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THE DIAGNOSTIC RELATIONSHIP BETWEEN *HELICOBACTER PYLORI*, PERIODONTAL DISEASES AND GASTROESOPHAGEAL REFLUX

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Summary

Background and objectives. The development of the digestive tract and periodontal diseases is influenced by several general risk factors. One of them is the tooth loss, due to periodontal diseases, which results in poor mastication of food and latter gastroesophageal reflux. The second major risk factor is bacterial microflora. Current research is relevant due to the controversial nature of the opinions on the general bacterial causes of the aforementioned diseases and their correlations.

The aim of the study is to confirm the hypothesis that the development of periodontal and gastroesophageal reflux diseases is influenced by the simultaneous colony development of *Helicobacter pylori* in periodontal pockets and the digestive tract.

Material and Methods. A total of 70 respondents were studied. Social and demographic indicators were analyzed based on the data of a questionnaire-based survey. The periodontium was studied clinically; community periodontal index of treatment needs was determined, a rapid urease test (Pronto Dry) was performed to detect bacteria. The gastroesophageal reflux disease was analyzed using an endoscope and via survey. The respondents were distributed into three test and three age groups. IBM SPSS Statistics 22 software was used for statistical analysis.

Results. A statistically significant correlation was found between the community periodontal index of treatment needs and Reflux symptom index ($p=0.004$), and urease test ($p<0.001$) and oral hygiene ($p<0.05$). No statistically significant correlations were determined between indices (RSI; RFS), age, gender, urease test ($p>0.05$).

Conclusions. The colony formation of *Helicobacter pylori* in periodontium has no impact on the gastroesophageal reflux disease but is significantly related to periodontal diseases. Thus, the oral cavity is the primary reservoir of internal *H. pylori* colonization.

Introduction

Modern medicine focuses on disease prevention and public health. Great attention paid to dentistry because sufficient oral hygiene and healthy mouth and teeth make up the basis of public health [1]. Periodontal diseases (PD) fall under two main categories – gingivitis and periodontitis. Gingivitis – inflammation of the gingiva in which the inflammatory process does not damage gingival junctional epithelium and bone tissue. On the other hand, periodontitis is a more severe periodontal disease, which results in periodontal pocket (PP) development, bone loss and increasing tooth mobility. Advanced periodontitis is the sixth most prevalent disease globally [2]. In Europe, 50 % of the adult population suffers from chronic periodontitis and 11 % of the adult population suffer from severe periodontitis [2,3]. These diseases are caused by a combination of local and systemic factors, microorganisms and macro organism individual qualities play a great role in their pathogenesis [4]. PP can contain up to 400 different species of microorganisms [4-6], including – *Helicobacter pylori (H. pylori)*, which is known to cause chronic periodontitis, mouth ulcers, stomatitis and oral cancer. *H. pylori* is a gram-negative microeosinophilic bacteria, ranging 2 to 4 μm in length and 0,5 to 1 μm in width, highly mobile due to the expression of flagella [7]. The single primary colonization location for this microorganism remains undetermined to this day. Primary human *H. pylori* colonization sites are thought to be oral cavity or

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gastrointestinal tract [7, 8]. While primary colonization site remains for undetermined, primary transmission routes – oral and faecal-oral, are well known [9]. Some authors suggest that improved oral hygiene and timely periodontal disease treatment would decrease the incidence of gastrointestinal tract diseases [10].

