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Estimation of time gaps between sexual partners and epidemic modeling of threshold effects

Tom Andersson

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Postal address:

Mathematical Statistics
Dept. of Mathematics
Stockholm University
SE-106 91 Stockholm
Sweden

Internet:

http://www.math.su.se/matstat

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Abstract

GOAL: The time gap between sexual partners is a risk factor in the spread of sexually transmitted infections (STI). A gap that is shorter than the infectious period implies a risk of transmission. The aim of this study is both empirical, methodological and theoretical: (1) to determine covariates affecting gap lengths, (2) to evaluate gap measures, and (3) to calculate effects of different gap lengths on R_0. METHODS: A Swedish sample of partnership data is used to calculate gaps from self-estimations of partnership timing, and to test for differences in age, sex, and self-reported unfaithfulness. A stochastic epidemic model with partnership dynamics for homogenous populations is used to define an alternative gap measure and to calculate R_0 for different gap lengths and partnership durations. RESULTS: Self-reported unfaithfulness is the only significant determinant of gap length. The effect of gap lengths on R_0 is marked for gaps less than six months, but the effect is attenuated for longer partnerships. CONCLUSIONS: Gap statistics based on self-reported estimations of partnership timing is biased. It presupposes at least two partnerships and favors higher rates of partnership change. More research is needed to generalize gap measures and results to heterogenous populations.

*Postal address: Mathematical Statistics, Stockholm University, SE-106 91 Stockholm, Sweden. E-mail: toma@fc.dsv.su.se. Supervisors: Tom Britton and Fredrik Liljeros.

Contents

1 Introduction

Extensive research has been conducted on the risks of concurrency, i.e. multiple and simultaneous sexual partners, for the spread of sexually transmitted infections (STI). It is considered an important risk factor [19][24][29] and a hallmark of core groups, i.e. individuals with high rates of partnership change and high levels of sexual activity [4][22][36]. With concurrent partners, infections can spread to a greater number of individuals.

Two studies of sexual behavior point to the more general risk of "time gaps" for the spread of STIs [18][26], i.e. the time between sexual partners. In this study, we restrict the definition of "gap" to *the time between the first sexual contact with the current or most recent sexual partner and the last sexual contact with the partner that directly preceded the current or most recent sexual partner*. If the gap length is shorter than the infectious period, sexual contact without protective measures is a risk of transmission. Knowledge of the covariates affecting gap length is of value in developing and designing preventive measures against STIs.

In this paper, the concepts of "negative gap" and "concurrency" will be kept apart. Concurrency is a discrete variable, referring to the number of concurrent partners, whereas negative gap is about partnership timing, a continuous variable. A long negative gap implies a long partnership. If the infectious period is shorter than the absolute value of the negative gap, the STI may be "trapped" and die out in the same way as it may be "trapped" and die out in a long positive gap. Thus, an absolute gap length shorter than the infectious period implies a risk of transmission of a STI.

In section 2.1., we summarize the results of the two previous gap studies [18][26]. Together, they point to significant age differences in absolute gap length, with shorter absolute gaps for younger age groups. Since younger age groups also demonstrate higher incidence of STIs, the results confirm partnership timing as a risk factor. However, the studies were based on selective sampling. Only respondents who provided information on more than one sexual partner were taken into account. By excluding individuals with less sexual experience, there is the risk of overestimating the rate of partnership change in the population, and underestimating the absolute gap length.

Consequently, in section 2.2., we define an alternative gap measure based on the queueing model $M/M/\infty$ [33]: *the gap length is the difference between "the inter-arrival time" - the time between the starting points of two successive partnerships - and "the service time" - the partnership duration*. This allows us to estimate gap length at the population level by means of the rates of partnership formation and dissolution. Furthermore, self-reported estimations on partnership timing, the rate parameters can be used to calculate the basic reproduction number.

In section 2.3., with the aim of evaluating threshold effects of gap lengths, we describe a stochastic epidemic model with partnership dynamics for a homogenous population [1]. We cite a formula for the basic reproduction number R_0 that takes partnership and infection dynamics into account, i.e. rates of partnership formation and dissolution, as well as rates of infection and recovery.

In section 3, we describe the data and the methods of analysis. We apply our two different gap measures to Swedish partnership data collected in a survey almost twenty years ago (1988) [22]. The survey is still of value for methodological reasons. It is a representative survey. It allows us to test several covariates of gap length, e.g. age, sex and self-reported unfaithfulness, and to apply and compare our two gap measures.

In section 4, we present the results of the statistical analysis. We also describe and analyze gap distributions for different sub-groups of subjects. We calculate R_0 for various gap lengths, partnership durations, and rates of infection and recovery. The purpose is to evaluate the relative threshold effects of these parameters, and thereby to define hypotheses for future studies.

2 Studies and Modeling of Gaps

2.1 Two previous gap studies

Kraut-Becher et al [26] carried out the first gap study. It is based on sex partnership data collected in the 1995 National Survey of Family Growth (NSFG), a cross-sectional survey of a representative sample of 10,847 women 15 to 44 years of age in the United States. The statistical analysis was done on a smaller sample of 2,768 women. Approximately 25% of the original sample was excluded because the women were not sexually active. Another 50% of the original sample was excluded due to missing information on partnership timing.

The gap study was restricted to positive gaps, but also included statistics on concurrency. Concurrency was measured in terms of the proportions of respondents reporting an overlap the last five years. About 25% of the subjects reported concurrency at some time during the last five years. Positive gaps were measured by self-estimation of end- and starting-points of sexual contact with successive partners. Statistical analysis demonstrated significant age differences in positive gap length, with shorter gaps among younger women (15-19), about 8 months, compared to 11 and 18 months for women 20-29 and 30-44 respectively. The conclusion was that more than half of the subjects changed partner within time periods shorter than infectious periods of some STIs.

Foxman et al conducted another gap study in 2006 ([18]. The gap statistics is based on a survey of sex partners among 1194 male and female residents in Seattle between 2003-2004. The initial sample consisted of 8683 households in Seattle, but 6101 (70.2%) did not meet requirements for participation - age 18-39 and fluency in English. The final analysis was based on 1051 individuals who agreed to participate and could provide information on partnership timing and sexual activity for the last five partners.

One third of the subjects reported at least one overlap. This is about 5-10% higher than the figure in the first study. Exact figures are missing. The average length of gaps, both positive and negative, was 60.8 days (SE: 29.9), about 2 months. Positive gaps averaged 354.1 days (SE: 19.1), almost 12 months, which is roughly the same figure as in the first study. The length of overlaps averaged 801.2 days (SE: 52.3), near 27 months.

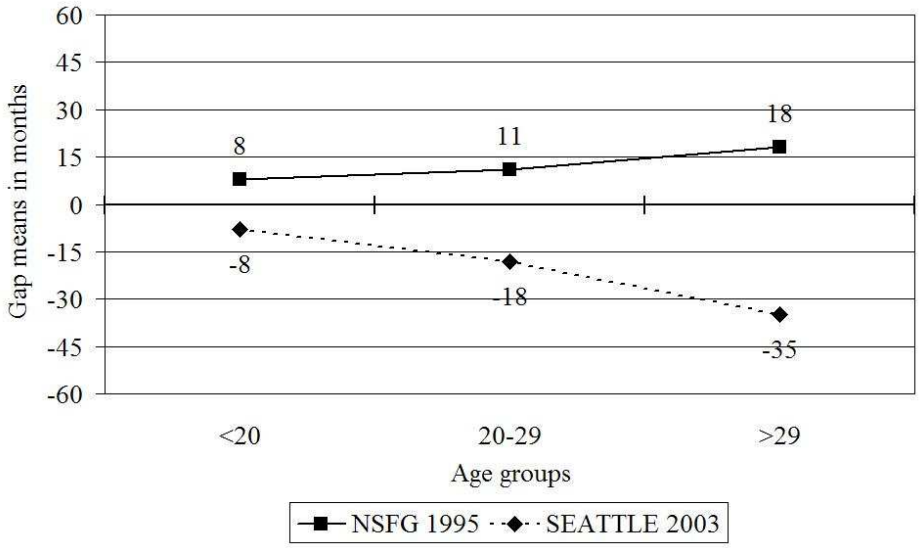
Age differences were reported for negative gaps, not for positive ones. No exact figures were given, but approximate figures can be deduced from a diagram, with shorter negative gaps among younger subjects: -250 days for the age group 18-19, -500 days for the age group 20-24, -600 days for the age group 25-29, -850 for the age group 30-34, and -1250 days for the age group 35-39.

