

Oral Cancer: Just the Facts

Denise M. Laronde, RDH, MSc; T. Greg Hislop, MDCM;
J. Mark Elwood, MD, MBA, FRCPC; Miriam P. Rosin, BSc, PhD

Auteure-ressource

Mme Laronde
Courriel : dlaronde@bccancer.bc.ca



SOMMAIRE

Le dépistage du cancer de la bouche devrait faire partie intégrante de la routine du clinicien. Le présent article examine les faits connus sur le cancer de la bouche qui ont rapport au dépistage. La pertinence de certains faits dans un cabinet dentaire particulier variera en fonction de la clientèle.

Pour les citations, la version définitive de cet article est la version électronique : www.cda-adc.ca/jcda/vol-74/issue-3/269.html

Oral cancer, although prevalent worldwide, is very common in some countries such as India, Pakistan and Taiwan, and in some areas of France.¹ Although less frequent in Canada, oral cancer was diagnosed in 3,200 people and responsible for 1,100 deaths in 2007.² To put this into perspective, currently in Canada, more cases of oral cancer are diagnosed in a year than cervical or ovarian cancer, and more deaths occur from oral cancer than from melanoma or cervical cancer.² With growing immigration from high-risk areas such as India, the number of cases of oral cancer will increase.

Since nasopharyngeal cancers are routinely included in oral cancer statistics, we estimate that dental professionals could potentially detect about 2,700 (84%) of these 3,200 cases. The distribution of the sites of oral and related cancers that can be detected by dental screening is shown in **Table 1**. For convenience, we refer to this group of 2,700 cancers as “oral cancers” in this paper.

Oral cancer is often diagnosed at an advanced stage, and the overall survival rate 5 years after diagnosis is about 62% for all sites combined² and 65% for the sites shown in **Table 1**. Survival has improved a little over the last several decades, perhaps because of a reduction in smoking.² Like

most cancers, the incidence of oral cancers rises with age (**Fig. 1**). Patients over 60 years of age are at the greatest risk; however, the incidence of oral cancer has increased in patients under 40 years of age, perhaps because of changing risk factors. About 6% of oral cancers occur among persons under 40 years of age. Although the overall ratio of males to females with oral cancer in Canada is 2:1, the ratio is almost 1:1 in patients under 40. The overall incidence in Canada is about 12 per 100,000 per year in men and 5 per 100,000 in women. Rates are somewhat higher in eastern than in western Canada.

What Can Be Done About This?

Early diagnosis of oral cancer through screening and early detection is critical. Survival is much better when the lesion is diagnosed at an early stage. For example, the 5-year survival rate for tongue cancer in the United States is 71% for stage 1 disease and 37% for late-stage disease.³ More than 40% of oral cancers are diagnosed at a late stage.³

Who Should Be Screened?

The first step in screening for oral cancer involves reviewing the patient’s medical history to assess various risk factors, including a past history of disease, specific risk-related habits,

Table 1 Distribution and survival rates for oral cancers amenable to dental screening and early detection by subsite: Canada, 2007

Subsite of cancer	Number of new cases (n = 2,690)	% distribution	5-year survival rate (mean 65)
Lip	341	13	94
Tongue	740	27	53
Gum and other mouth	509	19	60
Floor of mouth	281	10	53
Salivary gland	348	13	74
Tonsil	379	14	50
Oropharynx	93	3	50

Estimates based on data from the United States³ and Canada.²

medications and reasons for hospitalizations. A number of factors are associated with an increased risk of oral cancer (Table 2).^{1,5-8}

There is no single cause of oral cancer. It results from a variety of factors that operate over time and is dependent on each person's unique response to these factors. However, patients without obvious risk factors can develop oral cancer and premalignant lesions. The 2 most important modifiable risk factors for oral cancer are tobacco and alcohol consumption. Up to 75% of oral cancers may be attributed to exposure to tobacco or alcohol.^{6,9}

