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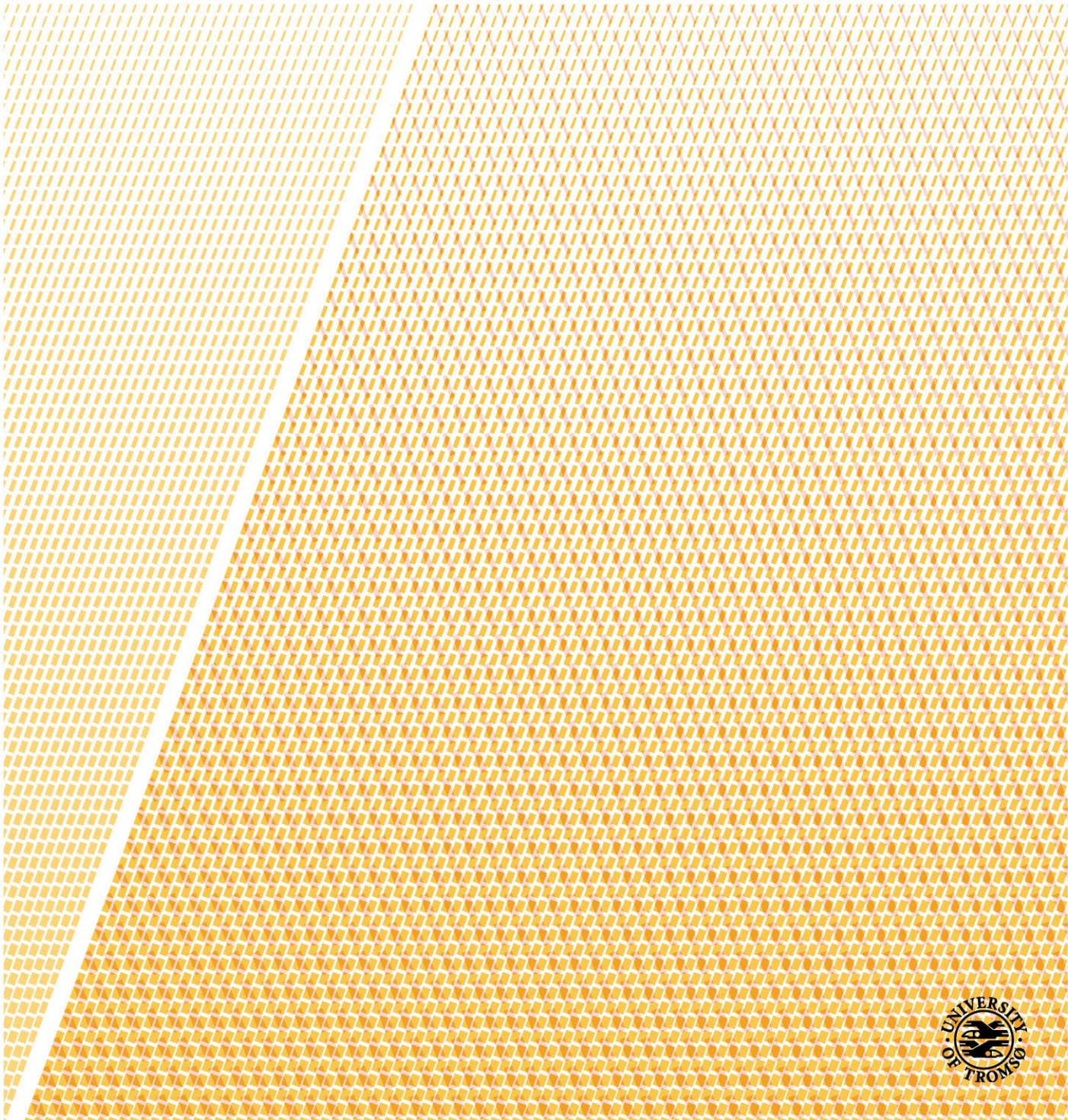
Periodontal health in Troms County, Northern Norway

Descriptive, subject level and site-specific analyses

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To Ingmar

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Abbreviations

| | |
|---------|--|
| AAP | American Academy of Periodontology |
| AC | Alveolar crest |
| ANOVA | Analysis of variance |
| BL | Bone loss |
| BOP | Bleeding on probing |
| CAL | Clinical attachment level |
| CDC | Centers for Disease Control and Prevention |
| CEJ | Cemento-enamel-junction |
| CFA | Confirmatory factor analysis |
| CFI | Comparative fit index |
| CI | Confidence interval |
| CPITN | Community Periodontal Index of Treatment Needs |
| DAS | Dental Anxiety Scale |
| df | Degrees of freedom |
| EFP | European Federation of Periodontology |
| GM | Gingival margin |
| ICC | Intraclass correlation coefficient |
| OHIP | Oral health impact profile |
| OHIP-14 | Short version of oral health impact profile |
| OHRQoL | Oral health-related quality of life |
| OPG | Orthopantomogram |
| PD | Probing depth |
| RBL | Radiographic bone loss |
| REC | Regional Committees for Medical and Health Research Ethics |
| RMSEA | Root mean square error of approximation |
| SD | Standard deviation |
| SE | Standard error |
| SOC | Sense of coherence |
| SRMR | Standardized root mean square residual |
| TLI | Tucker-Lewis index |
| TOHNN | Tromstannen – Oral Health in Northern Norway |

List of papers

This thesis is based on the following three papers, referred to by their corresponding roman numerals in the text.

- I. **Holde, G. E.**, Oscarson, N., Trovik, T. A., Tillberg, A., & Jönsson, B. (2017). Periodontitis Prevalence and Severity in Adults: A Cross-Sectional Study in Norwegian Circumpolar Communities. *Journal of Periodontology*, 88(10), 1012-1022.
- II. **Holde, G. E.**, Baker, S. R., & Jönsson, B. (2018). Periodontitis and quality of life: What is the role of socioeconomic status, sense of coherence, dental service use and oral health practices? An exploratory theory-guided analysis on a Norwegian population. *Journal of Clinical Periodontology*, 45(7), 768-779.
- III. **Holde, G.E.**, Jönsson, B., Oscarson, N., & Müller H.P. To what extent does smoking affect gingival bleeding response to supragingival plaque? Manuscript submitted.

Abstract

Background/Aim: Periodontal disease is one of the most prevalent diseases in the world. It is a common cause of tooth loss and has been related to social, psychological and physical impairment. There is a scarcity of epidemiological studies of periodontal disease in Norway. The overall aim of this thesis was to assess the burden of periodontitis in an adult population, and to explore risk factors, as well as health-promoting factors related to the disease.

Methods: The thesis is based on a cross-sectional study with a target population of adults 20 to 79 years old. Data were collected between October 2013 and November 2014 in Troms County, Northern Norway. Information about sociodemographic characteristics, behavioral factors, oral health-related quality of life, and sense of coherence was collected by self-reported questionnaire. Periodontal conditions were assessed with a full-mouth periodontal examination. Probing depth and bleeding on probing were measured at six sites per tooth. Radiographic bone loss was examined using orthopantomograms.

Results: According to the Centers for Disease Control and Prevention/American Academy of Periodontology case definitions, 50% of participants had periodontitis, of which 9% had severe periodontitis. Periodontitis prevalence increased markedly with age, was higher among men, and positively associated with smoking, lower levels of education, and lower income. Using Andersen's behavioral model of health services use, more social structure and stronger SOC was related to enabling resources, which in turn was associated with more use of dental services. More use of dental services was related to more periodontitis and more periodontitis was associated with increased oral health impacts. Self-perceived treatment need was not associated with use of dental services. Gingival bleeding was affected by local, behavioral and socioeconomic factors. Smoking reduced the general bleeding tendency of the gingiva and attenuated the response to supragingival plaque.

Conclusions: There is a high burden of periodontitis among adults in Troms County. Socioeconomic factors and smoking were main predictors of periodontitis. Smoking strongly affected the clinical expression of gingivitis. There is a complex relationship between population characteristics, use of dental services and oral health outcomes. Regular dental visiting habits did not reduce the likelihood of periodontitis.

1 Introduction

Periodontitis has been reported to affect major parts of populations [1, 2]. There is little information about the prevalence of periodontal disease among adults in Norway, and how it is distributed in the population. In order to plan appropriately for people's periodontal health care needs, information about number of persons with the disease is necessary. By identifying groups with higher risk of periodontitis, strategies aiming to prevent and control the disease can be developed.

Epidemiological studies provide knowledge about the prevalence of diseases and related risk factors, thus making valuable contributions to health care management and the planning and evaluation of preventive strategies.

This thesis aims to describe the periodontal health and disease in an adult population, not only measured by objective assessments of clinical signs and symptoms, but also as experienced by individuals. Examining clinical, behavioral and social factors related to periodontitis, and how these factors relate to each other, can give valuable insight into patterns of oral health-related behavior and its effect on the populations' periodontal status and perceived oral health.

1.1 Periodontitis

Periodontitis is an inflammatory disease affecting the soft and hard tissues surrounding the teeth. Microorganisms in dental plaque initiate the disease, and if it progresses, destruction of the supporting tissues of the teeth (i.e. periodontal ligament and alveolar bone) follows. Clinical features are attachment loss, periodontal pocket formation and reduced density and height of the alveolar bone surrounding the teeth [3]. Untreated, periodontitis can lead to loosening of teeth and potentially tooth loss. The disease is reported to have a negative impact on oral health-related quality of life and patients' daily lives [4-8].

1.1.1 Case definitions

For a long time, numerous case definitions for periodontitis have existed [9, 10]. For example, the International Dental Federation and The World Health Organization developed the Community Periodontal Index of Treatment Needs (CPITN) to indicate levels of periodontal conditions in populations [11]. The index has a score from 0 to 4 and is based on the clinical parameters bleeding on probing (BOP), calculus, and periodontal probing depth (PD). Scores are based on index teeth or the highest score from each sextant of teeth is recorded. The validity of the index has later been criticized [12-14]. Hugoson & Jordan developed a classification used in a series of epidemiological studies in Sweden, grouping individuals into five groups (healthy, gingivitis, and three groups of

periodontitis) based on BOP, PD and radiographic bone loss (RBL) [15]. A periodontal case definition for use in risk factor analysis was proposed by the 5th European Workshop in Periodontology, defining periodontitis as incipient and severe, based on presence and extent of clinical attachment loss (CAL) [16]. Also, the Centers for Disease Control and Prevention and the American Academy of Periodontology (CDC/AAP) developed a case definition for use in population-based surveillance [17, 18]. The case definition defines periodontitis as mild, moderate and severe based on PD and CAL. The CDC/AAP case definition was in 2015 (when the present study was performed) proposed as the standard for reporting chronic periodontitis in epidemiological studies.

1.1.2 Epidemiology of periodontal disease

Estimates of periodontitis have varied across populations. Comparison between different studies and different populations has not been straightforward, mainly because there has not been a universally accepted case definition of periodontitis. Further complicating comparison is the use of different examination protocols, e.g. use of index teeth and partial mouth recordings that are prone to misclassification of disease [19, 20].

The Global Burden of Disease have aimed to consolidate all epidemiological data about severe periodontitis, defined as “a CPITN score of 4, a clinical AL [attachment loss] more than 6 mm, or a gingival PD more than 5 mm” [21]. In 2017, it was estimated that severe periodontitis affected 10% of the global population [22].

In Norway, there have been few epidemiological studies of periodontitis, and none in a general adult population. Periodontal health was described using data from four epidemiological studies on 35-year olds in Oslo, carried out between 1973 and 2003 [23]. Periodontal status was assessed with CPITN and radiographic bone level measurements. The authors reported a decrease in prevalence of deep pockets (≥ 6 mm) from 22% in 1973 to 8% in 2003. The prevalence of RBL also decreased from 54% to 24%, respectively. In a study from 2012, using data from a national sample of old-age pensioners, it was found that periodontitis, defined as presence of $PD \geq 6$ mm, affected 33% of the examined population [24].

Repeated cross-sectional studies were also carried out in Sweden between 1973 and 2003. These studies included participants 20-80 years old, and defined periodontitis according to criteria by Hugoson & Jordan [25]. The prevalence of periodontitis was 39% in 2003, a marked decrease from 50% in 1973. Periodontal health was assessed in a Danish adult population using CPITN, and the

prevalence of PD \geq 6 mm was 6% in 35-44-year-olds and 20% in 65-74-year-olds [26]. Presence of PD \geq 6 mm was also assessed in a Finnish population of adults 30 years and older, where authors reported a prevalence of 21% [27].

There are several studies applying the CDC/AAP case definition. Table 1 lists studies from the last five years. The prevalence of total periodontitis (mild, moderate and severe) ranges from 1 to 81%.

Table 1. Studies reporting prevalence of periodontitis published between 2014 and 2019 using the CDC/AAP case definition.

| Author | Year | Country | Sample (n) | Age (yrs.) | Examination protocol | Periodontitis | |
|--------------------------|------|---------------|------------|------------|----------------------|---------------|------------|
| | | | | | | Total (%) | Severe (%) |
| Munoz-Torres et al. [28] | 2014 | United States | 147 | \geq 70 | FM, 4 sites | 60** | 21 |
| Al-Harathi et al. [29] | 2014 | Oman | 319 | 23-50 | FM, 6 sites | 46 | 2 |
| Marulanda et al. [30] | 2014 | Colombia | 355 | 16-35 | PM, 4 sites | 8* | |
| Petrutju et al. [31] | 2014 | Romania | 623 | 16-35 | FM, 3 sites | 1 | |
| Eke et al. [32] | 2015 | United States | 7,066 | \geq 30 | FM, 6 sites | 46 | 9 |
| Aimetti et al. [33] | 2015 | Italy | 736 | 20-75 | FM, 6 sites | 76 | 35 |
| Schützhold et al. [34] | 2015 | Germany | 3,622 | 20-84 | PM, 4 sites | 49** | 16 |
| | | | 913 | 35-44 | PM, 3 sites | 71** | 17 |
| | | | 755 | 65-74 | PM, 3 sites | 88** | 42 |
| Knight et al. [35] | 2015 | New Zealand | 2,048 | \geq 18 | FM, 3 sites | | 6 |
| Eke et al. [36] | 2016 | United States | 1,511 | \geq 65 | FM, 6 sites | 80 | |
| Wellapuli et al. [37] | 2017 | Sri Lanka | 1,400 | 30-60 | FM, 6 sites | 51 | |
| Kim et al. [38] | 2018 | Korea | 5,078 | \geq 50 | PM, 6 sites | 81 | 25 |
| Eke et al. [2] | 2018 | United States | 10,683 | \geq 30 | FM, 6 sites | 42 | 8 |
| Shariff et al. [39] | 2018 | United States | 907 | \geq 65 | FM, 6 sites | 80 | 23 |
| Bhat et al. [40] | 2018 | India | 873 | 35-54 | FM | 46** | |
| Schuch et al. [41] | 2019 | Brazil | 539 | 31 | FM, 6 sites | 37 | 14 |

FM: full-mouth, PM: partial mouth

*Only moderate periodontitis, **moderate and severe

Most of the variation can probably be attributed to the very different age groups included in the different studies. For general adult populations the prevalence of periodontitis seems to range between 40 and 50%, with the exception of Italian and German populations where prevalence was reported to be more than 70%. Estimates for the older-age populations are closer to 80%, while the lowest estimates are found in populations 35 years old and younger (1-37%).

1.1.3 Etiology and risk factors

Periodontal disease is a multifactorial disease [42]. There are factors that initiate, affect development and influence the clinical expression of the disease. Bacteria have to be present to initiate the disease, but most part of the periodontal destruction is due to the inflammatory and immunological host response [43]. Risk factors can be divided into environmental or behavioral factors (modifiable risk factors) and intrinsic factors or characteristics related to the individual (non-modifiable risk factors) [44].

1.1.3.1 Modifiable risk factors

One major modifiable risk factor is smoking. Smokers have increased susceptibility to periodontitis and greater severity and progression of the disease as compared to non-smokers (reviewed by Nociti et al. [45]). A meta-analysis of data from six studies (n = 2,361) revealed an overall odds ratio (OR) for severe periodontal disease of 2.82 (95% confidence interval [CI] 2.36-3.39) for smokers as compared to non-smokers [10]. Data from a large population-based study in the United States showed that current smokers were 4 times as likely to have periodontitis than never smokers (prevalence OR = 3.97; 95% CI, 3.20-4.93), with a dose-dependent relationship between number of smoked cigarettes and odds of periodontitis [46]. The authors further reported that about 50% of periodontitis cases were attributable to former or current smoking.

Diabetes, or rather hyperglycemia, is considered a modifiable risk factor of periodontitis.

Observational studies have shown that persons with diabetes have increased risk of periodontal destruction [47-49]. A recent meta-analysis of 13 studies showed that diabetes increases the risk of periodontitis onset and progression [50]. A higher level of periodontal destruction has been observed in dental patients with newly identified pre-diabetes and diabetes mellitus [51]. In a cross-sectional study, periodontitis was associated with poorly controlled type 2 diabetes, but not with pre-diabetes and well-controlled diabetes [52].

There is also evidence of an association between overweight/obesity and periodontitis from cross-sectional studies (reviewed by Suvan et al. [53]), with an overall OR of 2.13 (95% CI: 1.40-3.26). Results from a case-control study revealed that obese persons had 3 times higher odds of periodontitis compared to normal weight individuals [54].

Stress, in terms of financial strain, negative life events and psychological factors has been related to periodontitis in cross-sectional studies [55, 56]. It was also reported that coping ability seemed to modify the association. In longitudinal studies, results have shown that patients with passive coping

strategies and patients under psychosocial stress had poorer outcome of non-surgical periodontal treatment [57, 58].

Evidence of the influence of diet on periodontal health is emerging. Better-quality diet, specifically higher consumption of whole grains and fruit and lower consumption of red/processed meats were associated with lower odds of severe periodontitis [59]. Case-control and cohort studies have indicated that vitamin D and calcium may influence periodontal status [60, 61].

1.1.3.2 Non-modifiable risk factors

The genetic component of periodontitis has been examined in twin-studies, where it was found that monozygotic twins were more similar than dizygotic twins regarding clinical periodontal measures [62]. The authors concluded that about half of periodontitis variance is attributed to genetic variance. Recent investigations of the possible influence of genetic variation in host response on periodontitis has focused on immune-regulatory molecules, e.g. cytokines. Polymorphisms in cytokines such as interleukins, surface receptors and cyclooxygenase- and matrix metalloproteinase have been suggested to influence the risk for developing periodontitis [63-65].

Men are almost 2 times more likely to have severe periodontitis than women [66]. Previously, this difference was solely considered a consequence of different lifestyle and health behavior between genders [67]. Recent studies have suggested that sex hormones may play a role. High testosterone levels have been correlated with both prevalence and severity of periodontitis [68]. Results were, however, inconclusive in a review of testosterone levels and periodontitis [69].

Several studies show that the prevalence and severity of periodontal disease increases with age [2, 33, 66, 70, 71], probably due the chronic and cumulative nature of the disease. US adults 65 years and older were two times more likely to have periodontitis than adults 30-44 years old [66]. Mild and moderate periodontitis seem to account for most of the increase in periodontitis prevalence with age, as prevalence of severe periodontitis remained around 10% from the age of 45 years.

A review of socioeconomic status and periodontitis concluded that persons who are socioeconomically disadvantaged consistently have poorer periodontal status [72]. A meta-analysis showed that lower education increased odds of periodontitis almost two times, as compared to higher education [73]. Lower income has been related to higher levels of periodontitis in several cross-sectional studies [66, 74]

Systemic conditions characterized by neutrophil dysfunction (e.g. neutropenia, Papillon-LeFèvre syndrome and Down's syndrome) have been associated with periodontal disease (reviewed by Deas et al. and Khocht et al. [75, 76]).

1.1.4 Dental plaque and gingival inflammatory response

Dental plaque induces gingival inflammation, which is considered a necessary factor in the development of periodontitis [77]. Management of gingival inflammation is therefore essential in periodontal infection control, both as a primary prevention strategy for periodontitis and secondary prevention strategy for recurrent periodontitis [78]. While plaque control is the critical element in reducing gingival inflammation, there are many factors that can modify the gingival inflammatory response to plaque [79]. These factors include pregnancy [80], diabetes [81, 82], Down's syndrome [83, 84], interleukin-1 polymorphism [85], vitamin C and D deficiency [86, 87], anti-microbial and anti-inflammatory agents [88-90] and conditions affecting the immune system (e.g. HIV/AIDS, leukemia) [91, 92]. A recent experimental study of diet and gingivitis showed that an anti-inflammatory diet, especially vitamin D supplement, affected gingivitis [93]. Also, a host-dependent variation in gingivitis susceptibility have been investigated, with studies reporting a subject-specific gingival inflammatory response [94, 95]. Finally, smoking is an important environmental factor affecting gingival bleeding response to plaque. In both experimental and observational studies of gingivitis, it has been reported that smokers had similar or higher levels of dental plaque as compared to non-smokers, but less pronounced gingival inflammation as measured by BOP [96-104]. In these studies, individuals' proportion of sites with BOP and plaque have been correlated, meaning the results may not apply at the site level. The site-specific bleeding response to dental plaque have so far only been assessed in one study, where smoking did not reduce bleeding response to plaque [105].

The biomechanical effects of smoking on gingival inflammatory response remain unclear. There is limited evidence that tobacco smoke promotes gingival vasoconstriction in humans [106-110]. Studies have found a reduced number of gingival vessels or vessels of smaller caliber in smokers as compared to non-smokers [99, 111-113], indicating that smoking suppresses angiogenesis. It is also possible that thermally induced nerve damage in the oral cavity of smokers [114, 115], could have potential effects on the gingival vascular response. Additionally, tobacco smoking can alter the dental plaque composition and its inflammatory potential [116, 117]. Most importantly, cigarette smoking has been reported to affect the immune responses (reviewed by Sopori [118]). For example, a decreased level of pro-inflammatory biomarkers was found in the gingival crevicular

fluid of smokers with periodontitis, suggesting a reduced capacity to recruit inflammatory and immune cells [119].

1.2 Conceptual frameworks

1.2.1 Oral health-related quality of life

It is not only important to measure level of disease in a population, but also the subjective effects of disease on individuals. Oral health-related quality of life (OHRQoL) is a multidimensional concept of people's perspectives of oral health and the possible impact of oral conditions on their everyday functioning and well-being [120]. One of the most common instruments used to assess OHRQoL is the short form of the Oral Health Impact Profile (OHIP-14) [121]. The OHIP focuses on impairment with three functional status dimensions: social, psychological and physical, where all impacts are conceptualized as adverse outcome [122]. Responses are made on a five-point Likert scale, with response categories "never", "hardly ever", "occasionally", "fairly often" and "very often". A Norwegian version of the OHIP-14 has been developed and validated [123, 124].

Epidemiological studies have found that certain groups are more likely to report low OHRQoL. Persons with irregular dental visits and poor health-related behavior were more likely to have lower OHRQoL in a study of Israeli dental patients [125]. A study of Norwegian adults showed that poor self-rated oral health, irregular dental visits, fewer teeth, young age and female gender were associated with having oral problems [123]. High dental anxiety, low income, poor chewing ability and self-reported susceptibility to periodontitis predicted low OHRQoL in a study of Swedish women [126].

Several studies have shown that periodontitis, as clinically assessed, has an impact on OHRQoL. In two cross-sectional studies of UK adults, periodontal disease was associated with more oral health impacts [7, 127]. Similar results were found in two studies of Chinese adults, where both self-reported symptoms of periodontitis and clinically assessed periodontitis was associated with lower OHRQoL [4, 128]. A case-control study of British dental patients, showed that patients with periodontitis reported poorer OHRQoL than age- and gender-matched periodontally healthy patients [129]. Periodontal disease experience, measured by RBL, was also associated with reduced OHRQoL in a Swedish cross-sectional study [6]. Among Sri Lankan adults, severity of oral impacts increased with greater severity of periodontitis, where physical pain was the most common oral impact [5]. In a review, it was concluded that periodontal diseases affected OHRQoL, and that impairment increased with greater severity and extent of disease [130]. A randomized controlled

trial with Swedish periodontal patients showed that non-surgical periodontal treatment improved self-rated oral health [131].

1.2.2 Sense of coherence

Sense of coherence (SOC), a concept developed by Antonovsky in 1979, aims to explain why some individuals stay healthy, even after going through stressful situations in life, while others develop disease [132]. A person's level of SOC is based on "general resistance resources", a term coined by Antonovsky, which comprises characteristics that help a person cope and effectively avoid or deal with psychosocial stress [133]. SOC consists of three components, comprehensibility, manageability and meaningfulness, that together have an impact on health [132]. Comprehensibility is the ability to understand events in life as structured and clear in a cognitive way; manageability is the feeling of managing a situation and awareness of internal and/or external resources; and meaningfulness is the motivational factor, the belief that things in life are worthwhile and a reason to care and participate [133]. According to Antonovsky, these three components have a dynamic relationship and throughout life, people can have different levels of the different components. To measure SOC, Antonovsky developed a questionnaire called "The Life orientation questionnaire" [134]. Originally, the questionnaire contained 29 questions, but a shorter version with 13 questions has also been accepted as an instrument for measuring SOC [133, 135, 136]. The SOC scale has been validated in several Norwegian populations [137-140].

Studies have shown that SOC increases with age, education, income, marital status and social group; also, some studies have reported different SOC scores between genders [141-143]. SOC has been found to be important for adults' oral health in several recent studies including toothbrushing habits, eating fruit and vegetables, dental attendance, and OHRQoL [144-146]. The influence of SOC on oral health status has been assessed in a Swedish population, where a significant association between higher SOC scores and fewer teeth with PD \geq 4 mm was reported [147]. For an adult population in Finland it was reported that SOC was positively associated with adults' oral health, where a strong SOC was related to having more teeth, less caries and a lower extent of periodontal pockets [148]. The relationship between SOC and OHRQoL was explored in a population of middle-aged Swedish women, where low SOC predicted low OHRQoL [126]. In a randomized trial, it was assessed whether OHRQoL could be improved by increasing SOC levels, presenting experimental evidence that OHRQoL can be influenced by SOC [149]. SOC has also been explored as a predictor of pocket formation; however, an association between SOC and change in number of teeth with periodontal pockets was not found [150]. In a cross-sectional study of

Brazilian adults, an association between clinical periodontal health and SOC was not observed, while perceived periodontal disease was associated with a lower SOC [151].

1.2.3 Andersen's behavioral model of health service use

Andersen's behavioral model of health services use was initially developed in the late 1960s [152]. The model aimed to “assist the understanding of why families use health services; to define and measure equitable access to health care; [and] to assist in developing policies to promote equitable access [...]”[153]. According to Andersen (1995), the model originally focused on the family as the unit of analysis but shifted later to the individual as unit of analysis. The initial model (Figure 1) proposed that the individual's use of health services is a function of predisposing characteristics, factors that can enable or impede use and their perceived need for care [153].

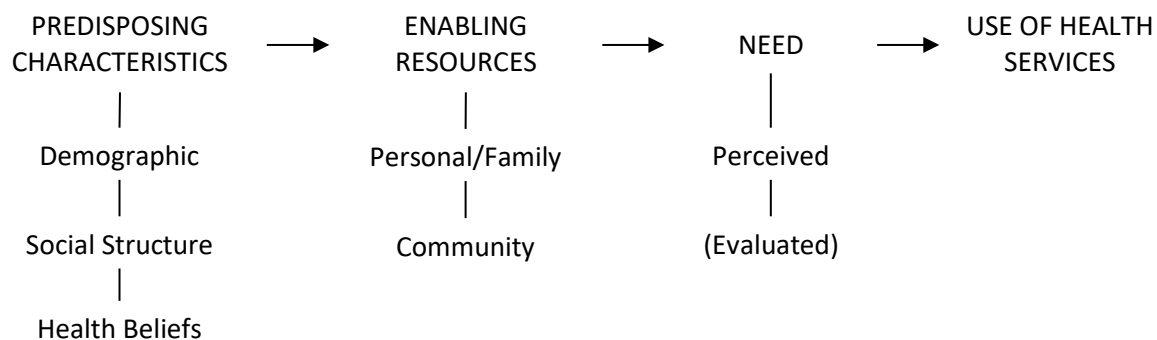


Figure 1. The initial behavioral model (1960s), figure after Andersen (1995)

Predisposing characteristics consist of demographic factors (e.g. age and gender), social structure (e.g. education, income and ethnicity), and health beliefs (e.g. attitudes and knowledge about health and health services) [153]. Andersen (1995) also discussed the possibility of adding genetic factors and psychological characteristics to predisposing characteristics.

Enabling resources represent the availability of health personnel and facilities and persons' means and competence to access and use health services, where economical means, health insurance, and travel and waiting times can be important measures [153].

Perceived need is a measure of how people view their own health and how they experience symptoms of disease, and whether or not they need to seek health care [153]. *Evaluated need* is the professional assessment of health status and need for care. In the model, perceived need is thought to facilitate the understanding of care-seeking, while evaluated need is more related to type of treatment provided [153].

Later, the model also included external environment, health status outcomes and personal health practices [153]. A review of studies from 1998-2011 found that Andersen's behavioral model has been applied in several studies investigating the use of health services [154]. Although the model was frequently used, findings were inconsistent and the authors concluded that there were considerable variations in how the variables were categorized. The model was tested in a Norwegian population in 1983 for physician use, where need was the primary determinant of physician service use [155]. Andersen's behavioral model has been tested in relation to dental care and oral health outcomes in two different general populations in the UK [156, 157], where the results provided support for Andersen's behavioral model, as applied to self-perceived oral health, and found that enabling resources and need were important predictors of use of services and oral health outcomes. The authors concluded that future research should consider incorporating other important factors into the model to increase its explanatory power. Such factors could be cost of treatment, dental anxiety, attitudes and health beliefs, as well as other key factors important for oral health and quality of life. SOC is an example of a factor affecting both oral health and OHRQoL.

1.3 Rational

Periodontal disease is one of the most prevalent diseases in the world [1]. It is a common cause of tooth loss and is related to impairment of social, psychological and physical function [7, 127-130, 158]. Moreover, the disease has been shown to be unequally distributed in the population, more commonly affecting groups with lower socioeconomic status and poorer oral-health related behavior [2, 67, 70].

There is a scarcity of epidemiological studies of periodontal disease in Norway [23, 24]. Northern Norway has a history of low dentist-to-patient ratio, and the lowest scores for self-assessed oral health in the country [159]. By assessing the periodontal status in the population in Troms County the Tromstannen – Oral health in Northern Norway (TOHNN) research project aimed to answer questions about the prevalence, severity and extent of periodontal disease and factors that could predict and/or affect oral health. By gaining knowledge about the prevalence and distribution of periodontal disease, a valuable source of data can be provided for the planning of dental health care services and allocation of resources in the region. By assessing different predictors and risk factors for periodontal disease, groups with higher risk can be identified, and preventive actions can be implemented both at population level and for the individual.

The population in Northern Norway has been reported to have less frequent use of dental services as compared to the rest of the population in Norway [159, 160]. A better understanding of which and how social and behavioral factors influence use of dental health services and its effect on oral health outcomes is valuable for the dental health care system in terms of optimizing use of and enabling access to dental health care. Furthermore, for clinicians, knowledge about factors affecting the clinical expression of periodontal diseases is essential in early diagnosis and treatment, especially in terms of risk assessment.

1.4 Aims

The overall aim of this thesis was to assess the burden of periodontitis in an adult population, and to explore which and how risk factors, as well as health-promoting factors are related to the disease and how it affects oral health-related quality of life.

The specific objectives were:

Paper I

- To describe the prevalence, severity and extent of periodontitis in circumpolar communities in Norway
- To examine differences in socio-demographic and behavioral characteristics of people with periodontitis

Paper II

- To utilize Andersen's behavioral model of health services use as the theoretical framework to explore the direct and indirect relationships between population characteristics, use of dental health care services, individuals' personal oral health practices, and periodontal health and self-reported oral health impacts
- To examine how sense of coherence, when incorporated into the model, was related to adult's oral health and to other key factors determining individual's oral health

Paper III

- To investigate the influence of smoking on the site-specific inflammatory response in the gingiva to supragingival plaque in a general adult population
- To assess local effects of smoking by examining whether smoking affects the association between plaque and gingival inflammation differently in different parts of the dentition

2 Materials and methods

This thesis is based on data from a dental health survey in Troms County, Northern Norway: Tromstannen – Oral Health in Northern Norway (TOHNN).

2.1 Study design and population

The study was a population-based, cross-sectional study with a target population of adults 20 to 79 years old. It included a structured questionnaire and clinical examination. All data were collected between October 2013 and November 2014 in Troms County, Norway (Figure 2). In January 2013, 112,253 people in the selected age range were registered as inhabitants of Troms County. Sample size estimation, based on a hypothesized 10% prevalence of severe periodontitis with a 95% confidence level and margin of error of 1.5%, indicated that we had to examine 1,516 individuals. Assuming a response rate of approximately 50%, the total sample size was estimated to 3,000 individuals. The sample was stratified by county region (Tromsø, Southern Troms and Northern Troms) according to proportion of inhabitants. Using a simple random sampling technique, 1,380 individuals from Tromsø, 1,320 individuals from Southern Troms, and 300 individuals from Northern Troms were selected from the population register by *Statistics Norway*.

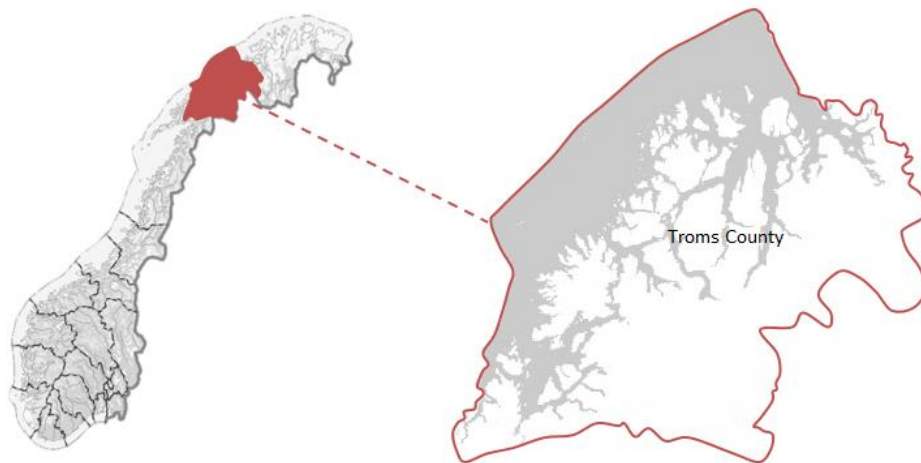


Figure 2. Troms County in Norway © Kartverket www.kartverket.no

2.2 Invitation procedure

Ninety-one of the selected individuals had moved out of the county or died, and were excluded from number of invited individuals. A letter of invitation was sent by mail, including information about the study aim, possible benefits and disadvantages of participation and clinical examination procedures. Potential participants were later contacted by telephone to confirm their decision to

participate or not. If stated, reasons for not participating were noted. Initial non-responders were contacted with an additional letter. Persons that chose to participate received the questionnaire, and forms for written consent and medical history prior to the clinical examination. A total of 1,986 individuals completed both the questionnaire and the clinical examination, resulting in a response rate of 68% (Figure 3).

