Using Centralized Laboratory Data to Monitor Trends in Herpes Simplex Virus Type 1 and 2 Infection in British Columbia and the Changing Etiology of Genital Herpes

Mark Gilbert, MD,[1,2] Xuan Li, MSc,[3] Martin Petric, PhD,[4] Mel Krajden, MD,[4] Judith L. Isaac-Renton, MD,[4] Gina Ogilvie, MD,[1,2] Michael L. Rekart, MD[1,2]

ABSTRACT

Objectives: Understanding the regional epidemiology of genital Herpes Simplex Virus (HSV) infections is important for clinical and public health practice, due to the increasing availability of type-specific serologic testing in Canada and the contribution of genital HSV-2 infection to ongoing HIV transmission. We used centralized laboratory data to describe trends in viral identifications of genital HSV in BC and assess the utility of these data for ongoing population surveillance.

Methods: Records of viral identifications (1997-2005) were extracted from the Provincial Public Health Microbiology & Reference Laboratory database. Classification as genital or other site was based on documented specimen site. We conducted a descriptive analysis of trends over time, and calculated odds of HSV-1 infection among individuals with genital herpes.

Results: Of 48,183 viral identifications, 56.8% were genital, 10.0% were peri-oral and 9.1% cutaneous; site was unknown for 22.9%. Among genital identifications, HSV-1 infection was more likely in females, younger age groups, and later time periods. The proportion of genital herpes due to HSV-1 increased over time from 31.4% to 42.8% in BC.

Conclusions: Our analysis of population-level laboratory data demonstrates that the proportion of genital herpes due to HSV-1 is increasing over time in BC, particularly among women and younger age groups; this has implications for clinical practice including the interpretation of type-specific serology. Provincial viral identification data are useful for monitoring the distribution of genital HSV-1 and HSV-2 infections over time. Improving clinical documentation of specimen site would improve the utility of these data.

Key words: Herpes simplex; herpes simplex, genital; epidemiology; Canada

La traduction du résumé se trouve à la fin de l'article.

Can J Public Health 2011;102(3):225-29.

Genital Herpes Simplex Virus (HSV) infection is one of the most prevalent sexually transmitted viral infections in North America.[1,2] Prevention of genital HSV infections will reduce the substantial associated clinical and psychological morbidity, and associated health care costs.[3,4] Prevention of genital HSV in women of reproductive age (particularly pregnant women in the third trimester) will reduce neonatal herpes infections, which although rare in Canada have severe morbidity and mortality.[5] Genital HSV-2 infection increases the likelihood of HIV acquisition and transmission[2,6] and interventions to prevent or treat HSV-2 infection have the potential to reduce the population transmission of HIV.[7,8] These reasons have led to calls for renewed efforts to control genital herpes nationally and for the development of a herpes vaccine.[9,10]

Genital herpes is caused by both HSV type 1 and HSV type 2 (HSV-1, HSV-2). HSV can be typed through viral identification methods on clinical specimens (i.e., viral isolation by culture, or nucleic acid amplification testing) or through type-specific serologic testing.[11] The latter inform seroprevalence surveys which in the past decade in Canada and the US have estimated the seroprevalence of HSV-1 from 51-62%, and HSV-2 from 10-19%.[12-15] While HSV-2 is almost always associated with genital herpes, HSV-1 (conventionally associated with oral-labial infection) is increasingly identified as a cause of genital infections globally.[16-22] Accordingly, seroprevalence data for HSV-1 are difficult to interpret and HSV-2 seroprevalence data underestimate the overall prevalence of genital HSV.[23] The population burden and epidemiologic trends in genital herpes infection for the province of British Columbia (BC) are currently unknown. Our objective was to examine the trends of laboratory-confirmed genital HSV infection for the province of BC

Author Affiliations

1. Division of STI/HIV Prevention and Control, BC Centre for Disease Control, Vancouver, BC
2. School of Population and Public Health, University of British Columbia, Vancouver, BC
3. University of Toronto, Toronto, ON
4. Provincial Health Services Authority Laboratories, Public Health Microbiology & Reference Laboratory, Vancouver, BC

Correspondence: Dr. Mark Gilbert, Division of STI/HIV Prevention and Control, BC Centre for Disease Control, 655 West 12th Avenue, Vancouver, BC V5Z 4R4, Tel: 604-707-5615, Fax: 604-707-5604, E-mail: mark.gilbert@bccdc.ca

