



GINGIVAL WETNESS IN PATIENTS WITH DRY MOUTH

ABSTRACT

Introduction: Gingival wetness can be diminished in the presence of dry mouth. Our objective was to comparatively assess oral mucosal/gingival wetness in conjunction with other saliva-related measures, in patients with dry mouth.

Materials and Method: Fourteen Sjögren's Syndrome (SS) patients and 14 control individuals were included in the study. Gingival wetness measurements were obtained from five selected sites. Whole salivary flow rate, residual wetness of six mucosal sites, minor salivary gland secretion rates, and periodontal parameters were also determined.

Results: Whole saliva flow rate was lower in dry mouth patients compared to controls, whereas there was no significant difference in minor salivary gland secretions between these groups. The patients with dry mouth had reduced lower labial wetness values. No significant difference was observed between the groups as to gingival wetness.

Conclusion: Present findings suggest that lower labial mucosal wetness is decreased in patients with dry mouth. However, gingival moisture is acceptably maintained in these patients. Mucosal wetness may have a potential benefit for the assessment of oral dryness however; the wide distribution range of most saliva-related measures needs to be taken into account to increase reliability.

Key Words: Aged; Saliva/secretion; Sjogren's Syndrome/diagnosis.

Burak DEMİRALP¹
Güliz N. GÜNCÜ¹
Nermin YAMALIK¹
Hasan HATİPOĞLU²
Erdem KARABULUT³
Reha ALPAR³
Haviye NAZZIEL-ERVERDİ⁴



AĞIZ KURULUĞU OLAN BİREYLERDEKİ DİŞETİ NEMLİLİĞİ

Öz

Giriş: Ağız kuruluğu olduğu durumlarda dişeti nemliliği azalabilir. Amacımız, ağız kuruluğu olan bireylerde, oral mukozal nemlilik ve dişeti nemlilik değerlerini diğer tükürükle ilişkili ölçümlerle karşılaştırmalı olarak değerlendirmektir.

Gereç ve Yöntem: 14 Sjögren sendromu olan hasta ve 14 kontrol bireyi çalışmaya dahil edilmiştir. Dişeti nemliliği değerleri beş ayrı bölgeden elde edilmiştir. Ayrıca, tüm tükürük akış hızı, altı mukozal bölgenin rezidüel nemliliği, minor tükürük bezlerinin salgı hızları ve periodontal parametreler de değerlendirilmiştir.

Bulgular: Tüm tükürük akış hızı ağız kuruluğu olan bireylerde kontrollere göre daha düşük değere sahiptir. Ancak minör tükürük bez sekresyonları değerlendirildiğinde gruplar arasında herhangi bir fark tespit edilememiştir. Ağız kuruluğu olan hastalarda alt dudak nemlilik değerlerinde de düşüş bulunmaktadır. Dişeti nemlilik değerleri gruplar arasında karşılaştırıldığında ise herhangi bir fark tespit edilememiştir.

Sonuç: Mevcut bulgular alt mukozal dudak nemlilik değerlerinin ağız kuruluğu olan bireylerde düşük olduğunu göstermektedir. Ancak bu hastalarda dişeti nemlilik değerlerinde herhangi bir değişiklik satpanamamıştır. Ağız kuruluğunu değerlendirmede mukozal nemlilikönemli bir yere sahip olabilir; ancak güvenilirliği arttırabilmek için tükürükle ilgili ölçümlerin geniş dağılımı göz önünde tutulmalıdır.

Anahtar Sözcükler: Yaşlı; Tükürük/sekresyon; Sjögren Sendromu/tanı.

İletişim (Correspondance)

Güliz N. GÜNCÜ
Hacettepe Üniversitesi Dişhekimliği Fakültesi
Periodontoloji ANKARA
Tlf: 0312 305 22 37
e-posta: guliz@hacettepe.edu.tr

Geliş Tarihi: 14/12/2009
(Received)

Kabul Tarihi: 16/01/2010
(Accepted)

¹ Hacettepe Üniversitesi Dişhekimliği Fakültesi
Periodontoloji ANKARA

² Dumlupınar Üniversitesi Araştırma ve Tedavi
Hastanesi Dental Klinik/Periodontoloji KÜTAHYA