No gap differences could be discerned for income, marital status, gender, age at first sex, and having same-sex partners. Other studies have demonstrated sex differences, men reporting more partners and overlaps [12][24][29][31]. The absence of sex differences may be due to the detailed survey questions. It forces the respondents to think carefully about their sexual partners, instead of giving general answers, for which reason the gender bias is reduced.

Furthermore, no significant changes in gap length could be discerned across partnerships over time, despite significant age differences. A possible explanation is the mean rate of partnership formation (1.81 per year). Considering that the maximum number of reported partnerships was limited to 5, this means that the estimated gaps do not stretch too far back in time, at most 5-10 years.

For the purpose of comparison with the first gap study, we have averaged the negative gap lengths for the first and second age groups, and for the fourth and fifth age groups. For the same reason, we re-scaled the negative gap length into months. In diagram 1, we render the positive gap lengths across age groups according to the first study (NSFG 1995), as well as the corresponding negative gap lengths according to the second study (SEATTLE 2003).

Diagram 1: Approximate gap means reported in previous gap studies



Together the two studies point to a gap and age trend. *The absolute gap length increases with age.* This agrees with research and studies that demonstrate higher rates of partnership change among adolescents, before people pass into stable relationships with occasional sidesteps.

2.2 Gap measures

The two previous gap studies conclude that a majority of time gaps between partnerships are shorter than the infectious periods of several STIs. The conclusion is problematic for both theoretical and methodological reasons.

First, subjects who do not, or cannot, report more than one partner were excluded from the studies. Thus, gap estimates are biased for subjects with higher rates of partnership turnover and shorter absolute gap lengths.

Second, in the second gap study, the average gap length was an average over positive and negative gaps over different numbers of partners for each respondent. This results in further bias for subjects with higher rates of partnership turnover and shorter absolute gap lengths.

Third, negative gaps are equated with overlaps (concurrency). The difference between long and short negative gaps is not taken into account. A long negative gap implies a long partnership without any recent concurrent partner. STIs may be trapped if the partnership duration is longer than the infectious period.

We cannot know if and to what extent the reported gap statistics is representative for the larger population. An important part of the problem is the gap measure - *self-reported estimations of partnership timing*. This gap measure presupposes more than one partner to report. Furthermore, it is put into practice in different ways in the two studies.

> We defined the gap to be the difference between the date of the first sex with a current or most recent partner and the date of last sex with a previous partner [26].

This gap definition was used in the first gap study. The concept of "previous partner" is not specified any further, but the most reasonable interpretation is "the partner that directly preceded the current or most recent partner". Partnership data were limited to 5 years before the interview, other than spouses and cohabiting partners for whom there was no time limit.

In the second gap study, there was no time limit on partnership data. Gaps were calculated for the last up to five sexual partnerships regardless of their occurrence in time. The gap statistics included all gap data for a respondent. This explains the more loose gap concept used in the second study.

> The length of time between the end of an individual's partnership with one sexual partner and the start of their next partnership [18].

This gap measure results in gap statistics based on 1-4 gaps (2-5 partners) for each subject, without any predefined limit to the occurrence of the partnerships in time. This raises five theoretical and methodological issues.

First, there are generational shifts in partnership dynamics. For example, during the last two decades, rates of partnership change have gone up in Sweden and Norway [17][35]. If gaps are collapsed over time, we cannot account for these changes.

Second, partnership sampling over longer time periods may result in higher sample losses among younger subjects with less sexual experience. We lack the relevant statistics to support or reject this hypothesis.

Third, the generalized gap measure gives more weight to subjects with many partners, since they have more gaps to report. This may result in a bias for shorter absolute gap lengths.

Fourth, to model the spread of STIs in populations, we must specify the prevalence of sexual behaviors and STIs at a particular time. A measure that collapses data over time is not suited as a parameter.

Fifth, the generalized gap measure introduces a potential bias in gap recall [16]. Recall is probably better for the most recent partners, whereas missing information on gaps increases with time. Thus, longer gaps may be underestimated.

The authors do not discuss these issues, but argue that the generalized gap measure strengthen the validity of gap statistics. However, to evaluate the methodological issues, we need to compare and evaluate different gap measures. Therefore, in the present study, we apply two different gap measures.

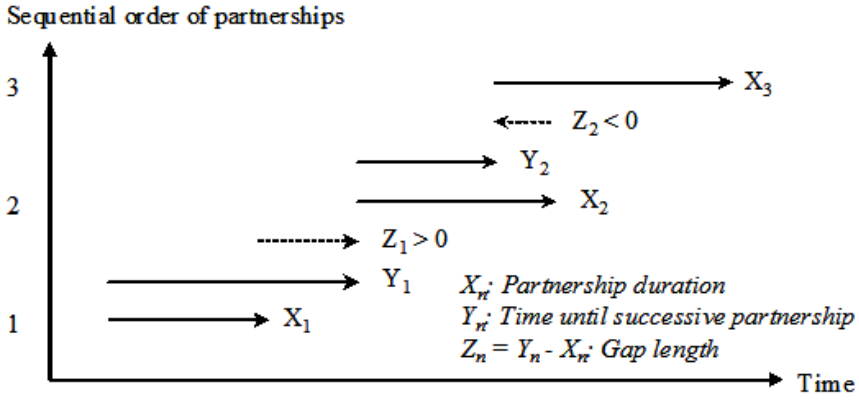
First, we use a modified version of the first gap measure: *the time in days between the date of the first sexual contact with the current or most recent sexual partner and the date of the last sexual contact with the partner that directly preceded the current or most recent sexual partner.* This is a gap measure at the individual level.

Second, we use of queueing theory to derive a representative gap measure at the population level. In the queueing model $(M/M/\infty)$[1][33], a single individual can be conceived as a partnership service station dissolving partnerships at a constant rate σ - the dissociation rate. The remaining individuals in the population are potential clients who arrive at the station at a constant rate ρ - the association rate.

The inter-arrival time, the length of time between two successive starting-points of two partnerships, is exponentially distributed with parameter ρ and expected value ρ^{-1}. The service time, the partnership duration, is exponentially distributed with parameter σ and expected value σ^{-1}.

In the queueing model $(M/M/\infty)$, *gap is a random variable Z defined by the difference between two independent random variables Y and X - the inter-arrival time for two successive partnerships and the duration of the first partnership in the succession.* In terms of partnership dynamics, the gap is the time between the end-point of partnership X and the starting-point of partnership Y directly succeeding partnership X (figure 1).