Acknowledgements: The authors gratefully acknowledge the assistance of the following individuals: Rob MacDougall, Laboratory Services, Provincial Health Services Authority Laboratories for assistance in extracting and preparing the laboratory dataset used in this analysis; Paul Hyeong-Jin Kim, STI/HIV Prevention and Control, BC Centre for Disease Control for assisting with data analysis; Dr. Paul Gustafson, University of British Columbia for advising on statistical methods; and Dr. Réka Gustafson, Vancouver Coastal Health and Rhonda Kropp, Community Acquired Infections Division, Centre for Infectious Disease Prevention and Control, Public Health Agency of Canada for reviewing this manuscript. No external sources of funding were used for any part of this study.

Conflict of Interest: None to declare.

Table 1. Factors Associated With Genital HSV-1 Infection in Individuals With a Genital Identification of HSV, BC, 1997-2005

Characteristic	HSV-1 Viral Identifications Number	%	HSV-2 Viral Identifications Number	%	Odds Ratio [95% CI] Unadjusted	Odds Ratio [95% CI] Adjusted
Sex						
Female	8000	40.3%	11,837	59.7%	1.73 [1.63-1.84]	1.60 [1.50-1.70]
Male	1793	28.1%	4597	71.9%		
Age Category (years)						
<15	129	68.6%	59	31.4%	8.08 [5.90-11.07]	8.20 [5.91-11.37]
15-29	6366	48.1%	6871	51.9%	3.42 [3.17-3.70]	3.43 [3.17-3.72]
30-44	2633	29.9%	6162	70.1%	1.58 [1.45-1.72]	1.65 [1.51-1.80]
≥45	1039	21.3%	3840	78.7%	Referent Category	Referent Category
Period						
1997-1999	2630	33.8%	5153	66.2%	Referent Category	Referent Category
2000-2002	2266	36.4%	3952	63.6%	1.12 [1.05-1.21]	1.19 [1.11-1.29]
2003-2005	5376	40.2%	8012	59.8%	1.32 [1.24-1.39]	1.43 [1.34-1.52]

(population 4.4 million people) using data from the Provincial Public Health Microbiology & Reference Laboratory (PHMRL), which conducts >90% of all provincial HSV testing. By improving our understanding of the regional epidemiology of genital herpes infection (including the distribution of HSV-1 and HSV-2), we hope to provide clinicians with information to guide counseling of patients and interpretation of serologic testing. With the possibility of new public health interventions and control strategies related to HSV in the future, we also assessed the usefulness of provincial laboratory testing for ongoing surveillance of genital herpes infections in British Columbia.

MATERIALS AND METHODS

Laboratory results for viral cultures between 1997 and 2005 were reviewed. Specimens consisting of lesion swabs were inoculated on Vero cell monolayers in respective wells of four microtitre plates under centrifugation (1000 rcf for 30 min). After incubation for two days, the monolayers on one plate were fixed and stained with a fluorescein conjugated monoclonal antibody reactive with both herpes types, and visualized by immunofluorescence microscopy. To identify the herpes virus type, respective wells on two additional plates inoculated in parallel were then fixed and stained with fluorescein conjugated type-specific herpes monoclonal antibody and visualized by immunofluorescence microscopy.

Laboratory records with a diagnosis of HSV by viral culture were extracted from the PPHRL database and nominal identifiers removed. Variables included in this analysis were age, sex, site of clinical specimen, test type, and result. Test results with unknown HSV type were excluded from further analysis; a small number of test results with identification of both HSV-1 and HSV-2 were counted twice during type-specific analysis (once for each HSV type). We defined genital herpes lesions as a positive result in specimens from sites in a "boxer short" distribution, namely: groin, pubic area, urethra, penis, vagina, vulva, cervix, clitoris, introitus, labia, perineum, anus, perianal, rectum, or buttocks. Specimens from other sites were classified as peri-oral (lip, mouth, oropharynx, throat, tongue, nasal, nasopharynx, nose, cheek, chin), ocular (eye, eyelid, conjunctiva, cornea), or other cutaneous lesions. All analyses were carried out in SPSS v16.0.1 (Apache Software Foundation, USA). We calculated chi-square test for trends, and among individuals with genital HSV identifications, calculated crude odds ratios and 95% confidence intervals [95% CI] of HSV-1 infection by sex, age category, and period; all were entered into a multivariate logistic regression model from which adjusted odds ratios (AOR) were obtained. Population rates were calculated using annual provincial population estimates.