³ Hacettepe Üniversitesi Tıp Fakültesi Biyoistatistik
ANKARA

⁴ Özel Klinik Periodontoloji İSTANBUL



INTRODUCTION

Saliva has been known to play a significant role in oral defense mechanisms (1). After each swallowing cycle, a layer of saliva, residual saliva, is left clinging to the soft- and hard-tissue surfaces of the mouth (2). The residual saliva functions as a moisture retainer, a protective barrier, a lubricant and a determinant for microbial colonization (3-5). The sensation of a dry mouth is perceived when there is insufficient mucosal wetting (6,7). Wolff & Kleinberg (7) showed that the lower the salivary flow rate, the thinner the residual saliva covering the oral mucosal surfaces. Nevertheless, the data regarding the relationship between the salivary flow and oral mucosal wetness is quite limited. Sjögren's syndrome is the prototype of salivary deficiency conditions (8). In this syndrome, the diminished salivary secretion leads to an increase in the incidence of dental caries (9),(10) and gingival inflammation (9, 11). Inflammatory gingival changes are also observed in mouth breathers and are generally attributed to irritation from surface dehydration (12). Gingiva is a part of oral mucosa. To our knowledge, normal gingival wetness (the thickness of residual saliva on gingiva) values are not known. It might be expected that gingival wetness could be diminished in the presence of dry mouth. However, no evidence has been available regarding this subject. Thus, this study was designed to investigate the thickness of residual saliva coating the gingiva and other selected mucosal surfaces in patients with dry mouth and to compare the results to healthy controls. Additionally the data were evaluated for the correlation of selected salivary and periodontal parameters.

MATERIALS AND METHOD

Study Population

Starting in 2004 all patients complaining of dry mouth were examined during the 3-year period. From this group 14 volunteers (all female, mean age, 46.1 years; range, 33 - 62 years) were selected for this study based on the presence of consistent oral dryness. They were asked to fill a questionnaire (based in part on questions of the "European classification criteria for Sjögren's syndrome") (13) about their possible characteristic sicca symptoms. All of these subjects had had the diagnosis of Sjögren's syndrome (11 primary, 3 secondary). Although ten patients were on therapy for sicca symptoms, all of the patients had a chief complaint of dry mouth (mean duration, 5.1 yrs; range, 1-15 yrs), thirteen patients complained of dry lip, and twelve patients had the complaints of

dry eye. The control group comprised of 14 age-matched systemically healthy female volunteers (mean age, 46.6 years; range, 43-57 years) with no complaints suggestive of salivary gland dysfunction (14). None of the control subjects had taken any medication known to affect the salivary flow rate for several months. Moreover, none of the participants had the habits of smoking and/or alcohol use.

The protocol of this study was approved by the Ethics Committee of Hacettepe University (# FON 03/6-14). All subjects were provided with the necessary information regarding the experimental design and their informed consents were obtained before any experimental process.

Probing depth, attachment loss, sulcus bleeding index (15), plaque index (16) and calculus index (17) were recorded to evaluate the periodontal status.

In order to minimize the variances in saliva, the circadian rhythm of this biologic fluid was considered, and all of the examinations and saliva sampling were carried out between 8:00 and 11:00 a.m. Before examinations, subjects had no meal-drink or tooth brushing.

Collection of Unstimulated Whole Saliva

After a rest of 5 min, each participant swallowed and then tilted her head forward with the chin near the chest and was instructed to avoid any lip or tongue movements, talking, or swallowing (18). The saliva was allowed to pool in front of the mouth for exactly 2 min without swallowing. It was then gently drooled into Sialometer™ (Oraflow Inc., Smithtown, NY, USA). The 2-min collection was repeated twice.

Quantification of Gingival/Oral Mucosal Wetness

Periotron 8000® (Oraflow Inc., Smithtown, NY, USA.) micro-moisture meter was used for quantification of gingival/oral mucosal wetness. (7, 19, 20). After a rest of 5 min the residual wetness of the following sites was determined. The gingival sites: (I-II) Labial gingival surfaces of the upper and lower right central incisors; (IIIIV) Buccal and palatal gingival surfaces of the maxillary left first molar; (V) Buccal gingival surface of mandibular left first molar. Mucosal sites: (I-V- in the midline) (I) lower labial mucosa, halfway between the vermilion border and the attachment of the lower lip to the labial frenum; (II) soft palatal mucosa, along the vibrating line; (III) upper labial mucosa, halfway between the vermilion border and the attachment of the upper lip to the labial frenum; (IV) anterior hard palatal mucosa, the palatal area including the incisive papilla; (V) anterior tongue, anterior part of the dorsal surface of the tongue approximately 5 mm



from the tip; (VI) buccal mucosa-1 cm from the commissure of the lip at the height of the occlusal plane.

