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ORIGINAL ARTICLE

The role of *Candida albicans* hyphae and *Lactobacillus* in denture-related stomatitis

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Abstract Denture-related stomatitis (DRS) is still a dilemma in removable prosthodontics. The aim of this study was to investigate the relationship of DRS with the presence of *Candida albicans* hyphae and *Lactobacillus*. A total of 91 patients wearing maxillary and mandibular complete dentures were included in the present study and tested mycologically as well as bacteriologically. A statistically significant association of DRS was found with denture age (p=0.003) and continuous denture wearing (p=0.015). Presence of *C. albicans* hyphae was shown to be significantly higher in DRS cases (p<0.01), and there was a statistically significant positive correlation between presence of hyphae and *C. albicans* (p<0.01). Another interesting finding was that DRS patients showed higher *Lactobacillus* counts in their saliva (p=0.04), as well as in the palate (p=0.028). *C. albicans* is an important factor in the development of DRS. Hyphae seem to facilitate the rise of *C. albicans* counts and be related to the inflammatory response of the tissues. *Lactobacillus* seems to play an important role in the presence of DRS, as well. In agreement with many other studies, the results of this study confirm the importance of denture age and continuous denture wearing in the development of DRS.

Keywords Denture stomatitis · Hyphae · *Candida* · *Lactobacillus* · Denture age · Continuous denture wearing

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Introduction

Denture-related stomatitis (DRS) is a common inflammatory process that mainly involves the palatal mucosa when it is covered by complete dentures [10, 33, 36, 46]. The etiology appears to be multifactorial; old age and the associated decline of the immune defense, systemic diseases, continuous denture wearing, increased age of denture, and lack of denture cleanliness resulting in the accumulation of plaque on the denture have all been proposed as predisposing factors [10,12, 21, 33, 36, 43, 45, 46].

Besides the above-mentioned predisposing factors, several studies have demonstrated an association between *Candida albicans* and DRS [5, 6, 8, 9, 18, 25, 35, 38, 41]. In comparison with other species of *Candida* such as *Candida tropicalis*, *Candida glabrata*, *Candida parapsilosis*, and *Candida krusei*, *C. albicans* was shown to have a very high occurrence in the oral cavity [1, 3, 27, 49, 50]; thus, the majority of candidiasis still seems to be caused by *C. albicans* [16, 31, 32, 48]. According to the results of many

studies showing a high incidence of *C. albicans* in the saliva of DRS patients in comparison to the control groups, it has been speculated that its presence is an important factor in the development of the disease. The higher prevalence of *C. albicans* was explained by the higher capability to adhere to mucosal surfaces, which is considered as the first step in the pathogenesis of oral candidiasis [19]. *C. albicans* forms complex biofilms consisting of a basal blastospore layer covered by a thick matrix composed of extracellular material and hyphal elements, which is thicker than less commonly pathogenic species, including *C. parapsilosis*, *C. glabrata*, and *C. tropicalis* [26]. *Candida*, which is primarily found in the plaque on the tissue surface of the denture rather than on or in the inflamed mucosa, produce toxins which act as chemical irritants [18]. However, an important amount of subjects with *C. albicans* in their saliva had no symptoms of DRS. Consequently, there must be factors that are important in the development of the disease besides the presence of *C. albicans*. A striking feature of *C. albicans* biology is its ability to grow in a variety of morphological forms. These range from unicellular yeast to true hyphae. Cellular differences between pseudohyphae and hyphae are revealed by septin localization [30]. This property of *C. albicans* could promote tissue penetration during the early stages of infection [48]. Hyphae have been observed to adhere to and invade host tissues more readily than the yeast form, suggesting that filamentous growth may contribute to the virulence of this major human pathogen [29]. The property of *Candida* species to adhere to mucosal surfaces is reported to be enhanced especially by the transition from blastospores to hyphae [2] and consequently contributing to the virulence of this pathogen [29].