Figure 1: A queueing model of gaps (M/M/∞)



The gap distribution is derived from the distributions of its constituents, the inter-arrival time (Y) and the service time (X), with their corresponding parameters ρ and σ. The formulas for calculating expected gap value, the overall value as well as conditional values for positive and negative gaps, and gap variance, are given below.

$$Y \sim exp\,(\rho) \tag{1}$$

$$X \sim exp\,(\sigma) \tag{2}$$

$$Z = Y - X \tag{3}$$

$$f_Z(z) = \int_0^\infty f_X(x) f_Y(z+x) dx \qquad \text{if } z \geq 0 \tag{4}$$

$$f_Z(z) = \frac{\rho\,\sigma}{\rho+\sigma}\, e^{-\rho z} \qquad \text{if } z \geq 0 \tag{5}$$

$$f_Z(z) = \int_{-z}^\infty f_X(x) f_Y(z+x) dx \qquad \text{if } z < 0 \tag{6}$$

$$f_Z(z) = \frac{\rho\,\sigma}{\rho+\sigma}\, e^{\sigma z} \qquad \text{if } z < 0 \tag{7}$$

$$E(Z) = E(Y) - E(X) = \rho^{-1} - \sigma^{-1} \tag{8}$$

$$V(Z) = V(Y) + V(X) = \rho^{-2} + \sigma^{-2} \tag{9}$$

$$E(Z|Z \geq 0) = \frac{\rho\sigma}{\rho+\sigma}\rho^{-2} \tag{10}$$

$$E(|Z||Z < 0) = \frac{\rho\sigma}{\rho+\sigma}\sigma^{-2} \tag{11}$$

This gap model enables a gap measure based on the association and dissociation rates ρ and σ at the population level, in contrast to the gap measure that relies on self-reported estimation of partnership timing at the individual level.

The model predicts lower gap variance for groups of individuals with higher mean rates of partnership formation and dissolution. This agrees with age trends in partnership dynamics, i.e. higher rates of turnover and shorter absolute gap lengths among younger persons.

The model also allows us to evaluate the effects of gap length on R_0. (See Appendix A for a short introduction to the basic reproduction number). As far as we know, there is no epidemic model that explicitly defines a gap parameter, but there are models that include ρ and σ. We can calculate relative effects of gap length by varying these parameters.

2.3 Stochastic epidemic modeling

A majority of epidemic models is based on the assumption of occasional sexual contact: *an infected individual makes occasional sexual contact with susceptible individuals at a regular rate.* These models cannot account for the function of partnership in the spread of STIs [23][24][29][34][36]. (See Appendix B for a short introduction to epidemic modeling).

Some deterministic models do take partnership dynamics into account. They have mainly been formalized by means of dynamic systems - systems of differential equations - that specify rates of pair formation and dissolution, as well as rates of infection and recovery [9][11][27][28].

The dynamic system approach extends the state space of the basic version of the SIR model (appendix B). Individuals are either single or paired, which enables evaluation of relative threshold effects of partnerships compared to isolated contacts. The drawback with this approach is the mathematical complexity that does not always allow for explicit calculations of R_0 [1]. Nor is the approach well suited to deal with concurrency, except when assuming severe restrictions on the number of concurrent partners [20].

An argument has been made that the dynamic system approach does allow for unlimited concurrency: "rapidly changing monogamous interactions" [15]. However, concurrency covers different types of overlapping partnerships, including concurrency with one steady and one causal partner, which cannot be equated with high rates of partnership formation and dissolution.

Altmann [1] proposes a different approach to epidemic modeling with partnership dynamics. He extends the SIR-model to include stochastic partnerships, i.e. partnerships as independent stochastic processes, thereby allowing for concurrency without predefined limits of the number of partners. The main idea is to track states of dyads, pairs of individuals.

A dyad is in one of two states, single or united. Furthermore, each individual in the dyad is in one of three disease states: susceptible, infected or recovered. Thus, there are 18 states in this dyadic model: 2x3x3. The basic version of the model assumes a homogenous population and Markov transitions between the states. Thus, the transitions are regulated by the following assumptions.

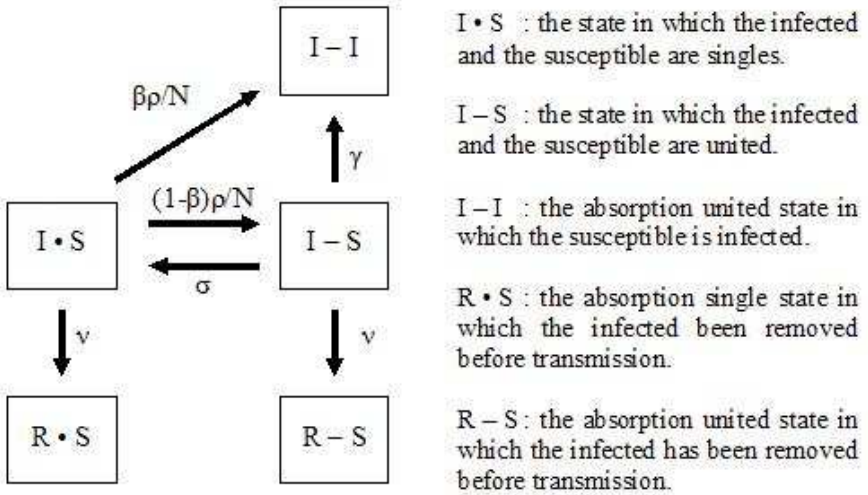
1. Constant rate of association (per individual): ρ/N
2. Constant rate of dissociation (per partnership): σ
3. Constant rate of infection from infectious partner: γ
4. Constant rate of recovery or removal from infection: ν

The parameter ρ is scaled by population size N to keep the total rate of association constant over different population sizes.

The model can be elaborated for heterogenous populations and/or generalized to semi- or non-Markov processes, but the Markov model is sufficient for our purposes. It accounts for partnership dynamics and keeps the mathematical formula for calculating R_0 simple and explicit. The derivation of R_0 that follows is a short summary of the derivation in Altmann [1].

We assume a homogenous and completely susceptible population of size $N+1$. We also assume that the stochastic process of partnership formation and dissolution has equilibrated when the infection is introduced for a random individual. The infected exists in N dyads. To calculate the probability of transmission in a dyad, we only need to deal with the reduced state space in figure 2.

Figure 2: The state space for calculating R_0 in a stochastic epidemic model with partnership dynamics *(Altmann, 1995:666)*



The state of the infected is given. This reduces the original state space of the Markov model from 18 to 12 states: 2 x 2 x 3. Furthermore, the states $I-I$, $R \cdot S$, and $R-S$ are absorption states. Thus, seven states that are dependent on these absorption states are excluded: $I \cdot I$, $R-I$, $I-R$, $R \cdot I$, $I \cdot R$, $R \cdot R$, and $R-R$. The five states in figure 2 remain.

When a dyad with the infected makes a transition from the single to the united state, there is a probability β that the infection gets transmitted at first contact. Thereafter, if not transmitted, the infection is transmitted at a constant rate γ when the infected and the susceptible are united in partnership.

To calculate the probability of transmission for a dyad, we calculate the probability of absorption into state $I-I$ as opposed to $R\cdot S$ or $R-S$. This is done with standard methods for Markov processes.