Tobacco

Tobacco smoke contains dozens of known carcinogens. The risk of oral cancer and premalignant lesions increases with the amount of tobacco consumed and the duration of tobacco use. This increased risk holds for all types and uses of tobacco, whether it is smoked as a cigarette, cigar, pipe or bidi (a small, hand-rolled cigarette commonly used in Asia), or used smokeless as a chew, plug or snuff. Stopping smoking reduces the risk of oral cancer and premalignant lesions, although it may take 10 to 20 years for a former smoker's risk to reduce to that of a nonsmoker.¹

Patients from South Asian countries may also chew betel quid or paan, a common habit in their culture. Betel quid is a carcinogenic complex mixture of plant components that frequently contains tobacco. Since immigrants from these countries tend to retain the level of oral cancer risk charac-

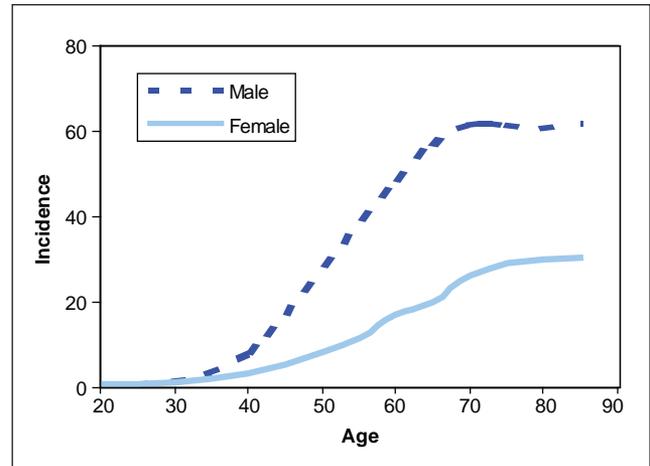


Figure 1: Annual incidence rate for new oral cancers by age and sex (number per 100,000 population). From U.S. SEER data.⁴

teristic of their country of origin for some time, they should be questioned about their consumption of betel quid.

Alcohol

Alcohol consumption is also a strong risk factor for oral cancer and premalignant lesions. The risk increases with increased consumption and duration of use of alcohol. Typically, one 8-ounce glass of beer, one 4-ounce glass of wine and 1 ounce of spirits have equal amounts of alcohol.¹⁰ In many studies,^{10,11} heavy drinking is defined as consumption of more than 14 to 21 drinks per week. Again, the risk of oral cancer decreases when alcohol is no longer consumed, but it takes many years for a drinker's risk to reduce to that of someone who has never been a drinker.¹

Tobacco and alcohol consumption work together synergistically, increasing the risk of oral cancer to more than 30 times that of those who do not smoke or drink.^{1,5,6} Heavy drinkers and smokers are also more likely to be diagnosed with late-stage disease. Ceasing to use tobacco and alcohol greatly reduces the risk of developing oral cancer and premalignant lesions.

Human Papilloma Virus

Having human papilloma virus (HPV) is a strong risk factor for oral cancers, especially when the lingual and palatine tonsils, the soft palate and the base of the tongue are involved. Of the more than 120 types of HPV, only a few are high-risk factors for oral cancer, primarily HPV-16 and HPV-18. Over 90% of HPV-positive oral cancers are HPV-16 positive.¹ Since risk factors for HPV infection include having a large number of sexual partners and first intercourse at a younger age, changing sexual practices in our society may increase the effect of HPV infection on the development of oral cancers and premalignant lesions, especially in younger adults.¹² The combination of smoking and HPV infection and of alcohol and HPV infection may have an additive effect.¹³

Table 2 Risk factors for oral cancers

Very strong risk factors (> 10-fold increased risk)
Increased age ¹
Using tobacco and alcohol, especially combined use (risks for heavy smokers and drinkers are increased more than 30-fold) ^{1,5,6}
Using smokeless tobacco, including snuff and chewing tobacco ⁶
Chewing betel quid, areca nut and paan ^{1,6}
Being immunologically compromised (e.g., after bone-marrow transplantation) ⁶⁻⁸
Strong risk factors (4- to 10-fold increased risk)
Smoking cigarettes ^{5,6}
Drinking alcohol ^{5,6}
Having a human papilloma virus infection, especially type 16 ^{1,5}
Moderate risk factors (≤ 4-fold increased risk)
Being male ¹
Smoking pipes and cigars ^{1,5,6}
Smoking marijuana ¹
Being exposed to environmental tobacco smoke ⁶
Having low fruit and vegetable intake ^{1,5,6}