For each measurement the individual was asked to swallow, open her mouth, and then a Sialopaper™ strip (frying-pan-shaped filter paper strips, measuring area 44.15mm²) (Oraflow Inc., Smithtown, NY, USA) was carried into the mouth with a pair of college tweezers and pressed against the mucosal surface for 5 sec with the forefinger of the investigator's right hand, which was sheathed in a dry surgical glove (7, 19, 20). The residual saliva was absorbed onto the strip. To eliminate the risk of evaporation, strips were immediately transferred to a chair-side located and previously calibrated Periotron 8000®. Saliva thicknesses were calculated by dividing the volume of saliva collected by the area of the Sialopaper™ strip (7).

Measurement of Minor Salivary Gland Secretions

After the measurement of mucosal wetness, minor salivary gland secretion rates were assessed on the lower labial mucosa (in the midline, halfway between the labial frenulum and vermillion border) and soft palatal mucosa (in the midline, along the vibrating line). After drying each site with gauze, a Sialopaper™ strip was placed there and retained with light finger pressure for 30 seconds to collect saliva secreted from the underlying mucosa. The saliva on the strip was quantified with the Periotron 8000® and flow rates were calculated in units of $\mu\text{l}/(\text{min cm}^2)$ of mucosal area.

Statistical Analysis

Data were analyzed statistically by using the SPSS for Windows software program (Chicago, IL,USA). A Student's

t-test was used for analyzing normally distributed variables and the Mann Whitney-U test was performed for analyzing abnormally distributed variables in Sjögren's syndrome and control groups. The relationships between various measures were examined by using the Spearman's rank correlation coefficient and $p < 0.05$ was considered statistically significant.

RESULTS

The mean flow rate of the unstimulated whole saliva was significantly lower in patients compared to controls ($p = 0.004$). However, no significant difference was observed in minor salivary gland secretion rates from the lower labial and soft palatal areas between the groups ($p > 0.05$) (Table 1).

In general, the patients had lower wetness values than the controls. However, this difference reached to a significance level only at the lower labial mucosa ($p = 0.001$) (Table 2). No significant difference was observed in gingival wetness values between the groups ($p > 0.05$) (Table 3). No significant difference was noted in periodontal findings (PI, CI, SBI, CAL, PD) between the groups (Table 4). Correspondingly, there was no significant difference in periodontal findings of the selected sites between these groups (not shown).

Correlations Analysis

In the patient group, no significant correlation was found between the unstimulated whole salivary flow rate and labial minor gland saliva secretion rate ($r = 0.013$, $p = 0.964$) or palatal minor gland saliva secretion rate ($r = 0.139$, $p = 0.635$). However, a statistically significant positive relationship was noted between the residual saliva thickness on the buccal

Table 1— Data Regarding Unstimulated Whole Saliva and Minor Salivary Glands

	Sjögren (n=14)	Control (n=14)	p
Unstimulated whole salivary flow rate (ml/min)	0.3±0.1 (0.1-0.5)	0.4±0.1 (0.3-0.6)	0.004
Minor salivary gland secretion rate ($\mu\text{l}/(\text{cm}^2\text{min})$)			
Lower Labial	6.1±4.4 (1.6-13.3)	6.3±3.0 (3.0-13.3)	0.52
Soft Palatal	4.5±3.2 (0.7-11.3)	6.0±3.3 (2.1-13.3)	0.15

All the results are expressed as mean \pm sd (minimum-maximum)

Student's t-test was used for analyzing normally distributed variables (unstimulated whole salivary flow rate) and Mann Whitney-U test was performed for analyzing abnormally distributed variables (minor salivary gland secretions) in Sjögren's syndrome and control groups

