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# SMOKING AND ORAL DISEASES- PERIODONTITIS

## DISSERTATION WORK IN HYGIENE, PREVENTIVE MEDICINE AND EPIDEMIOLOGY

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## DECLARATION

I hereby declare that this dissertation work was done on my own under the guidance of Assoc. Prof. MUDr. Jindra Šmejkalová Ph.D. at the Department of Hygiene and Preventive Medicine from the year 2004 to 2007.

Hradec Králové, 14th January 2008 .....

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**SUMMARY**

The primary aim of the study was to investigate the influence of cigarette smoking on periodontitis in Czech population. The secondary aim was to compare the results with those from Indian population, where the differences in culture and race, socioeconomic status, oral hygiene measures and practices, and the use of tobacco in different forms like chewing tobacco and smoking bidi in India would have an impact on the final outcome of the study.
The participants of this study were patients of dentists cooperating with the study. The inclusion criterion was age between 30-69 years. Two different sets of questionnaires were prepared; one for the Czech study population (in Czech language) and the other, with minor variations, for the Indian study population (in English). All participants of this study were requested to answer the questionnaire which included questions concerning their personal history, economic status, educational qualification, profession, general health status, food habits, frequency of dental visit, brushing habits, dental aids used and a detailed tobacco consumption history. Information collected on use of tobacco included current tobacco consumption status, duration and amount of tobacco use and form of tobacco use in case of Indian population. Possible forms of tobacco consumption in India that were considered in this study were: 1) tobacco with betel nuts and leaves, 2) tobacco alone, 3) bidi/chutta 4) cigarettes without filters, 5) cigarettes with filters, and 6) pipes and other forms. In case of cigarette or bidi/chutta smokers, they were classified into: 1) regular smokers 2) occasional smokers and 3) ex-smokers. Since the use of smokeless tobacco, mostly in the form of chewing tobacco is prevalent in India, we further classified the Indian study population into 'consumers but non-smokers' and 'non-consumers'. Consumers but non-smokers consisted of subjects who use tobacco in forms other than smoking and non-consumers consisted of subjects who never used tobacco (at the time of study or in the past) in any form. In case of Czech study population, the mode of tobacco consumption most prevalent was smoking in different forms like cigarettes, pipes and cigars.
The examiners in India and Czech Republic used a standard examination environment, standard equipment and followed detailed written instructions. Periodontal status of the respondents was assessed using CPITN index.

The Indian study population consisted of 580 males (72 %) and 225 females (28 %). The majority of respondents were male-consumers of tobacco. Among regular smokers, 98 % were males and among non-consumers 73.2 % were females. The percentage of female

'consumers but non-smokers' (75.2 %) was higher compared to that of males (24.8 %). Except age, all other variables like sex, education, preventive dental visits, brushing frequency and smoking habits significantly influenced the maximum CPITN (%) outcome. Non-consumers had a higher percentage of CPITN score 0 compared to consumers, indicating higher percentage of healthy periodontium in non-consumers. Consumers had a higher percentage of CPITN scores 2, 3 and 4 compared to non-consumers. Non-consumers also had a higher percentage of CPITN score 1 compared to consumers.

The Czech study population consisted of 339 males (49.9 %) and 340 females (50.1 %). Among regular smokers, 60.3 % were males and among non-smokers, 58.9 % were females. Except sex, all other variables like age, education, preventive dental visits, brushing frequency and smoking habits significantly influenced the maximum CPITN (%) outcome. Taking the CPITN scores in percentage of smokers and non-smokers in all sextants, non-smokers had higher percentage of healthy periodontium compared to smokers, smokers had lesser percentage of sites with bleeding on probing and higher percentage of sites with pocketing compared to non-smokers.

Comparison between percentage of scores according to CPITN categories of Czech and Indian population revealed that the Indian study population had a higher percentage of CPITN scores 0, 1 and 2 indicating healthy periodontium, bleeding on probing and supra-gingival or sub-gingival calculus respectively and a lower percentage of CPITN scores 3 and 4 indicating pocket depths up to 4-5 mm and 6 mm or more respectively compared to Czech study population.

## SOUHRN

Hlavním cílem studie bylo posoudit vliv kouření cigaret na onemocnění parodontu u české populace. Ve druhé fázi výzkumu jsme se pokusili porovnat získané výsledky s identickým šetřením provedeným u vzorku indické populace. V úvahu se přitom musely vzít kulturní a rasové rozdíly, různý socioekonomický stav, rozdíly v provádění ústní hygieny, odlišné způsoby užívání tabáku v Indii (např. žvýkání tabáku či kouření bidi), které mohly významně ovlivnit konečné výsledky. Studie se účastnili respondenti ve věku 30-69 let, kteří byli vyšetřeni jednotně instruovanými zubními lékaři. Pro potřeby šetření byly připraveny dvě

verze dotazníků, jedna pro českou populaci (v češtině) a druhá, s drobnými rozdíly, pro indickou populaci (v angličtině). Dotazník obsahoval otázky týkající se osobní anamnézy respondentů, jejich ekonomického postavení, vzdělání, profese, celkového zdravotního stavu, stravovacích návyků, četnosti návštěv u zubního lékaře a způsobu provádění ústní hygieny. Nedílnou součástí dotazníku byla detailní kuřácká anamnéza. Respondenti byli tázáni na to, zda, jak dlouho, v jakém množství a v jaké formě tabák užívají. V této studii byly brány v úvahu následující způsoby užívání: 1) tabákové palice a listy, 2) samotný tabák, 3) bidi/chutta, 4) cigarety bez filtru, 5) cigarety s filtrem, 6) dýmky a ostatní formy. Kuřáci cigaret nebo bidi/chutta byli navíc rozděleni do těchto skupin: 1) pravidelný kuřák, 2) příležitostný kuřák, 3) bývalý kuřák. Vzhledem k tomu, že v Indii je běžnější tabák žvýkat než kouřit, bylo dále u indických respondentů rozlišeno, zda jde o „konzumenta - nekuřáka" nebo „nekonzumenta". Skupinu „konzument - nekuřák" tvořili ti, kteří užívali tabák v jiné formě než kouřením a skupinu nekonzumentů ti, kteří nikdy tabák neužívali v žádné formě (v době výzkumu nebo v minulosti). U české populace přicházelo v úvahu pouze kouření tabáku v různých formách: cigarety, dýmky a doutníky.
Zubní lékaři participující na výzkumu v České republice a v Indii pracovali za standardních podmínek se standardním vybavením a postupovali dle podrobně popsaných instrukcí. Stav parodontu byl hodnocen s pomocí indexu CPITN.

Indický soubor tvořilo 580 mužů (72 %) a 225 žen (28 %). Většina respondentů byli muži - konzumenti tabáku. Muži představovali 98 % pravidelných kuřáků, naopak ženy tvořily 73,2 % nekonzumentů. Podíl žen v kategorii „konzument ale nekuřák" (75,2 %) byl oproti mužům (24,8 %) vyšší. Všechny sledované proměnné s výjimkou věku, tj. pohlaví, vzdělání, četnost preventivních návštěv u zubního lékaře, frekvence čištění zubů a užívání tabákových výrobků měly významný vliv na výslednou hodnotu indexu CPITN. V porovnání s konzumenty byl index CPITN s hodnotou 0 (zdravý parodont) zjištěn u vyššího počtu nekonzumentů. U této skupiny byl rovněž častější výskyt CPITN 1 (krvácení na dotyk). U konzumentů byla naopak zjištěna vyšší frekvence CPITN 2, 3 a 4.

Český soubor tvořilo 339 mužů (49,9 %) a 340 žen (50,1 %). Mezi pravidelnými kuřáky bylo 60,3 % mužů a mezi nekuřáky 58,9 % žen. Všechny sledované proměnné s výjimkou pohlaví, tj. věk, vzdělání, četnost preventivních návštěv zubního lékaře, frekvence čištění zubů a kouření měly významný vliv na zjištěnou hodnotu indexu CPITN. Při porovnání hodnot indexu CPITN u kuřáků a nekuřáků bylo zjištěno, že u nekuřáků byla ve všech sextantech

vyšší prevalence nálezů CPITN 0 (zdravý parodont) a 1 (krvácení na dotyk), zatímco u kuřáků byly častější nálezy CPITN 3, 4 (parodontální choboty).

Srovnávací analýza četností zastoupení hodnot indexu CPITN u české a indické populace prokázala, že v indické skupině se častěji vyskytovaly subjekty s CPITN 0, 1 a 2 (tj. zdravý parodont, krvácení na dotyk a supra-gingiválním či sub-gingiválním calculus), zatímco v českém souboru byly častější nálezy CPITN s hodnotami 3 a 4 (parodontální choboty).

# 1

# INTRODUCTION

Tobacco leaves and the smoke generated when they are burned contain over four thousand chemicals, the best known of which is nicotine, first isolated from tobacco leaves in 1828 by Posselt and Reimann. It is the nicotine that causes smokers to become addicted to tobacco, and the chemical itself is lethal in small doses. When tobacco smoke is inhaled, the nicotine passes quickly to every organ of the body. The brain and nervous system are stimulated by small doses and depressed by larger ones. Nicotine increases the heart rate and blood pressure, and may contribute directly to the excess of thrombosis and atheroma in smokers (31).

Today, tobacco represents the single most preventable cause of death in the world. Of 260 million deaths that occurred in the developing world between 1950 and 2000, it is estimated that 50 million were due to smoking. Globally, smoking related mortality is set to rise from 3 million annually (1995 estimate) to 10 million annually by 2030, with 70 % of these deaths occurring in developing countries (47). Smoking prevalence is lower among women than men in most countries, yet there are about 200 million women in the world who smoke, and in addition, there are millions more who chew tobacco. Approximately 22 % of women in developed countries and 9 % of women in developing countries smoke. Added to this, about 40 % of the world's children are exposed to Environmental Tobacco Smoke (ETS) in the home and a further 61 % in public places (94).

Portuguese introduced tobacco to India 400 years ago. Ever since, Indians have used tobacco in various forms. Two hundred years after the introduction of tobacco to India, the British introduced commercially produced cigarettes to India and established tobacco production in the country. Sixtyfive percentage of all men and 33 % of all women in India use tobacco in some form. In 1997, World Health Organization (WHO) reported the prevalence of tobacco habits in India to be: bidis (34 %), cigarettes (31 %), chewing tobacco (19 %), hookah (9 %), cigars-cheroots (5 %) and snuff (2 %). But the data reported by Cancer Patients' Aid Association of India in 2004 reveals the prevalence to be: bidis (40 %), cigarettes (20 %) and the remaining 40 % is consumed as chewing tobacco, pan masala, snuff, gutkha, mishri and tobacco toothpaste (29). Recent shifts in global tobacco consumption indicate that an estimated 930 million of the world's 1.1 billion smokers live in developing countries, with 182 million in India alone. By the year 2020, it is predicted that tobacco will account for 13 % of all deaths in India (135).

Czech Republic is among the ten countries with the highest rate of cigarette consumption worldwide. According to 2005 estimates by WHO, 31.1 % males and 20.1 % females smoke

in Czech Republic (42). Along with overall rise in cigarette smoking, there has been a significant increase in the reported cases of periodontitis in the country. Association between cigarette smoking and various oral diseases such as leukoplakia and oral cancers has been well documented but the role of cigarette smoking in the causation of periodontitis, however has not been widely investigated in Czech Republic.

People in the developing countries usually have limited access to dental care, more extensive gingivitis and higher levels of plaque and calculus than people in more economically developed societies (9). To better understand the epidemiology of periodontal disease, it is important to have detailed descriptions of the periodontal condition of groups with different genetic backgrounds, levels of dental care and economic development.

# 2

# LITERATURE REVIEW

# 2.1. TOBBACO

## 2.1.1. HISTORY OF TOBACCO

Tobacco and mankind have been associated in the same way as food and tea since before history began (103). *Nicotiana tabacum*, the plant now raised for commercial tobacco production, is probably of South American origin and *Nicotiana rustica*, the other major species, which was carried around the world, came from North America (31). Men came across them about 18,000 years ago when they migrated to the American continents from Asia across the Bering Straight land bridge. Tobacco is thought to have been cultivated since about 5000-3000 BC (103).

In 1762, Columbus found Native Americans growing and using tobacco, sometimes for its pleasurable effects but often for treatment of various illnesses. Some of his sailors observed natives of Cuba and Haiti smoking the leaves and subsequent European explorers and travellers corroborated both these observations. The name tobacco was originally applied to the plant in error. In fact, this term referred to the cane pipe, called a *tabaco* or *tavaco*, with two branches for the nostrils, which was used by the Native Americans for sniffing tobacco smoke. The tobacco itself was variously called; *petum*, *betum*, *cogioba*, *cohobba*, *quauhyetl*, *picietl* or *yietl*, and these names sometimes appeared in herbals of pharmacopoedias (31).

As early as 15 October 1492, Columbus noted that a man in a canoe near the island of Ferdinandina carried dried leaves because they were esteemed for their healthfulness. In the same year, two members of his crew observed people in what is now Cuba carrying a burning torch that contained tobacco, the purpose of which (it later emerged) was to disinfect and help ward off disease and fatigue. Snuffing of *cogioba* through the *tabaco* caused loss of consciousness, Columbus observed, and it is tempting to speculate that this property was used as an anesthetic for the trepanning operations, which were frequent at that time. Tobacco, probably mixed with lime or chalk, appears to have been used in these Native American populations as toothpaste to whiten the teeth, as observed by Nino and Guerra in 1500 and by Vespucci at about the same time in Venezuela. In 1529, a Spanish missionary priest, Bernadino de Sahagun recorded that breathing the odor of fresh green leaves of the plant relieved persistent headaches (31).

There are some uncertainty about which species of *Nicotiana* was first brought to Europe. Probably, it was the Flemish herbalist Rembert Dodoens, in Antwerp, who in 1554 published the earliest figure of *N. rustica*, in his *Cruydeboeck*, seemingly drawn from a specimen plant. In about 1560, according to Nicolas Monardes, the Spanish physician-botanist, the French ambassador to Lisbon, Jean Nicot, was presented with a herb by the keeper of a prison he was visiting. It was described as a strange plant brought from Florida and the ambassador had it planted in his garden. Nicot was so liberal and generous with tobacco that it became known as the ambassador's herb or *nocotiane*-the origin of the name by which we know it now (31). Almost from the onset, the Europeans described smoking as an evil and harmful practice. The first European smoker is reputed to have been imprisoned in a dungeon in Spain for 3 years by the inquisition for smoking in public on his return from America. The British first obtained their tobacco by plundering Spanish ships en route back from America. Sir Francis Drake brought tobacco back from his circumnavigation of the globe in 1580 while some tobacco may have been brought back from the Caribbean in the 1560s (103).

By 1525, tobacco trade had already been established between the Caribbean and India, extending soon afterwards to China, Japan and the Malay Peninsula. About the same time, the Portuguese and Spanish brought tobacco down the east cost of Africa, and by 1560 it was being used in Central Africa also. By the 17$^{th}$ century, tobacco was being produced in Russia, Persia, India and Japan (95).

Even though tobacco was used for medicinal purposes, there were those who questioned its efficacy. Philtaretes, a doctor writing in 1602, raised many criticisms, especially of the indiscriminate use of the herb for all diseases in all age groups without specific measured prescriptions. Vaughan in 1612 warned that tobacco could do much harm when abused. After the isolation of nicotine from tobacco leaves in 1828, the medical world became yet more mistrustful of tobacco as a general treatment, now aware that the plant contained a dangerous alkaloid (31).

## 2.1.2. SCIENTIFIC CLASSIFICATION

There are over sixty species of *Nicotiana* of which *Nicotiana tabacum* and *Nicotiana rustica* are more commonly seen (31).

| | |
|---|---|
| Kingdom: | Plantae |
| Division: | Magnoliophyta |
| Class: | Magnoliopsida |
| Order: | Solanales |
| Family: | Solanaceae |
| Genus: | Nicotiana |

## 2.1.3. FORMS OF TOBACCO INTAKE

- **Cigarettes:** Cigarettes are made from fine-cut tobacco, machine-rolled into a narrow cylindrical shape and wrapped in specially manufactured paper. Those manufactured in the US and India is blended with varying proportions of different grades of flue-cured and air-cured tobaccos and added burning agents. Cigarettes usually measure between 68 and 83 mm in length and approximately 8 mm in diameter, and range in weight from 700 to 1100 mg (including 5 % paper weight) (113).

- **Smoking Pipe:** A smoking pipe is a device used for smoking combustible substances such as tobacco and cannabis. The smoking pipe consists of a small chamber (the bowl) for the combustion of the substance to be smoked and a thin stem (shank) that ends in a mouthpiece.

- **Bidi:** A bidi (from Hindi) is a thin, often flavored Indian cigarette, handmade by rolling a dried rectangular piece of Temburni leaf (*Diospyros melanoxylon*) with 0.15-0.25 g of sun-dried tobacco and secured with a colored thread at one end (120). Temburni leaf accounts for about 60 % weight of a bidi. Bidis measure between 60 and 80 mm in length and range in weight from 400 to 600 mg (including wrapper) (113). They are smaller than regular cigarettes but more potent. Since they do not have filter and are wrapped in nonporous leaves, a

smoker needs to inhale more often and more deeply to keep them lit. One bidi produces three times more carbon monoxide and nicotine, and five times more tar than a regular cigarette (120).

- **Chutta:** A chutta is a type of small hand-made cigar, without a wrapper and a single tobacco leaf as a binder. It consists of air-cured and fermented tobacco folded into a dried tobacco leaf. Chuttas vary greatly in form, length, diameter and weight. Chuttas are usually without a filter and characterized by being open-ended and often have tapered mouthpieces. They are frequently associated with the remarkable habbit of "reverse" smoking, during which the burning end is held inside the mouth (113).

- **Kretek:** Kreteks are Indonesian cigarettes made with a complex blend of tobacco, cloves and a flavoring sauce. They are often called clove cigarettes containing approximately 60 % tobacco and 40 % shredded clove buds (34).

- **Shesha:** Shesha is a smoking device, widely used in the Arabian Peninsula, to smoke Jurak, which is a tobacco-fruit mixture cooked to produce a dark colored paste. Jurak is burnt by electrical device or charcoal. The produced smoke passes through water at the base of the shesha device and passes through a long tube before it is inhaled (7).

- **Argela:** The argela device looks like shesha, but is smaller in size, and the tobacco used with it (measel) differs from jurak in that it is pure tobacco mixed with different fragrances. It is also burned by charcoal and the smoke produced passes through the water base of the device and its tube before it is inhaled. The advantage of shesha and agela over conventional smoking is that nicotine dissolves in water, thus decreasing nicotine inhalation; decrease in tar content; and cooling and humidification (7).

- **Chewing tobacco:** It is a form of smokeless tobacco and is one of the oldest ways of consuming tobacco leaves. Native Americans in both North and South America chewed the leaves of the plant, frequently mixed with lime (31).

- **Snuff:** Snuff is a generic term for fine-ground smokeless tobacco products. In Sweden, snuff is locally known as snus. It is manufactured in a dry form to be used in the nasal cavity and in a moist form to be used in the oral cavity. Snus manufactured for oral use has moist ground tobacco mixed with an aqueous solution of water and other blending ingredients. This form of snuff is found in two types: 1) loose and, 2) portion-bag-packed (68).

- **Toombak:** The snuff used in Sudan is locally known as toombak. It is processed into a loose moist form, and its use is widespread in the country. Tobacco used for manufacturing toombak is of the species *Nicotiana rustica*, and the fermented ground powder is mixed with an aqueous solution of sodium bicarbonate. The resultant product is moist, with a strong aroma and highly addictive (68).

Some other forms of tobacco consumption existing in India includes: *Pan* (piper betel leaf filled with sliced areca nut, lime, catechu and other spices chewed with or without tobacco), *Pan-masala or Gutkha* (a chewable tobacco containing areca nut) and *Mishri* (a powdered tobacco rubbed on the gums as toothpaste) (121).