A dyad begins either in state $I\cdot S$ or $I-S$. Let $P_0(I\cdot S)$ denote the probability that the dyad starts in $I\cdot S$. Let $P_0(I-S)$ denote the probability that the dyad starts in $I-S$,

$$P_0(I\cdot S) = \frac{N\sigma}{\rho+N\sigma}, \tag{12}$$

$$P_0(I-S) = \frac{\rho}{\rho+N\sigma}. \tag{13}$$

Furthermore, let $P(I-I|I\cdot S)$ denote the probability of absorption into state $I-I$, starting from state $I\cdot S$. Let $P(I-I|I-S)$ denote the probability of absorption into state $I-I$, starting from state $I-S$. $P(I-I|I\cdot S)$ and $P(I-I|I-S)$ then satisfy the following system of equations,

$$\beta\rho/N+(1-\beta)\rho/NP(I-I|I-S) = (\rho/N+\nu)P(I-I|I\cdot S), \tag{14}$$

$$(\nu+\sigma+\gamma)P(I-I|I-S) = \gamma+\sigma P(I-I|I\cdot S). \tag{15}$$

Together with initial state probabilities, this system gives us the probability of transmission for a dyad.

The stochastic and independent nature of partnership processes implies that the secondary cases in a large population are unlikely to be partners. Thus, we calculate R_0 by summing up the probabilities of absorption over all dyads involving the infected. For large N, the equation [1] converges to

$$\lim_{N\to\infty} R_0 = \frac{\rho\gamma}{\sigma(\nu+\sigma+\gamma)}+\frac{\beta\rho}{\nu}+\frac{\rho\gamma(1-\beta)}{(\nu+\sigma+\gamma)\nu} \tag{16}$$

Later on, we will make use of this formula for calculating the basic reproduction number for various gap lengths by means of ρ and σ - the rates of association and dissolution - as well as for different values of γ and ν - the rates of infection and removal/recovery.

3 Data and Methods

3.1 The Gotland data

To address the theoretical and methodological issues raised in this paper, we will apply our gap measures to Swedish partnership data collected in a survey conducted on Gotland in 1988 [22]. Gotland is an island in the Baltic sea. At present, it has 57,500 inhabitants. Its demographics is similar to the general Swedish population.

The respondents were selected on the basis of a random sample of 1150 local residents in the age group 16-31. They were invited to come to schools and other public places on specified dates and times to answer a self-administered questionnaire. 779 individuals (70%), 432 women and 347 men, completed it.

One part of the questionnaire consisted of simple and direct questions regarding the subject's sexual experience and general background: Have you ever had intercourse? How old were you at your first intercourse? How old was your partner? How old were you at first intercourse with your second partner? How many different persons have you had intercourse with during your life? Have you ever been unfaithful to a partner? Age and sex?

Another part consisted of graphical representation of the subject's sexual contacts in the preceding 12 months. The subject was given a graph showing the months from July 1987 to June 1988. He or she was then asked to mark any causal contact with an 'X' on the time line, and each longer, stable relationship with a continuous horizontal line. For each partner, the subject was asked to give information on the partner's age, number of occasions in which they had had sexual intercourse, and whether a condom had been used.

General statistics from this study has already been published [22]. The authors reported results that agree with general findings. Men tend to report more sexual experience, more partners and sexual activity, although age at first sex is generally lower for women than for men. The authors also reported longer and increasingly stable partnerships with age, both for men and women. High-risk behavior, i.e. several partners in parallel or in quick succession, only occurred in about 10% of the subjects. The authors concluded that a rather small group of people have sexual contacts that put them at risk of acquiring or transmitting STIs, supporting the notion of a core-group.

In the previous gap studies, a majority of respondents were estimated at risk. Different sampling techniques and questionnaire design explain the discrepancy. No one was excluded in the Gotland study due to lack of sexual experience. Furthermore, the gap measures and estimates interact with the different time windows.

The time window for reporting sexual partners was smallest in the Gotland study, one year, moderate in the first American study, five years, and unlimited in the second American study. With a larger time window, a larger proportion of respondents will report concurrent partners at some point in time. The proportion of subjects who report at least one case of concurrency increases from 10% in the Gotland study, to 25% and 33% in the two American studies.

3.2 Methods of Analysis

The first study of the Gotland data was mainly descriptive in kind [22]. In this paper, we will present a more detailed analysis of the partnership data within the one-year time window - July 1987 to June 1988. For this one year time window, we have information on end- and starting points of partners and partnerships.

We will begin by presenting partnership and gap statistics based on self-reported estimations of partnership timing: the mean number of partners and the average gap length. Due to the restricted one-year time window, which cannot account for absolute gaps > 12 months, the average empirical gap length ought to be shorter than the gap estimates in the previous studies.

We then test for differences in age, sex and self-reported unfaithfulness with a three-way ANOVA. For this purpose, we have divided the subjects into three age groups (16 to 20, 21 to 25, and 26 to 31). Furthermore, we make a regression analysis of gap length on several other variables to evaluate the best predictors: age (continuous), sex (dichotomous), age of first sex (continuous), age difference between subject and previous partner (continuous), and number of sex acts with previous partner (continuous).

After the statistical analysis, we apply the Markov gap measure at the population level and derive gap estimates on the basis of monthly rates of association and dissociation. We compare the results of the two gap measures, as well as with the results from the two previous gap studies. Considering that the Markov gap measure is calculated at the population level, not excluding subjects with less sexual experience, it ought to result in longer gap estimates, as well as greater variance, than any gap measure based on self-reported estimation of partnership timing.

Markov gap estimates are thereafter calculated separately for two subpopulations, i.e. subjects who reported being unfaithful and faithful respectively. This division reflects core and non-core group behavior. First, unfaithfulness implies concurrency, multiple partners. This is a hallmark of core groups [6]. Second, unfaithfulness implies nondisclosure of information to sexual partners [14], which is also a central feature of core group behavior, where multiple sexual contacts are ignorant of each other. Together, concurrency and non-disclosure of information neutralize the regulating function of partnerships in the spread of STI.

By splitting the population into unfaithful and faithful, we control for core and non-core behavior at the population level. Considering that they represent different levels of sexual activity, the groups ought to demonstrate shorter and longer absolute gaps respectively.

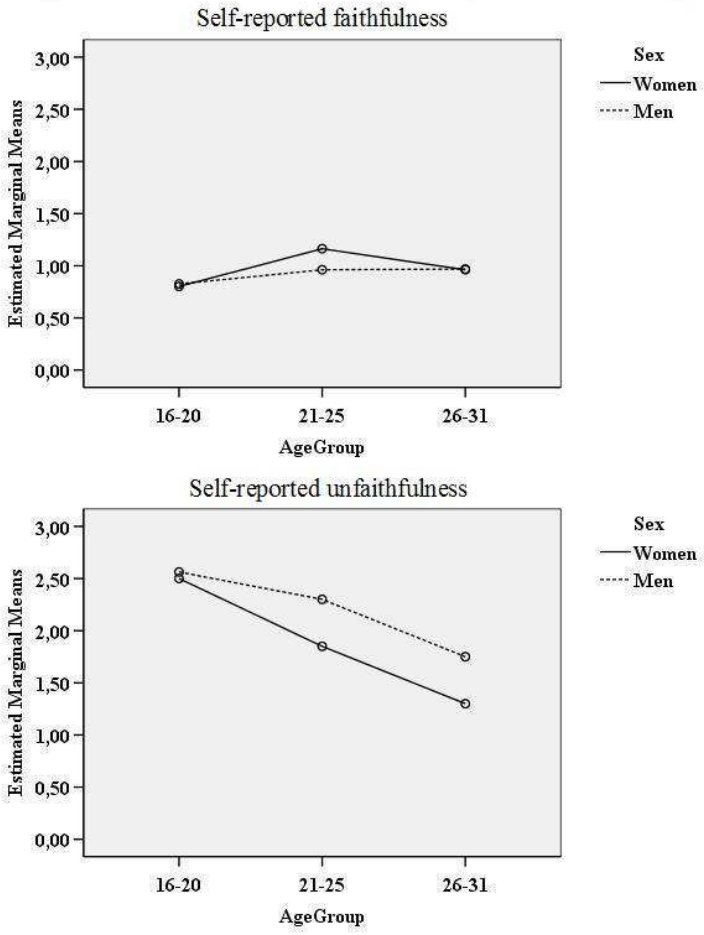
Finally, we use the Markov formula for R_0 to evaluate relative threshold effects of various gap lengths and partnership durations at the population level. We calculate and compare R_0 for the core and the non-core group respectively. We simulate and plot R_0 for (1) continuous variations of gap length, (2) discrete partnership durations (1, 6, and 36 months), and (3) two different types of infections, (F) fast infection with high transmission and recovery rates and (S) slow infection with low transmission and recovery rates [15][19].

