RESEARCH AND EDUCATION IN THE BATTLE AGAINST TOBACCO-RELATED ORAL DISEASES: TWO EXAMPLES FROM A DENTAL SCHOOL

PhD Thesis

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1. List of publications providing the basis and related to the topic of the thesis

List of publications providing the basis of the thesis:

I. **Antal M**, Forster A, Zalai Z, Barabas K, Spangler J, Braunitzer G, Nagy K. A video feedback-based tobacco cessation counselling course for undergraduates-preliminary results. *Eur J Dent Educ* 2013 Feb;17(1):e166-172. **IF: 1.012** (2012)

II. **Antal M**, Braunitzer G, Mattheos N, Gyulai R, Nagy K. Smoking as a permissive factor of periodontal disease in psoriasis. PLoS One. 2014 Mar 20;9(3):e92333. doi: 10.1371/journal.pone.0092333. eCollection 2014. **IF: 3.730** (2012)

Further publication related to tobacco use and/or oral health :

1. **Antal M**, Forster A, Zalai Z, Barabás K, Ramseier C, Nagy K. Attitudes of Hungarian dental professionals to tobacco use and cessation. *Cent Eur J Public Health* 2012 Mar;20(1):45-49.

2. **Antal M,** Ramseier A, Barabás K, Forster A, Zalai Z, Nagy K. A dohányzás megelőzése és a leszokás támogatásának lehetőségei. *Fogorv Sz.* 2012 Sep;105(3):99-103.

3. Nagy J, Braunitzer G, **Antal M**, Berkovits C, Novák P, Nagy K. Quality of life in head and neck cancer patients after tumor therapy and subsequent rehabilitation: an exploratory study. *Qual Life Res* 2013 Jun 4. [Epub ahead of print] PubMed PMID: 23733663. DOI:10.1007/s11136-013-0446-1. **IF: 2.412** (2012)

4. Mesmer C, Forster A, **Antal M**, Nagy K. Alsó részleges kivehető fogpótlást viselő páciensek mikrobiológiai és immunológiai vizsgálata peri-implantitiszes és egészséges kontrollcsoportba tartozó esetekben. (12 hónapos utánkövetés). *Fogorv Sz.* 2012 Jun;105(2):59-64.

5. Forster A, Velez R, **Antal M**, Nagy K. Width ratios in the anterior maxillary region in a Hungarian population – addition to the golden proportion debate. *J Prosthet Dent.* 2013; 110: 211-215. **IF: 1.724** (2012)

6. Baldea B, Furtos G, **Antal M**, Nagy K, Popescu D, Nica L. Push-out bond strength and SEM analysis of two self-adhesive resin cements: an in vitro study. *J Dent Sci.* 2013 Sep 1. 8(3):296-305 DOI:10.1016/j.jds.2013.01.007 **IF: 0.347** (2012)

7. Fráter M, Braunitzer G, Urbán E, Bereczki L, **Antal M**, Nagy K. In vitro efficacy of different irrigating solutions against polymicrobial human root canal bacterial biofilms. *Acta Microbiol Immunol Hung.* 2013 Jun; 60(2):187-199. doi:10.1556/AMicr.60.2013.2.9 **IF: 0.646** (2012)

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Book chapter:

Antal M, Nagy K (2013) : Tobacco use and cessation among dental professionals In: Péter Balázs: Increasing capacity for tobacco research in Hungary 2008-2013. 245 p. Budapest: Magyar Tudománytörténeti Intézet, 2013. pp. 151-174. (ISBN:978 615 5365 003)

2. Introduction

2.1. General introductory remarks and an outline of the thesis

Several chronic illnesses can be traced back to alcohol consumption, sedentary lifestyle, unhealthy nutrition and smoking. Smoking, in particular, is a global public health problem (Edwards, 2004). Tobacco use is identified as one of the most important risk factors for oral cancer, and the relevant WHO resolution (WHA60 A16) also encourages "steps to ensure that prevention of oral cancer is an integral part of national cancer-control programmes, and to involve oral-health professionals or primary health care personnel with relevant training in oral health in detection, early diagnosis and treatment" (Petersen, 2008). Oral cancer has a high rate of mortality, and more importantly, even if the patient survives, it can have a direct infulence on various oral functions like the ability to chew or to speak (Schuster et al., 2012), and also on the overall quality of life (Nagy et al., 2013). There were 14.1 million new cancer cases, 8.2 million cancer deaths and 32.6 million people living with cancer (within 5 years of diagnosis) in 2012 worldwide (GLOBOCAN 2012, WHO), which points to the outstanding importance of defining the controllable causative factors and devising preventive strategies.

Universities are distinguished venues of research, but also of education, and therefore in a university framework one can battle smoking-related oral diseases on two fronts: one is that of research where knowledge is produced, and the other is teaching, where it is disseminated. The present thesis follows this logic. First, after a review of the literature, a piece of clinical research is presented, where it is shown that smoking possibly has a permissive effect on the occurrence of severe periodontal disease in psoriatic patients. This is followed by the introduction of an educational method developed and introduced at our faculty, the Faculty of Dentistry at the University of Szeged. The method, which is now part of the curriculum of the faculty and also of other Hungarian and foreign dental faculties, was developed to enable dental students to offer tobacco cessation counseling by providing both theoretical knowledge and skills training.

2.2. Tobacco use and oral health

Tobacco use has been described to have several consequences on the human body, including several types of cancer. Of these, lung cancer has been the most common cancer in the world for several decades. In 2012, 1.8 million new cases have been registered worldwide, which is 12.9% of the incidence of all types of cancer (GLOBOCAN 2012, WHO). Cigarette smoking and other ways of tobacco use can be associated with approximately 75% of oral cancer cases (Rodriguez et al., 2004). The worldwide mortality of oral cancer was 82,000 in 1990, which, by 2010, reached 124,000 (Lozano et al., 2012). It must be noted, though, that even if the cancer patient survives (given that death is not a necessary consequence), the secondary deficits due to both the disease itself and the curative efforts can seriously affect oral functions (Schuster et al., 2012) and quality of life in turn. Although advanced prosthetics offer near optimal restorative solutions (Nagy et al., 2013), the survivor, having undergone the disease and its aftermath, still bears an immense psychosocial burden.

In Hungary, the incidence and mortality of cancers of the lip and oral cavity is the highest in Europe, and the incidence of lung, lip and oral cancers together - where smoking is the single highest risk factor - is the highest in the world (GLOBOCAN 2012, WHO).

Cancers of the lip and oral cavity, however, represent only a segment of the range of oral diseases associated with smoking. Population-based epidemiological studies have found that periodontitis is more common in smokers than in nonsmokers (Beck et al., 1990; Tomar et al., 2000), and the association between smoking and tooth loss has also been established (Hanioka et al., 2011). The association between tobacco use and acute ulcerative gingivitis (ANUG) was demonstrated as early as 1947 (Pindborg 1947). Smoking causes deeper periodontal pockets and greater atachment loss (Linden et al., 1994). The prevalence of furcation involvement is also higher in smokers, than in nonsmokers (Mullally et al., 1999), and the fact, that alveolar bone loss is greater in smokers has been documented since decades (Bergström et al., 1987).

Smoking can cause tooth discoloration (Alkhatib et al., 2005), and staining on the surface of dental restorations (Asmussen et al., 1986). There is evidence to suggest that smoking increases the chance of dental caries (Golpasand et al., 2013; Bendetti et al., 2013), but this is a matter of debate.

2.3. Links and associations between oral and extraoral diseases: the rationale behind the clinical study

Several oral conditions are associated with systemic diseases, and it is a well-known fact that the oral manifestations of such diseases can often be of diagnostic value.