Figure 1. Number of viral cultures by site and proportion by HSV type, BC, 1997-2005

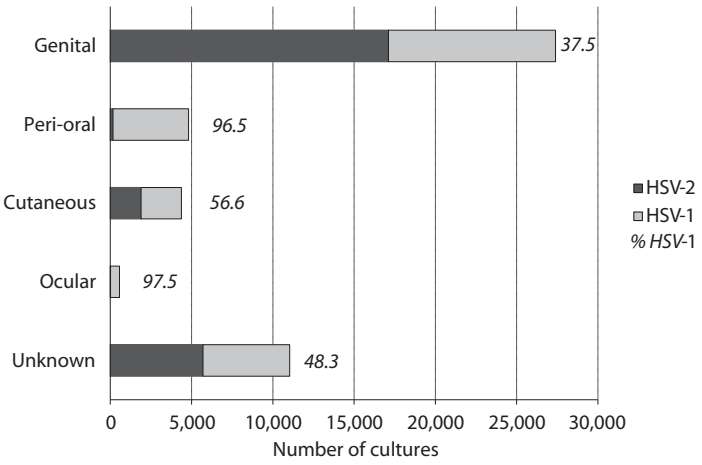
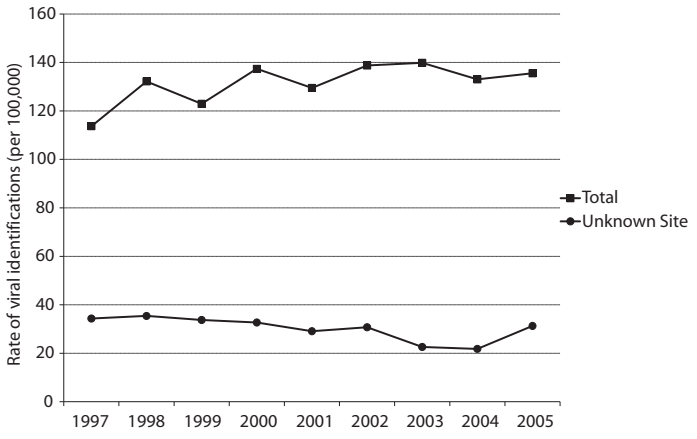