There are also studies revealing the importance of mutans streptococci and lactobacilli in the development of DRS [2, 7]. It is shown that subjects with a healthy lifestyle generally have better oral hygiene and thus lower *Lactobacillus* counts and less occurring denture stomatitis [45].

The purpose of this study has been to assess the importance of lactobacilli and the hyphae form of *C. albicans* in the etiology of DRS.

Materials and methods

In the period between 2004 and 2007, edentulous patients attending the Department of Removable Prosthodontics of Istanbul University, for a new denture, were examined and interviewed. Out of 108 patients who were wearing complete dentures longer than 6 months and showing various levels of DRS symptoms, a total of 71 voluntary patients were selected to include in our study group. Additionally, 20 subjects without any inflammatory occurrence on the palate were chosen as the control group. The criterion in the selection of the control group was, as in the study group, that the patient wore upper and lower complete dentures for a period longer than 6 months. The protocol of the whole project was reviewed by the Ethical Committee of Istanbul University, and each subject signed an informed consent form. The patients' age, gender, existence of a denture actually being used, denture age, frequency, and method of denture cleaning and dental history were recorded. Clinical examination was performed by the same investigator for standardization reasons. The type of dentures, presence, and localization of denture induced stomatitis.

In the case of DRS, the erythema was scored by using Newton's classification index [37]:

1. Slight inflammation (localized slight hyperemia)
2. Moderate inflammation (generalized erythema)
3. Severe inflammation (diffuse and papillary hyperplasia)

For the recording of the denture cleanliness, a subjective denture hygiene index was used to score the plaque at the intaglio surface in three groups.

1. Good: no or very little plaque
2. Fair: less than half of the denture base covered by plaque
3. Poor: more than half of the denture base covered by plaque

Collecting samples

In order to provide standardization for collected samples, the overall investigation was carried out at midmorning and at least 2 h after eating, drinking, or any hygiene procedure. From each patient, DRS cases as well as the control group, oral swab samples were collected from a triangular area of the palate and the tissue surface of the upper dentures. For direct microscopic examination and detection of *C. albicans* hyphae, scraped specimen from the inflamed tissue of the palate was collected by using a sterile lancet.

Microbiological procedures

Direct microscopical examination

The calcofluor white stain was used for the direct microscopical examination of saliva and smear samples [28]. The slides were examined with a fluorescent microscope (Olympus BH2-RFCA, Japan) for the detection of hyphae and budding yeast cells.

Culture procedures

Saliva samples were mixed well, and 1 ml was inoculated onto Sabouraud dextrose agar (SDA) supplemented with

chloramphenicol (50 mg/l) and penicillin (50 mg/l) for isolation of yeasts. For the isolation of *Lactobacillus* species, one loop (0.01 μl) of the same sample was plated onto Rogosa agar [52]. Swab samples were plated directly on SDA and Rogosa agar. SDA plates were incubated at 37°C in aerobic conditions for 24 h. Rogosa agar plates were incubated at 37°C in anaerobic conditions for 24 h [22, 52].

Identification of yeast species

The yeasts grown from saliva were expressed as colony forming units per milliliter (cfu/ml). The number of yeast colonies grown from swab specimens was evaluated as follows:

1. Light growth: Colonies are seen only at the first part of the agar plate.
2. Medium growth: Colonies are seen at the first and second part of the agar plate.
3. Heavy growth: Colonies are seen at the first, second, and third part of the agar plate.
4. Very heavy growth: Colonies are seen at all parts of the agar plate.

Morphologically, different colonies were picked and reinoculated onto SDA for purity. Identification of yeast isolates to the species level were based on standard methods such as chlamydospore production on corn meal agar supplemented with 1% Tween 80 and carbohydrate assimilation pattern by using the API ID 32°C (Biomerieux, Marcy I'Etoile, France). In addition, growth at 45°C, colony morphology, and chlamydoconidia production on Staib agar were used for discriminating *C. albicans* and *Candida dubliniensis*. *C. albicans* ATCC 10231 and *C. dubliniensis* CD36I were used as control strains [22].