For instance, avitaminosis due to malnutrition or metabolic can have severe oral consequences, such as stomatitis, cheilitis and glossitis (Field et al., 1995). Kwashiorkor, caused by protein deficiency, can lead to the well known noma (Heird et al., 2007) and the oral manifestation of amyloidosis can be macrgoglossia (Thibault et al., 2011). Plaque on the tongue or oral discolorations can be indicative of cardiovascular diseases, but these can also increase the likelihood of and/or aggravate periodontal disease. Furthermore, there is evidence to suggest that periodontal pathogens can modulate the process of atherosclerosis, thus playing a role in the development of coronary disease (Thomopoulos et al., 2011).

Numerous studies describe the interplay between systemic diseases and oral conditions. The most often studied of such conditions is periodontal disease. Periodontal disease is a destructive inflammatory disease of the supporting tissues of the teeth and is caused by specific microorganisms (Saini et al., 2009). These microorganisms, especially when accumulated in dental plaque, maintain a local immune reaction, which gives rise to the primary disease in situ. However, these pathogens, including Porphyromonas gingivalis, have the ability to invade the endothelium, whereby they easily access the circulation (Lundberg et al., 2008; Lu et al., 2009). The ensuing chronic bacteraemia can trigger pathological processes at distant sites, such as in the joints and the endocardium (Wegner et al., 2010), which eventually turn into clinical conditions like rheumatoid arthritis (Detert et al., 2010) and endocarditis.

Several studies demonstrated connection between periodontal disease and a considerable number of systemic conditions, and in most of these cases the connection cannot be put down simply to lasting bacteraemia. There is evidence to suggest, for instance, that moderate periodontal disease occurs more frequently among chronic kidney disease (CKD) patients as compared to individuals without the disease (Ioannidou et al., 2011). The same has been demonstrated in patients with diabetes

mellitus (Khader et al., 2006), and the effect appears to be the most pronounced in Type 2 cases (Pranckeviciene et al., 2014).

In the first part of the present thesis, a study about a similar, if less researched link is discussed. Since the first cross-sectional pilot study in 2010 (Preus et al., 2010) that raised the issue of the association between psoriasis and chronic destructive periodontal disease, surprisingly few studies have examined the claimed link. An increased risk of psoriasis among patients with chronic periodontitis has been detected (Keller et al., 2012) and Lazaridou et al. proposed that chronic plaque psoriasis patients should be closely followed-up by dentists for the adequate and early treatment of periodontitis, given an increased risk of the latter (Lazaridou et al., 2012). Evidence published so far unequivocally suggests that the two conditions are related. In our clinical study, we sought to test if the link does indeed exist (as suggested), but a new aspect was also added. As both smoking and psoriasis has been proposed as triggers and/or aggravating factors in periodontal disease (Beck et al., 1990; Tomar et al., 2000; Preus et al, 2010; Lazaridou et al., 2012), the new research objective was to find out about how smoking influences the severity of periodontal disease in the presence of psoriasis, that is, when both predisposing factors are present.

2.4. On the need to introduce tobacco cessation counseling into the dental curriculum

In the European Union, less than 10% of dentists smoke every day, and three quarters of all practices are totally smoke free (Allard, 2010), and in the US, some studies have reported smoking prevalence as low as 3% among dentists (Brothwell et al., 2004). However, in Eastern Europe, the situation is far from being that optimal. In our studies regarding this question, we found a decreasing, but still quite high percentage of smokers among dentists and dental students. In 2004, 35.5% reported to have smoked at least one day in the last month (Nagy et al., 2004), while in 2011, 22.3% of the dentists and 20.3% of the dental students defined themselves as smokers (Antal et al., 2012). In comparison, a 35% smoking rate was found among Jordanian dentists, and 83% of the smoking dentists were daily smokers (Burgan et al., 2003). In Laos, a smoking prevalence of 7.9% was reported among dental students (Sychareun ez al., 2013), while in Iran, 20.6% of a sample of dental students were found to use tobacco in different forms (Keshavarz et al., 2013). These data point out that smoking (and tobacco use in

general) among dental students and dentists shows a high geographical variability, and that Hungary is among the more affected areas. This means that the tobacco-related health-consciousness among Hungarian dental students and dentists is still not at the level that could shape their actual behavior. On one hand, this puts students' and dentists' health at risk, but on the other hand, a smoking dental professional cannot serve as a valid and congruent role model for cessation, which means that such professionals miss an important chance to support their smoking patients' health.

In terms of chances for cessation counselling, dentists seem to be in quite an advantageous situation; according to Garvey (Garvey et al., 1997), the expected quit rates are comparable with the succes of physicians. Furthermore, dentists are in a special situation, as they may be the first to notice damage caused by regular tobacco use, sometimes well before the actual manifestation of any kind of oral disease. Also, the dental office is a setting where it is „natural" to talk about oral problems that otherwise would possibly embarrass patients (such as bad breath or tooth discoloration). All in all, the dental office appears to be just the optimal venue for tobacco intervention in many respects, thus a possible and very promising strategy to reduce the prevalence of smoking-related oral diseases or even mortality is to increase the involvement of the dental team in tobacco prevention and cessation counselling (Walsh et al., 2005). To reach that end, the implementation of tobacco use- and cessation-related education for dental students and dentists is essential (Warren et al., 2005; Shibly et al., 2010).

3. Periodontal disease in psoriasis as determined by smoking: further evidence to support the immunomodulatory effect of cigarette smoke

3.1. Background and aims

Population-based epidemiological studies have found that periodontal disease is more common in smokers than in nonsmokers (Beck et al., 1990; Tomar et al., 2000). Beyond the obvious explanation that tobacco smoke is a local irritant, it has been shown that smoking favors colonization by specific periodontopathic bacteria (Kubota et al., 2011), modulates cellular immunity (Korn et al., 2005; Sopori, 2002; Sulivan et al., 2005) and favors the development and aggravation of autoimmune and immune-mediated conditions (Masdottir et al., 2000; Mathews et al., 1973), including psoriasis (Emre et al., 2012). Smoking, therefore, appears to be a risk factor for both periodontal disease and psoriasis. What is more interesting is that psoriasis and periodontal disease seem to be associated themselves. It was Yamada and colleagues in 1992 who first reported a case of psoriasis in which exacerbation of the cutaneous disease was accompanied by gingival epithelial changes and periodontal bursts (Yamada et al., 1992), but the first blinded, case-controlled study on the assumed association was published only eighteen years later by Preus and colleagues (Preus et al., 2010). This was the first study to seriously raise the issue of the possible association between the two conditions, followed by the studies of Lazaridou et al. (Lazaridou et al., 2013) and Keller and Lin (Keller et al., 2012), both of which corroborated the findings of the Preus group.

It is established that the immunopathogenesis of psoriasis (Schon et al., 2005) and the aggravation of periodontal disease (Ohlrich et al., 2009) is linked to altered T-lymphocyte - mediated immunity. Periodontal disease turns progressive when B-cell/plasma cell responses become dominant in the periodontal inflammatory process, which is preceded by the oligoclonal expansion of Th_2 lymphocytes. These lymphocytes stimulate B-cells and induce humoral immune responses by secreting interleukins IL-4, IL-5 and IL-10 (see Table 1). It is to be noted that Th_1 cells are also capable of stimulating B cells by secreting INF-γ. This can be an alternative way of shifting the immune response toward B cell dominance, especially in the context of suppressed T cell responses (as it happens in the oligoclonal expansion of Th_2 cells). Th_1 cells are

stimulated by IL-6, IL-8, IL-12, IL-18, TNF-α and INF-γ, the levels of all of which were found to be elevated in the serum of patients with chronic plaque psoriasis (Arican et al., 2005). It might not be too far-fetched to hypothesize that psoriasis can aggravate periodontal disease because enhanced Th_1 INF-γ secretion furthers the effects of Th_2 dominance. However, the clarification of the molecular level basis of the link between the two conditions was no subject of this study.

