity) were significantly less than the upper limit shown in Fig. 2, even if the measurement were approximate and placed only the loosest constraints on δ , one could still conclude that β was small—less than about 0.1.

In addition, our calculations give us an upper limit on the duration of immunity to GBS, which could have important policy implications. If the upper limit on the duration is low, it implies that natural immunity does not last very long. Assuming that natural and vaccine-derived immunity have similar lifetimes, this would imply that a vaccine would be of only modest utility in GBS prevention. A vaccine conferring short-term immunity might prevent some neonatal infections if pregnant women were inoculated late in their pregnancies or

given repeated booster shots, but it would not be a practical tool for long-term prevention of infection in nonpregnant adults.

Our results seem to indicate that the upper limit on duration of immunity to GBS ranges from about eight months to about three years, depending on serotype. At the low end, the figure of eight months for serotype V (the most common serotype in the empirical observations) is relatively short and calls into question the possible efficacy of a vaccine. Moreover, the eight month figure is only an upper limit—the actual duration of immunity could be substantially shorter. The other serotypes have higher upper limits on the duration of immunity. A duration close to the 1125 day limit calculated for serotype III, for instance, might be long enough to make vaccination a viable strategy in certain populations.

Alternatively, it is possible that all serotypes have similar periods of immunity and the variation in the limits computed in our calculation is just that—variation in the limits but not in the true duration figures. If this is the case, one can immediately draw several further conclusions. For example, if periods of immunity are roughly the same for all serotypes, none of them can exceed the eight-month limit for serotype V. Translated to the curves for other serotypes, this means the transmission probabilities β_m and β_f must be relatively small: less than about 0.08 per sex act for serotype Ia and less than about 0.03 for serotype III.

To make further progress more research is needed to determine either the duration of natural or vaccine-derived immunity to GBS or the probability per contact of infection, as the measurement of one would allow the calculation of the other from Fig. 2. As we have argued, even a crude measurement would significantly narrow the possible ranges for these parameters.

Studies of GBS's duration of immunity have been conducted in nonhuman species [24, 25] and in pregnant women with a focus on fetal immunity [4], because GBS has historically had the greatest clinical impact among neonates and is also an important fish pathogen. We are, however, aware of no estimates to date of the duration of immunity among nonpregnant adults. A single study of the antibody response to inoculation with polysaccharides from type III GBS showed that antibody responses are maximal at 2 weeks post-inoculation and remain at 21 weeks post-inoculation [26], although the study focused on only a single GBS serotype and a single type of vaccine. Nonetheless, our results are consistent with this study and underscore the fact that natural duration of immunity to GBS colonization is relatively short. Thus, a vaccine intended to protect infants would need to be administered during pregnancy, and one targeting non-pregnant adults would probably need to be administered annually.

We are aware of only one study that has attempted to estimate transmission probabilities for GBS (with the exception of vertical transmission from mother to child, for which the transmission probability is approximately 50%; however, only 1% of infants thus infected go on to develop GBS disease [27, 28]). Foxman et al. [29] followed a cohort of 78 couples in Brazil for about two months and observed only a single instance of male-to-female transmission of GBS serotype III and zero instances for any other serotype or for female-to-male transmission. Allowing for the observed serotype prevalence in the population and the rate of sexual contacts among study participants, the authors calculated an average male-to-female transmission probability of about 0.06 per sex act. With only a single instance of transmission this estimate is subject to large uncertainty; however, taking it at face value and referring to Fig. 2c (for serotype III), it implies that transmission falls on the lower arm of the curve, with small transmission probabilities and a duration of immunity of about 600 days, well below the upper limit. It is important to note that although we consider only sexual transmission of GBS, any figure for the duration of immunity inferred from measured

sexual transmission probabilities in this way should be valid for other modes of infection as well.

As is true of any mathematical model, ours has some limitations. First, the fact that we used a finite population in our model and did not allow individuals to enter or leave means that we may overestimate the probability of transmission: in a more complete model some incidence of infection would arise from outside sources. Such outside infections are represented in our model by the parameter λ , but we set this parameter to a very small value so that outside infection occurs at a negligible rate. In addition, although other modes of transmission are possible for GBS, our calculations have focused solely on sexual transmission. This may also cause us to overestimate the probability of sexual transmission, with the size of the correction depending on the relative importance of other transmission modes.