## 2.2. CIGARETTES

The cigarette machine was invented in the early 20$^{th}$ century and James B. Duke (1865-1925) established the British American Tobacco (BAT) Company (95). Manufactured cigarettes, made by a combination of hand and machine and later by machine alone, were first marketed in England in the 1850s. Their convenience, especially in the trenches in the First World War has resulted in them being the most popular nicotine delivery devices ever since, even more popular since their construction with cork tips and then filters. Unfortunately, the smoke from cigarettes is more acidic than that from pipes and cigars and requires inhalation into the lungs for effective uptake of nicotine, while the nicotine of pipes and cigars can more readily be absorbed through the oral mucosa. Uptake through the lung provides not only a more immediate sense of satisfaction to the smoker as the nicotine in short-circuited to the brain, but also exposes a much greater surface area of respiratory epithelium to the smoke, which

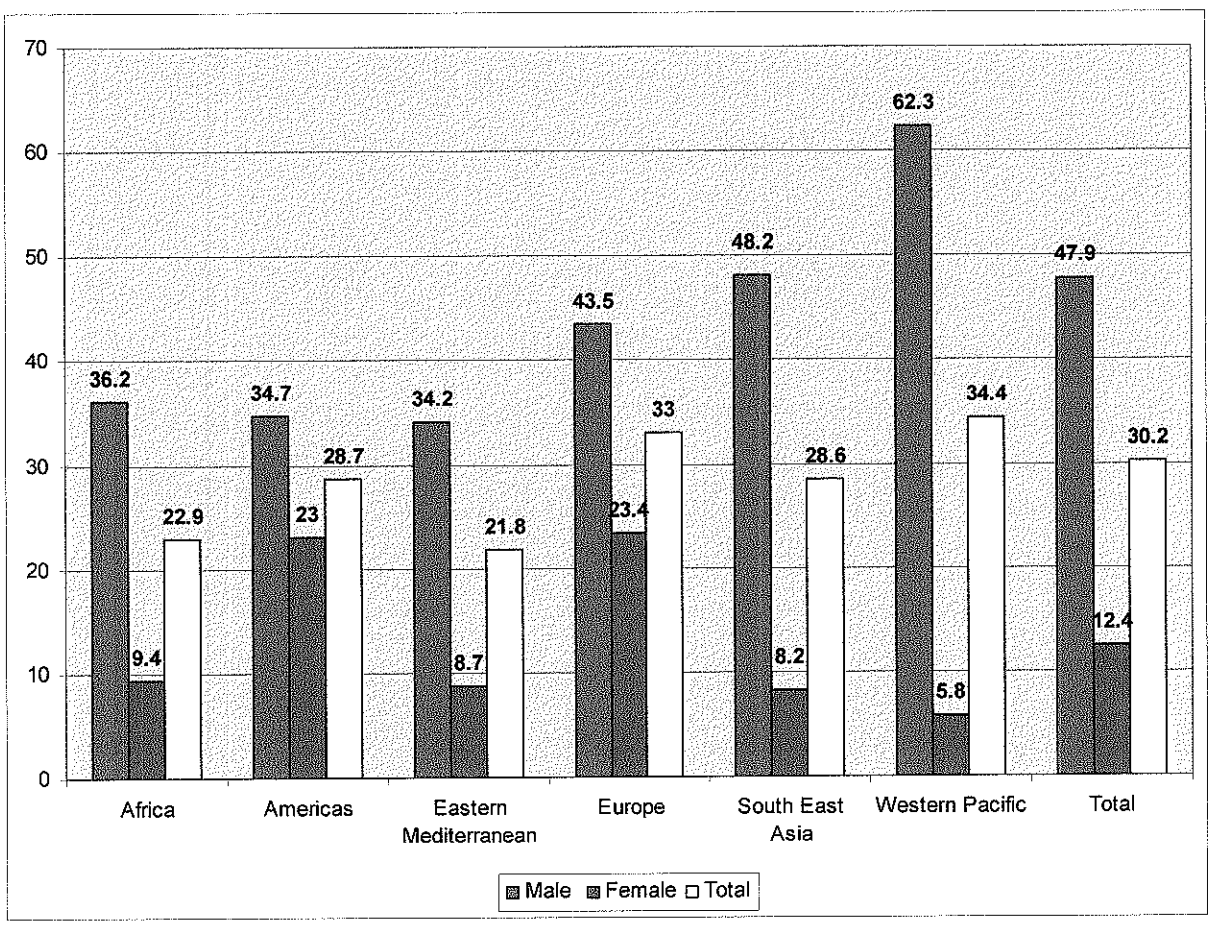
enhances its rapid absorption and potentiates the addictive nature of the product. Cigarette smoking has been taken up worldwide since then and has been described as 'a tragic accident of history' (103).

During the 1980s, as markets began to decline in developed countries, the transnational companies were looking even harder towards developing countries. Glowing accounts of successful tobacco marketing in Asia, and the future potential there, were given by the major companies; Philip Morris, British American Tobacco (BAT) and Rothmans (95).

### 2.2.1. SMOKING PREVALENCE BY GENDER

**Diagram 1**

Smoking prevalence among men and women aged 15 years and above by WHO region, 1998




(Source: http://www.who.int/gender/documents/Gender_Tobacco_2.pdf.)

## 2.2.2. INGREDIENTS OF CIGARETTES

The term "tobacco ingredient" may be defined as a substance (except water) that is added to tobacco during the manufacturing process and having a specific function on the final tobacco product. A "tobacco constituent" may be defined as a substance naturally present in tobacco. In some parts of the world, tobacco companies add ingredients to tobacco, either to increase the subjective characteristics of the smoke or, for example, to increase the moisture-holding capacity of tobacco. Tobacco ingredients are classified as flavors and additives.

Potentially, ingredients can undertake one or more of the following processes during tobacco combustion in the smoking process: 1) distil directly into smoke, 2) pyrolyse or oxidise and their products enter smoke, or 3) pyrolyse or oxidise and the reaction products react with tobacco and smoke constituents and effect their yields or generate other smoke products (11). As most of the ingredients used are highly volatile aroma compounds, they are transferred and changed into mainstream smoke (MS). Typical examples of these ingredients are menthol, anisole, benzyl alcohol, vaniline and certain essential oils. Many of these compounds or mixtures are also natural constituents of the tobacco leaf. Since most ingredients are added to the tobacco in very small amounts (a few parts per million) and because of the low transfer into MS, their contribution to MS composition is consequently also very low. Other typical ingredients are compounds of low volatility such as sugars, and fruit juice extracts, which contain predominantly sugars and aroma compounds. These low volatile ingredients are expected to behave essentially like the sugars naturally contained in the tobacco leaf and to contribute in a similar way to MS composition (126).

**Flavors:** Flavors impart a specific taste, flavor or aroma to a product. They may be used as casing ingredients or flavorings (sometimes referred to as top flavors). Casing ingredients are substances used to enhance the tobacco product's sensory quality by balancing sensory attributes and developing certain required taste and flavor characteristics. Casing ingredients are often recognized foodstuffs and are applied early in the manufacturing process to the pre-cut tobacco. They are applied to the cut and processed tobacco prior to cigarette manufacture, usually in parts per million quantities in a complex mixture in solution.

For example, Licorice extract (block, powder or liquid) may be applied to cigarette tobacco at levels of about 1 % to 4 % to enhance and harmonize the flavor characteristcs of smoke, to

improve moisture holding characteristics of tobacco, to reduce dryness in the mouth and throat, and it acts as a surface active agent for ingredient application (27).

**Additives:** Additives are substances used for a specific technological purpose in the manufacture of tobacco products. Typical tobacco ingredient additives include: Humectants – substances, which increase the moisture-holding capacity of the tobacco. Preservatives- substances that protect the product from deterioration caused by micro-organisms. Solvents- substances used to dissolve or dilute ingredients, without altering their function, in order to facilitate their handling and application. Binders and strengtheners- substances that make it possible to maintain the physical state of the product. Fillers- substances that contribute to the volume of the product without contributing significantly to odor, taste or flavor. In addition, there are some additives that are used as processing aids.

For example, Glycerin is applied to cigarette tobacco at levels in the range of about 1 % to 5 % to improve moisture-holding characteristics of tobacco and act as a surface-active agent for flavor application (26).

Justification for the use of tobacco ingredients cannot be based solely on their approved use in food since, potentially, they could decompose into other substances during tobacco combustion in the smoking process. Tobacco consists of nearly 4000 constituents and during the burning process, these are subjected to temperatures up to 950 °C in the presence of various levels of oxygen. Many types of chemical reaction take place, yielding at least 4800 chemical constituents in smoke. The effects of 450 tobacco ingredients added to tobacco on the forty-four "Hoffmann analytes" in mainstream cigarette smoke have been determined. These analytes are believed by regulatory authorities in the USA and Canada to be relevant to smoking-related diseases. The ingredients comprised of 431 flavors, 1 flavor/solvent, I solvent, 7 preservatives, 5 binders, 2 humectants, 2 process aids and 1 filter (10).

### 2.2.3. CHEMICAL CONTENTS OF CIGARETTE SMOKE AND SMOKELESS TOBACCO

Tobacco smoke is a complex mixture of thousands of compounds, of which approximately 400 have been measured in both mainstream smoke (MS) and sidestream smoke (SS). Environmental Tobacco Smoke (ETS) is composed primarily of SS, with lesser contributions

from the exhaled MS. This complex mixture of particles, gas- and vapor-phase components is rapidly diluted and dispersed after emission and undergoes changes in its physicochemical properties because of shifts in vapor-particle distributions, sorption and desorption of vapor-phase components on indoor surfaces, and chemical reactions. Some of the major components of ETS classified according to their physicochemical characteristics are: 1) very volatile organic compounds like Formaldehyde, Acrolein, Acetylaldehyde, 2) volatile organic compounds like Benzene, Toluene, Styrene, 3) semi-volatile organic compounds like Nicotine, Naphthalene, N-nitrosonornicotine, 4) particulate organic compounds like Benzo[a]pyrene, Solanesol and 5) gas-phase inorganic compounds like $CO_2$, $H_2O$, CO, $NH_3$ (38).

Some of the chemical agents detected in smokeless tobacco which may have a carcinogenic effect includes: 1) Benzo[a]pyrene - Coumarin, Ethyl carbamate, 2) volatile aldehydes - Formaldehyde, Acetaldehyde, Crotonaldehyde, 3) nitrosamines – Nitrosodimethylamine, Nitrosopyrrolidine, 4) nitrosamino acids – Nitrososarcosine, Nitrosoazetadine-2-carboxylic acid, 5) tobacco-specific nitrosamines – N'-nitrosonornicotine, N'-nitrosoanabasine, 6) inorganic compounds – Hydrazine, Arsenic, Nickel, Cadmium, 7) radioactive elements – Polonium-210, Uranium-235, Uranium-238 (64).

When smoked, the tobacco and additives in a cigarette undergo complex chemical processes to form smoke that contains harmful chemicals. So far, more than 4000 different substances have been identified in cigarette smoke. Around half of these substances are found in tobacco itself, the rest are produced as the tobacco burns.

Much literature is available on the harmful constituents of tobacco and tobacco smoke from Western tobacco/smoking products. There are no detailed and systematic data collected on the harmful constituents of Indian tobacco/tobacco smoke from popular Indian smoking products. Plant variety, cultivation, curing methods, and designs of the smoking products (including wrapper and the presence/absence of filters differing in efficiencies) are very different in India. These factors are known to influence the formation and yields/levels of toxic chemicals in tobacco and tobacco smoke in both mainstream (MS) and sidestream (SS) smoke. In a study, the comparison of the levels of dry Total Perticulate Matter (TPM) in both MS and SS from cigarettes, bidis and chuttas showed that the highest level of dry TPM were found in chutta followed by cigarette and bidi. Considering the levels of nicotine in

chutta/bidi/cigarette tobacco and comparing SS/MS ratio of nicotine in chutta/bidi/cigarette, it was evident that relative contribution of dry TPM and nicotine in SS of a bidi and a chutta is much lower than that of a cigarette, suggesting differences in the rate of burning, i.e., cigarette burning more efficiently than bidi and chutta probably because of added burning agent and uniform size and density of the tobacco (113).

A comparative chemical analysis of Indian bidi and American cigarette showed that bidi had a high content of carbon monoxide (7.7 vs. 3.5 %), Ammonia (284 vs. 180 micrograms), Hydrogen cyanide (903 vs. 445 nanograms), Phenol (250 vs. 150 micrograms), other volatile phenols (264 vs. 173 micrograms), Benzo(a)anthracene (9117 vs. 81 nanograms) and Benzo(a)pyrene (78 vs. 47 nanograms). However, tar, nicotine and other constituents were less in bidi smoke than in cigarette smoke (66). Bidi wrapper (tendu leaf) is less porous than cigarette paper and poor in combustibility, resulting in a higher intake of carbon monoxide nicotine and tar (120).

### 2.2.4. IMPACT OF NICOTINE

Tobacco is a complex chemical mixture of several thousand potentially toxic constituents and more than 5000 in its smoke. The nicotine in tobacco is mainly present as the pharmacologically more active [S]-enantiomer that elicits tobacco dependence. Tobacco smoking is further characterized by a transient constriction of the upper airways, a brief increase in blood pressure, respiration rate and heart rate, and various other physiological effects (e.g., relaxation in stress situations). These effects (referred to as "impact" or "kick") are attained via neuronal cholinergic activation and the release of neurotransmitters, like norepinephrine and dopamine. Cigarettes represent very efficient nicotine 'delivery devices' that enable much faster and more complete nicotine absorption, as compared to tobacco gum and nicotine sprays. Inhalation of the tobacco smoke of one cigarette (0.9-1.1 g of whole tobacco containing 6-11 mg of nicotine) rapidly delevers 1 to 3 mg of nicotine to the airways of the smoker. With in a few seconds, about 90 % of the nicotine is absorbed in the upper and lower airways, the rest is swallowed and time-delayed absorption happens via the intestinal route. Within 4 to 6 minutes following the smoking of one cigarette, peak arterial (about 45 ng/mL) and venous (about 25 ng/mL) blood nicotine levels are attained. A typical pack-per-day smoker absorbs about 20 to 40 mg nicotine per day (155). Nicotine has a short half-

life of approximately 30 minutes and is rapidly converted into its primary metabolite, cotinine. For tobacco users, the levels of nicotine reported to be found in saliva ranges from 96 ng to 1.6 mg/ml and in plasma, it ranges from 15 ng to 8 micrograms/ml. Cotinine has a longer half-life than nicotine and have been used to estimate intake of nicotine by its measurement in plasma, urine, or saliva of cigarette smokers. Levels of cotinine appear to remain relatively constant in active smokers over a long time (70). Only 5-10 % of nicotine is cleared renally with a $t_{1/2}$ elimination value of 2-3 hours, depending on urinary pH and flow rate. Only minor quantities of nicotine are excreted via saliva (155).

## 2.3. SYSTEMIC HEALTH EFFECTS OF CIGARETTE SMOKING

### 2.3.1. CANCERS ASSOCIATED WITH SMOKING

Lung cancer, cancers of the oral cavity, esophagus and larynx, cancer of bladder, kidney, pancreas, stomach, uterine cervix and vulvar, breast, penis, colorectal, liver, prostate and leukemia (41, 65, 151).

### 2.3.2. OTHER DISEASES ASSOCIATED WITH SMOKING

- Cardiovascular diseases: coronary heart disease, sudden cardiac death, cerebrovascular stroke, thrombangitis obliterans, atherosclerotic peripheral vascular disease and aortic aneurysm (151).
- Chronic obstructive pulmonary diseases (14).
- Peptic ulcer disease (47).
- Glaucoma and cataracts (33).

### 2.3.3. WOMEN'S HEALTH AND SMOKING

In addition to the health risks that women share with men, women face particular problems linked to tobacco use. These include:

- Female specific cancers such as cancer of cervix.
- Pregnancy related problems: Smoking in pregnancy causes increased risk of spontaneous abortion, ectopic pregnancy, low birth weight, higher perinatal mortality and, long-term effects on growth and development of the child (94).
- Decreased fertility.
- Smoking and contraceptive pills: The risk of heart attack, stroke and other cardiovascular disease in women is increased by approximately tenfold if they both smoke and use oral contraceptives.
- Menstruation and menopause: Smokers experience a greater prevalence of secondary amenorrhea and irregularity of periods and smoking causes women to reach natural menopause one to two years earlier than non-smokers or ex-smokers.
- Osteoporosis (47, 123).

### 2.3.4. HEALTH EFFECTS OF ENVIRONMENTAL TOBACCO SMOKE

ETS exposure has been shown to be an important cause of smoking related diseases especially since the case-control study of Trichopoulos in 1981 of the smoking habits of the spouses of non-smoking female residents of Athens, although the dangers of ETS to children were demonstrated in the 1970s (103). Professor Takeshi Hirayama's cohort study in 1981 on lung cancer in 91 000 non-smoking Japanese wives married to men who smoked was the first conclusive evidence on the harmfulness of passive smoking (94).

In the 1997 review of the literature by the National Health and Medical Research Council of Australia, it was estimated from 34 studies that people who never smoke but live with a smoker have a 30 % increased risk of developing lung cancer compared with people who never smoke and live with non-smoker. The report also reviewed 48 studies of the relationship between passive smoking and asthma and it was estimated that children exposed to ETS are about 40 % more likely to suffer from asthma symptoms than children who are not exposed. On the basis of 25 studies, it was also estimated that children exposed to ETS during the first 18 months of life have a 60 % increase in the risk of developing lower respiratory illnesses such as bronchitis, bronchiolitis and pneumonia. On the basis of 16 studies, it was estimated that the risk of heart attack or death from coronary heart disease was about 24 % higher in people who never smoked but are exposed to ETS (103).

## 2.4. EFFECTS OF SMOKING ON ORAL TISSUES

### 2.4.1. TOBACCO ASSOCIATED ORAL DISEASES AND CONDITIONS

- Oral cancer
- Oral leukoplakia: homogeneous, non-homogeneous leukoplakia and nodular erythroleukoplakia.
- Other tobacco-associated mucosal lesions: snuff dipper's lesion, smoker's palate (leukokeratosis nicotina palate), smoker's melanosis.
- Tobacco associated changes in teeth and supporting structures: tooth loss, periodontal disease, tobacco stains, tooth abrasion.
- Other tobacco-associated oral conditions: gingival bleeding, dental calculus, halitosis, leukoedema candidiasis, median rhomboid glossitis, hairy tongue (12).

## 2.5. PERIODONTITIS

The periodontium is a connective tissue organ, covered by epithelium that attaches the teeth to the bones of the jaws and provides a continually adapting apparatus for support of the teeth during function. The periodontium comprises four connective tissues, two mineralized and two fibrous. The two mineralized connective tissues are cementum and alveolar bone and the two fibrous connective tissues are periodontal ligament and lamina propria of the gingiva. The periodontium is attached to the dentin of the root of the tooth by cementum and to the bone of the jaws by the alveolar bone. The periodontal ligament occupies the periodontal space, which is located between the cementum and the periodontal surface of the alveolar bone (23).

The infection or inflammation of periodontium is known as periodontitis. The pathogenesis of periodontal disease involves a complex interplay between plaque bacteria and a susceptible host. Gingivitis precedes periodontitis, but it is not inevitable that periodontitis will follow gingivitis. In gingivitis, the inflammatory lesion is confined to the gingiva. By contrast, in periodontitis, inflammatory processes extend to affect all of the periodontal support structures (gingiva, periodontal ligament, cementum and alveolar bone), leading to the clinical signs of periodontitis. Breakdown of fibres of periodontal ligament occures, resulting in clinical loss

of attachment of the tooth to its supporting structures and resorption of alveolar bone follows. Pocket formation is evident, there is radiographic bone loss, and teeth may become mobile and may require extraction (119).

**Old Theory of Periodontitis:**

- Periodontal disease is inevitable following gingivitis.
- Periodontal disease is uniformly distributed in the population.
- Disease severity is correlated with plaque levels.
- There is linear progressive loss of attachment over time.
- The severity of periodontitis increases with age.

**New Theory of Periodontitis:**

- Gingivitis and mild periodontitis are common (seen in about 40-60 % of people).
- Approximately 10-15 % of the population exhibit advanced periodontitis.
- Gingivitis precedes periodontitis, but not all sites with gingivitis develop periodontitis.
- Periodontitis is not a natural consequence of aging.
- In some patients, periodontitis may progress with episodes of disease activity and periods of quiescence, in a non-linear manner (119).

### 2.5.1. CLASSIFICATION OF PERIODONTITIS

Classification of periodontitis according to world workshop in clinical periodontics (1989):

- Adult periodontitis.
- Early onset periodontitis.
  - Pre pubertal (generalized or localized)
  - Juvenile (generalized or localized)
- Rapidly progressive periodontitis.
- Necrotizing ulcerative periodontitis
- Periodontitis associated with systemic diseases.
  - Down's syndrome
  - Diabetes type-I
  - Papillon-Lefevre syndrome
  - AIDS

Other diseases

- Refractory periodontitis (28).