As tobacco smoking also interferes with immunity, favors colonization by periodontopathic bacteria and acts as a local irritant, we hypothesized that smoking may act as a trigger or permissive factor of periodontal disease in patients suffering from psoriasis. To test this hypothesis, the prevalence and severity of periodontal disease was assessed in a group smoking and non-smoking psoriasis patients and a group of smoking and non-smoking psoriasis-free controls. The specific question we sought an answer to was whether periodontal disease occurs more often and in a more serious form in smoking psoriasis patients than in non-smoking psoriasis patients. The primary aim of the study, therefore, was to find evidence for or against the hypothesized permissive role of smoking.

3.2. Methods

Data for the study were gathered in two stages: first, participants were administered a questionnaire (non-clinical stage), and then they underwent a full intraoral examination (clinical stage).

3.2.1. Participants and non-clinical data gathering

A hospital-based case-control study was conducted in 2012. Participants (n=82) were selected from the patients of the Psoriasis Outpatient Unit of the Department of Dermatology and Allergology, University of Szeged. The control group (n=89) was recruited from people attending mandatory lung screening in the same city and the same period, on a voluntary basis. Controls were age-matched to patients, but the sex ratios were also almost identical (see Table 2.). Required sample size was calculated with G*Power 3.1.5. (University of Kiel, Germany), a software designed especially for

statistical power and sample size computation (Faul et al., 2007). The software allows the computation of achieved statistical power (post-hoc) and required sample size (a priori). As mostly categorical variables were to be analyzed, a priori sample size estimation was performed for crosstabs/chi square/contingency tables, with the following input parameters: effect size (w): 0.5; α: 0.05;power (1-β): 0.85; DF: 3. Required sample size turned out to be n=50, but much more cases were available, whereby, also expecting dropout, it was decided that 100 cases and 100 controls would be examined. This way, even with the dropout of 18 cases and 11 controls, a statistical power over 0.9 was achieved.

CLINICAL STAGING (Fernandes et al., 2009)	PATHOLOGY/PATHOPHYSIOLOGY (Ohlrich et al., 2009)
1.NO CLINICAL SIGNS- no clinical attachment loss (CAL) or bleeding on probing (BOP)	(NO LESION- NOT CLASSIFIED EXPLICITLY IN OHLRICH ET AL.)
(GINGIVITIS-NOT CLASSIFIED EXPLICITLY IN FERNANDES ET AL.) (CPITN 1)	**1. INITIAL LESION** – up to 4 days following plaque accumulation. Polymorphonuclear leukocytes (PMN), complement activation, loss of connective tissue. Mast cells release tumor necrosis factor alpha, PMNs migrate into the gingival sulcus, but as the bacteria are protected by the biofilm, abortive phagocytosis occurs. PMNs release lysosomal contents, which leads to further tissue destruction.
2.EARLY PERIODONTITIS- CAL ≥1 mm in ≥2 teeth (CPITN 2)	**2.EARLY/STABLE LESION**- 7-21 days after plaque accumulation, clinically evident approximately from day 12. Dominantly macrophages and lymphocytes ($CD4^+$:$CD8^+$ 2:1). Perivascular inflammatory infiltrate. Intercellular spaces between epithelial cells widen, bacterial products infiltrate the gingival tissues at a higher rate. Escalation of response. If plaque removed, tissue remodeling can take place.
3.MODERATE PERIODONTITIS- 3 sites with CAL ≥4 mm and at least 2 sites with probing depth (PD) ≥3 mm (CPITN 3)	**3. ESTABLISHED OR PROGRESSIVE LESION**- dominantly a B cell/plasma cell response. High levels of IL-1 and IL-6: connective tissue loss, breakdown of bone.
4. SEVERE PERIODONTITIS- CAL ≥6 mm in ≥2 teeth and PD ≥5 mm in ≥1 site (CPITN 4)	**4. ADVANCED LESION**- Overt loss of attachment. High levels of IL-1, TNF α and PGE_2 stimulate fibroblasts and macrophages to produce matrix metalloproteases. The junctional epithelium progresses in apical direction (deepening periodontal pocket). Oligoclonal Th_2 ($CD4^+$) dominance.

Table 1. The applied clinical staging and the corresponding pathological/pathophysiological changes. The approximate CPITN stage is also given (in brackets).

	PSORIASIS		CONTROL
Age (mean±SD, years)	50.9±13.4		50.3±13.7
smoker subsample	48.5±14.0		43.3±12.4
nonsmoker subsample	54.7±12.9		52.9±14.7
Sex ratio F:M (n(%):n(%))	45(55%):37(45%)		44(49%):45(51%)
smoker subsample	24(60%):21(40%)		12(50%):12(50%)
nonsmoker subsample	23(49%):24(51%)		33(51%):32(49%)
Smoking (n(%))	35(43%)		24(27%)
Periodontal status in smokers (n)	**healthy:** -	reference category	**healthy:** 10
	early: 5	p<0.001	**early:** 8
	moderate: 13	p<0.001	**moderate:** 4
	severe: 17	n.s.	**severe:** 2
Periodontal status in nonsmokers (n)	**healthy:** 10	reference category	**healthy:** 25
	early: 13	n.s.	**early:** 19
	moderate: 18	p=0.05	**moderate:** 17
	severe: 6	p<0.05	**severe:** 4

Table 2. Descriptive statistics of the study and control groups with the characteristics of the subsamples. To keep the table transparent, percentages have been rounded to whole numbers. Note that no periodontally healthy smoker psoriasis patients were found. A trend analysis (χ^2 with adjusted residuals) revealed a significant trend (χ^2 =48.074, p=0.000), with non-smoker controls being the less likely to have any stage of periodontal disease and smoker patients being the most likely to have the severe stage. Significance values refer to the comparison of odds with healthy as the reference category (see also Table 3.).

Patients were eligible for the study if they were diagnosed with psoriasis (defined as ICD-10 L40.0-L40.9) by a dermatologist. The diagnosis of psoriasis was set up earlier than the beginning of this study in all cases, whereby dermatological examination was not regarded as a direct part of this study, neither was it listed on the informed consent forms as such. Exclusion criteria for both groups were determined based on the literature of the subject and included obesity (BMI≥30), excessive alcohol consumption, drug abuse, diabetes mellitus, oestrogen deficiency, diseases causing neutropenia and

local or systemic inflammatory conditions (other than psoriasis) (Genco, 1996; Genco et al., 2013). Dropout resulted partially from meeting one or more of the exclusion criteria (especially excessive self-reported alcohol consumption), but there were subjects who quit by their own will before the intraoral examination, and a number of subjects failed to provide tobacco use data.

Demographic and tobacco use data were collected by a questionnaire. Medical information on both controls and psoriasis patients was extracted from patient files and hospital records. Participants were divided into smoker and non-smoker groups, based on their self-reported current tobacco use. To minimize desirable response tendency in this crucial aspect, smoking was not pointed out to the participants as the target variable until all study procedures had been finished.

The study was approved by the Human Ethics Review Board of the University of Szeged (approval Nos. 2848 and 2879), and the study design conformed to the Declaration of Helsinki in all respects. Written informed consent was obtained from all participants.