Another limitation of the present approach is that our data focus on a population of young adults, rather than on the populations at high risk for GBS disease, such as the elderly. However, we know of no estimates in the literature indicating that horizontal transmission probabilities for GBS differ significantly between high-risk individuals and young, healthy individuals. GBS colonization prevalences are similar among healthy college students (30%) [20] and the elderly (25%) [30], and transmission by intimate contact and/or food likely occurs in elderly populations as well as college students. Pathogenicity (the probability of disease given colonization) does differ by host age and immune status, but there is no *a priori* reason to expect the same of our estimates of GBS transmission probabilities and durations of immunity.

In summary, we have developed a computer model of the spread of group B *Streptococcus* based on social and epidemiological parameters derived from studies of sexual transmission in a college dormitory population. By comparing the model's GBS prevalence predictions for different parameter choices against observed prevalences of three GBS serotypes, we have derived constraints on the possible values of the parameters. In particular, we have placed upper limits on the duration of immunity for each of the serotypes studied. These limits have implications for the effectiveness of vaccination strategies if one assumes that vaccine-derived and natural immunity last for about the same length of time. To more precisely estimate the duration of immunity we need a further piece of information, the probability per contact of GBS transmission, which is currently lacking in most cases. The one published figure for transmission probability, when combined with our results, suggests that the duration of immunity for GBS serotype III is about 600 days. Better transmission estimates are required to establish the epidemiological parameters of GBS infection and determine the best vaccination strategies to prevent invasive GBS disease.

Appendix

The general shape of the curves in Figure 2 can be understood in terms of the following argument. Ignoring for the moment population variation in the behavioral parameters, the average length of time for which an individual is susceptible is $1/c\beta$, the average time for which he/she is infected is $1/\gamma$, and the average time for which he/she is recovered is $1/\delta$. Thus the average total time between successive infections is $1/c\beta + 1/\gamma + 1/\delta$ and the fraction of that time spent in the infected state is equal to the disease prevalence p , or

$$p \simeq \frac{1/\gamma}{1/c\beta + 1/\gamma + 1/\delta}. \quad (1)$$

Solving for β then gives

$$\beta = \frac{1}{c(1-p)/p\gamma - c/\delta}. \quad (2)$$

In the present case, p and γ are known constants [20]. The quantity c actually has a distribution of values over the population, but if we ignore this and approximate it by a single value then Eq. (2) gives a direct relation between β and δ : as the mean duration of immunity $1/\delta$ increases so does β . The solid curves in Fig. 2 show the form of Eq. (2) for the best-fit choice of c in each case, and the curves fit well, indicating that this intuitive explanation may be a good guide to disease behavior in this case.

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Highlights

- > We model the spread of Group B Streptococcus in a population via sexual contact.
- > We estimate GBS's duration of immunity relative to its transmission probability.
- > We place upper limits on immune duration for three pathogenic GBS serotypes.
- > Our results can help inform GBS vaccination strategies.

Figure 1. Distributions of relationship lengths, gap lengths, and rates of sexual contacts for male and female college freshmen ($n = 299$) in the study of Foxman et al. [20].

Figure 2.

Probability per sex act that a susceptible individual will become infected, for females (β_f) and males (β_m), for various values of $1/\delta$, the average duration of immunity. The individual panels show (a) the general case (no serotype specificity), (b) serotype Ia, (c) serotype III, and (d) serotype V. Open circles represent the values of β_f and filled circles the values of β_m . Error bars are standard deviations on the sample mean for twenty repetitions of the calculation.

Table 1

Observed prevalences and average durations of colonization for three pathogenic GBS serotypes and for all serotypes combined, among the population of 462 first-year college students in the study of Foxman et al. [20].

Serotype	Gender	Prevalence	Mean Dur. Colonization (days)
Ia	Female	0.0679	67.1
	Male	0.0234	21.5
III	Female	0.0374	68.5
	Male	0.0185	34.1
V	Female	0.1138	57.8
	Male	0.0685	44.6
All	Female	0.3404	95.9
	Male	0.1996	59.5