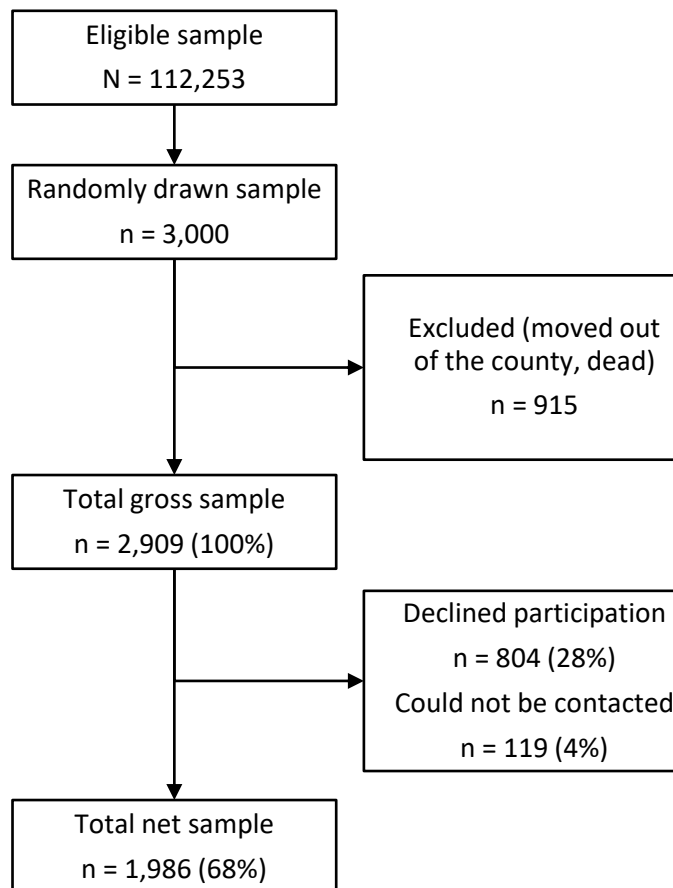


Figure 3. Flowchart of participants.

2.3 Questionnaire

The 16-page questionnaire was developed based on mainly previously used questions from comparable studies. All questions were self-reported by participants. The questionnaire was written in Norwegian and was tested on persons without scientific or dental background in order to identify potential issues and face validity. Briefly described, the questionnaire included questions about background characteristics (e.g. age, gender, ethnicity), socioeconomic factors, use of dental health services, perceived treatment needs, general health, food-frequency, subjective norms, normative beliefs and self-efficacy, sense of coherence (SOC-13), attitudes towards health, OHRQoL (OHIP-14), dental anxiety (Corah's Dental Anxiety Scale), and symptoms of pain (Appendix 1).

2.4 Clinical examination

Clinical examinations were performed in dental offices by 11 dentists with assisting dental nurses, all employed by the Public Dental Health Service in Troms County. All clinical data were registered in a computerized protocol (*Carestream T4*). Periodontal measurements were made in all individuals with natural teeth. PD and BOP was assessed at six sites per tooth (disto-buccal, mid-buccal, mesio-buccal, mesio-palatal/lingual, mid palatal/lingual, and disto-palatal/lingual) for all teeth. PD was measured to the closest millimeter, using a periodontal probe with single millimeter graduations. BOP was registered immediately after periodontal probing and was recorded dichotomously as present/not present. A modification of the Plaque Control Record was applied in order to assess dental plaque at four sites per tooth (distal, buccal, mesial and palatal/lingual) as present or not using a mouth mirror and periodontal probe [161]. No disclosing agent was used.

Radiographic bone level was assessed on orthopantomograms (OPG). Marginal bone levels of both distal and mesial surfaces of all teeth, excluding third molars, were measured linearly with a transparent plastic ruler [162] (see Figure 4). Alveolar bone level was measured in relation to the radiographic apex. The cemento-enamel junction (CEJ), alveolar crest (AC), and radiographically depicted root apex were used as reference points. If the CEJ was destroyed after restorative therapy, the apical margin of the restoration was used as a reference point. The AC was considered the most coronal point at which the periodontal ligament space had a constant width. If the CEJ or AC could not be determined for >20% of teeth, the participant was excluded from analysis. RBL was considered present at sites where the distance from the CEJ to the AC exceeded 2 mm, and was then categorized in 10% intervals from 1 to 10, as described by Skudutyte-Rysstad et al. [23].

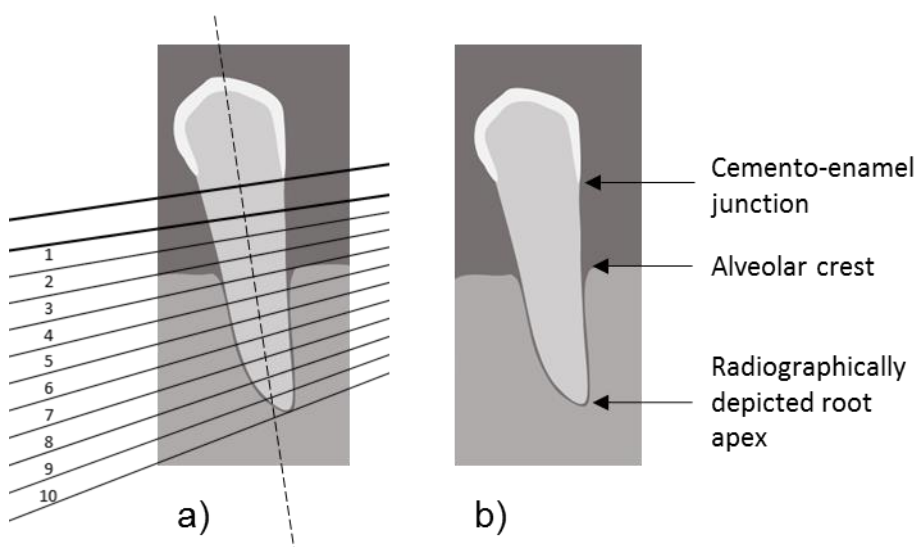


Figure 4. Illustration of bone level measurements. a) Bone level measurements using the ruler by Schei (1959), b) Reference points for bone level measurements.

2.5 Variables

All variables and statistical methods used in *Papers I-III* are listed in Table 2.

Table 2. List of variables and statistical methods used in each of the papers

| | Paper I | Paper II | Paper III |
|-----------------------|--|---|--|
| Outcome variable(s) | Periodontitis Periodontal probing depth Bone loss Bleeding on probing Plaque index | Periodontitis OHRQoL | Bleeding on probing |
| Independent variables | Age Gender Ethnicity Education Household income Demographic status Toothbrushing frequency Frequency of dental visits Smoking status Use of smokeless tobacco | Household income Urbanization Education Sense of coherence Costs of dental services Access to dental services Dental anxiety Treatment need Dental attendance Attendance orientation Toothbrushing frequency Smoking level | Dental plaque Periodontal probing depth Age Gender Education Household income Smoking status Smoking level BMI |
| Statistical methods | Chi-square test, one-way ANOVA, linear regression, z-test | Structural equation modelling, Chi-square, independent samples t-test | Multilevel modelling (three- level logistic regression model), Chi-square, independent samples t-test |

2.5.1 Outcome variables

2.5.1.1 Periodontitis case (Papers I-II)

Periodontitis was defined according to the CDC/AAP case definition (Table 3) [18, 32], which is based on PD and CAL. As CAL was not measured in the current study, the relationship between bone loss (BL) and CAL was explored. CAL and radiographically assessed BL was measured on 786 distal and mesial surfaces in a complementary sample. The ability of BL to predict CAL was modeled and estimated as: $CAL = 2.0 \text{ mm} + 1.3 \text{ BL}$. Using this model, BL was related to CAL, with BL categories 0, 1, 2, and 3 \approx 2, 3, 4-5 and 6 mm of CAL, respectively. Participants were classified with no, non-severe (mild and moderate combined) or severe periodontitis.

Table 3. CDC/AAP case definition for periodontitis

| Mild periodontitis | Moderate periodontitis | Severe periodontitis |
|--|---|--|
| ≥2 interproximal sites with ≥3 mm CAL and ≥2 interproximal sites with ≥4 mm PD (not on the same tooth) or one interproximal site with ≥5mm PD. | ≥2 interproximal sites with ≥4 mm CAL (not on the same tooth) or ≥2 interproximal sites with PD ≥5 mm (not on the same tooth) | ≥2 interproximal sites with ≥6 mm CAL (not on the same tooth) and ≥1 interproximal site(s) with ≥5 mm PD |

2.5.1.2 Periodontal measures (Paper I, III)

In *Paper I*, subjects' mean PD and BL were presented, as well as prevalence and extent of threshold values PD 4 and 6 mm, and BL categories 1 and 2. Mean percent BOP and mean percent plaque were reported. BOP was used as a binary response variable in *Paper III*. The six-sites measurements of BOP were collapsed to four assessments by using the maximum BOP of the two distal (disto-buccal, disto-palatal/lingual) and mesial (mesio-buccal, mesio-palatal/lingual) sites, respectively.

2.5.1.3 Person-reported outcomes (Paper II)

OHRQoL was assessed with the Norwegian version of the OHIP-14 [121, 123]. Response options on a five-point Likert scale were scored from 1 to 5, where “never” was coded as 1 and “very often” as 5. The higher the score, the greater oral health impacts were experienced. Cronbach's alpha for OHIP-14 was 0.89 [163]. Physical function was represented by responses to items 1–5 and 10; psychological function was represented by items 6–9; and social function was represented by items 11–14. Cronbach's alpha was 0.73 for physical function, 0.89 for psychological function, and 0.88 for social function, respectively.

2.5.2 Independent variables

2.5.2.1 Age and gender (Paper I, III)

Age was stratified in categories 20-34, 35-44, 45-54, 55-64, and 65-79 years in *Paper I*, while in *Paper III* age was categorized in four age groups, 20-34, 35-44, 45-69, and 60-79 years, and used as a continuous variable in some analyses. Gender was categorized as male or female.

2.5.2.2 Ethnicity (Paper I)

Ethnic background was defined by the question: “What is your ethnic background?” The three response options were Norwegian, Sámi, and other.

2.5.2.3 Socio-economic factors (Papers I-III)

Education was measured with the question: “What is your highest completed degree of education?” Response options were 1) less than high school, 2) high school, and 3) university level. Annual household gross income was reported in seven categories. In *Paper I* and *III*, income was collapsed into in three categories (low: $\leq 450,000$ NOK, intermediate: 451-900,000 NOK, and high: $>900,000$ NOK) according to national tertiles of household income in 2013 [164]. In *Paper II*, income was analyzed in four categories: $\leq 300,000$ NOK, 301– 450,000 NOK, 451–900,000 NOK, and $>900,000$ NOK.

2.5.2.4 Demographic status/urbanization (Papers I-II)

Demographic status was based on number of inhabitants and availability of dentists as a ratio of inhabitants per dentist. The municipality with the larger town (Tromsø) had the highest availability and was categorized as urban, two municipalities (Harstad and Lenvik) with smaller towns had the second highest availability and were categorized as suburban, and the remaining municipalities without towns had the lowest availability and were classified as rural.

2.5.2.5 Oral health-related behavior (Papers I-II)

Toothbrushing frequency was reported in six categories, from brushing less than once per week to two or more times per day. Toothbrushing was analyzed in three categories: less than daily, once per day, and twice or more per day.

Frequency of dental visits were reported in five categories: only when having problems, longer intervals than two years, every second year, every year, and more than once per year. For analysis, the two most frequent categories were combined to “once per year or more often”. Attendance orientation (*Paper II*) was measured with the question: “When do you use dental services?” The response options were seldom/never attend dental services, only when having pain or lost fillings, and having routine dental check-ups.

2.5.2.6 Sense of coherence

A Norwegian version of the 13-item SOC scale was used [133, 139]. Each question had 1-7 points, with a total score ranging from 13-91, where a high score indicates a strong SOC.

2.5.2.7 Resources related to dental services (Paper II)

Costs of dental services were assessed with the question: “Have you during the last two years refrained from dental services because you did not have enough money?” Response options were

yes or no. Access to dental services was assessed with the question: “Is it difficult for you to get routine dental health care?” Response options were yes, no, or I don’t know.

Dental anxiety was measured with the Norwegian version of Corah’s Dental Anxiety Scale (DAS) [165, 166]. For analysis the scores were reversed so higher scores represented less dental anxiety.

2.5.2.8 Treatment need (Paper II)

Treatment need was measured by the question: “If you had a dental appointment tomorrow do you think you would need dental treatment?” Response options were yes, no, or I don’t know.

2.5.2.9 Use of tobacco (Papers I-III)

Tobacco smoking was assessed with three questions: 1) Do you smoke on a daily basis? 2) How many cigarettes do you smoke each day? 3) For how many years have you been smoking? Number of years of past smoking was also registered. Use of Swedish type, low-nitrosamine, smokeless tobacco (snus) was assessed with the same questions. In *Papers II-III*, smoking level was defined by pack-years, categorized as non-smoker (no pack-years), light smoker (<20 pack- years) and heavy smoker (≥ 20 pack- years). One pack-year is defined as 20 cigarettes smoked per day for one year. Number of pack-years was calculated as (number of cigarettes per day/20) \times number of years smoked.

2.5.2.10 Body mass index (Paper III)

Height (m) and weight (kg) were measured at time of examination and body mass index (BMI, kg/m^2) was calculated. Persons were categorized as normal weight (<25 kg/m^2), overweight (25-29.9 kg/m^2), and obese (≥ 30 kg/m^2).

2.5.2.11 Periodontal measurements (Paper III)

Dental plaque was used as a binary response variable. PD was used as a continuous variable, centered on the mean value. PD measurements from six sites were collapsed to four assessments by using the maximum PD of the two distal (disto-buccal, disto-palatal/lingual) and mesial (mesio-buccal, mesio-palatal/lingual) sites, respectively.

2.6 Examiner reliability

Examiners were trained under supervision of a periodontist prior to data collection to reduce measurement bias. Inter-examiner agreement in PD measurements between the ten examiners and the periodontist, was assessed at site level. Congruency was compared to the nearest millimeter. The median ICC of agreement was 0.81 (range: 0.43 to 0.94). One examiner (GEH) performed all

measurements of RBL on OPGs. Test–retest agreement of site level measurements was assessed on two occasions with two sets of duplicate examinations of ten OPGs. In the first case, examinations performed at the beginning of the examination period were re-examined after three months, with an ICC of 0.78. In the second case, a second set of OPGs examined at the end of the examination period was re-examined after one week, with an ICC of 0.88.

2.7 Data analysis and statistical methods

2.7.1 Paper I

Data were analyzed using the IBM® SPSS® Statistics, version 24. Demographic and socioeconomic characteristics were presented as means (standard deviation [SD]) or numbers (proportions) for the total study population stratified by age. Differences in background characteristics between age groups were assessed with Pearson χ^2 test or one-way analysis of variance (ANOVA). Linear trend across age groups was tested with linear regression. Differences between groups were assessed with *z*-tests. Significance level was set at 0.05. A Lorenz curve was created with Microsoft Excel® and used to describe the distribution of PD \geq 4 mm in the population [167], where the cumulative proportion of the total population is plotted against the cumulative proportion of PD \geq 4 mm. A straight diagonal line would depict perfect equality, where every person would have the same number of PD \geq 4 mm. The extent to which the curve sags below the straight diagonal line indicates the degree of inequality of distribution. The Gini coefficient represents the area between the line of equality (diagonal) and the Lorenz curve, calculated using the Riemann sum estimate (middle sum). The higher the Gini coefficient, the more unequal the distribution is.

2.7.2 Paper II

Data were analyzed using the IBM® SPSS® Statistics, version 24 and AMOS 24. Variables were chosen according to Andersen's behavioral model of health services use [153]. Five latent variables, constructed from measured (indicator) variables were included: Social structure, SOC, enabling resources, use of dental services and oral impacts (Figure 5). The three measured variables for social structures were education, urbanization, and annual household income. SOC was represented by the three SOC components comprehensibility, manageability and meaningfulness. Enabling resources were measured with three measured variables: costs of dental services, access to dental services, and dental anxiety. Use of dental services was represented by the two measured variables frequency of dental attendance and attendance orientation. OHIP-14 was entered into the model as a latent variable with the three domains physical, psychological and social function as indicator variables.

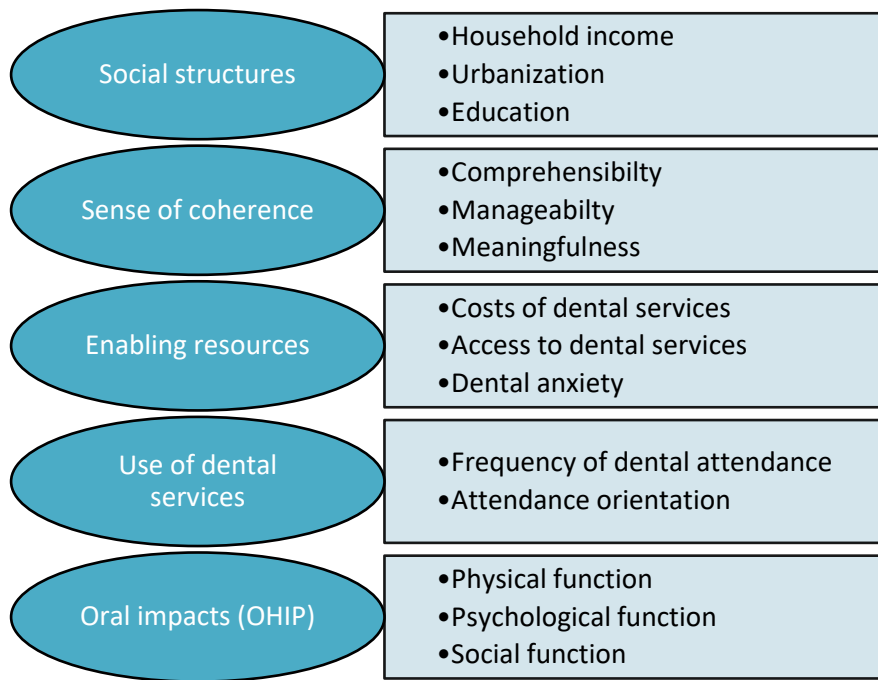


Figure 5. Latent variables with their respective indicator variables.

Structural equation modelling is based on two models: the measurement model and the structural model. The measurement model describes how indicator variables measure the latent variables (e.g. how income measures social structures), and is assessed with confirmatory factor analysis (CFA) [168]. The structural model describes the “causal” connections between the variables. Thus, the first step of the analysis was to test a first order CFA with social structures, SOC, enabling resources, use of dental services and OHIP-14 as the five latent constructs. Indicator variables were not allowed to load on more than one latent variable, nor were error terms allowed to correlate, with the exception of the three domains of the SOC construct.

In the second step of analysis, the structural model was tested, examining the direct and indirect relationships between the constructs as hypothesized in our revised Andersen’s behavioral model of health services use. In accordance with the model and with SOC as an additional population characteristic based on previous findings [145], 24 direct pathways were hypothesized (Figure 6).

AMOS estimates the total effects, which are made up of both direct effects (a path directly from one variable to another) and indirect effects (a path mediated through other variables). The indirect paths can comprise of several indirect effects. While some paths can be mediated through one variable, others can be mediated through several variables. Because of the presence of both non-normal and categorical data, the model was estimated using bootstrapping wherein multiple samples (n = 900+) are randomly drawn from the original sample. The CFA model is then estimated in each

dataset, and the results averaged. The maximum likelihood (ML) bootstrap estimates and standard errors (together with bias-corrected 95% CIs) are then compared with the results from the original sample to examine stability of parameters and test statistics [169]. As recommended, model fit was evaluated using a range of indices from three fit classes; absolute, parsimony adjusted and comparative [169, 170]. A χ^2/df ratio of <3.0 , RMSEA values <0.06 , CFI and TLI ≥ 0.9 , and an SRMR <0.08 were taken to indicate an acceptable model fit [170]. Results are presented as bootstrapped standardized regression weights (β).

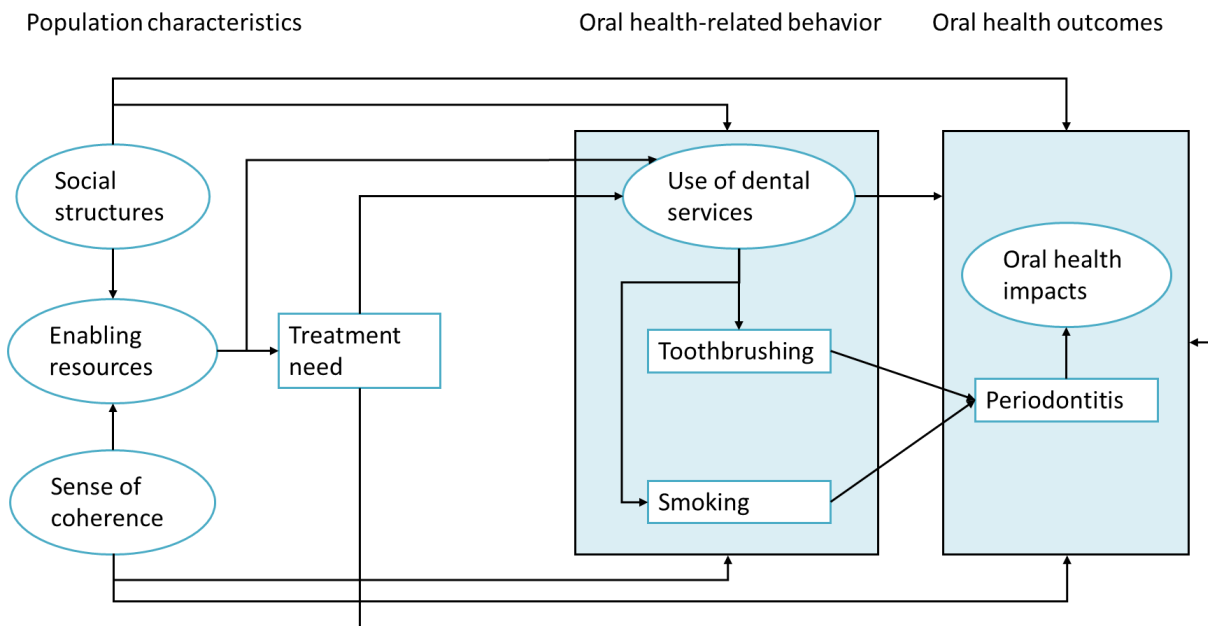


Figure 6. The 24 hypothesized pathways in the revised Andersen's model of health services use. Latent variables are in ellipses and indicator variables are in rectangles. Arrows represent hypothesized pathways. Arrows to boxes represents hypothesized pathways to each of the variables in the box.

2.7.3 Paper III

Data were analyzed using special software (*MLwiN* Version 3.02. Centre for Multilevel Modelling, University of Bristol). Descriptive data were analyzed using the IBM® SPSS® Statistics, version 25, and presented as means with SD or numbers with proportions in parentheses. Three-level (subject, tooth, and site), random intercept, logistic regression models were built (Figure 7), with BOP as the outcome. Plaque, PD, smoking status (non-smoker and smoker), age group, gender, education, income, BMI, and tooth type were entered as covariates. In order to assess how much smoking status modifies the association between plaque and BOP, interaction terms of “plaque \times smoking status” were included as well. Bleeding tendency was also assessed at different tooth types, i.e. upper anterior, lower anterior, upper posterior, and lower posterior teeth. In further analyses, the association between plaque and BOP was assessed in young adults (20-34 years old) and middle-aged adults (45-69 years old). Results are reported as regression coefficients, ORs and

respective 95% CIs. If considered necessary, p -values were derived from Wald tests. However, any inferential statistics (p -values, CIs) were intended to be exploratory, not confirmatory. No correction for multiple testing was done. P -values < 0.05 were considered as statistically noticeable.

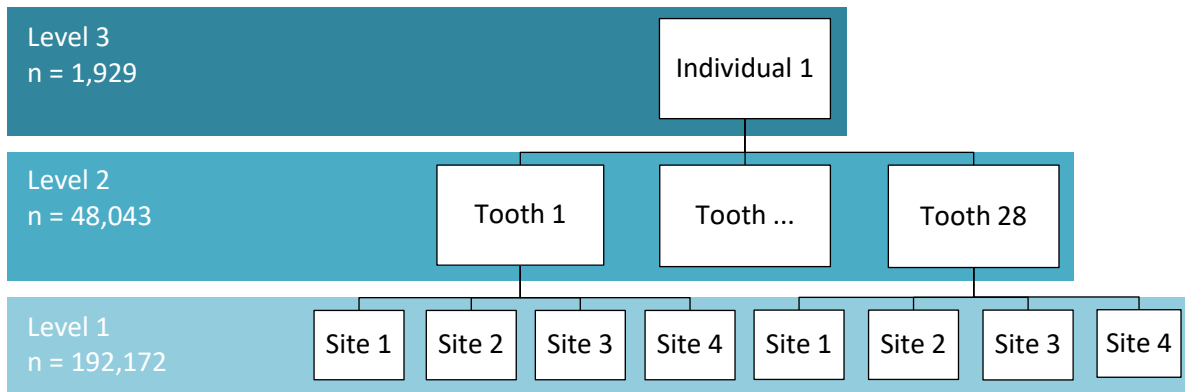


Figure 7. Hierarchical structure of the data.

2.7.4 Missing data

An overview of missing data is presented in Figure 8.

In *Paper I*, edentulous participants ($n = 51$), two participants with only one tooth, and 22 participants with incomplete periodontal records were excluded. The edentulous participants had a higher mean age (69.1 ± 8.4 years), lower level of education and a noticeable high proportion of smokers (32%) as compared to included participants (15%). Participants excluded because of few teeth or incomplete periodontal records were also older (mean age 58.3 ± 16.2 years) than the included participants, and had lower level of education, but had a lower proportion of smokers (7%).

In *Paper II*, missing data occurred at very low frequency (0–3.9%) except for one item in the OHIP-14 instrument (5.8%). An analysis of missing data pattern, computed by SPSS, showed that the missing values appeared to be missing at random. For all one-item variables, missing values were replaced with the median. When calculating SOC scores, individuals with more than three missing items were excluded from analysis. If three or fewer items were missing, they were replaced by the median value of the remaining SOC items for that individual [150]. For OHIP summary scores, individuals with more than two missing OHIP-items were excluded from analysis. When two or less items were missing, they were replaced with the sample median of the relevant OHIP-item [171]. Individuals with more than one missing item in the DAS-scale were excluded from analysis. When one item was missing, it was replaced with the median value of the remaining DAS items for that

individual. Re-analysis of data excluding individuals with any missing items did not change mean scores by more than one decimal place or frequency distributions by more than one percentage point, except for income that changed 2.4 percentage points (not reported). The excluded individuals did not differ noticeably in any of the key outcomes compared to those that were kept in the analysis (periodontitis: $\chi^2_{(2)} = 1.01, p = 0.605$; OHIP: $t_{(1892)} = 0.98, p = 0.328$).

In *Paper III*, edentulous participants ($n = 51$) and six participants with missing periodontal measures were excluded. Further, 168 participants were excluded because of missing values in education, income, smoking status and BMI. Excluded participants did not differ noticeably from included participants regarding main outcome variables. Mean percent BOP for excluded participants was 39.5%, and mean percent plaque was 46.9%, compared to 36.9% and 44.4%, respectively, for included participants (BOP: $t_{(1927)} = -1.48, p = 0.141$; plaque: $t_{(1927)} = -1.39, p = 0.165$).

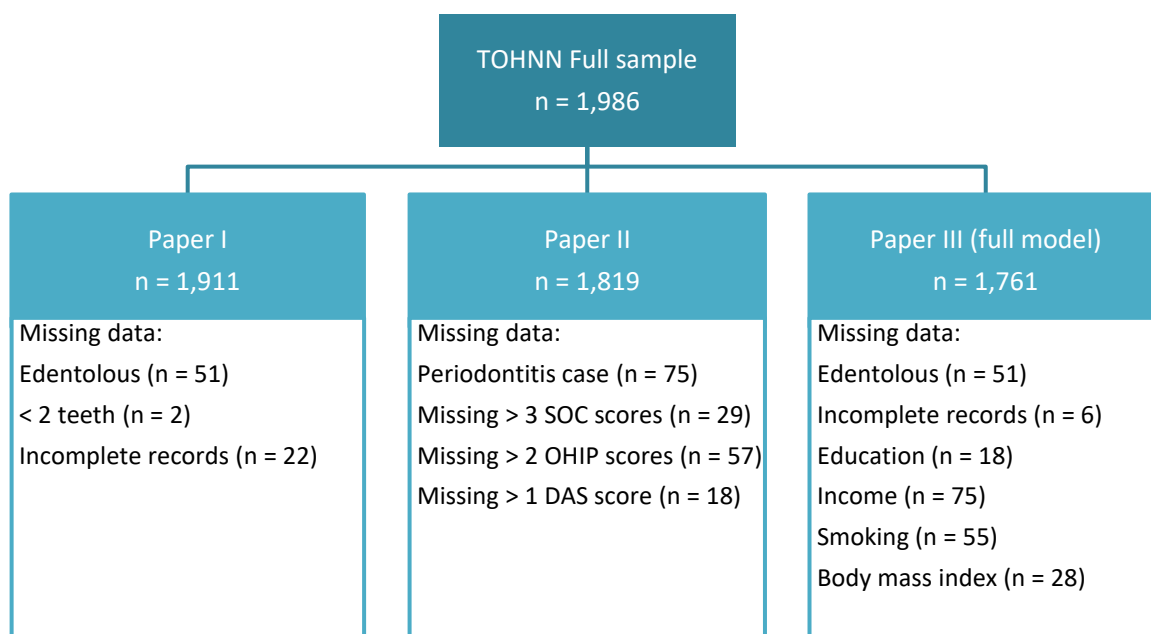


Figure 8. Overview of missing data in Papers I-III.

2.8 Ethics

The study was conducted in accordance with the Declaration of Helsinki. All invited persons received written information about the purpose of the study. They were informed that participation was voluntary and that they could withdraw participation at any time. The study was approved by the Regional Committees for Medical and Health Research Ethics North (22013/348/REC North), Norway (Appendix 2). All participants provided oral and written informed consent.

3 Summary of results

3.1 Paper I

The specific aim was to describe the prevalence, severity and extent of periodontitis in circumpolar communities in Norway, and to examine differences in socio-demographic and behavioral characteristics of people with periodontitis.

According to the CDC/AAP case definition, 50% of participants had periodontitis, of which 9% had severe periodontitis. Around 70% of all participants had at least one site with BL or PD ≥ 4 mm. BL was on average present in 27% of teeth, while PD ≥ 4 mm was found in 18% of teeth. The extent of BL and PD ≥ 4 mm also increased with age, but considerably more so for BL. The burden of PD ≥ 4 mm was unequally distributed in the population. The majority of deep PD was found in a minor part of the population. In the age group 20-34 years old, 94% of all PD ≥ 4 mm was found in 20% of participants. Disparities in the distribution of PD ≥ 4 mm decreased somewhat with older age. In the age group 65-79 years, 20% of the participants accounted for 80% of the total burden of PD ≥ 4 mm.

Periodontitis prevalence and severity increased markedly with age, from 16% in 20-34-year-olds to 81% in 65-79-year-olds. Periodontitis was more prevalent among men (57%) than among women (43%). When comparing prevalence of periodontitis in urban and rural municipalities, there was a higher prevalence in suburban and rural municipalities than in urban areas. In addition, prevalence increased with lower education and income. Smokers had a high prevalence of periodontitis (70%) as compared to non-smokers (45%).

3.2 Paper II

The specific aim was to utilize Andersen's behavioral model of health services use as the theoretical framework to explore the direct and indirect relationships between population characteristics, use of dental health care services, individuals' personal oral health practices, and periodontal health and self-reported oral health impacts.

Andersen's behavioral model explained a large part of the variance in use of dental services (58%) and oral health-related impacts (55%), and to a less extent the variance in periodontitis (19%).

Within this model, 18 direct paths and 12 indirect paths were statistically noticeable (Figure 9).

- More social structures ($\beta = 0.17$) and stronger SOC ($\beta = 0.72$) was related to more enabling resources. Enabling resources was, in turn, associated with a lower perceived treatment need ($\beta = -0.53$) and more use of dental services ($\beta = 0.99$).

- Higher self-perceived treatment need was related to more severe periodontitis ($\beta = 0.07$).
- More use of dental services was related to more frequent toothbrushing ($\beta = 0.12$) and more periodontitis ($\beta = 0.07$).
- More severe periodontitis was associated with increased oral health impacts ($\beta = 0.17$).
- A stronger SOC was associated with less oral health impacts ($\beta = -0.73$).
- There was no direct association between use of dental services and oral health impacts, only indirectly, mediated by periodontitis ($\beta = 0.01$).
- Social structures were only related to use of dental services, when mediated through enabling resources ($\beta = 0.18$).
- Self-perceived treatment need was not related to use of dental services.

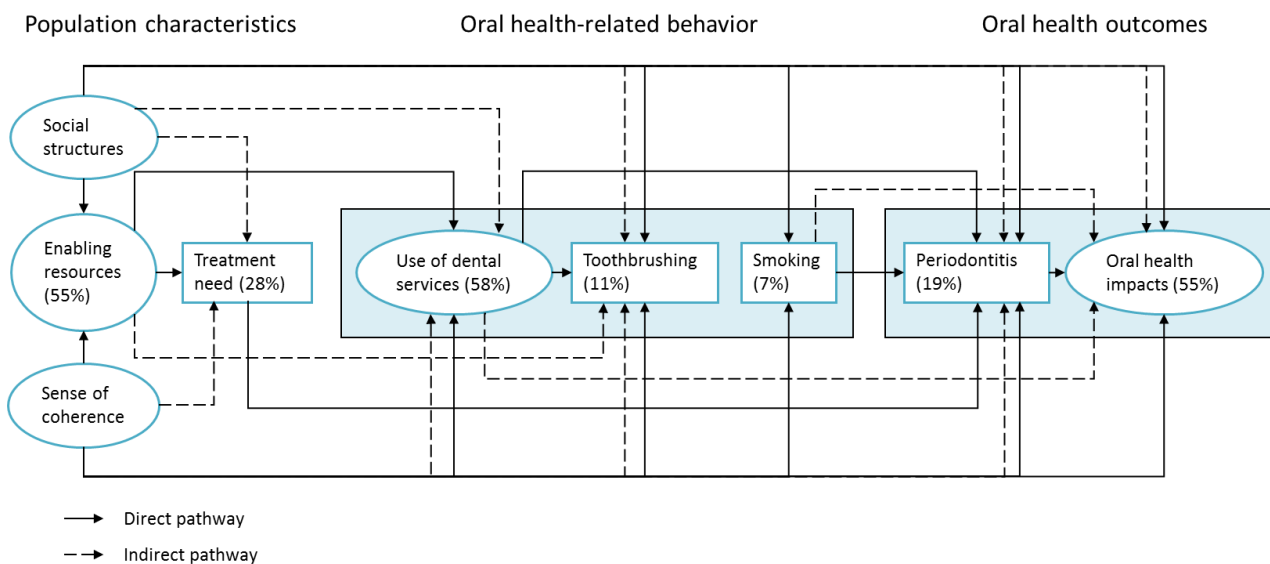


Figure 9. Statistically noticeable pathways in the revised Andersen's model of health services use. Solid lines represent direct pathways and dashed lines represent indirect pathways. (n%) = percentage of variance explained.

3.3 Paper III

The specific aim was to investigate the influence of smoking on the site-specific inflammatory response in the gingiva to supragingival plaque in a general adult population, and to assess local effects of smoking by examining whether smoking affects the association between plaque and gingival inflammation differently in different parts of the dentition.

When plaque was present at a site, the odds of BOP increased twofold. With every millimeter increase in PD, the odds for BOP increased almost threefold. Smoking reduced the odds of bleeding by 26%. The interaction term plaque by smoking also influenced the odds of BOP. Older age and

higher level of education both reduced the odds of bleeding, while overweight and obese persons had increased odds of BOP. Lower anterior teeth, and upper posterior to a less extent, were more likely to bleed upon probing as compared to upper anterior teeth.

Smoking reduced the bleeding tendency of the gingiva for both plaque-covered and plaque-free sites. In smokers, the OR of bleeding at plaque-free sites was 0.77 (95% CI: 0.68-0.88) as compared to non-smokers (OR: 1; ref., $p < 0.001$). While the odds of BOP at plaque-covered sites in non-smokers was increased twofold (OR: 2.12; 2.06-2.18), bleeding tendency was only slightly increased in plaque-covered sites in smokers (OR: 1.46; 1.28-1.66, $p < 0.001$). Heavy smoking (≥ 20 pack-years) further attenuated the association (Figure 10).