3.2.2. Disease staging and intraoral examination

The clinical staging of periodontal disease is still a matter of debate, even though the progression of the disease is well established in pathological terms (Ohlrich et al., 2009). The most widely used classification is the Community Periodontal Index of the WHO (CPITN) (Barmes, 1994). Applicable as it may be for clinical purposes, CPITN defines rather wide categories, which carries the risk of oversampling. Therefore we decided to use the more detailed (and thus more strict) staging proposed by Fernandes and colleagues (Fernandes et al., 2009). The staging requires the following parameters to be recorded: the number of missing teeth (excluding third molars), Plaque Index (PI; also known as the Silness-Löe Index), bleeding on probing (BOP; the presence or absence of bleeding within 15 sec after probing), probing depth (PD; in millimeters), and clinical attachment level (CAL; to describe the position of the soft tissue in relation to the cemento-enamel junction).

In order to record the above mentioned parameters, all patients received a full mouth periodontal examination, performed by a periodontist. BOP, PD and CAL were examined six sites per tooth (mesio-buccal, buccal, disto-buccal, disto-lingual, lingual,

mesio-lingual), with the exception of third molars. Williams probes (Hu-Friedy Manufacturing Co., Chicago, USA) were used. Table 1 shows the categories of the applied staging and the corresponding pathological/pathophysiological status.

3.2.3. Statistical Analysis

The study and control groups were compared by smoking status. To express the probability that a member of a given group develops a given clinical degree of periodontal disease, multinomial logistic regression was conducted and the odds ratios were calculated. In the multinomial model, disease severity (healthy, early, moderate, severe) was defined as the outcome variable, smoking status (smoker or non-smoker), and group (psoriasis or control) were defined as factors, and smoking intensity (expressed as the number of cigarettes smoked per day) was defined as a covariate. Descriptive statistics and odds ratios were calculated in SPSS 17.0 (SPSS, Inc., Chicago, USA).

3.3. Results

3.3.1. The population

Table 2. shows the descriptive statistics of both the study and the control groups. Females were slightly over-represented in the psoriasis group (n_{female}=45, n_{male}=37) while in the control group the sexes were represented almost perfectly equally (n_{female}=44, n_{male}=45). Crosstab analysis with χ^2 test was conducted to find out if sex was significantly associated with disease severity, but the association turned out to be non-significant for both patients (χ^2 =5.184 p= 0.189) and controls (χ^2 = 3.029, p=0.387), whereby sex was not included in the multinomial regression. Patients and controls were age-matched (mean age for the entire sample: 50.6 years). The division into four groups by diagnosis (psoriasis or control) and smoking status (yes or no) for the purposes of the analysis did not change these parameters significantly, that is, the ratios remained representative of the whole sample. Group membership (i.e. patient or control) was shown to be significantly associated with disease severity (χ^2= 27.337, p=0.000).

In the psoriasis group 35 persons defined themselves as smoker (43%). The number of smokers was lower among the control group, only 24 persons defined themselves as smoker (27%). In the psoriasis+smoker group no patient could be categorized as periodontally healthy. 14% of this group had early, 37% moderate and 49% severe periodontal disease. Among non-smoker patients 21% were classified as healthy, 28% as having early, 39% as having moderate and 12% as having severe periodontal disease. 42% of smoking controls was periodontally healthy, 33% had early, 17% moderate and 8% severe periodontal disease. Nonsmoking controls were periodontally healthy in 38%, 29% had early periodontal disease, while in 26% of the cases the disease presented in its moderate and in 7% in its severe form. These data reflect opposing tendencies between smoker patients and non-smoker controls, that is, while in the smoker patient subsample the severe stage of periodontal disease is the most frequent, and no periodontally healthy patient is seen, in the non-smoker control subsample exactly the opposite can be observed (Figure 1.).

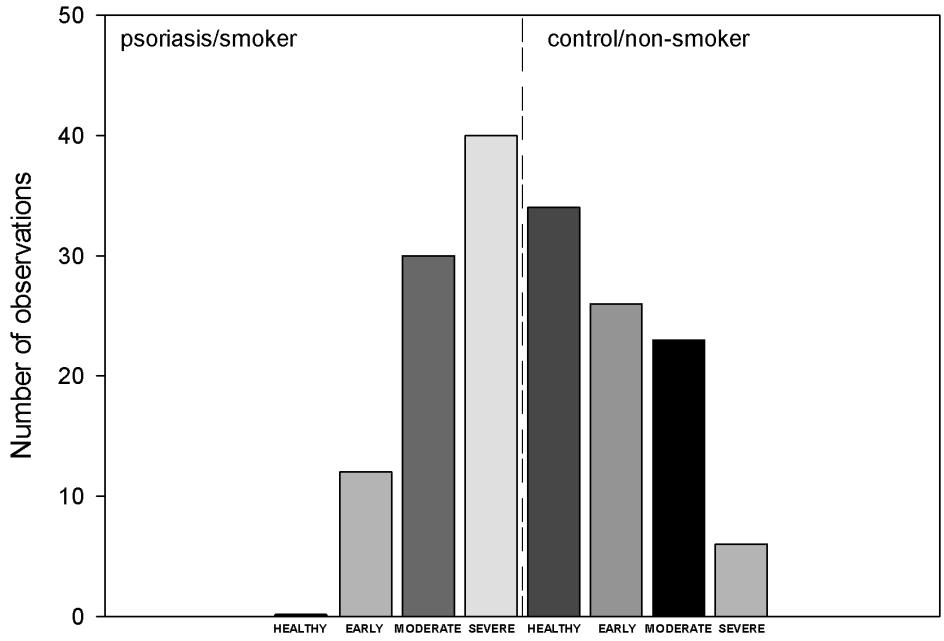

Figure 1. Opposing tendencies: while in the smoker patient subsample the severe stage of periodontal disease was the most frequent, and no periodontally healthy patient was seen, in the non-smoker control subsample exactly the opposite was observed.

3.3.2. Smoking history and smoking intensity

In terms of smoking history (expressed in years) and their intensity of smoking (expressed as the number of cigarettes smoked per day), controls and psoriasis patients turned out to be quite similar. Smoking controls had smoked for a mean of 20.9 years (mode: 20 years, SD: 11.97), and they smoked a mean of 16 cigarettes a day (mode: 20 cigarettes, SD: 11.67). Smoking patients had smoked for an average of 16.57 years (mode: 20 years, SD: 12.37), and they smoked an average of 13 cigarettes a day (mode: 20 cigarettes, SD: 8.57). Indeed, the two groups exhibited no statistically significant difference in either of these parameters (MWU=315, n1=35, n2=24, p=0.17, years; MWU=758, n1=35, n2=24, p=0.57, cigarettes). The crosstab analysis revealed that smoking status in itself was significantly associated with disease severity only in patients (χ^2= 18.258, p=0.000). In controls, no significant association was seen (χ^2= 0.935, p=0.812). However, when the data were re-coded so that the outcome variable was not disease severity, but the occurrence of any stage of the disease, the association was highly significant (χ^2= 89.000, p=0.000). As for smoking history and intensity, the built-in likelihood ratio test of the multinomial logistic regression module of SPSS revealed that smoking history had a significant effect on disease severity (χ^2 = 11.099, p=0.011), but the effect of smoking intensity was not significant (χ^2= 30.862, p=0.504).

3.3.3. Odds Ratios

Odds ratios with significance and confidence intervals are given in Table 3. Odds ratios were calculated in a multinomial logistic regression model. Smokers and controls were compared stagewise, according to the applied classification. The factors of the model were smoking status (smoker or non-smoker) and group (patient or control), and the covariate was smoking history (in years). Five comparisons were done, that is, non-smoker controls were compared with smoker controls (OR_{EARLY}: 1.053 , $OR_{MODERATE}$: 0.588, OR_{SEVERE}: 1.250), with non-smoker patients (OR_{EARLY}: 1.944 , $OR_{MODERATE}$: 2.647, OR_{SEVERE}: 4.373) and with smoker patients (OR_{EARLY}: 1.711 , $OR_{MODERATE}$: 2.500, OR_{SEVERE}: 24.278), smoker patients were also compared with non-smoker patients (OR_{EARLY}: 6.082 , $OR_{MODERATE}$: 1.712, OR_{SEVERE}: 4.480), and smoker controls were compared with smoker patients (OR_{EARLY}: 1.904 , $OR_{MODERATE}$: 9.900, OR_{SEVERE}: 2.589).