Digestive tract and periodontal diseases tend to have negative impact on quality of life. Oral cavity is the first organ of the digestive tract, from which the food reaches gastrointestinal tract and is digested and absorbed. It has been proven that disorders in digestive system may impact PD [11]. Digestive system disorders may result in tooth loss in 23 to 36 % of the patients, which in turn, may cause functional, as well as, aesthetic issues. PD may result and insufficient nutrition, when food is poorly masticated resulting in difficult digestion, dyspepsia and gastroesophageal reflux disease (GERD) [3]. GERD is prevalent in 10 % of the population, but only 15 % of the cases exhibit increased peptic acidity [12]. It is believed that insufficient oral hygiene may contribute to GERD pathogenesis and prevalence, because different microflora occupy oral cavity and different parts of gastrointestinal tract [3, 13]. No scientific consensus has been reached regarding *H. pylori* colonization of oral cavity and the rest of digestive system [3]. PD causes like *Porphyromonas gingivalis*, *Actinobacillus actinomycetemcomitans*, *Tannerella forsythia*, *Treponema denticola, Prevotella intermedia* have been thoroughly researched over the years, but there are only few publications regarding *H. pylori* colonization in periodontal tissue link to GERD [14]. Other type of bacteria, such as *Streptococcus salivarius, Haemophilus influenzae,* can be found in the oral cavity. These species, when affected by adverse factors, such as, antibiotics, may produce urase [15]. However *Str. Salivarius* or *Haemophilus spp.* produced urease is more than 400 times less potent than urease produced by *H. pylori*, which leads authors to believe that other gastrointestinal microbiota does not affect *H. pylori* RUT identification [15].

While main *H. pylori* contamination routes – oral and oral-faecal, are known, primary reservoir of internal *H. pylori* colonization is still widely debated [4,16,17]. Rapid urease test (RUT) conventionally is used to determine *H. pylori* infection, but histochemical dye test is recommended in addition to RUT [15]. It is believed that proper oral hygiene and PD treatment might prevent some diseases of the digestive tract, GERD included [13].

The aim of this study is to test the hypothesis, that primary colonization location for *H. pylori* is oral cavity from where the bacteria spreads to the rest of digestive system and to determine if number of colonies in periodontal tissue is linked to the quality and rate of oral hygiene, as well as, GERD.

Materials and Methods

The study took place in LUHS Department of Dental and Oral Pathology and Department of Otorhinolaryngology. Patients referred to LUHS Otorhinolaryngology outpatient facility for GERD evaluation and healthy volunteers were included in this study. Participant age ranged from 24 to 83 years. During primary visit, participants were asked to fill out a questionnaire and a GERD symptom index scale based on a Likert scale (RSI). Rigid laryngeal video endoscopy images and subgingival plaque sample for urease test were obtained from each participant during the same visit. The control group consisted of GERD free participants, while the patient group was made up of patients with healthy periodontal tissue and patients with PD. Participants with a history of diabetes, oncologic diseases, HIV positive patients, participants who have used antibiotics or nonsteroidal anti-inflammatory drugs in the past two month period, as well as, pregnant women were not included in this study.

Participant survey. Anonymous questionnaire was used to obtain social-demographic data, relevant medical history, personal plaque control habits (IOH), professional oral hygiene (POH) rate, complaints, like hoarseness, persistent curling, increased laryngeal secretion, posterior rhinorrhea, difficulty when swallowing, cough after eating or while laying down, persistent cough, persistent feeling of asphyxiation, feeling foreign body in the pharynx, retrosternal burning sensation, dyspepsia, acid reflux and other laryngeal problems, as well as, treatment response and harmful habits [18]. Specialized Likert based Reflux symptom index (RSI) scale was filled out (ranging from 0 to 45 points where score $\geq$ 13 was considered pathologic) [18].

Endoscopic GERD evaluation. Laryngeal endoscopic evaluation was carried out using XION EndoSTROB DX rigid 90˚ endoscope (XION GmbH, Berlin, Germany, 2012). Obtained laryngeal, together with, nasopharyngeal and oral findings were included in GERD diagnostic scale. Reflux finding score (RFS) was determined taking into account vestibular fold, subglottic and posterior laryngeal site mucosa redness, swelling, hypertrophy, as well as, granulomas and thick mucus presence in the larynx (Figure 1). Total score ranged from 0 to 26 points where $\geq$7 points suggested GERD [18].

