Herpes Simplex Virus Type 2 Shedding in Human Immunodeficiency Virus–Negative Men Who Have Sex with Men: Frequency, Patterns, and Risk Factors

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We investigated the frequency, site, and risk factors for herpes simplex virus (HSV) shedding in 30 healthy men who have sex with men. Subjects collected daily HSV culture samples from genital, perianal, and oral areas for 100 days and maintained diaries of signs and symptoms. Sixteen men (53.3%) shed HSV-2, and 9 (56.3%) of 16 men who were also HSV type 1 (HSV-1) shed HSV-1. Overall, HSV-2 was isolated on 3.1% of the days; 68% of the isolations were on days that lesions did not occur. HSV-2 shedding was predominantly perianal (83.3%), HSV-1 was isolated on 2.1% of the days; 23 of 24 HSV-1 isolates were from oral areas. Rates of perianal or genital shedding were 6.6% on the days that participants reported prodromal symptoms and 1.9% on the days that participants did not report prodromal symptoms (P < .001). Men seropositive for both HSV-1 and HSV-2 were significantly more likely to shed HSV-2 (odds ratio, 4.1; 95% confidence interval, 1.4–11.9) than were HSV-2–seropositive men.

HSV–2–seropositive men who have sex with men have frequent subclinical HSV-2 shedding, usually from the perianal area, and more frequent prodromal HSV-2 shedding.

A recent population-based seroprevalence study estimated that 22% of the adult population in the United States is infected with herpes simplex virus type 2 (HSV-2) [1]. Both sexual behavior and a variety of demographic factors appear to influence the prevalence of HSV-2 infection. Studies of men who have sex with men (MSM) that were performed during the late 1980s and mid-1990s indicated that the prevalence of HSV-2 infection among HIV-negative MSM varied from 26% [2] to 40% [3]. HSV-2 seroprevalence is considerably higher, up to 70%, among HIV-infected MSM [4, 5], whereas up to 95% of HIV-positive persons have antibodies to HSV type 1 (HSV-1), HSV-2, or both [6]. HSV-2 is the predominant cause of genital ulcer disease in the United States and has been linked to acquisition of HIV infection [5–9]. This epidemiological association has been strengthened by recent demonstration of high HIV RNA copy numbers in herpes lesions during symptomatic herpes recurrences in HIV-infected men, demonstrating the biological plausibility for HSV infection facilitating HIV transmission [10].

Given the high HSV-2 seroprevalence and the possible amplification of HIV transmission in the presence of HSV-2 infection, a better understanding of the natural history of HSV shedding and symptomatic recurrences of HSV infection may help direct prevention of both HSV-2 and HIV infections. Several recent studies have shown that the frequency of HSV reactivation among immunocompetent men and women is higher than previously appreciated [11–13]. Because little is known about HSV shedding in MSM, we undertook a prospective cohort study to evaluate the frequency of, anatomic location of, and risk factors for clinical and subclinical HSV-1 and HSV-2 shedding among HIV-negative MSM, by collecting daily samples for viral cultures and detailed information about prodromal symptoms and symptomatic recurrences.

Materials and Methods

Study population. From February through June 1996, we recruited 30 HIV-seronegative HSV-2–seropositive MSM for a pro-

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Informed consent was obtained from all study participants, and the human experimentation guidelines of the Human Subjects Review Committee at the University of Washington were followed in the conduct of the clinical research.

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spective study of the frequency and anatomic sites of subclinical and symptomatic HSV shedding. The men were participants in the HIV Network for Prevention Trials Vaccine Preparedness Study in Seattle. Men were eligible to participate in the HIV Network for Prevention Trials if they were HIV-negative and reported any anal intercourse in the prior 12 months. All Seattle participants in the HIV Network for Prevention Trials underwent western blot testing for HSV, and those with antibodies to HSV-2 were invited to enroll in a consecutive 100-day study of HSV-2 shedding. History of symptomatic genital herpes was not required for participation. Men who were being treated with suppressive acyclovir at the time of screening were not eligible to participate.