Significantly higher odds of developing moderate ($p=0.05$) and severe ($p<0.05$) periodontal disease were found when non-smoker patients were compared against non-smoker controls (the periodontal risk of psoriasis alone). The highest odds ratio resulted from the comparison of smoker patients with non-smoker controls (24.278, $p<0.001$), which is 4.43 times higher than the combination of the individual odds of smoking (1.250, n.s.) and psoriasis (4.373, $p<0.05$). It must be noted, though, that the high odds ratio had a rather wide confidence interval. When compared with non-smoker patients, smoker patients' odds of developing periodontal disease was significantly higher at all three stages ($p<0.001$). Finally, as compared to smoker controls, smoker patients' odds of developing early or moderate periodontal disease were also significantly higher ($p<0.001$), but no significant difference was observed regarding the severe stage of the disease.

The covariate smoking history was significantly associated with disease severity in both patients ($\chi^2 = 12.204$, $p=0.014$) and controls ($\chi^2 = 9.869$, $p=0.02$). The odds ratios were positive for the moderate and severe stages in both groups ($OR_{CONTROL,\ MODERATE}$: 1.145, CI:0.999-1.312; $OR_{CONTROL,\ SEVERE}$: 1.164, CI: 0.963-1.408; $OR_{PATIENT,\ MODERATE}$: 1,112, CI: 0.939-1.318; $OR_{PATIENT,\ SEVERE}$:1.185, CI: 0.998-1.406), indicating that a longer smoking history significantly increases the odds that the moderate or severe stage of the disease occurs, as compared to the early stage.

COMPARISON	STAGE	OR	Sig.	95% CI (lower ;upper)
SMOKER CONTROLS (vs. non-smoker controls)	**early**	1.053	n.s.	0.394;3.177
	moderate	0.588	n.s	0.158;2.187
	severe	1.250	n.s	0.197;7.942
NON-SMOKER PATIENTS (vs. non-smoker controls)	**early**	1.944	n.s.	0.706;5.353
	moderate	2.647	*p=0.05*	0.985;7.113
	severe	4.373	*p<0.05*	1.114;17.169
SMOKER PATIENTS (vs. non-smoker controls)	**early**	1.711	n.s	0.618;4.732
	moderate	2.500	n.s	0.924;6.761
	severe	24.278	*p<0.001*	5.207;113.189
SMOKER PATIENTS (vs. non-smoker patients)	**early**	6.082	*p<0.001*	1.516;2.440
	moderate	1.722	*p<0.001*	3.535;3.689
	severe	4.480	*p<0.001*	1.651-6.851
SMOKER PATIENTS (vs. smoker controls)	**early**	1.904	*p<0.001*	1.202; 3.016
	moderate	9.900	*p<0.001*	1.568;6.262
	severe	2.589	n.s.	2.108;2.695

Table 3. Odds ratios by clinical staging. In each case, the ratios express the odds that a member of the given category (printed in capitals) develops the given stage of periodontitis in comparison with the members of another category (in brackets). Significance levels and 95% confidence intervals (with lower and upper limits) are also given.

3.4. Discussion

In the present study it was demonstrated that psoriasis patients who smoke are at an approximately sixfold higher risk of developing severe periodontal disease, as compared to psoriasis patients who do not smoke. Psoriasis in itself increases the likelihood of severe periodontal disease to 4.373 (as compared to non-smoker controls), while smoking appears to increase this to 24.278. In other words, the risk of severe periodontal disease in psoriasis is approximately six times higher in smokers than in non-smokers. It would be reasonable to infer that this increase in risk is merely the

result of the individual risk factors adding up. To test that possibility, odds ratios were combined and the synergy factor was calculated.

Combined odds expresses the probability of a given condition if all combined risk factors are present. For samples of similar size, combination of odds is done by averaging their logs ($logOR_1/logOR_2$). For psoriasis alone (4.373) and smoking alone (1.250), the combined odds of severe periodontal disease is 6.60, far below the observed 24.278. From this it may be conferred that what is observed in smoker patients is not simply the odds of psoriasis and smoking added up. Computation of the synergy factor corroborates this suspicion. The synergy factor was devised by Cortina-Borja and co-workers (Cortina-Borja et al., 2009) to allow the estimation of whether two factors are in a positive or negative interaction in terms of bringing about a given condition, based on their individual odds. The factor is calculated as $SF = OR_{12}/(OR_1 \times OR_2)$, where OR_{12} expresses the combined odds of the examined factors. SF=1 expresses the simple addition of the odds, SF<1 means negative interaction (antagonism) and SF>1 denotes positive interaction (synergy). The value of the synergy factor for the odds of psoriasis and smoking is 1.2, indicating a synergistic effect. It seems, therefore, that smoking acts as a permissive factor.

It was also found that the association between smoking status and disease severity is significant only in the patient group, which is also to support that exposure to cigarette smoke and psoriasis act together toward the aggravation of periodontal disease. The lack of significant association, however, should not be interpreted as evidence that cigarette smoke alone does not cause periodontal disease. Evidence suggests that it does (Tonetti 1998), and the additional analysis also showed that when the target variable is the occurrence of periodontal disease, the association is highly significant. From this it might be inferred, that smoking increases the odds of periodontal disease in both controls and psoriasis patients, but psoriasis patients are perdisposed to the more severe forms of the disease.

A further interesting result is the significant association of smoking history with the occurrence of the moderate and severe forms of periodontal disease in both groups. Together with the lack of significant association with the number of cigarettes smoked per day, it suggests that the key factor in the proposed permissive role of cigarette smoke is chronic exposure, apparently regardless of how heavy a smoker one is.

As no study before has dealt with the effects of smoking on periodontal disease severity

in psoriasis, it is beyond our limitations to give an exact explanation of these findings. However, enough is known about the pathogenesis of psoriasis and periodontal disease and the pathophysiological effects of smoking to allow the articulation of the elements of a hypothesis, which we briefly delineate here.

As pointed out by Ohlrich and co-workers, the most readily observable difference between the early and later stages of periodontal disease is that in the later stages the inflammatory response is dominated by B lymphocytes, instead of T lymphocytes (Ohlrich et al., 2009). It is in the context of this exaggerated and maladaptive B cell/plasma cell response that periodontal tissue destruction and bone breakdown occurs. Our propsed hypothesis, therefore, centers around this T-to-B shift, which appears to be the key event in the aggravation of periodontal disease, and how cigarette smoke might play a permissive role in the process. Here we concentrate only on a putative immunological backrgound, while we understand that additional factors, like the detrimental effect of smoking on gingival circulation (Mavropoulos et al., 2007) can also contribute to the end result.

It is well established that psoriasis is associated with a millieu of inflammatory mediators that favors a shift toward B cell responses. Serum levels of the inflammatory mediators IL-6, IL-8, IL-12, IL-18, TNF-α and INF-γ were found to be elevated in plaque psoriasis [17]. These mediators are all documented to activate B cells (Hirano et al., 1986; Falkoff et al., 1983; Durali el at., 2003; Rieckmann et al., 1991). TNF-α is also a potent stimulant of fibroblasts and macrophages, which, upon activation, release matrix metalloproteases (Ohlrich et al., 2009). As mentioned before, periodontal disease itself is associated with Th_2 expansion. This, on one hand, suppresses T cell responses, but Th_2 cells, in the presence of antigen, can stimulate B cells too (Del Prete, 1998). Therefore, it might be argued that psoriasis increases susecptibility to destructive immune responses in general by shifting the immune response profile toward B cell responses, which would explain the original observation - also corroborated by this study- that patients with psoriasis develop periodontal disease more often than psoriasis-free individuals.