Figure 2. Total positive identifications of HSV by specimen site, BC, 1997-2005

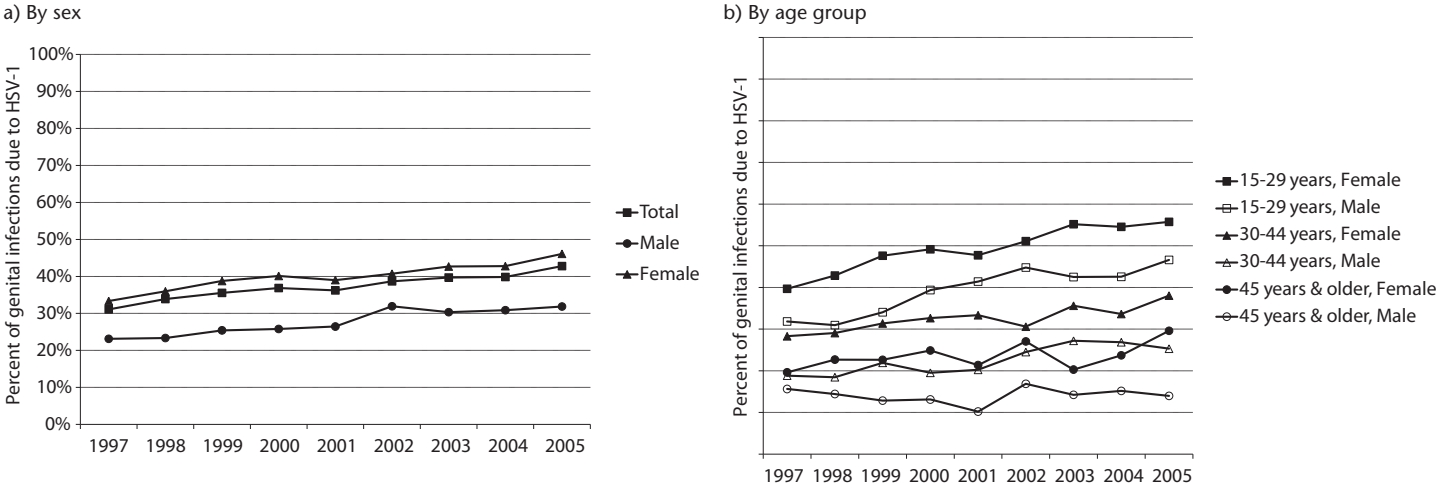


Genital HSV has been a laboratory-reportable disease in British Columbia since 1983 for the purpose of public health surveillance and control. As this study was part of routine surveillance, ethical approval was not sought.

RESULTS

We identified 48,546 laboratory reports between 1997 and 2005 with a viral identification of HSV including: 23,254 (47.9%) reports of HSV-1, 24,883 (51.3%) reports of HSV-2, 23 (0.05%) reports of both HSV-1 and HSV-2, and 386 (0.8%) reports of unspecified type. Of the 48,183 typed identifications, the majority (27,389; 56.8%) were from genital sites, with 37.5% and 62.5% due to HSV-1 and

Figure 3. Annual percentage of genital HSV infections due to HSV-1, BC, 1997-2005



HSV-2 infection respectively (Figure 1). We identified 4,818 reports of HSV in peri-oral specimens (10.0% of all viral identifications; 96.5% HSV-1, 3.5% HSV-2) and 4,371 reports of HSV from other cutaneous sites (9.1% of all viral identifications; 56.6% HSV-1, 43.4% HSV-2). A large proportion (22.9%) of specimens lacked information regarding site of infection (ranging from 16.1% to 30.9% per year; overall 48.3% HSV-1 and 51.7% HSV-2). The provincial HSV identification rate increased over the study period from 113.7 per 100,000 in 1997 to 135.5 per 100,000 in 2005 ($p<0.005$, Figure 2).

Among individuals with genital HSV identifications, females (AOR 1.60 [1.50-1.70]) were more likely to have HSV-1 infection, and an increasing gradient by younger age category and later time periods in likelihood of HSV-1 infection was apparent (Table 1). Over time, the proportion of genital herpes infections due to HSV-1 has been increasing in BC, from 31.4% in 1997 to 42.8% in 2005 (Figure 3a). This affects both sexes and is most pronounced among younger age groups (Figure 3b); all trends are significantly increasing ($p<0.05$) with the exception of males aged 45 years and older.

DISCUSSION

This is the first Canadian study to examine provincial trends in genital herpes infection over time and to assess the utility of these data for public health surveillance, made possible by access to centralized laboratory data for HSV testing in BC. We found that the rate of provincial HSV identifications has increased over time; however, a substantial number of annual HSV infections have no data on specimen site each year and misclassification of genital lesions is likely (as reflected by the large proportion of HSV-2 infections among specimens from cutaneous or unknown sites). Hence we are unable to assess population-based genital herpes rates.

Among all individuals with genital HSV infection, we found females and younger age groups to be more likely to have infection due to HSV-1, and time period was independently associated.[16] HSV-1 currently accounts for the majority of genital infections in younger age groups; the proportion of genital HSV-1 infection has been significantly increasing over time in both sexes and in almost all age groups in BC. Knowledge of this trend is important for clinicians as genital herpes due to HSV-1 infection is more likely to present with a symptomatic first episode, have a milder clinical course (fewer recurrences), and have reduced asymptomatic viral shedding.[2,24,25] This knowledge is also important for appropriate interpretation of type-specific serologic testing for HSV which is increasingly available in Canada (i.e., that HSV-1 antibodies may be related to both genital and non-genital infection, whereas HSV-2 antibodies are primarily related to genital infection).[11] While genital HSV-1 following genital HSV-2 infection is rare, genital HSV-1 infection does not protect completely against subsequent genital HSV-2 infection, which may explain the genital co-infections identified in this study.[2]