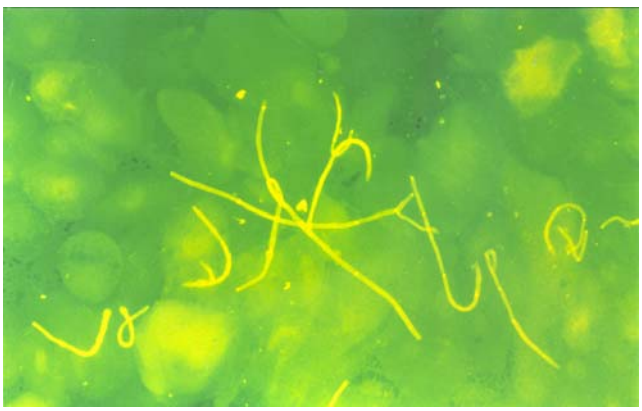

Fig. 1 Hyphae from a patient's smear sample (×200, prepared with calcofluor white)

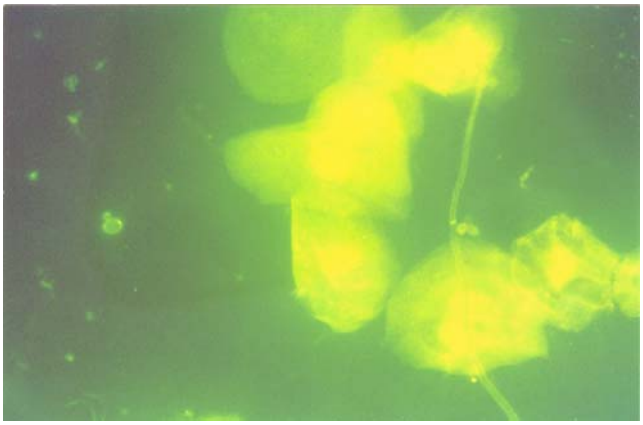

Fig. 2 Hyphae and yeast cells from a patient's smear sample (×400, prepared with calcofluor white)

Identification of *Lactobacillus* species

Rogosa agar was used as a selective media for the isolation of *Lactobacillus* species. All different colonies grown on Rogosa agar plates were examined by Gram stain. Gram-positive *Bacillus* strains were replated into lactobacilli MRS broth tubes supplemented with glucose, maltose, mannitole, or saccharose. Growth in this broth and esculin hydrolysis was used for identification [52].

Whitney–Mann *U* and Chi-square tests were used for statistical analysis of the results.

Table 1 The differences of the patient age, denture age, and *Candida albicans* counts in saliva, candidal growth on the palate and the tissue surface of the denture base, hyphae growth in saliva, on the palate, and continuous denture wearing habit between the patients with and without DRS is shown

	Patients		*p*
	With DRS (*n*=71)	Without DRS (*n*=20)	
Patient age (year)	62.17±9.4	69.95±9.8	0.006
Denture age (year)	9.5±8.2	4.7±6.6	0.003
Candida count in saliva (cfu/ml)	2,281.6± 3,803.6	305.3± 674.9	0.001
Candida growth in palate (%)	66.2	25	0.002
Candida growth in denture (%)	81.7	30	0.000
Hyphea growth in saliva (%)	95	5	0.001
Hyphea growth in palate (%)	100	0	0.001
Lactobacillus growth in saliva (%)	87.3	65	0.028
Continuous denture wearing (%)	85.9	60	0.015

Table 2 The effect of hyphae presence on *Candida albicans* count and *Lactobacillus* growth is shown

	Hyphae									
	In saliva					On the palate				
	Presence (n=24)		Absence (n=67)		p	Presence (n=13)		Absence (n=78)		p
	Mean	SD	Mean	SD		Mean	SD	Mean	SD	
Candida albicans count (cfu/ml)	5,308.5	5,184.7	607.4	1,055.8	0.001	6,959.6	6,011.4	995.2	1,810.1	0.001
Lactobacillus growth (%)	95.8		77.6		0.036	92.3		78.2		0.218

Results

The microscopic images of *C. albicans* hyphae and yeast cells from a patient's smear sample were shown in Figs. 1 and 2.