How, then, might cigarette smoke boost this effect? We propose that toll-like receptor 4 (TLR 4) can play a key role here. Toll-like receptors are preferentially expressed on immune cells, and mediate immune responses. TLR4 on the surface of Th1 cells

activate these cells upon binding to bacterial lipopolysaccharides to secrete INF-γ , a potent B cell activator (Netea et al., 2005). Normally, this is part of the immune response to bacterial colonization of the gingival sulcus. However, if TLR 4 is overexpressed, the response may become exaggerated and maladaptive. Pace and co-workers demonstrated that cigarette smoke causes upregulation of TLR4 in airway epithelial cells of smokers with COPD, and they proposed that this upregulation might be the reason why a dominantly Th1-regulated immune response is seen in the airways of COPD patients (Pace et al., 2008). The authors also demonstrated that cigarette smoke increased epithelial cells' ability to bind bacterial lipopolysaccharides (Pace et al., 2008). TLR4 upregulation was demonstrated in the psoriatic skin too (Curry et al., 2003). It is not known if cigarette smoke has the same effect in the periodontal tissues (and on the surface of Th1 cells), but it seems plausible to assume that TLR 4 upregulation can be the key step in the destructive T- to B- shift in periodontal disease in the context of an already B-biased inflammatory mediator environment. Based on the literature, it can be proposed that the examination of the effects of cigarette smoke on TLR4 expression in the gingival tissues could be a logical next step toward the understanding of our observations, which we are planning to undertake.

4. Introducing tobacco cessation counseling into the dental curriculum

Considering that, as mentioned under 2.2. and 2.4., Hungary is a leading country in terms of tobacco-related morbidity and mortality, with a notable percentage of smokers among dental students and dentists, and that the dental office could be a distinguished venue of tobacco use interventions if well-prepared professionals were available who could also serve as a role model, we decided to introduce a new element into the curriculum of the Faculty of Dentistry at the University of Szeged. This new curricular element was intended to provide dental students both theoretical and practical training in cessation counseling, so as to enable them to offer such counseling and also to increase their tobacco-related health-consciousness. The second part of the thesis describes the methodology of this new curricular element, and also discusses the initial experiences.

4.1. Cessation support by oral health professionals

The importance of cessation support interventions done by oral health professionals is getting widely recognized. The dental practice setting has been described as providing a unique opportunity to assist tobacco users in achieving abstinence or even quitting tobacco (Christen et al., 1990; Ramseier et al., 2010; Needleman et al., 2010). More specifically, evidence suggests that behavioral interventions for tobacco cessation conducted in the dental office and involving an oral examination component may increase the quit rates among cigarette smokers (Carr et al., 2012). It is true that quit rates are similar to those achieved by physicians and other healthcare providers (Warnakulasuriya 2002), but oral health professionals recognize patient tobacco use more readily (Block et al., 1999). Furthermore, dentists are in a special situation, as they may be the first to detect damage caused by regular tobacco use, sometimes well before the actual manifestation of any kind of oral disease. Thus, a possible and very promising strategy to reduce the prevalence of smoking- related oral diseases or even mortality is to increase the involvement of the dental team in tobacco prevention and cessation counseling (Walsh et al., 2005).

Several tobacco cessation support methodologies are known, from brief advice and quitline referral (Ebbert et al., 2007) to video-based cessation support with phone follow-up (Andrews et al., 1999; Severson et al., 2009). Motivational Interviewing (MI) is often part of these approaches (Lando et al., 2007). MI is a patient-oriented method aimed at increasing the patients' inner motivation to change their behavior by exploring and resolving ambivalence (Miller et al., 2002). As dentists have only a limited timeframe at their disposal for cessation support, it has been proposed that the shorter, questionnaire-based form of MI, better known as "Brief Motivational Interviewing" (BMI) should be used in the dental practice (Rollnick et al., 1997). If BMI is used in the framework of the 5As approach (ask, advise, assess, assist, arrange follow-up) (Needleman et al., 2010) and pharmacological augmentation is also applied (such as nicotine replacement therapy), quit rates up to 15% can be achieved in a three-month period, compared to the 9% seen with other cessation support approaches (Binnie et al., 2007).

4.2. Smoking prevention in the dental practice: a new course in the curriculum

With all the information available in the literature and our experience of dentist-patient communication in mind, we proposed a new course titled „Smoking prevention in the dental practice" in the period 2008-2009. The course proposal was accepted by the Education Committee of the dental school in November 2009, and the first course started in February 2010, the spring semester of the academic year 2009-2010. The Semmelweis University in Budapest followed suit in 2011. In brief, the course consists of a sixty-minute-long theoretical class per week and four practice sessions. During the practice sessions students have the chance to practice various dentist-patient communication situations with professional actors. These encounters are recorded and the video is analyzed. So far 70 students have finished the course.

4.2.1. Aims of the course

The ultimate aim of the course is that dental students be able to help patients with behavioral and pharmaceutical cessation techniques. A further -similarly important- goal is to provide students the theoretical basis and practical skills that will make them

congruent role models for cessation. Throughout the course, three tenets are emphasized, so that students can appreciate the importance of cessation support as a dentist's task: the first is that although 75%-85% of smokers want to quit smoking (Garvey et al., 1997), only a small percentage can succeed without professional help. Second, that approximately 17% of smokers are ready to give up smoking within the next six months at any given time (Walsh et al., 2005). Third, that the relationship between the dentist and the patient opens up a platform for personal guidance and consultation. This increases the probability of successful cessation (Warren et al., 2008). The message behind these three tenets is that there is a real need for support, there is readiness to accept it, and that the position of a dentist is especially suitable for efficient support. A more general aim of the course as a whole is to sensitize participants to the problem of smoking-related diseases and the importance of smoking prevention and smoke-free life.

4.2.2. The team

The educational team of this program is a multidisciplinary one, which involves a professor of dental sciences, two dentists, a specialist of preventive medicine, a general practicioner, a communication specialist, and four specially trained professional actors.

4.2.3. Course structure

The course has been designed in a problem-based learning (PBL) framework, which, particularly over the past two decades, has become increasingly common in dental education (Shibly, 2010; Davis et al., 2010). It comprises three main, consecutive phases, as described below (see Table 4. for an item-wise course description).

The first phase is a theoretical preparation phase. In this first phase students acquire a substantial amount of background knowledge on various aspects of nicotine addiction in physiological and pathophysiological terms, and about psychological tobacco dependence as well. This phase of the course follows the lines laid out by Ramseier et al. (Humair et al., 2003). Several topics of the theoretical sessions are covered by guest lecturers, so that students have the chance to learn directly from professionals of the different fields involved.