### 2.5.2. PATHOGENESIS OF PERIODONTITIS

An awareness of the importance of the host response in periodontal pathogenesis began to develop in the 1970s and 1980s. Peripheral blood neutrophils collected from patients with what was then termed localized juvenile periodontitis (now called localized aggressive periodontitis) were found to have a defective response to chemotactic stimuli, indicating that failure of a host protective mechanism led to increased susceptibility to disease (87). Throughout the 1980s and 1990s, research focused on mediators of the periodontal inflammatory response to the presence of plaque. In particular, these included the prostanoids (e.g. prostaglandin $E_2$, which stimulates alveolar bone resorption) and the cytokines (including the interleukins and tumor necrosis factor) (50). An important family of enzymes, the matrix metalloproteinases (which includes collagenases) was also identified as having a key role in connective tissue breakdown in inflamed periodontal tissues (127).

In the mid-1980s, investigators at the Forsyth Institute introduced a theoretical model to describe the nature of the progression of periodontal disease, collectively referred to as the 'random burst theory'. According to this theory, periodontal tissue support is lost during short, acute episodes of disease activity (bursts), followed by prolonged periods of quiescence. Thus, the loss of attachment recorded by sequential probing assessments is thought to reflect the cumulative effect of such repeated episodes. Since then, a number of publications have re-visited the issue of linear *versus* episodic disease progression. In this context, it must also be realized that, as long as disease progression is measured by linear measurements of vertical attachment loss along the root surface, 'bursts' of activity will be the de facto favored alternative, because the magnitude of the detectable progression is directly dependent on the incremental readings of the periodontal probe (24).

In case of established gingivitis, following plaque accumulation at the gingival margin, there has been infiltration of the connective tissues by numerous defense cells, particularly neutrophils (polymorphonuclear leukocytes or PMNs), plasma cells, monocytes/macrophages and lymphocytes. As a result of accumulation of these defense cells, there has been disruption of the normal anatomy of the connective tissues, with breakdown of collagen fibres to create

space to accommodate the infiltrating defense cells. Blood vessels are dilated; there is vascular proliferation and further collagen loss. The tissues are swollen and the free gingival margin is enlarged and rounded.

In case of periodontitis, histologically, there is proliferation of the junctional epithelium following attachment loss and further destructive events in the connective tissues in response to plaque irritation. The lesion is no longer localized, and the inflammatory cell infiltrate extends apically and laterally into the underlying connective tissues, including the periodontal ligament and the alveolar bone. Alveolar bone loss is evident and there is breakdown of fibres of the periodontal ligament. The accumulation of plaque bacteria in the gingival sulcus results in the release of microbial substances (chemotactic factors such as lipopolysaccharide-LPS, microbial peptides), which cross the junctional epithelium and enter the gingival connective tissues. Epithelial and connective tissue cells are thus stimulated to produce inflammatory response in the tissues. Blood vessels dilate (vasodilatation) and become more permeable to fluid and cells. Fluid accumulates in the tissues and defense cells migrate from the capillaries, up to a chemotactic concentration gradient towards the source of the chemotactic stumulus, bacteria and their products in the gingival sulcus. Thus, there is accumulation of fluids and cells in the tissues and the gingiva becomes erythematous and edematous (119). Finally, Gilthrope et al. in the year 2003 confirmed the cyclical nature of periodontal disease progression using multilevel modeling, and proposed that the 'linear' and 'burst' theories of periodontal disease progression are a manifestation of essentially the same phenomenon, i.e., of the sequential deterioration and repair that occur at the individual tooth sites over time (53).

### 2.5.3. CASE DEFINITION OF PERIODONTITIS IN EPIDEMIOLOGICAL STUDIES

Epidemiology is concerned with the prevalence, severity and distribution of a disease on the population level as well as associations with putative causal or other affecting factors (86, 115). The definition of the specific outcome under investigation is essential in all epidemiological studies. However, the global periodontal literature has been plagued by a number of case definitions of periodontal disease. Studies have used an array of clinical signs and symptoms such as gingivitis, bleeding on probing (BoP), pocket depth (PD), clinical attachment loss (CAL) as well as radiographically assessed alveolar bone loss (18).

Because of inconsistencies in the use of the above disease indicators, large variations in the definition of periodontitis are inevitable. In addition, combinations of disease indicators, such as pocket depth and clinical attachment loss at specific levels have also been used under the rationale that they represent both cumulative tissue destruction (CAL) and current pathology (PD) (6). To further complicate the issue, there is wide variation in the threshold values used in the definition of a 'case', regardless of the indicators used, as well as in the definitions of incident or progressive disease. Finally, studies have also used tooth loss as an additional outcome variable in the context of risk assessment (32).

Although several studies are focused on the role of the same risk factors, a direct comparison of odds ratio or relative risk between studies is hard. Another issue that needs to be accounted for in terms of case definitions is the use of full- or partial-mouth recording parameters such as CAL and/or PD. National, large-scale epidemiologic studies have usually used partial-mouth recording methodologies, such as the system used in National Health and Nutrition Examination Survey (NHANES) III study, which examined only two sites in two randomly selected quadrants under the assumption that these measurements are representative of the full-mouth status (2). In contrast, smaller scale studies are more likely to have used full-mouth examination methodologies (46). Several studies have documented that the use of partial-mouth examinations usually leads to an underestimation of both the prevalence and the severity of the disease (79), which, in combination with the lack of a uniform case definition of periodontitis, has an inevitable effect on inferences, related to risk and prognostic factors (15). For the above-mentioned reasons, it appears that a consensus decision on the adoption of uniform criteria for both prevalent and incident periodontitis is essential in order to advance analytical epidemiological research of periodontitis in the future.

Another major problem in periodontal epidemiology is that periodontal disease cannot be assessed as an active process, but only as a present status by historic evidence by means of surrogate parameters, with rather poor correlation with a tangible effect for the subjects (such as tooth retention or no discomfort) (54).

## 2.5.4. POSSIBLE RISK FACTORS OF PERIODONTITIS OTHER THAN CIGARETTE SMOKING

A risk factor can be defined as characteristics of the person or environment that, when present, directly result in an increased likelihood of a person getting a disease and when absent, directly result in a decreased likelihood (17). It is important to make the distinction that risk factors are associated with a disease but do not necessarily cause the disease. Risk factors may be modifiable or non-modifiable. Modifiable risk factors are usually environmental or behavioral in nature whereas non-modifiable risk factors are usually intrinsic to the individual and therefore not easily changed. Non-modifiable risk factors are also known as determinants (147).

The manifestation and progression of periodontitis, a multifactorial disease with microbial dental plaque as the initiator, is influenced by a variety of determinants and factors. They include subject characteristics, social and behavioral factors, systemic factors, genetic factors, tooth-level factors, microbial composition of dental plaque and other emerging risk factors (108).

**Modifiable risk factors:**

**Microbiota:** In a classic paper in 1994, Haffajee and Socransky (58) adapted Koch's postulates to be used in the identification of periodontal pathogens and proposed the following criteria: (i) association, i.e., elevated odds ratios in disease; (ii) elimination, i.e., conversion of disease to health when bacteria are suppressed; (iii) development of a host response; (iv) presence of virulence factors; (v) evidence from animal studies corroborating the observations in humans; and (vi) support from risk assessment studies. Based on the above criteria, the Consensus Report of the 1996 World Workshop in Periodontics identified three species namely; *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis* and *Bacteroides forsythus* recently renamed *Tanerella forsythensis* as causative factors for periodontitis (24).

Microbial dental plaque has long been recognized as the initiator of periodontal disease. The Specific Plaque Hypothesis suggested that specific bacterial species are causative for periodontal disease. Putative periodontal pathogens include Gram-negative species such as *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis, Bacteroides forsythus* and *Eikenella corrodens* (89). Over the last decade, interesting data have emerged on the

prevalence of these causative bacteria in different populations, in states of both periodontal health and disease. Studies performed in children (140, 159) that analyzed plaque from the gingival crevice, tooth surface, and the dorsum of the tongue revealed that sizeable proportions of subjects harbored *Actinobacillus actinomycetemcomitans, Porphyromonas gingivalis* and *Tanerella forsythensis* despite absence of overt gingival inflammation. A comparably high carrier state was documented in studies that sampled infants, children, adolescents and adults with good clinical periodontal status (75, 82, 96).

Thus, contrary to the conclusions of earlier culture-based studies that these bacteria occur infrequently in periodontally healthy oral cavities and behave as exogenous pathogens, the above studies that have used molecular techniques for bacterial identification demonstrate the contrary. However, both the prevalence of and the level of colonization by these pathogens have been shown to vary significantly between populations of different racial or geographic origin (57, 90, 129).

An alternative hypothesis, the Non-Specific Plaque Hypothesis, stated that disease results from the physical mass of subgingival organisms present, and occurs once a certain threshold has been reached. In other words, it is the quantity of plaque that is important rather than the quality (143). More recently, cluster analysis of subgingival plaque has demonstrated that certain species frequently occur together in 'complexes' (131). These complexes have been colour-coded purple, yellow, green, orange and red, representing a progression from health (characterized by a predominantly gram-positive, aerobic, non-motile microflora) to disease (characterized by a gram-negative, anaerobic, motile microflora). The red complex of species (*P.gingivalis, B.forsythus* and *Treponema denticola*) is strongly associated with the clinical signs of periodontitis. The orange complex (including *Pervotella intermedia, Fusobacterium nucleatum, Peptostreptococcus micros* and *Campylobacter rectus*) is also associated with periodontitis, but less strongly than the red complex (131). Plaque in patients with periodontitis tends to contain an increased proportion of red and orange complex species compared to plaque from periodontally healthy patients (158). In diseased patients, there is also a decrease in the proportion of *Actinomyces* species, which are found in large numbers in periodontal heath (158).

A critical development in the understanding of periodontal microbiology was the concept that plaque exists in biofilms (35), which can be defined as matrix enclosed bacterial populations

adherent to each other and/or surfaces or interfaces (110). Biofilms are complex bacterial communities that form in aqueous environments where there is a regular nutrient source. Within the biofilm, there is primitive homeostastis, a primitive circulatory system (for waste elimination and nutrition supply) and a degree of metabolic co-operation. The biofilm has evolved to protect individual bacteria, so that the bacteria are highly resistant to killing by phagocytosis and antimicrobial drugs.

Realization of the importance of plaque biofilms, together with knowledge gained from the cluster analyses of the periodontal microflora, has led to the most current concept of the role of bacteria in periodontitis, the Environmental Plaque Hypothesis (60). This hypothesis suggests that the entire subgingival microbial environment is the important determinant of the role of bacteria in the development of disease. Approximately 300-500 bacterial species have been identified as being able to colonize a periodontal pocket but, of these, only 20-30 are considered to be pathogenic. Therefore, for periodontitis to develop, not only is a susceptible host required (an essential prerequisite), but also pathogenic species must develop in sufficiently high quantities within the subgingival plaque biofilm (119). Data generated in the past decade have enhanced the knowledge on a number of specific microbial risk factors for periodontitis, but have also clarified the significance of the concept of bacterial load rather than that of mere positive colonization (24).

**Tooth factors**: Various aspects of tooth anatomy, variations in tooth positions such as malalignment, crowding and migration or tipping of a tooth distal to an edentulous area have all been shown to be associated with clinical manifestations of periodontal disease. Occlusal discrepancies and pulpal involvement also contribute to the periodontal destruction (108).

**Socioeconomic status**: Previous studies have documented differences in periodontal health by socioeconomic indicators, i.e., income and education but these indicators have rarely been investigated as independent variables of main interest. These studies can be summarized as those reporting (i) higher rates of disease for subjects with low socioeconomic status in cross-tabulations between outcomes and socioeconomic indicators or (ii) that racial/ethnic differences persisted after adjustment for socioeconomic indicators in multivariate analyses. Regardless, socioeconomic indicators are robust markers of periodontitis. Their role in periodontal disease can be attributed to differential access to resources and opportunities that may influence preventive behaviors. Evidence also suggests that education has a greater

influence than income in favorably affecting the level of periodontitis in the population (24). Studies have also stated that psychosocial measures of stress associated with financial strain are significant risk indicators for periodontal disease in adults (52).

**Diabetes Mellitus**: Studies performed over the last decade have expanded the available evidence on the role of diabetes mellitus as a major risk factor for periodontitis, especially in subjects with poor metabolic control and a long duration of the disease. Studies suggest a two-way relationship between diabetes and periodontitis, with more pronounced periodontal tissue destruction in people with diabetes and also a poorer metabolic control of diabetes in subjects with periodontitis. Evidence from cross-sectional and longitudinal studies suggests that, irrespective of the case definition used for periodontitis, subjects with diabetes have higher prevalence, extent and severity of periodontal disease. These observations are consistent for both Type-1 and Type-2 diabetes (24).

**Obesity:** The biological plausibility of a potential link between obesity and periodontitis has been suggested to involve the hyper-inflammatory state and the aberrant lipid metabolism prevalent in obesity, as well as the pathway of insulin resistance, which may collectively result in an enhanced breakdown of the periodontal tissue support (24). A number of recent studies point to a positive association between obesity, defined as body mass index (BMI) $\geq$ 30, and periodontitis (4, 128, 157).

**Osteoporosis:** A number of cross-sectional studies of limited sample size and largely confined to postmenopausal women have suggested that women with low bone mineral density are more likely to have clinical attachment loss, gingival recession and/or pronounced gingival inflammation (100, 142). However, studies that failed to report such an association have been published as well (91, 153). Based on these studies, it has been hypothesized that the systemic loss of bone density in osteoporosis may, in combination with hormone action, heredity, and other host factors, provide a host system that is increasingly susceptible to the infectious destruction of periodontal tissue (150).

**HIV infection:** Although several publications of the last decade reported increased prevalence and severity of periodontitis in HIV-positive subjects when compared to controls (97, 107, 125), other studies are either not supportive of this notion or indicate that the

differences in periodontal status between HIV-seropositive and HIV-seronegative subjects are much more limited than earlier believed (36, 83, 149).

**Psychosocial factors:** The mechanisms by which psychological stress may affect periodontal health are complex. It has been suggested that one of the plausible pathways may involve behavioral changes leading to smoking and poor oral hygiene that, in turn, may affect periodontal health (51).

**Non-modifiable factors**

**Age:** The relationship between age and periodontitis is not straightforward. Early evidence demonstrates that both the prevalence and severity of periodontitis increase with increasing age, suggesting that age may be a marker for periodontal tissue support loss. However, the belief that periodontitis is a disease of the elderly has been challenged over the years. Instead of indicating an increased susceptibility to periodontitis in older people, this 'age effect' can conceivably represent the cumulative effect of prolonged exposure to true risk factors. Moreover, it is established that periodontitis may have its onset in youth and early adulthood, rather than in older years. Therefore, a subject's susceptibility level to periodontal disease appears to be more important than age, and subjects with high susceptibility manifest the disease at an earlier age. Notably, the effect of age appears to be different for probing depth and clinical attachment loss. Specifically, while there is a pronounced effect of increasing clinical attachment loss with age, the effect on probing depth appears to be minimal. Interestingly, the effect of age on clinical attachment loss has been found to be reduced after adjusting for covariates such as oral hygiene levels or access to dental care services. However, studies have often failed to adjust for important covariates such as systemic diseases (e.g., diabetes) and health-risk behaviors (e.g., smoking) in the older population. Therefore, the literature on the effect of age on periodontitis needs to be interpreted with caution (24).

**Gender:** Although there is no established, inherent difference between men and women in their susceptibility to periodontitis, men have shown to exhibit worse periodontal health than women. This difference has been documented in different populations and has been traditionally thought to be a reflection of better oral hygiene practices and/or more utilization of dental health care services among women. On the other hand, periodontitis is a bacterial infection determined to a large extent by the host immuno-inflammatory response to the

bacterial challenge. Although gender-specific differences in these responses have not been unequivocally demonstrated, it is biologically plausible that such differences do, in fact, exist (24).

**Race/ethnicity:** Although differences in the prevalence of periodontitis between countries and across continents have been demonstrated, no consistent differences across racial/ethnic groups have been documented when age and oral hygiene are accounted for. However, race/ethnicity is usually a social construct that determines an array of opportunities in the society such as access, status and resources. As a result, race/ethnicity and socioeconomic status are strongly intertwined, suggesting that the pervasive racial/ethnic effect is the result of residual confounding by socioeconomic status because of the unequal meaning of socioeconomic status indicators across racial/ethnic groups (24).

**Genetic factors:** Specific genotypes have been identified and linked to periodontal destruction. Polymorphisms of IL-1, IL-1 Beta and IL-1RN genotypes have been identified as potential risk factors for periodontal destruction. In a study that evaluated these polymorphisms and smoking, it was found that being positive for the composite IL-Alpha/ IL-Beta polymorphism in smokers resulted in four times the risk of significant attachment loss compared to genotype-negative smokers. Polymorphism of the tumor necrosis factor (TNF-Alpha) gene has been suggested as a possible risk factor for periodontitis (108). Evidence from classical twin studies suggests that genetic determinants are significant modifiers of the periodontitis phenotype (98, 130) but the role of single-nucleotide polymorphisms remain unclear. After Korman's seminal work (80) reporting an association of a composite genotype based on specific polymorphisms in the interleukin-1 (IL-1) gene cluster with severe periodontitis in non-smokers, there has been an exponential increase in publications that examined a plethora of gene polymorphisms as severity markers of periodontitis. Typically, the majority of cross-sectional studies reported positive associations between the investigated polymorphisms and the extent or the severity of periodontitis. The results, however, are not unequivocal, as the strength of the reported associations is not uniformly consistent across populations, the frequency of occurrence of these polymorphisms appear to vary extensively between ethnic groups, the subject samples involved are generally of limited size, the definitions of the outcome variable (periodontitis) vary considerably, and adjustments for other important covariates and risk factors have frequently not been performed (24).

## 2.6. EFFECTS OF SMOKING ON PERIODONTAL HEALTH

Cigarette smoking is a significant risk factor for periodontal disease (139), demonstrated by an increased loss of attachment (5, 67, 123), development and progression of periodontal inflammation (51, 70) and increased gingival recession (104). It has been estimated that smoking accounts for half of all periodontal diseases. There is epidemiological evidence, which shows that cigarette smoking is a stronger risk factor for the presence of periodontitis compared to the presence of certain suspected periodontal pathogens (40). The number of cigarettes smoked per day is a major risk determining factor, doubling the risk for those in the lowest consumption category and increasing it six fold in the subgroup smoking more than thirty cigarettes per day (144, 156). Former smokers have lower rates of periodontitis than present smokers (3, 25, 72, 81, 134, 148). Longitudinal studies indicate that periodontal disease may progress faster in smokers in comparison to non-smokers (16).

### 2.6.1. EFFECTS ON IMMUNE RESPONSE

Smoking affects various aspects of the host immune response and the mechanisms by which smoking enhances periodontal degradation are said to be the cumulative effect of elevation of putative periodontal pathogens and modulation of the host inflammatory and immune response (40, 144, 148). Smoking does not alter the composition of bacterial plaque but it has been observed that the host's response to bacterial plaque is disturbed (92). Chronic periodontitis is said to be influenced by an interaction of host immune mechanism and environmental factors (48). Experimental studies on plaque-induced gingivitis in humans suggest that clinical signs of gingival inflammation, namely, redness, bleeding and exudation are not as prominent in smokers as in non-smokers (88, 146). Even though the primary etiology of periodontal disease is bacteria, the host response determines a patient's susceptibility to disease. There is enough evidence, which indicates that smoking affects the innate and immune host responses (13, 72, 78). It has been observed that the hemorrhagic responsiveness of periodontium is lowered in smokers compared to non-smokers (20). The findings of decreased inflammation and reduced gingival crevicular fluid volumes in smokers compared to non-smokers suggest that smoking impairs gingival blood flow. Although smokers actually have significantly higher numbers of neutrophils, the first line of defense against bacterial infection, in peripheral circulation, their function is impaired. Neutrophils

have shown decreased chemotaxis, phagocytosis, and adherence in smokers. Integrin expression and protease inhibitor production is also affected (72). On the other hand, smoke exposure of unstimulated neutrophils has been shown to elevate the oxidative burst, which could enhance tissue destruction through direct toxic effect.

Antibody production is another protective host mechanism that is altered by smoking. Smoking decreases serum IgG concentrations generally and decreases $IgG_2$ antiboby production in patients with early onset periodontitis (59, 101). Smokers have demonstrated reduced titres of serum IgG to periodontal pathogens like Pervotella Intermedia and Fusobacterium Nucleatum and the level of $IgG_2$ against Actinobacillus Actinomycetemcomitans is lower in smokers compared to non-smokers. The proliferative response of T-cells to antigens is decreased by long term exposure of cigarette smoke (104, 148).