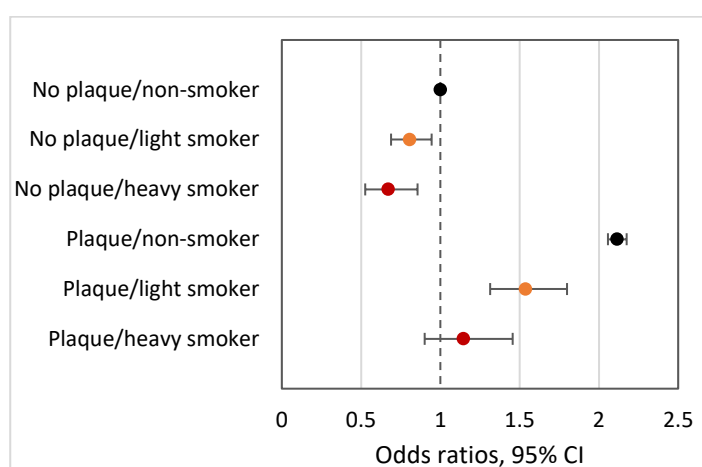


Figure 10. Odds ratios and 95% CIs for BOP in non-smokers and light and heavy smokers as derived from 3-level random intercept model of bleeding on probing adjusted for pocket depth and gender, age, education, income, and body mass index.

When restricting the data to younger adults (20-34 years old), smoking had only a slight effect on the association between plaque and BOP. For plaque-free and plaque-covered sites differences in ORs were not statistically noticeable ($p = 0.221$ and $p = 0.235$, respectively).

While general bleeding tendency differed somewhat in different parts of the dentition, the effect of smoking on gingival bleeding response to plaque did not differ across tooth types. The odds of BOP were reduced in all parts of the dentition, upper and lower anterior and posterior teeth, as compared to non-smokers ($\chi^2_{(4)} = 32.04$, $p < 0.001$), with no noticeable difference between tooth types.

4 Discussion

4.1 General discussion of results

4.1.1 Prevalence, severity and extent of periodontitis

Results from *Paper I* suggest that half of the adults in the target population had periodontitis, with 9% severe periodontitis. Severity and extent of the disease increased with age, and prevalence was highest among people with lower education and current smoking habit. This was in accordance with previous studies [2, 32, 66].

The prevalence of deeper PD (≥ 6 mm) was, for a comparable age group, in range with results from a 35-year old population in Oslo [23]. RBL was, on the other hand, twice as prevalent in the current study as compared to the Oslo-study. In a nationally representative sample of Norwegian elderly pensioners (≥ 67 years), the reported prevalence of deeper PD (≥ 6 mm) was consistent with results from *Paper I* for the same age group [24].

When comparing results with other studies applying the CDC/AAP case definition, estimates were quite similar to those reported for United States adults [2] (see Table 1). European studies have reported considerably higher estimates of periodontitis [33, 34, 70]. About three out of four adults (20-75 years old) in Northern Italy had moderate or severe periodontitis [33]. For a national sample of German adults (35-44 years old) prevalence of moderate or severe periodontitis was two times higher than estimates for the same age group in the current study [70]. Total estimates of periodontitis were not that different between German and Norwegian seniors (≥ 65 years), although severe periodontitis was twice as prevalent among German seniors. Discrepancies between studies could partially be explained by differences in the underlying characteristics of the study populations. For example, smoking was more prevalent in the Italian and German populations (24% and 35% respectively) [33, 70], as compared to the current study (15%). Different levels of education could also have influenced the estimates of periodontitis.

For a Swedish population, total periodontitis prevalence was somewhat lower as compared to estimates from *Paper I*, while prevalence of severe periodontitis was about the same [25]. Periodontitis cases were, however defined according to Hugoson & Jordan [15], where criteria for number of affected teeth are stricter compared to the CDC/AAP case definition. Comparing results with studies using other case definitions of periodontitis is not straightforward, as different definitions can produce very different estimates of periodontitis [172]. For periodontal parameters,

prevalence of PD \geq 6 mm was comparable with results from Finland [27]. In a Danish population, prevalence of PD \geq 6 mm was considerably lower for corresponding age groups [26].

After the World Workshop on the Classification of Periodontal and Peri-implant Diseases and Conditions in 2017, a new classification was presented, including a new case definition of periodontitis [173]. The new periodontitis case definition is based on staging and grading, where stages I-IV describes severity and complexity based mainly on CAL/RBL and PD, local or general extent refer to proportion of teeth affected, while grades A-C indicate progression rates slow, moderate and rapid [174]. When defining periodontitis according to the new case definition by the AAP/EFP [174-176] using RBL and PD, prevalence of periodontitis was 48%, with 21% stage III-IV (severe) periodontitis (Table 4).

Table 4. Prevalence of periodontitis as defined by the AAP/EFP case definition.

| Non periodontitis case | | | | |
|-------------------------------|----------------|-------------------|---------------------|--------------|
| | Healthy | Gingivitis | RP* | Total |
| | n (%) | n (%) | n (%) | n (%) |
| Total | 73 (3.8) | 655 (34.3) | 265 (13.9) | 993 (52.0) |
| Periodontitis case | | | | |
| | Stage I | Stage II | Stage III-IV | Total |
| | n (%) | n (%) | n (%) | n (%) |
| Total | 154 (8.1) | 366 (19.2) | 398 (20.8) | 918 (48.0) |
| Age group (yrs.) | | | | |
| 20-34 | 15 (3.3) | 12 (2.6) | 13 (2.8) | 40 (8.7) |
| 35-44 | 31 (8.1) | 62 (16.1) | 40 (10.4) | 133 (34.5) |
| 45-54 | 56 (14.3) | 93 (23.7) | 74 (18.9) | 223 (56.9) |
| 55-64 | 35 (9.4) | 107 (28.7) | 141 (37.8) | 283 (75.9) |
| 65-79 | 17 (5.7) | 92 (30.7) | 130 (43.3) | 296 (79.7) |
| Gender | | | | |
| Female | 88 (9.0) | 171 (17.5) | 151 (15.5) | 410 (42.1) |
| Male | 66 (7.1) | 195 (20.8) | 257 (26.4) | 452 (48.3) |

RP: reduced periodontium; PD: periodontal probing depth

* PD < 4mm

A comparison of the two case definitions shows that half of the mild cases were defined as stage II, while two fifths of moderate cases were defined as stage III-IV (Table 5). The difference can be explained by different criteria for PD, which is 5 mm or more in the CDC/AAP definition of severe, and 4 mm and more in the definition of stage III-IV by AAP/EFP. A few mild and some moderate cases were defined as non-periodontitis cases (healthy/gingival inflammation on a reduced

periodontium). An advantage of the new case definition is perhaps an increased ability to distinguish between “active” periodontal cases and persons with a history of periodontitis (periodontal disease stability) or persons with periodontal disease remission/control [176]. A distinction between periodontally active and stable cases might affect associations with different exposure variables. Despite some differences between the two case definitions, the weighted kappa for agreement between the two case definitions was 0.78 (95% CI: 0.76-0.80), indicating good agreement.

Table 5. Cross-tabulation of the CDC/AAP and AAP/EFP case definitions of periodontitis.

| | NP case | Stage I | Stage II | Stage III-IV | Total |
|----------|---------|---------|----------|--------------|-------|
| NP case | 864 | 63 | 33 | 6 | 966 |
| Mild | 16 | 91 | 131 | 18 | 256 |
| Moderate | 113 | 0 | 192 | 211 | 516 |
| Severe | 0 | 0 | 10 | 163 | 173 |
| Total | 993 | 154 | 366 | 398 | 1,911 |

NP: non-periodontitis case. Weighted kappa: 0.78 (0.76-0.80)

4.1.2 Use of dental services and oral health outcomes

According to Andersen’s model of health services use, enabling resources and perceived need for care would predict use of health services [153]. As presented in *Paper II*, enabling resources was associated with more use of dental services, while self-reported treatment need was not. Findings from two British populations applying Andersen’s model, reported need as the main predictor of oral health behavior [156, 157]. A study of dental attendance among adult Finns also found perceived need for care to be a predictor of use of dental services in logistic regression analysis [177]. In Norway, there has been a tradition of regular dental visits, independent of oral symptoms or pain [178], which might differ from UK and Finnish populations. Also, Andersen did hypothesize that enabling resources and need would have different abilities to predict use of health care, depending on what type of service that was examined [152]. For example, use of health services related to serious health problems and conditions would mostly be explained by need and demographic characteristics. Use of dental services can be considered more elective, thus, explained to a greater extent by social structures, health beliefs and enabling resources [153]. For the current study population, enabling resources was the main predictor of use of dental services, both as a direct effect and as a mediator for predisposing characteristics. Social structures (education, income and urbanization) were not directly associated with use of dental services, only when mediated via enabling resources. While prior research has shown that socioeconomic factors are important determinants of dental service utilization [179-181], results from *Paper II* indicate that higher level

of education, income and urbanization in itself does not increase people's likelihood of regular dental visits. Use of dental services is only affected when people perceived that they have the necessary resources to attend dental services.

Paper I and *II* demonstrated that socioeconomic and behavioral factors are associated with periodontitis. According to results from *Paper II*, social structures (education, income and urbanization) and SOC were important factors in predicting both clinically measured and self-reported oral health outcomes. Higher education, income and urban residency with higher availability of dentists were related to lower levels of periodontitis in *Paper I*. This is consistent with previously published literature, where socioeconomic factors have been related to periodontitis [2, 32, 33, 66, 70, 182]. How socioeconomic factors influence the etiological pathway of periodontitis is not well understood. It could be explained by differences in oral health-related behavior, access to dental health care, and norms for seeking treatment. As shown in *Paper II*, socioeconomic factors were related both directly and indirectly (via. e.g. enabling resources and smoking) to periodontitis. Higher education, high income and availability to dentists was also associated with less oral health impacts. This was in contrast to findings by Baker [156], where there was no direct association between socioeconomic status and self-reported oral health outcomes. Other studies of socioeconomic factors and subjective oral health support the current findings, reporting socioeconomic inequalities in oral health-related impacts [183-185].

A stronger SOC was, interestingly, related to worse periodontal status (*Paper II*). In previous studies of SOC and periodontitis, SOC has been related to self-perceived periodontal disease [151]. Some studies have found a weak association between SOC and extent of periodontal pockets (PD ≥ 4 mm) [147, 148], while other studies have not found any relationship between SOC and clinically assessed periodontitis [150, 151]. SOC is, after all, a psychological concept of how persons view their own lives, and it is plausible that it primarily affects the way individuals perceive their own health, rather than their clinically assessed health. It should also be considered that both SOC and periodontitis are positively correlated with age, which is not included in the model, and could be a potential confounder of the association between SOC and periodontitis. To further investigate this, the model could be tested in different age groups, where it could be assessed whether or not the association between SOC and periodontitis changes. Analysis in *Paper II* also revealed that SOC was the main predictive factor of oral health-related impacts. A stronger sense of coherence decreased the likelihood of having oral health impacts, which is in line with results from previous studies [145, 186].

Self-reported treatment need was directly and positively associated with periodontitis, although estimates were rather small. The small estimates could be explained by the fact that treatment need was not specified as periodontal treatment need, but included all needs related to oral health. Surprisingly, treatment need was not related to oral health impacts, in contrast to previous studies of Andersen's behavioral model and dental health [156, 157]. How participants understood the term "treatment need" could also have had an impact on the results. Treatment need is a broad term, and can range from a need for simple tooth cleaning to more extensive dental treatment.

Smoking was identified as a factor strongly associated with periodontitis in both *Paper I* and *II*. Regular use of dental services was related to more frequent tooth brushing, but did not affect smoking. While advice and guidelines on smoking cessation should be an important part of dental services, the current results indicate that dental services are not effective in reducing tobacco smoking.

Results presented in *Paper II* showed that there was a weak, and interestingly positive, association between use of dental services and periodontitis, meaning persons with frequent dental visiting habits had a slightly higher likelihood of having periodontitis. In bivariate analysis in *Paper I*, persons with yearly dental visits and persons only seeing dental care for acute problems did not differ in level of both non-severe and severe periodontitis. These results contradict the assumption that regular and prevention-oriented dental attendance should prevent or control periodontitis. A study of US males from 1994 found that utilization of dental services was not predictive of the extent and severity of periodontitis [187]. The same was reported for Swedish older adults, where regular dental visitors retained more teeth but had the same periodontal conditions as infrequent visitors [188]. Altogether, this calls into question the effectiveness of use of dental services in relation to periodontitis prevention and control, how it affects oral health-related behavior and raises concerns about possible under-diagnosis or failed/ineffective treatment of periodontal disease. On the other hand, it could also result from the fact that persons undergoing periodontal treatment would have more frequent dental visits. When examining regular dental attendees in more detail, periodontitis prevalence was highest among those using dental services more than once per year and lowest among those with biennial dental visits (data not shown). Moreover, successful treatment and control of periodontitis is dependent both on patient cooperation in periodontal infection control and provision of appropriate interventions and treatment by the dental care provider. Without information about what kind of dental care or treatment that was given, it is not possible to draw any conclusions about the effectiveness of dental services use related to periodontitis prevalence.

Use of dental services had no direct effect on oral health impacts, and was only indirectly related via periodontitis. Frequent and regular dental attendance was not associated with oral health impacts in a Norwegian cohort of aging people [189]. In other studies, routine dental attendance was reported to decrease oral health impacts [125, 156].

4.1.3 Clinical expression of gingival inflammation

As presented in *Paper III*, the clinical expression of gingival inflammation, measured by BOP, was strongly affected by smoking, with a dose-dependent effect. The results are in line with site-specific analyses of data collected in a population based epidemiological study conducted in the US [190], and in an observational study of dental patients in Italy [191].

Smoking additionally attenuated the bleeding response to dental plaque, which was further attenuated with increasing tobacco-exposure in terms of pack-years (*Paper III*). From previous experimental gingivitis studies and observational studies, it has been concluded that smoking reduces the gingival inflammatory response to plaque [96-104]. These conclusions have been drawn based on the association between subject's mean values of plaque and gingival bleeding. When site-specific data are aggregated and analyzed on a higher level, it can be defined as an ecological study [192]. This means that associations found at the subject level cannot be inferred to the site level. Based on the evidence mentioned above, it can be concluded that smokers on average have less gingival bleeding in relation to level of plaque as compared to non-smokers. It is not clear, however, whether the gingival bleeding response to plaque was actually attenuated at the site. There are few studies with site-specific analysis of BOP and smoking where plaque is included as a covariate. In the study of a US population plaque was not measured, but calculus was, which can be considered a proxy to plaque [190]. The authors reported a strong effect of sub- or supragingival calculus on BOP in never smokers, which was gradually attenuated in former, light and heavy smokers [190], supporting findings from *Paper III*. In a longitudinal study of 19-30 year old German soldiers, smoking did not affect the bleeding response to dental plaque [105]. When stratifying analysis on age groups, the bleeding response to plaque was to a much lesser extent affected by smoking in the youngest age group (20-34 years old). The lack of or reduced effect of smoking in young adults could be explained by the duration of tobacco-exposure. In the current study, there was a low number of smokers in the youngest age group (n = 47), and only one person with high tobacco-exposure.

Results from *Paper III* showed that the bleeding tendency of the gingiva was affected by several other factors, which were local factors (plaque, PD and tooth type), age, education level, and

overweight/obesity. No difference in BOP was found between males and females or between different levels of income. Compared to previous studies with site-specific analysis of BOP, effects of plaque and PD on BOP are consistent [190, 191]. For tooth types, posterior teeth were reported to have increased risk of BOP in Italian dental patients [191]. The conflicting results regarding tooth type could be due to the missing of an important (causal) factor, i.e. plaque, in the analysis of the above-mentioned study. For example, posterior teeth had more bleeding, but also more plaque and deeper PD. When adjusting for plaque and PD, posterior teeth were not more likely to bleed upon probing than other tooth types (data not shown). Higher age reduced the odds of bleeding with an apparent threshold effect at 45 years. A study of experimental gingivitis found that older persons developed more gingivitis than younger persons [193], while no difference in bleeding probability according to age was reported among Italian dental patients [191]. Gender differences in BOP have been reported, although, in opposite directions [190, 191]. Several studies have shown that lower socioeconomic status is related to more BOP or gingival inflammation [190, 191, 194, 195]. Socioeconomic factors were, as presented in *Paper II*, associated with toothbrushing directly and indirectly via enabling resources and use of dental services, presenting oral health-related behavior as a possible mechanism by which socioeconomic factors are related to gingival bleeding.

Obesity has been associated with periodontitis with several possible mechanisms proposed, e.g. increased inflammatory response, change in dental plaque amount and composition, or both [196]. Common risk factors, e.g. lifestyle-related factors, such as smoking, diet and insulin sensitivity could also have an impact on the respective association [53]. As presented in *Paper III*, overweight and obesity increased the bleeding tendency of the gingiva; however, persons with higher body mass index also had higher levels of plaque (Table 6).

Table 6. Mean percent BOP and plaque according to BMI categories

| BMI category | Mean BOP (%) | Mean plaque (%) |
|---------------|--------------|-----------------|
| Normal weight | 26.8 | 40.4 |
| Overweight | 30.7 | 45.0 |
| Obese | 33.5 | 47.9 |

When including plaque in a model of BOP and BMI categories, estimates for BMI categories changed by about 20%, indicating that part of the effect of overweight/obesity on BOP is due to increased levels of plaque. Whether plaque is a confounder or a mediator to the association between BOP and overweight/obesity depends on whether or not plaque is considered to be on the causal pathway (Figure 11) [197].

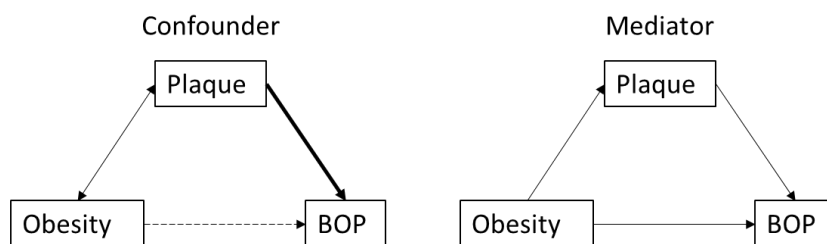


Figure 11. The possible structures of confounding and mediation in the association between obesity and BOP.

If obesity influences the quantity and/or composition of dental plaque [198], plaque would be a mediator. In that case, when both plaque and overweight/obesity are included in the model as covariates, the estimates for overweight/obesity represent only the direct effects, and not those mediated through plaque.

Results from *Papers I-III* shows that gingival bleeding and periodontal destruction are associated with many of the same factors. For example, higher education is associated with less gingival bleeding and less periodontitis. For smoking, the effect on BOP and periodontitis seem to be in opposite directions. On one hand, smoking reduces gingival bleeding tendency and response to dental plaque, while on the other hand smoking is associated with increased levels and severity of periodontitis. This could be explained by dual effects of smoking, i.e. toxic effects and immunosuppressive effects [118]. Nicotine is considered the main immunosuppressive constituent of cigarette smoke [199], and has even been suggested as a potential therapeutic agent in some chronic inflammatory diseases [200, 201].

There are many factors other than smoking that can modulate the bleeding response to plaque, such as pregnancy, diabetes, Down's syndrome, diet, anti-microbial and anti-inflammatory agents, and conditions affecting the immune responses (reviewed by Tatakis et al. [79]). These factors have not been controlled for in the current study. For example, vitamin D has been shown to affect gingival inflammation [86, 93]. This could have an effect on the results, considering that both smoking and obesity have been associated with lower levels of vitamin D [202, 203]. Furthermore, host-dependent variations in gingivitis susceptibility could also have an impact on the results [94, 95]. Plaque and BOP were only measured at one time-point, and it is possible that time of examination relative to eating and toothbrushing could have affected plaque levels. However, there is evidence that the distribution of plaque is consistent over time. In a longitudinal study of young adults, well-defined, symmetric and consistent patterns of dental plaque distribution were observed [204].

4.2 Methodological considerations

In all health and disease research, error and bias are inevitable. It is therefore important to recognize the potential sources of error in order to avoid making false conclusions.

4.2.1 Study design

Cross-sectional studies examine disease and risk factor patterns in populations, thus providing major contributions to the population burden of both risk factors and disease [205]. Cross-sectional studies also seek associations between risk factors and diseases and can generate and test hypothesis [205]. A major limitation of cross-sectional studies is that disease outcome and exposure is measured at the same time, meaning temporal relationships cannot be established and causality cannot be concluded [205]. The study was regional, and results cannot be directly inferred to other geographic regions. Differences in socio-demographic characteristics and oral health-related behaviors of the current study population should be carefully considered when extrapolating findings to other populations.

4.2.2 Validity and reliability

Validity is the degree to which conclusions drawn from a study are justified when study methods and characteristics of study participants are taken into consideration [206]. External validity, or generalizability, is the degree to which the results of a study can be applied to populations or groups that did not participate in the study [206]. Internal validity is the degree to which an observation or measurement can be shown to be true and accurate (the lack of bias and systematic error), and is a prerequisite for external validity [207]. Reliability refers to the stability of a repeated measurement, where lack of reliability may result from variation between observers or instruments of measurement [206].

4.2.2.1 Selection bias and generalizability

Selection bias is distortion that can result from the selection of study subjects where the characteristics of the study population differ systematically from those of other populations [206], and can be a threat to the external validity of the study. The aim of this study was to estimate the burden of periodontitis in the adult population in Troms County. In order to generalize or apply the results to the whole adult population of Troms County, the selected study population had to be representative for all adults living there. Participants were selected through random sampling methods. By having the same sampling fraction in each of the three stratified regions, every person in the county had the same chance of being selected. All adults, 20-79 years old, listed in the population register with a permanent address in Troms County, were eligible for participation in the

study, including persons living in institutions. Only persons without a registered phone number were excluded. Although this only applied to a minor fraction of the population, certain subgroups could have been excluded with unknown impact on the results.

The representativeness of the data is also dependent on the participation rate. To get a high level of participation can be challenging as participation rates for epidemiological studies have declined during the past decades [208, 209]. The following steps were taken to facilitate participation: In an effort to reach as many potential participants as possible and increase visibility of the study, it was advertised in local newspapers, on radio and on social media. Written invitations were sent by mail and was followed up by a phone call. Non-responders were re-contacted by postal letter.

Participants could choose from five different dental clinics located in five geographical regions of the county. They were also able to choose the time of the examination, and in some cases, evening hours were offered. To accommodate persons with long travelling distances or travelling difficulties, participants were offered reimbursement for travelling expenses and, if necessary, taxi services were arranged. A small incentive for participation was given (gift card of 150 NOK).

Finally, participants received written feedback about examination results. At the end of the study, the response rate was 68%, which is considered high compared to similar studies [23, 210, 211].

Flexibility of the time of examination is likely to have had a positive impact on participation [212]. Calling all participants by phone took a lot of time and effort. Reaching participants during working hours could also be challenging. Recent studies have reported that short text message (SMS) reminders can be an effective way to increase participation [212, 213]. In addition to sending SMS reminders of scheduled times for examinations, SMS could have been an efficient mode of first contact, especially among younger participants and even older participants hesitant to answer unknown callers.

Even with a high participation rate, selection bias might still occur if the non-response is not random [214-216]. To be able to assess the potential non-response bias differences between the non-responders and responders have to be examined. For the non-responders, the only information available was age, gender and address. If the non-responders gave a reason for not participating this was registered. When comparing responders and non-responders we found that non-responders were older and comprised a higher proportion of men than the responders (Table 7). There was no difference between responders and non-responders regarding what district they lived in. Frequently reported reasons for not participating among the oldest age groups were health problems, travelling difficulties and no need for dental care. Information about educational level or other socioeconomic factors were not available for non-responders. The educational level of the participants was,

however, higher compared to countywide levels. Forty-one percent of the study participants had university level education compared to 29% of the Troms County population in 2013 [217]. This indicates that persons with higher level of education were more inclined to participate. The possible overrepresentation of persons with higher level of education could underestimate prevalence of periodontitis, as higher education has been associated with less periodontitis [66, 72]. On the other hand, we experienced that people who had not seen a dentist for many years, e.g. for economic reasons, saw the study as an opportunity to get a free of charge full dental examination. If participation was also affected by the need for dental care, it would overestimate disease levels, opposing the effect of higher education. The underrepresentation of older adults could affect the generalization to the older population in Troms County. Health problems were reported as a common reason for not participating among older adults, and could have resulted in an underestimation of periodontitis among older adults, as poor general health and poor oral health are related, directly or through common risk factors [185-187]. Not attending because of “no need for dental care” could have the opposite effect and overestimate the prevalence of periodontitis. This should be taken into consideration when drawing conclusions about the older age groups.

Table 7. Characteristics of participants and non-participants

| Characteristic | Participants | | Non-participants | | P-value* |
|------------------|--------------|------|------------------|------|----------|
| | n | % | n | % | |
| Age group, yrs. | | | | | <0.001 |
| 20-34 | 478 | 24.1 | 178 | 19.3 | |
| 35-44 | 408 | 20.5 | 158 | 17.1 | |
| 45-54 | 379 | 19.1 | 163 | 17.3 | |
| 55-64 | 396 | 19.9 | 182 | 19.7 | |
| 65-79 | 325 | 16.4 | 242 | 26.2 | |
| Gender | | | | | 0.008 |
| Male | 967 | 49 | 499 | 54 | |
| Female | 1019 | 51 | 424 | 46 | |
| District | | | | | 0.723 |
| Tromsø | 897 | 45 | 403 | 45 | |
| Harstad | 403 | 20 | 204 | 24 | |
| Målselv/Finnsnes | 471 | 24 | 217 | 21 | |
| Storslett | 215 | 11 | 99 | 11 | |

* P-value for differences between groups using the χ^2 -test.

Survival bias is another type of selection bias. For analysis of periodontitis, participants who had less than two teeth or incomplete periodontal records were excluded. However, persons who have

lost all or most of their teeth could have lost them because of severe periodontitis. This means that persons with the most severe form of disease could possibly be excluded from the study. These participants might have a higher level of exposure or an entirely different exposure than participants with periodontitis but who have kept their teeth. For example, there was a noticeable difference in proportion of smokers between excluded persons due to edentulousness and included persons (Table 8). Not including edentulous participants when tooth loss could be a result of severe periodontitis could affect both burden of periodontitis and the association between exposure and disease. To investigate this in more detail, information about previous oral disease experience would be necessary, and ideally assessed in a longitudinal study.

Table 8. Distribution of periodontitis risk factors among included and excluded participants.

| | Included persons | Excluded – less than 2 teeth | Excluded – incomplete periodontal records | <i>P</i> -value* |
|------------------------|---------------------|---------------------------------|--|------------------|
| Year of age, mean (SD) | 47.3 (15.3) | 69.0 (8.2) | 56.2 (17.2) | <0.001 |
| Smoking status, n (%) | | | | 0.003 |
| Smoker | 283 (14.9) | 16 (30.8) | 1 (4.5) | |
| Non-smoker | 1,615 (85.1) | 36 (69.2) | 21 (95.5) | |
| Education, n (%) | | | | <0.001 |
| Less than high school | 273 (14.4) | 36 (69.2) | 8 (38.1) | |
| High school | 826 (43.6) | 15 (28.8) | 9 (42.9) | |
| University level | 795 (42.0) | 1 (1.9) | 4 (19.0) | |

* *P*-value for differences between groups using the χ^2 -test.

4.2.2.2 Misclassification

Misclassification is a type of information bias and is defined as “the erroneous classification of an individual, a value or an attribute into a category other than that to which it should be assigned” [206]. Misclassification can occur as a result of recall or reporting bias, observer bias or imprecise or poorly calibrated measurement instruments.

The information collected from the questionnaire could be affected by recall or reporting bias, which happens when participants are not correctly recalling past events or selectively reporting sensitive information or what they think is the “right” answer. Misclassification of exposure variables can have different effects on the association with the outcome depending on the type of misclassification [218]. Non-differential (random) misclassification generally underestimates the strength of the association. While differential misclassification (non-random), when measurement error differs between groups that are compared, can both under- and overestimate the strength of the association. For example, persons with periodontitis might have understated the amount of smoking

because they know smoking is bad for disease prognosis. This would underestimate the association between smoking and periodontitis. On the other hand, persons with periodontitis could also have been more interested in risk factors of the disease and be more likely to recall or report certain exposures, resulting in overestimation of the association. While the reported level of smoking in the current study was the same as for national estimates [219], misclassification of smoking cannot be completely ruled out, as overestimation in some groups and underestimation in others may have resulted in overall about right estimates. The use of previously used and validated questions and instruments also help limit the potential reporting bias. Not all questions and instruments have been tested in a Norwegian or Northern Norwegian population, which could be a potential source of bias with unknown consequences for the results.

The clinical periodontal examination is intricate and time consuming. PD was measured at six sites for up to 28 teeth, resulting in up to 168 measurements per participant. Many factors could have influenced the accuracy of the measurements. The probing pressure and the experience of the investigator, local factors like calculus or bleeding of the gums, and the investigators pre-knowledge of disease or exposure status could all have influenced the measurements. Both outcome and exposure like smoking can be difficult to blind for the investigator. Severe periodontitis is often apparent with visual inspection of the teeth and gums and could have caused investigators to make more careful measurements compared to persons with less visible signs of periodontitis. The examinations were carried out by 11 investigators. In order to minimize observer bias all investigators were carefully trained ahead of the examinations and standard protocols were followed making all measurements in the same way and order. The variability between examiners (inter-observer variability) were assessed between one of the investigators (an experienced periodontist) serving as a “gold standard” and each of the remaining 10 investigators in 10 different cases. Measurements could not be done 11 times on one participant, as the repeated probing would influence the measurements. PD is a continuous variable measured to the closest millimeter, and variability was assessed with intraclass correlation coefficients (ICC). The ICCs of agreement ranged from 0.43-0.94 (Table 9). To further investigate the variability between the “gold standard” and the investigators the mean difference was assessed. Eight of the 10 investigators measured on average more shallow periodontal probing depths compared to the “gold standard”. This means that the periodontal probing depth could have been underestimated. However, 90-100% of the measured difference between the “gold standard” and the investigators were within ± 1 mm.

Table 9. Variability in periodontal pocket measurements between the "gold standard" and each of the 10 investigators

| | ICC | Mean difference (gold standard – investigator) (mm) | Proportion of measurement differences within ± 1 mm (%) |
|-----------------|------|---|---|
| Investigator 1 | 0.94 | 0.2 | 100 |
| Investigator 2 | 0.84 | 0.1 | 100 |
| Investigator 3 | 0.77 | 0.2 | 94 |
| Investigator 4 | 0.90 | 0.1 | 100 |
| Investigator 5 | 0.56 | 0.1 | 94 |
| Investigator 6 | 0.78 | -0.1 | 100 |
| Investigator 7 | 0.43 | 0.6 | 90 |
| Investigator 8 | 0.93 | -0.2 | 100 |
| Investigator 9 | 0.87 | 0.7 | 90 |
| Investigator 10 | 0.75 | 0.3 | 100 |

In order to estimate the prevalence and severity of periodontitis using the CDC/AAP case definition, CAL had to be estimated from radiographic bone level. The validity of the method was tested in 19 cases using both true and predicted clinical attachment levels. The intraclass correlation coefficient between true and predicted clinical attachment level was 0.81. Validity was further assessed with a Bland-Altman plot [220], where the difference between the true clinical attachment level and the predicted clinical attachment levels and the mean of the two measures were plotted against each other (Figure 12).

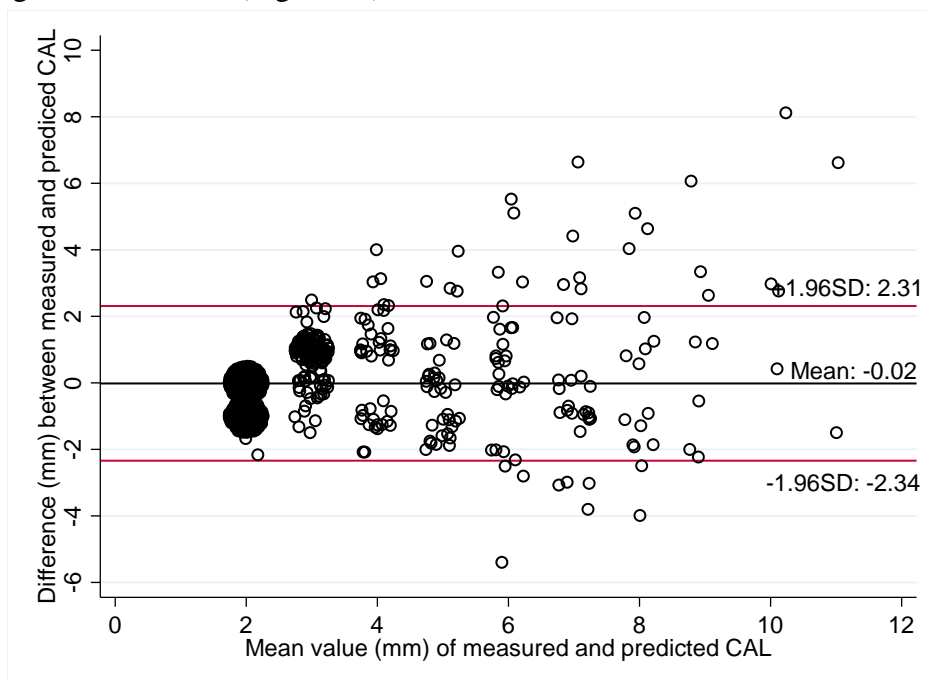


Figure 12. Bland-Altman plot illustrating the relationship between difference and mean values of measured and predicted CAL.

The mean difference between measured and predicted CAL was -0.02, meaning that overall, there was a slight tendency of overestimation of CAL. With higher levels of CAL, the difference between the two methods increased, and so did the tendency to underestimate CAL. This means that the ability of radiographic attachment level to predict clinical attachment level depended on the severity of attachment loss, with less reliable estimates for more severe attachment loss. The clinical attachment level was, however, estimated within ± 1 mm in 90% of measurements using the prediction model. The predicted levels of clinical attachment were not used to produce estimates of CAL in the study population. They were only used to apply periodontitis case definitions based on threshold values of: ≥ 3 mm, ≥ 4 mm and ≥ 6 mm CAL. In Table 10, periodontitis categories using both measures are presented. The use of predicted clinical attachment level misclassified one of the 19 cases (5%) with no periodontitis instead of mild to moderate periodontitis. Agreement between the two methods were tested with Cohens Kappa, with a value of 0.91.

Table 10. Periodontitis categories based on true and predicted clinical attachment level

| Periodontitis category | Using true clinical attachment levels, n (%) | Using predicted clinical attachment levels, n (%) |
|-----------------------------|--|---|
| No-periodontitis | 8 (42) | 9 (47) |
| Mild-moderate periodontitis | 3 (16) | 2 (11) |
| Severe periodontitis | 8 (42) | 8 (42) |

RBL was examined on dental radiographs by one investigator. The five different dental offices had x-ray machines from altogether three different manufacturers, which could have affected the quality of the radiographs and thereby the measurements of RBL. The positioning of the jaw and exposure time could also have affected the radiographic quality. Twenty-two participants were excluded because of poor quality of radiographs. The poor quality was mainly a result of suboptimal positioning either due to operating skills or functional limitations of the participant (e.g. unable to stretch the neck).

Bias due to error in periodontal measurements could have been reduced by limiting the number of investigators or by intensifying training and calibration of the investigators. CAL could, ideally, have been measured directly to avoid possible underestimation as a result of the indirect approach through radiographic bone level. The decision not to measure CAL was a compromise made due to time constraints of the examination. Nevertheless, measurements of CAL would not have been free of error [221-223].

4.3 Ethical considerations

Informed consent protects the rights and safety of patients in research [224], and is an essential part of ethical research. The Norwegian Act on Medical and Health Research [225] states that “consent must be obtained from participants in medical and health research, unless otherwise laid down in law. Consent must be informed, voluntary, express and documented.” To ensure that consent is valid the participant’s ability to understand and evaluate the research project must be taken into consideration, and the researcher is obliged to ensure that the information is comprehended.