A study clinician provided information about clinical signs and symptoms of oral and genital herpes and instructed the participants on methods of oral, genital, and perianal self-examination to detect herpes lesions. The participants were also taught how to obtain swabs for HSV cultures, as described elsewhere [12]; swabs were obtained daily at home from 4 sites: the perianal area, penile skin, meatus of the urethra, and mouth. For the penile skin, subjects were instructed to swab the entire ventral and dorsal surface of the penis; for the perianal area, they swabbed the external anus and inserted the swab into the anus. The men were instructed to obtain all culture specimens upon awakening. Participants recorded sexual activity and the dates of onset, duration, and location of oral, genital, or perianal symptoms and lesions on daily logs. Four clinic visits were scheduled at 2, 5, 10, and 14 weeks after enrollment; culture samples from the same four sites were obtained by the clinician at these visits. In addition, participants were asked to come to the study site for interim visits at the first sign or symptom of a herpes outbreak. All genital herpes lesions were noted by the clinician, and a specimen for viral culture was obtained from the lesion.

Laboratory methods. Western blot assays were performed to detect antibodies to HSV-1 and HSV-2, as described elsewhere [14]. Dacron swabs (Baxter Healthcare, Deerfield, IL) for HSV cultures were placed into viral transport media and were inoculated in triplicate into wells of human diploid fibroblast cultures. The wells were observed for 2 weeks; all cultures demonstrating cytopathic effects were confirmed and typed by direct immunofluorescence [15].

Statistical analysis. The overall rate of HSV-2 shedding was defined as the number of days of HSV-2–positive cultures of specimens from the perianal area, penis, urethra, or mouth, divided by the total number of days on which culture specimens were obtained. Similarly, the overall rate of HSV-1 shedding was defined as the number of HSV-1–positive cultures of specimens from any site divided by the total number of days on which culture specimens were obtained from the subset of subjects with antibodies to both HSV-1 and HSV-2. We defined a herpes recurrence as ≥1 consecutive days on which oral, penile, or perianal lesions were reported by the participant or observed by the clinician. Lesions observed by the participant that were culture-negative and judged by the clinician as not consistent with genital herpes were not counted as recurrences. Subclinical genital or perianal HSV shedding was defined as an HSV-positive culture of a specimen from the perianal area, penis, or urethra on ≥1 consecutive days in the absence of reported or observed genital or perianal lesions. Subclinical oral shedding was defined as an HSV-positive culture of a specimen from the mouth without reported or observed oral lesions. The rate of subclinical HSV shedding was computed as the number of days on which HSV was isolated divided by the number of days on which culture specimens were obtained and no lesions were reported.

The association between prodromal symptoms (defined as itching, burning, or tingling) at any time and HSV shedding was evaluated in the following manner: the overall rate of subclinical HSV shedding among all participants was calculated on days when they reported genital, perianal, or oral prodromal symptoms and on days when they did not report such symptoms. Reported prodromal symptoms antedating a herpes recurrence within 1 week were also evaluated.

Risk factors for total and subclinical HSV-1 and HSV-2 shedding were evaluated by using logistic regression, grouping the cultures for each participant [16]. This method adjusts for the correlation of culture results for an individual by estimating a scale factor; this scale factor accounts for the greater variability among subjects than that assumed in the standard logistic regression model. Unadjusted and adjusted ORs and 95% CIs were calculated for the following factors: age at enrollment, HSV serostatus (seropositive for both HSV-1 and HSV-2 vs. HSV-2–seropositive only), history of outbreaks of HSV infection, and number of episodes of anal sex during the study period.

Results

Study population. The demographic and clinical characteristics of the 30 study participants are shown in table 1. The mean age of the men was 41 years, and 90% of the study participants were white. Fourteen men (46.7%) had antibodies to HSV-2 only, and 16 (53.3%) had antibodies to both HSV-1 and HSV-2. Nineteen participants (63.3%) reported a history of symptomatic HSV infection (median duration of HSV infection from the first observed episode, 21 years). All 30 men were HIV-seronegative at enrollment, and none seroconverted from the mouth without reported or observed oral lesions. The rate of subclinical HSV shedding was computed as the number of days on which HSV was isolated divided by the number of days on which culture specimens were obtained and no lesions were reported.