### 2.6.2. ALVEOLAR BONE LOSS

A higher amount of alveolar bone destruction has been seen in smokers (21, 77, 123) and the severity of destruction was also found to be more in smokers compared to non-smokers (21, 22, 77). A dose-response effect on alveolar bone has been seen, accelerating the bone loss with higher amount and longer duration of tobacco consumption (7). The bone mineral content among smokers were found to be 10-30 % lower compared to non-smokers in a longitudinal cohort study and it was speculated that constituents of tobacco smoke may alter the metabolism of vitamin D or influence hormonal states (48). The periodontal bone height and frequency of diseased sites (probing depth > 4 mm) remained stable among non-smokers and smokers who quit before the baseline examination in a long term prospective study, but became worse among subjects who continued to smoke (145). Women who smoke one pack of cigarettes per day throughout their adult lives will have an average deficit in bone density of 5-8 %, by the time they reach menopause. Estrogen metabolism is altered in female smokers, and the deficiency of estrogen is associated with elevations of IL-1, IL-6 and Tumor Necrosis Factor-alpha (TNF), which affect both alveolar and systemic bone status (73).

### 2.6.3. PERIODONTAL PATHOGENS

Powerful reducing agents such as carbon monoxide contained in tobacco smoke produce a substantial immediate reduction of redox potential at mucosal surfaces. The powerful physico-chemical reducing activity of carbon monoxide is probably a direct mechanism to promote growth of anaerobes at superficial sites than simple anaerobiosis (45). Several studies suggest that the types of bacteria in smokers and non-smokers did not vary significantly but smoking may alter the quality of the flora. A lower oxygen tension in the periodontal pocket of smokers may be favorable for the growth of anaerobic bacteria (73). The oxidation-reduction potentials in dental plaque have been shown to be decreased by smoking, possibly encouraging growth of anaerobic bacteria (59). Plaque formation appears not to be influenced by smoking in several experimental gingival settings, however, there seems to be an altered gingival response to supragingival plaque in smokers. In a steady state situation, in some cross-sectional studies, smokers seem to have more plaque and calculus than non-smokers. It has been conjectured that distinctive personal characteristics of smokers may be responsible for a general trend for neglecting health issues (104).

### 2.6.4. EFFECTS ON WOUND HEALING AND RESPONSE TO PERIODONTAL THERAPY

Smokers have a poorer response to periodontal therapy compared to non-smokers (70, 92, 118). Various studies suggest that smoking adversely effects healing after various forms of periodontal therapy. In one study, researchers found that ex-smokers were similar to non-smokers in their response to therapy, suggesting that, quitting smoking may help in healing (56, 72, 156). Smokers were found to have lesser reduction in periodontal depth and lesser clinical attachment gain after treatment compared to ex-smokers or non-smokers (74). In a six-year longitudinal study, non-smokers had approximately 50 % higher rate of improvement in probing depth and clinical attachment levels after periodontal therapy than smokers (73).

Chemical products and toxins in tobacco smoke may delay wound healing by impairing the biologic progression of healing and by inhibiting the basic cellular functions responsible for its initiation. Smoking has a strong negative influence on regenerative therapy, which includes osseous grafting, guided tissue regeneration or a combination of these treatments. The revascularization of bone and soft tissues is impaired by smoking which could have a strong

influence on wound healing, particularly related to regenerative, periodontal and implant therapies. Volatile components of cigarette namely Acrolein and Acetaldehyde may inhibit gingival fibroblast attachment and proliferation. Fibroblasts, which are exposed to nicotine, produce less fibronectin and collagen and more collagenase and these negative effects on fibroblast functions could influence wound healing and progression of periodontitis (73).

### 2.6.5. POSSIBLE MECHANISMS FOR THE NEGATIVE PERIODONTAL EFFECTS OF SMOKING:

- Vascular alterations
- Altered neutrophil function
- Decreased IgG production
- Decreased lymphocyte proliferation
- Increased prevalence of periopathogens.
- Altered fibroblast attachment and function
- Difficulty in eliminating pathogens by mechanical therapy.
- Negative local effects on cytokine and growth factor production (72).

# 3

# COMMUNITY PERIODONTAL INDEX OF TREATMENT NEEDS (CPITN)

The Community Periodontal Index of Treatment Needs (CPITN) was developed for the 'Joint Working Committee' of the World Health Organization (WHO) and Federation Dentaire Internationale (FDI) by Jukka Ainamo, David Barmes, George Beagrie, Terry Cutress, Jean Martin, and Jennifer Sandro-Infirri in 1982 (117). The index was evaluated by a group of experts and a final version was released in 1983. In 1987, the CPITN was incorporated into the WHO manual 'Oral Health Survey, Basic Methods'. Since then, the CPITN index has been widely used to measure the level of periodontal diseases and treatment needs in populations (19). CPITN has been used frequently in periodontal epidemiology during the last decades. Although originally intended as a screening procedure for epidemiological purposes, the CPITN has been adapted for other purposes; in a promotional role in developing periodontal health awareness programmes, for initial screening and for monitoring changes in periodontal needs of individuals in clinical practice (117).

**Scope and Purpose:** The CPITN procedure is recommended for epidemiological surveys of periodontal health and it uses clinical parameters and criteria relevant to planning and prevention of periodontal diseases and it records the common treatable conditions, namely periodontal pockets, gingival inflammation and dental calculus. The CPITN is not intended as a comprehensive assessment of total past and present periodontal disease experience and it does not record irreversible changes such as gingival recession or other deviations from periodontal health such as tooth mobility or loss of periodontal attachment (117). The CPITN does not include the measures of intensity of inflammation, precise identification of pocket depths or differentiation between supra-gingival and sub-gingival calculus (37). The advantages of CPITN Index are: 1) its use renders knowledge about the high prevalence and low severity of periodontal disease among populations, 2) simplicity in recording and, 3) world-wide application allowing for international comparisons (122).

**Procedure for CPITN:** The dentition is divided into six parts (sextants) for assessment of periodontal conditions. The sextants begin from the maxillary right sextant, proceeding in a clockwise manner and finishing in the mandibular right sextant. At least six points on each tooth (mesiobuccal, midbuccal, distobuccal, distooral, midoral and mesiooral) are examined by gently "walking the probe" around the tooth and for each sextant, only the highest score based on the highest probing value obtained on any tooth in that sextant, is recorded (37).

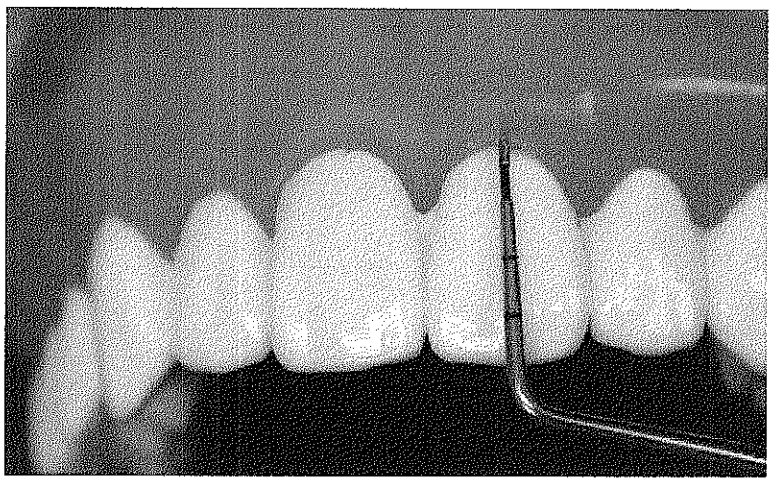
**CPITN Probe:** The recommended periodontal probe for use with CPITN was first described by WHO. This porbe was designed for two purposes, namely, measurement of pocket depth and detection of sub-gingival calculus. The probe is both thin in the handle and is of very light-weight (5 gms) (117). The probe has a 0.5 mm diameter ball tip, which enhances detection of subgingival calculus or over hanging restorative margins and limits false readings from over-measurement of probing depths. It also has a color-coded band extending 3.5 mm to 5.5 mm from the tip, which facilitates rapid interpretation of probing depths and markings at 8.5 mm, 11.5 mm and 15.2 mm. The CPITN probe is gently inserted into the gingival pocket and the depth of penetration read against the color-coded band (37). A tooth is probed to determine pocket depth and to detect sub-gingival calculus and bleeding response. The probing force can be divided into a 'working component'- to determine pocket depth and a 'sensing component'- to detect sub-gingival calculus. The working force should not be more than 25 gms.

**Codes and Criteria:**

| Code | The description of condition |
|---|---|
| Code 0 | Healthy periodontal tissues |
| Code 1 | Bleeding on gentle probing |
| Code 2 | Supragingival and/or subgingival calculus |
| Code 3 | Shallow pockets up to 4-5 mm |
| Code 4 | Deep pockets 6 mm or more |
| Code X | Sextant excluded (when less than two teeth present) |

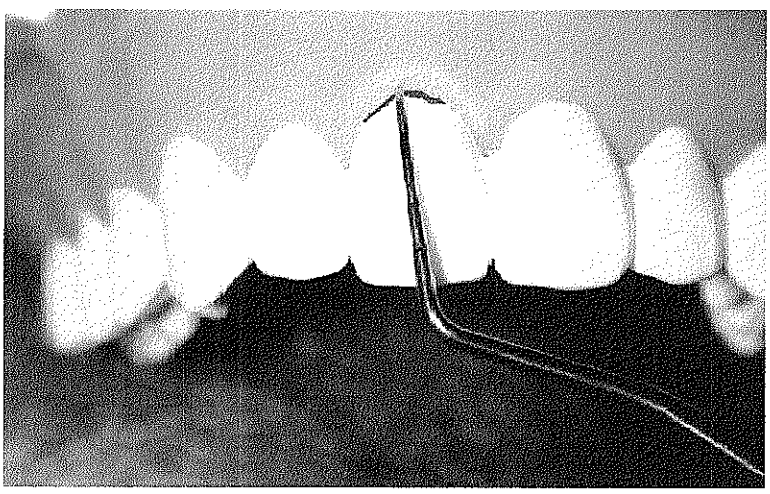
*A description of CPITN codes and the corresponding criteria is explained in the following page with pictures.*

**CODE 0**



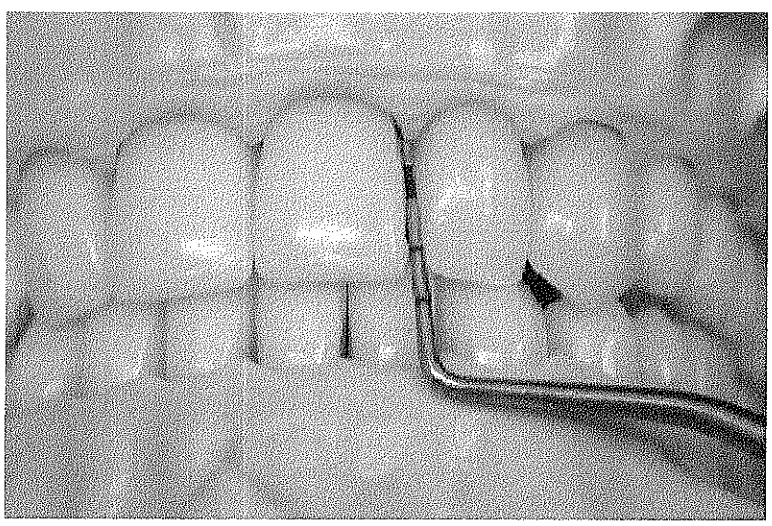
The color code is visible, no bleeding, calculus or pocket.

**CODE 1**



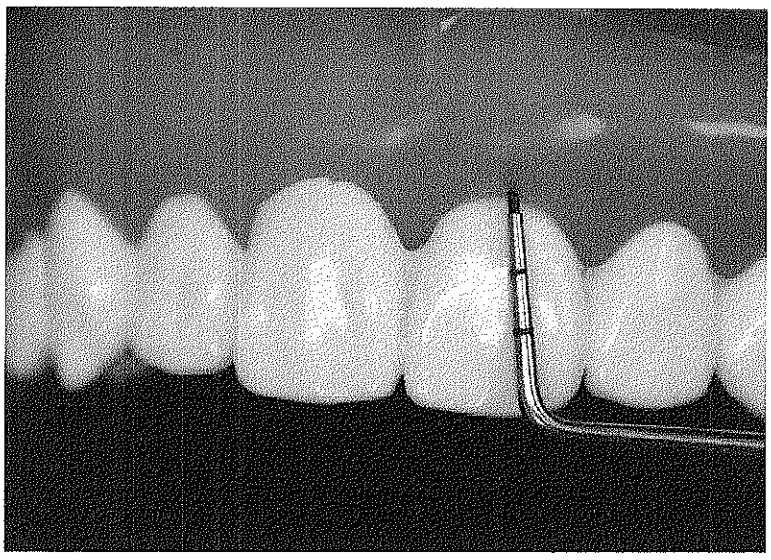
Bleeding observed during or after probing.

**CODE 2**



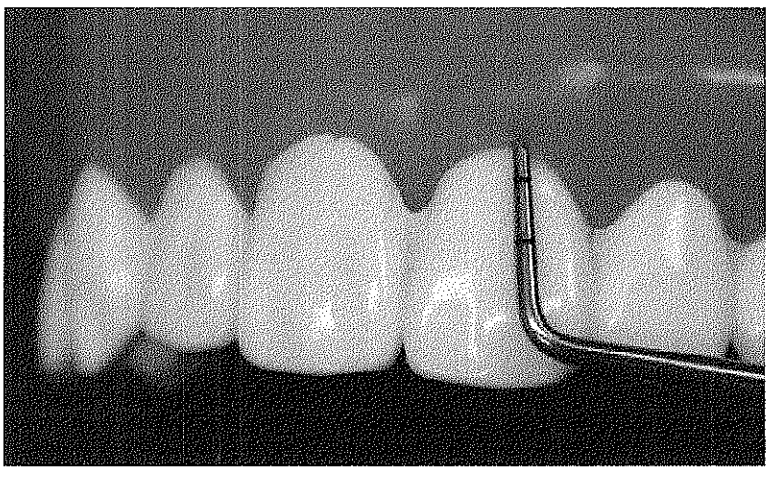
Supra-gingival or sub-gingival calculus detected on probing.

**CODE 3**



Pathological pocket of 4 mm to 5 mm present. The color code of the probe is only partially visible.

**CODE 4**



Pathological pocket of 6 mm or more present. The color code of the probe is not visible.

# 4

# AIM OF THE STUDY

The primary aim of the study was to investigate the influence of cigarette smoking on periodontitis in Czech population. The secondary aim was to compare the results with those obtained from Indian population, taking into consideration that the differences in culture and race, socioeconomic status, oral hygiene measures and practices, and the use of tobacco in different forms like chewing tobacco and smoking bidi in India would have an impact on the final outcome of the study.

# 5

# MATERIALS AND METHODS

Epidemiological studies on periodontal status have used a variety of indicators and indices. The most commonly used indices are the Periodontal Index from Russel, the Periodontal Disease Index of Ramfjord and also, the Community Periodontal Index of Treatment Needs (CPITN). CPITN Index was used for this study. The study was approved by the Ethics Committees of SRM University and Charles University in Prague and was conducted at SRM Dental College and Hospital, Chennai, India with the cooperation of three dentists and at the Department of Dentistry, Teaching Hospital, Hradec Králové and also by three private dental practitioners in Hradec Králové, Czech Republic simultaneously from July 2005 to February 2007.

## 5.1. STUDY POPULATION

Several studies used a representative sample (55, 62), whereas most studies used a convenience sample such as volunteers or patients attending dental clinics (49, 112) for collecting data. The participants of this study were patients of dentists cooperating with the study. The inclusion criterion was age between 30-69 years. Completely edentulous patients were excluded from the study. The participants were informed about the purpose of the study, an informed consent was taken and at the end of clinical examination, each participant was given instructions regarding dental treatment needs.

## 5.2. QUESTIONNAIRES

Two different sets of questionnaires were prepared; one for the Czech study population (in Czech language) and the other, with minor variations, for the Indian study population (in English). All participants of this study were requested to answer the questionnaire which included questions concerning their personal history, economic status, educational qualification, profession, general health status, food habits, frequency of dental visit, brushing habits, dental aids used and a detailed tobacco consumption history. Information collected on use of tobacco included current tobacco consumption status, duration and amount of tobacco use and form of tobacco use in case of Indian population. Possible forms of tobacco

consumption in India that were considered in this study were: 1) tobacco with betel nuts and leaves, 2) tobacco alone, 3) bidi/chutta, 4) cigarettes without filters, 5) cigarettes with filters, and 6) pipes and other forms. In case of cigarette or bidi/chutta smokers, they were classified into: 1) regular smokers 2) occasional smokers and 3) ex-smokers. Regular smokers were defined as individuals who, at the time of examination, smoked at least one cigarette daily. Occasional smokers were individuals who smoked less than one cigarette per day. Former or ex-smokers were defined as individuals who smoked at least 1 cigarette per day for 6 consecutive months and don't smoke at least for the past 6 months from the time of the study. Since the use of smokeless tobacco, mostly in the form of chewing tobacco is prevalent in India, we further classified the Indian study population into 'consumers but non-smokers' and 'non-consumers'and the following definitions were considered appropriate in the context of the present study: 'Consumers but non-smokers' consisted of subjects who use tobacco in forms other than smoking and 'non-consumers' consisted of subjects who never used tobacco (at the time of study or in the past) in any form. In case of Czech study population, the most prevalent mode of tobacco consumption was smoking in different forms like cigarettes, pipes and cigars.

(*A copy of the Czech and Indian questionnaires is added in the appendix.*)

## 5.3. CLINICAL EXAMINATION

The examiners in India and Czech Republic used a standard examination environment, standard equipment and followed detailed written instructions. The following clinical evaluations were performed: 1) oral mucosal findings; 2) presence or absence of gingivitis; 3) recording CPITN index.

Mouth mirror and WHO-621 Trinity probe manufactured by Medin (Medin, a.s. Vlachovická 619, 592 31 Nové Město na Moravě, Česka Republika) set to give a constant probing force of 20-25 g as recommended, were used for the study in India and Czech Republic. At least six points on each tooth (mesiobuccal, midbuccal, distobuccal, distooral, midoral and mesiooral) were examined by gently "walking the probe" around the tooth and for each sextant, only the highest score based on the highest probing value obtained on any tooth in that sextant, was recorded.

## 5.4. DATA ANALYSIS

The statistical analysis of the data included the classification of data and calculation of frequencies and was performed by NCSS 2004 program. Mann-Whitney test or Kolmogorov-Smirnov test was used for comparing two groups (consumers and non-consumers or smokers and non-smokers) of quantitative data (e.g., age) and Kruskal-Wallis analysis of variance with multiple comparison tests was used for the five groups (regular smokers, occasional smokers, ex-smokers, 'consumers but non-smokers' and non-consumers) according to tobacco use. Chi-square test of independence in contingency tables or Fisher's exact test was used for qualitative data (e.g., CPITN, education, preventive dental visits, brushing habits, smoking habits) and level of significance was $\alpha = 0.05$.

# 6

# RESULTS

# 6.1. INDIAN RESULTS

The total study population was classified into consumers who used tobacco in different forms like smoking, chewing, snuff etc and non-consumers who never used tobacco (at the time of study or in the past) in any form. For detailed description, consumers were further classified into regular smokers, occasional smokers, ex-smokers and 'consumers but non-smokers'. The population under study consisted of 574 males (72 %) and 225 females (28 %). A detailed classification of the subjects according to tobacco use and gender is given in table 1. Majority of respondents were male-consumers of tobacco and taking both the genders into consideration, the mean age of consumers was 47.3 and that of non-consumers was 40.3.

**Table 1:** Classification according to tobacco use and gender ($p < 0.001$; $\chi^2$ test)

| | Males | Females | Total | Age (mean) | Age (SD) |
|---|---|---|---|---|---|
| Consumers | 536 | 121 | 657 | 47.30 | 11.26 |
| Non-consumers | 38 | 104 | 142 | 40.32 | 10.44 |

Table 2 demonstrates the detailed tobacco consumption history of the subjects. Among regular smokers, 98 % were males and among non-consumers 73.2 % were females. The percentage of female 'consumers but non-smokers' (75.2 %) was higher compared to that of males (24.8 %).