In my own experience from the study, the comprehension of information varied among individuals. There was also a difference in what kind of information and which details were important for different persons. Some wanted to know if there would be any discomfort and how long the examination lasted, while other wanted to know how the collected information would be used and if we could guaranty the confidentiality of the data. We tried to make the invitation letter as easily readable and understandable as possible without excluding necessary information. The purpose of the study and the examination procedures were always explained prior to the clinical examination. Towards the end of the recruitment procedure a simplified version of the invitation letter was sent to persons we had not yet been able to contact, referring to the more detailed original invitation enclosed. This approach with a splitting of information, as has been suggested [226], resulted in a response from around 40% of those we were previously unable to reach. The use of a split information approach from the beginning of the study could have contributed to comprehension of information and might have increased response.

Dental care is by many people considered to be of high cost. By offering a free of charge dental examination individuals with economic challenges or without access to dental care may be more likely to participate out of need for care rather than the voluntariness to research. Also, a small financial compensation was offered. Financial compensation is something that could influence people to participate that otherwise would not. Especially large amounts of money could cause people to expose themselves to a greater risk. The amount of financial compensation should be seen in relation to the risks of the study, to avoid coercive force exerted on specific social groups e.g. those with economic challenges and/or poor access to health care. It is difficult to define what is acceptable encouragement and what is undue influence. Fisher [227] states that “undue influence balances on the interpretation of what might induce someone to participate against his or her better judgment”. Both the risk involved in participating in the study and the amount of financial compensation was considered low, hence, there should be a low risk of undue influence.

In terms of minimizing potential harm to participants, taking radiographs for research purposes could be discussed. A standard set of oral radiographs were taken for all participants, unless they were pregnant or it was otherwise not recommended or wanted by the participant. To minimize unnecessary radiographic exposure, recent x-rays, if they could be accessed, were used, and the participants were offered a copy of radiographs from the study, which could be used by their regular dental practitioner or hygienist. Optimized protocols with the lowest radiographic exposure producing acceptable image quality were used. The oral radiographs provided a valuable diagnostic source for several dental conditions. Combined with the re-use of these radiographs, the potential benefits outweigh the low risk of harm due to radiographic exposure (radiation doses of four intraoral radiographs and one OPG equivalent to a few days of background radiation [228]).

Although REC North approved the recruitment procedure for this study, calling participants by phone could have put pressure on people to participate and made declining participation more uncomfortable. On the other hand, it could be argued that by calling the potential participants they would get the chance to ask questions and receive information about the study to enable an informed consent. Furthermore, when calling, those who had not received the invitation, misplaced it, or confused it with advertisement got a chance to participate and thereby preventing exclusion of these groups. If the participant does not have a relationship of dependence with the caller, and the caller uses neutral non-emotional language and respects the potential participant's decision to decline or not receive information about the study, I argue that follow-up by phone would be ethically defensible.

5 Conclusions

Using observational data, this thesis provides new information about the prevalence, severity and extent of periodontitis in an adult population in Troms County, Northern Norway. Periodontitis is a common disease among adults. Socioeconomic factors and smoking were main predictors of periodontitis, and smoking also strongly affected the clinical expression of gingivitis. There is a complex relationship between population characteristics, use of dental services and oral health outcomes. The specific conclusions were:

- There is a high burden of periodontitis among adults in Troms County, Northern Norway, and the severity and extent of the disease is unequally distributed in the population
- Socioeconomic factors affect oral health-related behaviors, such as toothbrushing and smoking, and when mediated via perceived resources also the use of dental services
- Smoking was positively associated with periodontitis prevalence and severity, and had a strong and dose-dependent effect on the clinical expression of gingival inflammation
- Frequent use of dental services was associated with more toothbrushing, but not with reduced likelihood of periodontitis
- Oral health-related quality of life was to a greater extent affected by social structures and sense of coherence, and to a lesser extent by periodontitis
- Andersen's behavioral model of health services use was found to be a useful tool in identifying predictors of dental service utilization and oral health-related quality of life and how these relate to each other

6 Future perspectives

This thesis presents a high burden of periodontitis in Troms County, Northern Norway and disparities in periodontitis prevalence according to age, gender and socioeconomic factors. The findings call attention to further investigation of differences between population groups. Results from this thesis can provide an important contribution to the dental health services in the region in terms of planning periodontal health care needs and preventive actions. The incorporation of staging and grading of periodontitis according to the new case definition by the AAP/EFP in epidemiological studies could provide additional information about complexity levels and the need for advanced treatment in the population. How determinants and risk factors relate to the new definition of periodontitis would be interesting to further explore, especially the effect of a distinction between periodontal disease stability and periodontitis cases. This makes it possible to assess the effect of risk factors on the cumulative lifetime experience of periodontitis as well as the effect on current disease status.

The findings in *Paper II* suggest that how persons perceive their own resources in terms of access to dental services and dental anxiety is the main determinant of use of dental services. Although a large part of the population is regularly using dental services, it would be interesting to look into groups with irregular dental visits and assess how dental services can be made more easily accessible for the population. There is a need for more knowledge about the effectiveness of dental health care utilization related to periodontitis prevention and control. Studies of periodontal treatment prevalence could answer questions about possible underdiagnosis of periodontitis. Assessing experiences and attitudes among dental personnel related to periodontitis diagnosis and treatment by applying mixed (quantitative and qualitative) research methods could present interesting aspects. A longitudinal study of oral health applying Andersen's behavioral model of health services use, could validate the present finding and identify alternative explanations by testing possible reciprocal associations, such as use of dental services and periodontitis.

As presented in *Paper III*, tobacco smoking reduces the clinical expression of gingival inflammation, with consequences for early diagnosis and risk assessment of periodontal disease. More knowledge about smoking's effects on the gingival and periodontal inflammatory responses is necessary, and whether these responses represent solely negative or possibly to some extent positive effects. In particular studies collecting site-specific data of periodontal parameters in combination with microbiologic and biomedical testing could produce new insights into mechanisms at the periodontal site.

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Papers I-III

Paper I

Periodontitis Prevalence and Severity in Adults: A Cross-Sectional Study in Norwegian Circumpolar Communities

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Background: The aim of this study is to describe the prevalence, severity, and extent of periodontitis in the adult population of circumpolar communities in Norway using data from the Tromstannen–Oral Health in Northern Norway study.

Methods: In this cross-sectional survey, data were collected from a randomized population sample (aged 20 to 79 years) in Northern Norway. Periodontal conditions were assessed for 1,911 dentate adults with a full-mouth periodontal examination. Probing depth (PD) and bleeding on probing were measured at six sites per tooth. Radiographic bone loss (BL) was examined using orthopantomograms.

Results: According to the Centers for Disease Control and Prevention/American Academy of Periodontology case definitions, 49.5% of participants had periodontitis, and 9.1% had severe periodontitis. Periodontitis prevalence and severity increased with age. Extent of BL and PD \geq 4 mm also increased with age, but more rapidly and to a greater extent for BL. Prevalence of periodontitis was higher among men and varied between urban and rural areas. Periodontitis prevalence was positively associated with smoking, lower levels of education, and income.

Conclusions: This study reveals a high burden of periodontitis among adults living in circumpolar communities in Norway. The results showed sociodemographic disparities regarding periodontitis and highlights the importance of further investigation of factors influencing periodontal health. *J Periodontol* 2017;88:1012-1022.

KEY WORDS

Alveolar bone loss; arctic regions; dental health surveys; epidemiology; periodontitis.

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Periodontitis is a common disease among adults; its prevalence is reported by European and United States studies to range from 31% to 76%.¹⁻⁶ Severe forms of the disease affect 11% of the global population.⁷ Differences in demographic characteristics and levels of exposure to various risk factors among different populations can partially explain the wide range in the prevalence of periodontal disease, but this variance can also be the result of differences in periodontal examination protocols and case definitions among studies using different measures of periodontitis.⁸⁻¹¹ To enable a comparison between populations, the Joint EU/USA Periodontal Epidemiology Working Group has proposed standards for reporting the prevalence and severity of chronic periodontitis (CP).¹²

Periodontal disease is considered a major public health problem.¹³ It is reported to have a negative impact on oral health–related quality of life and the lives of patients, including impairment, functional limitations, discomfort, and disability.¹⁴⁻¹⁶ Consequently, it is important to gain knowledge about the periodontal condition of a population; by collecting reliable and comparable periodontal data, researchers can contribute to global estimates of the burden of periodontitis.⁷

Knowledge about prevalence of periodontal disease in the general adult population of Norway has been lacking. A nearly 40-year-old study (1979) described

periodontal conditions in a coastal community in Northern Norway (N = 297) in patients aged 20 to 69 years).¹⁷ Other studies have described periodontal conditions exclusively in age cohorts (35 year) in Oslo between 1973 and 2003 (N = 543)¹⁸ and in a national random sample (N = 394) of elderly pensioners.¹⁹ Northern Norway has a history of low dentist-to-patient ratio, and in a national health interview survey of living conditions, it was reported that Northern Norway had the poorest self-reported dental health and the least frequent use of dental health services.²⁰ Additionally, large geographic disparities with respect to tooth loss and denture wearing have been reported in Norway.²¹ There is a need for studies estimating the burden of periodontitis and possible risk factors in the northern part of the country to aid the planning of dental health care services in the region. Furthermore, knowledge of periodontal conditions in these northern communities could be of interest for other regions with similar living conditions because there are few studies describing periodontal conditions.^{2,5,22-26} Studies of periodontitis prevalence in circumpolar countries provide only national estimates or estimates from regions south of the Arctic Circle,^{2,5,22-24} or they focus on indigenous populations.^{25,26} This is the first epidemiologic study in the general adult population of an entire Norwegian county. The aim is to describe the prevalence, severity, and extent of periodontitis in circumpolar communities in Norway, according to the recommended standards for measuring CP,¹² as well as to examine differences in the sociodemographic and behavioral characteristics of people with periodontitis.

MATERIALS AND METHODS

Study Design and Data Collection

To describe periodontal conditions, data from a dental health survey²⁷ in Northern Norway (Tromstannen–Oral Health in Northern Norway [TOHNN]) were used. The TOHNN study is a population-based, cross-sectional representative study with a target population of adults aged 20 to 79 years, living in Troms County, Norway. Troms County is one of three Norwegian counties located north of the Arctic Circle. Tromsø, one of the largest cities within the Arctic Circle, surrounded by islands, fjords, and mountain peaks, and the gateway to the Polar Seas, is included in the catchment area. In January 2013, 112,253 people in the selected age group inhabited the county. A power calculation, with a 95% confidence interval and a margin of error of 1.5%, indicated that 1,516 individuals needed to be examined to be able to describe the prevalence of severe periodontitis when hypothesizing a 10% prevalence as reported in the literature.⁷ The total sample (N = 3,000) was based on a 50% attendance rate experienced in other epidemiologic studies in Norway.^{18,28-30} To obtain

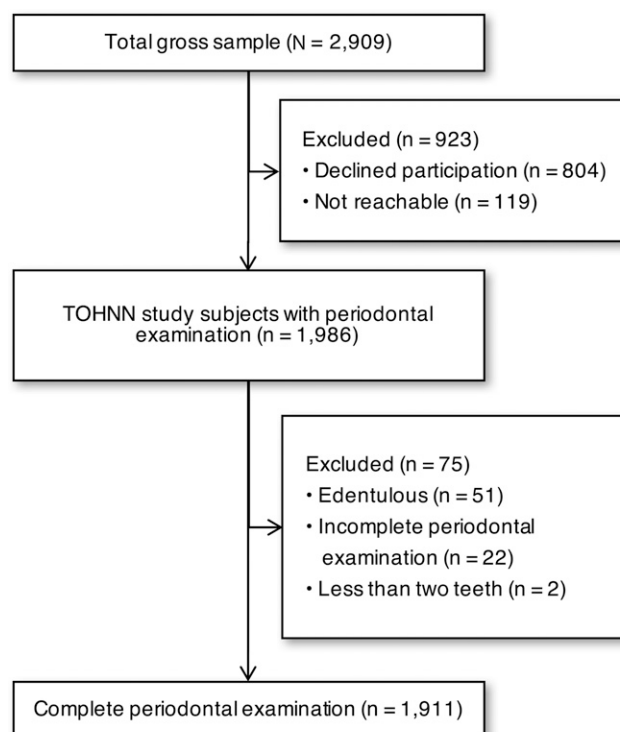


Figure 1.
Flowchart of study participants.

a representative selection of all regions in the county, the sample was stratified on three different areas: Tromsø (51,110 people: 46%), Southern Troms County (49,740 people: 44%), and northern Troms County (11,403 people: 10%). Three thousand individuals were selected by simple random sampling technique from the population register by Statistics Norway, resulting in 1,380 people from Tromsø, 1,320 people from Southern Troms County, and 300 people from Northern Troms County.

A total of 2,909 individuals was invited to participate in the study by a letter of invitation. Initial non-responders were contacted with an additional letter. Details of the invitation procedure have been described previously.²⁷ The study included a questionnaire and a clinical dental examination, and was completed by 1,986 (68.3%) participants. The study was approved by the Regional Committees for Medical and Health Research Ethics, Tromsø, Norway (2013/348/REC North). All participants provided written informed consent.

Information on sociodemographic characteristics, behaviors, and comorbidities were collected by self-reported questionnaire. The questionnaire covered questions about the following: 1) self-perceived ethnicity; 2) education; 3) annual household gross income (analyzed in three categories according to the

national tertiles of gross household income in 2013); 4) diabetes mellitus (DM); 5) toothbrushing frequency; 6) frequency of dental visits; 7) smoking; and 8) Swedish type, low-nitrosamine, smokeless tobacco (snus) use. Smoking was assessed with three questions: 1) Do you smoke on a daily basis? 2) How many cigarettes do you smoke each day? 3) For how many years have you been smoking? Number of years of past smoking was also registered. Use of snus was assessed with the same questions. Age was stratified in categories 20 to 34, 35 to 44, 45 to 54, 55 to 64, and 65 to 79 years. To assess urban–rural disparities, municipalities were categorized into the following three groups: 1) the municipality with the largest city (Tromsø) was classified as urban; 2) two municipalities (Harstad and Lenvik) with smaller towns were classified as suburban; and 3) the remaining municipalities without towns were classified as rural.

Periodontal examinations were performed on all individuals with natural teeth. Twenty-two participants were excluded because of incomplete periodontal examinations, and 51 (2.6%) were identified as edentulous; two participants had only one tooth and were excluded because of case definition criteria of measurements from two or more teeth. This resulted in 1,911 participants (936 males and 975 females, aged 20 to 79 years; mean age: 47.3 ± 15.3 years) with complete periodontal examinations (Fig. 1). Examinations were performed in a dental office by 11 calibrated dentists (employed by The Public Dental Health Service in Troms County, including authors GEH, NO, and AT) assisted by dental nurses. Bleeding on probing (BOP) and periodontal probing depth (PD) were assessed at six sites per tooth for all teeth. Third molars and implants were excluded from analysis. Periodontal PD was measured to the closest millimeter with a periodontal probe with single millimeter graduations.¹¹ Orthopantomograms (OPG) were used to assess radiographic bone level.^{2,18} Marginal bone levels of both distal and mesial surfaces of all teeth, excluding third molars, were measured linearly with a transparent plastic ruler.³¹ Alveolar bone level was measured in relation to the radiographic apex. The cemento-enamel junction (CEJ), alveolar crest (AC), and radiographically depicted root apex were used as reference points. If the CEJ was destroyed after restorative therapy, the apical margin of the restoration was used as a reference point. The AC was considered the most coronal point at which the periodontal ligament space had a constant width. If the CEJ or AC could not be determined for >20% of teeth, the participant was excluded from analysis. Bone loss (BL) was considered present at sites in which distance from the CEJ to the AC exceeded 2 mm and was categorized in 10% intervals as 1 to 10, as described by Skudutyte-Rysstad et al.¹⁸ A modified plaque index

(PI) was used,³² recording plaque at four sites per tooth as present or not using a mouth mirror and periodontal probe.

Periodontal Case Definition and Periodontal Parameters

To estimate the prevalence and severity of periodontitis, a categorical case definition was necessary. The Joint EU/USA Periodontal Epidemiology Working Group has suggested Centers for Disease Control and Prevention/American Academy of Periodontology (CDC/AAP) case definitions for reporting of periodontitis in epidemiologic studies.¹² These case definitions are based on PD and clinical attachment level (CAL) with the following definitions: 1) severe periodontitis: at least two interproximal sites with ≥ 6 mm CAL (not on the same tooth) and at least one interproximal site(s) with ≥ 5 mm PD; 2) moderate periodontitis: at least two interproximal sites with ≥ 4 mm CAL (not on the same tooth) or at least two interproximal sites with PD ≥ 5 mm (not on the same tooth); and 3) mild periodontitis: at least two interproximal sites with ≥ 3 mm CAL and at least two interproximal sites with ≥ 4 mm PD (not on the same tooth) or one interproximal site with ≥ 5 mm PD. In this study, CAL was unknown. To be able to define periodontitis according to the CDC/AAP case definition in the present sample, the relationship between radiographic BL and CAL was inferred from results in a complementary sample. To get a wide range of CALs, eight patients visiting the periodontal clinic at the Public Dental Health Service Competence Center of Northern Norway, Tromsø, Norway, were examined, along with 11 patients with no or mild periodontitis (by GEH). PD and CAL were measured clinically on all proximal surfaces ($n = 786$), and proximal bone levels on OPGs were recorded. BL was measured as a proportion of the root, categorized in 10% intervals, 1 to 10. CAL was measured in millimeters indirectly by first measuring the PD (distance from the gingival margin [GM] to the bottom of the pocket), followed by measuring distance from the CEJ to the GM. If the GM was coronal to the CEJ, the measurement was given a negative value and subtracted from the PD measurement. To assess the ability of measured radiographic bone loss to predict measured CAL, the following model was tested by linear regression:

$$CAL = \beta_0 + \beta_1 \cdot BL$$

In this formula, β_0 was the intercept and the value for CAL when $BL = 0$, and β_1 was the difference in CAL for each one-unit difference in BL. The model was statistically significant ($F_{1, 786} = 1,616.20$, $P < 0.001$)

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Table 1.

Characteristics of Study Participants With Periodontal Examination (N = 1,911) Stratified by Age and in Total

| Characteristics | Age Groups (years) | | | | | P Value* | Total |
|--------------------------------------|--------------------|------------|------------|------------|------------|----------|--------------|
| | 20 to 34 | 35 to 44 | 45 to 54 | 55 to 64 | 65 to 79 | | |
| Number of participants | 461 | 385 | 392 | 373 | 300 | | 1,911 |
| Proportion of target population, % | 1.5 | 1.7 | 1.8 | 1.9 | 1.6 | | 1.7 |
| Males, n (%) | 203 (44.0) | 187 (48.6) | 190 (48.5) | 194 (52.0) | 162 (54.0) | 0.062 | 936 (49.0) |
| Ethnicity, n (%) | | | | | | 0.780 | |
| Norwegian | 445 (96.5) | 377 (98.2) | 379 (96.7) | 364 (97.6) | 292 (97.7) | | 1,857 (97.3) |
| Sámi | 7 (1.5) | 3 (0.8) | 5 (1.3) | 6 (1.6) | 3 (1.0) | | 24 (1.2) |
| Other | 9 (2.0) | 4 (1.0) | 8 (2.0) | 3 (0.8) | 4 (1.3) | | 28 (1.5) |
| Education, n (%) | | | | | | <0.001 | |
| University level | 190 (41.4) | 222 (58.0) | 171 (43.8) | 133 (36.2) | 79 (26.8) | | 795 (42.0) |
| High school | 245 (53.4) | 141 (36.8) | 168 (43.1) | 165 (45.0) | 107 (36.3) | | 826 (43.6) |
| Less than high school | 24 (5.2) | 20 (5.2) | 51 (13.1) | 69 (18.8) | 109 (36.9) | | 273 (14.4) |
| ‡Household income, n (%) | | | | | | <0.001 | |
| ≥105,499 USD | 69 (15.8) | 122 (32.4) | 101 (26.1) | 65 (18.0) | 13 (4.7) | | 370 (20.1) |
| 52,750 to 105,498 USD | 179 (41.1) | 194 (51.5) | 208 (53.7) | 214 (59.3) | 114 (41.3) | | 909 (49.5) |
| <52,750 USD | 188 (43.1) | 61 (16.2) | 78 (20.2) | 82 (22.7) | 149 (54.0) | | 558 (30.4) |
| Demographic status, n (%) | | | | | | <0.001 | |
| Urban | 226 (49.0) | 194 (50.4) | 187 (47.7) | 142 (38.1) | 117 (39.0) | | 866 (45.3) |
| Suburban | 133 (28.9) | 108 (28.1) | 129 (32.9) | 134 (35.9) | 88 (29.3) | | 592 (31.0) |
| Rural | 102 (22.1) | 83 (21.6) | 76 (19.4) | 97 (26.0) | 95 (31.7) | | 453 (23.7) |
| Toothbrushing frequency, n (%) | | | | | | <0.001 | |
| Twice daily | 314 (68.9) | 279 (73.0) | 301 (77.0) | 282 (77.1) | 182 (62.1) | | 1,358 (71.9) |
| Once daily | 121 (26.5) | 92 (24.1) | 81 (20.7) | 77 (21.0) | 87 (29.7) | | 458 (24.3) |
| Less often than daily | 21 (4.6) | 11 (2.9) | 9 (2.3) | 7 (1.9) | 24 (8.2) | | 72 (3.8) |
| Frequency of dental visit, n (%) | | | | | | <0.001 | |
| Yearly | 145 (31.5) | 183 (48.2) | 232 (59.2) | 249 (67.3) | 196 (66.2) | | 1,005 (53.0) |
| Every other year | 85 (18.5) | 61 (16.0) | 53 (13.5) | 36 (9.7) | 20 (6.8) | | 255 (13.4) |
| Less often than every other year | 81 (17.6) | 47 (12.4) | 34 (8.7) | 28 (7.6) | 24 (8.1) | | 214 (11.3) |
| Only for acute problems | 149 (32.4) | 89 (23.4) | 73 (18.6) | 57 (15.4) | 56 (18.9) | | 424 (22.3) |
| Smoking status, n (%) | | | | | | 0.003 | |
| Never smoker | 402 (87.6) | 309 (81.3) | 311 (79.7) | 291 (78.4) | 259 (86.9) | | 1,572 (82.8) |
| Former smoker | 10 (2.2) | 9 (2.4) | 6 (1.5) | 10 (2.7) | 8 (2.7) | | 43 (2.3) |
| Current smoker | 47 (10.2) | 62 (16.3) | 73 (18.7) | 70 (18.9) | 31 (10.4) | | 283 (14.9) |
| §Smokeless tobacco use, n (%) | | | | | | <0.001 | |
| Never user | 321 (69.8) | 324 (85.3) | 353 (90.5) | 343 (93.0) | 291 (99.3) | | 1,632 (86.2) |
| Former user | 4 (0.9) | 4 (1.0) | 3 (0.8) | 0 (0) | 0 (0) | | 11 (0.6) |
| Current user | 135 (29.3) | 52 (13.7) | 34 (8.7) | 26 (7.0) | 2 (0.7) | | 249 (13.2) |
| DM, n (%) | 3 (0.7) | 5 (1.3) | 10 (2.6) | 20 (5.4) | 34 (11.8) | <0.001 | 72 (3.8) |
| Tooth count in dentates, mean (SD) | 27.2 (1.6) | 26.9 (1.7) | 26.1 (2.3) | 24.0 (4.6) | 19.1 (7.0) | <0.001† | 25.0 (4.7) |

Data are presented as means (SD) or as numbers with percentages given in parentheses. USD = US Dollars.

* P value for differences between groups using the χ^2 test.

† P value for differences between groups using one-way ANOVA.

‡ Average household income in Norway for 2013: 85,665 US dollars.

§ Swedish type, low-nitrosamine, smokeless tobacco.

|| Excluding third molars.

and explained 67% of the variance. All parameters of the model were significant ($P < 0.001$) and estimated as follows:

$$\text{CAL} = 2.0 \text{ mm} + 1.3\text{BL}$$

Using this model, measured radiographic BL was related to measured CAL with BL categories 0, 1, 2, and 3 corresponding to 2.0, 3.3, 4.6, and 5.9 ($\approx 2, 3, 5,$ and 6) mm of CAL, respectively, and could be used to apply the CDC/AAP case definition. Using predicted measures of CAL to apply the CDC/AAP case definition accurately defined 95% of cases previously defined using direct measures of CAL, with an intraclass correlation coefficient (ICC) of 0.95.

To provide a detailed description of periodontal status, the prevalence and extent (proportion of sites and teeth affected) of threshold values (PD ≥ 4 and ≥ 6 mm, and BL $> 0\%$ and $> 10\%$) were presented. For case definitions, mild and moderate periodontitis were

combined into one category, labeled “non-severe” periodontitis.^{5,33} Total periodontitis was defined as the presence of either severe or non-severe periodontitis, reported as “periodontitis.” BOP and PI were presented according to periodontitis severity and age group.

Examiner Reliability

Examiners were trained under supervision of a periodontist (NO) prior to data collection to reduce measurement bias. Interexaminer agreement in PD measurements between the 10 examiners and the periodontist (NO), was assessed at site level. Congruency was compared to the nearest millimeter. The median ICC of agreement was 0.81 (range: 0.43 to 0.94). One examiner (GEH) performed all measurements of radiographic BL on OPGs. Test-retest agreement of site-level measurements was assessed on two occasions with two sets of duplicate examinations of 10 OPGs. In the first case, examinations performed at the beginning of the examination period

Table 2.

Prevalence and Extent of BL and PD, and Overall Mean BL and PD by Age Group and in Total

| Measure of Periodontitis | Age Groups (years) | | | | | P Value for Trend* | Total |
|-------------------------------|--------------------|------------|------------|------------|------------|--------------------|------------|
| | 20 to 34 | 35 to 44 | 45 to 54 | 55 to 64 | 65 to 79 | | |
| BL, % (SE) | | | | | | | |
| Prevalence | | | | | | | |
| BL $> 0\%$ | 28.0 (2.1) | 68.1 (2.4) | 84.7 (1.8) | 97.1 (0.9) | 99.3 (0.5) | < 0.001 | 72.4 (1.0) |
| BL $> 10\%$ | 4.8 (1.0) | 20.3 (2.1) | 35.5 (2.4) | 66.2 (2.5) | 78.3 (2.4) | < 0.001 | 37.7 (1.1) |
| BL, mean (SE) | | | | | | | |
| Proportion of sites/mouth (%) | | | | | | | |
| BL $> 0\%$ | 1.7 (0.2) | 7.3 (0.6) | 18.8 (1.1) | 38.5 (1.5) | 52.7 (1.6) | < 0.001 | 21.5 (0.6) |
| BL $> 10\%$ | 0.2 (0.04) | 0.8 (0.1) | 3.8 (0.5) | 10.8 (1.0) | 17.8 (1.3) | < 0.001 | 5.9 (0.3) |
| Proportion of teeth/mouth (%) | | | | | | | |
| BL $> 0\%$ | 2.9 (0.04) | 11.1 (0.8) | 25.1 (1.3) | 48.1 (1.5) | 63.0 (1.6) | < 0.001 | 27.4 (0.7) |
| BL $> 10\%$ | 0.3 (0.1) | 1.4 (0.2) | 5.4 (0.7) | 14.6 (1.1) | 23.6 (1.5) | < 0.001 | 8.0 (0.4) |
| Mean BL (%) | 0.2 (0.03) | 0.8 (0.1) | 2.5 (0.2) | 5.8 (0.3) | 8.8 (0.5) | < 0.001 | 3.2 (0.1) |
| PD, % (SE) | | | | | | | |
| Prevalence | | | | | | | |
| PD ≥ 4 mm | 54.9 (2.3) | 65.7 (2.4) | 71.4 (2.3) | 80.2 (2.1) | 81.0 (2.3) | < 0.001 | 69.5 (1.1) |
| PD ≥ 6 mm | 5.9 (1.1) | 12.7 (1.7) | 17.6 (1.9) | 30.6 (2.4) | 33.0 (2.7) | < 0.001 | 18.7 (0.9) |
| PD, mean (SE) | | | | | | | |
| Proportion of sites/mouth (%) | | | | | | | |
| PD ≥ 4 mm | 2.9 (0.3) | 4.2 (0.4) | 6.1 (0.5) | 9.3 (0.7) | 10.3 (0.8) | < 0.001 | 6.2 (0.2) |
| PD ≥ 6 mm | 0.1 (0.1) | 0.3 (0.1) | 0.7 (0.2) | 1.1 (0.2) | 1.3 (0.2) | < 0.001 | 0.6 (0.1) |
| Proportion of teeth/mouth (%) | | | | | | | |
| PD ≥ 4 mm | 10.0 (0.8) | 14.3 (1.0) | 18.0 (1.1) | 25.4 (1.4) | 27.4 (1.6) | < 0.001 | 18.3 (0.5) |
| PD ≥ 6 mm | 0.5 (0.2) | 1.1 (0.2) | 2.4 (0.4) | 4.0 (0.5) | 4.2 (0.5) | < 0.001 | 2.3 (0.2) |
| Mean PD (mm) | 2.0 (0.02) | 2.1 (0.02) | 2.1 (0.02) | 2.2 (0.03) | 2.3 (0.04) | < 0.001 | 2.1 (0.01) |

Values are given as means or percentages with SE in parentheses. BL of $> 0\%$ and $> 10\%$ relating to degree of clinical attachment loss ≥ 3 and ≥ 5 mm, respectively.

* P value for linear trend across age groups.

were re-examined after 3 months, with an ICC of 0.78. In the second case, a second set of OPGs examined at the end of the examination period was re-examined after 1 week, with an ICC of 0.88.

Statistical Analyses

Data were analyzed using statistical software.[¶] Demographic and socioeconomic characteristics are presented as means (standard deviation [SD]) or numbers (proportions) for the total study population stratified by age. Differences in background characteristics between age groups were assessed with Pearson χ^2 test or one-way analysis of variance (ANOVA). Radiographic BL and PD are presented as means (standard error [SE]) and proportions (SE) of affected sites and teeth per mouth for the total study population and by age group. PD is presented using measurements from all six sites per tooth. Tests of linear trend across age groups of BL and PD were estimated using linear regression models for continuous variables and logistic regression for binary variables. Prevalence of periodontitis is presented as proportions (SE). Overall estimates of total, severe, and non-severe periodontitis were standardized to the age distribution of the 2013 Troms County population. The group with the lowest prevalence of periodontitis served as a referent group within each category, and the absolute difference from this group in percentage points was calculated. Differences between groups were assessed with z-tests, with a significance level set at 0.05. Additionally, BOP and PI are presented as means (SD) for the total study population and for subpopulations stratified by severity of periodontal disease according to the CDC/AAP case definition. The Lorenz curve was created with a spreadsheet software[#] and used to describe the distribution of PD ≥ 4 mm in the population,³⁴ where the cumulative proportion of total population is plotted against the cumulative proportion of PD ≥ 4 mm. A straight diagonal line would depict perfect equality, where every person would have the same number of PD ≥ 4 mm. The extent to which the curve sags below the straight diagonal line indicates the degree of inequality of distribution. The Gini coefficient represents the area between the line of equality (diagonal) and the Lorenz curve, calculated using the Riemann sum estimate (middle sum). The higher the Gini coefficient, the more unequal the distribution is.

RESULTS

Study Population

The mean age of participants was 47.3 ± 15.3 years, and 51% were women (Table 1). About 45% of participants resided in urban areas and 42% reported having a university level education. Of the

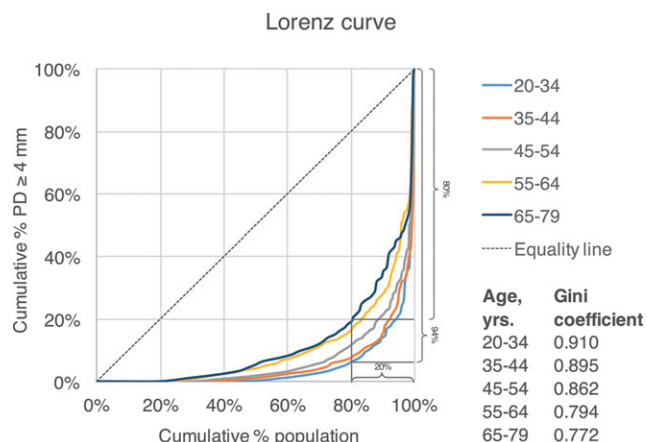


Figure 2.

Proportional distribution of sites with PD ≥ 4 mm in different age groups. Each point of the curves denotes the proportion of the population (x-axis) responsible for the proportion of the total burden of PD ≥ 4 mm (y-axis) in respective age groups. For example, in patients aged 65 to 79 years, 20% of the population accounted for 80% of the total burden of PD ≥ 4 mm, whereas in patients aged 20 to 34 years, 20% of the population accounted for 94% of the total burden of PD ≥ 4 mm.

examined population, $\approx 20\%$ were categorized in the highest income group. Prevalence of DM was 3.8%. For oral hygiene habits, the majority reported brushing their teeth at least twice daily. Fifteen percent were current smokers. Mean number of teeth present was 25. Fifty-two participants (2.7%) reported ethnicities other than Norwegian.

Radiographic BL and Periodontal PD

In Table 2, the prevalence and extent of radiographic BL and PD are presented by selected thresholds. Prevalence of radiographic BL spiked from the age of 35 years, reaching almost 100% in the 65- to 79-year-old age group. The extent of BL also increased rapidly with age. Prevalence of PD ≥ 4 mm was high across all age groups. The extent of PD ≥ 4 mm increased with age but to a lesser degree than BL. Figure 2 presents distribution of PD ≥ 4 mm in the population stratified by age group. Number of sites with PD ≥ 4 mm was unequally distributed in the population for all age groups. Although more than four in five 65 to 79 year olds had some sites with PD ≥ 4 mm, the majority (80%) of all sites with PD ≥ 4 mm was contributed by 20% of individuals in the age group. Disparities in the distribution of sites with PD ≥ 4 mm increased with decreasing age; 20% of 20 to 34 year olds accounted for 94% of all sites with PD ≥ 4 mm.

[¶] SPSS Statistics for Windows, v24.0, IBM, Armonk, NY.

[#] Excel 2013 for Windows, Microsoft, Redmond, WA.