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<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Value</th>
</tr>
</thead>
<tbody>
<tr>
<td>Age in y, mean (range)</td>
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</tr>
<tr>
<td>Race or ethnicity</td>
<td></td>
</tr>
<tr>
<td>White</td>
<td>27 (90)</td>
</tr>
<tr>
<td>Black</td>
<td>2 (6.7)</td>
</tr>
<tr>
<td>Latino</td>
<td>1 (3.3)</td>
</tr>
<tr>
<td>HSV serotype</td>
<td></td>
</tr>
<tr>
<td>HSV-2 only</td>
<td>14 (46.7)</td>
</tr>
<tr>
<td>HSV-1 and HSV-2</td>
<td>16 (53.3)</td>
</tr>
<tr>
<td>History of HSV recurrence(s)</td>
<td></td>
</tr>
<tr>
<td>Genital or perianal only</td>
<td>6 (20)</td>
</tr>
<tr>
<td>Oral only</td>
<td>6 (20)</td>
</tr>
<tr>
<td>Genital or perianal and oral</td>
<td>7 (23.3)</td>
</tr>
<tr>
<td>None</td>
<td>11 (36.7)</td>
</tr>
<tr>
<td>Years of HSV infection, median (range)*</td>
<td></td>
</tr>
<tr>
<td>Genital or perianal</td>
<td>11 (0.25–21)</td>
</tr>
<tr>
<td>Oral</td>
<td>26 (7–37)</td>
</tr>
</tbody>
</table>

NOTE. Data are no. (%) of subjects unless stated otherwise.

* For the 19 men with a history of HSV infection.
during the study. The median length of follow-up was 100 days (range, 49–132 days), with a median of 85 days on which participants collected culture specimens.

**Frequency of HSV shedding.** Viral culture specimens were collected on 2474 (86.6%) of 2856 days of follow-up. A total of 9823 specimens for HSV cultures were obtained from the mouth, perianal area, penis, and urethra; 9617 (97.9%) of these cultures yielded valid interpretable results. At least 1 culture was positive for HSV-2 during follow-up for 16 (53.3%) of the 30 men. Overall, HSV-2 was isolated on 76 (3.1%) of the days. The frequency of HSV-2 shedding among the 16 MSM from whom HSV-2 was isolated ranged from 1.3% to 26.3% of the days on which culture specimens were obtained. At least 1 culture for 9 (56.3%) of the 16 HSV-1–seropositive men was positive for HSV-1 during follow-up. The overall rate of HSV-1 shedding among participants seropositive for both HSV-1 and HSV-2 was 2.1%, with HSV-1 isolated on 24 days; individual rates of shedding among those men who shed HSV ranged from 1% to 23.7%.

**Anatomic sites of HSV reactivation.** HSV was isolated from the perianal region in 14 men, the genital skin in 4, the urethra in 2, and the mouth in 16. Of the 78 isolates of HSV-2, 65 (83.3%) were from perianal swabs. The rate of perianal HSV-2 shedding was 2.7%. HSV-2 shedding was rarely penile, urethral, or oral (table 2). In contrast, HSV-1 shedding was almost exclusively oral; of the 24 HSV-1 isolates, 23 were from the mouth, and 1 was from the perianal area.

The duration and anatomic location of HSV-2 shedding for 5 representative study participants are shown in figure 1. Subject 1 shed both HSV-1 and HSV-2 without detectable lesions and shed either HSV-1 or HSV-2 from the mouth on 2 different days. Subject 2 had a culture-negative lesion after 1 day of subclinical HSV-2 shedding and had 4 other episodes of subclinical perianal HSV-2 shedding for 1 or 2 days. Subjects 3 and 4 had culture-positive genital and perianal recurrences, and subject 4 also had subclinical HSV-1 shedding from the mouth on 2 occasions. Subject 5 had subclinical HSV-2 shedding from the perianal area on multiple occasions for 1 day each and had 2 culture-negative oral lesions.