**Table 2:** Detailed tobacco consumption history of respondents ($p < 0.001$; $\chi^2$ test)

| | Males (n) | Males (%) | Females (n) | Females (%) | Total (n) | Total (%) |
|---|---|---|---|---|---|---|
| Regular smokers | 433 | 98.0 | 9 | 2.0 | 442 | 100.0 |
| Occasional smokers | 33 | 94.3 | 2 | 5.7 | 35 | 100.0 |
| Ex-smokers | 35 | 89.7 | 4 | 10.3 | 39 | 100.0 |
| Consumers but non-smokers | 35 | 24.8 | 106 | 75.2 | 141 | 100.0 |
| Non-consumers | 38 | 26.8 | 104 | 73.2 | 142 | 100.0 |

Taking educational qualification of respondents into consideration, except for the group of consumers but non-smokers, more than 50 % of respondents of all other groups (smokers and

non-consumers) had university graduation and the least percentage of graduates (37.6 %) were found to be among the group of 'consumers but non-smokers'. A detailed description is given in table 3.

**Table 3:** Educational qualification of respondents (%) ($p < 0.05$; $\chi^2$ test)

| | No education | Basic education | High school education | University graduation |
|---|---|---|---|---|
| Regular smokers | 4.8 | 14.3 | 30.3 | 50.7 |
| Occasional smokers | 11.4 | 11.4 | 17.1 | 60.0 |
| Ex-smokers | 5.1 | 15.4 | 23.1 | 56.4 |
| Consumers but non-smokers | 10.6 | 16.3 | 35.5 | 37.6 |
| Non-consumers | 8.5 | 9.2 | 22.5 | 59.9 |

Comparing educational qualification and form of tobacco consumption, among subjects without any education, having only basic education or having high school education, a higher percentage of them used tobacco with betel nut and leaves and smoked bidi or chutta. But in case of graduates, 58.4 % were abstinent from using tobacco with betel nuts and leaves and 56.1 % were abstinent from using bidi or chutta compared to 30.1 % and 7.8 % using these forms of tobacco consumption respectively ($p<0.001$).

Considering the income of respondents, among those who disclosed their income, occasional smokers and ex-smokers had higher income (above 10,000 Indian Rupees) compared to other groups and a higher percentage of regular smokers (36.5 %) had lower income compared to other groups of smokers and non-consumers. A detailed description is given in table 4.

**Table 4:** Percentage of respondents in given income group (in Indian Rupees)

($p < 0.001$; $\chi^2$ test)

| Income/month | 500-1500 | 1500-3500 | 3500-6500 | 6500-10,000 | Above 10,000 | Not willing to disclose |
|---|---|---|---|---|---|---|
| Regular smokers | 1.8 | 36.5 | 23.4 | 12.9 | 13.8 | 11.6 |
| Occasional smokers | 5.7 | 28.6 | 20.0 | 14.3 | 25.7 | 5.7 |
| Ex-smokers | 2.6 | 12.8 | 12.8 | 15.4 | 25.6 | 30.8 |
| Consumers but non-smokers | 7.8 | 27.7 | 26.2 | 14.2 | 5.7 | 18.4 |
| Non-consumers | 2.1 | 22.5 | 24.6 | 14.8 | 7.7 | 28.2 |

Table 5 demonstrates the distribution of subjects in percentage, according to tobacco consumption in relation to participation in preventive dental check-ups. Irrespective of tobacco consumption, 64.6 % of respondents visited the dentist only when they had some dental problems. 20.4 % of non-consumers visited dentists twice a year and was highest among the groups.

**Table 5:** Percentage of respondents participating in preventive dental check-ups

($p=0.0019$; $\chi^2$ test)

| | Twice a year | Once a year | When having problem(s) | Never before |
|---|---|---|---|---|
| Regular smokers | 15.2 | 15.8 | 65.8 | 3.2 |
| Occasional smokers | 2.9 | 25.7 | 60.0 | 11.4 |
| Ex-smokers | 12.8 | 23.1 | 59.0 | 5.1 |
| Consumers but non-smokers | 7.1 | 23.4 | 62.4 | 7.1 |
| Non-consumers | 20.4 | 12.0 | 65.5 | 2.1 |
| Total | 14.0 | 17.3 | 64.6 | 4.1 |

Taking the tooth brushing frequency of respondents into consideration, irrespective of tobacco consumption history, majority of respondents (> 70 %) brushed their teeth once daily. Further details are given in table 6.

**Table 6:** Tooth brushing frequency of respondents (%) ($p < 0.001$; $\chi^2$ test )

| | Once daily | 2 times/day | 3 times/day |
|---|---|---|---|
| Regular smokers | 87.8 | 10.9 | 1.4 |
| Occasional smokers | 71.4 | 22.9 | 5.7 |
| Ex-smokers | 79.5 | 20.5 | 0.0 |
| Consumers but non-smokers | 75.9 | 22.7 | 1.4 |
| Non-consumers | 75.4 | 20.4 | 4.2 |

Table 7 presents the distribution of respondents in percentage, according to tobacco consumption in relation to oral mucosal findings and gingivitis. 22.7 % of respondents in the group of 'consumers but non-smokers' that mainly consisted of chewing tobacco users (tobacco with betel nuts and leaves) and 12.9 % of regular smokers had some oral mucosal changes or lesions.

**Table 7:** Percentage of respondents with oral mucosal findings and gingivitis

| | Oral mucosal findings ($p<0.001$); $\chi^2$ test | Gingivitis ($p<0.001$); $\chi^2$ test |
|---|---|---|
| Regular smokers | 12.9 | 23.1 |
| Occasional smokers | 8.6 | 51.4 |
| Ex-smokers | 5.1 | 25.6 |
| Consumers but non-smokers | 22.7 | 29.1 |
| Non-consumers | 2.8 | 46.5 |

Nearly twentyfour percentage of respondents using tobacco with betel nuts and leaves, 21.1 % smoking bidi/chutta ($p<0.001$) and 11 % smoking cigarettes with/without filters had some oral mucosal changes or lesions but it was present only in 2.8 % of non-consumers. 28.8 % of

respondents using tobacco with betel nuts and leaves and 23.8 % smoking cigarettes with/without filters had gingivitis but it was present in 46.5 % of non-consumers ($p<0.001$).

The maximum CPITN score was calculated in percentage, by taking the maximum or worst findings from six sextants (max CPITN).

The max CPITN (%) in consumers and non-consumers are shown in table 8. Non-consumers had a higher percentage of CPITN score 0 compared to consumers, indicating higher percentage of healthy periodontium in non-consumers. Consumers had a higher percentage of CPITN scores 2, 3 and 4 compared to non-consumers. Non-consumers also had a higher percentage of CPITN score 1 compared to consumers.

**Table 8:** CPITN scores (%) in consumers and non-consumers ($p<0.001$; $\chi^2$ test)

| | CPITN 0 | CPITN 1 | CPITN 2 | CPITN 3 | CPITN 4 |
|---|---|---|---|---|---|
| Consumers | 6.2 | 39.3 | 38.4 | 12.6 | 3.5 |
| Non-consumers | 19.0 | 47.2 | 23.9 | 7.0 | 2.8 |

The percentage of CPITN score 0, indicating healthy periodontium, in subjects using tobacco with betel nuts and leaves, smoking bidi/chutta and smoking cigarettes with/without filters were lower, demonstrated by a score of 5.9 %, 1.5 % and 5.6 % respectively compared to that of non-consumers demonstrated by CPITN score of 19 %. The percentage of CPITN score 1, indicating bleeding on probing, was lower in subjects smoking cigarettes with/without filters and those using tobacco with betel nuts and leaves (35.7 % and 39.7 % respectively) and slightly higher in respondents smoking bidi/chutta (48.9 %) compared to non-consumers (47.2 %). Considering the pattern of smoking, percentages of CPITN score 0 and score 1 in regular smokers (smoking bidi/chutta and/or cigarette) were 3.8 % and 35.3 % respectively compared 19 % and 47.2 % respectively in non-consumers ($p<0.001$). Considering each sextant separately, the percentage of CPITN score 0 was higher in non-consumers in all sextants compared to consumers as demonstrated in table 9.

**Table 9:** CPITN score (%) of consumers and non-consumers in each sextant:

| | CPITN 0 | CPITN 1 | CPITN 2 | CPITN 3 | CPITN 4 |
|---|---|---|---|---|---|
| $1^{st}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 20.4 | 50.4 | 23.6 | 5.1 | 0.5 |
| Non-consumers | 45.8 | 30.3 | 20.4 | 2.8 | 0.7 |
| $2^{nd}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 29.4 | 54.0 | 13.3 | 2.8 | 0.5 |
| Non-consumers | 58.5 | 30.3 | 8.5 | 2.8 | 0.0 |
| $3^{rd}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 21.3 | 48.3 | 24.9 | 4.4 | 1.1 |
| Non-consumers | 47.5 | 30.2 | 16.5 | 4.3 | 1.4 |
| $4^{th}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 18.7 | 42.0 | 29.2 | 8.2 | 1.9 |
| Non-consumers | 49.3 | 32.4 | 11.3 | 6.3 | 0.7 |
| $5^{th}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 25.3 | 49.8 | 18.7 | 5.4 | 0.8 |
| Non-consumers | 54.3 | 33.6 | 9.3 | 2.9 | 0.0 |
| $6^{th}$ sextant ($p<0.001$; $\chi^2$ test) | | | | | |
| Consumers | 19.7 | 41.9 | 28.4 | 8.2 | 1.7 |
| Non-consumers | 50.4 | 28.1 | 14.4 | 5.8 | 1.4 |

Analysing the influence of sex on maximum CPITN score (%), it was evident that females had a higher CPITN score of 0 and 1 and males had a higher CPITN score of 2. Details are given in table 10.

**Table 10:** Influence of sex on max CPITN (%) ($p < 0.001$; $\chi^2$ test)

| Max CPI | Females | Males | Total |
|---|---|---|---|
| 0 | 16.0 | 5.6 | 8.5 |
| 1 | 47.1 | 38.2 | 40.7 |
| 2 | 20.0 | 42.0 | 35.8 |
| 3 | 11.1 | 11.8 | 11.6 |
| 4 | 5.8 | 2.4 | 3.4 |
| Total | 100.0 | 100.0 | 100.0 |

Analysing the influence of age on maximum CPITN (%), it was evident that there was no significant difference in percentage of CPITN scores 0, 1, 2, 3 and 4 among the compared age groups i.e., younger age group (30-49 years) and older age group (50-69 years). Details are shown in table 11.

**Table 11:** Influence of age on max CPITN (%) ($p = 0.71309$, $\chi^2$ test)

| Max CPI | Younger age group (30-49 years of age) | Older age group (50-69 years of age) | Total |
|---|---|---|---|
| 0 | 9.6 | 6.7 | 8.5 |
| 1 | 40.5 | 40.9 | 40.7 |
| 2 | 35.1 | 36.9 | 35.8 |
| 3 | 11.6 | 11.7 | 11.6 |
| 4 | 3.2 | 3.7 | 3.4 |
| Total | 100.0 | 100.0 | 100.0 |

Table 12 gives a detailed description of the influence of education on maximum CPITN (%). It was evident that university graduates followed by high school educated respondents had a higher percentage of max CPITN 0 (10.4 % and 8.2 % respectively) and respondents with no education followed by respondents with basic education had a higher percentage of max CPITN 4 (9.3 % and 5.5 % respectively).

**Table 12:** Influence of education on max CPITN (%) ($p = 0.000016$; $\chi^2$ test)

| Max CPI | No education | Basic education | High school education | University graduation | Total |
|---|---|---|---|---|---|
| 0 | 5.6 | 3.7 | 8.2 | 10.4 | 8.5 |
| 1 | 24.1 | 32.1 | 37.7 | 46.9 | 40.7 |
| 2 | 35.2 | 42.2 | 38.5 | 32.6 | 35.8 |
| 3 | 25.9 | 16.5 | 11.7 | 8.4 | 11.6 |
| 4 | 9.3 | 5.5 | 3.9 | 1.7 | 3.4 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 13 describes the influence of preventive dental visits on max CPITN (%). It was obvious that respondents attending preventive dental check-ups twice a year had a higher percentage of healthy periodontium. Respondents visiting dentist only when having some dental problems or those who never visited a dentist before had a higher percentage of shallow pockets.

**Table 13:** Influence of preventive dental visits on max CPITN (%) ($p = 0.000371$; $\chi^2$ test)

| Max CPI | Twice a year | Once a year | When having problem | Never before | Total |
|---|---|---|---|---|---|
| 0 | 18.8 | 6.5 | 7.0 | 6.1 | 8.5 |
| 1 | 42.9 | 47.8 | 38.6 | 36.4 | 40.7 |
| 2 | 29.5 | 34.8 | 38.0 | 27.3 | 35.8 |
| 3 | 7.1 | 9.4 | 12.8 | 18.2 | 11.6 |
| 4 | 1.8 | 1.4 | 3.7 | 12.1 | 3.4 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 14 describes the influence of tooth brushing frequency on max CPITN (%). It was obvious that respondents brushing their teeth 3 times a day had a higher percentage of maximum CPITN 0 compared to respondents brushing less frequently but it was also evident that the same group of respondents had a higher percentage of maximum CPITN 4 compared to other groups.

**Table 14:** Influence of tooth brushing frequency on max CPITN (%) ($p = 0.000218$; $\chi^2$ test)

| Max CPI | Once daily | Twice daily | 3 times/day | Total |
|---|---|---|---|---|
| 0 | 6.5 | 16.8 | 25.0 | 8.5 |
| 1 | 40.6 | 40.8 | 43.8 | 40.7 |
| 2 | 38.1 | 26.4 | 12.5 | 35.8 |
| 3 | 11.4 | 13.6 | 6.3 | 11.6 |
| 4 | 3.3 | 2.4 | 12.5 | 3.4 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 |

Analysing the influence of tobacco consumption on max CPITN (%), it was evident that non-consumers had a higher percentage of max CPITN 0 compared to other groups, regular smokers had a higher percentage of max CPITN 1 and 2 and regular smokers followed by 'consumers but non-smokers' had a higher percentage of max CPITN 3. A detailed description is given in table 15.

**Table 15:** Influence of tobacco consuming on max CPITN (%) ($p < 0.001$; $\chi^2$ test)

| Max CPI | Regular smokers | Occasional smokers | Ex-smokers | Consumers but non-smokers | Non-consumers | Total |
|---|---|---|---|---|---|---|
| 0 | 3.8 | 14.3 | 12.8 | 9.9 | 19.0 | 8.5 |
| 1 | 35.3 | 48.6 | 61.5 | 43.3 | 47.2 | 40.7 |
| 2 | 44.8 | 25.7 | 23.1 | 25.5 | 23.9 | 35.8 |
| 3 | 13.3 | 11.4 | 2.6 | 13.5 | 7.0 | 11.6 |
| 4 | 2.7 | 0.0 | 0.0 | 7.8 | 2.8 | 3.4 |
| total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 16 gives a detailed description of the various independent variables (sex, age, education, preventive dental visits, brushing habits and smoking habits) and the corresponding max CPITN scores that influenced the respective variable to be significant. It was evident that gender, education, preventive dental visits, brushing frequency and smoking habits had a significant influence on the periodontal status ($p<0.001$) where as age of the respondents had no significant influence on the periodontal health ($p=0.717$).

**Table 16:** Influence of chosen variables on maximum CPITN

($\chi^2$ test of independence in contingency tables)

| Variable | p-value | Chi-Square Contribution Section (main resultes) |
|---|---|---|
| Sex | 0*** | max CPI-0<br>- more frequently found in women<br>max CPI-2<br>- more frequently found in men<br>max CPI- 4<br>- more frequently expressed in women |
| Age | 0.713 | - no significant difference between younger (30-49 years of age) and older (50-69 years of age) group of respondents |
| Education | 0.000016*** | max CPI-1<br>- more frequently present in university graduated respondents<br>max CPI-3 and 4<br>- more frequently found in group of respondents with no education<br>- less frequently expressed in university graduated respondents |
| Preventive dental visits | 0,000371 *** | max CPI-0<br>- most frequently found in respondents participating in preventive dental visits 2 times a year<br>max CPI-4<br>- most frequently expressed in respondents not participating in dental preventive visits |
| Brushing frequency | 0,000218 *** | max CPI-0<br>- most frequently seen in respondents brushing their teeth 3 times daily, following by those brushing their teeth 2 times daily<br>max CPI-4<br>- most frequently found in respondents brushing their teeth 3 times daily |
| Smoking | 0*** | max CPI-0<br>- most frequently found in the group of non-consumers<br>- less frequently expressed in the group of regular smokers<br>max CPI-2<br>- more frequently found in regular smokers<br>max CPI-4<br>- most frequently expressed in the group of 'consumers but non-smokers' |

## 6.2. CZECH RESULTS

The Czech study group consisted of 339 males (49.9 %) and 340 females (50.1 %). A detailed classification of the subjects according to smoking history and gender is given in table 17 and the age characteristics are given in table 18. Among regular smokers, 60.3 % were males and among non-smokers, 58.9 % were females.

**Table 17:** Classification according to tobacco use and gender (number and percentage)

| | Regular smokers | | Occasional smokers | | Ex-smokers | | Non-smokers | | Total | |
|---|---|---|---|---|---|---|---|---|---|---|
| | n | % | n | % | n | % | n | % | n | % |
| Males | 91 | 60.3 | 26 | 63.4 | 61 | 64.2 | 161 | 41.1 | 339 | 49.9 |
| Females | 60 | 39.7 | 15 | 36.6 | 34 | 35.8 | 231 | 58.9 | 340 | 50.1 |
| Total | 151 | 100.0 | 41 | 100.0 | 95 | 100.0 | 392 | 100.0 | 679 | 100.0 |

**Table 18:** Age characteristics of respondents

| | Mean ± SD | Median | 25th percentile | 75th percentile |
|---|---|---|---|---|
| Regular smokers | 43.8 ± 10.6 | 43 | 34 | 52 |
| Occasional smokers | 39.1 ± 9.1 | 36 | 31.5 | 45 |
| Ex-smokers | 46.9 ± 10.6 | 46 | 38 | 56 |
| Non-smokers | 44.3 ± 11.3 | 42 | 34 | 53 |

Taking the educational qualification of respondents into consideration, 41.3 % of regular smokers had lower education (Basic school) where as 50.5 % of ex-smokers and 42.7 % of non-smokers had high school education. A detailed description is given in table 19. Irrespective of smoking habits, 43.6 % of respondents had high school education.

**Table 19:** Educational qualification of respondents in percentage ($p < 0.001$; $\chi^2$ test)

| | Regular smokers | Occasional smokers | Ex-smokers | Non-smokers | Total |
|---|---|---|---|---|---|
| Basic school | 41.3 | 17.1 | 23.2 | 22.0 | 26.1 |
| Skilled | 8.7 | 12.2 | 7.4 | 7.2 | 7.8 |
| High school | 40.0 | 48.8 | 50.5 | 42.7 | 43.6 |
| University graduation | 10.0 | 22.0 | 18.9 | 28.1 | 22.5 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Considering the income of respondents, among those who disclosed their income, irrespective of smoking habits, 47.1 % of respondents had high income (> 10,000 CZK). A detailed description is given in table 20.

**Table 20:** Percentage of respondents in given income group (in Czech Crowns)
($p = 0.035$; $\chi^2$ test)

| Income/month | Regular smokers | Occasional smokers | Ex-smokers | Non-smokers | Total |
|---|---|---|---|---|---|
| Not willing to disclose | 12.7 | 7.3 | 8.8 | 12.1 | 11.5 |
| < 5000 CZK | 6.7 | 9.8 | 3.3 | 2.6 | 4.0 |
| 5000-10,000 CZK | 29.3 | 24.4 | 31.9 | 35.9 | 33.2 |
| > 10,000 CZK | 43.3 | 53.7 | 54.9 | 46.0 | 47.1 |
| Do not know | 8.0 | 4.9 | 1.1 | 3.4 | 4.2 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 21 demonstrates the distribution of subjects in percentage, according to smoking habits in relation to participation in preventive dental check-ups. Irrespective of smoking habits, 66.2 % of respondents visited dentists for preventive dental check-ups twice a year. Further details are shown in the table.