Table 3.**Distribution of Participants Classified According to CDC/AAP Case Definition by Sociodemographic, Behavioral, and Comorbidity Variables (proportions)**

| Characteristics | n | Periodontitis (CDC/AAP Case Definition) ¹¹ | | | | | | | | |
|--------------------------------------|-------|---|-------|--------------------------|--------|-------|--------------------------|-------|--------|--------------------------|
| | | Non-severe | | | Severe | | | Total | | |
| | | % | (SE) | Absolute difference (pp) | % | (SE) | Absolute difference (pp) | % | (SE) | Absolute difference (pp) |
| Total | 1,911 | 40.4 | (1.1) | | 9.1 | (0.7) | | 49.5 | (1.1) | |
| *Total, Age standardized | | 39.2 | (1.1) | | 8.8 | (0.6) | | 48.2 | (1.1) | |
| Age group (years) | | | | | | | | | | |
| 20 to 34 | 461 | 15.8 | (1.7) | Ref. _a | 0.2 | (0.2) | Ref. _a | 16.1 | (1.7) | Ref. _a |
| 35 to 44 | 385 | 33.0 | (2.4) | 17.2 _b | 1.6 | (0.6) | 1.4 _a | 34.6 | (2.4) | 18.5 _b |
| 45 to 54 | 392 | 46.4 | (2.5) | 30.6 _c | 7.4 | (1.3) | 7.2 _b | 53.8 | (2.5) | 37.7 _c |
| 55 to 64 | 373 | 57.9 | (2.6) | 42.1 _d | 18.0 | (2.0) | 17.8 _c | 75.9 | (2.2) | 59.8 _d |
| 65 to 79 | 300 | 57.7 | (2.9) | 41.9 _d | 23.7 | (2.5) | 23.5 _c | 81.3 | (2.3) | 65.2 _d |
| Sex | | | | | | | | | | |
| Females | 975 | 35.7 | (1.5) | Ref. _a | 6.9 | (0.8) | Ref. _a | 42.6 | (1.6) | Ref. _a |
| Males | 936 | 45.2 | (1.6) | 9.5 _b | 11.4 | (1.0) | 4.5 _b | 56.7 | (1.6) | 14.1 _b |
| Ethnicity | | | | | | | | | | |
| Norwegian | 1,857 | 40.3 | (1.1) | 7.0 _a | 9.0 | (0.7) | 4.8 _a | 49.4 | (1.2) | 11.9 _a |
| Sámi | 24 | 33.3 | (9.8) | Ref. _a | 4.2 | (4.2) | Ref. _a | 37.5 | (10.1) | Ref. _a |
| Other | 28 | 46.4 | (9.6) | 13.1 _a | 14.3 | (6.7) | 10.1 _a | 60.7 | (9.4) | 23.2 _a |
| Education | | | | | | | | | | |
| University | 795 | 35.2 | (0.7) | Ref. _a | 4.7 | (0.7) | Ref. _a | 39.9 | (1.7) | Ref. _a |
| High school | 826 | 41.8 | (1.1) | 8.3 _b | 10.4 | (1.1) | 5.7 _b | 52.2 | (1.7) | 12.3 _b |
| Less than high school | 273 | 50.6 | (2.3) | 6.2 _c | 17.6 | (2.3) | 12.9 _c | 68.1 | (2.8) | 28.2 _c |
| Annual household income [†] | | | | | | | | | | |
| ≥105,499 USD | 370 | 34.3 | (2.5) | Ref. _a | 4.6 | (1.1) | Ref. _a | 38.9 | (2.5) | Ref. _a |
| 52,750 to 105,498 USD | 909 | 42.6 | (1.6) | 8.3 _b | 8.1 | (0.9) | 3.5 _a | 50.7 | (1.7) | 11.8 _b |
| <52,750 USD | 558 | 40.5 | (2.1) | 6.2 _{a,b} | 14.0 | (1.5) | 9.4 _b | 54.5 | (2.1) | 15.6 _b |
| Demographic status | | | | | | | | | | |
| Urban | 866 | 34.0 | (1.6) | Ref. _a | 8.0 | (0.9) | Ref. _a | 41.9 | (1.7) | Ref. _a |
| Suburban | 592 | 46.1 | (2.1) | 12.1 _b | 10.3 | (1.3) | 2.3 _a | 56.4 | (2.0) | 14.5 _b |
| Rural | 453 | 45.0 | (2.3) | 11.0 _b | 9.7 | (1.4) | 1.7 _a | 54.8 | (2.3) | 12.9 _b |
| Frequency of dental visits | | | | | | | | | | |
| Yearly | 1,005 | 46.2 | (1.6) | 20.3 _b | 9.8 | (0.9) | 2.9 _a | 55.9 | (1.6) | 24.1 _b |
| Every other year | 255 | 25.9 | (2.7) | Ref. _a | 6.9 | (1.5) | Ref. _a | 31.8 | (2.9) | Ref. _a |
| Less often than every other year | 214 | 29.4 | (3.1) | 3.5 _a | 8.9 | (1.9) | 2.0 _a | 38.3 | (3.3) | 6.5 _a |
| Only for acute problems | 424 | 40.6 | (2.4) | 11.2 _b | 9.7 | (1.4) | 2.8 _a | 50.2 | (2.4) | 18.4 _b |
| Smoking status | | | | | | | | | | |
| Never smoker | 1,572 | 37.9 | (1.2) | Ref. _a | 7.4 | (0.7) | Ref. _a | 45.4 | (1.3) | Ref. _a |
| Former smoker | 43 | 46.5 | (7.7) | 8.6 _{a,b} | 11.6 | (4.9) | 4.2 _{a,b} | 58.1 | (7.6) | 12.7 _{a,b} |
| Current smoker | 283 | 51.6 | (3.0) | 13.7 _b | 18.4 | (2.3) | 11.0 _b | 70.0 | (2.7) | 24.6 _b |

Table 3. (continued)**Distribution of Participants Classified According to CDC/AAP Case Definition by Sociodemographic, Behavioral, and Comorbidity Variables (proportions)**

| Characteristics | n | Periodontitis (CDC/AAP Case Definition) ¹¹ | | | | | | | | |
|------------------------------------|-------|---|--------|--------------------------|--------|-------|--------------------------|-------|--------|--------------------------|
| | | Non-severe | | | Severe | | | Total | | |
| | | % | (SE) | Absolute difference (pp) | % | (SE) | Absolute difference (pp) | % | (SE) | Absolute difference (pp) |
| Smokeless tobacco use [‡] | | | | | | | | | | |
| Never user | 1,632 | 42.0 | (1.2) | 23.8 _a | 9.6 | (0.7) | 4.4 _a | 51.6 | (1.2) | 24.3 _a |
| Former user | 11 | 18.2 | (12.2) | Ref. _{a,b} | 9.1 | (9.1) | 3.9 _a | 27.3 | (14.1) | Ref. _{a,b} |
| Current user | 249 | 28.1 | (2.9) | 9.9 _b | 5.2 | (1.4) | Ref. _a | 33.3 | (3.0) | 6.0 _b |

Differences between groups were assessed with z-test. Different subscript letters denotes significant differences in periodontitis prevalence between characteristics at the 0.05 level. Ref. = reference; pp = percentage points. USD = US dollars.

* Standardized to age distribution of the 2013 Troms County population.

† Average household income in Norway for 2013: 85,665 US dollars.

‡ Swedish type, low-nitrosamine, smokeless tobacco.

Prevalence and Severity of Periodontal Disease

The estimated prevalence and distribution of periodontitis by age and sex, as well as socioeconomic status, oral health-related behaviors, and tobacco use are presented in Table 3. According to the CDC/AAP case definition,¹¹ prevalence of total periodontitis was estimated to be 49.5% ± 1.1% (SE), with 9.1% severe periodontitis and 40.4% non-severe periodontitis (mild and moderate periodontitis combined). Prevalence of periodontitis increased with age; in the oldest age group, it was five times higher than in the youngest age group. Periodontitis was more prevalent among men (56.7%) than women (42.6%). When comparing prevalence of periodontitis in urban and rural municipalities, there was a higher prevalence in suburban and rural municipalities than in urban areas. In addition, prevalence increased with lower education and income and current smoking habit. Prevalence of severe periodontitis was highest in the 65- to 79-year age group and in current smokers (Table 3).

BOP and PI

Mean BOP was 30%, and this was consistent across age groups (Table 4). BOP increased with level of severity of periodontitis, with a mean of 25.4% for persons with no periodontitis, 33.2% for persons with non-severe periodontitis, and 41.7% for persons with severe periodontitis. Stratified by severity of periodontitis, BOP varied more across age groups. Mean PI was 44.2%, and this increased with severity of periodontitis and age.

DISCUSSION

Results suggest that half of adults in the target population had periodontitis; approximately four in 10 had non-severe periodontitis, and only one in 10

had severe periodontitis. That severity and extent of the disease increased with age was expected because periodontitis often is seen as a chronic disease and cumulative with time.³⁵ Prevalence of periodontitis was highest among people with lower education and a current smoking habit.

The majority of the study population was healthy, educated, and reported making regular dental visits and practicing good oral hygiene. Participants had a high educational level; 42% had university-level education compared with the nationwide percentage of 35%.³⁶ Educational level was highest in the largest municipality,²⁷ where the Arctic University of Norway and University Hospital of North Norway are located, contributing to the high number of persons with university-level education. The proportion of persons with university-level education in suburban and rural municipalities was equivalent to the national average.²⁷ Smoking and DM had the same rates as national averages and estimates, with 15% and 4%, respectively.^{37,38}

Periodontitis in Europe and the United States

Comparing the findings in this study with previous findings in Norway is not straightforward because different measures of periodontitis have been used. The prevalence of PD ≥6 mm (Table 2) was in the range of the results from the Oslo study in comparable age groups (8%),¹⁸ whereas prevalence of BL was considerably higher in the present study than among 35 year olds in Oslo in 2003 (24%). In the study of Norwegian elderly pensioners (≥67 years), prevalence of at least one tooth with PD ≥6 mm was reported to be 33%,¹⁹ consistent with results in the current study for the same age group. Conversely, prevalence of severe periodontitis was only half of

Table 4.**BOP and Plaque Score by Severity of Periodontitis Stratified by Age Group and in Total**

| Measures of Gingival Inflammation and Dental Plaque | Age Group (years) | | | | | Total Mean (SD) |
|---|-----------------------|-----------------------|-----------------------|-----------------------|-----------------------|--------------------|
| | 20 to 34 Mean (SD) | 35 to 44 Mean (SD) | 45 to 54 Mean (SD) | 55 to 64 Mean (SD) | 65 to 79 Mean (SD) | |
| Mean BOP (%) | 31.9 (18.3) | 27.2 (16.2) | 28.6 (16.7) | 30.3 (20.1) | 32.4 (21.6) | 30.0 (18.6) |
| No periodontitis | 29.4 (16.6) | 23.2 (13.9) | 22.7 (13.3) | 20.6 (13.9) | 23.2 (21.1) | 25.4 (15.7) |
| Non-severe periodontitis | 45.0 (21.2) | 34.8 (17.3) | 32.0 (17.4) | 31.2 (20.1) | 31.0 (20.1) | 33.2 (18.8) |
| Severe periodontitis | — | 30.8 (25.0) | 43.7 (16.4) | 40.5 (21.1) | 43.0 (21.5) | 41.7 (20.6) |
| Total periodontitis | 44.8 (21.1) | 34.6 (17.6) | 33.6 (17.7) | 33.4 (20.7) | 34.5 (21.2) | 34.8 (20.0) |
| Mean plaque score (%) | 45.2 (22.9) | 40.0 (21.0) | 42.6 (21.5) | 44.3 (22.4) | 49.7 (23.4) | 44.2 (22.4) |
| No periodontitis | 43.4 (22.5) | 38.4 (21.2) | 39.0 (19.9) | 38.7 (21.2) | 45.7 (25.3) | 40.9 (21.8) |
| Non-severe periodontitis | 54.5 (22.7) | 43.7 (20.3) | 44.6 (22.2) | 45.2 (21.6) | 47.5 (22.1) | 46.2 (21.9) |
| Severe periodontitis | — | 31.3 (19.0) | 51.6 (23.3) | 49.1 (25.0) | 58.2 (23.1) | 52.7 (24.2) |
| Total periodontitis | 54.6 (22.6) | 43.1 (20.3) | 45.6 (22.4) | 46.1 (22.5) | 50.6 (22.9) | 47.4 (22.5) |

Periodontitis categories according to the CDC/AAP case definitions.¹¹

— = no data available.

what was found in the current study. One explanation could be the case definition used for severe periodontitis (at least three periodontal pockets ≥ 6 mm) by Norderyd et al.¹⁹ and possibly the partial-mouth recording (one site per tooth), which could have provided biased estimates of periodontitis.³⁹⁻⁴¹

The present findings were comparable with prevalence reported in a Swedish study: 39% for periodontitis of any severity and 11% for severe periodontitis.² However, a different case definition for periodontitis was used, and direct comparison should be made with caution. In comparison with studies applying the CDC/AAP case definition, prevalence of periodontitis in this study concurred with prevalence reported for United States adults: 46% for periodontitis of any severity and 9% for severe periodontitis.⁵ Prevalence reported in German and Italian studies evaluating periodontitis with the CDC/AAP case definition was considerably higher. For German adults (aged 35 to 44 years), prevalence of periodontitis and severe periodontitis was reported to be 71% and 17%, respectively.⁴ For adults aged 20 to 75 years in Northern Italy, estimated prevalences of periodontitis and severe periodontitis were 76% and 35%, respectively.⁶ Discrepancies between studies could partially be explained by differences in the underlying characteristics of the study populations. There was a larger proportion of current and former smokers in the German and Italian studies compared with the present study,^{4,6} and proportions of people with middle and high levels of education were greater in the present study compared with other countries.

Differences in Periodontal Health

The present study showed differences in the population regarding the distribution of periodontitis, which was in accordance with other reported data.^{4,5,42}

These discrepancies could be explained by differences in oral health-related behavior, access to dental health care, and norms for seeking treatment. However, in bivariate analysis, persons making annual dental visits did not have less periodontitis than persons with less frequent dental visits; rather, it was the opposite. This could be a result of neglected important aspects of prevention dentistry, undertreatment or underdiagnosis, or that people categorized with yearly dental visits also include those undergoing treatment (e.g., periodontal treatment).

The most notable differences in periodontitis were across age groups, with $>80\%$ of persons aged ≥ 65 years affected (Table 4). Although a large number of seniors had periodontitis, the burden of PD ≥ 4 mm was not equally distributed in the population (Fig. 2). A small proportion of the senior population accounted for the majority of PD ≥ 4 mm, meaning that there was a subgroup of seniors with more extensive periodontitis. The number of natural teeth in seniors is increasing.⁴³ Based on an estimate that every fifth person in Norway will be at least 70 years old in 2060,⁴⁴ it is important for dental health care services to be capable of detecting individuals with periodontitis at an early stage and for preventive measures to be implemented. Clinicians should be trained in and adopt methods that have been reported to be effective in improving oral health-related behaviors,⁴⁵⁻⁴⁷ and the role of dental hygienists and dentists with special knowledge of prevention and oral health promotion should be emphasized in all parts of the country.

Strengths and Limitations

There are some limitations to note. Only slightly more than half (57.3%) of adults ≥ 65 years old responded, which could have caused biased results for

this age group. The most common reasons for not participating were health issues in combination with travel difficulties and no subjective need or interest in participating (e.g., wearing dentures).²⁷ Additionally, more men than women ≥ 65 years old participated, which might have resulted in overestimation of periodontitis prevalence for men.

The indirect approach to CAL by predicting CAL from BL could have led to errors in case definitions and possible underestimation of periodontitis. Variance in the ability of BL to predict CAL increased with increasing values of CAL. However, use of threshold values of CAL ≥ 4 and ≥ 6 mm to define cases minimized errors of high measures of CAL. Finally, geographic disparities, including the low periodontist-to-patient ratio in northern Norway, different living conditions, culture, and attitudes toward health, should be considered when extrapolating estimates to other regions and countries.

The study also has several strengths, including the high participation rate and full-mouth examination protocol. Furthermore, to the best knowledge of the authors, this was the first study to apply the recommended standards for reporting CP, enabling future comparisons across studies and contributing to the understanding of the global burden of periodontitis.

CONCLUSIONS

This study reveals a high burden of periodontitis in circumpolar communities in Norway, with half of the adult population affected. Sociodemographic disparities regarding periodontitis were shown, highlighting the importance of further investigation of factors influencing periodontal health. Results from this study contribute new knowledge and will be valuable in planning dental health care and population-based preventive actions.

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Paper II

Periodontitis and quality of life: What is the role of socioeconomic status, sense of coherence, dental service use and oral health practices? An exploratory theory-guided analysis on a Norwegian population

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Abstract

Aim: To utilise Andersen's behavioural model for health services' use as the theoretical framework to examine direct and indirect relationships between population characteristics, oral health behaviours and periodontitis and oral health impacts.

Materials and methods: The model was tested in a general adult population ($n = 1,886$) in Norway, using structural equation modelling. Socioeconomic status, sense of coherence (SOC), dental anxiety, perceived treatment need, oral health behaviours and oral health impact profile (OHIP-14) were collected through questionnaire. Periodontal examinations consisted of full-mouth recordings.

Results: Andersen's model explained a large part of the variance in use of dental services (58%) and oral health-related impacts (55%), and to a less extent periodontitis (19%). More social structure and stronger SOC was related to more enabling resources, which in turn was associated with more use of dental services. More use of dental services was related to more periodontitis and more periodontitis was associated with increased oral health impacts. A stronger SOC was associated with less oral impacts. There was no association between use of dental services and oral health impacts.

Conclusions: The result demonstrated complex relationships between population characteristics, oral health-related behaviours and oral health outcomes. Socioeconomic factors and smoking were main predictors of periodontitis. Regular dental visiting habits did not, however, reduce the likelihood of periodontitis.

KEYWORDS

Andersen's behavioural model for health services' use, chronic periodontitis, dental anxiety, dental health services, health behaviour, health-related quality of life, oral health, sense of coherence, socioeconomic factors

1 | INTRODUCTION

Periodontitis is a common disease amongst adults with a prevalence reported by European and US studies ranging from 31% to 76% (Aimetti et al., 2015; Bernabe & Marcenes, 2010; Eke et al.,

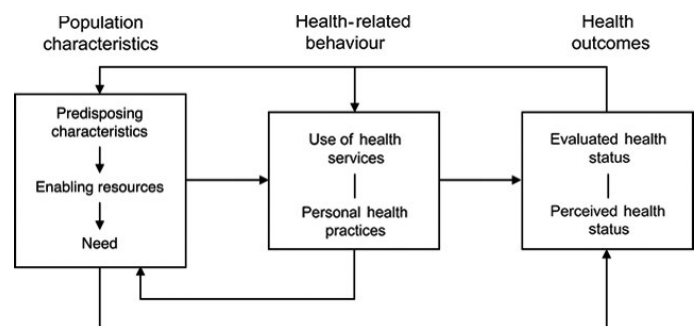
2015; Holde, Oscarson, Trovik, Tillberg, & Jonsson, 2017; Holtfreter, Kocher, Hoffmann, Desvarieux, & Micheelis, 2010; Hugoson, Sjodin, & Norderyd, 2008). Severe forms of the disease affect around 11% of the global population (Kassebaum et al., 2014). To be able to develop preventive strategies for periodontal disease it is important

to understand characteristics associated with periodontitis. Several risk factors such as age, gender, socioeconomic status (Genco & Borgnakke, 2013), smoking (Calsina, Ramon, & Echeverria, 2002), and oral hygiene habits (Zimmermann et al., 2015) have been associated with the progression and severity of periodontitis.

As periodontitis is a complex disease with biological, behavioural and social risk factors, it is important not only to examine the individual influence of each factor but also to examine the periodontal risk network as a whole. To be able to do this, there is a need for a conceptual model to underpin the research and, alongside this, a more comprehensive statistical analysis. At present, one of the ways to explore the interrelationship between several contributing factors simultaneously is to utilise theoretically driven structural equation modelling (SEM). SEM is a powerful statistical technique that allows simultaneous testing of complex direct and indirect (mediated) relationships between variables specified within a priori model (Kline, 2015). So far, studies using SEM in relation to periodontitis have examined the relationship between psychological factors and periodontal health (Alkan, Cakmak, Yilmaz, Cebi, & Gurgan, 2015), impact of psychological factors on the relationship between periodontal status and quality of life (Wright et al., 2017), gingivitis and the interaction of oral health-related behaviours (Furuta et al., 2011), or the relationship between periodontitis and specific systemic diseases (Fisher, Taylor, West, & McCarthy, 2011; Rebelo, de Castro, Rebelo Vieira, Robinson, & Vettore, 2016). No study to date has focused on determinants of oral health care practices and use of dental health services and their relationship with periodontitis and oral health-related quality of life.

Andersen's behavioural model of health services' use (Andersen, 1968, 1995) has been used as the conceptual framework in several studies of health care utilisation. It was originally developed to predict and explain why and how people use health care services by integrating predisposing/social structural factors (e.g. income, education, physical environment), enabling resources (e.g. having the means to use available health services) and need for health care (e.g. how people view their need for care). These different population characteristics would, according to the model, help understand why some people are more likely to seek health care. As such, the model suggests that that different factors would be of differential importance depending on the seriousness of the health problem. The model has, during the last three decades, been further extended and developed adding personal health practices and health outcomes/status (Andersen, 1995) (Figure 1).

FIGURE 1 Model of health services' use and health outcomes based on Andersen's behavioural model (1995)



Clinical Relevance

Scientific rationale for the study: To examine how population characteristics are related to oral health behaviour, and how this in turn, is related to periodontitis and oral health impacts.

Principal findings: Self-perceived resources were a key determinant of use of dental services. Regular dental visiting habits did not reduce the likelihood of having periodontitis.

Practical implications: The results contradicted the assumption that regular and prevention-oriented dental attendance should prevent or control periodontitis.

The extended Andersen behavioural model for health services' use (1995) has been tested in relation to dental care and oral health outcomes in two different general populations in the UK (Baker, 2009; Marshman et al., 2012). The results were in line with Andersen's model, however, the authors concluded that other important factors needed to be incorporated within the model to increase its usefulness for understanding dental access and oral health outcomes. Such factors include the cost of treatment as well as key psychosocial factors previously identified as important for oral health and quality of life (e.g. sense of coherence, dental attitudes). Sense of coherence (SOC) is a salutogenic concept and "a specific way of viewing life as comprehensible, manageable and meaningful" (Antonovsky, 1987). It has been found to be important for adults' oral health in several recent studies including toothbrushing habits, eating fruit and vegetables, dental attendance, and oral health-related quality of life (Elyasi et al., 2015; Gupta, Robinson, Marya, & Baker, 2015; Savolainen et al., 2005).

The aim of this study was to utilise Andersen's behavioural model for health services' use as the theoretical framework to explore the direct and indirect relationships between population characteristics, use of dental health care services, individuals' personal oral health practices, and periodontal health and self-reported oral health impacts. In addition, we incorporated within the model, SOC, in order to examine how it was related to adult's oral health and to other key factors determining individual's oral health. This exploratory model was tested in a general adult population with data from the

Tromstannen Oral Health in Northern Norway (TOHNN) study (Holde, Oscarson, Tillberg, Marstrander, & Jonsson, 2016) using structural equation modelling (SEM).

2 | MATERIALS AND METHODS

2.1 | Study design and participants

The TOHNN study was a cross-sectional study of adults 20–79 years old in Troms County, Norway. The randomized sample included 2,901 individuals. The estimated minimum sample size for the structural equation model with an effect size of 0.1, power of 0.8, five latent variables, 4 observed variables, and a probability set at 0.05 was 1,599 participants.

Data were collected between October 2013 and November 2014, with 1,986 participants (68%). The regional committee for medical and health research ethics of the University of Tromsø, Norway, approved the study (2013/348/REK Nord). All participants provided written informed consent.

2.2 | Selection of variables

Variables were chosen according to Andersen's behavioural model for health services' use (1995) and with reference to the two previous studies that had tested the model for oral health (Baker, 2009; Marshman et al., 2012). The latent and measured variables used in the analysis are summarised below. Detail of each construct, its operationalisation, measures including response options and scoring, can be seen in Supporting Information Table S1.

2.3 | Population characteristics

Predisposing characteristics were measured with two latent variables: social structures and sense of coherence. The three measured (indicator) variables for social structures were education, annual household income, and urbanisation. Education was divided into three categories (primary/middle school, high school and university level) and income four categories: (a) $\leq 300,000$ NOK, (b) 300,001–450,000 NOK, (c) 450,001–900,000 NOK, and (d) $>900,000$ NOK. Urbanisation was used as an indicator of number of inhabitants and availability of dentists as a ratio of inhabitants per dentist. The municipality with the larger town had the highest availability and was categorised as urban, two municipalities with smaller towns had the second highest availability and were categorised as suburban, and the remaining municipalities without towns had the lowest availability and were classified as rural. SOC was assessed with the Norwegian version (Eide, 1991) of Antonovsky's (1993) "The orientation to life questionnaire," comprising 13 items. The three indicator variables were represented by the three SOC dimensions: comprehensibility (five items); manageability (four items); and meaningfulness (four items).

Enabling resources was measured with three indicator variables: declined treatment due to costs, perceived difficulty accessing a

dentist (each assessed with one question), and dental anxiety (assessed with the Norwegian version of Corah's Dental Anxiety Scale (DAS) (Corah, Gale, & Illig, 1978; Kvale, Berg, & Raadal, 1998). For analysis, the DAS-score was reversed so higher scores represented less dental anxiety.

Treatment need was measured as an observed variable and assessed with one item: "If you saw a dentist tomorrow, do you think you would need treatment?" Response option was: yes, don't know or no.

2.4 | Oral health-related behaviours

Oral health-related behaviours were represented by personal health practices and use of dental services. Toothbrushing frequency was measured as one item. Smoking was measured by pack-years categorised as non-smoker (no pack-years), light smoker (<20 pack-years) and heavy smoker (≥ 20 pack-years). Use of dental services was measured as a latent variable with two indicators: attendance orientation (assessed with the question "For what reason do you seek dental services?") and frequency of attendance (assessed with the question "How often do you attend dental services?"). Response options are presented in Table 1.

2.5 | Oral health outcomes

Oral health outcomes included both clinical- and person-reported measures. The clinical measure was periodontitis. Clinical examinations were performed in a dental office by 11 calibrated dentists with assisting dental nurses. Bleeding on probing (BoP) and periodontal pocket depth (PD) were assessed at six sites per tooth for all teeth. Third molars and implants were excluded from the analysis. For a more comprehensive description of the periodontal assessment see Holde et al., 2016, 2017. Periodontitis was defined using case definitions developed by the Centers for Disease Control and Prevention and the American Academy of Periodontology (CDC/AAP) (Eke, Page, Wei, Thornton-Evans, & Genco, 2012; Eke et al., 2015). According to this definition, participants were classified with no, non-severe or severe periodontitis. Person-reported oral health was assessed with the Norwegian version of oral health impact profile (OHIP-14) (Dahl, Wang, Skau, & Ohrn, 2011; Slade, 1997), a measure of people's perceptions of the social impact of oral disorders on their well-being. Chronbach's alpha for OHIP-14 was 0.89. In line with similar SEM studies using the OHIP-14 (see Baker, 2009), person-reported oral health impacts were represented in the model as a latent variable with the three sub-scales—psychological, physical and social impacts—as the indicator variables. Responses to Items 1–2, 3–4, 5 and 10 were summed to represent physical function (range 3–15); Items 6–7 and 8–9 were summed to represent psychological function (range 2–10); Items 11–12 and 13–14 were summed to represent social function (range 2–10). Chronbach's alpha for physical function was 0.73, psychological function 0.89, and social function 0.88, respectively.

TABLE 1 Items from the THONN-questionnaire that reflects a revised Andersen's behavioural models different concepts and constructs. N = 1,819

| Variable | N/Mean | %/SD | Min-max |
|---|--------|------|---------|
| <i>Predisposing characteristics</i> | | | |
| Social structures (Latent variable) | | | |
| Education | | | |
| Primary/middle school | 247 | 13.6 | |
| High school | 803 | 44.1 | |
| University | 769 | 42.3 | |
| Income (household annually) | | | |
| <300,000 NOK | 250 | 13.7 | |
| 300,001–450,000 NOK | 589 | 32.4 | |
| 450,001–900,000 NOK | 620 | 34.1 | |
| >900,000 NOK | 360 | 19.8 | |
| Urbanisation (availability to dentists) | | | |
| Rural | 418 | 23.0 | |
| Suburban (small towns) | 567 | 31.2 | |
| Urban (lager town) | 834 | 45.8 | |
| Salutogenic factors (Latent variable) | | | |
| Sense of coherence (SOC) | 68.5 | 10.5 | 25–90 |
| Comprehensibility | 25.5 | 4.8 | 5–35 |
| Manageability | 20.9 | 3.8 | 4–28 |
| Meaningfulness | 22.1 | 3.6 | 8–28 |
| Enabling resources (Latent variable) | | | |
| Declined treatment due to costs | | | |
| Yes | 354 | 19.5 | |
| No | 1,465 | 80.5 | |
| Difficulty attending dental services | | | |
| Yes/Don't know | 317 | 17.4 | |
| No | 1,502 | 82.6 | |
| Dental anxiety scale | 7.7 | 3.3 | 4–20 |
| Need (observed variable) | | | |
| Perceived treatment need | | | |
| Would not need treatment | 465 | 25.6 | |
| Don't know | 695 | 38.2 | |
| Would need treatment | 659 | 36.2 | |
| <i>Oral health-related behaviour</i> | | | |
| Personal health practices | | | |
| Toothbrushing (observed variable) | | | |
| Less than daily | 68 | 3.7 | |
| Once per day | 468 | 24.1 | |
| Twice per day | 1,313 | 72.2 | |
| Smoking habits (observed variable) | | | |
| Non-smoker | 1,553 | 85.4 | |
| Light smoker (<20 pack-years) | 196 | 10.8 | |
| Heavy smoker (≥20 pack-years) | 70 | 3.8 | |

(Continues)

TABLE 1 (Continued)

| Variable | N/Mean | %/SD | Min-max |
|--|--------|------|---------|
| Use of dental services (latent variable) | | | |
| Attendance orientation | | | |
| Seldom/never attend DS | 282 | 15.5 | |
| Only when problem (pain, lost fillings) | 358 | 19.7 | |
| Having routine recall/check-up | 1,179 | 64.8 | |
| Frequency of dental attendance | | | |
| Only when having problems | 403 | 22.2 | |
| Longer intervals than 2 years | 202 | 11.1 | |
| Every second year | 244 | 13.4 | |
| Every year | 970 | 53.3 | |
| Oral health outcomes | | | |
| Clinical | | | |
| Periodontitis diagnosis ^a (observed variable) | | | |
| No periodontitis | 922 | 50.7 | |
| Non-severe periodontitis ^b | 734 | 40.3 | |
| Severe periodontitis | 163 | 9.0 | |
| Oral health impacts (person-reported) | | | |
| OHIP-14 (Latent variable) | 19.4 | 6.5 | 14–70 |
| OHIP physical | 8.7 | 2.9 | 6–30 |
| OHIP psychological | 6.0 | 2.9 | 4–20 |
| OHIP social | 4.7 | 1.8 | 4–20 |

Notes. DS: dental services.

^aCDC/AAP case definitions for reporting periodontitis in epidemiological studies; ^bMild and moderate periodontitis combined.

2.6 | Data analysis

Data were analysed using the IBM® SPSS® Statistics, version 24 and AMOS 24. For analysis, eligible individuals had to have complete periodontal recordings and two or more teeth in order to be diagnosed according to the CDC/AAP case definition for periodontitis. The classification was with the following definitions: (a) severe periodontitis: at least two interproximal sites with ≥ 6 mm clinical attachment loss (CAL) (not on the same tooth) and at least one interproximal site(s) with ≥ 5 mm PD; (b) moderate periodontitis: at least two interproximal sites with ≥ 4 mm CAL (not on the same tooth) or at least two interproximal sites with PD ≥ 5 mm (not on the same tooth); and (c) mild periodontitis: at least two interproximal sites with ≥ 3 mm CAL and at least two interproximal sites with ≥ 4 mm PD (not on the same tooth) or one interproximal site with ≥ 5 mm PD.

Missing data occurred at very low frequency (0%–3.9%) except for one item in the OHIP-14 instrument (5.8%). An analysis of missing data pattern, computed by SPSS, showed that the missing values appeared to be missing at random. For all one-item variables, missing values were replaced with the median. When calculating SOC scores, individuals with more than three missing items were excluded from analysis. If three or fewer items were missing they were replaced by the median value of the remaining SOC items for that individual (Kanhai et al., 2014). For OHIP summary scores, individuals with more than two missing OHIP-items were excluded from

analysis. When two or less items were missing, they were replaced with the sample median of the relevant OHIP-item (Slade et al., 2005). Individuals with more than one missing item in the DAS-scale were excluded from analysis. Where one item was missing, it was replaced with the median value of the remaining DAS items for that individual. Re-analysis of data excluding individuals with any missing items did not change mean scores by more than one decimal place or frequency distributions by more than one percentage point, except for income that changed 2.4 percentage points (not reported). The excluded individuals did not differ significantly in any of the key outcomes (periodontitis and oral health impacts) compared to those that were kept in the analysis.

In order to identify whether the indicators chosen to measure the five latent constructs were acceptable, confirmatory factor analysis was used (CFA). CFA is the first in the two-stage process of SEM (the measurement model) (Kline, 2015). CFA provides information on how indicator items (e.g. income) measure underlying (latent) constructs (e.g. social structures). The initial step of the analysis was to test a first order CFA with social structures, SOC, enabling resources, use of dental services and oral health impacts (OHIP-14) as the five latent constructs. Scale items (indicators) representing each of the five latent constructs are detailed in Table 1 (see also Figure 2). Items were not allowed to load on more than one construct nor were error terms allowed to correlate, with the exception of the three domains of the SOC construct (Figure 2).

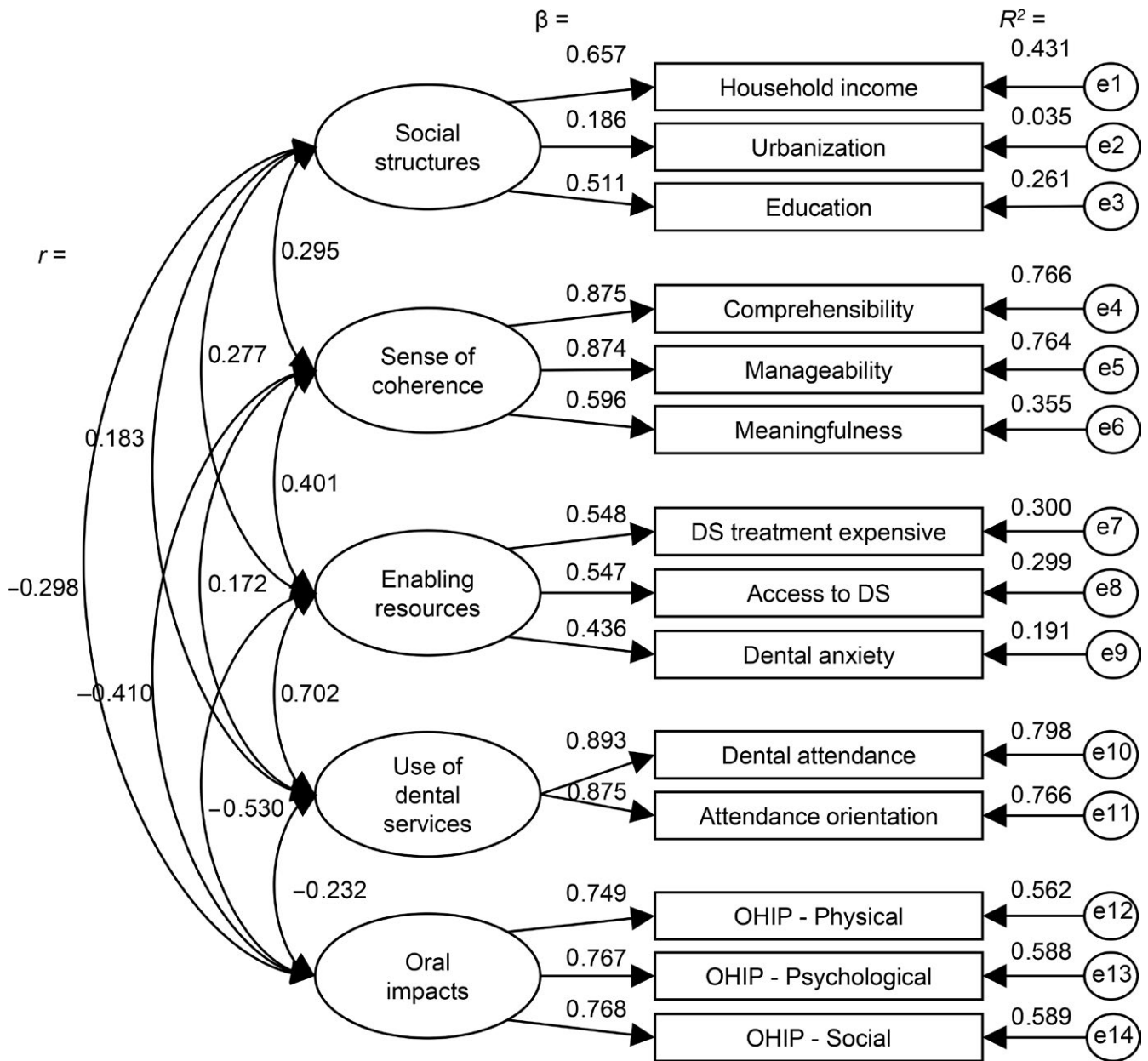


FIGURE 2 Bootstrapped ML standardized estimates for the confirmatory factor analysis. All figures $p < 0.01$

Following specification of the measurement model, the next step in the analysis was to test a structural model which examined the direct and indirect relationships between the constructs as hypothesised in our revised Andersen’s behavioural model for health services’ use. In accordance with the model and with SOC as an additional predisposing factor based on findings from Gupta et al. (2015), 24 direct pathways were hypothesised. Population characteristics: social structures and SOC (higher scores) would predict more enabling resources. Enabling resources would in turn predict patients’ perceived treatment need. More enabling resources would relate to less perceived treatment need. Social structure, SOC, enabling and treatment need would predict use of dental services, where more social structure, greater SOC, more enabling resources and less treatment need would relate to more use of dental services. Social

structures, SOC, enabling resources, treatment need and use of dental services would predict periodontal health, which in turn would predict oral impacts, with more severe periodontitis relating to more oral impacts. In addition, social structure and SOC would directly predict use of dental services, personal oral health practices (toothbrushing and smoking), periodontitis, and oral impacts. Use of dental services would predict personal oral health practices and oral impacts. At last, personal oral health practices would predict periodontitis. The full model can be seen in Supporting Information Figure S1.