Few studies have examined population-based trends in laboratory-confirmed genital herpes infections. Surveillance data from genitourinary medicine and general practice clinics in the United Kingdom have demonstrated an increasing number of diagnoses of genital herpes over time,[26] and in the United States the total number of initial visits to physicians' offices for genital herpes has also been steadily increasing.[27] Other regions have demonstrated an increasing trend in the proportion of genital herpes due to HSV-1, with a greater proportion among females and at younger age groups.[16-23,28,29] The most likely explanation for this trend is reduced acquisition of HSV-1 infection in childhood (e.g., changes in living conditions affecting acquisition of oral-labial herpes) and increased susceptibility to HSV-1 at age of sexual debut, or changes in oral sex behaviour resulting in increased oral-labial to genital transmission of HSV-1.[23,30] Population-level seroprevalence surveys provide some proof for this theory, having demonstrated a reduction in the overall seroprevalence of HSV-1 over time, particularly among younger ages and in some ethnic groups.[12]

We acknowledge the following limitations. These data are representative of a tested population and genital herpes may be diagnosed based on clinical examination or history without laboratory testing.[4] Accordingly these data do not reflect the true population burden and may not reflect true population trends. Additionally, symptoms of primary and recurrent genital herpes infections may be atypical or vary in clinical severity, and may be undiagnosed (which may explain why only 14.2% of individuals with antibodies to HSV-2 in a large US seroprevalence survey reported receiving a diagnosis of genital herpes).[12] We also do not have denominator data on the number of tests performed over this time period and cannot account for the influence of changes in test volume or pat-

tern, and as data were de-identified we were unable to identify multiple testing episodes per individual and distinguish new from recurrent infections (which may lead to underestimating the proportion of genital herpes due to HSV-1 due to the lower likelihood of recurrence compared to HSV-2). We did not include type-specific serologic tests or nucleic acid amplification test results in our analysis as these had limited use during the study period. The use of laboratory data for surveillance purposes has inherent limitations, including errors related to incomplete or erroneous completion of test requisition forms or data entry errors (as suggested by our findings related to HSV-2 infections in specimens from unknown or other cutaneous sites). Similarly, data on sex and age were missing for 4.2% and 1.1% of genital specimens, respectively. Finally, we postulate that differences in the severity of clinical presentation between genital HSV-1 and HSV-2 infection may lead to differences in rates of diagnostic testing; for example, primary episodes of genital HSV-1 are more likely to be symptomatic than first episodes of HSV-2 (due to the modulating effect of previous HSV-1 infection).

In conclusion, we have demonstrated that an increasing proportion of genital herpes in British Columbia is caused by HSV-1, which accounts for over 50% of all new genital herpes infections among individuals less than 30 years of age. This information is important to communicate to clinicians in order to guide client counseling and appropriate ordering and interpretation of serologic and clinical specimen HSV testing (e.g., positive HSV-1 serology may indicate genital infection), and for informing public health sexual health education programs (e.g., to youth regarding potential for oral-labial to genital transmission of HSV-1 infection). We have also demonstrated the value of using centralized laboratory data on type-specific HSV infection for public health surveillance, which is likely most robust for monitoring the percentage distribution of genital HSV-types over time than population rates. Such data can be used for evaluation of public health control strategies for HSV infection, including future vaccine programs. Understanding trends in HSV-related laboratory data is a useful complement to surveillance data from seroprevalence surveys, and is required in order to interpret seroprevalence data appropriately. Being able to identify unique individuals undergoing viral identification testing for HSV (i.e., to distinguish between first and recurrent episodes), monitoring the total volume of HSV testing, and improving the documentation of clinical specimen sites would enhance the utility of centralized laboratory data for HSV infection in British Columbia. Understanding the trends in genital herpes in our province may also be enhanced by monitoring incident genital herpes infections in defined cohorts with higher quality demographic information and enhanced data on sexual behaviours, such as among clients attending sexually transmitted infection clinics around the province. These recommendations may be relevant for surveillance programs in other provinces in Canada and globally.