**Table 21:** Percentage of respondents participating in preventive dental check-ups (p=0.0138; $\chi^2$ test)

| | Regular smokers | Occasional smokers | Ex-smokers | Non-smokers | Total |
|---|---|---|---|---|---|
| 2 times a year | 57.3 | 61.0 | 58.9 | 71.9 | 66.2 |
| Once a year | 22.7 | 22.0 | 25.3 | 18.6 | 20.6 |
| Once in 2 years | 2.7 | 2.4 | 5.3 | 3.6 | 3.5 |
| Less frequently | 8.7 | 9.8 | 5.3 | 3.3 | 5.2 |
| Never | 8.7 | 4.9 | 5.3 | 2.6 | 4.4 |

Table 22 shows the tooth brushing frequency of respondents. Irrespective of smoking habits, 75.5 % of respondents brushed their teeth twice daily. A higher percentage of regular smokers brushed their teeth less frequently compared to other groups and a higher percentage of non-smokers brushed their teeth 3 times a day or more, compared to other groups. Further details are given in the table.

**Table 22:** Tooth brushing frequency of respondents (%) (p = 0.0504; $\chi^2$ test)

| | Regular smokers | Occasional smokers | Ex-smokers | Non-smokers | Total |
|---|---|---|---|---|---|
| 3 times/day or more | 9.3 | 2.4 | 5.3 | 13.0 | 10.5 |
| 2 times/day | 70.9 | 82.9 | 83.2 | 74.7 | 75.5 |
| Once daily | 15.2 | 12.2 | 9.5 | 11.0 | 11.8 |
| Less frequently | 4.6 | 2.4 | 2.1 | 1.3 | 2.2 |

The analysis of data concerning the influence of cigarette smoking on oral mucosa of the respondents revealed that oral mucosal lesions were relatively rare among smokers and non-smokers, indicating that smoking had no significant influence on the oral mucosal health. Table 23 gives a detailed description.

**Table 23:** Percentage of oral mucosal lesions in smokers and non-smokers (p=0.011, Fisher's Exact Test)

| Oral mucosal lesions | Smokers | Non-smokers | Total |
|---|---|---|---|
| Absent | 96.9 | 99.5 | 98.4 |
| Present | 3.1 | 0.5 | 1.6 |
| Total | 100.0 | 100.0 | 100.0 |

Taking the CPITN scores in percentage of smokers and non-smokers in each sextant into consideration, in all sextants, non-smokers had higher percentage of healthy periodontium compared to smokers, smokers had lesser percentage of sites with bleeding on probing and higher percentage of sites with pocketing compared to non-smokers. A detailed description is given in table 24.

**Table 24:** CPITN score (%) of smokers and non-smokers in each sextant:

| | CPITN 0 | CPITN 1 | CPITN 2 | CPITN 3 | CPITN 4 |
|---|---|---|---|---|---|
| $1^{st}$ sextant (p=0.076; $\chi^2$ test) | | | | | |
| Smokers | 5.6 | 27.2 | 13.9 | 34.5 | 7.3 |
| Non-smokers | 9.1 | 33.7 | 13.8 | 29.5 | 6.8 |
| $2^{nd}$ sextant (p=0.016; $\chi^2$ test) | | | | | |
| Smokers | 17.4 | 31.7 | 24.0 | 15.0 | 5.2 |
| Non-smokers | 25.3 | 37.3 | 19.3 | 10.4 | 3.7 |
| $3^{rd}$ sextant (p=0.021; $\chi^2$ test) | | | | | |
| Smokers | 7.0 | 23.7 | 16.4 | 32.8 | 9.8 |
| Non-smokers | 11.5 | 31.3 | 17.2 | 25.6 | 6.8 |
| $4^{th}$ sextant (p=0.043; $\chi^2$ test) | | | | | |
| Smokers | 5.6 | 30.0 | 14.3 | 30.3 | 11.5 |
| Non-smokers | 10.2 | 35.2 | 14.1 | 27.2 | 6.5 |
| $5^{th}$ sextant (p=0.007; $\chi^2$ test) | | | | | |
| Smokers | 4.2 | 14.3 | 58.9 | 15.7 | 3.5 |
| Non-smokers | 8.6 | 18.8 | 59.0 | 8.1 | 3.1 |
| $6^{th}$ sextant (p=0.011; $\chi^2$ test) | | | | | |
| Smokers | 5.9 | 27,9 | 15,3 | 30,0 | 11,5 |
| Non-smokers | 10.2 | 37,1 | 14,4 | 24,5 | 6,8 |

Table 25 shows a detailed description of the influence of sex on max CPITN (%). It was evident that there were no significant differences in percentage of CPITN scores 0, 1, 2, 3 and 4 among females and males.

**Table 25:** Influence of sex on max CPITN (%) ($p = 0.2605$; $\chi^2$ test)

| Max CPI | Females | Males | Total |
|---|---|---|---|
| 0 | 1.5 | 1.5 | 1.5 |
| 1 | 16.4 | 11.7 | 14.0 |
| 2 | 29.2 | 25.8 | 27.5 |
| 3 | 34.7 | 40.5 | 37.6 |
| 4 | 18.2 | 20.6 | 19.4 |
| Total | 100.0 | 100.0 | 100.0 |

Table 26 gives a detailed description of the influence of age on max CPITN (%). It was evident that the younger age group (30-49 years) had a higher percentage of max CPITN 0 and 1 and lesser percentage of max CPITN 3 and 4 compared to older age group (50-69 years).

**Table 26:** Influence of age on max CPITN (%) ($p < 0.001$; $\chi^2$ test)

| Max CPI | Younger age group (30-49 years of age) | Older age group (50-69 years of age) | Total |
|---|---|---|---|
| 0 | 2.0 | 0.5 | 1.5 |
| 1 | 18.3 | 4.9 | 14.1 |
| 2 | 30.7 | 20.5 | 27.5 |
| 3 | 33.9 | 45.9 | 37.6 |
| 4 | 15.1 | 28.3 | 19.3 |
| Total | 100.0 | 100.0 | 100.0 |

Analysing the influence of education on max CPITN (%), it was obvious that the respondents with university graduation had a higher percentage of max CPITN 0 and the respondents with

basic education had a higher percentage of max CPITN 4. A detailed description is given in table 27.

**Table 27:** Influence of education on max CPITN (%) ($p = 0.0028$; $\chi^2$ test)

| Max CPI | Basic education | Skilled | High school education | University graduation | Total |
|---|---|---|---|---|---|
| 0 | 0.6 | 2.0 | 1.8 | 2.0 | 1.5 |
| 1 | 11.6 | 15.7 | 14.1 | 16.3 | 14.1 |
| 2 | 18.0 | 31.4 | 31.0 | 30.6 | 27.5 |
| 3 | 37.8 | 39.2 | 38.0 | 35.4 | 37.5 |
| 4 | 32.0 | 11.8 | 15.1 | 15.6 | 19.4 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Analysing the influence of preventive dental visits on max CPITN (%), it was obvious that the respondents who never visited a dentist before had a higher percentage of max CPITN 4 compared to that of respondents attending preventive check-ups. A detailed description is given in table 28.

**Table 28:** Influence of preventive dental visits on max CPITN (%) ($p < 0.001$; $\chi^2$ test)

| Max CPI | 2 times a year | Once a year | Once in 2 years | Less frequently | Never before | Total |
|---|---|---|---|---|---|---|
| 0 | 1.8 | 0.0 | 0.0 | 6.3 | 0.0 | 1.5 |
| 1 | 17.4 | 9.7 | 9.5 | 0.0 | 3.4 | 14.1 |
| 2 | 30.8 | 22.4 | 23.8 | 21.9 | 10.3 | 27.5 |
| 3 | 35.2 | 42.5 | 47.6 | 37.5 | 44.8 | 37.6 |
| 4 | 14.8 | 25.4 | 19.0 | 34.4 | 41.4 | 19.3 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 29 gives a detailed description of the influence of tooth brushing frequency on max CPITN (%). It was evident that respondents brushing their teeth less frequently had a higher percentage of max CPITN 4 compared to those brushing more frequently.

**Table 29:** Influence of tooth brushing frequency on max CPITN (%) ($p < 0.001$, $\chi^2$ test)

| Max CPI | 3 times daily | 2 times daily | Once daily | Less frequently | Total |
|---|---|---|---|---|---|
| 0 | 3.1 | 1.2 | 1.3 | 6.7 | 1.5 |
| 1 | 18.8 | 14.8 | 7.8 | 0.0 | 14.0 |
| 2 | 29.7 | 29.3 | 19.5 | 0.0 | 27.5 |
| 3 | 29.7 | 38.3 | 40.3 | 33.3 | 37.6 |
| 4 | 18.8 | 16.4 | 31.2 | 60.0 | 19.4 |
| Total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Analysing the influence of tobacco consumption on max CPITN (%), it was evident that non-smokers had a higher percentage of max CPITN 0 compared to other groups and regular smokers had a lower percentage of max CPITN 1 and a higher percentage of max CPITN 4 compared to other groups. A detailed description is given in table 30.

**Table 30:** Influence of tobacco consuming on max CPITN (%) ($p = 0.0071$; $\chi^2$ test)

| Max CPI | Regular smokers | Occasional smokers | Ex-smokers | Non-smokers | Total |
|---|---|---|---|---|---|
| 0 | 1.4 | 0.0 | 0.0 | 2.1 | 1.5 |
| 1 | 9.0 | 19.5 | 9.9 | 16.4 | 14.0 |
| 2 | 23.4 | 31.7 | 28.6 | 28.3 | 27.5 |
| 3 | 35.2 | 34.1 | 48.4 | 36.2 | 37.6 |
| 4 | 31.0 | 14.6 | 13.2 | 16.9 | 19.4 |
| total | 100.0 | 100.0 | 100.0 | 100.0 | 100.0 |

Table 31 gives a detailed description of the various independent variables (sex, age, education, preventive dental visits, brushing frequency and smoking habits) and the corresponding max CPITN scores that influenced the respective variable to be significant. It was evident that age ($p<0.001$), education ($p<0.01$), preventive dental visits ($p<0.001$), brushing frequency ($p<0.001$) and smoking habits ($p<0.01$) had a significant influence on the

periodontal status where as gender of the respondents had no significant influence on the periodontal health (p=0.261).

**Table 31:** Influence of chosen variables on CPITN (maximum) scores.

($\chi^2$ test of independence in contingency tables)

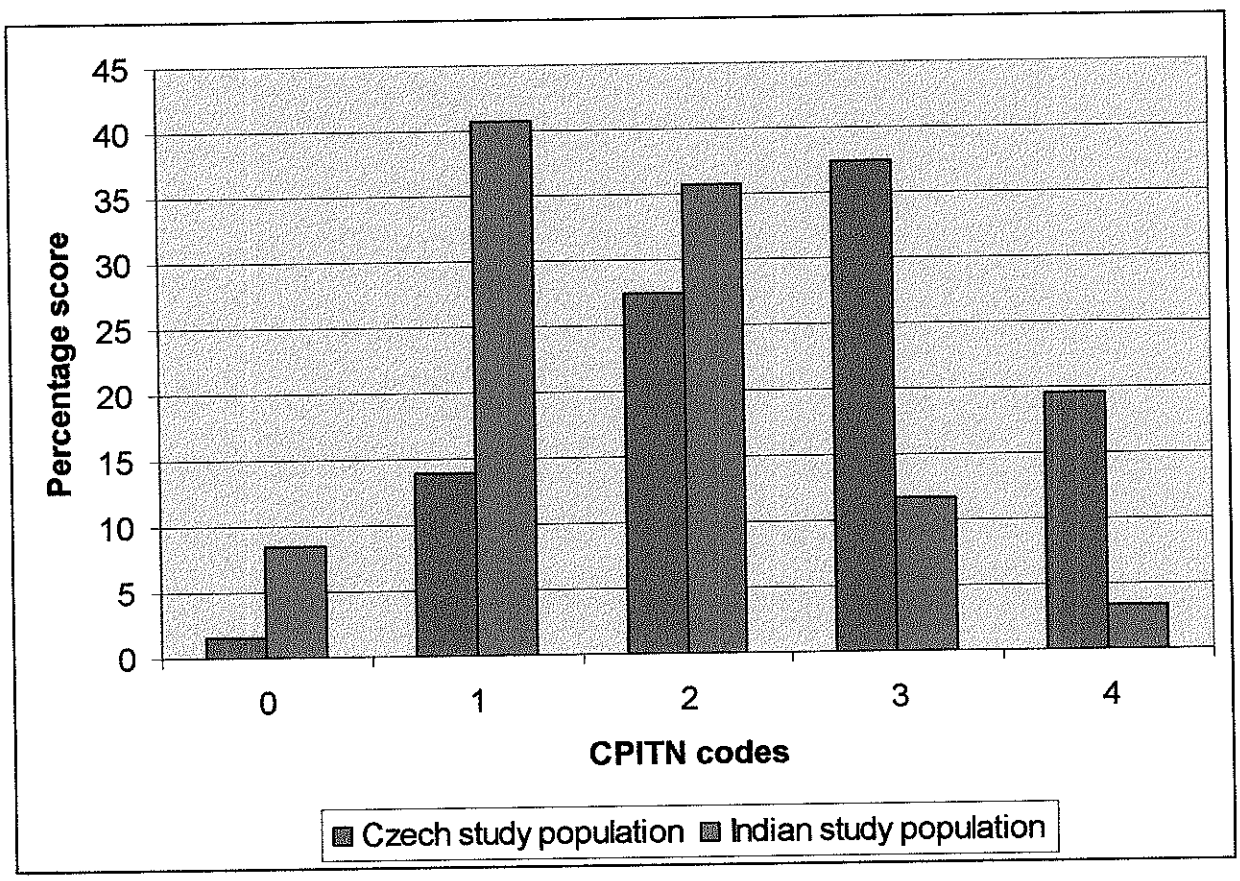
| Variable | p-value | Chi-Square Contribution |
|---|---|---|
| Sex | 0.2605 | No significant difference. |
| Age | 0 *** | Max CPI-3,4<br>- More frequently in the age group 50-69 years.<br>Max CPI-1,2<br>- Less frequently in the age group 50-69 years. |
| Education | 0.002847 ** | Max CPI-4<br>- More frequently in respondents with basic education. |
| Preventive dental visits | 0.000060 *** | Max CPI-4<br>- Less frequently in respondents participating in preventive check-ups twice a year.<br>- More frequently in respondents not participating in preventive check-ups. |
| Brushing frequency | 0.000154 *** | Max CPI-4<br>- More frequently in respondents brushing their teeth once daily or less.<br>- Less frequently in respondents brushing their teeth twice daily. |
| Smoking | 0.007109 ** | Max CPI-4<br>- More frequently in regular smokers.<br>Max CPI-3<br>- More frequently in former (ex) smokers.<br>Max CPI-1<br>- Less frequently in regular smokers. |

## 6.3. COMPARATIVE ANALYSIS OF CPITN SCORES OF CZECH AND INDIAN STUDY POPULATION

Comparison between percentage of scores according to CPITN categories of Czech and Indian population revealed that the Indian study population had a higher percentage of CPITN scores 0, 1 and 2 indicating healthy periodontium, bleeding on probing and supra-gingival or sub-gingival calculus respectively and a lower percentage of CPITN scores 3 and 4 indicating pocket depths up to 4-5 mm and 6 mm or more respectively compared to Czech study population.

**Diagram 2**

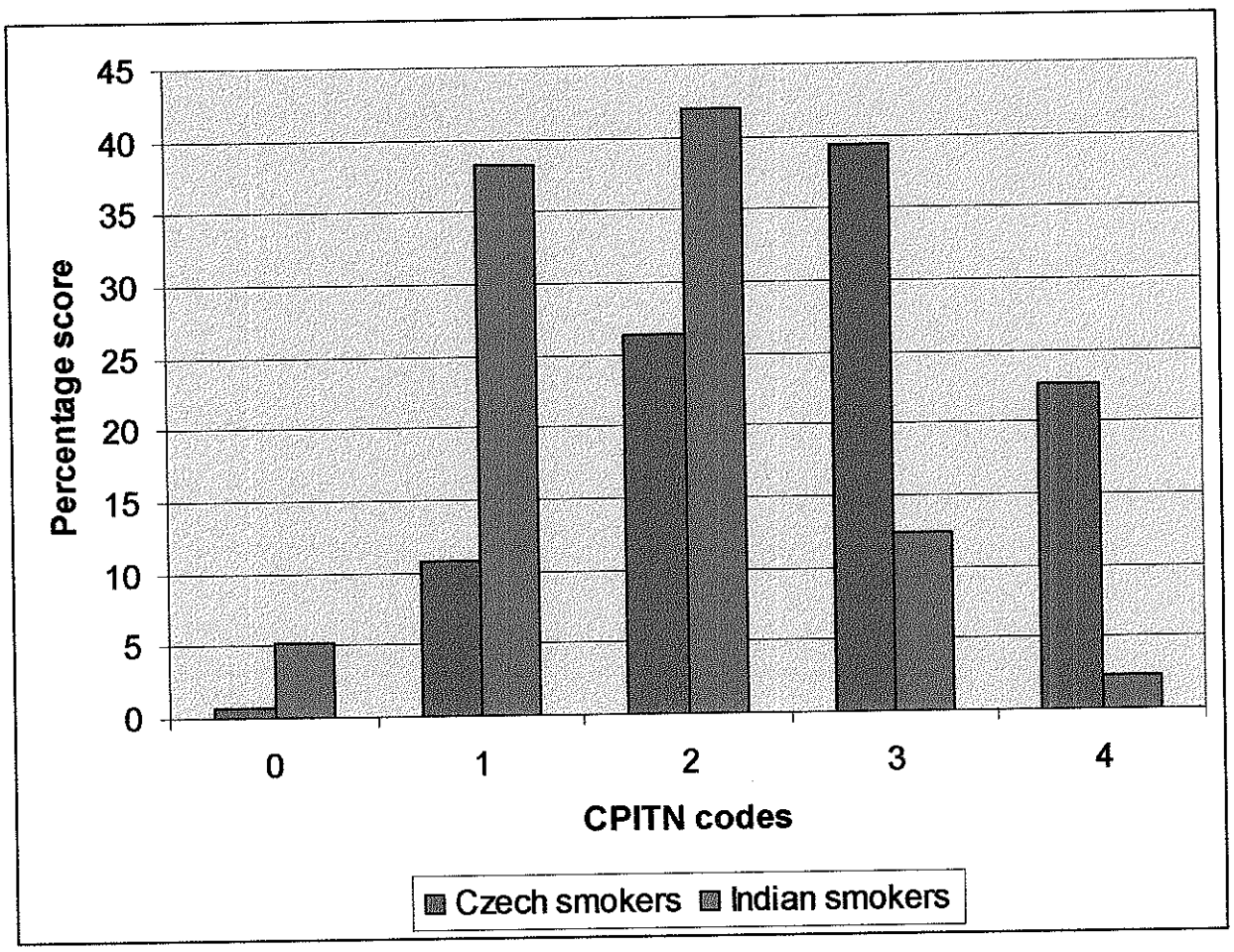
Comparison between percentage of scores according to CPITN categories of Czech and Indian study population ($p<0.001$; $\chi^2$ test)



Comparison between the percentage of scores of smokers according to CPITN categories of Czech and Indian study population revealed that the smokers in Indian study population had a higher percentage of CPITN scores 0, 1 and 2 indicating healthy periodontium, bleeding on probing and supra-gingival or sub-gingival calculus respectively and a lower percentage of CPITN scores 3 and 4 indicating pocket depths up to 4-5 mm and 6 mm or more respectively compared to the smokers in the Czech study population.