Periodontological examination. Sterile periodontal kit has been chosen for periodontal examination: periodontal probe was used to measure gingival sulcus or PP depth of each tooth. Gathered periodontological data was used for the community periodontal index of treatment needs (CPITN) determination: 0 – healthy periodontium; 1- bleeding gums, 2 – supra- or subgingival calculus, faulty fillings or artificial crowns; 3 –4 to 5 mm PP; 4 – 6 mm and deeper PP. Obtained data was included in the clinical evaluation pro-

tocol. According to CPITN patients fell under one of four categories: 0 – no treatment necessary; I - oral hygiene instruction necessary; II – oral hygiene instruction, supra- and subgingival scaling, filling, crown correction needed; III – oral hygiene instruction, supra- and subgingival scaling and complex treatment necessary [19].

Urease testing. For *H. pylori* testing subgingival soft and mineralized plaque was obtained from gingival sulcus or PP using sterile universal curette. Testing sample was selectively obtained from either maxilla or mandible periodontium at the deepest measured PP. Pronto Dry New, GASTREX (France 2011) rapid urease test (RUT) was used to determine *H. pylori* involvement. Test sample was placed on RUT indication field. RUT color change was observed and captured after 1 minute, 20 minutes and 1 hour. According to manufacturer instructions, possible indication field colors ranged from yellow (negative) to red (positive test) [11]. Five distinct color change patterns were established: negative test (0), weak urease reaction (1), normal urease reaction (2), strong urease reaction (3) and very strong urease reaction (4). More saturated redness indicated greater severity of the infection.

Statistical analysis. Statistical data analysis was carried out with IBM SPSS Statistics 22 (Statistical Package for Social Sciences). Serial number was assigned to each participant to protect their privacy. Data obtained from the questionnaire and laryngeal endoscopy was used. Significance level of 0.05 for testing of statistical hypothesis was chosen. Averages with standard deviations and median were calculated. Kolmogorov–Smirnov test was used to test for normality in quantitative variables. Student t-test and one-way ANOVA were used to determine statistically significantdifference between groups. The Wilcoxon rank-sum test, Kruskal-Wallis test and Mann-Whitney U test were used for nonparametric statistics. Factorial analysis was used to condense and index primary variables and to form scales. Factorial analysis was performed by building a correlation matrix. Primary variable method and VARIMAX rotation were used. Kaiser-Mayer-Olkin (KMO) coefficient was calculated to evaluate matrix adequacy. Cronbach's alfa was used to determine scale internal consistency. Binary logistic regression was performed for forecasting. Receiver Operating Characteristic curve (ROC) was used to determine the sensitivity, specificity and area under the curve (AUC) in RUT of periodontal tissue.

Results

70 individuals were included in the study. Age ranged from 24 to 83 years. According to clinical findings, subjects were assigned to one of three groups (I; II;

Table 1. Sample social characteristics.
χ^2 – chi squared, p – statistical significance, SD – standard deviation. Krustal-Wallis test was used for nonparametric quantitative value comparison; Mann-Whitney test was used for multicomparison.

Characteristic total	Sample (n=70)	Group		
		I gr.(n=19)	II gr. (n=16)	III gr.(n=35)
Gender, n (%):				
male	24 (34.3)	5 (26.3)	6 (37.5)	13 (37.1)
female	46 (65.7)	14 (73.7)	10 (62.5)	22 (62.9)
		χ^2=0.736, df=2, p=0.692		
Average age (SD)	46.4 (12.9)	54.3(13.1)	42.1(12.5)	44.0 (11.3)
Median [25-75%]	44 [37.0-55.5]	55.0 [43.0-64.0]*,**	39.5 [34.0-49.8]*	42.0 [37.0-54.0]**
		χ^2=8.821, df=2, p=0.012; * p=0.012, ** p=0.009		
Habitat n (%):				
urban	63(90,0)	18 (28.6)	15 (23.8)	30 (47.6)
rural	7(10,0)	1 (5.3)	1 (6.3)	5 (14.3)
		χ^2=1.438, df=2, p=0.487		

Table 2. Clinical periodontal examination results.
χ^2 – chi squared, p – statistical significance, obtained using Monte Carlo method.