4 Results

4.1 Gotland partnership and gap statistics

The mean number of sexual partners during the one-year time window is 1.12 (SD: 1.16); median 1.00. A three-way ANOVA of the number of partners ($R^2 = 0.144$) demonstrates main effects of age ($F(2, 761) = 11.68$, $p = 0.001$) and unfaithfulness ($F(1, 761) = 111.03$, $p < 0.001$), as well as an interaction effect of these factors ($F(2, 761) = 10.78$, $p < 0.001$) (cf. Diagram 2). The effect of unfaithfulness (partial $\eta^2 = 0.127$) is stronger than the effect of age (partial $\eta^2 = 0.018$). There is no main effect of sex.

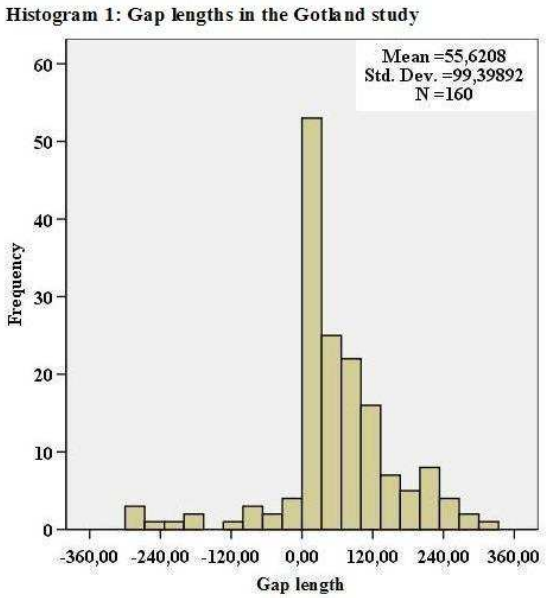
Diagram 2: No. partners per year by age, sex and faithfulness



The mean number of partners for subjects with self-reported unfaithfulness and faithfulness are 1.87 and 0.93 respectively (SD: 1.66 and 0.90). The mean number for the age groups are 1.03, 1.25, and 1.10 (SD: 1.43, 1.19, and 0.73).

The mean age of subjects with self-reported unfaithfulness and faithfulness are 24.57 and 22.61 respectively (SD: 4.60 and 5.00). The difference is significant ($t(772) = 4.36$, equal variances assumed, $p < 0.001$).

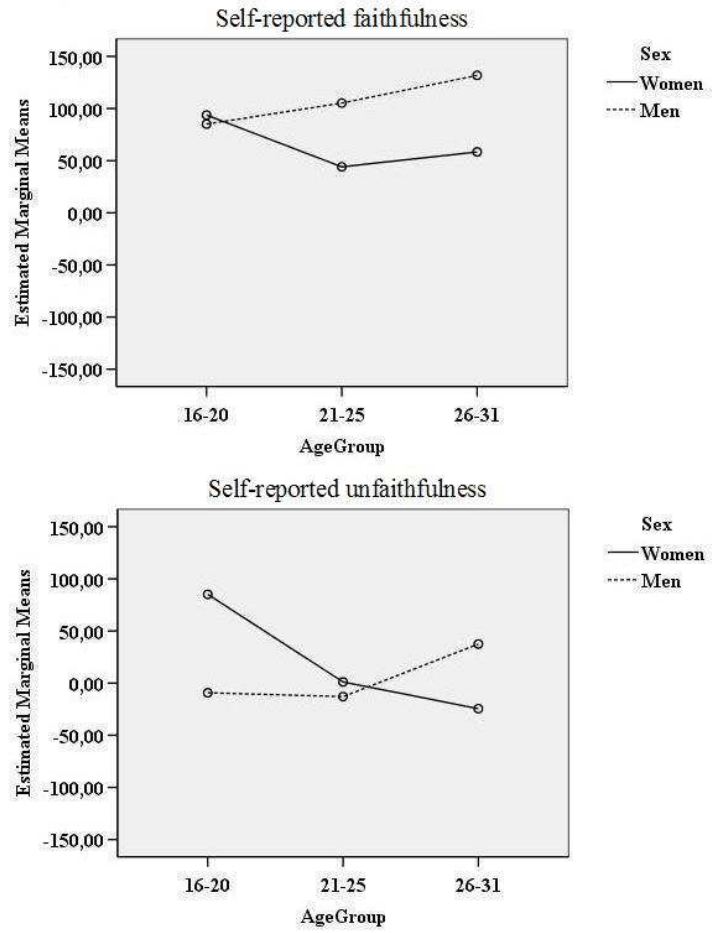
160 subjects (20.5%) report sexual contacts with two or more persons. The mean value of the most recent gap is 55.62 days (SD: 99.40); median 44.15 (cf. histogram 1). 19 gaps (11.9%) are negative. The average positive and negative gaps were 78.80 and -116.40 days (SD: 6.09 and 24.21).



A three-way ANOVA of the gap length ($R^2 = 0.244$) demonstrates a main effect of self-reported unfaithfulness ($F(2, 145) = 19.50$, $p < 0.001$). The mean values of gaps for subjects with self-reported faithfulness and unfaithfulness are 85.51 and 11.60 days respectively (SD: 74.49 and 115.66). The ANOVA cannot confirm main effects of age group or sex, but a weak interaction between these factors, with longer gaps for men and shorter for women ($F(2, 145) = 4.75$, $p \approx 0.01$, $\eta^2 = 0.061$) (diagram 3 on next page).

Linear regression analysis of gap length confirms unfaithfulness to be the only significant predictor among several variables: age (continuous), sex (dichotomous), age of first sex (continuous), age difference between subject and previous partner (continuous), and number of sex acts with previous partner (continuous). Linear regression analysis of gap length for each variable demonstrated significant effects of unfaithfulness and age, with shorter gaps for unfaithfulness and with age. When tested together, as well as pair-wise and step-wise with the remaining variables, only unfaithfulness proved to be a robust predictor for a linear regression model ($F(1, 156) = 24.16, p < 0.001, t = -4.916, p < 0.001$, standardized $\beta = -0.366$).

Diagram 3: Mean gaps by age, sex and faithfulness



4.2 Markov estimates and distributions

Tables 1-3 on the following page present the calculations of the Markov estimate of gap length for the whole sample (Table 1), the core group (Table 2), and the non-core group (the whole sample excluding the core group) (Table 3).

Monthly rates of partnership association (ρ) were calculated on the basis of the number of new partnership per month and subject. Monthly rates of partnership dissociation (σ) were calculated on the basis of the proportion of dissolved partnerships per month.