**Table 2—** Oral Mucosal Wetness (residual saliva thickness) at Distinct Sites

Residual saliva thickness (μm)	Sjögren (n=14)	Control (n=14)	p
Lower labial mucosa	9.6 \pm 5.7 (0.2-19.7)	24.1 \pm 18.4 (10.4-66.4)	0.001
Soft palatal mucosa	9.6 \pm 8.0 (1.6-29.0)	21.0 \pm 18.0 (2.7-59.8)	0.07
Upper labial mucosa	17.7 \pm 13.7 (3.0-51.9)	19.5 \pm 14.5 (10.0-67.3)	0.50
Buccal mucosa	36.4 \pm 26.3 (3.9-67.3)	37.2 \pm 18.2 (10.4-67.3)	0.78
Anterior hard palatal mucosa	12.3 \pm 19.4 (1.1-66.4)	19.4 \pm 25.0 (1.4-66.4)	0.07
Anterior tongue	27.2 \pm 18.7 (5.4-65.7)	34.8 \pm 17.3 (6.6-63.2)	0.27

All the results are expressed as mean \pm sd (minimum-maximum).

Student t-test was used for analyzing normally distributed variables (Residual saliva thickness on anterior tongue) and Mann Whitney-U test was performed for analyzing abnormally distributed variables (Residual saliva thickness on lower labial, soft palatal, upper labial, buccal and anterior hard palatal mucosa) in Sjögren's syndrome and control groups.

Table 3— Gingival Wetness (residual saliva thickness) at Selected Natural Tooth Sites

Residual Saliva Thickness (μm)	Maxillary Left Molar	Mandibular Left Molar	Maxillary Right Central Incisor	Mandibular Right Central Incisor
Sjögren (n= 14)	24.0 \pm 21.8 (0.2-67.3)	30.4 \pm 22.0 (2.7-66.4)	16.7 \pm 14.0 (0.9-48.7)	13.5 \pm 18.7 (0.9-66.4)
Control (n= 14)	20.7 \pm 21.0 (4.3-67.3)	25.6 \pm 20.7 (2.9-67.3)	19.8 \pm 16.4 (4.8-66.4)	12.5 \pm 15.4 (2.3-38.3)

RST: Residual saliva thickness (μm) at natural tooth sites

All the results are expressed as mean \pm sd (minimum-maximum)

Mann Whitney-U test was performed for analyzing abnormally distributed variables in Sjögren's syndrome and control groups

Table 4— Periodontal Findings (all sites)

	Sjögren (n=14)	Control (n=14)	p
Plaque index (PI)	0.9 \pm 0.6 (0.3-1.6)	0.6 \pm 0.4 (0.0-1.1)	0.07
Calculus index (CI)	0.2 \pm 0.3 (0.0-0.9)	0.4 \pm 0.8 (0.0-3.1)	0.51
Sulcus bleeding index (SBI)	1.1 \pm 1.3 (0.1-4.8)	0.6 \pm 0.8 (0.0-2.5)	0.21
Clinical attachment level (CAL)	2.6 \pm 0.7 (1.8-4.1)	2.4 \pm 1.3 (1.1-5.3)	0.54
Probing depth (PD)	2.3 \pm 0.6 (1.6-3.6)	2.4 \pm 0.6 (1.8-3.8)	0.48

All the results are expressed as mean \pm SD (minimum-maximum)

Student's t-test was used for analyzing normally distributed variables (PI) and Mann Whitney-U test was performed for analyzing abnormally distributed variables (CI, SBI, CAL, PD) in Sjögren's syndrome and control groups



mucosa and palatal minor gland saliva secretion rate ($r=0.611$, $p=0.020$). Similar positive correlation in residual saliva thickness was observed between the labial and soft palatal mucosa ($r=0.799$, $p=0.001$). No significant association was found between the unstimulated whole salivary flow rate and residual saliva thickness on the labial mucosa ($r=0.497$, $p=0.070$) or that on the soft palatal mucosa ($r=0.065$, $p=0.824$).

In the control group a significant correlation was detected between unstimulated whole salivary flow rate and labial minor gland saliva secretion rate ($r=0.611$, $p=0.020$). A significant positive correlation was also found in residual saliva thickness between the anterior hard palate and anterior tongue ($r=0.674$, $p=0.008$). Additional significant correlations were noted between the palatal minor gland saliva secretion rate and the residual saliva thickness on the upper labial mucosa ($r=0.588$, $p=0.027$); and the anterior hard palate ($r=0.557$, $p=0.038$). However, no significant association was observed between the unstimulated whole salivary flow rate and residual saliva thickness on the anterior hard palate ($r=0.225$, $p=0.439$).