The differences of the patient age, denture age, and *C. albicans* counts in saliva, candidal growth on the palate and the tissue surface of the denture base, *C. albicans* hyphae growth in saliva, on the palate, and continuous denture wearing habit between the patients with and without DRS were shown in Table 1. Chi-square and Mann–Whitney *U* tests were used to analyze the differences.

The subjects in which *C. albicans* hyphae were observed in their saliva and on the palate were analyzed for the *C. albicans* counts using Mann–Whitney *U* test and for the *Lactobacillus* growth using Chi-square test (Table 2). The *C. albicans* counts were found statistically significant higher in the subjects with hyphae in their saliva or on the palate, compared to the subjects without hyphae.

However, *Lactobacillus* growth was found statistically higher only in subjects with *C. albicans* hyphae in their saliva. For the subjects with *C. albicans* hyphae growth on the palate, there was no statistically significant difference.

Although there was a noticeable increase in *C. albicans* counts in saliva due to the clinical intensity (Newton classification) of DRS, no statistical significant differences were found. In patients with papillary hyperplasia (Newton type 3), the *Lactobacillus* incidence was higher on the palate; however, this difference was also not supported statistically (Table 3).

Discussion

C. albicans is the most common type of microorganism found in oral candidal infections [17, 51]. The higher prevalence of *C. albicans* was explained by the higher capability to adhere to mucosal surfaces, which is considered as the first step in the pathogenesis of oral candidiasis [19]. The results of the present study indicate that *C. albicans* counts are higher in patients with DRS compared with the healthy group of patients. The high capability of *Candida* species to adhere to mucosal surfaces is reported to be enhanced especially by the transition from blastospores to hyphae [2], which is a striking feature of *C. albicans* biology. This property of *C. albicans* could promote tissue penetration during the early stages of infection [48]. In healthy environment, the host's defense systems must be preventing the infection. There is some evidence that oral epithelial cells may present a topical defense response to *Candida* [34]. As Budtz-Jorgensen et al. had pointed out to the importance of *C. albicans* hyphae in DRS [8], in this study, the presence of hyphae seemed to facilitate the *C. albicans* colonization.

It was suggested that the denture plaque on tissue surfaces of dentures must have an irritating effect on the mucosa [5, 12, 14, 23]. Denture plaque is composed of many different microorganisms, particularly of bacteria and *C. albicans* [11, 13, 42]. *Lactobacillus* counts, as well as the presence of yeasts in saliva, were found to be associated with the secretion rate of saliva [44]. Salivary components and the cleansing action of the tongue are a part of the hosts

Table 3 The relation between clinical intensity and *Lactobacillus* growth and *Candida albicans* counts is shown

	Clinical intensity of DRS					
	Newton 1 (n=30)		Newton 2 (n=27)		Newton 3 (n=14)	
	Mean	SD	Mean	SD	Mean	SD
Candida albicans count (cfu/ml)	1,578,700	2,717,119	2,374,037	4,677,758	3,609,429	3,822,830
Lactobacillus growth (%)	86.7		85.2		92.9	

defense balance. The isolation of oral tissues under a denture, especially a large area under a complete maxillary denture that seals and forms its own microenvironment, represents a local alteration that disturbs the normal balance [45]. In addition to the duration dentures are worn, it was suggested that DRS is associated with the amount of tissue covered by a denture. DRS prevalence was found to be higher in complete maxillary denture cases [37, 33, 36, 40, 46, 51]. Reduced salivary flow and dependant simultaneous changes in protein content, electrolyte content, buffering capacity, antibodies, and nonspecific antimicrobials of whole saliva are considered to be the main causes of the frequently detected rapid development of post-radiation caries and the quantitative and qualitative shifts in oral microflora. These patients are reported to be at high risk for rampant dental caries because of the loss of buffering capacity in their saliva and a shift in the microbial flora favoring an increase in *Streptococcus mutans* and lactobacilli [4, 15].