Diet and Vitamins

A diet rich in fruits and vegetables, particularly fruit, reduces the risk of oral cancer and premalignant lesions.^{5,14} Several studies^{1,5} have shown that higher levels of vitamin C or carotene consumption reduce the risk of oral cancer. The potentially increased risk associated with meat consumption is less clear.¹ Results of intervention studies involving dietary change or dietary supplements have shown no clear evidence of benefit.¹

Other Issues

Studies of the role of marijuana in oral cancer are scarce. Marijuana smoke contains many of the same carcinogens found in tobacco smoke¹⁵ and has 4 times the tar burden.¹⁶

Studies⁶⁻⁸ have reported that immunosuppressed patients (due to medications, bone-marrow transplants or disease) have an increased risk of oral cancer and premalignant lesions. One study⁸ reported an 11-fold increased risk of oral cancer for bone-marrow transplant patients. This risk increases with time, after transplantation.

Eating spicy or hot foods, using mouthwash, or having poor oral hygiene, missing or broken teeth, or dentures do not seem to cause oral cancer. Reports^{1,5} of higher risks associated with infrequent tooth-brushing and with missing teeth may be due to other factors such as smoking. Reported increased risks in some occupational groups, such as rubber workers and cooks, may also be due to such factors.^{1,5,6}

Common Fallacies about Oral Cancer

Oral cancer happens only to smokers and alcohol drinkers.

About 25% of oral cancers occur in people with no history of tobacco or alcohol use.

Oral cancer occurs only in the elderly.

Although the risk of oral cancer increases with age, it can occur at any age and seems to be increasing in patients less than 40 years of age.

The risk of oral cancer does not decrease once a patient quits smoking.

The risk of oral cancer is reduced for former smokers and approaches that of a nonsmoker after many years.

It is not my job to question patients about their tobacco and alcohol use.

A comprehensive health history should include questions about patients' tobacco and alcohol use, including the duration of use and amount consumed. Talking to your patients about tobacco and alcohol cessation may play an important role in the prevention of disease.

I can quickly screen for oral cancer as I complete other parts of my treatment.

Screening for oral cancer is a 3-part process: the review of the health history, the extraoral examination and the intraoral examination. cursory looks are not sufficient because areas such as the posterior lingual vestibule, the soft palate, tonsils, the floor of the mouth, and the posterior lateral and ventral tongue can easily be missed.

What Risk Information Should Be Recorded and Updated in the Patient's Chart?

Questions about tobacco and alcohol consumption are a vital part of the screening process and should be recorded in the patient's chart. Frequency (current and past use), and amount and duration of use should be recorded and updated regularly. This information may indicate the need to counsel

patients about tobacco and alcohol cessation. Finally, screening should be done regularly because oral cancer can occur in patients without any apparent risk factors. ♦

THE AUTHORS

Acknowledgments: Ms. Laronde is supported by a Michael Smith Foundation for Health Research/BC Cancer Foundation Senior Trainee Award.

Ms. Laronde is a dental hygienist and PhD candidate, applied science, Simon Fraser University and BC Oral Cancer Prevention Program, BC Cancer Agency/Cancer Research Centre, Vancouver, British Columbia.

Dr. Hislop is an epidemiologist and clinical professor, medicine, University of British Columbia and senior scientist in the cancer control research department, BC Cancer Agency/Cancer Research Centre, Vancouver, British Columbia.

Dr. Elwood is an epidemiologist and clinical professor, medicine, University of British Columbia and vice-president, Family and Community Oncology, BC Cancer Agency/Cancer Research Centre, Vancouver, British Columbia.

Dr. Rosin is a translational scientist and professor, applied science, Simon Fraser University, medicine, University of British Columbia and director, BC Oral Cancer Prevention Program, BC Cancer Agency/Cancer Research Centre, Vancouver, British Columbia.

Correspondence to: Ms. Denise M. Laronde, BC Oral Cancer Prevention Program, BC Cancer Agency/Cancer Control Research Centre, 675 West 10th Ave., Vancouver, BC V5Z 1L3.

The authors have no declared financial interests.

