

physicians. However, the majority of people seeking mental health care prefer to be addressed as patients, which leads us to believe that there is no reason to deviate from the current medical vocabulary.

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HIV infection and risk behaviours in young gay and bisexual men

We have several concerns about the interpretation of the findings of a recent cohort study of sexual behaviour and HIV infection among young men who have sex with men in Vancouver.¹ Of the 11 subjects who became seropositive, 3 reported having injected drugs and 1 having shared needles. The other 2 may also have shared needles; this practice is often underreported because of its social undesirability and poor recall related to the effect of the drugs. Thus, the 3 subjects may have been infected through injection rather than through sex with other men. In fact, injection was significantly associated with HIV infection ($p <$

0.001) whereas sharing needles was not ($p = 0.06$), and HIV incidence among injection drug users during this period was extremely high (18.6 per 100 person-years²).

We also question the inclusion of the man who had an indeterminate result at baseline in the seroconverter group; subjects in a cohort study should be susceptible at study entry. Excluding the 3 subjects who injected drugs and the seroconverter yields an HIV incidence of 1.1–1.3 per 100 person-years (depending on whether the seroconverter also injected drugs). This is similar to the HIV incidence of 1.05 per 100 person-years we observed in men under 30 years old who have sex with men in Montreal from 1996 to 1999 (unpublished data). We believe HIV incidence among men who have sex with men should be calculated excluding those with other risk factors or, alternatively, calculations should be made separately for subjects with and without other risk factors.

Finally, the authors concluded that levels of unsafe sex increased over time on the basis of the proportion of subjects reporting safe sex at baseline who reported unsafe sex at follow-up. In Montreal we found that sexual behaviour is dynamic; a large proportion (51%) of those who practised unsafe sex at baseline practised only safe sex at follow-up,³ which resulted in similar proportions of subjects reporting unsafe sex at baseline and follow-up despite the fact that about 10% of those reporting safe sex at baseline reported unsafe sex at follow-up. Therefore, risky sexual behaviour among both those with safe and those with unsafe

sexual practices at baseline must be examined at follow-up.

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[The authors respond:]

We recalculated the HIV infection rate based on all years and on whether a subject had reported that they had ever injected drugs. These new person-time estimates of HIV incidence are based on 18 subjects who became HIV positive after their baseline seronegative test, 8 more than in our published study¹ and excluding the person with the baseline indeterminate result. The incidence rate has been revised to 1.2 per 100 person-years (95% CI 0.6–1.7) (Table 1) since the paper

Table 1: Incidence of HIV infection among study participants, by study year and category

Study year	All participants ($n = 617$)		Noninjection drug users ($n = 555$)		Injection drug users ($n = 61$)	
	New infections	Rate (95% CI)	New infections	Rate (95% CI)	New infections	Rate (95% CI)
1	1	1.0 (0.0–2.8)	1	1.0 (0.0–3.1)	0	–
2	6	1.7 (0.3–3.1)	3	0.9 (0.0–2.0)	3	11.4 (0.0–24.3)
3	2	0.5 (0.0–1.1)	1	0.2 (0.0–0.7)	1	3.2 (0.0–9.4)
4	5	1.2 (0.1–2.2)	4	1.0 (0.0–2.0)	0	–
5	4	1.8 (0.0–3.6)	4	2.0 (0.0–4.1)	0	–
All years	18*	1.2 (0.6–1.7)	13	0.9 (0.4–1.4)	4	3.1 (0.6–6.1)

*Data regarding injection drug use were unavailable for 1 seroconverter, who was identified through anonymous database linkage.

was written. Gay and bisexual men who injected drugs have a higher incidence rate (3.1 per 100 person-years [95% CI 0.6–6.1]) than those who did not (0.9 per 100 person-years [95% CI 0.4–1.4]). Robert Remis and colleagues are correct in assuming that our rate of HIV incidence among noninjecting participants is similar to that reported for men under 30 years in the Omega cohort (1.0 per 100 person-years from 1996 to 1999). However, the annualized incidence rates presented in Table 1 for noninjecting gay and bisexual men indicate that HIV infection appears to be increasing in this population.

Remis and colleagues felt that we should have reported follow-up risky sexual behaviour among participants who had safe as well as unsafe sexual practices at baseline. Of the 285 men with regular partners, 89 (31.2%) reported having unprotected insertive anal sex and 100 (35.0%) reported having unprotected receptive anal sex in the year before the baseline visit. At 1-year follow-up, 66 (74.1%) of the 89 subjects and 71 (71.0%) of the 100 subjects reported having unprotective insertive and receptive anal sex respectively. Of the 279 men with casual partners, 46 (16.5%) reported having unprotected insertive and 36 (12.4%) unprotected receptive anal sex in the year before the baseline visit. Of these men, 21 (46.6%) and 16 (44.4%) reported having had unprotected insertive and receptive anal intercourse respectively by the time of their first follow-up visit. In combining these

data with other information presented in our paper,¹ the odds of relapse among men with regular partners increased 2-fold for both unprotected insertive intercourse (odds ratio 2.2, 95% CI 1.4–3.7) and receptive anal intercourse (odds ratio 1.9, 95% CI 1.4–3.0). Among men with casual partners, similar odds were observed for unprotected insertive intercourse (odds ratio 1.7, 95% CI 1.0–2.8), but the odds for receptive anal intercourse were not significantly increased (odds ratio 1.3, 95% CI 0.7–2.3). These new data along with the findings originally presented in our article confirm the high HIV rates and sexual risk behaviour in our cohort.

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Different centuries, same old story

The recent *Escherichia coli* outbreak in Walkerton, Ont., is remarkably similar to a cholera outbreak in Hamburg, Germany, in 1892. However, it is not the outbreak of waterborne disease that makes these stories similar but the delays in warning citizens of the emerging epidemics threatening them.

In Hamburg, the first person to die from cholera was a building worker who inspected a sewage outlet on Aug. 15. Although his vomiting and diarrhea were consistent with cholera, an official diagnosis could not be made without a cultured bacillus. Another building worker became ill with the same symptoms and died Aug. 17. However, gastrointestinal upset was not uncommon during the summer months in Hamburg and local doctors were not persuaded to take the time to investigate the cause of each illness. Physicians did not attempt to culture the bacillus until Aug. 20. In the meantime, others began to show signs of infection: 2 people on the 16th, 4 more on the 17th and 12 on the 18th. By Aug. 19, 31 patients had received treatment.

Although "official" confirmation of the cholera outbreak had been received by Aug. 22, the chief medical officer and the Senate chose not to warn people to boil water, and the contamination was not publicized until Aug. 24. By then, every part of the city had been infected and thousands of citizens had unsuspectingly consumed the infected water; they soon became ill with cholera and began to infect others. By the time the cholera outbreak was fully contained almost 17 000 people had been infected and 8600 had died. The outbreak of 1892 killed 13.4% of the population of Hamburg; it killed as many people as all other cholera outbreaks in Germany during the 19th century.¹

Although several public inquiries and investigations are taking place to ascertain just what went wrong in Walkerton, the Hamburg outbreak does illus-

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