Statistically significant correlations were also detected between the labial minor gland saliva secretion rate and wetness values of the labial gingival surface of the lower right central incisor ($r=0.531$, $p=0.05$) and buccal gingival surface of mandibular left first molar ($r=0.636$, $p=0.015$). In both groups no association was found between the clinical periodontal findings and any of the saliva-related measures.

DISCUSSION

Dry mouth is believed to be sensed when the flow rate of unstimulated whole saliva is approximately 0.1 ml/min. However, it is well known that the severity of dryness does not correlate directly with a reduction of salivary flow (21-23). In the present study, although all the patients complained of dry mouth, the mean unstimulated whole-saliva flow rate was 0.3ml/min. It has been implied that insufficient mucosal wetting leads to sensation of dryness and measurement of mucosal wetness is suggested as one of the diagnostic methods for assessing dry mouth (7,19). Wolff and Kleinberg (7) drew attention to reduced mucosal wetness in hyposalivators. Won et al (20) analyzed mucosal wetness in individuals with normal salivary function. Then, Lee et al (19) reported low wetness values in hyposalivators at almost all the mucosal sites. The present data showed reductions in mucosal wetness values in patients compared to controls, but the differ-

ence between the groups reached a significance level only on the lower labial mucosa. A statistically significant positive relationship was also observed in wetness values between the lower labial and soft palatal mucosa. Collectively, although differences existed as to the sites that were affected, the results of the previous studies and the present findings confirmed the presence of reduced mucosal wetness in patients with dry mouth.

Our data revealed that there was no difference in minor salivary gland secretions between the patient and control groups. Moreover, in the patient group, no significant correlation was found between the unstimulated whole salivary flow rate and minor salivary gland secretions. In contrast, a significant correlation was detected in the control group between the unstimulated whole salivary flow rate and labial minor gland saliva secretion rate. These findings suggest that dry mouth may not always be associated with reduced secretions of minor salivary glands. Correspondingly, Lee et al. (19) reported that the function of the minor salivary glands was less affected and well preserved in patients with dry mouth.

To our knowledge, the present study is the first report describing gingival wetness values both in normal individuals and in patients with dry mouth. No statistically significant difference was detected in gingival wetness values between the groups. These findings may suggest that dry mouth does not lead to any reduction in residual saliva covering gingival surfaces. It was also interesting to note that 13 patients were complaining of lip dryness, and residual saliva thickness on labial mucosa was very low in this group. However, minor labial gland secretion rates and the amount of residual saliva on the labial gingival surface of the anterior teeth were not any different from those in healthy controls. Accordingly, in the control group significant correlations were detected between labial minor gland saliva secretion rates and labial gingival wetness values of the lower incisors. These findings may suggest that gingival moisture is acceptably maintained in patients with dry mouth, probably owing to the regular minor salivary secretions.

Sjögren's syndrome is demonstrated to be a condition that influences the periodontal status of affected patients (9, 11). However, in the present study no significant difference was noted in periodontal findings between the two groups and no association was found between the clinical periodontal findings and any of the saliva-related measures.

Likewise, Tseng et al (24) found no difference between Sjögren's syndrome patients and healthy controls as to peri-



odontal parameters, but no information was provided in their report indicating the severity of the salivary disease. In our study, most of the patients were using systemic medications to relieve the symptoms of dryness that could have contributed to similar periodontal characteristics noted in both groups. Accordingly, gingival wetness values from both the groups were comparable.

This study was limited by its small sample size. A wider range of volunteer participants could not be achieved. In conclusion, the results of this study suggest that lower labial mucosal wetting is insufficient in patients with dry mouth complaints. However, gingival moisture is acceptably maintained in these patients. Further studies investigating gingival wetness values in mouth breathers or in patients with severe xerostomia may provide valuable information about saliva related changes on gingiva.

ACKNOWLEDGEMENTS

This study was supported by Hacettepe University Scientific Research Unit (Grant No.03D03201001).