Characteristic	Total (n=70)	Group		
		I (n=19)	II (n=16)	III (n=35)
POH/year, n (%):	38(54.3)	11(57.9)	8(50.0)	19(54.3)
		χ^2=0.218, df=2, p=0.897		
Two times daily IOH, n (%):	52(74.3)	11(57.9)	14(87.5)	27(77.1)
One time daily	18(25.7)	8(42.1)	2(12.5)	8(22.9)
		χ^2=4.284, df=2, p=0.117		
CPITN, n (%):				
healthy periodontium (0)	51(72.9)	0*,**	16(100.0) *	35(100)**
mineralized plaque (2)	1(1.4)	1(5.3)	0	0
4 to 5 mm. PP (3)	11(15.7)	11(57.9) *,**	0*	0**
6 mm and deeper PP (4)	7(10.0)	7(36.8) *,**	0*	0**
		χ^2=70.0, df=6, p<0.001, *,** p<0.05		

III – control gr.). Three distinct age groups were formed: A group was <40 yrs. (n=24 (34.3%)); B group was 40 to 52 yrs. (n=22 (31.4%)); C group was > 52 yrs. (n=24(34.3%)) (p=0.005). Average sample age was 46.4 (12.9) yrs. Gender and average age distribution are presented in Table 1.

Periodontological examination. Data obtained from clinical periodontal examination, CPITN index score, dental survey is presented in Table 2.

Statistically significant correlation was found between CPITN and A, B, C age groups (r=0.334, p=0.005). Subjects aged 50 and older had a statistically significantly less healthy periodontal tissue and deeper than 4 mm PP, when compared to other age groups (p=0.001).

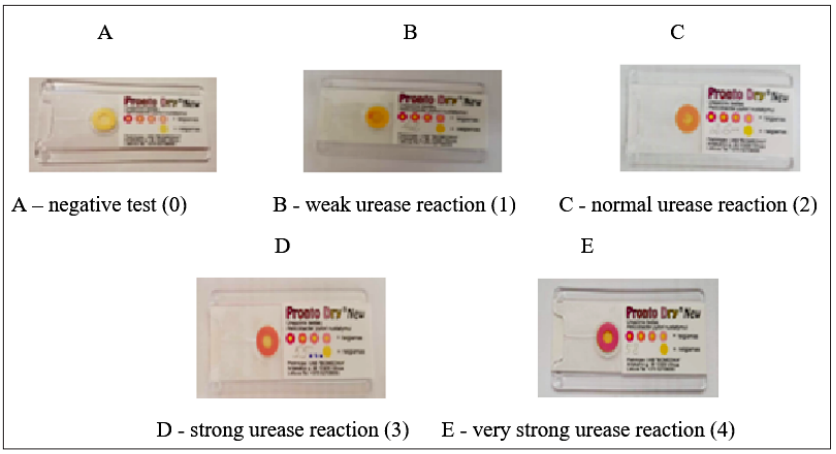
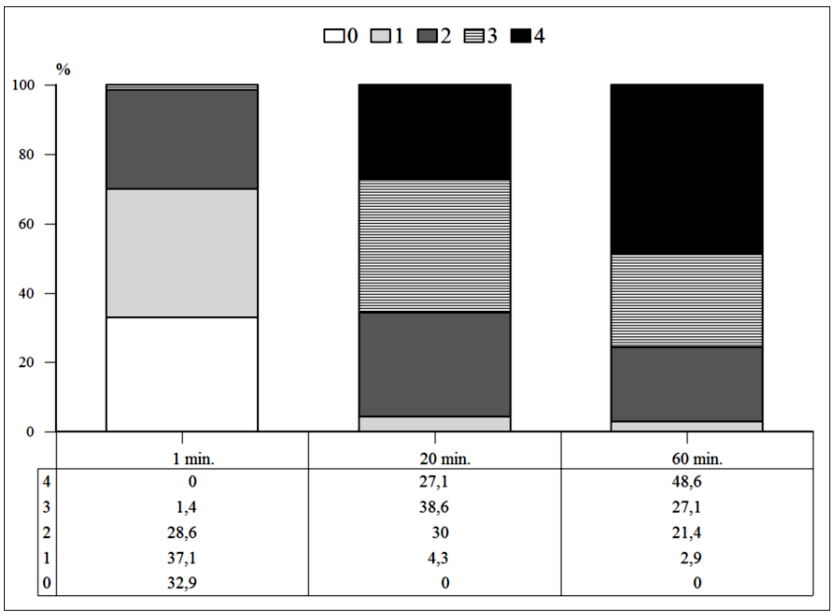