AMOS estimates the total effects, which are made up of both direct effects (a path directly from one variable to another, for example social structures → enabling) and indirect effects (a path mediated through other variables, for example social structures → need via enabling resources). Because of the presence of both non-normal and categorical

data, the model was estimated using bootstrapping wherein multiple samples ($n = 900+$) are randomly drawn from the original sample. The CFA model is then estimated in each dataset, and the results averaged. The ML bootstrap estimates and standard errors (together with bias-corrected 95% confidence intervals [CIs]) are then compared with the results from the original sample to examine stability of parameters and test statistics (Brown, 2006). Proportions of total effects (%) were calculated for direct and indirect effects. In cases where the direct and indirect effects had opposing directions, the proportion of the total effect could not be calculated because of suppression effect.

As recommended, model fit was evaluated using a range of indices from three fit classes; absolute, parsimony adjusted and comparative (Brown, 2006; Hu & Bentler, 1999). A χ^2/df ratio of <3.0 , RMSEA values <0.06 , CFI and TLI ≥ 0.9 and an SRMR <0.08 were taken to indicate an acceptable model fit (Hu & Bentler, 1999).

3 | RESULTS

In the final analysis, 1,819 of 1,986 participants were included (923 women, mean age 47.1 ± 15.2 years). Forty-nine per cent ($n = 897$) of participants had periodontitis, of which 9.0% ($n = 163$) had severe

periodontitis. Proportions, mean values and range for each variable used in the model are presented in Table 1.

The measurement model was an acceptable fit on four of the five a priori indices (see Table 2, Model 1). The standardised estimates for this five-factor measurement model can be seen in Figure 2. Factors (latent variables) are in ellipses, items (indicator variables) are in rectangles and residual error terms in circles. All item loadings were significant (<0.001) and in the expected direction. The correlations between the five latent factors ranged between -0.53 and 0.71 , indicating that they had acceptable discriminant validity (i.e. <0.85).

The structural model was an acceptable fit to the data meeting four of the five a priori criteria (Table 2, Model 2). Within this model, eighteen paths were significant (Figure 3). In this model, 55%, 28%, 58%, 19% and 55% of the bootstrapped variance was accounted for in enabling resources, need, use of dental services, periodontitis and oral health impacts, respectively.

The direct effects are presented in Table 3. More of the social structures (greater income, higher educational level and urbanisation) ($\beta = 0.17$) and a stronger SOC ($\beta = 0.72$) was linked to more enabling resources. More enabling resources was, in turn, linked to lower perceived treatment need ($\beta = -0.53$) and more use of dental services ($\beta = 0.99$). Higher self-reported treatment need was related to more severe

TABLE 2 Fit indices for the measurement and structural models

| Model | χ^2/df | p | RMSEA | (90% CI) | CFI | TLI | SRMR | Criteria fitted |
|-------|-------------|-------|--------------|---------------|--------------|--------------|--------------|-----------------|
| 1 | 4.938 | 0.000 | 0.047 | (0.042–0.052) | 0.966 | 0.953 | 0.037 | 4 |
| 2 | 4.948 | 0.000 | 0.047 | (0.043–0.051) | 0.949 | 0.931 | 0.050 | 4 |

Notes. Figures in bold are those that meet the a priori model fitting criteria.

CFI: comparative fit index; CI: confidence interval; df: degrees of freedom; Model 1: measurement model; Model 2: periodontal structural model; RMSEA: root mean square error of approximation; SRMR: standardised root mean square residual; TLI: Tucker-Lewis Index; χ^2 : chi-square.

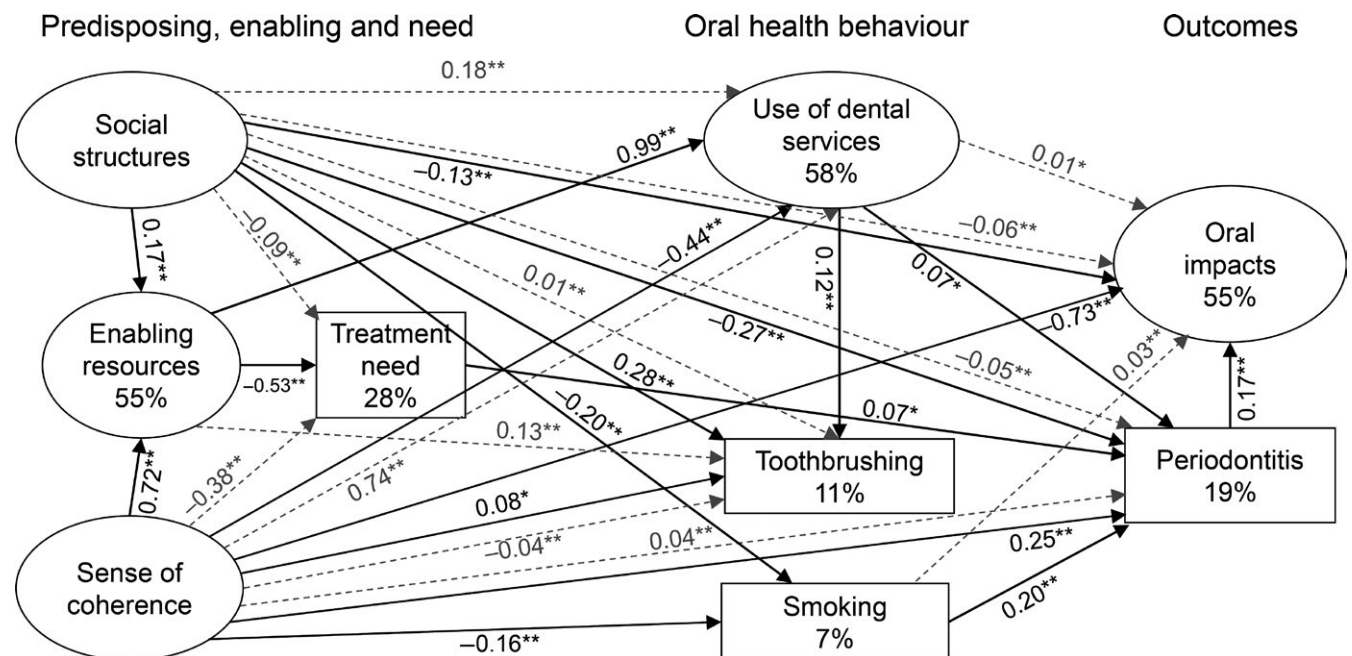


FIGURE 3 Bootstrapped standardised estimates for the revised Andersen's behavioural model for health services' use. Solid lines = direct effect; dashed lines = indirect effect. * $p < 0.05$, ** $p < 0.01$

TABLE 3 Direct and indirect effects for the Andersen's model (20–79 years old)

| Effect | β | Bootstrap SE | Bias-corrected 95% CI | p | % of total effect |
|----------------------------------|---------|--------------|--------------------------|-------|-------------------|
| Direct effects | | | | | |
| Social structures—enabling | 0.173 | 0.044 | 0.090/0.266 | 0.001 | 100 |
| SOC—enabling | 0.718 | 0.056 | 0.614/0.831 | 0.002 | 100 |
| Enabling—treatment need | -0.528 | 0.023 | -0.578/-0.483 | 0.001 | 100 |
| Treatment need—use of DS | -0.065 | 0.036 | -0.132/0.012 | 0.107 | 100 |
| Use of DS—toothbrushing | 0.122 | 0.030 | 0.070/0.187 | 0.001 | 100 |
| Use of DS—smoking | -0.025 | 0.032 | -0.086/0.038 | 0.473 | 100 |
| Use of DS—periodontitis | 0.074 | 0.032 | 0.010/0.136 | 0.025 | - ^a |
| Use of DS—oral impacts | 0.011 | 0.050 | -0.080/0.116 | 0.790 | 50 |
| Toothbrushing—periodontitis | -0.025 | 0.026 | -0.077/0.025 | 0.324 | 100 |
| Periodontitis—oral impacts | 0.169 | 0.042 | 0.095/0.263 | 0.001 | 100 |
| Social structures—use of DS | -0.062 | 0.048 | -0.154/0.024 | 0.153 | - ^a |
| Social structures—toothbrushing | 0.277 | 0.033 | 0.219/0.340 | 0.002 | 95 |
| Social structures—smoking | -0.198 | 0.031 | -0.257/-0.138 | 0.002 | 99 |
| Social structures—periodontitis | -0.273 | 0.035 | -0.342/-0.202 | 0.003 | 86 |
| Social structures—oral impacts | -0.126 | 0.040 | -0.208/-0.049 | 0.001 | 69 |
| SOC—use of DS | -0.436 | 0.163 | -0.799/-0.231 | 0.002 | - ^a |
| SOC—toothbrushing | 0.085 | 0.042 | 0.002/0.164 | 0.046 | 70 |
| SOC—smoking | -0.156 | 0.042 | -0.241/-0.079 | 0.002 | 96 |
| SOC—periodontitis | 0.246 | 0.048 | 0.160/0.342 | 0.002 | - ^a |
| SOC—oral impacts | -0.726 | 0.057 | -0.835/-0.618 | 0.002 | - ^a |
| Enabling—use of DS | 0.990 | 0.167 | 0.782/1.390 | 0.002 | 97 |
| Treatment need—periodontitis | 0.072 | 0.028 | 0.014/0.125 | 0.014 | - ^a |
| Treatment need—oral impacts | 0.032 | 0.038 | -0.053/0.103 | 0.445 | 74 |
| Smoking—periodontitis | 0.198 | 0.024 | 0.151/0.243 | 0.002 | 100 |
| Indirect effects | | | | | |
| Social structures—treatment need | -0.091 | 0.024 | -0.145/-0.049 | 0.001 | 100 |
| Social structures—use of DS | 0.177 | 0.053 | 0.086/0.300 | 0.002 | - ^a |
| Social structures—toothbrushing | 0.014 | 0.006 | 0.006/0.028 | 0.001 | 5 |
| Social structures—smoking | -0.003 | 0.004 | -0.012/0.003 | 0.324 | 1 |
| Social structures—periodontitis | -0.045 | 0.012 | -0.068/-0.023 | 0.002 | 14 |
| Social structures—oral impacts | -0.056 | 0.016 | -0.094/-0.030 | 0.001 | 31 |
| SOC—treatment need | -0.379 | 0.035 | -0.456/-0.318 | 0.001 | 100 |
| SOC—use of DS | 0.736 | 0.173 | 0.530/1.175 | 0.001 | - ^a |
| SOC—toothbrushing | 0.037 | 0.011 | 0.019/0.064 | 0.001 | 30 |
| SOC—smoking | -0.007 | 0.010 | -0.026/0.012 | 0.410 | 4 |
| SOC—periodontitis | -0.040 | 0.017 | -0.079/-0.012 | 0.009 | - ^a |
| SOC—oral impacts | 0.026 | 0.028 | -0.018/0.086 | 0.309 | - ^a |
| Enabling—use of DS | 0.034 | 0.019 | -0.006/0.067 | 0.101 | 3 |
| Enabling—smoking | -0.026 | 0.034 | -0.090/0.042 | 0.457 | 100 |
| Enabling—toothbrushing | 0.125 | 0.039 | 0.069/0.214 | 0.001 | 100 |
| Enabling—periodontitis | 0.029 | 0.035 | -0.041/0.091 | 0.429 | 100 |

(Continues)

TABLE 3 (Continued)

| Effect | β | Bootstrap SE | Bias-corrected 95% CI | <i>p</i> | % of total effect |
|------------------------------|---------|--------------|-----------------------|----------|-------------------|
| Enabling—oral impacts | -0.001 | 0.055 | -0.095/0.122 | 0.986 | 100 |
| Treatment need—smoking | 0.002 | 0.003 | -0.002/0.009 | 0.323 | 100 |
| Treatment need—toothbrushing | -0.008 | 0.005 | -0.021/0.000 | 0.063 | 100 |
| Treatment need—periodontitis | -0.004 | 0.003 | -0.013/0.000 | 0.081 | — ^a |
| Treatment need—oral impacts | 0.011 | 0.007 | -0.002/0.027 | 0.101 | 26 |
| Use of DS—periodontitis | -0.008 | 0.007 | -0.021/0.006 | 0.266 | — ^a |
| Use of DS—oral impacts | 0.011 | 0.006 | 0.002/0.024 | 0.025 | 50 |
| Toothbrushing—oral impacts | -0.004 | 0.005 | -0.015/0.004 | 0.287 | 100 |
| Smoking—oral impacts | 0.033 | 0.009 | 0.017/0.055 | 0.001 | 100 |

Notes. CI: confidence interval; DS: dental services; SE: standard error; β : bootstrapped standardised estimate.

^aCould not be calculated because of suppression effect.

periodontitis ($\beta = 0.07$). More use of dental services was related to more frequent toothbrushing ($\beta = 0.12$) and more periodontitis ($\beta = 0.07$). More severe periodontitis was linked to increased patient-reported oral health impacts ($\beta = 0.17$). In addition, more of the social structures were associated with more frequent toothbrushing ($\beta = 0.28$), less likelihood of smoking ($\beta = -0.20$), less periodontitis ($\beta = -0.27$), and less oral health impacts ($\beta = -0.13$). A stronger SOC was associated with less use of dental services ($\beta = -0.44$), more frequent toothbrushing ($\beta = 0.09$), less likelihood of smoking ($\beta = -0.16$), more periodontitis ($\beta = 0.25$) and less oral health impacts ($\beta = -0.73$). At last, more smoking was linked to more severe periodontitis ($\beta = 0.20$). The following five direct effects were not significant; treatment need-use of services, use of services—oral health impacts, toothbrushing-periodontitis, social structure-use of services, treatment need-oral health impacts.

There were twelve significant indirect paths (Table 3). More social structures was linked to lower perceived treatment need ($\beta = -0.09$), more use of dental services ($\beta = 0.18$), more frequent toothbrushing ($\beta = 0.01$), less severe periodontitis ($\beta = -0.05$) and less oral health impacts ($\beta = -0.06$). Stronger SOC was related to less perceived treatment need ($\beta = -0.38$), more use of dental services ($\beta = 0.74$), more frequent toothbrushing ($\beta = 0.04$) and less severe periodontitis ($\beta = -0.04$). More enabling resources was linked to more toothbrushing ($\beta = 0.13$). More use of dental services was associated with increased oral health impacts ($\beta = 0.01$). More smoking was associated with increased oral health impacts ($\beta = 0.03$). These are total indirect paths, which comprise of separate indirect effects. Some paths consist of one potential effect (e.g. stronger SOC is linked to less perceived need via enabling resources), whilst some indirect paths can consist of multitude potential effects (e.g. social structures may be linked to less oral health impacts via more enabling resources, less perceived treatment need and less periodontitis).

4 | DISCUSSION

The model explained a large amount of the variance in both use of dental services and oral health impacts, supporting use of Andersen's

behavioural model for health services' use for explaining factors related to oral health. Enabling resources were found to be a key factor in predicting use of dental services. Absence of dental anxiety, not having declined treatment due to costs and no perceived difficulty accessing a dentist increased the likelihood of regular dental visits. Social structures only affected use of dental health services via enabling resources. A stronger SOC was directly linked to less likelihood of using dental services. However, for the indirect effect, when the inter-relationships between all variables in the model are considered, the association between SOC and use of dental services changed direction. When mediated through enabling resources a stronger SOC was related to more use of dental services. Enabling resources also influenced perceived treatment need, where individuals with dental anxiety and perceived difficulty accessing a dentist were more likely to report a higher treatment need. Self-reported treatment need was not, however, significantly associated with use of dental services, as reported by both Baker (2009) and Marshman et al. (2012) as the main predictor of oral health behaviour. A study of dental attendance amongst adult Finns also found perceived need for care to be a predictor of use of dental services in logistic regression analysis (Raittio, Kiiskinen, Helminen, Aromaa, & Suominen, 2014). Frequency of participants with regular dental visiting habits was similar to reports from the other studies. Perceived treatment need was, on the other hand, notably higher in the current study where only one in four reported no need for treatment. For the current study population, use of dental services seem to be influenced by other factors than perceived need that is enabling resources—directly and as a mediator for predisposing characteristics.

Social structures (education, income and urbanisation) and SOC were important factors in predicting both clinically measured and self-reported oral health outcomes. Higher education, income and availability of dentists decreased the likelihood of periodontitis. This is supported by the literature where socioeconomic factors have been related to periodontitis (Borrell & Crawford, 2012; Eke et al., 2016; Petersen & Ogawa, 2012). A stronger SOC was, interestingly, related to worse periodontal status. In previous studies of SOC and periodontitis, SOC has been related to self-perceived periodontal

disease (Cyrino, Costa, Cortelli, Cortelli, & Cota, 2016) but no relationship has been reported between SOC and clinical measures of periodontitis (Kanhai et al., 2014). As SOC is a psychological concept of how a person views their own life, it is plausible that it affects the way individuals perceive their own health, independent of their clinically measured health. It should also be considered that both SOC and periodontitis are positively correlated to age, which is not included in the model, and could be a potential mediator of the association between SOC and periodontitis.

Having higher education, income and availability to dentists was also associated with less oral health impacts. This was in contrast to findings by Baker (2009) where there was no direct association between socioeconomic status and self-reported oral health outcomes. Other studies of socioeconomic factors and subjective oral health support the current findings, reporting socioeconomic inequalities in oral health-related impacts (Guarnizo-Herreno et al., 2014; Raittio et al., 2015). SOC was the main predictive factor for oral health-related impacts, where a stronger sense of coherence decreased the likelihood of having oral health impacts. This is in line with results from previous studies (Gupta et al., 2015; Savolainen et al., 2005). Self-reported treatment need and use of dental services had no direct effect on oral health impacts. This is again in contrast to findings by Baker (2009) and Marshman et al. (2012). Routine dental attendance was reported to have a protective effect on oral health-related quality of life in other studies (Almoznino et al., 2015).

It is interesting that the use of dental services was related to a higher likelihood of having periodontitis. This result is in contrast to the assumption that regular and prevention-oriented dental attendance should prevent or control periodontitis. Also, in bivariate analysis, persons with yearly dental visits and persons only seeing a dentist for acute problems did not differ in regards to prevalence of both non-severe and severe periodontitis (Holde et al., 2017), further contradicting this assumption. Here, the sample was cross-sectional. Thus, whilst the data were modelled based on the causal ordering hypothesised within Andersen's model, such ordering does not imply a causal effect (Holland, 1988). In Andersen's revised model, many of the key relationships are hypothesised as being bi-directional; for example, seeking treatment (dental service use) may influence clinical outcomes but also vice versa. That is, persons diagnosed with periodontitis would have more frequent dental visits when undergoing periodontal treatment and maintenance. Further, successful control of initiation and progression of periodontitis is dependent both on patient cooperation in plaque control and provision of appropriate interventions and treatment by the dental practitioner. A study of US males from 1994 found that utilisation of dental services was not predictive of the extent and severity of periodontitis (Brown & Garcia, 1994). The same was reported for Swedish older adults, where regular dental visitors retained more teeth but had the same periodontal conditions as infrequent visitors (Renvert, Persson, & Persson, 2011). This questions the effectiveness of utilisation of dental services in relation to periodontitis prevention and control, and could be an indication of under-diagnosis or under-treatment of periodontal disease. However, to investigate this

in more detail, more information would be required about participant's dental history and treatment. In addition, the testing of such reciprocal relationships needs to be incorporated into a longitudinal validation study. Such a study would allow cross-validation of the present model and possible alternative explanations such as those proposed above.

4.1 | Strengths and limitations

Study limitations include the cross-sectional study design. As all variables were measured at the same point in time, the present analysis does not attempt to identify cause and effect relationships but rather was an exploratory theory-driven analysis which aimed to examine the complex relationship between several contributing factors. It would be useful, however, in future observational studies to utilise newer statistical tools such as marginal structural models, which allow for estimation of direct effects using a counterfactual scenario and which allow more assumptions about causality to be made (Robins, Hernan, & Brumback, 2000; VanderWeele, 2012). It would also be interesting in future research to examine in more detail, and longitudinally, the mechanisms by which SOC may influence oral health behaviours (e.g. smoking) and, in turn, both clinical and person-centred oral health outcomes.

Whilst the level of periodontitis in the current sample was comparable to those reported in European and US studies (Aimetti et al., 2015; Bernabe & Marcenes, 2010; Eke et al., 2015; Holtfreter et al., 2010; Hugoson & Koch, 2008), findings regarding use of dental services should be cautiously extrapolated to other regions and countries, as the structure of dental services might differ.

The study also has several strengths. This is the first study to test Andersen's behavioural model for health services' use with periodontitis as an outcome and to incorporate SOC within the model to examine its relationship to oral health and its determinants. Furthermore, the results validate previous findings regarding utilisation of dental services and periodontitis by including multiple determinants rather than one or two as in previous studies, but also by assessing these using complex statistical methods that allow for testing of not just direct effects but also indirect effects. Thereby, giving information on, not only, *what* variables are related but also *how* they are related.

5 | CONCLUSIONS

The present study highlights the complex relationships between population characteristics, oral health-related behaviours and oral health outcomes. Enabling resources was found to be a key determinant in the use of dental services. Socioeconomic factors and smoking were main predictors of periodontitis. Regular dental visiting habits did not, however, reduce the likelihood of periodontitis. There is a need for more knowledge about the effectiveness of dental health care utilisation related to periodontitis prevention and control.

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CONFLICT OF INTEREST

The authors have stated explicitly that there is no conflict of interest in connection with this article.

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SUPPORTING INFORMATION

Additional supporting information may be found online in the Supporting Information section at the end of the article.

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SUPPORTING INFORMATION

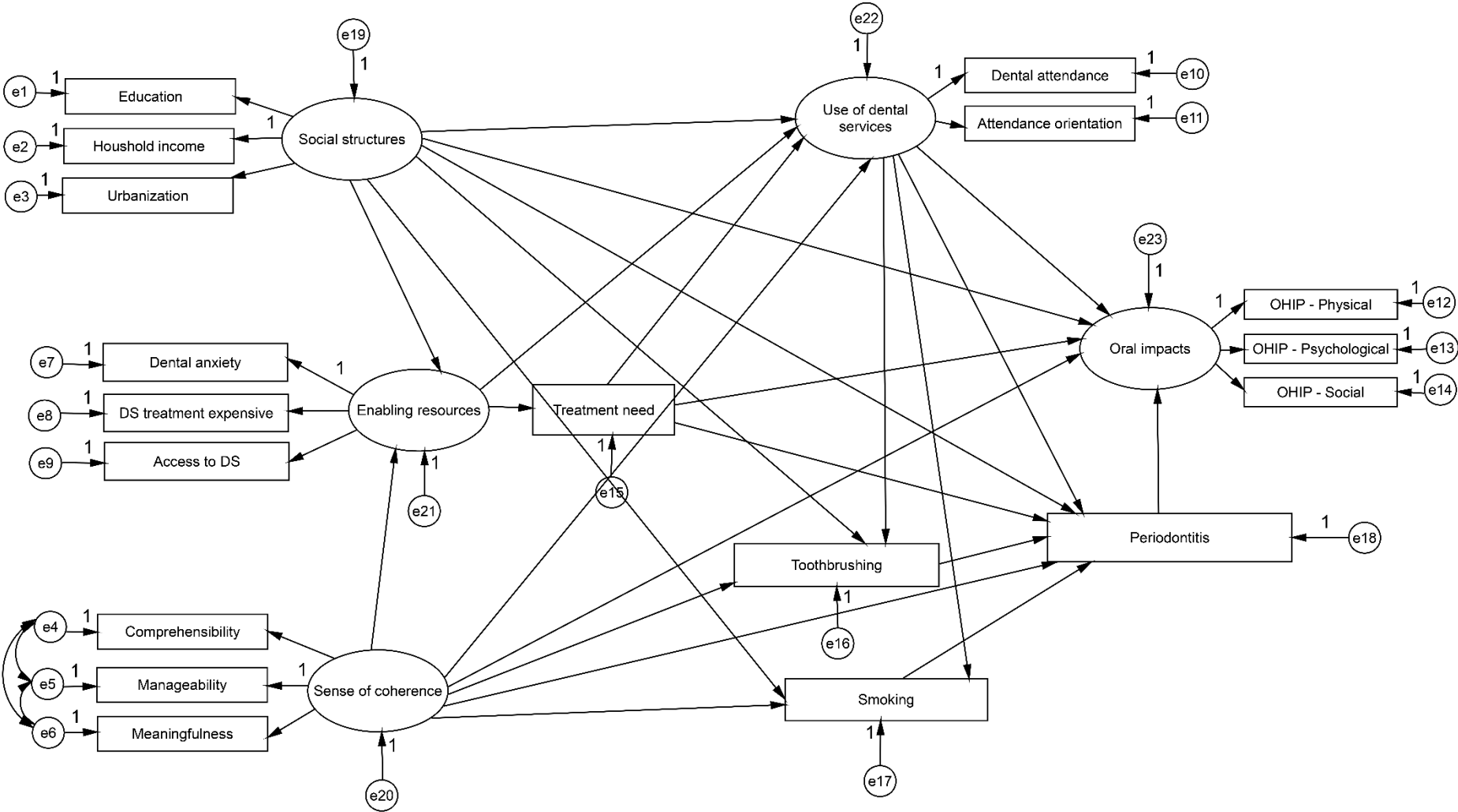
Supplementary Table 1. Detail of each construct, its operationalisation, measures including response options and scoring.

| Variable | Measure | Reference | No item/ dental examination | Response set | Scoring/interpretation | Cronbach's alpha |
|-------------------------------------|--|------------------------------|-----------------------------------|---|--|---------------------|
| POPULATION CHARACTERISTICS | | | | | | |
| Predisposing /social structures | Education | - | 1 | <i>'What is the highest level of school you have completed?'</i> Primary/middle school = 1, High school = 2, University = 3 | Higher scores more pre-disposing | - |
| | Annual household income | - | 1 | <i>'≤300,000NOK' = 1, '>300,000-450,000NOK' = 2, '>450,000- 900,000NOK' = 3, '900,000+ NOK' = 4.</i> | Higher scores more pre-disposing | - |
| | Urbanization | - | 1 | <i>'Rural/municipalities with widespread settlement' = 1, 'Suburban/municipalities with smaller towns' = 2, 'Urban/municipalities with larger towns' = 3.</i> | Higher scores more pre-disposing | - |
| Predisposing /salutogenic resources | Sense of coherence | Antonovsky 1993 Eide 1991 | 13 | <i>An example of item: 'Do you have the feeling that you don't really care about what goes on around you?'</i> 7 point Likert scale ranging from 1 to 7. The sum scores from 13–91. | Higher scores indicate stronger SOC = more pre-disposing | 0.84 |
| Enabling recourses | Declined treatment due to costs | - | 1 | <i>'Have you during the last two years refrained from dental services because you did not have enough money?'</i> 'Yes'= 1 and 'No' = 2. | Higher scores more resources | - |
| | Difficulty attending dental health care services | Marshman et al. 2012 | 1 | <i>'Is it difficult for you to get routine (e.g. check-up and fillings) dental health care?'</i> 'Yes/don't know" = 1,'No' = 2 | Higher scores more resources | - |

| | | | | | | |
|---------------------------|--------------------------------------|---------------------------------|--------------------|---|--|------|
| | Dental anxiety (DAS) | Corah's 1969, Kvale et al. 1997 | 4 | 5-point Likert scale yield sum scores from 4 to 20. | Scores reversed: Higher scores indicate less dental anxiety = more resources | 0.92 |
| Needs | Respondents perceived treatment need | Marshman et al. 2012 | 1 | <i>'If you saw a dentist tomorrow, do you think you would need treatment?'</i> 'I would not need treatment' = 1, 'Don't know' = 2, and 'I would need treatment' = 3. | Higher scores more needs | - |
| ORAL HEALTH BEHAVIORS | | | | | | |
| Personal health practices | Toothbrushing frequency | - | 1 | <i>'How often do you brush your teeth?'</i> 'Twice a day' = 3, 'once a day' = 2, and 'not daily' = 1 | Higher scores more frequent brushing | - |
| | Smoking status | - | 1 | Smoking status was categorized in three groups based on number of pack years: 'Non-smoker' = 1, 'Light smoker' = 2, 'Heavy smoker' = 3. | Higher scores more smoking | - |
| Use of dental services | Frequency of dental attendance | Marshman et al. 2012 | 1 | <i>'How often do you attend dental services?'</i> 'Only when having problems' = 1, 'Longer intervals than 2 years' = 2, 'Every second year' = 3, 'Every year' = 4. | Higher scores more frequent use | - |
| | Attendance orientation | Marshman et al. 2012 | 1 | <i>'When do you use dental services?'</i> 'Seldom/never attend DHCS' = 1, 'Only when having problems (pain, lost fillings)' = 2, 'Having routine recall/check-up' = 3. | Higher scores more frequent use | - |
| ORAL HEALTH OUTCOMES | | | | | | |
| Clinical outcomes | Periodontitis | Eke et al. 2015 | Dental examination | Periodontitis was categorized in three groups: 'Healthy' = 1, 'non-severe' = 2, 'severe' = 3. | Higher scores more periodontitis. | - |

| | | | | | | |
|-------------------------------------|--------------------------------------|-----------------------|----|--|--|------|
| Person-reported oral health outcome | Oral Health impact profile (OHIP-14) | Slade 1997, Dahl 2011 | 14 | 5-point Likert scale coded as never (1), hardly ever (2), occasionally (3), fairly often (4), and very often (5). The sum scores from 14-70. | Responses to item 1-5, and 10 represent physical function; item 6-9 psychological function; items 11-14 represents social function. The higher the score the greater oral health impacts were experienced. | 0.89 |
|-------------------------------------|--------------------------------------|-----------------------|----|--|--|------|

Supplementary Figure 1. Full structural model with all direct hypothesised pathways.



DS = Dental services

- 1) Population characteristics: social structures (i.e. high education, high income, living in a larger town with high availability to dental services) and SOC (higher scores) would predict more enabling resources (i.e. no difficulty in accessing dental services, no decline of treatment due to costs, and no dental anxiety).
- 2) Enabling resources would in turn predict patients' perceived treatment need. More enabling resources would relate to less perceived treatment need.
- 3) Social structure, SOC, enabling and treatment need would predict use of dental services, where more social structure, greater SOC, more enabling resources and less treatment need would relate to more use of dental services.
- 4) Social structures, SOC, enabling resources, treatment need and use of dental services would predict periodontal health, which in turn would predict oral impacts, with more severe periodontitis relating to more oral impacts.
- 5) Additionally, social structure and SOC would directly predict use of dental services, personal oral health practices (toothbrushing and smoking), periodontitis, and oral impacts. Use of dental services would predict personal oral health practices and oral impacts. Finally, personal oral health practices would predict periodontitis

Paper III

Title:

To what extent does smoking affect gingival bleeding response to supragingival plaque?

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Running title: Smoking and gingival bleeding response to plaque

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Abstract

Background and objective: The aim of this study was to investigate whether and to what extent smokers have a different inflammatory response in the gingiva to supragingival plaque and to assess if this differs in different regions of the dentition.

Methods: Data from a representative sample of 1,911 adults (20-79 yr old) in Northern Norway was analyzed. Periodontal examinations consisted of full-mouth recordings of periodontal probing depth (PD), bleeding on probing (BOP), and presence of supragingival plaque. Smoking status and background characteristics were self-reported by questionnaire. The association between plaque and BOP was assessed in several three-level (subject, tooth, and site) random intercept logistic regression models adjusted for PD, smoking status, socioeconomic factors, and body mass index. In a further model, it was assessed whether the association between supragingival plaque and BOP differed in different parts of the dentition.

Results: For plaque-free sites, bleeding tendency was lower in smokers, the odds ratio (OR) was 0.773 with a 95% confidence interval of 0.678-0.881 as compared to non-smokers (OR: 1; ref., $p < 0.001$). The odds of BOP at plaque-covered sites in non-smokers was increased twofold (OR: 2.117; 2.059-2.177). Albeit bleeding tendency was slightly increased in plaque-covered sites in smokers, it was considerably lower as compared to plaque covered sites in non-smokers (OR: 1.459; 1.282-1.662, $p < 0.001$). Heavy smoking (≥ 20 pack-years) further attenuated the association. In smokers, the odds of BOP was reduced in all parts of the dentition, lower and upper anterior and posterior teeth ($\chi^2_{(4)} = 32.043$, $p < 0.001$). When restricting the data to younger adults (20-34 yr old), smoking had only a slight effect on the association between plaque and BOP. For plaque-free and plaque-covered sites differences in ORs were not statistically noticeable ($p = 0.221$ and $p = 0.235$, respectively).

Conclusions: Smoking considerably attenuates the site-specific association between plaque and BOP with a dose dependent effect. The effect of smoking did not differ across tooth types.

Introduction

Smoking increases susceptibility to periodontitis and is associated with higher levels of periodontal destruction,¹ but also reduces the inflammatory response to dental plaque in the gingiva.² Gingival inflammation is considered a key risk factor for the development and progression of periodontitis.^{3,4} Therefore, it is important to investigate the extent to which smoking affects the gingival bleeding response to dental plaque.

In studies of experimental gingivitis it has been reported that smokers and non-smokers presented similar levels of dental plaque, while the severity of gingival inflammation was less pronounced in smokers as compared to non-smokers.⁵⁻⁹ This was also demonstrated in observational studies where smokers had similar, or even higher, levels of plaque than non-smokers but less gingival bleeding after probing.¹⁰⁻¹² What the above-mentioned studies have in common is that the relationship between gingival bleeding and plaque has been studied using subjects' mean values. Respective associations have been designated as ecological correlations.¹³ In ecological studies, data are analyzed at a higher level, e.g. at the population or group level, instead of the individual level. When data are analyzed in aggregate form, associations found at the population or group level cannot be inferred to the individual.¹⁴ The same applies for the association between subjects' mean gingival bleeding and mean plaque levels. The gingival inflammatory response to plaque occurs locally at the tooth site, so the (causal) relationship between plaque and gingival inflammation is preferably studied at the site-level in order to avoid bias and confounding, the so-called ecological fallacy.¹⁵

Site-level analysis of the effects of smoking on gingival bleeding have been assessed in some studies. A large study of a representative sample of the United States population showed that smoking had a strong and dose-dependent, suppressive effect on gingival bleeding after probing at the site level.¹⁶ In a study of Italian dental patients the odds for a site to bleed on probing was lower in smokers as compared to non-smokers.¹⁷ Plaque was not considered in these studies, and consequently the possible site-specific effects of smoking on the (causal) association between plaque and gingival bleeding was not studied.

Possible effects of smoking were, however, explicitly addressed in a six-month longitudinal experiment conducted in a cohort of young adults with mild gingivitis.¹⁸ In a steady state, where participants were asked not to alter oral hygiene habits, heavy smokers consistently presented with higher plaque and calculus scores. In this study, site-specific analyses did not reveal evidence for an enhanced or attenuated association between plaque and bleeding on probing.