**Diagram 3**

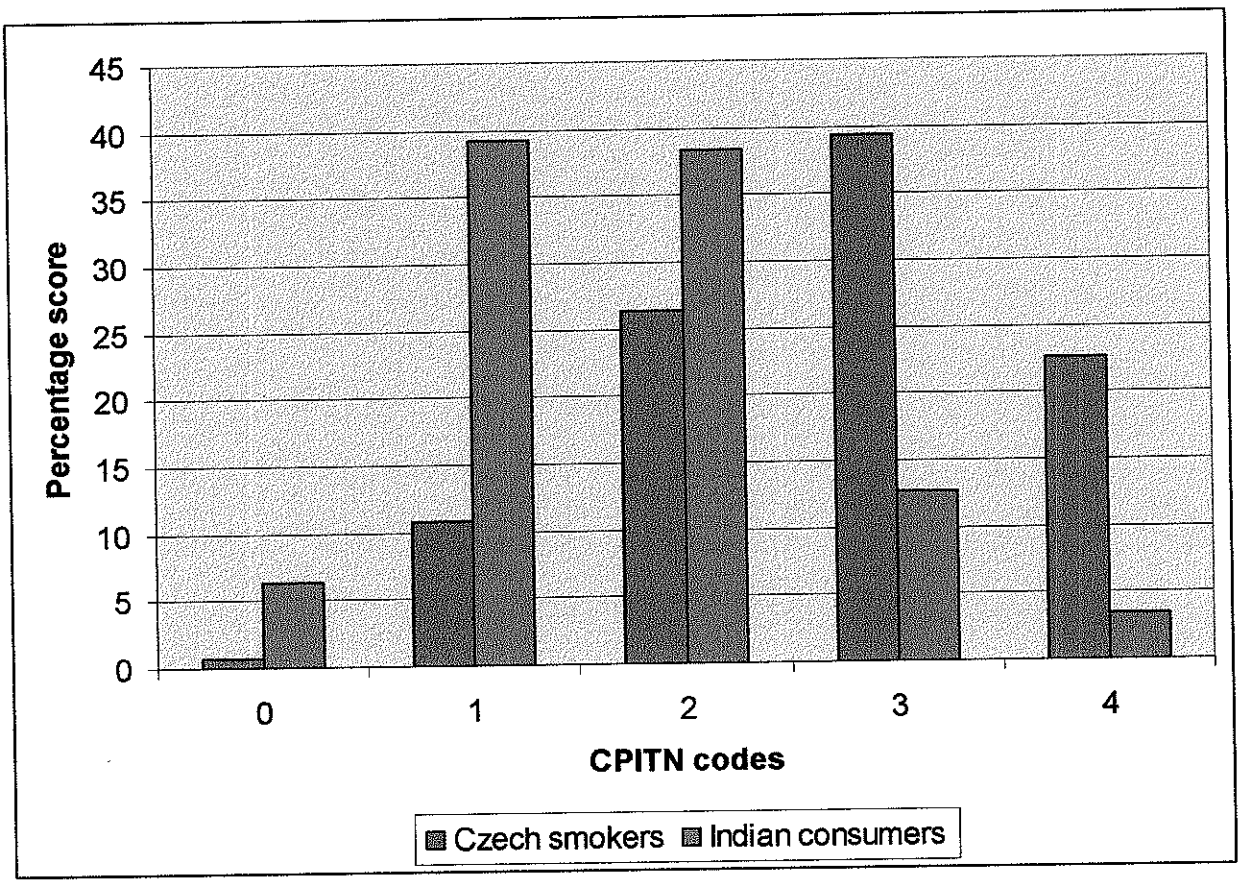
Comparison between percentage of scores of smokers according to CPITN categories of Czech and Indian study population ($p<0.001$; $\chi^2$ test)



Comparison between percentage of scores of smokers (Czech) and consumers (Indian) according to CPITN categories of Czech and Indian study population revealed that the consumers in the Indian study population had a higher percentage of CPITN scores 0, 1 and 2 indicating healthy periodontium, bleeding on probing and supra-gingival or sub-gingival calculus respectively and a lower percentage of CPITN scores 3 and 4 indicating pocket depths up to 4-5 mm and 6 mm or more respectively compared to the smokers in the Czech study population.

**Diagram 4**

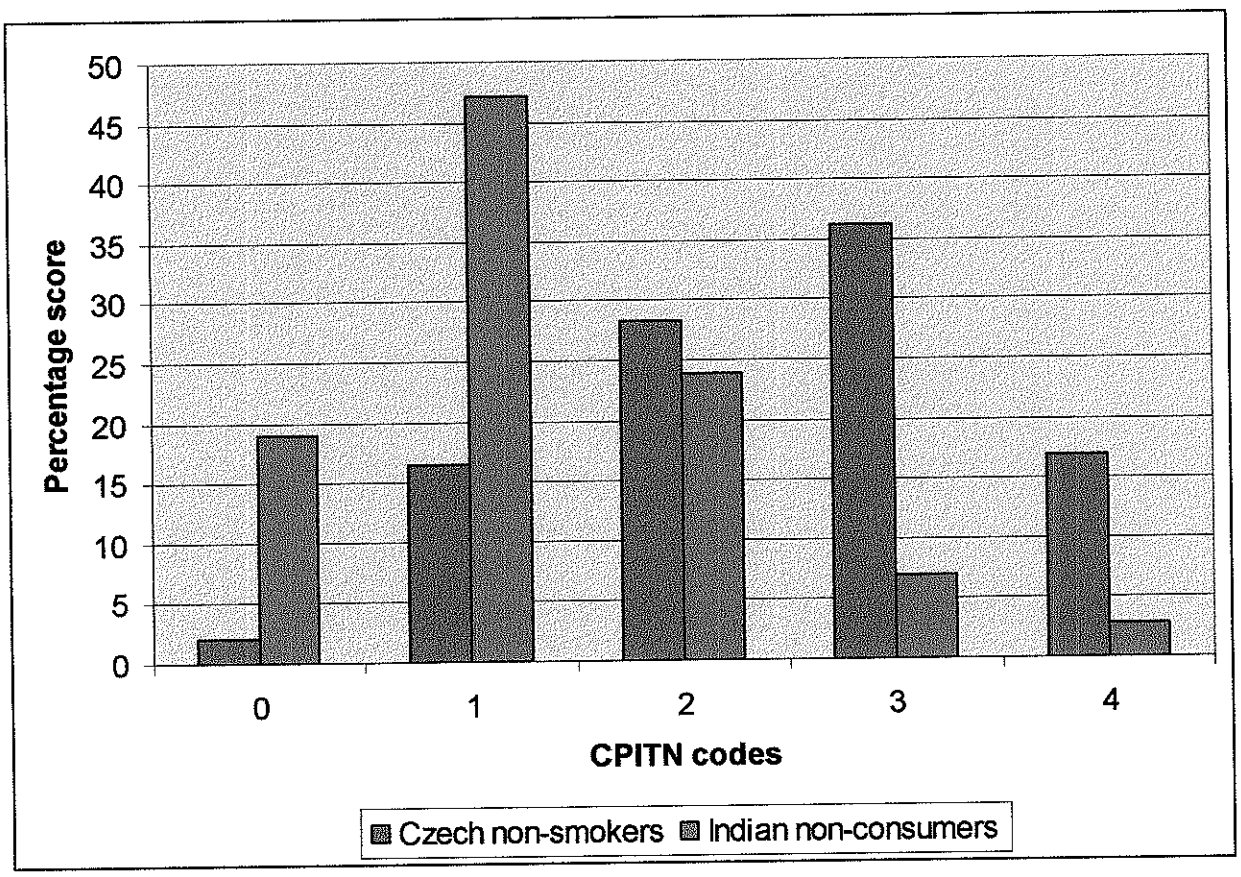
Comparison between percentage of scores of smokers (Czech) and consumers (Indian) according to CPITN categories of Czech and Indian study population ($p<0.001$; $\chi^2$ test)



Comparison between percentage of scores of non-smokers (Czech) and non-consumers (Indian) according to CPITN categories of Czech and Indian study population revealed that the non-consumers in the Indian study population had a higher percentage of CPITN scores 0 and 1 indicating healthy periodontium and bleeding on probing respectively and a lower percentage of CPITN scores 2, 3 and 4 indicating supra-gingival or sub-gingival calculus, pocket depths up to 4-5 mm and 6 mm or more respectively compared to the non-smokers in the Czech study population.

**Diagram 5**

Comparison between percentage of scores of non-smokers (Czech) and non-consumers (Indian) according to CPITN categories of Czech and Indian study population ($p<0.001$; $\chi^2$ test)



# 7

# DISCUSSION

Tobacco consumption may be the greatest single cause of premature death in the developed world and, increasingly, in the less developed countries. The US Surgeon General has been quoted as saying that 'smoking represents the most extensively documented cause of disease ever investigated in the history of biomedical research' (30).

One of the conclusions to emerge from a review of longitudinal studies in the 1996 World Workshop in Periodontics was that 'the interaction between environmental and subject related factors does not have to be constant in geographically or racially different populations'. Most of the studies that have identified tobacco as a true risk factor for periodontitis have been carried out in the West and have focussed on the habit of tobacco smoking (114). The present study samples were taken from a population of a developing country characterized by varying standards of oral hygiene measures and practices, socioeconomic status, and peculiar modes of tobacco consumption and also from a population of a developed country where the most commonly practiced mode of tobacco consumption is smoking and also where the medical insurance based preventive dental visits are more frequently utilized. Given that the final expression of periodontitis is based on the complex interactions occuring between host, microbial and environmental factors (109), it was perceived that the contribution of tobacco as a risk factor for periodontitis in this perticular setting might be worthy of investigation and comparison. The present study was intended to augment scientific knowledge about the effects of tobacco on periodontitis by re-examining this relationship in two different socio-behavioral contexts.

Self-reported oral health questionnaires are used widely in epidemiological oral health investigations because they are time and cost-effective and provide detailed information on subjects in a single health examination. The validity and reliability of these questionnaires are crucial if the self-assessed data on oral health and disease is to be useful (1). Ho et al. (63) concluded that although information provided by the subject may not be as accurate as laboratory testing when screening general and oral health of populations, it is nevertheless a valuable source of data that can be utilized cost-effectively in research studies. In a meta-analysis, Patric et al. (116) concluded that self-reports of smoking are accurate in most studies. Thus, a questionnaire enquiry was presumed to be appropriate for this study. Certain questions (e.g., regarding medical history, food habits etc) were asked but excluded from the present analysis since these were beyond the scope of this study and is discussed elsewhere.

The criteria for recognizing periodontal disease severity differ among various studies, with some using mean clinical attachment level or percentage sites with clinical attachment level about a certain cut off point, some using radiographic assessments of bone level, and others using the combination of clinical attachment level and probing depth measurements (145). Investigators have recognized that mean values of any index alone is not adequate enough to describe the nature of periodontal disease in populations because of the marked variation between and within subjects. Despite the fact that most clinical indicators used in the CPITN are either inadequately sensitive or specific as predictors for the detection of groups or individuals at risk of periodontal disease and that it does not adequately reflect the distribution of periodontal disease in a population (93), its use renders knowledge about the high prevalence and low severity of periodontal disease among populations (122, 137). A study measuring CPITN by probing on 2 sites (mesiobuccal and buccal) per tooth, and all around each tooth reported that even though probing on 2 sites saves time during examination and facilitates examiner calibration, it leads to considerable underestimation of periodontal problems (19). Thus, taking all these into consideration and also the simplicity in recording and its world-wide application allowing for international comparisons, CPITN Index was used for this study by probing all around each tooth to assess periodontal conditions. A CPITN score of 3 and 4 was considered as presence of periodontitis. The index parameters for evaluation of periodontal conditions were bleeding, calculus and pockets.

The Czech study population consisted of patients attending the Department of Dentisty, Teaching Hospital, Hradec Králové and also patients attending three private dental practitioners who were cooperating with the study. This was done to eliminate selection bias because most of the patients attending Department of Dentistry, Teaching Hospital, Hradec Králové were referred by private practitioners for specialized treatment of advanced periodontitis. The Indian study population was drawn from SRM Dental College and Hospital, Chennai, India.

The minimum age for participating in the study was fixed as 30 years under the assumption that majority of tobacco users start the habit in their teenage or early adulthood and considering the fact that it takes some time for its destructive effect or clinical manifestation to be obvious.

**Tobacco use and other variables**

This study showed that smoking rate was high among the males of the representative Czech and Indian sample compared to females. A higher smoking rate among Czech males compared to females was in agreement with the reports of the WHO in the year 2005 (42). The social and cultural norms in India may preclude women from smoking. This social and cultural pressure may prevent some female smokers from reporting their true smoking status and thus, may have affected the study results. A similar trend of smoking status was reported in a study conducted in India (121) and in Vietnam (44). However, in case of the Indian representative sample, the percentage of females in the group of 'consumers but non-smokers' was higher compared to that of males. This may be explained by the fact that tobacco chewing is a common practice among females of lower economic strata in India. It was also evident that a higher percentage of respondents with better education, i.e., graduates, abstained from using chewing tobacco or beedi/chutta smoking. This may be probably because these forms of tobacco, being much cheaper compared to cigarettes, are commonly used by people belonging to lower economic class and thus becoming an issue of status concern for the educated people. These results are in agreement with results of studies conducted in India by Subramanian SV et al. (133) and Sorensen G et al. (135).

A review by Harwood et al. stated that initially, those of higher socioeconomic status (SES) were more likely to smoke cigarettes. However, since the first Surgeon General's report in 1964, the profile of cigarette smokers has reversed. Cigarette smokers now are more likely to be poor and less educated. The well educated may have higher levels of health literacy, and were more responsive to messages of health-promoting and disease-prevention behaviors and beliefs. Additionally, the poor may have least information on the health risks of smoking, the fewest resources, and the least access to cessation services. Socioeconomic disadvantage is associated with persistent smoking, and consequently the burden of smokng-related disease falls disproportionately on those with lower SES (61).

Taking the educational qualification of respondents into consideration, regular smokers of the Czech sample and 'consumers but non-smokers' of the Indian sample having lower educational qualification is consistent with the results of previous studies conducted in other developed countries and in India. Researchers of a study conducted in Australia noted that those leaving in low socioeconomic status areas were more likely to smoke (106). A study

published in the Journal of Canadian Dental Association stated that a higher percentage of current smokers had education less than high school (99) and a study conducted in America stated that inequalities in smoking exist, as evidenced by persistent class-based disparities and the growing number of smokers in the lower socioeconomic groups (132). A similar trend was noted by researchers in India where chewing tobacco was more commonly used by people with lower education (133).

Taking income of respondents into consideration, regular smokers of Indian sample having lower income compared to other groups is in agreement with other studies conducted in India in this regard (133).

Majority of Czech respondents visiting the dentist twice a year for dental check-ups can be explained by the fact that the dental check-up is covered by the Social and Health Insurance existing in Czech Republic. Results of studies conducted in other European countries like that conducted in Spain revealed that only 13 % of the study population visited dentist for regular check-up at least once a year (92). Where as, majority of Indian respondents visiting the dentist only when having some dental problems can be explained by multiple factors such as high cost of dental treatment, lesser accessibility to dentists, lack of dental health awareness amongst the poor or non-inclusion of yearly regular dental check-up that is required for medical health insurance in India (133).

A study conducted in Spain revealed that 46 % of subjects brushed their teeth at least once daily (92) where as, a higher percentage of Czech respondents brushed their teeth twice daily. A majority of Indian respondents brushing their teeth once daily was consistent with the results of a study conducted in India (133).

Indian respondents using tobacco with betel nut and leaves, smoking bidi/chutta and smoking cigarettes with/with out filters having higher percentage of oral mucosal lesions or changes compared to non-consumers may be due to the local irritation of oral mucosa caused by using these forms of tobacco. These results are in agreement with the results of previous studies conducted in this regard (141). No significant influence of smoking on oral mucosa of the Czech respondents may be due to the fact that they are not exposed to other tobacco forms like chewing.

**CPITN and other variables**

The gender of the Indian respondents significantly influenced the maximum CPITN outcome where as the gender of Czech respondents had no influence on the maximum CPITN outcome. Several periodontal diseases have been found to be more prevalent among males, even after oral hygiene, socioeconomic status and age have been taken into account and hormonal conditions have been proposed to explain the difference (55). It has been reported that although there is no established, inherent difference between men and women in their susceptibility to periodontitis, men have been shown to exhibit worse periodontal health than women and this difference has been documented in different populations (24). Females of the Indian study population having a better periodontal profile compared to males may be due to better oral hygiene practices and/or more utilization of oral health care services or due to the differences in tobacco consumption habits. In the Indian study population, females smoked less where as in the Czech study population, a higher percentage of females were found to be regular smokers. Possible explanations for diverging results when comparing the Czech and Indian results may be due to population characteristics as well.

Age of the Indian respondents had no significant influence on the maximum CPITN outcome where as the age of Czech respondents significantly influenced the maximum CPITN outcome. Early evidence demonstrated that both the prevalence and severity of periodontitis increase with increasing age, suggesting that age may be a marker for periodontal tissue support loss. Later, it was established that periodontitis might have its onset in youth and early adulthood, rather than older years (24). A national survey conducted in 1986 in Brazil using CPITN methodology to assess the periodontal status estimated that 5.2 % and 7.4 % of subjects in the age groups 35 to 44 and 50 to 59 years had one or more teeth with probing depth of $\geq$ 5.5 mm (CPITN 4) (136) and another national survey in the United Kingdom using CPITN estimated that 42 % of 35 to 44 year olds and 70 % of 55 to 64 year olds had CAL > 3.5 mm (102). Older age group (50-69 years) of the Czech study population having a higher percentage of maximum CPITN scores 3 and 4, indicating pathological pocket, may be due to the cumulative effect of prolonged exposure to true risk factors including cigarette smoking. Again, diverging results when comparing the Czech and Indian results may be due to population characteristics.

Education significantly influenced the maximum CPITN outcome of both Czech and Indian study population. Previous studies have documented differences in periodontal health by socioeconomic indicators, i.e., income and education but these indicatiors have rarely been investigated as independent variables of main interest. Socioeconomic indicators are robust markers of periodontitis. Their role in periodontal disease can be attributed to differential access to resources and opportunities that may influence preventive behaviours. Evidence also suggests that education has a greater influence than income in favourably affecting the level of periodontitis in the population (24). University graduates of both Czech and Indian study population having a higher percentage of maximum CPITN score 0, indicating healthy periodontium, can be attributed to the factors mentioned above.

Preventive dental visits and brushing frequency significantly influenced the maximum CPITN outcome of both Czech and Indian population. A general principle in preventive efforts towards chronic diseases is to focus on changeable causal or modifying factors. Regarding periodontal diseases, such factors are those related to life style such as oral hygiene, regularity of dental visits and tobacco use. It seems that mainly the mild and moderate forms of periodontitis are influenced by preventive actions such as plaque control, utilization of dental services and an improvement of the general health attitudes of the societies. Comparative studies between the Eastern European countries and the Western societies showed that socioeconomically less-developed Eastern European countries displayed a higher fraction with mild-to-moderate periodontitis than the Western well-developed societies. Particularly, the Scandinavian countries where a comprehensive public dental health care system with emphasis on prevention and regular dental visits has existed for more than 100 years, displayed high proportions of healthy subjects and even a low prevalence of severe periodontal disease (54). One study reported that the subjects who had a dental checkup at least once a year had significantly less gingivitis, calculus and periodontal pockets compared to those who made less frequent visits (84). However, the same group of authors failed to find a relationship between dental insurance and improved periodontal health (85), although those with insurance were more likely to visit the dentist (69). Differences among the populations of the world in terms of periodontal status, oral cleanliness and oral health behaviour probably reflect the social and economic development of the various regions. Cultural differences may also affect the attitudes towards dental health and dental care in populations (54). Lack of use

of preventive care may reflect a general attitude toward preventive care, differences in willingness or ability to pay for dental services or differences in the availability of dental care.

The findings from this study that non-smokers/non-consumers exhibiting a higher percentage of healthy periodontium compared to smokers/consumers corroborates the results of several previous studies (16, 139, 145, 154, 156). This study also reconfirmed the relationship between smoking and a reduced gingival bleeding on probing, which has been well documented in previous studies (43, 105, 144). This may be due to vasoconstrictive action of nicotine and as a result of a profound influence on vascular dynamics and cellular metabolism (20).

Smoking as a strong and consistent risk indicator for periodontitis with the presence of calculus, an indicator of oral hygiene, in logistic regression model has been documented (44) and smokers have been reported to exhibit a low awareness of their health (138). From this study, it was evident that smokers or consumers of tobacco had a higher percentage of supragingival and/or subgingival calculus compared to non-consumers. A study conducted in Lithuania using CPITN index reported that smoking negatively influenced CPITN index. According to their findings, the CPITN index of the subjects who smoke was significantly higher than that of non-smokers and the smokers had higher number of sextants with calculus compared to non-smokers (76). Where as, studies conducted elsewhere showed that periodontal conditions as measured by CPITN were not significantly different among smokers and non-smokers (8).

As proposed by Gelskey, smoking meets the majority of nine criteria for causation according to Hill to varying degrees.

- Strength of association: Cross-sectional and case-control studies demonstrate a moderate to strong association between smoking and periodontitis.
- Consistency: Multiple studies of various designs (cross-sectional, case-control and longitudinal) and in various populations have demonstrated an association between smoking and periodontal attachment loss.
- Specificity: Disease progression slows in patients who quit smoking as compared to those who continue to smoke.

- Temporality: Longitudinal studies show that smokers do not respond as well to periodontal therapy as non-smokers.
- Biologic gradient: There is dose-response effect in that heavy smokers have increased disease severity compared to light smokers.
- Biologic plausibility: The biologic plausibility of the explanation of the relationship between smoking and periodontitis is supported by tobacco's adverse impact on microbial and host response parameters.
- Coherence: The effects of smoking on periodontitis are consistent with our knowledge of the natural history of periodontal disease.
- Analogy: Periodontal effects of smoking are analogous to other adverse smoking-related general health effects.
- Experiment: evidence not currently available (48).