**Subclinical HSV shedding.** For all study participants, no signs or symptoms of genital or oral herpes were present on 72 of the 100 days on which an HSV-positive culture specimen was obtained. Subclinical genital or perianal shedding occurred on 68% of the total days on which HSV-1 or HSV-2 was isolated from the genital or perianal area, whereas subclinical oral shedding accounted for 84% of the days on which HSV-1 was isolated. Fifteen men (50%) had subclinical HSV-1 or HSV-2 shedding from the perianal area, penis, or urethra; the overall rate of subclinical shedding was 2.2%, with individual rates of subclinical shedding ranging from 1% to 23.7% among those men who shed virus. As with total shedding, the rate of subclinical HSV-2 shedding was the highest from the perianal site, compared with the rates of subclinical HSV-2 shedding from the penis and urethra (table 2). Nine (56.3%) of 16 participants with antibodies to both HSV-1 and HSV-2 had subclinical oral shedding (overall rate of subclinical oral HSV-1 shedding, 1.7%; range, 1.1%–9.5% among those who shed HSV-1). Characteristics of subclinical and clinical shedding. The characteristics of clinical and subclinical HSV shedding are shown in table 3. The 19 men who shed either HSV-1 or HSV-2 had 44 episodes of viral shedding from the perianal area, penis, or urethra. Of these 44 episodes, 33 (75%) occurred entirely without lesions, 8 (18.2%) occurred on days that genital lesions occurred, and 3 (6.8%) spanned days on which lesions did and did not occur. There were 17 episodes of oral HSV shedding, of which 15 (88.2%) occurred without any lesions and the remaining 2 (11.8%) occurred when oral lesions were present. The mean duration of both subclinical genital or perianal shedding and oral shedding was 1.4 days, compared with mean durations of 2.2 and 2 days for symptomatic genital or perianal and oral shedding, respectively. Only 1 man shed HSV from multiple sites on the same day; HSV-2 was isolated from the penis, urethra, and perianal area subclinically on 1 day.

**Recurrent herpes lesions.** Fourteen (46.7%) of the 30 study participants had 44 episodes of HSV reactivation from the genital and perianal area, penis, or urethra; 9617 (97.9%) of these cultures yielded valid interpretable results. At least 1 culture was positive for HSV-2 during follow-up for 16 (53.3%) of the 30 men. Overall, HSV-2 was isolated on 76 (3.1%) of the days. The frequency of HSV-2 shedding among the 16 MSM from whom HSV-2 was isolated ranged from 1.3% to 26.3% of the days on which culture specimens were obtained. At least 1 culture for 9 (56.3%) of the 16 HSV-1–seropositive men was positive for HSV-1 during follow-up. The overall rate of HSV-1 shedding among participants seropositive for both HSV-1 and HSV-2 was 2.1%, with HSV-1 isolated on 24 days; individual rates of shedding among those men who shed HSV ranged from 1% to 23.7%.

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**Recurrent herpes lesions.** Fourteen (46.7%) of the 30 par-
Participants had herpes lesions during the study follow-up. These 14 persons reported 18 separate episodes of genital lesions (perianal, 11; genital, 7) and 17 episodes of oral lesions. The mean duration of lesions was 5 days (perianal, 5.6 days; genital, 4.1 days; oral, 5.0 days). Twelve (34.3%) of the 35 symptomatic herpes recurrences were observed by the clinician during a clinic visit, whereas the other 23 were self-reported. Cultures were positive for HSV-2 in 8 (44%) of the 18 episodes of penile or perianal lesions, and cultures were positive for HSV-1 in 2 (12%) of the 17 episodes of oral lesions. Of the remaining 25 culture-negative episodes, 18 were self-reported recurrences (genital, 3; perianal, 4; oral, 11), and 7 were clinician-observed recurrences (genital, 1; perianal, 2; oral, 4), in which samples were obtained by the clinician.

Men with a history of genital or perianal herpes recurrences were twice as likely as men without such a history to develop symptomatic recurrences; however, there was no association of history of oral herpes with shedding rates. Seven (53.8%) of the 13 men with a self-reported history of symptomatic genital or perianal herpes developed genital or perianal lesions during follow-up, compared with 4 (23.5%) of the 17 men without a history of symptomatic genital or perianal herpes ($P = .1$).

### Prodromal symptoms

Seventeen men (56.7%) reported prodromal symptoms, defined as itching, burning, or tingling. These symptoms occurred on 125 days of the study and were most commonly perianal (57%), genital (32%), or oral (18%). Six (4.8%) of these days on which prodromal symptoms occurred antedated a herpes recurrence within 1 week. The rate of viral shedding was 9% on the days that perianal prodromal symptoms were noted, compared with a shedding rate of 1.8% on the days on which a prodrome or lesions did not occur (table 2). The shedding rate was also higher (2.5%) on the days that genital prodromal symptoms occurred than on the days on which such symptoms did not occur (0.1%). Oral prodrome was not associated with oral shedding.

