

## Section 6

# ORAL INFECTIONS

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### 6.1 Oral infections and benign lesions

The mucosal lining of the mouth can serve as a site and portal for oral infections. These infections include oral candidiasis, herpes labialis, herpetic gingivostomatitis, oral papillomas, recurrent aphthous ulcers, oral lichen planus, and angular cheilitis. The prevalence of oral infections varies by type of infection. Persons who are immunocompromised due to diseases or disorders such as HIV/AIDS are more susceptible to most oral infections. The following is a brief description of some of the more common oral infections.

#### Infections

*Oral candidiasis* is a fungal infection that manifests as white plaque on the palate and oral mucosal regions and may adhere to soft and hard tissue surfaces such as dentures (Patton & van der Horst, 1999; Kleinman et al., 1991). Oral candidiasis may be a manifestation of HIV infection, chemotherapy or radiotherapy to the head and neck, xerostomia, medications, and other systemic conditions (US DHHS, 2000). A prevalence of between 43% and 93% has been reported among HIV-infected patients (Samaranayake, 1992). In addition, there is a significant association between oral candidiasis and Sjogren's syndrome (Rhodus et al., 1997). Diagnosis is based on clinical appearance and laboratory identification of hyphae. Long-term morbidity includes impairment in nutritional intake due to discomfort, and bleeding in oral tissues.

*Herpes labialis* is caused by herpes simplex virus 1 (HSV-1). Lesions occur on lips, palate, and gingiva subsequent to an earlier primary HSV-1 infection (see herpetic gingivostomatitis below). Oral mucocutaneous herpetic ulcers may persist for prolonged periods. The prevalence of recurrent herpes lesions has been estimated between 15% and 40% (Scully, 1989). Less frequently, similar lesions may be due to a genitourinary HSV-2 infection.

*Herpetic gingivostomatitis* is a common manifestation of primary herpes simplex infection. The main complications include dehydration, oral lesions, malaise, and fever. In most cases, individuals must have an active lesion to transmit the virus. Prior to the development of lesions, patients may remain in a state of primary infection, where symptoms are limited to malaise and fever, without presentation of clinical lesions. The oral lesions usually last one or two weeks and are limited to discrete areas of ulceration (Amir et al., 1999; Matusow et al., 1992).

*Oral papillomas*, caused by the human papillomavirus (HPV), appear as single or multiple nodules. Diagnosis can be made by visual examination of the lesion, biopsy, or HPV strain typing. Immune suppression increases the occurrence of lesions. Treatment consists of surgical removal of lesions. However, lesions may reappear in the presence of latent viral particles (Patton & van der Horst, 1999).

## Benign lesions

*Recurrent aphthous ulcers* (RAU) are of unknown cause. The most common form of RAU usually lasts up to two weeks and is recurrent. The prevalence of RAU varies from 5% to 25% (US DHHS, 2000). The ulcers are painful and make eating and swallowing difficult. Risk factors for the recurrence of lesions include emotional stress, socioeconomic status, smoking, nutritional deficiencies, sensitivity to food dyes, and denture use.

*Oral lichen planus* occurs on the skin as well as oral mucosa. Oral lesions consist of lacy white and red plaque formations. Most cases of oral lichen planus are of unknown origin; however, some cases have been shown to be associated with hepatitis C infection (Kleinman et al., 1991; Lodi et al., 2000).

*Angular cheilitis* is a reactive process that causes inflammation of the lips, affecting the corners of the mouth. Common causes of angular cheilitis include infections, nutritional deficiencies, allergies to oral products and cosmetics, chapping from cold environments, dermatitis, anti-acne medication, and immunosuppression from AIDS. Secondary complications include bacterial infections and fungal infections such as candidiasis that may exacerbate symptoms (Rogers & Bekic, 1997; Burt & Eklund, 1999).

### SOURCE OF DATA

Analyses reported here are based on the Third National Health and Nutrition Examination Survey (NHANES III) 1988-1994, National Center for Health Statistics, Centers for Disease Control and Prevention. The dental exam included a component to determine the presence of oral lesions and infections. Data analyses reported here come from the dental exam and reflect current cases of infection at the time of the exam. There were insufficient data to analyze candidiasis, herpetic gingivostomatitis, and lichen planus.