The toxic effects of plaque masses in contact with oral mucosa for extended periods of time are predictable and similar as in the periodontal patient. Continuous denture wearing could be an attribute, strengthening this effect [47] as observed in our study, too. Individuals wearing their complete dentures 24 h a day were reported to show more DRS [20, 24, 47]. Due to the deterioration of the dentures in time, such as the polished surfaces fit to the underlying tissues and the occlusion, dentures could become more irritant to the mucosa and more open to candidal and bacterial colonization [6]. In this manner, the results of this present study showed a statistically significant relation between denture age and *C. albicans* counts.

C. albicans does not necessarily cause disease; there have to be some other predisposing factors [39]. The most important result of this study was that it showed the importance of *C. albicans* hyphae in the development of DRS. The presence of hyphae seems to facilitate the candidal colonization on the mucosal surfaces and raising the *C. albicans* counts.

The results of this study indicated an association between *Lactobacillus* counts and *C. albicans* hyphae incidence. The explanation of this positive correlation might be the above-mentioned relation between oral hygiene and microorganism growth. Poor oral hygiene and the covered palate might enhance both the *Lactobacillus* and *C. albicans* hyphae growth in the oral cavity because of the missing rinsing action of saliva. Although our results showed no statistically significant association between continuous denture wearing and microorganism growth, this factor should be further investigated in higher patient numbers to draw conclusions.

Epidemiological facts about DRS, such as gender, age of patients, and the type and localization of dentures, are factors which are not possible to manipulate, whereas motivation of the patient not to wear the dentures at night and renewing the dentures in reasonable time intervals would be an easy measure to reduce the prevalence of DRS dramatically. It would be of importance to concentrate on patient information and motivation on hygiene for prophylactic purposes.

Conclusions

1. *C. albicans* must be an important factor in the formation of denture-related stomatitis since its counts in DRS patients are statistically higher than in healthy subjects.
2. Hyphae seem to facilitate the rise of *C. albicans* counts and be related to the inflammatory response of the tissues.
3. *Lactobacillus* seems to play an important role in the presence of DRS as well.
4. In agreement with many other studies, the results of this study confirm the importance of denture age and continuous denture wearing in the development of DRS.

Conflict of interest The authors declare that they have no conflict of interest.

References

1. Arendorf TM, Walker DM (1987) Denture stomatitis: a review. J Oral Rehabil 14:217–227
2. Baena-Monroy T, Moreno-Maldonado V, Franco-Martínez F et al (2005) Candida albicans, Staphylococcus aureus and Streptococcus mutans colonization in patients wearing dental prosthesis. Med Oral Patol Oral Cir Bucal 10:27–39
3. Barbeau J, Seguin J, Goulet JP et al (2003) Reassessing the presence of Candida albicans in denture-related stomatitis. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 95:51–59
4. Brown LR, Dreizen S, Handler S et al (1975) Effect of radiation-induced xerostomia on human oral microflora. J Dent Res 54:740–750
5. Budtz-Jorgensen E, Bertram U (1970a) Denture stomatitis I: the etiology in relation to trauma and infection. Acta Odontol Scand 28:71–92
6. Budtz-Jorgensen E, Bertram U (1970b) Denture stomatitis II: the effect of antifungal and prosthetic treatment. Acta Odontol Scand 28:283–304
7. Budtz-Jorgensen E, Mojon P, Banon-Clement JM et al (1996) Oral candidosis in long-term hospital care: comparison of edentulous and dentate subjects. Oral Dis 2:285–290
8. Budtz-Jorgensen E, Stenderup A, Grabowski M (1975) An epidemiologic study of yeasts in elderly denture wearers. Community Dent Oral Epidemiol 3:115–119
9. Budtz-Jorgensen E (1970) Denture stomatitis III. Histopathology of trauma- and Candida-induced inflammatory lesions of the palatal mucosa. Scand J Dent Res 28:551–579
10. Budtz-Jorgensen E (1981) Oral mucosal lesions associated with the wearing of removable dentures. J Oral Pathol 10:65–80