Topic	Allocated time	Course phase
The epidemiology of smoking	2 hours	TP
National and international epidemiology, with special emphasis on oral diseases	2 hours	TP
The chemistry of cigarette smoke. The pathophysiology of smoking.	2 hours	TP
The behavioral background of smoking	2 hours	TP
Smoking as addiction	1 hour	TP
Risk groups	1 hour	TP
Behavioral modification	1 hour	PP
Clinical interventions and preventing relapse	1 hour	PP
Options for assisting tobacco cessation. Pharmacologically and/or psychologically supported cessation.	4 hours	PP
BMI/5As	2 hours	PP
Individual consultation	1 hour	IP
Consultation on the phone	1 hour	IP
Video simulation	4 hours	IP
Course evaluation	1 hour	-

Table 4. Course structure. TP: theoretical preparatory phase; PP: practical preparatory phase; IP: interventional practice

The second phase comprises practical preparation. In this phase, students practice skills on each other after that they are trained how to use practical interventional techniques, most importantly the technique of MI and BMI. As the efficacy of MI and BMI in connection with dependent behavior is supported by several studies (Logan et al., 1999; Andersen, 2007; Benowitz, 2008; Rollnick et al., 1999), we put emphasis on its knowledge, both in theory and practice. Students learn that the most important aspects they have to assess are patients' readiness to change (Cornuz et al., 2002), how important that change for the patient is (Rollnick et al., 1997), and how much the patient

is convinced that the change can actually be made (Koerber et al., 2003). Students are familiarized with the evaluation of the BMI questionnaires, and they are also taught the patient guidance styles to be used with BMI (Fiore et al., 2008).

Students also obtain information about evidence-based methods for tobacco use cessation, including the well-established 5As Method (WHO, 2012) and its combination with pharmacotherapeutic solutions.

In the practical part of this phase, students get a written manual, which describes the BMI protocol step-by-step, summarizes the 5As Method, and contains scenario scripts, which describe the ways, concepts and methods of tobacco cessation support. With the help of this manual, students have the chance to practice their newly acquired skills in pairs, based on made-up situations

In the third, intervention practice phase, students participate in an interactive, videofeedback-supported communication training, which is the real methodological novelty of this approach. To our knowledge, our Faculty has been the first to integrate a videofeedback- and simulation-based undergraduate tobacco cessation counseling course into a university curriculum worldwide.

With the help of professional actors real-life dental encounters are simulated, these encounters are recorded and then evaluated together with the actor, students and teachers. The simulated situations are complex ones, based on expert experience. The situations center around three basic patient attitudes: seeking help with cessation, hesitation, and refusal (see Table 5 for an example).

Both verbal and nonverbal skills are evaluated, so that students are able to learn about and gain a deeper understanding of the particularities of communication in clinical situations through direct involvement. Written feedback (in the form of an evaluation sheet) is given by both a teacher and the actor playing the patient. Points to be evaluated by the teacher include various aspects of verbal and nonverbal communication, history taking, and the ability to getting medical information through, while the patient sheet gives an opportunity to aspects like friendliness, openness toward the psychological concerns of the patient or conversational style. Both the teacher and the actor rate each particular aspect from 0 to 2 (See Table 6. for a selection of items and evaluation). Students' videos are also discussed in a group training session, where expert feedback on desirable communicative elements and ones to be avoided is provided.

Situation # 3.: The patient who refuses to quit
Description: It is the patient's first appointment. He is a typical hedonist who considers harmful habits as part of enjoying life, so much so that these almost seem to be part of his identity. Several years of drinking and smoking have done persistent harm to his oral cavity, which is aggravated by poor oral hygiene. His teeth are discolored and loose, his gums are atrophic. He seeks help with these problems, however, quitting smoking is the last thing he wants to hear about, what is more, he seems to take even the mention of it as an offence. **Dental status:** gingival atrophy, plaques and severe calculus formation. **Educational goal:** To learn how tho approach such patients, how to gain their confidence, and how to make them understand that they share a goal with the dentist, which is reaching a solution to their own problem. This should be done by establishing a positive tone and guiding the patient toward the realization that the majority of their problems stem from smoking, rather than directly telling them so. It should be made clear that the dentist accepts the patient's present decision, but at the same time cessation help should be offered in case the patient changed their mind. Agressive reactions are best left without any response, and in no way should they result in counter-agression. At the end of the appointment the patient should possibly get an appointment card for the next appointment, on which the accessibilities of the nearest cessation center are given.

Table 5.: An example of the scenarios for the video-feedback practice.

Aspect	**Evaluation**		
Nonverbal communication			
Communicates with facial expressions	**0**	**1**	**2**
Verbal communication			
Introduces himself/herself	**0**	**1**	**2**
History taking			
Asks questions regarding the patient's environment	**0**	**1**	**2**
Diagnosis			
Succeeds in making the patient understand the essence of the diagnosis/problem	**0**	**1**	**2**
Providing information			
The patient has got involved in therapeutic decisions to a satisfactory extent	**0**	**1**	**2**
Rapport			
Managed to establish a good rapport	**0**	**1**	**2**

Table 6. Items from teachers' video evaluation sheet. The main aspects/categories are printed in bold. For each category only one item is given here as an example. Evaluation: 0- failed to do so; 1- partially succeeded in doing so; 2- carried it out in a satisfactory manner.

The students who have finished the course are working with real patients now, which means an excellent opportunity for efficacy- and progress assessment. For that reason, evaluation sheets from the first two semesters of the course have been saved, and now patients treated by former course attendees are asked to do the same evaluation. This

way it will be possible to tell if the students can apply the knowledge and skills acquired in the course with real situations as well, and progress assessment at the individual level will be possible as well.

4.2.4. Evaluation of the course- student feedback

As this course has not been part of the curriculum for long, it would be difficult to evaluate it in terms of actual cessation support efficacy. Therefore, we concentrate on student feedback, which is an important measure of how effective the course may be and how well it fits into the curriculum. Obviously, if students find a course useful, interesting and well-constructed, the efficacy of that course in terms of knowledge transfer increases. Furthermore, such a course is likely to generate more interest among future students as well.

Here we present the results of the student evaluation of our latest course. A short questionnaire about the course and its methodology was administered to attendees. Altogether 40 questionnaires were completed. Students were also asked to give a descriptive evaluation. The questionnaire was approved by the Institutional Review Board at the University of Szeged.

Three questions were asked. The first one addressed general satisfaction with the course (Did the course fulfill your expectations?), the second one asked participants to evaluate the theoretical part (Please evaluate the theoretical education during the course.), while the third one sought to assess satisfaction with the videofeedback part (Please evaluate the videofeedback session.). Participants were instructed to rate these aspects on a five-level Likert scale, where 1 meant poor, 2 meant below average, 3 meant average, 4 meant good and 5 meant excellent. Results are presented in Figure 2. To summarize the results, more than 90 ($n>36$) percent of the respondents rated all aspects as excellent or good, which signifies a high level of satisfaction. Only five percent ($n=2$) indicated average general satisfaction and two and a half percent ($n=1$) rated the course in general as poor. Neither the theoretical nor the videofeedback sessions were rated lower than average. Satisfaction with the videofeedback sessions was remarkable: 77.5 percent rated them as excellent.

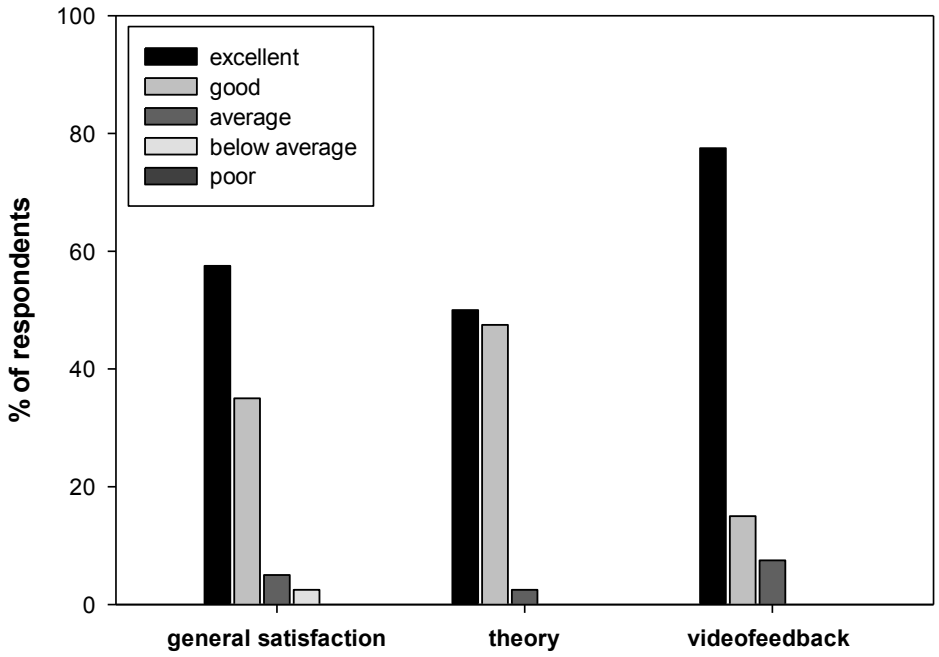

Figure 2. Results of the student satisfaction survey. Results are represented as the percentage of the total number of respondents who rated a given aspect at the same level. It is obvious that the majority of participants rated all aspects as excellent or good, which signifies a high level of satisfaction.