- Angular cheilitis was experienced by 2.5% of the adult U.S. population, 5.7% experienced herpes labialis, 1.9% experienced papillomas/ warts, and 3.0% experienced recurrent aphthous ulcerations. These infections differ greatly in their demographic distribution. Small numbers hinder analyses of significant differences.

- **Differences by race/ethnicity (Figure 6.1.1)**

- A greater percentage of both non-Hispanic whites and Mexican Americans experienced angular cheilitis than non-Hispanic blacks.
- A greater percentage of both Mexican Americans and non-Hispanic whites experienced herpes labialis than non-Hispanic blacks.
- A greater percentage of Mexican Americans than non-Hispanic blacks experienced recurrent aphthous ulcerations.

- **Differences by education (Figure 6.1.2)**

- The percentage of persons with either angular cheilitis or papillomas decreases with increasing education.

- **Differences by gender and federal poverty level**

- No differences were observed.

*Bullets reference data that can be found in Table 6.1.1.*

### REFERENCES

- Amir J, Harel L, Smetana Z, Varsano I. The natural history of primary herpes simplex type 1 gingivostomatitis. *Pediatr Derm* 1999;16:259-63.
- Burt BA, Eklund SA. *Dentistry, Dental Practice, and the Community*, 5th ed. Philadelphia, PA: WB Saunders, 1999.
- Kleinman DV, Swango PA, Niessen LC. Epidemiologic studies of oral mucosal conditions. *Community Dent Oral Epidemiol* 1991;19:129-40.
- Lodi G, Carrozzo M, Harris K, Piatelly A, Teo CG, Porter SR. Hepatitis C virus associated oral lichen planus. *J Oral Pathol Med* 2000;29(1):39-42.
- Matusow RJ. Acute primary herpetic gingivostomatitis in non-immunocompromised adults and HSV-1 isolation technique. *Compendium* 1992;13:662-66.

Patton LL, van der Horst C. Oral infections and other manifestations of HIV disease. *Oral Infect* 1999;13:879-900.

Rhodus NL, Bloomquist C, Liljemark W, Bereuter J. Prevalence, density, and manifestations of oral *Candida albicans* in patients with Sjogren's syndrome. *J Otolaryngol* 1997;26:300-5.

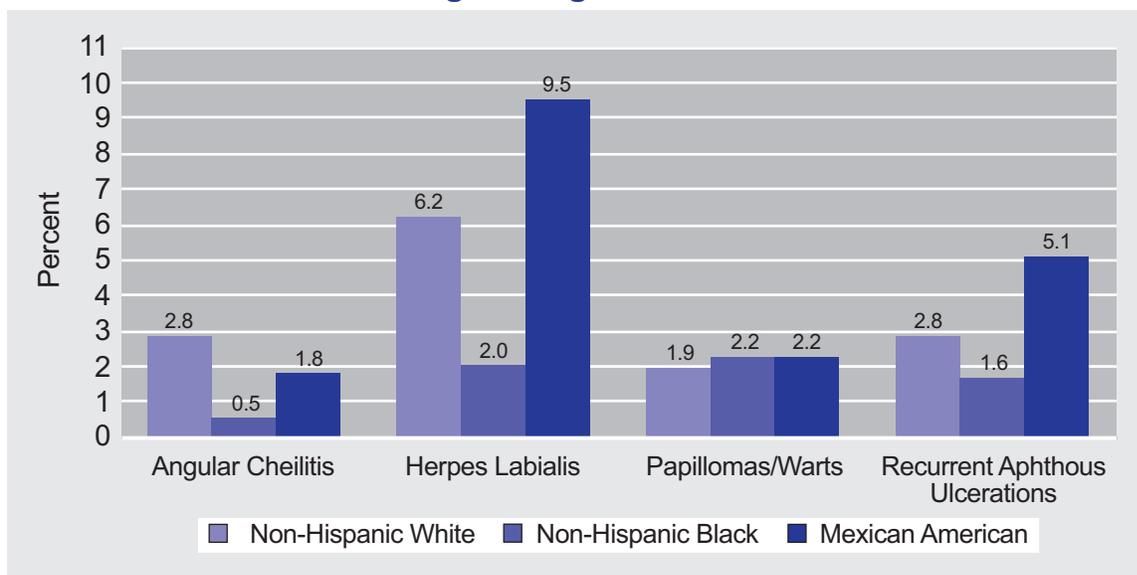
Rogers RS, Bekic M. Diseases of the lips. *Semin Cutan Med Surg* 1997;16(4):328-36.