REFERENCES

1. Bulkacz J CF. Defense mechanisms of the gingiva. In: Clinical Periodontology: Newman MG, Takei HH, Carranza FA, editors. Philadelphia: W.B. Saunders Company. 2002, pp 254-62.
2. Dawes C. A mathematical model of salivary clearance of sugar from the oral cavity. *Caries Res* 1983;17:321-34.
3. Gibbons RJ, Qureshi JV. Selective binding of blood group-reactive salivary mucins by *Streptococcus mutans* and other oral organisms. *Infect Immun* 1978;22:665-71.
4. Hatton MN, Loomis RE, Levine MJ, et al. Masticatory lubrication. The role of carbohydrate in the lubricating property of a salivary glycoprotein-albumin complex. *Biochem J* 1985;230:817-20.
5. Tabak LA, Levine MJ, Mandel ID, et al. Role of salivary mucins in the protection of the oral cavity. *J Oral Pathol* 1982; 11:11-7.
6. DiSabato-Mordarski T, Kleinberg I. Measurement and comparison of the residual saliva on various oral mucosal and dentition surfaces in humans. *Arch Oral Biol* 1996;41:655-65.
7. Wolff M, Kleinberg I. Oral mucosal wetness in hypo- and normosalivators. *Arch Oral Biol* 1998;43:455-62.
8. Fox RI. Sjogren's syndrome. Pathogenesis and new approaches to therapy. *Adv Exp Med Biol* 1998;438:891-902.
9. Najera MP, al-Hashimi I, Plemons JM, et al. Prevalence of periodontal disease in patients with Sjogren's syndrome. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod* 1997; 83:453-7.
10. Soto-Rojas AE, Kraus A. The oral side of Sjogren syndrome. Diagnosis and treatment. A review. *Arch Med Res* 2002;33:95-106.
11. Celenligil H, Eratalay K, Kansu E, et al. Periodontal status and serum antibody responses to oral microorganisms in Sjogren's syndrome. *J Periodontol* 1998;69:571-7.
12. Carranza FA, Hogan EL. Gingival Enlargement. In: Clinical Periodontology: Newman MG, Takei HH, Carranza FA (Eds). Philadelphia: W.B. Saunders Company 2002, 279-96.
13. Vitali C, Bombardieri S, Moutsopoulos HM, et al. Preliminary criteria for the classification of Sjogren's syndrome. Results of a prospective concerted action supported by the European Community. *Arthritis Rheum* 1993; 36:340-7.
14. Fox PC, Busch KA, Baum BJ. Subjective reports of xerostomia and objective measures of salivary gland performance. *J Am Dent Assoc* 1987; 115: 581-4.
15. Mühlemann HR, Son, S. Gingival sulcus bleeding-a leading symptom in initial gingivitis. *Helv Odontol Acta* 1971;15:107-13.
16. Silness J, Løe, H. Periodontal disease in pregnancy. II. Correlation between oral hygiene and periodontal condition. *Acta Odontol Scand* 1964; 22: 121-35.
17. Greene JC, Vermillion JR. The simplified oral hygiene index. *J Am Dent Assoc.* 1964; 68:7-13.
18. Haveman CW, Redding SW. Dental management and treatment of xerostomic patients. *Tex Dent J* 1998;115:43-56.
19. Lee SK, Lee SW, Chung SC, et al. Analysis of residual saliva and minor salivary gland secretions in patients with dry mouth. *Arch Oral Biol* 2002; 47: 637-41.
20. Won S, Kho H, Kim Y, et al. Analysis of residual saliva and minor salivary gland secretions. *Arch Oral Biol* 2001;46:619-24.
21. Spielman A, Ben-Aryeh H, Gutman D, et al. Xerostomia—diagnosis and treatment. *Oral Surg Oral Med Oral Pathol* 1981;51:144-7.
22. von Knorring L. Changes in saliva secretion and accommodation width during short-term administration of imipramine and zimelidine in healthy volunteers. *Int Pharmacopsychiatry* 1981;16:69-78.
23. Donatsky O, Johnsen T, Holmstrup P, et al. Effect of saliment on parotid salivary gland secretion and on xerostomia caused by Sjogren's syndrome. *Scand J Dent Res* 1982;90:157-62.
24. Tseng CC. Periodontal status of patients with Sjogren's syndrome: a cross-sectional study. *J Formos Med Assoc* 1991;90:109-11.