Thus, taking the above criteria and the findings from this study into consideration, the significant effect of tobacco use on the prevalence of periodontitis is confirmatory as reported by several researchers who have investigated the tobacco-periodontal relationship in alternative cultures. Nevertheless, oral hygiene practices, preventive dental visits, and socioeconomic status may be considered as risk factors for periodontitis.

**Limitations of the study**

Certain limitations should be taken into consideration when interpreting the results of this study. The validity of self-reported smoking is often questioned because of the widespread belief that smokers are inclined to underestimate the amount smoked or to deny smoking altogether (116). In addition, self-reported exposure to ETS may require detailed questionnaire items (71). The cumulative effect of periodontal destruction over time such as attachment loss, recession and loss of alveolar bone were not recorded by the CPITN index since it was originally constructed for the assessment of treatment needs (111). The full CPITN version used in the present study, although examining all teeth, could be considered a partial instrument since the multiplicity of sites in not considered for the diagnosis when a single score is applied for each sextant. Thus the index scores may not fully reflect the true periodontal condition. Another limitation was that the plaque levels were not recorded during the dental examination. Consequently, adjustments for plaque could not be made in the

present analysis. However, most studies reported similar plaque levels for smokers and non-smokers (20, 59) and no difference between smokers and non-smokers with regard to plaque accumulation could be observed in experimental gingival studies (39, 88). Another limitation was the inability to generalize our findings to the Indian and Czech population. Our study group was a convenient sample and was not randomized. A comparison of our data with recent studies was not always possible because of differences in methodology.

# 8

# CONCLUSIONS

Within the limitations of this study, the following conclusions were drawn:

- Non-smokers/non-consumers had a higher percentage of healthy periodontium and sites with bleeding on probing demonstrated by higher percentage of CPITN scores 0 and 1 respectively, compared to smokers/consumers.
- Smokers/consumers had higher percentage of shallow and deep pockets demonstrated by higher percentage of CPITN score 3 and 4 respectively, compared to non-smokers/non-consumers.
- Age of the respondents significantly influenced the outcome of CPITN scores in case of the Czech study population where as age did not have any influence on the outcome of CPITN scores of Indian study population.
- Gender of the respondents significantly influenced the outcome of CPITN scores in case of Indian study population but had no influence on the outcome of CPITN scores of Czech study population.
- Education, participation in dental preventive check-ups, frequency of tooth brushing and smoking significantly influenced the outcome of CPITN scores in both the study populations.
- Regular smokers and 'consumers but non-smokers' of the Indian study population demonstrated a higher percentage of oral mucosal changes or lesions compared to other groups but smoking had no significant influence on oral mucosa of the Czech respondents.
- Majority of Indian respondents visited dentists only when they had some dental problems and brushed their teeth once daily where as majority of Czech respondents visited dentists for regular dental check-ups twice a year and brushed their teeth twice daily.
- The comparative analysis of CPITN scores of Czech and Indian study population revealed that irrespective of higher percentage of Czech respondents participating in regular dental check-ups twice a year and brushing their teeth twice daily, they had worse periodontal findings (lower percentage of healthy periodontium and higher percentage of shallow and deep pockets) compared to Indian respondents.

# 9

# IMPLICATIONS OF THE STUDY

Research about the relation between smoking and periodontal health and disease has provided not only important knowledge about smoking as one of the most decisive factors for the onset and progression of the disease but has, in addition, offered new insight into the characteristics of this particular disease. The understanding that periodontal disease may develop in response to environmental factors like smoking suggests that the traditional view (paradigm) of the disease as a reaction to plaque infection is no longer tenable and has to be replaced by a new concept that also takes into consideration other factors. Once one environmental factor has been identified, there are probably others to be detected. The current understanding of the role of smoking thus raises questions about the appropriateness of the prevailing paradigm focusing on 'infection'. A re-evaluation of chronic periodontal disease and its causality based on a broader environmental perspective is warranted. The necessity for a conceptual reorientation is not unique to periodontal disease. Over the past decades epidemiological research has brought about a transition in that non-infectious diseases have replaced infectious diseases as the most common cause of morbidity and mortality in the industrialized world. According to this paradigm, chronic diseases are considered preventable problems caused by multiple determinants in our environment. Chronic periodontal disease as a multifactorial non-infectious problem well conforms to such a concept.

Because smoking is associated with over 40 various diseases, it is evident that the periodontal tissues share a susceptibility to smoking with 40 other tissues or organs of the body. The self-evident conclusion to be drawn is that the periodontal tissues react to or are influenced by functions that regulate the body as a whole. As a further consequence, periodontal disease can be regarded as a 'systemic' rather than a 'local' disease. The end stage of periodontal destruction is the loss of masticatory function, and along with the gradual breakdown of the periodontal tissues and ensuing pocket formation, the root surfaces become microbially colonized or infected. Whether or not microbial colonization is necessary for the progression of periodontal disease associated with smoking is not known, but it may amplify the problem and contribute to acute exacerbations.

The current understanding of the importance of tobacco smoking as the most potent risk factor for chronic periodontal disease now has to be applied to the clinical management of the disease. Treatment of smoker patients not including corrective measures against the smoking behavior should be regarded as unethical. As a first step, the smoking patient has to be informed about the fact that the treatment outcome will be less beneficial than normally

expected or not beneficial at all, and that the risk of failure is overwhelming. Secondly, the hazardous influence of smoking has to be considered in the overall patient management. Here the cutting down on smoking is the most important part. The patient, no doubt, decides on his/her smoking, but the therapist makes the decisions on the therapy well aware that, in the long run, the prognosis of periodontal treatment for patients who continue to smoke is poor.

Although counselling on smoking and the implementation of strategies for smoking cessation are important tasks, the great challenge to the periodontal profession for the future is to counteract the initiation of smoking in new generations. For this purpose, periodontology and periodontal health care have to become more oriented towards the public. Because smoking habits are established early in life, the prevention of periodontal disease has to be encouraged among children and adolescents. Every young individual who starts smoking is a potential patient for the future deliverers of periodontal care. It can be readily realized that reducing smoking in the population will not only reduce individual discomfort but also public expenditure. The socioeconomic impact of smoking on the individual and public cost of periodontal care, surprisingly, has been paid only little attention to.

**Role of dental professionals in smoking cessation:**

The World Dental Federation (FDI) adopted a Position Statement on Tobacco in 1996 in which all oral health professionals were urged to integrate tobacco use prevention and cessation services into their routine and daily practice (124). There are important links between smoking and oral health that provide a unique opportunity for action by the dental team. The early effects of smoking are often clearly evident on the facial and oral tissues. These changes can be visible to patients and are reversible on successful cessation. They can therefore be used as a marker of the impact of smoking on the body and in a motivational counselling. Halitosis and tooth staining are also common concerns of smokers and can be used as motivations for quitting.

Smoking cessation delivered in primary care has been shown to be very effective. Recently published effectiveness reviews have systematically evaluated the evidence and identified an international consensus on smoking cessation recommendations. Very brief advice lasting less than 3 minutes given by a health professional will help an additional 2 % of smokers to successfully stop smoking. With more intensive support lasting up to 10 minutes, plus nicotine replacement therapy (NRT) an additional 6 % of smokers will quit.

In an attempt to encourage dentists to become more involved, a smoking cessation protocol has been developed to give advice and support in the most effective manner in the dental practice. To be effective, smoking cessation advice needs to be tailored to the smokers' circumstance and experience. The five A's model is a simple and quick means of identifying smokers who want to quit and how best to support them in their quit attempt (152). The five A's model is described below:

*Ask*: Ask patients about smoking. A system should be implemented that ensures that every patient at every visit is asked about tobacco use, and the answer documented in the patient's record.

*Advice*: Advice all smokers to stop. A prescriptive approach should be avoided. Rather, the health care professional or the dentist should provide the patients with information and advice, reinforcing the patients' own motivation when possible and emphasizing the benefits of stopping. Immediate benefits will often motivate the patients more effectively than long-term benefits.

*Assess*: Assess the patient's willingness to stop. If the patient is willing to make an attempt to quit, dentist should assist the patient. If a patient is not at all interested in stopping, it is rarely beneficial to push the patient.

*Assist*: Assist the patient in stopping. If a patient has a desire to stop, the dentist should help the patient set a realistic quitting date which should be soon but not immediately so that the patient has time to prepare.

*Arrange*: Arrange follow-up contact. Follow-up contacts are very important as the chances of a successful outcome are improved when patients know that their progress will be reviewed (124).

*Nicotine replacement therapy (NRT)*: One of the most difficult problems in quitting is dealing with the inevitable cravings for nicotine. A course of nicotine replacement therapy helps with this and when used correctly, doubles the cessation rate. A variety of products are now available on the market (gum, patch, nasal spray, inhalator and microtabs).

Reported barriers to providing smoking advice include time and cost pressures, concerns regarding the effectiveness of interventions, inadequate training and a lack of appropriate resources (152). National and local dental societies and associations should become involved in the tobacco control programs which should include activities such as educating the public on the health hazards of environmental tobacco use, promoting smoke-free restaurants and

theatres, and prohibiting smoking in public places. Dental schools or colleges have to incorporate into their curriculum not only the harmful effects of smoking but also practical training in clinical intervention, thus, graduating the next generation of dentists with competency in assessing and treating tobacco use.

This study extends information on the relationship of tobacco use to periodontitis in two different study populations. The findings highlight the need for preventive strategies aimed at very young individuals, particularly as many of those who smoke take up the habit as teenagers. The conclusions drawn from this study can also be used for further longitudinal studies, especially for studying in detail about the factors that have influenced the Indian study sample to have a better periodontal profile with the study sample visiting dentist and brushing their teeth less frequently compared to the Czech study sample.

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# APPENDIX

## APPENDIX I

**Czech questionnaire**

**Dotazník pro účastníky studie zabývající se vlivem kouření na orální zdraví**

Respondent číslo

*(Zaškrtněte laskavě políčko ☐ u vybrané odpovědi či doplňte údaj na vytečkovaný řádek.)*

**1. Osobní údaje**

1.1. Pohlaví: ☐ Muž ☐ Žena

1.2. Věk: .....

1.3. Ukončené školní vzdělání: 1.3.1. ☐ základní/vyučen

1.3.2. ☐ vyučen s maturitou

1.3.3. ☐ středoškolské s maturitou

1.3.4. ☐ vysokoškolské

1.4. Čistý měsíční příjem na člena Vaší rodiny činí přibližně:

1.4.1. ☐ do 5 tisíc Kč 1.4.2. ☐ 5 – 10 tisíc Kč 1.4.3. ☐ nad 10 tisíc Kč

1.4.4. ☐ nevím 1.4.5. ☐ odmítám odpovědět

1.5. Zaměstnání/profese (např. učitel, dělník, úředník): .....

**2. Zdravotní stav**

2.1. Trpíte nějakou chronickou chorobou? 2.1.1. ☐ ano 2.1.2. ☐ ne

2.2. Jestliže ano, kterých orgánů se potíže týkají?

2.2.1. ☐ srdce a cév 2.2.2. ☐ ledvin a močového ústrojí

2.2.3. ☐ dýchacího ústrojí 2.2.4. ☐ psychiky

2.2.5. ☐ pohybového ústrojí 2.2.6. ☐ cukrovka

2.2.7. ☐ trávicího ústrojí 2.2.8. ☐ jiných orgánů, kterých .....

2.2.9. ☐ prodělal(a) jsem nádorové onemocnění léčené cytostatiky či ozařováním

2.2.10. ☐ odmítám odpovědět

2.3. Užíváte dlouhodobě nějaké léky? 2.3.1. ☐ ano, jaké .....

2.3.2. ☐ ne

**3. Jak často chodíte na preventivní prohlídky k zubnímu lékaři?**

3.1. ☐ 2x ročně 3.2. ☐ 1x za rok 3.3. ☐ 1x za 2 roky

3.4. ☐ méně často 3.5. ☐ nechodím na preventivní prohlídky

**4. Hygiena dutiny ústní**

4.1. Zuby si čistím: 4.1.1. ☐ dutiny 3x denně a častěji 4.1.2. ☐ 2x denně

4.1.3. ☐ 1x denně 4.1.4. ☐ méně často

4.2. Uveďte, jaké pomůcky ústní hygieny pravidelně používáte:

4.2.1. ☐ zubní kartáček 4.2.2. ☐ mezizubní kartáček

4.2.3. ☐ dentální nit 4.2.4. ☐ ústní vody

4.2.5. ☐ jiné, jaké .....

4.3. Zaškrtněte, která z následujících vět je ve Vašem případě pravdivá:

4.3.1. ☐ Po vyčištění zubů večer již nikdy nic nejím (ani jablko) ani nepiji sladké nápoje či pivo nebo mléko.

4.3.2. ☐ Po vyčištění zubů večer již většinou nic nejím.

4.3.3. ☐ Po vyčištění zubů večer jím pouze ovoce.

4.3.4. ☐ Po vyčištění zubů večer ještě konzumuji nějaké jídlo nebo piji sladké nápoje.

4.4. Napište, jak často konzumujete následující potraviny či nápoje:

| | Pravidelně denně | Několikrát týdně | Několikrát do měsíce | Méně často | Nikdy |
|---|---|---|---|---|---|
| Cukrovinky (dorty, čokoláda, sušenky, bonbóny) | | | | | |
| Slazené nápoje (limonáda, kola, džus) | | | | | |
| Tučné výrobky | | | | | |
| Ovoce a zelenina | | | | | |
| Pivo a víno | | | | | |
| Destiláty | | | | | |

**5. Kuřácká anamnéza**

5.1. ☐ **Pravidelný kuřák** *(Kouří pravidelně minimálně 1 cigaretu denně.)*

Kouřím … let … cigaret denně.

5.2. ☐ **Příležitostný kuřák** *(Kouří méně než 1 cigaretu denně.)*

Kouřím … let … cigaret za týden.

5.3. ☐ **Bývalý kuřák**

Kouřil(a) jsem … let … cigaret denně (… cigaret týdně) minimálně 6 měsíců po sobě jdoucích a nyní již 6 měsíců nekouřím

*V případě, že jste kouřil(a) kratší dobu než 6 měsíců a nyní již nekouříte, zaškrtněte bod 5.4.*

5.4. ☐ **Nekuřák**

Nikdy jsem nekouřil(a) déle než 6 měsíců.

Děkuji Vám za vyplnění dotazníku.

**Výsledek preventivní prohlídky**

**(vyplní lékař)**

Respondent číslo: .....

**Nález na ústní sliznici:** ☐ normální

☐ abnormální (jaký .....)

**Zánětlivé změny na dásních:** ☐ ano ☐ ne

**Stav parodontu podle CPI:**

| 17/16 | 11 | 26/27 |
|---|---|---|
| | | |
| | | |
| 47/46 | 31 | 36/37 |

0 = zdravý parodont
1 = krvácení
2 = zubní kámen
3 = p. chobot 4 – 5 mm
4 = p. chobot nad 5 mm
X = nezjištěno

**APPENDIX II**

**Indian questionnaire**

**Questionnaire for respondents participating in the study about influence of smoking on oral health**

Respondent no:.....

*(Kindly fill in the box □ with appropriate answer.)*

**1. Personal data**

1.1. Sex: □ Male □ Female

1.2. Age: .....

1.3. Educational qualification: 1.3.1. □ No education

1.3.2. □ Basic-Till 5th std.

1.3.3. □ High school-Till 10th std.

1.3.4. □ Graduation

1.4. Income (per month in Indian Rupees):

1.4.1. □ 500-1500 1.4.2. □ 1500-3500 1.4.3. □ 3500-6500

1.4.4. □ 6500-10,000 1.4.5. □ above 10,000

1.4.6. □ Not willing to disclose

1.5. Profession (example: teacher, doctor, house-wife, laborer...): .....

**2. Health status**

2.1. Are you suffering from any chronic diseases? 2.1.1. □ Yes 2.1.2. □ No

2.2. If yes, which?

2.2.1. □ Hypertension 2.2.2. □ Diabetes

2.2.3. □ Astma 2.2.4. □ Chronic lung diseases (T.B., Pneumonia)

2.2.5. □ Urinary tract infection 2.2.6. □ Neurological diseases

2.2.7. □ Gastro-intestinal diseases 2.2.8. □ Hepatic diseases

2.2.9. □ Bone and joint diseases 2.2.10. □ Malignant diseases

2.2.11. □ Any other diseases.....

2.3. Are you on any long term medication? 2.3.1. □ Yes 2.3.2. □ No

2.3.3. □ If Yes, which drug?.....

2.3.4. □ Yes, but I don't know the name of the drug

**3. How often do you visit a dentist?**

3.1. □ 2 times/year 3.2. □ once/year 3.3. □ visit only when I have problem

3.4. □ never before

**4. Brushing habits**

4.1. Frequency of brushing teeth:

4.1.1. □ once daily 4.1.2. □ twice daily 4.1.3. □ 3 times a day

4.1.4. □ never

4.1.5. □ Do you take any food/drinks after brushing in the night? □ Yes □ No

4.2. Dental aids used for oral hygiene:

4.2.1. □ tooth brush and tooth paste 4.2.2. □ tooth brush alone

4.2.3. □ tooth brush and tooth powder 4.2.4. □ dental floss

4.2.5. □ mouth wash 4.2.6. □ motorized tooth brush

4.2.7. □ tooth pick

4.2.8. □ Any other aids (example: neem stick, charkcoal…) ……..

4.4. Details of food and drinking habits:

| | Regularly/ daily | Several times/week | Several times/month | Less frequently | Never |
|---|---|---|---|---|---|
| Sweets (cakes, chocolates, biscuits, ice cream etc) | | | | | |
| Sweet drinks (coke, fruit juices etc) | | | | | |
| Fruits and vegetables | | | | | |
| Alcoholic drinks | | | | | |

**5. Form of tobacco consumption:**

5.1. □ tobacco with betel nut and leaves 5.2. □ tobacco alone

5.3. □ bidi/chutta etc 5.4. □ cigarettes without filters

5.5. □ cigarettes with filters 5.6. □ pipes/other forms

**6. Smoking history**

6.1. □ **Regular smokers** *(Smokes minimally one cigarette per day.)*

I smoke……years……cigarettes per day.

6.2. □ **Occasional smoker** *(Smokes less than one cigarette per day.)*

I smoke……years……cigarettes per week.

6.3. □ **Ex-smoker**

I smoked…..years…..cigarettes per day (…..cigarettes per week)

6.4. □ **Never smoked**

Thank You for filling the questionnaire.

**Clinical data**

(To be filled by dentist)

Respondent no: .....

**Oral mucosal findings:** □ normal

□ abnormal (findings .....)

**Gingivitis:** □ Yes □ No

**Periodontal Index (CPI):**

| 17/16 | 11 | 26/27 |
|---|---|---|
| | | |
| | | |
| 47/46 | 31 | 36/37 |

0 = Healthy periodontium
1 = Bleeding on probing
2 = Calculus present
3 = Pocket 4 – 5 mm
4 = Pocket 6 mm or more
X = Excluded sextant

# 11

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# 12

# ABBREVIATIONS

**AIDS-** Acquired Immunodeficiency Syndrome

**BAT-** British American Tobacco

**BMI-** Body Mass Index

**BoP-** Bleeding on Probing

**CAL-** Clinical Attachment Loss

**CPITN-** Community Periodontal Index of Treatment Needs

**ETS-** Environmental Tobacco Smoke

**FDI-** Federation Dentaire Internationale

**HIV-** Human Immunodeficiency Virus

**IgG-** Immunoglobulin G

**IL-** Interleukin

**MS-** Mainstream Smoke

**NHANES-** National Health And Nutrition Examination Survey

**NRT-** Nicotine Replacement Therapy

**PD-** Periodontal Depth

**PMN-** Polymorphonuclear leukocytes

**SES-** Socioeconomic Status

**SS-** Sidestream Smoke

**TNF-** Tumor Necrosis Factor

**TPM-** Total Particulate Matter

**WHO-** World Health Organization

# 13

# LIST OF AUTHOR'S PUBLICATIONS

## Original articles

1. **Jacob V.**, Vellappally S., Smejkalova J., Rajkumar K., Cermakova E., Fiala Z., Rakesh V. Somanathan, Shriharsha P.: Tobacco use and its effect on periodontal health in India. Cent. Eur. J. Med. (accepted).
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