This article has been peer reviewed.

References

1. Mayne S, Morse D, Winn D. Cancers of the oral cavity and pharynx. In: Schottenfeld D, Fraumeni J, Jr, editors. Cancer epidemiology and prevention. 3rd ed. New York: Oxford University Press; 2006. p. 674–96.
2. Canadian Cancer Society, National Cancer Institute of Canada. Canadian cancer statistics 2007. Toronto (ON); 2007. Available: www.cancer.ca/vgn/images/portal/cit_86751114/36/15/1816216925cw_2007stats_en.pdf (accessed 2008 Mar 7).
3. Piccirillo J, Costas I, Reichman M. Cancers of the head and neck. In: Ries LA, Young JL, Keel GE, Eisner MP, Lin YD, Horner MJ, editors. SEER survival monograph: cancer survival among adults: U.S. SEER Program, 1988-2001, Patient and tumor characteristics. NIH Pub. No. 07-6215. Bethesda (MD): National Cancer Institute, SEER Program; 2007. p. 7–22.
4. SEER (Surveillance Epidemiology and End Result). U.S. National Institutes of Health. National Cancer Institute; 2007. Available from URL: www.seer.cancer.gov (accessed 2008 Feb 16).
5. Mucci L, Adami H. Oral and pharyngeal cancer. In: Adami H, Hunter D, Trichopoulos D, editors. Textbook of cancer epidemiology. New York: Oxford University Press; 2002. p. 115–32.
6. Quon H, Hershock D, Feldman M, Sewell D, Weber R. Cancer of the head and neck. In: Abeloff M, Armitage J, Niederhuber J, Kastan M, McKenna W, editors. Clinical oncology. 3rd ed. Orlando: Churchill Livingstone, an imprint of Elsevier; 2004. p. 1499–500.
7. Bhatia S, Louie AD, Bhatia R, O'Donnell MR, Fung H, Kashyap A, and others. Solid cancers after bone marrow transplantation. *J Clin Oncol* 2001; 19(2):464–71.
8. Curtis RE, Rowlings PA, Deeg HJ, Shriner DA, Socie G, Travis LB, and others. Solid cancers after bone marrow transplantation. *N Engl J Med* 1997; 336(13):897–904.
9. La Vecchia C, Tavani A, Franceschi S, Levi F, Corrao G, Negri E. Epidemiology and prevention of oral cancer. *Oral Oncology* 1997; 33(5):302–12.
10. Franceschi S, Levi F, Dal Maso L, Talamini R, Conti E, Negri E, and other. Cessation of alcohol drinking and risk of cancer of the oral cavity and pharynx. *Int J Cancer* 2000; 85(6):787–9.
11. Hashibe M, Brennan P, Benhamou S, Castellsague X, Chen C, Curado M, and others. Alcohol drinking in never users of tobacco, cigarette smoking in never drinkers, and the risk of head and neck cancer: pooled analysis in the

International Head and Neck Cancer Epidemiology Consortium. *J Natl Cancer Inst* 2007; 99(10):777–89.

12. D'Souza G, Kreimer AR, Viscidi R, Pawlita M, Fakhry C, Koch WM, and others. Case-control study of human papillomavirus and oropharyngeal cancer. *New Engl J Med* 2007; 356(19):1944–56.

13. Smith EM, Ritchie JM, Summersgill KF, Hoffman HT, Wang DH, Haugen TH, and other. Human papillomavirus in oral exfoliated cells and risk of head and neck cancer. *J Natl Cancer Inst* 2004; 96(6):449–55.

14. World Cancer Research Fund International and American Institute for Cancer Research. Mouth, pharynx, and larynx. In: Food, nutrition, physical activity and the prevention of cancer: a global perspective. Washington (DC): AICR; 2007. p. 245–9.

15. Hashibe M, Ford DE, Zhang ZF. Marijuana smoking and head and neck cancer. *J Clin Pharmacol* 2002; 42(11 Suppl):1035–75.

16. Lingen M, Sturgis EM, Kies MS. Squamous cell carcinoma of the head and neck in nonsmokers: clinical and biologic characteristics and implications for management. *Curr Opin Oncol* 2001; 13(3):176–82.