Table 1: Gap estimation based on monthly partnership changes for all subjects

# 779	# Pair-formation	ρ	ρ^{-1}	# Partnership	# Separation	σ	σ^{-1}	gap = ρ^{-1}-σ^{-1}
January	87	0,112	8,954	464	43	0,093	10,791	-1,837
February	56	0,072	13,911	477	37	0,078	12,892	1,019
Mars	39	0,050	19,974	479	46	0,096	10,413	9,561
April	37	0,047	21,054	470	33	0,070	14,242	6,812
May	49	0,063	15,898	486	46	0,095	10,565	5,333
June	45	0,058	17,311	485	46	0,095	10,543	6,768
July	28	0,036	27,821	467	25	0,054	18,680	9,141
August	54	0,069	14,426	496	44	0,089	11,273	3,153
September	42	0,054	18,548	494	43	0,087	11,488	7,059
October	67	0,086	11,627	518	67	0,129	7,731	3,896
November	62	0,080	12,565	513	63	0,123	8,143	4,422
December	63	0,081	12,365	513	53	0,103	9,679	2,686
Mean		***0,067***	***16,204***			***0,093***	***11,370***	***4,834***
SD		*0,021*	*5,130*			*0,021*	*2,910*	*3,321*

Table 2: Gap estimation based on monthly partnership changes for core group

# 149	# Pair-formation	ρ	ρ^{-1}	# Partnership	# Separation	σ	σ^{-1}	gap = ρ^{-1}-σ^{-1}
January	31	0,208	4,806	125	14	0,112	8,929	-4,122
February	27	0,181	5,519	138	19	0,138	7,263	-1,745
Mars	14	0,094	10,643	133	22	0,165	6,045	4,597
April	18	0,121	8,278	129	20	0,155	6,450	1,828
May	21	0,141	7,095	130	19	0,146	6,842	0,253
June	20	0,134	7,450	131	19	0,145	6,895	0,555
July	10	0,067	14,900	122	10	0,082	12,200	2,700
August	20	0,134	7,450	132	19	0,144	6,947	0,503
September	21	0,141	7,095	134	26	0,194	5,154	1,941
October	32	0,215	4,656	140	27	0,193	5,185	-0,529
November	26	0,174	5,731	139	26	0,187	5,346	0,385
December	25	0,168	5,960	138	24	0,174	5,750	0,210
Mean		***0,148***	***7,465***			***0,153***	***6,917***	***0,548***
SD		*0,044*	*2,866*			*0,033*	*1,977*	*2,196*

Table 3: Gap estimation based on monthly partnership changes for non-core group

# 626	# Pair-formation	ρ	ρ^{-1}	# Partnership	# Separation	σ	σ^{-1}	gap = ρ^{-1}-σ^{-1}
January	55	0,088	11,382	337	28	0,083	12,036	-0,654
February	28	0,045	22,357	337	16	0,047	21,063	1,295
Mars	23	0,037	27,217	344	23	0,067	14,957	12,261
April	19	0,030	32,947	340	13	0,038	26,154	6,794
May	27	0,043	23,185	354	25	0,071	14,160	9,025
June	25	0,040	25,040	354	27	0,076	13,111	11,929
July	17	0,027	36,824	344	14	0,041	24,571	12,252
August	34	0,054	18,412	364	25	0,069	14,560	3,852
September	20	0,032	31,300	359	16	0,045	22,438	8,863
October	35	0,056	17,886	378	40	0,106	9,450	8,436
November	36	0,058	17,389	374	37	0,099	10,108	7,281
December	37	0,059	16,919	374	28	0,075	13,357	3,562
Mean		***0,047***	***23,405***			***0,068***	***16,330***	***7,074***
SD		*0,017*	*7,568*			*0,022*	*5,701*	*4,286*

The monthly rates of association and dissociation were used to calculate monthly inter-arrival times and service times. In turn, these served to estimate the average monthly gap length (8).

The Markov gap estimate for all subjects is 4.83 months, or 145 days. The empirical gap mean in the previous section was 55.6 days. The divergence is to be expected considering the one-year time-window. The discrepancy should be less for subjects with self-reported unfaithfulness, since core behavior means shorter absolute gaps, and larger for self-reported faithfulness, since non-core behavior means longer absolute gaps.

For the core group, the Markov gap estimate is 0.548 months (SD: 2.20 months), which is more in line with the empirical gap estimate of 11.60 days. The largest divergence is found for subjects with self-reported faithfulness: 212.2 days (7.07 months, SD: 4.29 months)and 85.5 days respectively.

On the next page, we show plots of the theoretical gap distributions (5)(7) for the core and the non-core group. The main difference is the tighter distribution for the core group, less variance, which is a consequence of more frequent changes and shorter duration. Previous empirical gap studies identify this gap pattern as more typical for younger age groups, whereas the gap distribution for the non-core group is more typical for older age groups.

The expected positive gap $E(Z|Z \geq 0)$ (10) is 3.4 months for the core group and 12.6 months for non-core group. The expected negative gap $E(Z|Z < 0)$ (11) is 3.2 months for the core group and 5.9 months for non-core group.

For a M/M/∞-system, the stationary distribution of the number of customers is Poisson distributed with parameter ρ/σ. Applied to our data, the expected number of partners is 0.72 for the whole sample, 0.96 for the core group, and 0.68 for the non-core group. The Poisson estimate can be compared with the proportion of time that the subjects find themselves engaged in a partnership. Since the one year time window is a more representative time frame for the core group, we will only make the comparison for this group.

The average duration of the first noted partnership is 238.4 days for the core group (SD: 13.0). The average gap length is 11.2 days. Thus, the proportion of time in partnership is 0.955, which agrees well with the Poisson estimate0.96. Taken together, the short gap length and the short partnership duration for the core group mean partnership changes well within the infectious periods of several STIs.

Diagram 4: Theoretical gap distribution with ρ = 0.148 and σ = 0.154 (core group)

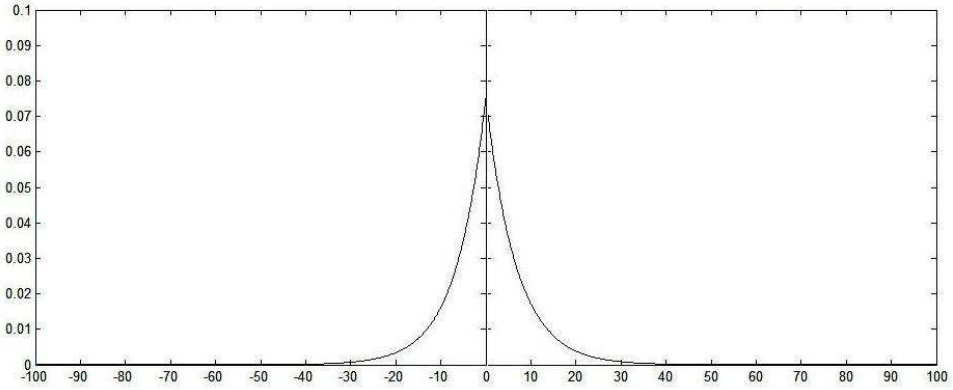
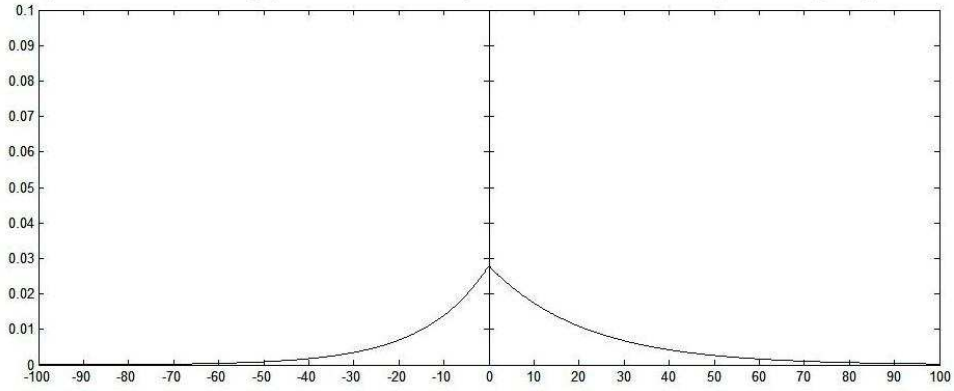


Diagram 5: Theoretical gap distribution with ρ = 0.047 and σ = 0.069 (non-core group)



4.3 Epidemic threshold effects

To calculate R_0 based on Altmann's model (16), five parameters need to be specified, besides the rates of partnership association and dissociation, (1) the probability of transmission at first contact (β), (2) the rate of transmission with a partnership (γ), and (3) the rate of recovery (removal) from infection (ν). We will use two classes of fictive but realistic parameter values that correspond to two types of infections: (1) fast infection (F) - high transmission and recovery rates - and (2) slow infection (S) - low transmission and recovery rates.