11. Budtz-Jorgensen E (1974) The significance of Candida albicans in denture stomatitis. Scand J Dent Res 82:151–190
12. Cannon RD, Chaffin WL (1999) Oral colonization by Candida albicans. Crit Rev Oral Biol Med. 10:359–383
13. Catalan A, Herrera R, Martinez A (1987) Denture plaque and palatal mucosa in denture stomatitis: scanning electron microscopic and microbiologic study. J Prosthet Dent 57:581–586
14. Chandra J, Mukherjee PK, Leidich SD et al (2001) Antifungal resistance of candidal biofilms formed on denture acrylic in vitro. J Dent Res 80:903–908
15. Cowman RA, Baron SS, Glassman AH et al (1983) Changes in protein composition of saliva from radiation induced xerostomia patients and its effect on growth of oral streptococci. J Dent Res 62:336–340
16. Cross LJ, Williams DW, Sweeney CP et al (2004) Evaluation of the recurrence of denture stomatitis and Candida colonization in a small group of patients who received itraconazole. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 97:351–358
17. Dar-Odeh NS, Shehabi AA (2003) Oral candidosis in patients with removable dentures. Mycoses 46:187–191
18. Davenport JC (1970) The oral distribution of candida in denture stomatitis. Br Dent J 129:151–156
19. Dorocka-Bobkowska B, Konopka K, Duzgunes N (2003) Influence of antifungal polyenes on the adhesion of Candida albicans and Candida glabrata to human epithelial cells in vitro. Arch Oral Biol 48:805–814
20. Fenlon MR, Sherriff M, Walter JD (1998) Factors associated with the presence of denture related stomatitis in complete denture wearers: a preliminary investigation. Eur J Prosthodont Restor Dent 6:145–147
21. Guggenheimer J, Moore PA, Rossie K et al (2000) Insulin-dependent diabetes mellitus and oral soft tissue pathologies. II. Prevalence and characteristics of Candida and candidal lesions. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 89:570–576
22. Hazen KC, Howell SA (2007) Candida, cryptococcus, and other yeasts of medical importance. In: Murray PR, Baron EJ, Jorgensen JH, Landry ML, Pfaller MA (eds) Manual of clinical microbiology. 9th edn. ASM, Washington DC, pp 1762–1788
23. Iacopino AM, Wathen WF (1992) Oral candidal infection and denture stomatitis: a comprehensive review. J Am Dent Assoc 123:46–51
24. Jeganathan S, Payne JA, Thean HP (1997) Denture stomatitis in an elderly edentulous Asian population. J Oral Rehabil 24:468–472
25. Jenkinson H, Harish C (1990) Coaggregation of Streptococcus sanguis and other Streptococci with Candida albicans. Infec Immun 58:1429–1436
26. Kuhn DM, Chandra J, Mukherjee PK et al (2002) Comparison of biofilms formed by Candida albicans and Candida parapsilosis on bioprosthetic surfaces. Infection and Immunity 70(2):878–888 Feb
27. Kulak-Ozkan Y, Kazazoglu E, Arikan A (2002) Oral hygiene habits, denture cleanliness, presence of yeasts and stomatitis in elderly people. J Oral Rehabil 29:300–304
28. Larone DH (ed) (2002) Medically important fungi: a guide to identification, 4th ed. ASM, Washington DC
29. Leberer E, Ziegelbauer K, Schmidt A et al (1997) Virulence and hyphal formation of Candida albicans require the Ste20p-like protein kinase CaCla4p. Curr Biol 7:539–546