Samaranayake LP. Oral mycoses in HIV infection. *Oral Surg Oral Med Oral Pathol* 1992;73(2):171-80.

Scully C. Herpes simplex virus (HSV). In: Millard HD, Mason DK, eds. *World Workshop on Oral Medicine*, 1988 Jun 19-25. Chicago: Year Book Medical Publishers, 1989.

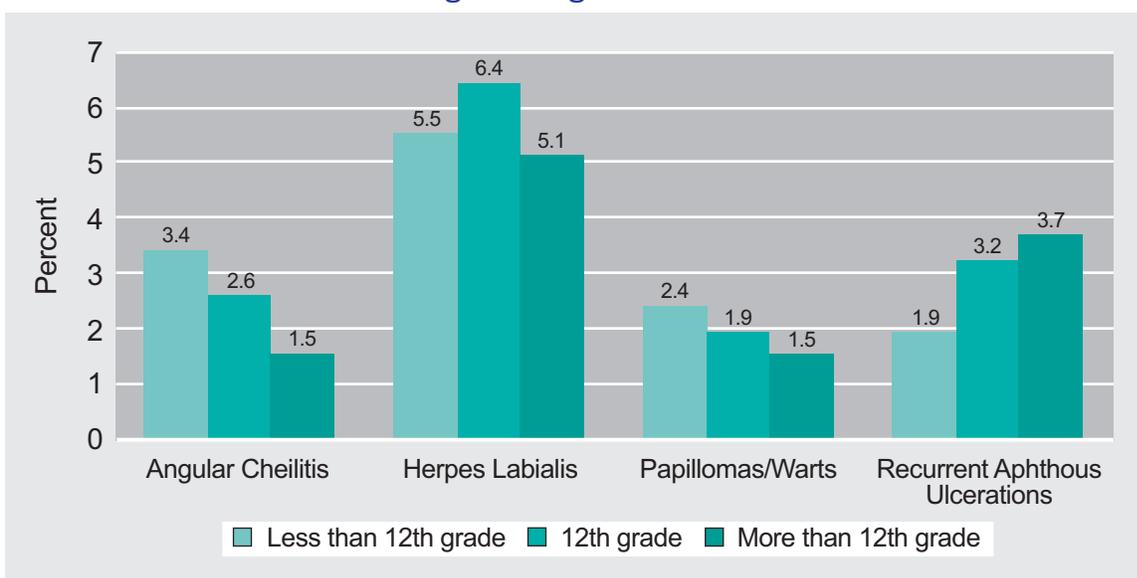
U.S. Department of Health and Human Services. *Oral Health in America: A Report of the Surgeon General*. Rockville, MD: U.S. Department of Health and Human Services, National Institute of Dental and Craniofacial Research, National Institutes of Health, 2000.

**Figure 6.1.1. Prevalence of oral infections and benign lesions by race/ethnicity among adults aged 18 and older**



Data source: The Third National Health and Nutrition Examination Survey (NHANES III) 1988-1994, National Center for Health Statistics, Centers for Disease Control and Prevention.

**Figure 6.1.2. Prevalence of oral infections and benign lesions by education among adults aged 18 and older**



Data source: The Third National Health and Nutrition Examination Survey (NHANES III) 1988-1994, National Center for Health Statistics, Centers for Disease Control and Prevention.