REFERENCES

1. Mertz KJ, Trees D, Levine WC, Lewis JS, Litchfield B, Pettus KS, et al. Etiology of genital ulcers and prevalence of human immunodeficiency virus coinfection in 10 US cities. The Genital Ulcer Disease Surveillance Group. *J Infect Dis* 1998;178:1795-98.
2. Corey L, Wald A. Genital herpes. In: Holmes K, Sparling P, Stamm WE, et al. (Eds.), *Sexually Transmitted Diseases*. New York, NY: McGraw Hill, 2008;399-437.
3. Tao G, Kassler WJ, Rein DB. Medical care expenditures for genital herpes in the United States. *Sex Transm Dis* 2000;27:32-38.
4. Romanowski B, Zdanowicz YM, Owens ST. In search of optimal genital herpes management and standard of care (INSIGHTS): Doctors' and patients' perceptions of genital herpes. *Sex Transm Inf* 2008;84:51-56.
5. Kropp RY, Wong T, Cormier L, Ringrose A, Burton S, Embree JE, et al. Neonatal herpes simplex virus infections in Canada: Results of a 3-year national prospective study. *Pediatrics* 2006;117:1955-62.
6. Freeman EE, Weiss HA, Glynn JR, Cross PL, Whitworth JA, Hayes RJ. Herpes simplex virus 2 infection increases HIV acquisition in men and women: Systematic review and meta-analysis of longitudinal studies. *AIDS* 2006;20:73-83.
7. Celum C, Wald A, Hughes J, Sanchez J, Reid S, Delany-Moretlwe S, et al. Effect of aciclovir on HIV-1 acquisition in herpes simplex virus 2 seropositive women and men who have sex with men: A randomised, double-blind, placebo-controlled trial. *Lancet* 2008;371:2109-19.
8. Watson-Jones D, Weiss HA, Rusizoka M, Changalucha J, Baisley K, Mugeye K, et al. Effect of herpes simplex suppression on incidence of HIV among women in Tanzania. *N Engl J Med* 2008;358:1560-71.
9. Corey L, Handsfield HH. Genital herpes and public health: Addressing a global problem. *JAMA* 2000;283:791-94.
10. Hook EW, Leone P. Time to translate new knowledge into practice: A call for a national genital herpes control program. *J Infect Dis* 2006;194:6-7.
11. Ratnam S, Severini A, Zahariadis G, Petric M, Romanowski B. The diagnosis of genital herpes - beyond culture: An evidence-based guide for the utilization of polymerase chain reaction and herpes simplex virus type-specific serology. *Can J Infect Dis Med Micro* 2007;18:233-40.
12. Xu F, Sternberg MR, Kottiri BJ, McQuillan GM, Lee FK, Nahmias AJ, et al. Trends in herpes simplex virus type 1 and type 2 seroprevalence in the United States. *JAMA* 2006;296:964-73.
13. Patrick DM, Dawar M, Cook DA, Krajden M, Ng HC, Rekart ML. Antenatal seroprevalence of herpes simplex virus type 2 (HSV-2) in Canadian women: HSV-2 prevalence increases throughout the reproductive years. *Sex Transm Dis* 2001;28:424-28.
14. Singh AE, Romanowski B, Wong T, Gourishankar S, Myziuk L, Fenton J, et al. Herpes simplex virus seroprevalence and risk factors in 2 Canadian sexually transmitted disease clinics. *Sex Transm Dis* 2005;32:95-100.
15. Howard M, Sellors JW, Jang D, Robinson NJ, Fearon M, Kaczorowski J, et al. Regional distribution of antibodies to herpes simplex virus type 1 (HSV-1) and HSV-2 in men and women in Ontario, Canada. *J Clin Microbiol* 2003;41:84-89.
16. Forward K, Lee S. Predominance of herpes simplex virus type 1 from patients with genital herpes in Nova Scotia. *Can J Infect Dis* 2003;14:94-96.
17. Gray E, Morgan J, Lindeman J. Herpes simplex type 1 versus Herpes simplex type 2 in anogenital herpes; a 10 year study from the Waikato region of New Zealand. *N Z Med J* 2008;121:43-50.
18. Haddow LJ, Dave B, Mindel A, McPhie KA, Chung C, Marks C, et al. Increase in rates of herpes simplex virus type 1 as a cause of anogenital herpes in western Sydney, Australia, between 1979 and 2003. *Sex Transm Infect* 2006;82:255-59.
19. Manavi K, McMillan A, Ogilvie M. Herpes simplex virus type 1 remains the principal cause of initial anogenital herpes in Edinburgh, Scotland. *Sex Transm Dis* 2004;31:322-24.
20. Ribes JA, Steele AD, Seabolt JP, Baker DJ. Six-year study of the incidence of herpes in genital and nongenital cultures in a central Kentucky medical center patient population. *J Clin Microbiol* 2001;39:3321-25.
21. Scoular A, Norrie J, Gillespie G, Mir N, Carman WF. Longitudinal study of genital infection by herpes simplex virus type 1 in Western Scotland over 15 years. *BMJ* 2002;324:1366-67.
22. Tran T, Druce JD, Catton MC, Kelly H, Birch CJ. Changing epidemiology of genital herpes simplex virus infection in Melbourne, Australia, between 1980 and 2003. *Sex Transm Infect* 2004;80:277-79.
23. Lafferty WE, Downey L, Celum C, Wald A. Herpes simplex virus type 1 as a cause of genital herpes: Impact on surveillance and prevention. *J Infect Dis* 2000;181:1454-57.
24. Engleberg R, Carrell D, Krantz E, Corey L, Wald A. Natural history of genital herpes simplex virus type 1 infection. *Sex Transm Dis* 2003;30:174-77.
25. Langenberg AG, Corey L, Ashley RL, Leong WP, Straus SE. A prospective study of new infections with herpes simplex virus type 1 and type 2. Chiron HSV Vaccine Study Group. *N Engl J Med* 1999;341:1432-38.
26. The UK Collaborative Group for HIV and STI Surveillance. Testing Times. HIV and Other Sexually Transmitted Infections in the United Kingdom: 2007. London, Health Protection Agency, Centre for Infections, 2007.
27. Sexually Transmitted Disease Surveillance 2007. Division of STD Prevention, Department of Health and Human Services, Centers for Disease Control and Prevention, Atlanta, Georgia, 2008.
28. Roberts CM, Pfister JR, Spear SJ. Increasing proportion of herpes simplex virus type 1 as a cause of genital herpes infection in college students. *Sex Transm Dis* 2003;30:797-800.
29. Ryder N, Jin F, McNulty A, Grulich AE, Donovan B. Increasing role of herpes simplex virus type 1 in first-episode anogenital herpes in heterosexual women and younger men who have sex with men, 1992-2006. *Sex Transm Infect* 2009;85:416-19.
30. Lowhagen GB, Tunback P, Andersson K, Bergstrom T, Johannisson G. First episodes of genital herpes in a Swedish STD population: A study of epidemi-