30. Liu H (2001) Transcriptional control of dimorphism in Candida albicans. Curr Op Microbiol 4:728–735
31. Meiller TF, Jabra-Rizk MA, Baqui A et al (1999) Oral Candida dubliniensis as a clinically important species in HIV-seropositive patients in the United States. Oral Surg Oral Med Oral Pathol Oral Radiol Endod 88:573–580
32. Merz WG (1990) Candida albicans strain delineation. Clin Microbiol Rev 3:321–334
33. Mikkonen M, Nyyssonen V, Paunio I et al (1984) Prevalence of oral mucosal lesions associated with wearing removable dentures in Finnish adults. Community Dent Oral Epidemiol 12:191–194
34. Murakami M, Lopez-Garcia B, Braff M et al (2004) Postsecretory processing generates multiple cathelicidins for enhanced topical antimicrobial defense. J Immunol 172:3070–3077
35. Nanetti A, Stancari F, Ferri M et al (1993) Relationship between Candida albicans and denture stomatitis: a clinical and microbiological study. New Microbiol 16:287–291
36. Nevalainen MJ, Narhi TO, Ainamo A (1997) Oral mucosal lesions and oral hygiene habits in the home-living elderly. J Oral Rehabil 24:332–337
37. Newton AV (1962) Denture sore mouth. Br Dent J 112:357–360
38. Nikawa H, Egusa H (2001) Alteration of the coadherence of Candida albicans with oral bacteria by dietary sugars. Oral Microbiol Immunol 16:279–284
39. Oksala E (1990) Factors predisposing to oral yeast infections. Acta Odontol Scand 48:71–74
40. Pires FR, Santos EB, Bonan PR et al (2002) Denture stomatitis and salivary Candida in Brazilian edentulous patients. J Oral Rehabil 29:1115–1159
41. Radford DR, Challacombe SJ, Walter JD (1999) Denture plaque and adherence of Candida albicans to denture-base materials in vivo and in vitro. Crit Rev Oral Biol Med 10:99–116
42. Renner RP, Lee M, Andors L et al (1979) The role of C. albicans in denture stomatitis. Oral Surg Oral Med Oral Pathol 47:323–328
43. Rossie K, Guggenheimer J (1997) Oral candidiasis: clinical manifestations, diagnosis and treatment. Pract Periond Aest Dent 9:635–641
44. Sakki TK, Knuuttila M (1996) Controlled study of the association of smoking with lactobacilli, mutans streptococci and yeasts in saliva. Eur J Oral Sci 104:619–622
45. Sakki TK, Knuuttila ML, Läärä E et al (1997) The association of yeasts and denture stomatitis with behavioral and biologic factors. Oral Surg Oral Med Oral Pathol Oral Radio Endod 84:624–629
46. Sherman RG, Prusinski L, Ravenel MC et al (2002) Oral candidosis. Quintessence Int 33:521–532
47. Shulman JD, Rivera-Hidalgo F, Beach MM (2005) Risk factors associated with denture stomatitis in the United States. J Oral Pathol Med 34:340–346
48. Sudbery P, Gow N, Berman J (2004) The distinct morphogenic states of Candida albicans. Trends Microbiol 12:317–324
49. Webb BC, Thomas CJ, Willcox MD et al (1998) Candida-associated denture stomatitis. Aetiology and management: a review. Part 2. Oral diseases caused by Candida species. Aust Dent J 43:160–166
50. Wingard JR (1995) Importance of Candida species other than C albicans as pathogens in oncology patients. Clin Infect Dis 20:115–125
51. Zegarelli DJ (1993) Fungal infections of the oral cavity. Otolaryngol Clin North Am 26:1069–1089
52. Zimbro MJ, Power DA (eds.) (2003) Difco&BBL manual. Becton Dickinson Company, Maryland