### Risk factors associated with HSV shedding

Risk factors for total and subclinical HSV-2 shedding were evaluated for all 30 men by logistic regression analyses. In both the unadjusted and adjusted models, men seropositive for both HSV-1 and HSV-2 were more likely to shed HSV-2 than were men with HSV-2 infection only (OR, 4.1; 95% CI, 1.4–11.9, adjusting for age, history of HSV infection, and frequency of engaging in anal sex; table 4). Likewise, men with antibodies to both HSV-1 and HSV-2 were also more likely than men with HSV-2 in-
fication only to shed HSV-2 subclinically (adjusted OR, 4.9; 95% CI, 1.4–17.4). Age and frequency of engaging in anal sex during the study were not associated with total or subclinical HSV-2 shedding. History of genital or perianal herpes recurrences was associated with higher rates of total and subclinical shedding, although the difference did not reach statistical significance.

Risk factors for total and subclinical HSV-1 shedding were assessed by logistic regression for the 16 men with antibodies to HSV-1. There were no significant predictors, including age, history of symptomatic oral HSV infection before the study, or prodromal symptoms during the study, for either total or subclinical HSV-1 shedding (data not shown). Our power for this analysis was limited by the smaller number of men who shed HSV-1 and, among them, a lower rate of HSV-1 shedding.

Discussion

This intensive 100-day study of rates of, anatomic site of, and risk factors for HSV shedding in 30 HIV-negative HSV-2-seropositive MSM demonstrates that reactivation of both HSV-1 and HSV-2 is more frequent than clinically appreciated. Overall, we isolated HSV-2 on 3.1% of the days, a rate similar to that for HIV-negative heterosexual men and women [17, 18]. Several important observations were generated from this study. The perianal region was the predominant site of HSV reactivation, as was found in previous studies of MSM [12, 13]; overall, 83.3% of HSV-2 isolates were from the perianal region. Our study also indicated that the frequency of clinically apparent perianal lesions among MSM is higher than that among heterosexual men and women [17], in that 11 of 18 clinical recurrences were perianal. The rate of subclinical shedding from the perianal region was particularly high on days when men noted prodromal symptoms. On those days, men may have had unrecognized perianal herpes lesions, since it is difficult for study participants to observe this area.

As with previous studies, we observed a wide variability in shedding rates among those men who shed HSV at some point during the study: from 1.3% to 26.3% of the days on which HSV-2 shedding occurred and 1% to 23.7% of the days on which HSV-1 shedding occurred among men seropositive for both HSV-1 and HSV-2. Of interest, a reported history of recurrences was not a significant predictor of subclinical or clinical shedding. These data provide a rationale for routinely performing HSV-2–specific serologies on all sexually active MSM at risk for transmitting or acquiring HSV infection.

The most novel finding of our study was the high shedding rates on days when patients noted prodromal symptoms; rates of perianal or genital shedding were 6.6% on days when participants noted symptoms consistent with a herpes prodrome, compared with 1.9% on days when they did not note prodromal symptoms. Prodromal symptoms antedating symptomatic recurrences of HSV infection are common, but whether this occurrence is accompanied by HSV reactivation on mucosal surfaces has been controversial. Our data indicate that such symptoms are often associated with HSV shedding. These prodromal symptoms, such as hyperesthesia and local irritative symptoms, probably represent reactivation of HSV-2 from the sensory root ganglia, with subsequent epithelial reactivation. Because the participants were not examined daily in the clinic, some of the symptoms coded as prodromes may actually have represented subtle herpes lesions that the participant missed or could not visualize because of location. However, even if there is some misclassification in what is defined as a prodrome versus mild clinical signs not detected by the study participants, our finding of higher shedding rates on days when participants noted prodromal symptoms is potentially useful clinically to encourage patients to recognize prodromes and lesions, as well as in counseling about the high rate of viral shedding and potential transmission during such episodes. Unfortunately, on most days when subclinical shedding occurred, the participants were truly asymptomatic.

The data showing a substantially higher rate of shedding of HSV-2 on days when the men had prodromal symptoms suggest that persons who are HSV-2-seropositive should be counseled

Table 3. Characteristics of subclinical and clinical shedding of herpes simplex virus (HSV) during 100 days of follow-up in 30 HIV-seronegative men who have sex with men.