1. $\beta = 0.25$, $\gamma = 1.5$, and $\nu = 0.15$
2. $\beta = 0.01$, $\gamma = 0.1$, and $\nu = 0.025$

With our previous estimates of ρ and σ for the core and non-core groups, we get the following result (superscript F = fast infection and S = slow infection),

$R_0^F = 0.878$ for the non-core group,

$R_0^F = 1.661$ for the core group,

$R_0^S = 1.329$ for the non-core group,

$R_0^S = 2.504$ for the core group.

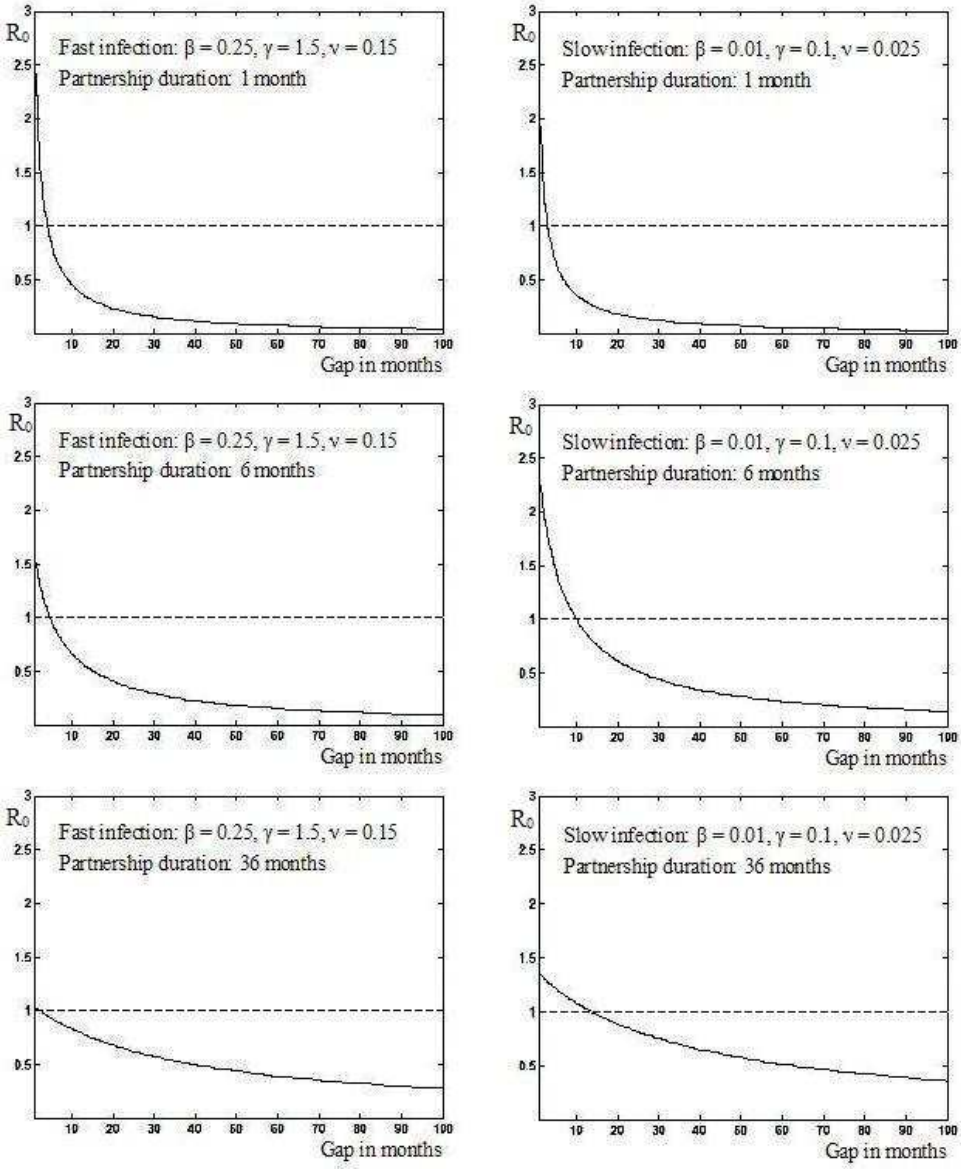
R_0 is almost doubled for the core group, irrespectively of the type of disease.

Our Markov gap estimates are based on a sample of young subjects. Adults manifest lower rates. Therefore, we calculated R_0^F and R_0^S for $\rho = 0.014$ and $\sigma = 0.017$, corresponding to partnership durations of 58.8 months and gaps of 12.6 months. This results in $R_0^F = 0.827$ and $R_0^S = 0.976$. Thus, prolonging partnership duration by several years and doubling the gap length do not radically effect R_0.

To make a more systematic evaluation of the threshold effects of gap length, R_0 was plotted against gap length, for three partnership durations (1, 6, and 36 months) and for fast and slow infections. The plots are given in figure 3 on the next page.

For the fast infection, the critical gap length for which $R_0 < 1$ is rather stable across partnership durations, about 5 months or less. For slow infections, the critical gap length changes to a larger extent across partnership durations, 5-15 months. The plots illustrate that changes in the threshold effect of gaps are larger for small gaps, less than 10 months, and for partnerships of short duration, six months and less.

Figure 3: Plots of R_0 against positive gap length for three partnership durations (1, 6, and 36 months) and two types of infection (fast and slow)



5 Conclusions

The statistical analysis of the Gotland gap data demonstrates that core and non-core behavior is a stronger determinant of gap length than age and sex. The risk of core group behavior is also marked compared to non-core behavior. The Markov gap measure results in an average gap length of 0.55 months and 7.07 months for the non-core group. The expected positive gap length is 3.4 months for the core group and 12.6 for the non-core group. The calculations of R_0 indicate critical gap lengths of approximately 5-10 months.

We could not confirm a significant linear age effect on gap length. There are several explanations for this. First, the age span of the respondents was tighter and lower in the Gotland study, 16-31 compared to 15-44 and 18-39 in the American studies. The tighter span makes age differences less prominent. Another explanation is the one-year time window.

A one-year time window cannot account for absolute gap lengths > 12 months. According to the previous studies, the average absolute gap length for adults lies above that. In fact, the one-year time window is biased for younger subjects. The mean age is 21.54 (SD: 4.36) for subjects reporting at least two sexual partners, and 23.36 (SD: 5.06) for subjects reporting at most one sexual partner, which is a significant difference ($t(281) = 4.561$, equal variances not assumed, $p < 0.001$).