So far, possible effects of smoking on the association between plaque and gingival inflammation have not been studied in a representative sample. Therefore, the aim of the present study was to investigate the influence of smoking on the site-specific inflammatory response in the gingiva to supragingival plaque in a general adult population. A second aim was to assess local effects of smoking by examining whether smoking affects respective association differently in different parts of the dentition.

Materials and methods

Study population

This is a secondary analysis of data from a dental health survey in Northern Norway (Tromstannen – Oral Health in Northern Norway [TOHNN]). The TOHNN study was a cross-sectional study of adults 20 to 79 yr old living in Troms County, Norway. The randomized sample included 2,901 individuals. The sampling and invitation procedures have been described in detail elsewhere.^{19,20} Data were collected between October 2013 and November 2014, with 1,986 participants completing the clinical examination and questionnaire. The Regional Committee for Medical and Health Research Ethics North, Norway, approved the study (2013/348/REC North). All participants provided written informed consent.

Inclusion and exclusion criteria

All subjects with two or more natural teeth were included in the analysis (n = 1,933).

Individuals with incomplete periodontal recordings (n = 4) were excluded. This resulted in

1,929 individuals (946 males and 983 females, aged 20-79 yr; mean age \pm standard deviation: 47.5 ± 15.3 yr).

Clinical examinations

Examinations were performed in dental offices by 11 calibrated dentists (employed by the Public Dental Health Service in Troms County) assisted by dental nurses. Measurements were made for all teeth, however third molars and implants were excluded from analysis. Bleeding on probing (BOP) has been recognized as a universally applicable means to describe local gingival inflammation in epidemiological studies.²¹ BOP and periodontal probing depth (PD) were originally assessed at six sites per tooth. PD was measured to the nearest millimeter with a periodontal probe with single millimeter gradations. BOP was registered immediately after periodontal probing, and was recorded dichotomously as present/not present. A modification of the Plaque Control Record was applied,²² in order to assess dental plaque at four sites per tooth (distal, buccal, mesial and palatal/lingual) as present or not using a mouth mirror and periodontal probe. No disclosing agent was used. For site-level analysis of PD and BOP, only four sites were considered: distal, buccal, mesial and palatal/lingual. Thus, the six-site measurements of PD and BOP were collapsed to four assessments by using the maximum PD or BOP of the two distal (disto-buccal, disto-palatal/lingual) and mesial (mesio-buccal, mesio-palatal/lingual) sites, respectively. Height (m) and weight (kg) were measured at time of examination and body mass index (BMI, kg/m^2) was calculated. Inter-examiner reliability of PD measurements has been reported elsewhere.²⁰

Questionnaire

Information about demographics, socioeconomic factors, behaviors, and health were collected by self-reported questionnaire. Age was stratified in categories 20-34, 35-44, 45-69, and 70-79. Education was categorized as less than high school, high school, and university level. Annual household income was analyzed in three categories (high, intermediate, low) according to national tertiles of household income in 2013.²³ Smoking was assessed by

smoking status (daily smoker: yes/no), number of cigarettes per day and number of years smoking. Smoking was further categorized by smoking status (non-smoker and smoker) and smoking level (non-smoker, light smoker <20 pack-years, heavy smoker ≥20 pack-years). One pack-year is defined as 20 cigarettes smoked per day for one year. Number of pack-years was calculated as (number of cigarettes per day/20) × number of years smoked. Former smokers (n = 42) were excluded from analysis because of unclear reporting of former smoking status. Missing data in other co-variables also resulted in exclusion from analysis. See Table 1 for number of excluded participants in each category.

Statistical analysis

Descriptive data are presented as means with standard deviations (SD) or numbers with proportions in parentheses. Three-level (subject, tooth, and site), random intercept, logistic regression models were built, with BOP as the outcome. A detailed description of the models can be found in Supporting Material 1.

Plaque, PD, smoking status (non-smoker and smoker), age group, gender, education, income, BMI and tooth type were entered as covariates. In order to assess how much smoking status modifies the association between plaque and bleeding on probing, interaction terms of “plaque × smoking status” were included as well. Bleeding tendency was also assessed at different tooth types, i.e. upper anterior, lower anterior, upper posterior, and lower posterior teeth. In further analyses, the association between plaque and BOP was assessed in young adults (20-34 yr old) and middle-aged adults (45-69 yr old). Results are reported as regression coefficients, odds ratios (OR) and respective 95% confidence intervals (CI). If considered necessary, *p*-values were derived from Wald tests. However, any inferential statistics (*p*-values, CIs) were intended to be exploratory, not confirmatory. No correction for multiple testing was done. *P*-values < 0.05 were considered as statistically noticeable.

Data were analyzed using special software (*MLwiN*, version 3.02, Centre for Multilevel Modelling, University of Bristol, Bristol, UK). For details, see Supporting Material 1.

Results

There were 1,929 dentate individuals with 192,172 sites with complete records of BOP, plaque and PD. Because of missing values in education, income, smoking status and BMI, the final model included 1,761 individuals with 176,220 sites. Mean percent BOP for excluded participants was 39.5%, and mean percent plaque was 46.9%, compared to 36.9% and 44.4%, respectively, for included participants (BOP: $t_{(1927)} = -1.48$, $p = 0.141$; plaque: $t_{(1927)} = -1.39$, $p = 0.165$). Characteristics of the study population are presented in Table 1.

Estimates of three-level random intercept models of BOP are listed in Table 2. According to the null model (without covariates), on average 34% gingival units bled upon probing. The reason for the discrepancy with the respective figure in Table 1 (37%) might be explained by the fact that the latter was calculated based on aggregate data. In the null model the variance partition coefficient (VPC) was 0.236, meaning 23.6% of the total variance was attributable to differences between subjects. In the model with main effects, plaque, PD and smoking, the OR of BOP when plaque was present at a site was (exponential of 0.733) 2.08 (95% CI: 2.03; 2.14). PD had an even stronger influence on the odds of BOP. With every millimeter increase in PD the odds for BOP increased by a factor of 2.82 (2.78; 2.87). On the other hand, being a smoker drastically decreased the odds of BOP. The OR was 0.744 (0.659; 0.840).

In order to examine whether smoking is an effect modifier in the association between plaque and BOP, the full model was set up with main effects, the interaction term “plaque × smoking” and further covariates, age groups, gender, education, income, and BMI (Table 2). Older age and higher level of education both reduced the odds of bleeding, while overweight and obese persons had increased odds of BOP. Interestingly, not only plaque and smoking status, but also the interaction term “plaque × smoking” strongly influenced the odds of BOP. Figure 1 displays three different, fully adjusted, models of BOP. With a site without plaque in a non-smoking subject as reference, ORs and 95% CIs were calculated for sites with and without plaque in non-smokers and smokers. Regarding the total sample, there was

apparently a very strong attenuating effect of smoking on the association between plaque and BOP ($p = 1.12 \times 10^{-4}$ and $p = 1.92 \times 10^{-8}$ for non-plaque covered and plaque covered sites, respectively). As age group appeared to have also an effect on the association, two separate models were set up with low and high proportion of smokers. Estimates of the models are listed in Table S4. Interestingly, in the youngest age group OR were only slightly lower in smokers ($p = 0.221$ and $p = 0.235$, respectively). In contrast the attenuating effect of smoking was even stronger in 45-69 yr olds ($p = 6.85 \times 10^{-4}$ and $p = 1.92 \times 10^{-6}$, respectively).

When considering the effect of lifetime tobacco exposure (pack-years), ORs for BOP were further attenuated in particular in heavy smokers (Fig. 2, Table S1). For example, for plaque-free sites, the OR was 0.807 (0.689; 0.945) in light smokers, and 0.671 (95% CI: 0.526-0.856) in heavy smokers as compared to non-smokers ($\chi^2_{(2)} = 16.190$, $p = 3.05 \times 10^{-4}$). For sites covered with plaque, light and heavy smokers had ORs for BOP of 1.537 (1.314; 1.799) and 1.146 (0.901; 1.456), while it was for non-smokers 2.115 (2.057; 2.175), $\chi^2_{(2)} = 37.756$, $p = 6.33 \times 10^{-9}$).

Table 4 presents ORs for BOP in different parts of the dentition in smokers as compared to non-smokers. Estimates of the model are listed in Table S2. As compared to non-smokers, the odds of BOP was reduced in all parts of the dentition, with ORs ranging between 0.685 (0.596; 0.787) for lower anterior teeth and 0.773 (0.675; 0.886) for lower posterior teeth ($\chi^2_{(4)} = 32.043$, $p = 1.88 \times 10^{-6}$). Interestingly, smokers had more plaque as compared to non-smokers, in all parts of the dentition ($\chi^2_{(4)} = 15.234$, $p = 0.004$, Table S3), with no difference between tooth types.

Discussion

The present analysis of data collected in a representative sample of adults in Northern Norway confirmed that smokers had less gingival bleeding upon probing than non-smokers. The results are in line with site-specific analyses of data collected in a population-based

epidemiological study conducted in the US.¹⁶ In that study, authors had observed that the odds ratio of bleeding upon probing was 0.53 in adults smoking even ≤ 10 cigarettes per day as compared to never smokers. It further decreased in heavy smokers. While presence of plaque was not assessed in that study, authors report a strong effect of sub or supragingival calculus (in a way a proxy for plaque) on BOP in never smokers, which was gradually and largely attenuated in former, light, and heavy smokers. The effect of heavy smoking was, in fact, so strong that sites with calculus in heavy smokers showed less than or the same bleeding as calculus-free sites in non-smokers.

In the present study, BOP was reduced in all parts of the dentition in smokers. In addition, the association between supragingival plaque and BOP was largely attenuated in smokers. For example, while, as compared to sites without plaque, the odds of BOP was more than twice as large at plaque-covered sites in non-smokers, the OR for plaque-covered sites in smokers was only slightly increased to 1.45 in smokers. Thus, smoking was a strong effect modifier of the (causal) relationship between plaque and gingival inflammation.

The bleeding response was not so much affected by smoking in younger adults (20-34-year-olds), a result that is in line with observations made in a 6-month longitudinal experiment in 19-30 yr old soldiers of the German Armed Forces who had been asked not to change oral hygiene habits.¹⁸ A possible explanation for these observations could be that young smokers have not been exposed to tobacco long enough to affect the bleeding response. Moreover, when considering the lifetime exposure of tobacco in terms of pack-years in the present study, the bleeding response was attenuated with a dose-dependent effect for light and heavy smokers, also indicating that the effect of smoking depends on the duration or amount of exposure.

In general, the odds of BOP was higher at lower anterior teeth as compared to other teeth when adjusted for plaque, PD, and subject-level covariates (Table S2). Differences in OR of BOP for tooth types was also reported in a retrospective study of dental patients in Italy.¹⁷ These authors found that posterior teeth were more likely to bleed upon probing than anterior

teeth. In that study, differences were rather small, and, additionally, presence of plaque was not adjusted for. When considering that posterior teeth have more plaque than anterior teeth,^{24,25} these results are probably due to the missing of an important (causal) factor in the analysis. In the present study, when considering smokers as compared to non-smokers, bleeding tendency was reduced in all parts of the dentition with no noticeable difference between tooth types. This is in agreement with results of the above-mentioned population-based study in the US, where authors reported no difference in the effect of smoking on gingival bleeding tendency between different tooth groups or jaws.¹⁶

Our results also considered other factors associated with gingival bleeding. For example, the association of PD (a proxy for subgingival plaque) with BOP was very strong. With each millimeter increase, the odds of BOP increased almost threefold. This is consistent with results from previous studies where the OR of BOP was increased twofold per mm increase in PD,¹⁷ or when comparing sites with increased PD to healthy sites (PD 0-3 mm).¹⁶ Higher age (≥ 35 yr) reduced the odds of bleeding by around 30%, apparently with a threshold effect, as gingival bleeding did not vary among persons 45 yr old and older. A study of experimental gingivitis found that older persons developed more gingivitis than younger persons,²⁶ while no difference in bleeding probability according to age was reported among Italian dental patients.¹⁷ In the present study, there was no difference in bleeding tendency between males and females. In previous site-specific analyses, differences in gingival bleeding between genders have been reported, however in both directions.^{16,17} In the present study, persons with higher education were less likely to bleed on probing, while income was not related to bleeding tendency. Previous studies have reported that people with lower income were more likely to show gingival bleeding,¹⁶ and that lower education was related to more BOP or gingival inflammation.^{27,28} In our study, overweight and obesity increased the bleeding tendency of the gingiva; however, higher body mass index was also associated with higher plaque levels. Obesity has been associated with periodontitis with several possible mechanisms proposed, i.e. increased inflammatory response, change in dental plaque

amount and composition, or both.²⁹ Our results indicate that overweight/obesity is associated with more gingival bleeding, and partly through increased levels of plaque. In particular, there was no noticeable interaction between plaque and overweight/obesity (Table S5), meaning BMI, in contrast to smoking, is not an effect modifier as regards the association between plaque and bleeding on probing.

The underlying mechanisms of smoking and its effect on gingival bleeding are somewhat unclear. There is limited evidence that tobacco smoke promotes gingival vasoconstriction in humans.³⁰⁻³⁴ There is some evidence of tobacco-induced suppressed angiogenesis, where a reduced number of gingival vessels or vessels of smaller caliber has been found in smokers relative to non-smokers.^{6,35-37} Thermally induced nerve damage in the oral cavity of smokers,^{38,39} could potentially affect the microvascular response of the gingiva.² Additionally, tobacco smoking alters the dental plaque composition.⁴⁰ Findings from a large study of the human oral microbiome in U.S. adults indicate that smoking promotes an anaerobic oral environment and a bacterial community with a reduced capability of degrading toxic components of cigarette smoke.⁴⁰ Furthermore, it has been proposed that smoking can suppress oral pathogens' production of short chain fatty acid, which can influence components of immune and healing responses, thereby presenting an additional mechanism for reducing vascular response to dental plaque.⁴¹ Most importantly, cigarette smoking has been reported to affect the immune responses.⁴² For example, decreased levels of pro-inflammatory biomarkers in smokers with periodontitis suggests a reduced capacity to recruit inflammatory and immune cells, which may explain the enhanced susceptibility to periodontitis,⁴³ and the reduced bleeding response to plaque.

There are many factors, other than smoking, that can modify the gingival inflammatory response to plaque, which have not been controlled for in the current study. Such factors include pregnancy, diabetes, Down's syndrome, vitamin C deficiency, anti-microbial and anti-inflammatory agents and conditions affecting the immune system (reviewed by Tatakis et al.⁴⁴). For example, toothpaste containing the antibacterial compound triclosan was shown to

attenuate the association between plaque and BOP in a randomized controlled trial.⁴⁵ Additionally, studies have shown that diet, and especially vitamin D, can affect gingivitis.^{46,47} Both smoking and obesity have been associated with lower levels of vitamin D in a population based study in Northern Norway.⁴⁸ Finally, the host-dependent variation in gingivitis susceptibility should be considered. In several studies, a subject-specific gingival inflammatory response has been reported, and “high- and low-responders”,⁴⁹ or “fast and slow responders” identified.⁵⁰

With increased focus on the inflammatory nature of periodontitis, host modulation therapy is an emerging treatment strategy for managing periodontitis, aiming to control the inflammation in order to control the infection.⁵¹ In this aspect, smoking’s effect on periodontal disease should be considered, where gingival inflammation is reduced, but periodontal destruction is increased. Smoking has on one hand toxic effects and on the other hand immunosuppressive effects.⁴² The latter might be the reason why incidence and/or severity of some inflammatory diseases has been reported to be reduced in smokers.⁵²⁻⁵⁴ Nicotine, the main immunosuppressive constituent of cigarette smoke, has even been suggested as a potential therapeutic agent in chronic inflammatory diseases such as ulcerative colitis.^{55,56}

As an epidemiological survey, the study has several limitations that need to be critically addressed. The study design was cross-sectional, so no causal relationships can be concluded. BOP and plaque was only measured at one time-point, and assumes a steady state plaque environment.²⁴ Examiners were not calibrated for measurements of the main outcome, BOP, as in a study of agreement and association of gingival bleeding after repeated probing it had been concluded that the reliability of an invasive diagnostic, such as BOP, could not really be determined, and that associations between repeat BOP were generally weak.⁵⁷ To precisely assess the dose dependent effect of smoking on the gingival bleeding response to plaque, information about amount and duration of smoking would be highly desirable. There was no objective measure of smoking, e.g. measuring serum cotinine levels. Smoking history was self-reported in a questionnaire, presenting a potential source of

imprecise smoking estimates. Nevertheless, reported smoking frequency was close to national estimates.⁵⁸ Furthermore, number of cigarettes smoked per day reported by smokers could have varied over time, potentially resulting in a non-accurate calculation of pack-years. Some of the persons that reported as non-smokers could have been former smokers. Previous studies have reported a suppressive effect on gingival bleeding among former smokers, albeit small, as compared to smokers.¹⁶ In the present study, for models including all covariates, 166 participants had been excluded because of missing values in questions about education, income, smoking and BMI. However, there were only small differences in BOP and plaque levels between the excluded and included participants.

Despite these limitations, this is, to the best of our knowledge, the first study to assess the influence of smoking on the gingival inflammatory response to supragingival plaque in a general adult population. Moreover, multilevel analysis confirms previous evidence of the attenuating effect of smoking on the inflammatory response to dental plaque at the site level.

In conclusion, analyses of data from a population-based epidemiological study in Northern Norway show that smoking reduces the general bleeding tendency of the gingiva but also attenuates the site-specific association between plaque and gingival bleeding. The extent of the attenuation is dependent on tobacco exposure, where heavy smoking further attenuates the association between gingival bleeding and plaque. The effect of smoking did not differ between different regions of the dentition. A reduced inflammatory response to dental plaque indicates that there might be a need for different strategies for periodontal infection control among smokers and non-smokers. BOP might not be a reliable measure of inflammation in smokers.

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Figure legends

Figure 1. The association between plaque and BOP in non-smokers and smokers in the total sample, and in age groups 20-34 and 45-69 yr. Odds ratios and 95% confidence intervals (CI) for BOP as derived from 3-level random intercept models adjusted for pocket depth, gender, age, education, income, and body mass index.

Figure 2. Odds ratios (OR) and 95% confidence intervals (CI) for BOP in non-smokers and light and heavy smokers as derived from 3-level random intercept model of bleeding on probing adjusted for pocket depth, gender, age, education, income, and body mass index.

Table 1. Characteristics of the study population

| Individual related variables (level 3) | N = 1,929 |
|--|--------------|
| Age, years [mean (SD)] | 47.5 (15.3) |
| Age group [n (%)] | |
| 20-34 years | 462 (24.0) |
| 35-44 years | 386 (20.0) |
| 45-69 years | 926 (48.0) |
| 70-79 years | 155 (8.0) |
| Gender [n (%)] | |
| Female | 983 (51.0) |
| Male | 946 (49.0) |
| Education [n (%)] | |
| University level | 796 (41.3) |
| High school | 835 (43.3) |
| Less than high school | 280 (14.5) |
| Missing | 18 (0.9) |
| Income [n (%)] | |
| High | 371 (19.2) |
| Intermediate | 917 (47.5) |
| Low | 566 (29.3) |
| Missing | 75 (3.9) |
| Smoking [n (%)] | |
| Smoker | 284 (14.7) |
| Non-smoker | 1,590 (82.4) |
| Missing | 55 (2.9) |
| Smoking level [n (%)] | |
| Heavy smoker | 74 (3.8) |
| Light smoker | 180 (9.3) |
| Non-smoker | 1,590 (82.4) |
| Missing | 85 (4.4) |
| Diabetes [n (%)] | 71 (3.7) |
| BMI (kg/m ²) [n (%)] | |
| Normal weight (<25) | 656 (34.0) |
| Overweight (25-29.9) | 773 (40.1) |
| Obese (≥30) | 472 (24.5) |
| Missing | 28 (1.4) |
| BOP score* [mean (SD)] | 37.1 (19.9) |
| Plaque score* [mean (SD)] | 44.6 (22.7) |
| Tooth-related variables (level 2) | N = 48,043 |

| | |
|----------------------------------|---------------|
| Tooth type [n (%)] | |
| Upper anterior teeth | 10,734 (22.3) |
| Lower anterior teeth | 11,374 (23.7) |
| Upper posterior teeth | 12,790 (26.6) |
| Lower posterior teeth | 13,145 (27.4) |
| Site-related variables (level 1) | N = 192,172 |
| PD, mm [mean (SD)] | 2.1 (1.0) |
| BOP, % [mean (SD)] | 36.6 (48.2) |
| Plaque, % [mean (SD)] | 43.6 (49.6) |

SD: standard deviation; BMI: body mass index; BOP: bleeding on probing; PD: probing depth
 *Subjects' averages

Table 2. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression models of bleeding on probing.

| | Null model (1) | Main effects (2) | Full model (3) |
|--|----------------|------------------|----------------|
| | Estimate (SE) | Estimate (SE) | Estimate (SE) |
| <i>Fixed effects</i> | | | |
| β_{0jk} (intercept) | -0.649 (0.024) | -1.020 (0.025) | -0.823 (0.092) |
| Plaque vs. no plaque | | 0.733 (0.013) | 0.750 (0.014) |
| PD (centered on mean) | | 1.038 (0.008) | 1.039 (0.009) |
| Smoker vs. non-smoker | | -0.296 (0.062) | -0.258 (0.067) |
| Plaque x smoker | | | -0.114 (0.039) |
| Female vs. male | | | 0.041 (0.046) |
| Age group (reference: 20-34 years) | | | |
| 35-44 years | | | -0.309 (0.069) |
| 45-69 years | | | -0.388 (0.058) |
| 70-79 years | | | -0.357 (0.098) |
| Education (reference: less than high school) | | | |
| High school | | | -0.084 (0.072) |
| University level | | | -0.219 (0.076) |
| Income (reference: low income) | | | |
| Intermediate income | | | 0.063 (0.054) |
| High income | | | 0.010 (0.071) |
| BMI (reference: normal weight) | | | |
| Overweight | | | 0.149 (0.053) |
| Obese | | | 0.306 (0.060) |
| <i>Random effects</i> | | | |
| v_{0k} (subject level variance) | 1.022 (0.035) | 0.831 (0.030) | 0.773 (0.029) |
| u_{0jk} (tooth level variance) | 0.026 (0.008) | 0.144 (0.011) | 0.144 (0.011) |
| VPC | 0.236 | 0.195 | 0.184 |

SE: standard error; PD: probing depth; BMI: body mass index; VPC: variance partition coefficient (VPC = $\sigma^2_v / (\sigma^2_v + \sigma^2_u + \pi^2/3)$, see supporting material 1)

Table 3. Odds ratios (OR) and 95% confidence intervals (CI) as derived from a fully adjusted 3-level random intercept model of BOP in smokers in different parts of the dentition as compared to non-smokers.

| Tooth type | OR | 95% CI | <i>p</i> -value |
|-----------------------|-------|----------------|-----------------------|
| Upper anterior teeth | 0.710 | (0.616; 0.819) | 2.30×10^{-6} |
| Lower anterior teeth | 0.685 | (0.596; 0.787) | 9.44×10^{-8} |
| Upper posterior teeth | 0.725 | (0.631; 0.832) | 4.62×10^{-6} |
| Lower posterior teeth | 0.773 | (0.675; 0.886) | 2.19×10^{-4} |

Supporting Material 1

Description of the multilevel model

Supporting Material 2

Table S1. Estimates (with standard error in parenthesis) of a 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, including smoking level.

Table S2. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, including tooth type.

Table S3. Odds ratios (OR) and 95% confidence intervals (CI) as derived from a 3-level random intercept model of plaque in current smokers in different parts of the dentition, compared to non-smokers. Adjusted for gender, age, education, income, and body mass index.

Table S4. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, in age groups 20-34 and 45-69 years.

Table S5. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression models of bleeding on probing, including interaction term of plaque × BMI

Figure 1

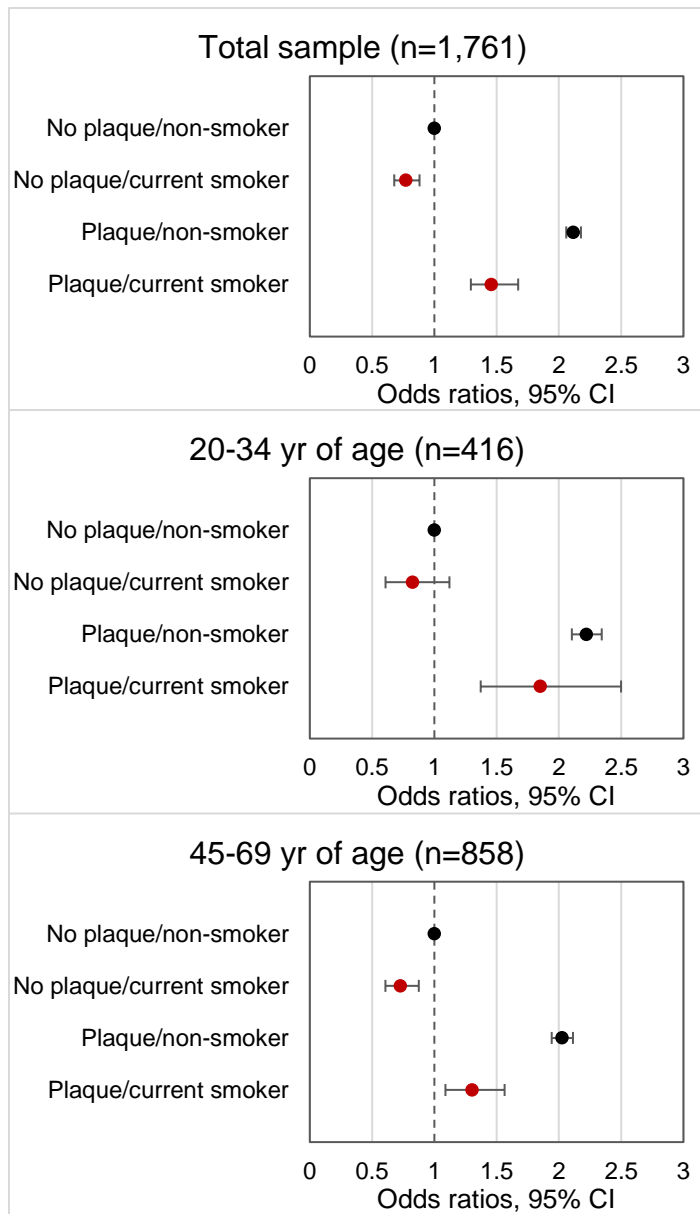
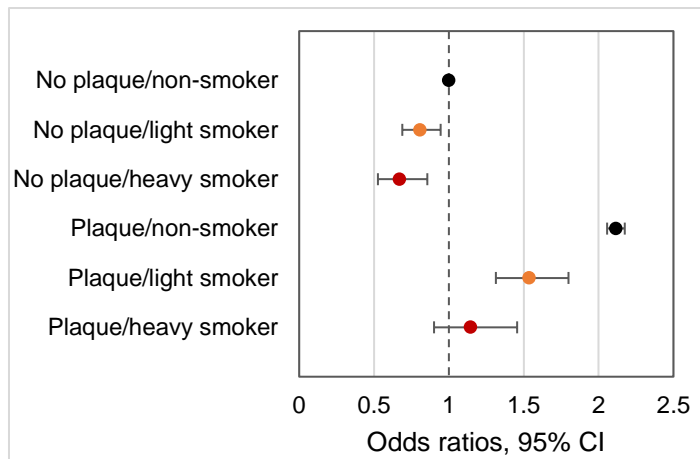


Figure 2



Supporting material 1

Description of the multilevel model

Three-level (subject, tooth, and site), random intercept, logistic regression models were built, with BOP as the outcome. The binary response BOP_{ijk} equals 1 if bleeding occurred after probing at site i , in tooth j nested within subject k and 0 if bleeding did not occur. The model can be written as,

$$\begin{aligned} BOP_{ijk} &\sim \text{Binominal}(1, \pi_{ijk}) \\ \text{logit}(\pi_{ijk}) &= \beta_{0jk} + \beta_1 x_{1ijk} + \beta_2 x_{2jk} + \beta_3 x_{3k} \\ \beta_{0jk} &= \beta_0 + v_{0jk} + u_{0k} \\ v_{0jk} &\sim N(0, \sigma_{v0jk}^2) \\ u_{0k} &\sim N(0, \sigma_{u0k}^2) \\ \text{var}(BOP_{ijk} | \pi_{ijk}) &= \pi_{ijk}(1 - \pi_{ijk}) \end{aligned}$$

with teeth and subjects as higher levels where the intercept β_0 is allowed to vary randomly about teeth and subjects. Additional site, tooth and subject related explanatory variables (e.g., x_{1ijk} , x_{2jk} and x_{3k} , respectively) are then added to the model to allow for respective effects. The level 2 (tooth) and level 3 (subject) random variation is described by the terms v_{jk} and u_k , respectively. Random effects at the tooth and subject level are assumed to follow a normal distribution with mean 0 and variances σ_{v0k}^2 and σ_{u0jk}^2 , respectively. Variance partition coefficients (VPC) were calculated by applying a latent variable approach.¹ Thus, $VPC = \sigma_v^2 / (\sigma_v^2 + \sigma_u^2 + \pi^2/3)$.

Data were analyzed using special software (*MLwiN*, version 3.02, Centre for Multilevel Modelling, University of Bristol, Bristol, UK). In particular, quasi-likelihood methods using a linearization method based on a Taylor series expansion which transforms a discrete response model to a continuous response model were applied. After linearization, the model was then estimated using iterative generalized least squares with 2nd order penalized quasi-

likelihood approximation.² Model assumptions were confirmed through analysis of residuals generated by the software.

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Supporting material 2

Estimates of the three-level random intercept model of BOP where smoking was categorized by smoking level (non-smoker, light smoker and heavy smoker) are listed in Table S1. Both light and heavy smoking had a noticeable effect on BOP. Light smoking reduced the odds of BOP with a factor of (exponential of -0.215) 0.807 (95% CI: 0.688; 0.945), while the effect of heavy smoking was even greater, reducing the odds of BOP with a factor of (exponential of -0.399) 0.671 (0.526; 0.856).

Table S1. Estimates (with standard error in parenthesis) of a 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, including smoking level.

| | Smoking level |
|--|----------------|
| | Estimate (SE) |
| <i>Fixed effects</i> | |
| β_{0jk} (intercept) | -0.829 (0.093) |
| Plaque vs. no plaque | 0.749 (0.014) |
| PD (centered on mean) | 1.040 (0.009) |
| Smoking level (reference: non-smoker) | |
| Light smoker | -0.215 (0.081) |
| Heavy smoker | -0.399 (0.124) |
| Plaque x smoking level | |
| Plaque x light smoker | -0.104 (0.046) |
| Plaque x heavy smoker | -0.213 (0.079) |
| Female vs male | 0.035 (0.046) |
| Age group (reference: 20-34 years) | |
| 35-44 years | -0.308 (0.069) |
| 45-69 years | -0.389 (0.059) |
| 70-79 years | -0.355 (0.099) |
| Education (reference: less than high school) | |
| High school | -0.068 (0.073) |
| University level | -0.202 (0.077) |
| Income (reference: low income) | |
| Intermediate income | 0.059 (0.054) |
| High income | 0.008 (0.071) |
| BMI (reference: normal weight) | |
| Overweight | 0.144 (0.054) |

| | |
|-----------------------------------|---------------|
| Obese | 0.307 (0.060) |
| <i>Random effects</i> | |
| v_{0k} (subject level variance) | 0.772 (0.029) |
| u_{0jk} (tooth level variance) | 0.141 (0.011) |
| VPC | 0.184 |

SE: standard error; PD: probing depth; BMI: body mass index; VPC: variance partition coefficient

Table S2 lists the estimates of three-level random intercept models of BOP where tooth type is included as a covariate. The estimates for tooth types did not change noticeably from Model 1 to Model 3. Adjusted for age, gender, socioeconomic factors, and body mass index, the odds of BOP increased for lower anterior teeth and slightly increased for upper posterior teeth as compared to upper anterior teeth. The ORs were (exponential of 0.315) 1.37 (95% CI: 1.32; 1.43) and (exponential of 0.054) 1.06 (1.01; 1.10), respectively.

Table S2. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, including tooth type.

| | Model 1 | Model 2 | Model 3 |
|--|----------------|----------------|----------------|
| | Tooth type | + interaction | Full model* |
| | Estimate (SE) | Estimate (SE) | Estimate (SE) |
| <i>Fixed effects</i> | | | |
| β_{0jk} (intercept) | -1.109 (0.027) | -1.105 (0.028) | -0.910 (0.093) |
| Plaque vs. no plaque | 0.732 (0.013) | 0.732 (0.013) | 0.734 (0.014) |
| PD (centered on mean) | 1.064 (0.009) | 1.064 (0.009) | 1.064 (0.009) |
| Current smoker vs. non-smoker | -0.304 (0.062) | -0.335 (0.071) | -0.342 (0.072) |
| Tooth type (reference: upper anterior) | | | |
| Lower anterior | 0.308 (0.018) | 0.311 (0.020) | 0.315 (0.020) |
| Upper posterior | 0.054 (0.018) | 0.050 (0.019) | 0.054 (0.020) |
| Lower posterior | 0.004 (0.018) | -0.011 (0.019) | -0.010 (0.020) |
| Smoking × tooth type | | | |
| Current smoker × lower anterior | | -0.021 (0.052) | -0.037 (0.054) |
| Current smoker × upper posterior | | 0.026 (0.051) | 0.020 (0.052) |
| Current smoker × lower posterior | | 0.104 (0.050) | 0.085 (0.052) |
| <i>Random effects</i> | | | |
| v_{0k} (subject level variance) | 0.827 (0.030) | 0.828 (0.030) | 0.768 (0.029) |
| u_{0jk} (tooth level variance) | 0.132 (0.011) | 0.133 (0.011) | 0.132 (0.011) |
| VPC | 0.195 | 0.195 | 0.183 |

SE: standard error; PD: probing depth; VPC: variance partition coefficient

*Adjusted for age, gender, education, income, and body mass index

In order to assess level of plaque in different parts of the dentition of smokers as compared to non-smokers, ORs were derived from a three-level random intercept model of plaque (Table S3). Smokers had increased odds of plaque in all parts of the dentition as compared to non-smokers. The OR ranged from 1.17 in lower anterior teeth to 1.33 in upper anterior teeth. However, there were no statistically noticeable difference between tooth types.

Table S3. Odds ratios (OR) and 95% confidence intervals (CI) as derived from a 3-level random intercept model of plaque in current smokers in different parts of the dentition, compared to non-smokers. Adjusted for gender, age, education, income, and body mass index.

| | OR | 95% CI | p-value |
|-----------------------|-------|----------------|---------|
| Upper anterior teeth | 1.331 | (1.125; 1.575) | 0.001 |
| Lower anterior teeth | 1.169 | (0.990; 1.380) | 0.066 |
| Upper posterior teeth | 1.265 | (1.073; 1.492) | 0.005 |
| Lower posterior teeth | 1.198 | (1.016; 1.413) | 0.032 |

To assess the possible different associations between plaque and BOP in various age groups, two separate three-level random intercept models were set up: age group 20-34 year olds with low proportion of smokers, and age group 45-69 year olds with high proportion of smokers. Smoking did not have a statistically noticeable effect on BOP in the youngest age group, while the opposite was true for the 45-69 year old age group (Table S4).