<table>
<thead>
<tr>
<th>Characteristic</th>
<th>Without lesions</th>
<th>With lesions</th>
<th>Without lesions</th>
<th>With lesions</th>
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</thead>
<tbody>
<tr>
<td>Total no. of d of shedding</td>
<td>51</td>
<td>24</td>
<td>21</td>
<td>4</td>
</tr>
<tr>
<td>Duration of shedding episodes, mean d (range)</td>
<td>1.4 (1–3)</td>
<td>2.2 (1–4)</td>
<td>1.4 (1–3)</td>
<td>2 (1–3)</td>
</tr>
<tr>
<td>No. (%) of shedding episodes lasting:</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1 d</td>
<td>24 (66.7)</td>
<td>5 (45.5)</td>
<td>12 (80)</td>
<td>1 (50)</td>
</tr>
<tr>
<td>2 d</td>
<td>9 (25)</td>
<td>1 (9)</td>
<td>1 (6.7)</td>
<td>0 (0)</td>
</tr>
<tr>
<td>3–4 d</td>
<td>3 (8.3)</td>
<td>5 (45.5)</td>
<td>2 (13.3)</td>
<td>1 (50)</td>
</tr>
</tbody>
</table>

* Five men had both subclinical and clinical genital or perianal HSV shedding; 1 man had both subclinical and clinical oral HSV shedding.

* Three episodes of genital or perianal HSV shedding occurred on days on which lesions did and did not occur; these 3 episodes are included in both columns.
about the types of symptoms compatible with herpes prodromes, as well as the range of mucocutaneous lesions caused by herpes. At those times, there may be greater potential for transmitting infection. Avoidance of sex should particularly be encouraged during prodromal symptoms, just as it is for lesions. Episodic antiviral therapy may also be of benefit during herpes prodromes to decrease HSV shedding. An ongoing randomized trial of daily suppressive antiviral therapy for HSV-2-discordant couples will eventually provide data on the efficacy of antivirals to reduce rates of HSV-2 transmission. However, until these data are available, HSV-2-seropositive persons should be encouraged to use condoms to reduce the risk of transmitting HSV-2 to their uninfected partners.

Multivariate analysis suggested that MSM who were seropositive for both HSV-1 and HSV-2 were more likely to shed HSV-2 than were men who were HSV-2–seropositive only. A similar but weaker association has been observed for HIV-infected MSM [12]. However, studies involving HSV-2–infected women have not shown this association [17, 19]. This observation warrants further study. Our finding differs from that of a previous cohort study of pregnant women at risk for HSV infection [20]; in that study, it was demonstrated that there is partial cross-protection against acquisition of HSV-1 in women with antibodies to HSV-2, but cellular immune response or other factors may be more important than antibody with respect to the frequency of shedding and reactivation.

We did not have adequate power to assess predictors of HSV-1 shedding in the men who shed HSV-1 by means of multivariate analyses. However, it is interesting to note that, even though an increasing proportion of HSV-2 infections and HSV-positive rectal cultures among MSM attending the Harborview STD Clinic in Seattle has been associated with HSV-1 [21], we observed very little perianal HSV-1 shedding in this study of MSM coinfected with HSV-1 and HSV-2. This finding may reflect infrequent acquisition of rectal HSV-1 infection in this group of men or rare reactivation of HSV-1 below the waist [22].

The limitations of our study include occasional missing data; the average number of days on which culture specimens were not obtained by study participants was 16, which could have changed the total shedding rates. However, if all missing culture specimens are assumed to be negative, the rates of clinical and subclinical shedding do not significantly change. In approximately two-thirds of the symptomatic herpes recurrences, participants did not present to the study clinic, and thus clinical findings could not be confirmed by the clinician. Clinical confirmation of self-reported symptoms may have been especially helpful for oral lesions, given that culture specimens were positive for HSV on only one-third of the days on which participants reported oral lesions. However, other investigators have also reported infrequent isolation of HSV-1 from oral herpes lesions [23]. Last, given the episodic nature of HSV shedding and the interperson variability of shedding rates, even a 100-day period could be insufficient to detect individuals with infrequent shedding.

Our findings indicate that HIV-negative HSV-2–seropositive MSM shed HSV an average of 3.1% of the 100–day study period and shed HSV-2 primarily from the perianal area. Most of the shedding was subclinical. Although shedding was more common on days when participants noted prodromal symptoms, most shedding was not accompanied by any symptoms. Current approaches for prevention should include identification of HSV-2–seropositive persons through targeted serological screening with type-specific tests, education of HSV-infected persons about recognition of visible lesions and prodromal symptoms, and counseling about condom use and safe sex practices.

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References