Figure 1. RUT possible outcomes.



	1 min.	20 min.	60 min.
4	0	27,1	48,6
3	1,4	38,6	27,1
2	28,6	30	21,4
1	37,1	4,3	2,9
0	32,9	0	0

Figure 2. General RUT results proportional distribution during different time frames and groups.

RUT analysis. RUT was performed to determine *H. pylori* involvement in periodontal tissue (n=70). RUT color change was observed and captured after 1 min., 20 min. and 1 hour. Possible outcomes and analysis results are presented in Figure 1 and Figure 2.

Based on Wilcoxon Rank Sum test, statistically significant general RUT indicator field change was observed after 1 min. and 2 min. ($z=7.635$, $p<0.001$); after 20 min. and 60 min ($z=7.51$, $p<0.001$).

RUT results between different groups were statistically significantly different in different indicator color observation times Table 3.

ROC demonstrated RUT sensitivity of 57.9%, specificity of 80.0% and AUC of 81.5% at cut-off value of 1.

Sociodemographic criteria, which were connected to CPITN index values and IOH, had statistically significant impact on RUT outcomes: after 1min RUT value was observed statistically significantly more often for subjects that brush their teeth 2 times daily, rather than subjects that brush their teeth once a day. After 20 and 60 min. mark very strong urease reaction (4) was statistically significantly observed more often for subjects, which brush their teeth one time during day when compared to subjects that brush their teeth 2 times daily. Corresponding data is presented in Table 4.

Subjects with PP equal or greater than 4 mm GUT value of 4 was observed significantly often. On the other hand, RUT score of 0 or 2 was statistically significantly common when healthy periodontal tissue was present. RUT and CPITN correlations are presented in Table 5.

GERD result analysis. Likert scale based reflux symptom index (RSI) showed strong internal consistency coefficient of 0,9. According to data obtained from RSI subjects were assigned to two groups: 1 group – subjects without GERD (RSI<13 points; n=49 (70%)) and 2 group – present GERD (RSI≥13; n= 21 (30%)). (I gr. vs II gr. $p>0.05$; II gr. vs III gr. $p<0.001$; I gr. vs III gr. $p<0.001$)

(χ^2=31.075; df=2, p<0.001).

Average RSI score for the total sample was 8.3 (SD 9.0). Average RSI score in responders with healthy periodontal tissue was statistically significantly lower (5.8 (SD 8.1)) than in PP of 4mm and deeper group (14.9 (SD 8.5)) (p<0.001).

RSI did not show dependence on age, gender or RUT score. Spearman correlation analysis showed statistically significant weak correlation between RSI and CPITN groups (r=0.344, p=0.004): 10(20.0%) subjects with healthy periodontium and 10(55.6%) contained pocket depth ≥4 mm were in RSI group 2. A binary logistic regression model with 95% confidence determined that patients with RSI ≥13 points are 5 times (CI=1.568-15.942) more likely to have 4 mm and greater PP.

Endoscopic laryngeal evaluation performed on groups I and II research groups, and control group was not analyzed. According RFS score two groups were divided: A group – RFS < 7 points and B group – RFS ≥7 points. Average RFS score was 8.4(SD 3.2) (Table 6).