Table S4. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression model of bleeding on probing, in age groups 20-34 and 45-69 years.

| | 20-34 year olds | 45-69 year olds |
|--|-----------------|-----------------|
| | Estimate (SE) | Estimate (SE) |
| <i>Fixed effects</i> | | |
| β_{0jk} (intercept) | -1.075 (0.215) | -1.017 (0.124) |
| Plaque vs. no plaque | 0.798 (0.027) | 0.706 (0.021) |
| PD (centered on mean) | 1.150 (0.018) | 0.997 (0.012) |
| Current smoker vs. non-smoker | -0.192 (0.156) | -0.317 (0.093) |
| Plaque x current smoker | 0.010 (0.090) | -0.123 (0.053) |
| Female vs. male | 0.122 (0.089) | -0.006 (0.068) |
| Age (centered on mean) | -0.011 (0.012) | -0.004 (0.005) |
| Education (reference: less than high school) | | |
| High school | -0.073 (0.203) | -0.096 (0.094) |
| University level | -0.307 (0.209) | -0.158 (0.101) |
| Income (reference: low income) | | |
| Intermediate income | 0.110 (0.100) | -0.064 (0.082) |
| High income | 0.126 (0.141) | -0.106 (0.109) |
| BMI (reference: normal weight) | | |
| Overweight | 0.298 (0.099) | 0.141 (0.083) |
| Obese | 0.263 (0.122) | 0.343 (0.090) |
| <i>Random effects</i> | | |
| v_{0k} (subject level variance) | 0.677 (0.051) | 0.833 (0.044) |
| u_{0jk} (tooth level variance) | 0.097 (0.021) | 0.187 (0.017) |
| VPC | 0.167 | 0.193 |

SE: standard error; PD: probing depth; BMI: body mass index; VPC: variance partition coefficient

Table S5 presents estimates of the fully adjusted three-level random intercept model of BOP, where the interaction term “plaque x BMI” was included to assess if there was an interaction between plaque and BMI. The interaction term was not statistically noticeable, meaning BMI did not act as an affect modifier to the association between BOP and plaque.

Table S5. Estimates (with standard error in parenthesis) of 3-level (site, tooth, and subject), random intercept, logistic regression models of bleeding on probing, including interaction term of plaque x BMI

| | Full model |
|--|----------------|
| | Estimate (SE) |
| <i>Fixed effects</i> | |
| β_{0jk} (intercept) | -0.823 (0.093) |
| Plaque vs. no plaque | 0.750 (0.023) |
| PD (centered on mean) | 1.039 (0.009) |
| Current smoker vs. non-smoker | -0.258 (0.067) |
| Plaque x current smoker | -0.113 (0.039) |
| Female vs. male | 0.041 (0.046) |
| Age group (reference: 20-34 years) | |
| 35-44 years | -0.309 (0.069) |
| 45-69 years | -0.389 (0.058) |
| 70-79 years | -0.358 (0.098) |
| Education (reference: less than high school) | |
| High school | -0.084 (0.072) |
| University level | -0.218 (0.076) |
| Income (reference: low income) | |
| Intermediate income | 0.063 (0.054) |
| High income | 0.011 (0.071) |
| BMI (reference: normal weight) | |
| Overweight | 0.157 (0.055) |
| Obese | 0.292 (0.062) |
| Plaque x BMI | |
| Plaque x overweight | -0.015 (0.030) |
| Plaque x obese | 0.027 (0.035) |
| <i>Random effects</i> | |
| v_{0k} (subject level variance) | 0.774 (0.029) |
| u_{0jk} (tooth level variance) | 0.145 (0.011) |
| VPC | 0.184 |

SE: standard error; PD: probing depth; BMI: body mass index; VPC: variance partition coefficient

Appendices

Appendix 1

Tromstannen – Oral Health in Northern Norway

Original Norwegian versions of:

Invitation letter

Informed written consent form

Questionnaire



HAR DU SPØRSMÅL OM STUDIEN ELLER DELTAKELSE?

Kontakt gjerne prosjektleder Nils Oscarson

på tlf. 77 78 90 00 eller e-post nils.oscarson@tromsfylke.no

ØNSKER DU SELV Å TA KONTAKT VIA E-POST FOR TIMEBESTILLING?

Du kan bruke følgende e-postadresse tknn@tromsfylke.no og angi «Tromstannen» i emnefeltet



INVITASJON

TIL Å DELTA I STUDIE OM TANNHELSE I TROMS FYLKESKommUNE

Formålet med studien er å kartlegge tannhelsen i Troms Fylkeskommune



BAKGRUNN OG HENSIKT

Dette er et spørsmål til deg om å delta i en forskningsstudie for å kartlegge tann- og munnhelseforhold i befolkningen i Troms fylkeskommune. Hensikten med studien er å beskrive tannhelsen for å kunne tilby alle et likeverdig tannbehandlingstilbud basert på den enkeltes behov. Dette vil være et viktig bidrag for å fremme tannhelsen i befolkningen i kommunen. Du er invitert for du er mellom 20-79 år og bor i Troms fylkeskommune. Studien gjøres i regi av Tannhelsetjenesten i Troms fylkeskommune, Tannhelsetjenestens Kompetansesenter for Nord-Norge og i samarbeid med institutt for klinisk odontologi, Universitetet i Tromsø.

HVA INNEBÆRER STUDIEN?

Du vil bli kontaktet per telefon og dersom du velger å delta i studien vil du tilbys tid for en kostnadsfri tannundersøkelse til en tannklinikk som passer best for deg å komme til. Verdien av denne tannundersøkelsen ville vært cirka kr 1 000,-. Hvis du bor langt fra klinikken vil du bli tilbudt kompensasjon for reisekostnader. Vi vil også be deg om å fylle ut et spørreskjema. Skjemaet har noen spørsmål om bruk av tannhelsetjenesten, hvordan du oppfatter forhold knyttet til din egen tann- og munnhelse, litt om kosthold og røykevaner samt noen spørsmål om økonomi, arbeid og etnisk tilhørighet. Det vil ta omtrent 15 minutter å fylle ut spørreskjemaet.

Tannlegen vil undersøke tennene dine og munnhulen ved å ta et røntgenbilde, notere antall tenner, fyllinger og eventuelle synlige hull samt undersøke om du har tannkjøtt sykdom, forandringer i munnslimhinner, problem med kjeveledd og hvis du har et behandlingsbehov. Undersøkelsene vil gjennomføres av spesialtrene tannlege-team. Data vil registreres i et dataprogram. Vi ber om tillatelse til at data om deg brukes til forskning.

GODTGJØRELSE

Du som deltar i undersøkelsen vil i tillegg til fri undersøkelse og dekning av reisekostnader få en gave/verdikupong til en verdi av kr 150,-. Dessuten vil du delta i trekningen av tre iPad nettbrett og tjue elektriske tannbørster.



FORDELER OG ULEMPER

Det forventes ikke noen risikoer forbundet med tannundersøkelsen. Du vil få en tilbakemelding på den kliniske undersøkelsen med eventuell anbefaling om behov for behandling og som du har mulighet å ta med til din ordinære tannklinikk. Du kan reservere deg mot å få denne informasjonen.



HVA SKJER MED INFORMASJONEN OM DEG?

Informasjonen som registreres om deg skal kun brukes slik som beskrevet i hensikten med studien. Etter de att data er innsamlet vil dataene anonymiseres. Alle opplysningene vil bli behandlet uten navn og fødselsnummer eller andre direkte gjenkjenning opplysninger. Det vil ikke være mulig å identifisere deg i resultatene av studien når disse publiseres.

FRIVILLIG DELTAKELSE

Det er frivillig å delta i studien. Du kan når som helst, og uten å oppgi noen grunn, trekke ditt samtykke til å delta i studien. Det får ingen konsekvenser for deg dersom du takker nei til invitasjonen i forhold til fremtidig kontakt med tannhelsetjenesten. Dersom du ønsker å delta, undertegner du samtykkeerklæringen på siste side. Om du nå sier ja til å delta, kan du senere trekke tilbake ditt samtykke uten at det påvirker din øvrige behandling.

RETT TIL INNSYN OG SLETING AV OPPLYSNINGER OM DEG OG SLETING AV PRØVER

Hvis du sier ja til å delta i studien, har du rett til å få innsyn i hvilke opplysninger som er registrert om deg. Du har videre rett til å få korrigert eventuelle feil i de opplysningene vi har registrert. Dersom du trekker deg fra studien, kan du kreve å få slettet innsamlet informasjon, med mindre disse opplysningene allerede er inngått i analyser eller brukt i vitenskapelige publikasjoner.

ØKONOMI

Studien finansieres gjennom forskningsmidler fra Tannhelsetjenesten i Troms fylkeskommune og støtte fra statlige midler. Ingen av finansildene har interessekonflikter knyttet til prosjektet.



FORSIKRING

Deltakerne er dekket gjennom pasientskadeerstatningsloven

INFORMASJON OM UTFALLET AV STUDIEN

Resultater av undersøkelsen vil publiseres i rapporter og internasjonale anerkjente vitenskapelige tidsskrifter. Sammenfatning av resultatene vil også kunne publiseres ulike mediekkanaler.

SAMTYKKE

Dersom du ønsker å delta i studien, undertegner du en samtykkeerklæring, svarer på et spørreskjema og leverer begge til tannhelsepersonalet ved tannklinikken i forbindelse med undersøkelsen.



TROMSTANNEN

- munnhelse i Troms Fylkeskommune



TROMS fylkeskommune
ROMSSA fylkkasuohkanl

Samtykkeerklæring

Jeg har lest informasjonen i "Invitasjon til å delta i studie om tannhelse i Troms fylkeskommune" og samtykker til å delta i studien Tromstannen – munnhelse i Troms fylkeskommune.

Jeg er kjent med at opplysningene behandles strengt konfidensielt og at jeg når som helst kan trekke meg fra deltakelse.

Personopplysninger (deltaker):

Navn:

Fødselsnummer (11 siffer):

(Signatur, dato)

Delen under fylles ut av tannhelsepersonell ved tannklinikken.

Jeg bekrefter å ha gitt informasjon om studien

(Signert, rolle i studien, dato)

Dersom du ønsker å delta i studien må du fylle ut denne samtykkeerklæringen, og levere den til tannhelsepersonalet ved tannklinikken i forbindelse med undersøkelsen.



TROMSTANNEN

- munnhelse i Troms Fylkeskommune



TROMS fylkeskommune
ROMSSA fylkkasuohtkanl

Takk for at du vil delta i denne undersøkelse. På denne måten vil du bidra til økt kunnskap om tannhelse og bruk av tannhelsetjenester i befolkningen i Tromsfylke.

For spørsmål med flere valg, sett kun ett kryss hvis det ikke er beskrevet annerledes

1. Hvor gammel er du?år

2. Kjønn:

- Mann
 Kvinne

3. Fødested?

- Norge
 Annet (beskriv):.....

4. Hvis du er født i utlandet: Hvor lenge har du bodd i Norge? år

5. Hvilken utdanning har Du, hva er din høyeste fullførte grad?

- Grunnskole
 Videregående skole
 Høyskole, universitet

6. Hva er din, din fars og din mors etniske bakgrunn?

| | Norsk | Samisk | Annet, beskriv |
|----------|--------------------------|--------------------------|--------------------------------|
| Min | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Min fars | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Min mors | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

7. Hvilken type arbeid/livsopphold har du? (sett ett eller flere kryss)

- Fastlønnnet, heltid
 Fastlønnnet, deltid
 Sesongarbeid
 Selvstendig næringsdrivende
 Arbeidsledig
 Hjemmeværende
 Alderspensjon
 Uførepensjon
 Annet (beskriv):.....

8. Dersom du er selvstendig næringsdrivende, hvilken type næring jobber du i? (sett ett eller flere kryss)

- Forretningsvirksomhet
- Fiske
- Reindrift
- Jordbruk
- Annet (spesifiser):.....

9. Hvor stor er familiens/husstandens bruttoinntekt per år?

- Under kr 150 000
- Kr 150 000 – 300 000
- Kr 301 000 – 450 000
- Kr 451 000 – 600 000
- Kr 601 000 – 750 000
- Kr 751 000 – 900 000
- Over kr 900 000

10. Hva er din familiesituasjon?

- Eneforsørger uten hjemmeboende barn
- Eneforsørger med hjemmeboende barn
- Gift / samboer uten hjemmeboende barn
- Gift / samboer med hjemmeboende barn

11. Hvor mye penger tror du det maksimale du kan bruke på din tannbehandling hvert år?

- Mindre enn 500 kroner
- Kr 500 – 1000
- Kr 1001 – 2000
- Kr 2001 – 3000
- Kr 3001 – 7000
- mer enn 7000 kroner
- Prisen er uvesentlig

12. Hvordan bruker du tannhelsetjenesten?

- Blir regelmessig innkalt av tannlege eller tannpleier
- Melder meg regelmessig for undersøkelse
- Melder meg når jeg har vondt eller har mistet en fylling
- Bruker ikke å gå til tannlege så ofte

13. Har du i løpet av de siste 2 årene helt konkret utsatt å gå til tannlege fordi du ikke hadde penger til å betale regningen?

- Ja Nei

14. Dersom du vært hos tannlegen i løpet av de siste 2 årene, utførte du den behandlingen tannlegen anbefalte, eller førte kostnadene til at du enten avsto fra eller utførte rimeligere behandling enn du fikk anbefalt?

- Utførte den behandling tannlegen anbefalte
- Utførte rimeligere behandling enn anbefalt
- Avsto fra behandlingen på grunn av høye kostnader

15. Dersom du vært hos tannpleier i løpet av de siste 2 årene, utførte du den behandlingen tannpleieren anbefalte, eller førte kostnadene til at du enten avsto fra eller utførte rimeligere behandling enn du fikk anbefalt?

- Utførte den behandling tannpleieren anbefalte
- Utførte rimeligere behandling enn anbefalt
- Avsto fra behandlingen på grunn av høye kostnader

16. Hvor får du din tannbehandling utført?

- Offentlige tannhelsetjenesten
- Privat
- Både privat og offentlige tannhelsetjenesten
- Tannlegeutdanningen

17. Går du regelmessig til tannlege/tannpleie?

- Ja, mer enn en gang i året
- Ja, hvert år
- Ja, hvert annet år
- Ja, med lengre mellomrom enn 2 år
- Nei, bare for akutte problemer

18. Hvis du ikke går regelmessig, hva er den viktigste grunnen til dette? (Angi kun **ett** alternativ)?

- Har ikke hatt behov for tannbehandling
- Det er lang ventetid hos tannlegen
- Jeg har ikke blitt innkalt
- Avstanden til tannklinikken
- Mangler tannleger/tannpleier
- Økonomiske grunner
- Er ikke interessert
- Jeg er redd eller engstelig for å gå til tannlege/tannpleier
- Anser at det er ikke er nødvendig
- Det er ubehagelig
- Annet.....

19. Hvordan er helsen din? Sett ett kryss på en skala der 1 er svært dårlig og 5 svært god

| | | | | | | |
|--------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|-----------|
| | 1 | 2 | 3 | 4 | 5 | |
| Dårlig | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært god |

20. Hvordan er tannhelsen din? Sett ett kryss på en skala der 1 er svært dårlig og 5 svært god

| | | | | | | |
|--------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|-----------|
| | 1 | 2 | 3 | 4 | 5 | |
| Dårlig | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært god |

21. Er du fornøyd med tennene dine eller protesene? Angi svaret på en skala der 1 er svært misfornøyd og 5 er svært fornøyd

| | | | | | | |
|------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|---------------|
| | 1 | 2 | 3 | 4 | 5 | |
| Svært misfornøyd | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært fornøyd |

22. Bruker du regelmessig noen reseptbelagte medisiner?

- Nei
 Ja

Hvis du svaret Ja, hvilken eller hvilke reseptbelagte medisiner du tar regelmessig? Marker det eller de alternativene som passer

- Ja, blodtrykkssenkende medisin
 Ja, hjerte eller hjertekrampe medisin
 Ja, kolesterolsenkende midler
 Ja, beroligende middel eller sovemidler
 Ja, midler mot depresjon
 Ja, midler mot andre psykiske lidelser
 Ja, midler mot magesår eller magekatarr
 Ja, smertestillende
 Ja, kortison eller andre medisiner mot inflammasjoner
 Ja, midler mot astma/allergi
 Ja, p-piller
 Ja, hormon medisin (eks østrogen)
 Ja, for forbrenning/metabolismen (eks levaxin)
 Ja, insulin
 Ja, annen. Angi hvilken / hvilke

.....
Tar du regelmessig så kalte naturlegemidler?

- Ja
 Nei

23. Har du diabetes? Ja Nei

Hvis ja, når fikk du diagnosen? Angi årstall

Hvis ja, hvilken type av diabetes Type 1 Type 2

24. Røyker du daglig? Ja Nei

Hvis ja, hvor mange sigaretter røyker du per dag?

Hvor mange år har du røykt?

25. Bruker du snus? Ja Nei

Hvis ja, hvor mange doser bruker du i uka?

Hvor mange år har du brukt snus?

26. Hvor ofte drikker du følgende alkoholholdige drikker?

| | Ganger per måned | | | | Ganger per uke | | | | | | Ganger per dag | | |
|----------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| | 0 | 1 | 2 | 3 | 1 | 2 | 3 | 4 | 5 | 6 | 1 | 2 | 3+ |
| a. Øl | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| d. Sider | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| e. Rusbrus | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| e. Rødvvin /hvitvin | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| g. Dessertvin/likør/sherry | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| h. Sterksprit | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

27. Hvor ofte spiser du følgende matvarer?

| | 0 | 1 per uke | 2-3 per uke | 4-6 per uke | 1 per dag | 2-3 per dag | 4+ per dag |
|---|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| Is | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Smågodt, sjokolade, lakris etc. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Boller, småkaker, søte kjeks, lefser etc. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Potetgull / Ostepop / Tortillachips etc. | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Sukkerholdige pastiller/harde karameller | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Fersk frukt, tørket frukt | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

28. Hvor ofte drikker du følgende?

| | 0 | 1 per uke | 2-3 per uke | 4-6 per uke | 1 per dag | 2-3 per dag | 4+ per dag |
|--|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| Kaffe med sukker | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Te med sukker/honning | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Usukret brus (lett brus/kunstig søtet) | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Brus med sukker | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Drikk yoghurt, «Smoothie» | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Sjokoladedrikk | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Juice, nektar | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

29. Hvor ofte pusser du vanligvis tennene dine? (Angi kun **ett** alternativ)

- Sjeldnere enn 1 gang per uke
- 1 gang per uke
- 2-3 ganger per uke
- 4-6 ganger per uke
- 1 gang daglig
- 2 eller flere ganger daglig

30. Bruker du selv noen av følgende hjelpemidler- og i tilfelle hvor ofte?

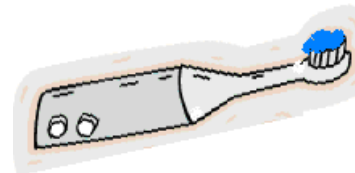
| | Sjeldnere/aldri | Noen ganger i måneden | Noen ganger i uka | Daglig |
|------------------|-----------------|-----------------------|-------------------|--------|
| Fluortannkrem | | | | |
| Tanntråd | | | | |
| Mellomromsbørste | | | | |
| Tannstikkere | | | | |
| Fluortabletter | | | | |
| Skyllevæske | | | | |
| Protesebørste | | | | |

31. Hvor mye tannkrem bruker du på tannbørsten din? Sett ett kryss for alternativet som best passer for deg.



Du som bruker elektrisk tannbørste, sett ett kryss for alternativet som best passer for deg.

- Legger tannkrem en gang på min elektriske tannbørste (som på bilden)
- Legger tannkrem to ganger på min elektriske tannbørste
- Legger tannkrem på tennene før jeg bruker en elektrisk tannbørste



32. Hvis du skyller munnen etter tannpuss, hvor mye vann bruker du? Sett ett kryss for alternativet som best passer for deg

En håndfull

To håndfull



Et halvt glass vann

Et fullt glass vann



33. Nedenfor følger noen utsagn. Vi er interessert i å vite hva som passer med din oppfatning i forhold til de ulike utsagnene? Angi svaret på en skala fra 1 til 7

- Å pusse tennene med fluortannkrem to ganger om dagen i to minutter forebygger tannkjøttbetennelse og hull i tennene:

Usannsynlig 1 2 3 4 5 6 7 Sannsynlig

- Å pusse tennene to ganger om dagen i to minutter med fluortannkrem i de neste seks månedene vil være:

Bortkastet tid 1 2 3 4 5 6 7 Vel brukt tid

- De nærmeste, familie og nære venner synes det er bra at jeg pusser tennene mine to ganger om dagen med fluortannkrem:

Helt uenig 1 2 3 4 5 6 7 Helt enig

- De fleste pusser tennene med fluortannkrem to ganger om dagen i to minutter i henhold til råd fra tannlege og tannpleieren:

Helt uenig 1 2 3 4 5 6 7 Helt enig

- Jeg har tenkt å pusse tennene med fluortannkrem to ganger om dagen i to minutter de neste seks månedene:

| | | | | | | | | |
|-------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Usannsynlig | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Sannsynlig |

Hvor sikker er du på at du vil pusse tennene to ganger om dagen i følgende situasjoner? Angi svaret på en skala fra 1 til 7.

- a) Når du er på ferie:

| | | | | | | | | |
|-------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|-------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Ikke sikker | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Helt sikker |

- b) Når du har mye å gjøre (f. eks veldig mye arbeid):

| | | | | | | | | |
|-------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|-------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Ikke sikker | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Helt sikker |

34. Mestring. Dette er noen spørsmål som er rettet til forskjellige aspekter ved våre liv. Hvert spørsmål har syv mulige svar. Vær snill å merke av det tallet som uttrykker best ditt svar, tallene 1 og 7 er de mest ytterliggående. Dersom utsagnet under tall 1 er det rette for deg, sett ett kryss under tallet 1. Dersom utsagnet under tall 7 er det rette for deg, sett ett kryss under tallet 7. Hvis du føler noe annet, sett ett kryss ved det tallet som best uttrykker det du føler. Vær vennlig å gi bare ett svar til hvert spørsmål.

- Føler du i bunn og grunn at du ikke bryr deg om hva som skjer rundt deg?

| | | | | | | | | |
|---------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|------------|
| | 7 | 6 | 5 | 4 | 3 | 2 | 1 | |
| Svært sjelden eller aldri | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært ofte |

- Har det hendt at du var overrasket over hvordan personer som du trodde du kjente godt, oppførte seg?

| | | | | | | | | |
|-------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------|
| | 7 | 6 | 5 | 4 | 3 | 2 | 1 | |
| Aldri | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Alltid |

- Har det hendt at du ble skuffet over personer som du stolte på?

| | | | | | | | | |
|-------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------|
| | 7 | 6 | 5 | 4 | 3 | 2 | 1 | |
| Aldri | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Alltid |

- Inntil nå har livet ditt hatt:

| | | | | | | | | |
|-------------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|----------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Ingen klare mål eller hensikt | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Meget klare mål og hensikt |

- Føler du at du blir urettferdig behandlet?

| | | | | | | | | |
|------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|---------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Svært ofte | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært sjelden eller aldri |

- Hvor ofte føler du at du er i en uvant situasjon og at du ikke vet hva du skal gjøre?

| | | | | | | | | |
|------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|---------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Svært ofte | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært sjelden eller aldri |

- Å utføre dine daglige gjøremål er:

| | | | | | | | | |
|--|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|-----------------------------------|
| | 7 | 6 | 5 | 4 | 3 | 2 | 1 | |
| En kilde til stor glede og tilfredsstillelse | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | En kilde til smerte og kjedsomhet |

- Har du svært motstridende følelser og tanker?

| | | | | | | | | |
|------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|---------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Svært ofte | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært sjelden eller aldri |

- Hender det at du har følelser inni deg som du ikke ønsker å ha?

| | | | | | | | | |
|------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|---------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Svært ofte | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært sjelden eller aldri |

- Mange mennesker, selv karaktersterke, føler seg noen ganger som tapere i visse situasjoner. Hvor ofte har du følt det slik?

| | | | | | | | | |
|-------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|------------|
| | 7 | 6 | 5 | 4 | 3 | 2 | 1 | |
| Aldri | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Svært ofte |

- Når noe har hendt, har du vanligvis oppdaget at du:

| | | | | | | | | |
|---|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|------------------------|
| | 1 | 2 | 3 | 4 | 5 | 6 | 7 | |
| Overvurderte eller undervurderte betydningen av det | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | Du vurderte det riktig |

- Hvor ofte føler du at det er liten mening i de tingene du gjør daglig?

1 2 3 4 5 6 7

Svært ofte Svært sjelden eller aldri

- Hvor ofte har du følelser som du ikke er sikker på at du kan holde under kontroll?

1 2 3 4 5 6 7

Svært ofte Svært sjelden eller aldri

35. Hvor viktig er tennene for deg utfra følgende fem utsagn? Angi svaret på en skala der 1 er svært uviktig og 5 er svært viktig.

- At tennene er pene når jeg snakker og smiler

1 2 3 4 5

Svært uviktig Svært viktig

- At jeg kan tygge uten problemer

1 2 3 4 5

Svært uviktig Svært viktig

- At jeg har frisk pust/god ånde

1 2 3 4 5

Svært uviktig Svært viktig

- At jeg ikke får hull i tennene mine

1 2 3 4 5

Svært uviktig Svært viktig

- At jeg har friskt tannkjøtt

1 2 3 4 5

Svært uviktig Svært viktig

36. Nedenfor stilles noen spørsmål om du i løpet av det siste året har hatt noen problemer eller ubehag på grunn av dine tenner eller protese (gebiss) eller på grunn av andre forhold i munnen.

| | Aldri | Sjelden | Av og til | Ganske ofte | Ofte |
|---|--------------------------|--------------------------|--------------------------|--------------------------|--------------------------|
| Har du på grunn av dine tenner, forhold i munnen eller protesen: | | | | | |
| - Opplevd at mat har gitt deg ubehag? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Hatt en dårlig kost/kostsammensetning? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Måttet avbryte måltider? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Hatt vanskeligheter med å uttale ord eller lage spesielle lyder? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Har din smaksans blitt endret/dårligere på grunn av dine tenner, forhold i munnen eller protesen? | | | | | |
| <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Har du på grunn av dine tenner, forhold i munnen eller protesen | | | | | |
| - Følt deg usikker? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Følt deg spent eller stresset? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Hatt problemer med å slappe av? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Kjent deg brydd / flau? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Har du i løpet av det siste året hatt smerte eller vondt i tennene, i munnen eller på grunn av protesen? | | | | | |
| <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| Har du på grunn av dine tenner, forhold i munnen eller protesen | | | | | |
| - Vært irritabel overfor andre mennesker? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Hatt vanskeligheter med dine vanlige gjøremål? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Følt at livet i sin alminnelighet var mindre tilfredsstillende? | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |
| - Ikke kunnet å fungere i hverdagen | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> | <input type="checkbox"/> |

37. Dersom du visste at du skulle til tannlegen i morgen, hva ville du føle? (Angi kun **ett** alternativ)

- Jeg ville se frem til det som en ganske hyggelig opplevelse
- Det ville være det samme for meg, ikke bety noe
- Det ville gjøre meg litt urolig
- Jeg ville bli redd for at det skulle bli ubehagelig og vondt
- Jeg ville bli svært redd med tanke på hva tannlegen kanskje skulle gjøre

Når du venter på tannlegens venteværelse, eller venter på å bli hentet til tannlegen, hvordan føler du deg da? (Angi kun **ett** alternativ)

- Avslappet
- Litt urolig
- Anspent, nervøs
- Redd, engstelig
- Så redd at jeg av og til begynner å svette eller nesten føler meg syk

Når du sitter i tannlegestolen og venter på at tannlegen skal begynne behandlingen, hvordan føler du deg da? (Angi kun **ett** alternativ)

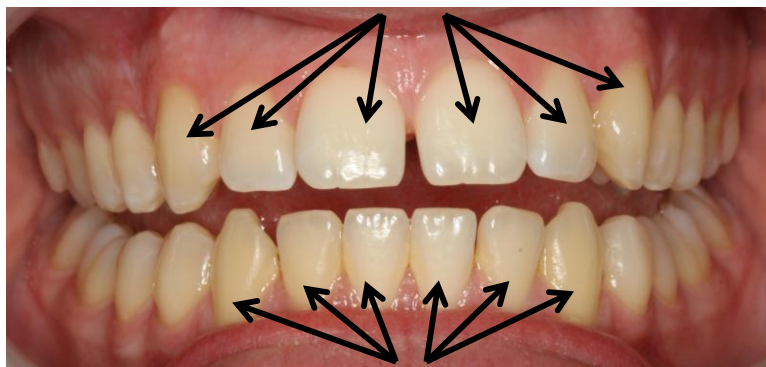
- Avslappet
- Litt urolig
- Anspent, nervøs
- Redd, engstelig
- Så redd at jeg av og til begynner å svette eller nesten føler meg syk

Tenk deg at du sitter i tannlegestolen og skal få tennene rensset og pusset. Mens du sitter og venter på at tannlegen skal finne instrumentene som brukes til å skrape og pusse med, hvordan føler du deg da? (Angi kun **ett** alternativ)

- Avslappet
- Litt urolig
- Anspent, nervøs
- Redd, engstelig
- Så redd at jeg av og til begynner å svette eller nesten føler meg syk

38. Hvis du har dine egne tenner og mistet en eller flere tenner, hvor viktig er det å erstatte tap av en eller flere tenner om det gjelder en tann i fortannsområdet (de tenner markerte nedenfor med piler på bildet)? Angi på en skala der 1 er svært uviktig og 5 svært viktig

1 2 3 4 5
 Svært uviktig Svært viktig



Om det gjelder en tann i sidene (de tenner markerte nedenfor med piler på bildet)? Angi på en skala der 1 er svært uviktig og 5 svært viktig.

1 2 3 4 5
 Svært uviktig Svært viktig



Om det gjelder en tann lengre bak i munnen (de tenner markerte nedenfor med piler på bildet)? Angi på en skala der 1 er svært uviktig og 5 svært viktig.

1 2 3 4 5
 Svært uviktig Svært viktig



39. Dette spørsmålet besvares bare om du har dine egne originale tenner intakt.

Hvis du mister en eller flere tenner, hva slags behandling foretrekker du?
(Angi kun ett alternativ)

- Fast bru på egne naturlige tenner
- Avtakbar gebiss
- Krone eller bru på implantater
- Ingen behandling

40. Hvis du har tannerstatninger i form av en fast bro / brygge eller avtakbar gebiss, er du generelt fornøyd eller misfornøyd med?

- Ja, veldig fornøyd
- Ja, ganske fornøyd
- Nei, ikke veldig fornøyd
- Nei, ikke i det hele tatt fornøyd
- Har ingen slik erstatning

41. Hvis du mangler en eller flere tenner, og har valgt å ikke erstatte dem, hvorfor har du ikke erstattet dem? Flere alternativer kan velges.

- Har ikke ønsket noen erstatning
- Behandling er for dyrt
- Har prøvd løstenner/gebiss, men det har ikke fungert
- Er blitt frarådet av tannlege
- Har takket nei på grunn av frykt for tannbehandling

42. Dette spørsmålet er for deg som mangler en eller flere tenner og allerede har en erstatning for dem. Hvis du ikke ønsker implantatbehandling, beskriv det alternativ som passer best for deg?

- Implantater er for stort inngrep i kroppen
- Implantater er for dyrt
- Jeg er redd operasjoner
- Jeg er redd for ukjente bivirkninger av implantater
- Erstatningen jeg har fungerer fint
- Annet.....

43. Har du i det siste året opplevd klikk / lyder fra kjeveledd?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

44. Har du i det siste året hatt smerter fra kjeven eller ansikt?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

45. Har du i det siste året opplevd at det var vanskelig å gape stort?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

46. Har du i det siste året opplevd at kjeven er sperret/låst?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

47. Har du i det siste året opplevd en stikkende/brennende følelse i munnen?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

48. Har du i det siste året opplevd munntørighet?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

49. Har du i det siste året hatt hodepine?

- Nei
- Ja, noen ganger
- Ja, omtrent en gang i uka
- Ja, flere ganger i uka
- Ja, daglig

Takk for ditt verdifulle bidrag!

Alle oppgaver i spørreskjemaet og ved undersøkelsen vil bli behandlet konfidensielt.

Har Du spørsmål kan Du ringe til noen av personene nedenfor eller sende en e-post

Nils Oscarson

Gro Eirin Olsen Holde

77 78 90 00 (resepsjon)

77 78 90 30 (resepsjon)

nils.oscarson@tromsfylke.no

gro.olsen.holde@tromsfylke.no

Spørsmål om bruk av tannhelsetjenesten

1. Er det vanskelig for deg å gå til rutinemessige tannhelsesjekker?

- Ja
- Vet ikke, husker ikke
- Nei

2. Dersom du skulle til tannlegen i morgen, tror du at du ville hatt behov for tannbehandling da?

- Ja, jeg ville trengt behandling
- Vet ikke
- Nei, jeg ville ikke trengt behandling

3. Går de nærmeste, familie og nære venner regelmessig til tannlege/tannpleie?

- Ja
- Vet ikke
- Nei

4. Synes de nærmeste, familie og nære venner at du skal gå regelmessig til tannlege/tannpleie?

- Ja
- Vet ikke
- Nei

5. Hvor stor betydning har avstanden til tannklinikken for at du skal komme dit på regelmessige kontroller og behandlinger?

- Avgjørende betydning
- Stor betydning
- Liten betydning
- Ingen betydning

Appendix 2

Ethical approval

Region:
REK nord

Saksbehandler:

Telefon:

Vår dato:
08.04.2013
Deres dato:
19.02.2013

Vår referanse:
2013/348/REK nord
Deres referanse:

Vår referanse må oppgis ved alle henvendelser

Nils Oscarson

2013/348 Tromstannen-munnhelse i Troms Fylkeskommune

Forskningsansvarlig: Tannhelsetjenesten

Prosjektleder: Nils Oscarson

Vi viser til søknad om forhåndsgodkjenning av ovennevnte forskningsprosjekt. Søknaden ble behandlet av Regional komité for medisinsk og helsefaglig forskningsetikk (REK nord) i møtet 21.03.2013. Vurderingen er gjort med hjemmel i helseforskningsloven (hfl.) § 10, jf. forskningsetikklovens § 4.

Prosjektleders prosjekttale

I en rapport fra Nasjonalt folkehelseinstitutt (2009) konstateres at man vet relativt lite om voksne befolkningens tannhelse i Norge. Videre konstateres at en viss andel av befolkningen av ulike grunner ikke går til tannlege. Sosial- og Helsedirektoratet (2005) slår i sin rapport «Gradientutfordringen» fast sammenhengen mellom sosiale ulikheter eller sosioøkonomiske forskjeller og tannhelse. Kunnskap om tannhelseforhold, eventuelle forskjeller i helse og årsakssammenhenger, er nødvendig for å kunne planlegge tannhelsetjenesten. Tannhelsetjenesten bør kunne tilby alle forskjellige grupper som lever etter ulike kulturelle og yrkesmessige livsbetingelser, et likeverdig tannbehandlingstilbud, basert på den enkeltes spesifikke behov. Det vil øke muligheten for en fremtidig god munnhelse for alle. Det overordnede målet med prosjektet er å kartlegge tannhelseforhold, inkludert mulige risikofaktorer- og årsakssammenhenger, i den voksne befolkningen (20-79 år) i Troms fylkeskommune.

Vurdering

Komiteen har ingen innvendinger til prosjektet.

Vedtak

Med hjemmel i helseforskningsloven § 10 og forskningsetikkloven § 4 godkjennes prosjektet.

Sluttmelding og søknad om prosjektendring

Prosjektleder skal sende sluttmelding til REK nord på eget skjema senest 30.06.2021, jf. hfl. § 12. Prosjektleder skal sende søknad om prosjektendring til REK nord dersom det skal gjøres vesentlige endringer i forhold til de opplysninger som er gitt i søknaden, jf. hfl. § 11.

Klageadgang

Du kan klage på komiteens vedtak, jf. forvaltningslovens § 28 flg. Klagen sendes til REK nord. Klagefristen er tre uker fra du mottar dette brevet. Dersom vedtaket opprettholdes av REK nord, sendes klagen videre til Den nasjonale forskningsetiske komité for medisin og helsefag for endelig vurdering.

Med vennlig hilsen

May Britt Rossvoll
sekreteriatsleder

Kopi til: peter.marstrander@tromsfylke.no