Only 11.9% of the gaps measured by self-estimation of partnership timing were negative in the Gotland study, compared to one third of the subjects in the most recent American study. The reason for the discrepancy is unclear. One explanation could be the age differences, i.e. the younger and tighter age span in the Gotland study. Perhaps the proportion of negative gaps increases with age. Another explanation could be cultural differences in sexual behavior.

Selective sampling for subjects with higher levels of sexual activity results in gap statistics that is not representative for the general population. With self-reported estimations of partnership timing, which excludes subjects with less sexual experience, we get an average gap length of almost two months in the Gotland study, whereas the Markov gap estimate predicts almost five months at the population level.

More research is needed to evaluate gap measures and estimates. We need systematic comparisons and statistical analysis of the effects of using selective sampling and different time-windows. Furthermore, we need to be more clear about the concepts in use, in particular measures including "negative gap". In this study, "negative gap" denotes the timing of partnerships, whereas previous studies equate negative gaps with concurrency and overlaps.

For modeling purposes, an independent gap parameter is of limited value. In this study, it is derived from the general partnership dynamics, i.e. as-

sociation and dissociation rates. Furthermore, its epidemic effect interacts with partnership duration. Considering the problems of empirical gap measures and estimation, it would then seem more reasonable to define the gap parameter in terms of general partnership dynamics.

The epidemic function of gaps is more complex than its relation to the infectious period. Gaps of equal size are not always equal in function. If monogamous partnerships are long enough, STI with an infectious period many times shorter than partnership duration will have little chance to be passed on, irrespectively of the time gap between current and next partnership. This supports our initial discussion of negative time gaps. A long negative gap implies a long partnership and less risk of transmission since the STI is trapped in the partnership.

Consequently, negative gaps should not be equated with concurrency as a risk factor in the spread of STIs. Concurrency refers to the number of partners at a particular moment, wheras time gaps refer to the timing of partnerships, i.e. the time distance between starting- and end-points of partnerships. Thus, the relevant epidemic risk factor may be the absolute gap length (time distance). Everything else equal, longer absolute gaps imply lower risks of transmission; shorter absolute gaps, higher risks of transmission. This is a hypothesis for future research.

More research is needed to validate or refute a queueing model of gaps and corresponding stochastic models. For example, we should consider and evaluate different distributions than the exponential one [30]. More research is also needed to understand and explain the partnership dynamics and the threshold effects in heterogenous populations and over time [13]. In this study, we have kept the analysis of core and non-core groups apart. A more realistic and relevant modeling must take their interaction into account.

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A The basic reproduction number R_0

The phrasing varies - the reproductive ratio or the reproductive rate - but the meaning is the same. The reproduction number is a measure of the potential of an infection to reproduce and spread itself in a population.

> The reproductive number is the ratio of "births" of new infections to the "deaths" of old infections [19].

If $R > 1$, the epidemic tends to increase. If $R < 1$, it tends to decrease. $R = 1$ is a point of equilibrium. However, R is not constant during an epidemic. It is greater than 1 when the epidemic starts to grow, and less than 1 when the spread abates [19].

The basic reproduction number R_0 restricts the general concept to the initiation phase of epidemics. It is a measure of the take-off potential of an infectious disease, a key parameter in epidemic modeling. The following definition holds for homogenous populations.

> R_0 is the average number of individuals directly infected by an infectious case during his or her entire infectious period, when he or she enters a totally susceptible population [21].

There are several ways to formalize the parameter depending on the model. The following equation is a starting-point for epidemic models in continuous time: $R_0 = \gamma/\nu$.

γ is the infection rate, the number of new infectious contacts per unit time. ν is the rate of recovery from infection. The equation is derived from the SIR model (appendix B).

B Epidemic models

There is a variety of epidemic models [2][3][5][10]. They can be divided into different kinds depending on their formal and mathematical properties [20]. Here is a list of the main distinctions.

> *Compartmental* versus *distributional* models refer to the progression of the STI, whether the infection state is stable (compartmental) or change in some way throughout the infectious period (distributional).
>
> *Discrete* versus *continuous* models refer to the transition time between states, whether the time is discrete or continuous.
>
> *Deterministic* versus *stochastic* models refer to the type of transition between states, whether transitions are fully determined or occur at random in the population.
>
> *Group* versus *individual* models refer to the level of analysis. Epidemic modeling often involves homogenous groups at some level, whereas simulations are usually based on networks of individuals.

Besides these formal properties, epidemic models take various forms depending on the determinants of concern [25]. For example, the SIR-model - a compartmental model - includes a removed state (R) that is relevant for some STIs (Chlamydia), but not others (HIV). Other compartmental models include other states and/or transitions.

> SIS: Susceptible → Infectious → Susceptible
>
> SEIR: Susceptible → Exposed → Infectious → Removed
>
> MSIR: Immune → Susceptible → Infectious → Removed

The classic SIR-model, the Kermack-McKendrick model, involves three infection states: Susceptible (S), Infected (I), and Recovered (R). Individuals pass from being susceptible to infected, and than from infected to recovered, or removed. The transitions between the states are governed by a set of differential equations and boundary conditions.

$$\frac{dS}{dt} = -\gamma IS \tag{17}$$

$$\frac{dI}{dt} = \gamma IS - \nu I \tag{18}$$

$$\frac{dR}{dt} = \nu I \tag{19}$$

$$N = S(t) + I(t) + R(t) \tag{20}$$

$$R_0 = \gamma/\nu \tag{21}$$

N is the constant number of individuals in the population. $S(t)$, $I(t)$ and $R(t)$ are the numbers of susceptibles, infected, and recovered at time t. γ and ν are transition rates between the infection states. γ is the transition rate from susceptible to infected per susceptible and per infected. ν is the rate of transition from infected to recovered.

The SIR model rests on the following set of assumptions: (1) fixed population size, i.e. no births or deaths; (2) homogeneous population with no social structures or relations; (3) constant rate of infection; (4) constant rate of recovery; (5) instantaneous infection after contact; (6) and independent infection and recovery rates. Assumptions of this kind are necessary to reduce real-world complexity and enable modeling in the early stages, but they must be continuously refined to develop models that make better approximations to real processes. This has also been done with the SIR model. Several of the assumptions and parameters have been revised and refined, not least the infection rate.

In its most simple form, the infection rate is a summary of both human sexual behavior, the contact rate, and the infectivity of the disease, the transmission rate. However, there are good reasons for keeping these parameters apart. Due to evolutionary pressures of the immune system, there is a trade-off between the infectivity of the disease and its duration. Highly infectious organisms are short-lived. They mobilize the immune system. The reaction is fast. Less infectious organisms do not elicit extensive reactions and may therefore survive for longer time [3][19]. Consequently, transmission and recovery rates are not independent, but outcomes of interactions between the immune system and the nature of the infectious organism.

Higher transmission rates imply higher recovery rates. Lower transmission rates imply lower recovery rates. For this reason, more elaborated epidemic models of STIs usually split the infection rate γ into two parameters: the contact rate β and the transmission rate ν, to distinguish behavioral and biological factors in the spread of STIs,

$$R_0 = \alpha\beta/\nu. \tag{22}$$

α is the probability of transmission per sexual contact, and β is the sexual contact rate, i.e. the number of new sexual contacts per unit time per infected individual. The contact rate is a behavioral measure, whereas the transmission probability is a measure of the biology of infection. This split of the infection rate parameter into two, the contact rate and the probability of transmission, is an example of how epidemic models are elaborated in response to empirical considerations. The inclusion of partnership formation and dissociation - partnership dynamics - is another example.