RFS did not exhibit statistically significant dependence on age, gender, RUT or CPITN (p>0.05). RFS score in responders with healthy periodontal tissue and PP ≥4 mm did not differ statistically significantly.

Discussion

It is known that periodontal and GERD related diseases are more common among the elderly. According to a literature review, elderly and women are at greater risk of chronic periodontitis [20]. Also, females are more likely to suffer from GERD, which was noted in data published by Mercut et al [20]. More females participated in our study. Data pool was deemed sufficient; most subjects came from urban areas. Questionnaire obtained answers demonstrate that subjects partake in personal, as well as, professional oral hygiene procedures. Due to a lack of information participants rarely had knowledge about periodontal disease prevalence in the family.

CPITN index was purposely chosen for this study. The index verifies the necessity for periodontal treatment. By determining CPITN, two statistically significant groups emerged – subjects with periodontal disease and healthy test participants. Furthermore, a direct correlation between CPITN and age was determined – older patients were prone to periodontal disease and were in need of periodontal treatment. This study only partially confirmed these claims because no significant correlation between gender and CPITN was determined.

While the link between *H. pylori* and periodontal diseases is well established, but many questions, like primary colonization site and contamination routes, remain up for debate [6;16;21;22].

The results of this study concur with the results obtained by Asqah et al. and Hu et al. that oral *H. pylori* colonies found in periodontal pockets intensify the progression of periodontal diseases [6,11]. Obtained results partially confirm

Table 3. RUT proportional distribution during different observation stages by group.
χ^2 – chi squared, p – statistical significance, obtained using Monte Carlo method.

RUT	Group		
	I	II	III
After 1 min., n(%):			
0	0	3(18.8)	20(57.1)
1	8(42.1)	10(62.5)	8(22.9)
2	10(52.6)	3(18.8)	7(20.0)
3	1(5.3)	0	0
	χ^2=26.29, df=6, p=0.036; **p=0.005		
After 20 min., n(%):			
1	0	1(6.3)	2(5.7)
2	1(5.3)	3(18.8)	17(48.6)
3	6(31.6)	10(62.5)	11(31.4)
4	12(63.2)	2(12.5)	5(14.3)
	χ^2=25.25, df=6, p=0.011; **p=0.001		
After 60 min., n(%):			
1	0	0	2(5.7)
2	0	1(6.3)	14(40.0)
3	1(5.3)	7(43.8)	11(31.4)
4	18(94.7)	8(50.0)	8(22.9)
	χ^2=31.75, df=6, p=0.008; *p=0.001		

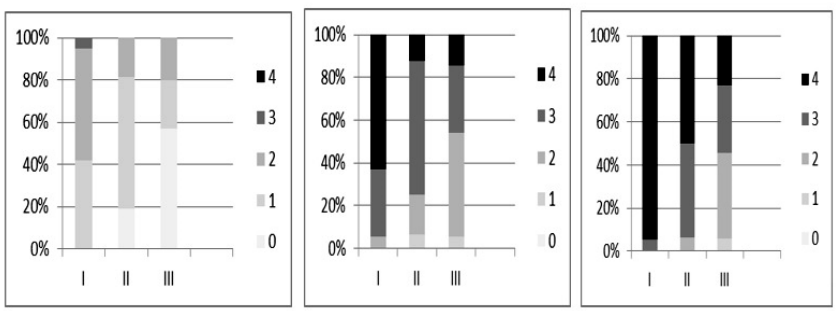


Figure 3. RUT proportional distribution during different observation stages by group.

Table 4. RUT result distribution by IOH.
χ^2 – chi squared, p – statistical significance, obtained using Monte Carlo method.