As for the descriptive evaluations, they could be divided into two large groups: those which expressed students' satisfaction with the course and those making technical suggestions. The first group included comments like *"I found it very useful. Actually, students on the general medicine track should take this course as well"* or *"What I liked about this course is that it deals with a completely new topic, which has not been dealt with before in the official curriculum. What is more, we can use what we have learnt not only in our dental practice, but in our private lives as well"* and *"Now I understand why it is difficult for smokers to give up tobacco use!"* Typical comments from the second group are

"Real smokers should be involved in the course, so that we would have a direct chance to ask them how they feel about the way we communicate" or *"A wider variety of scenarios for the video sessions would be useful."*

Finally, it is important to point out that the ratings of the corresponding points on the student evaluation forms filled by course teachers and actors largely overlapped.

4.2.5. A discussion of the findings

Although the course is too recent to be evaluated in terms of actual cessation support efficacy, student feedback shows that it was welcome and possibly filled a long-standing void experienced by students too. That is, student responses revealed that there exists a communicative gap between smokers and non-smokers in connection with smoking ("Real smokers should be involved…"; "Now I understand why..."), which is possibly related to the increasing (also legal) stigmatization of smoking on one hand and the lack of ongoing professional communication on the other. That smoking is something "bad" is increasingly and righteously realized, however, at the same time, smokers possibly perceive stigmatization, which may make them hide their habit or feel guilty about it, so that finally they will rather not talk to their dentist about their tobacco-related problems. This is just complicated by the fact that dentists who themselves smoke will shy away from the topic too, possibly so as not to lose patients' respect. One might say that tobacco-related popular communication operates mostly at the emotional level, inducing an uncertain and maybe uncanny feeling that there is "something wrong" with people who smoke. We believe that a key aspect in professional tobacco prevention is that tobacco use should not be demonized as a personality defect. Prevention and cessation counseling require an atmosphere of unconditional acceptance based on firm scientific knowledge of the multifactorial nature of tobacco dependence and also a firm knowledge of the most efficient methods. It is against such a background that efficient patient education and cessation support can happen. We are positive that our course is a step toward that goal.

The survey and written feedback show that the novel aspect of this course, simulation with videofeedback, turned out to be a great success. Obviously, this method gives a "closest to the real thing" experience, one step beyond training in pairs with peers, but still without the risks of working with real patients. This means a smoother transition from the classroom to reality with the chance of learning about one's strengths and weaknesses, supported by professional feedback. It is very important to emphasize that teacher observations on student performance largely overlapped with actor ratings, which indicated that video recording offers an optimal tool of evaluation.

We have learned a lot from students' direct personal reactions during the course as well. For instance, students who had taken the course admittedly only because they needed credits became interested and actively involved by the end of the sessions. More importantly, we know about students who quit smoking under the influence of the course, which is a very important, if unplanned effect - as the course was not directed at student cessation. A third, and not less important observation was that, as they were learning about smoking in detail, participants gradually gave up their "blaming" attitude towards smokers, and they finally became able to establish a rapport that was optimal for behavioral interventions.

Pieces of student feedback suggesting technical changes are being considered, and they will be used for the further development of the course, whenever possible.

In conclusion, our recently introduced tobacco prevention and cessation counseling course for undergraduate students has been successful in several respects.

First and foremost, it was almost unequivocally popular with students, who welcomed it as a long missed, interesting and useful curricular element. This means that students were actually interested in what was taught, which is obviously a factor that increases the success of knowledge and skills transfer.

Second, the reactions of students corroborated our conviction that in order to increase the efficacy of professional cessation help efforts, "serious talk" about smoking is essential, to take these efforts beyond the popular imagery of fighting against some widely defined evil, and to get rid of the communicative block resulting from the stigmatization of smoking. A course like ours offers facts, evidence-based methods and the chance to put the newly acquired knowledge to the test immediately. This latter element makes the whole process a very intense learning experience.

Third, simulation with video feedback, the key element of our new course, has proven to be a very useful method. By its introduction it became possible for students to gain next-to-real experience with counseling and to have professional feedback on their performance, without risking real-life consequences. It also turned out that the video recordings can be used very well to assess how well a particular student is doing, as video-based teacher ratings largely overlapped with the ratings of actor questionnaires.

Fourth, by using standard questionnaires administered to actual patients, each student can monitor their own development from time to time, and thus the long-term efficacy of the course can be assessed as well.

Finally, we believe that the introduction of this course into the curriculum of the Faculty of Dentistry at the University of Szeged has filled a long-standing void, and we are also positive that the actual cessation support efficacy results will corroborate its usefulness. At the same time we suggest that simulation with video feedback can be a very strong element of any behavioral intervention program, as it offers the chance to put theory into practice immediately, but without the risks of immediate real-life application. Therefore, we believe that this methodology should be an integral part of tobacco intervention teaching methodologies.

5. Thesis summary and recapitulation

Rolling back tobacco-related oral diseases, or at least keeping them at bay is a task of utmost importance in a country which occupies a leading position in terms of both tobacco-related mortality and the number of oral cancer cases. The task is immense, and the possible approaches are countless. In this thesis, two examples from our recent work in the field have been presented, which are tiny steps in the whole process, but we do believe that such tiny steps add up to real solutions.

The fact that one does dentistry at a university faculty puts one at a double advantage: one is that one has access to the infrastructure to do research and gain a deeper understanding of phenomena, and the other is that one can disseminate knowledge and shape future dentists' understanding of those phenomena.

Therefore, the examples chosen for the purposes of this thesis came from these two major segments of university work.

As for the research segment, we showed that smoking puts psoriasis patients at a massively increased risk of severe periodontal disease, the immediate message of which is that such patients should stay away from smoking as much as they can, so that further deterioration of their already poor health and quality of life can be prevented. At the level of scientific interpretation, this result points to the massive immunomodulatory capacity of cigarette smoke, and marks the path for further research into how cigarette smoke can give rise to secondary pathology in chronic inflammatory conditions.

In the second part of the thesis, an entirely new university course was introduced, which was developed at our faculty in order to enable students to provide tobacco cessation support and also to address the problem of high levels of smoking among oral health professionals. This course - now offered in two consecutive semesters- is based on a complex approach, that is, a strong theoretical priming is completed by skills training and practice. A central element of the practical part is videofeedback, which we were the first to introduce into the methodology of cessation support training. The initial results are promising, and although it would be too early to say about anything about the efficacy of the programme in terms of actual quit rates, the change in student attitudes can be felt already.

These results set the directions for the years to come, and the work goes on in the firm belief that even if smoking is a disease of mankind one cannot ultimately cure, one must still care.

6. Acknowledgements

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APPENDIX:

COPIES OF THE PUBLICATIONS PROVIDING THE BASIS OF THE WORK