ology and transmission by the use of herpes simplex virus (HSV) typing and specific serology. *Sex Transm Infect* 2000;76:179-82.

Received: April 13, 2010
Accepted: November 8, 2010

RÉSUMÉ

Objectifs : En raison de la disponibilité croissante du dépistage sérologique spécifique de type au Canada et du rôle de l'infection génitale à HSV-2 dans la transmission continue du VIH, il est important, pour les cliniciens et les praticiens de la santé publique, de connaître l'épidémiologie régionale des infections par le virus herpès simplex génital (HSV). À l'aide de données de laboratoire centralisées, nous décrivons les tendances des identifications virales de l'herpès génital en Colombie-Britannique et nous évaluons l'utilité de ces données pour la surveillance continue de l'herpès dans la population.

Méthode : Les dossiers d'identifications virales (1997-2005) sont extraits de la base de données *Provincial Public Health Microbiology & Reference Laboratory.* Notre classification des infections (génitales ou d'autres sites) est basée sur le site de prélèvement indiqué en dossier. Nous avons effectué une analyse descriptive des tendances au fil du temps et calculé les probabilités d'infection à HSV-1 chez les sujets atteints d'herpès génital.

Résultats : Sur 48 183 identifications virales, 56,8 % étaient génitales, 10 % étaient péribuccales, et 9,1 % étaient cutanées; dans 22,9 % des cas, le site n'était pas indiqué. Parmi les identifications génitales, l'infection à HSV-1 était plus probable chez les femmes, dans les groupes d'âge les plus jeunes et durant les périodes plus tardives. La proportion d'herpès génital dû à HSV-1 a augmenté au fil du temps en Colombie-Britannique, passant de 31,4 % à 42,8 %.

Conclusion : Notre analyse des données de laboratoire en population montre que la proportion de l'herpès génital dû à HSV-1 s'accroît au fil du temps en Colombie-Britannique, tout particulièrement chez les femmes et les jeunes, ce qui a des conséquences pour la pratique clinique, y compris l'interprétation de la sérologie spécifique de type. Les données provinciales des identifications virales sont utiles pour surveiller la distribution des infections génitales à HSV-1 et à HSV-2 au fil du temps. En améliorant la consignation du site de prélèvement dans les dossiers cliniques, on rehausserait l'utilité de ces données.

Mots clés : herpès; herpès génital; épidémiologie; Canada