RUT	Total (n=70)	IOH	
		2 t/d (n=52)	1 t/d (n=18)
After 1 min., n(%):			
0	23(32.9)	21(40.4) *	2(11.1) *
1	26(37.1)	19(36.5)	7(38.9)
2	20(28.6)	12(23.1)	8(44.4)
3	1(1.4)	0	1(5.6)
	χ^2=8.533, df=3, p=0.032; * p=0.026		
After 20 min., n(%):			
1	3(4.3)	3(5.8)	0
2	21(30.0)	19(36.5) *	2(11.1) *
3	27(38.6)	21(40.4)	6(33.3)
4	19(27.1)	9(17.3) **	10(55.6) **
	χ^2=11.299, df=3, p=0.011; * p=0.047; ** p=0.002		
After 60 min., n(%):			
1	2(2.9)	2(3.8)	0
2	15(21.4)	14(26.9)	1(5.6)
3	19(27.1)	15(28.8)	4(22.2)
4	34(48.6)	21(40.4) *	13(72.2) *
	χ^2=11.299, df=3, p=0.08; * p=0.023		

Table 5. Total sample group RUT result distribution by CPITN.
χ^2 – chi squared, p – statistical significance.

RUT	CPITN	
	Healthy periodontal tissue (n=51)	≥4 mm PP (n=17)
After 1 min.,n(%):		
0	23(46.0) *	0 *
1	18(36.0)	7(38.9)
2	9(18.0) **	10(55.6) **
3	0	1(1.5)
	χ^2=17.77, df=3, p<0.001; * p=0.001; ** p=0.003	
After 20 min.,n(%):		
1	3(6.0)	0
2	20(40.0) *	1(5.6) *
3	20(40.0)	5(27,.8)
4	7(14.0) **	12(66.7) **
	χ^2=19.84, df=3, p<0.001; * p=0.003; ** p=0.001	
After 60 min., n(%):		
1	2(4.0)	0
2	15(30.0) *	0*
3	18(36.0) **	1(5.6) **
4	15(30.0) ***	17(94.4) ***
	χ^2=22.19, df=3, p<0.001;* p=0.011; ** p=0.02; *** p<0.001	

our hypothesis that primary *H. pylori* reservoir is oral cavity and extent of colonization is directly linked to oral hygiene and oral health. Similar findings were reported by Anand and Kamath et al., who reported that *H. pylori* habitat is soft and mineralized plaque, which sustains *H. pylori* integration [16,21]. RUT indicated *H. pylori* involvement in all patients. Test group with deep periodontal pockets exhibited statistically significantly stronger RUT indicator color change than group with healthy periodontal tissue. Latter group exhibited milder RUT indicator change after 1 min. with insignificant color change during further observations. Personal oral hygiene habits had a statistically significant impact on RUT outcomes – patients with lacking in personal oral hygiene had higher concentration of *H. pylori* in periodontal pockets and their RUT often indicated maximum pigmentation. These observations are in agreement with data published by Asqah et al. and Anand et al. [11,16].

CPITN and RUT exhibited statistically significant direct correlation – higher CPITN values meant more intense RUT color. These findings do not concur with conclusions by Kariya et al. that *H. pylori* is not pathogenic to oral cavity and needs anaerobic environment found in gastrointestinal tract to proliferate [22]. We believe that a low oxygen environment found in deep periodontal pockets is sufficient for *H. pylori* and the bacteria not only impacts other microorganisms in periodontal pockets, as described by Hu et al., but also plays a significant role by itself [6].

Based on the RSI findings we can partially confirm the second part of our hypothesis, but RSI score only allows to suspect GERD, but does not confirm the diagnosis. Conclusive GERD diagnosis can only be obtained by endoscopic evaluation combined with RFS. In our study, we were unable to find a statistically significant link between RFS, CPITN, RUT, patients' age or gender. Based on the latter, we can only speculate that patients with periodontal diseases and high CPITN score may exhibit symptoms attributed to GERD. Due to the lack of RSI, RFS and RUT statistically significant correlation we cannot agree to findings of Xue et al., which describe *H. pylori* presence in the oral cavity in patients with GERD [23]. Binary logistic regression analysis predicts that patients with periodontal diseases may exhi-

Table 6. Patient groups by RFS.

Group	n	A gr.	p	B gr.	p
I gr.	5 (14.3%)	3 (15.8%)	p=1.0	16 (84.2(%)	p=1.0
II gr.	30 (85.7%)	2 (12.5%)		14 (87.5%)	

bit high RSI scores, but that does not prove the colonization.

By comparing our findings to data found during literature review we cannot claim with great confidence that we absolutely agree with data published, which states that GERD is directly linked to *H. pylori* periodontal tissue involvement [3,11,16]. For this purpose, greater data pools must be gathered.

Conclusions

In this study, we have not found significant connection among gastroesophageal reflux, periodontal disease and *H. pylori* presence in periodontal tissues. On the other hand, we found a significant connection between GERD survey and periodontal pathology. Moreover, CPITN correlates with Reflux symptom index, patient's age and the amount of *H. pylori* colonies in periodontal pockets. It is important to mention that Reflux symptom index does not approve the final diagnosis of GERD. According to our results, it could be concluded that oral cavity is the primary reservoir of internal *H. pylori* colonization as rapid urease test data has a considerable relation with oral hygiene and periodontal diseases.

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HELICOBACTER PYLORI DIAGNOSTINĖS PRIEDANČIO AUDINIŲ IR GASTROEZOFAGINIO REFLIUKSO LIGOS SĄSAJOS

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Raktažodžiai: Helicobactery pylori, priedančio ligos, lėtinis periodontitas, gastroezofaginio refliukso liga.

Santrauka

Problemos aktualumas ir darbo tikslas. Virškinamojo trakto ir periodonto ligas sukelia keletas bendrų rizikos veiksnių. Vienas iš jų − sergant periodonto audinių ligomis, prarandami dantys, maistas prasčiau smulkinamas ir virškinamas, vystosi gastroezofaginio refliukso liga. Kitas − bakterinė mikroflora. Tai prastina individo gyvenimo kokybę. Temos nagrinėjimas aktualus dėl kontroversiškų nuomonių apie bendras šių ligų etiologijos bakterines priežastis ir sąsajas. Tyrimo tikslas − patvirtinti hipotezę, kad periodonto ir gastroezofaginio refliukso ligų išsivystymą sukelia bakterijos *Helicobacter pylori* kolonizacija periodonto audiniuose.

Medžiaga ir metodai. Tyrime dalyvavo 70 individų. Pagal anketinės apklausos duomenis išanalizuoti jų socialiniai – demografiniai rodikliai, kliniškai ištirti periodonto audiniai, apskaičiuotas periodonto ligų gydymo reikmių indeksas, atliktas greitasis ureazės testas *H. pylori* diagnozuoti, anketinis ir endoskopinis gastroezofaginės ligos ištyrimas. Tiriamieji suskirstyti į tris tiriamąsias (I; II; III) ir tris amžiaus (A; B; C) grupes. Statistinei duomenų analizei naudotas programinis paketas IBM SPSS Statistics 22. Tikrinant statistines hipotezes, reikšmingumo lygmuo $p<0,05$.

Rezultatai. Nustatytas reikšmingas ryšys tarp RSI ir CPITN ($p=0,004$), tačiau tarp RSI, amžiaus, lyties ir GUT $p>0,05$. Tarp RFS, amžiaus, lyties, GUT, CPITN reikšmingo ryšio nenustatyta ($p>0,05$). CPITN reikšmingai skyrėsi tarp tiriamųjų ($p<0,05$) ir amžiaus grupių ($p=0,005$). GUT reikšmingai susijęs su CPITN ($p<0,001$) ir burnos higiena ($p<0,05$).

Išvados. Šio tyrimo duomenimis, *H. pylori* kolonizacija periodonto audiniuose neturi reikšmės gastroezofaginės ligos išsivystymui, tačiau reikšmingai susijusi su priedančio audinių ligomis. Todėl galime teigti, kad burnos ertmė yra pirminė bakterijos *H. pylori* kolonizacijos vieta.

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