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# Prevalence of periodontitis and DMFT index in patients with Crohn's disease and ulcerative colitis

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***Abstract***

**Aim:** To compare the prevalence of periodontal disease and the decayed, missing and filled teeth (DMFT) index in patients with Crohn's disease (CD) and ulcerative colitis (UC) with those without these diseases.

**Material and Methods:** Ninety-nine CD (39.0 SD ± 12.9 years), 80 UC (43.3 SD ± 13.2) and 74 healthy controls (40.3 SD ± 12.9) were compared for DMFT index and presence of periodontitis. Probing pocket depth (PPD), clinical attachment loss (CAL), bleeding on probing (BOP), plaque and DMFT index were measured on all subjects. The presence of periodontitis was defined as having CAL $\geqslant 3$ mm in at least four sites in different teeth.

**Results:** Significantly more patients with UC (90.0%; $p < 0.001$) and CD (81.8%; $p = 0.03$) had periodontitis than controls (67.6%). Among smokers, UC patients had significantly more periodontitis. CD had a greater mean DMFT score (18.7 *versus* 13.9; $p = 0.031$) compared with controls and UC had greater median PPD (2.2 *versus* 1.7 mm; $p < 0.0001$) than controls. Among non-smokers, CD (2.4 mm; $p < 0.0001$) and UC showed deeper pockets (2.3 mm; $p < 0.0001$) compared with controls (1.5 mm). UC had a greater mean DMFT score (15.3 *versus* 12.1; $p = 0.037$) compared with controls.

**Conclusions:** CD and UC patients had higher DMFT and prevalence of periodontitis than controls, but smoking was an effect modifier.

Key words: attachment loss; Crohn's disease; DMFT; inflammatory bowel disease; oral lesions; periodontitis; ulcerative colitis

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Inflammatory bowel disease (IBD) encompasses two distinct chronic intestinal disorders: Crohn's disease (CD) and ulcerative colitis (UC) (Podolsky 2002, Bouma & Strober 2003). The pathogenesis of IBD is still elusive as no agent or mechanism has explained all aspects of the disease. However, it is known that distinct immune abnormalities play a major role in the initiation and perpetuation of IBD (MacDonald et al. 2000).

Extra-intestinal manifestations in both forms of IBD can occur in the joints, eyes, skin, mouth and liver (Greenstein et al. 1976, Veloso et al. 1996, Jiang et al. 2006). Oral lesions may coincide, precede or follow the onset of the intestinal symptoms (Greenstein et al. 1976, Ghandour & Issa 1991, Williams et al. 1991). The prevalence of oral manifestations in IBD varies between 0% and 9% in adults (Basu 1976, Greenstein et al. 1976, Lisciandrano et al. 1996, Dupuy et al. 1999).

A high prevalence of caries has been reported in CD patients (Bevenius 1988, Sundh & Emilson 1989, Schütz et al. 2003); in comparison, little or no information is known about the prevalence of dental caries in UC patients. To date,

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only two cross-sectional studies have been performed examining the prevalence and severity of periodontitis in patients with IBD (Flemmig et al. 1991, Grössner-Schreiber et al. 2006). Flemmig et al. (1991) examined 46 CD patients and 61 UC patients. The periodontal examination was carried out at two sites (mid – and mesiobuccal) of all present teeth in two quadrants. They showed that periodontitis was more prevalent but not more severe in this population. However, there was no control group and the results were compared with the assessment of Oral Health of United States Adults. The study by Grössner-Schreiber et al. (2006) examined 62 patients with IBD and compared the results with 59 matched healthy controls, and the periodontal examination was performed in two quadrants. They found that patients with IBD had more sites of periodontitis with attachment loss of at least 4 and 5 mm although no significant differences were found between IBD patients and healthy controls. Furthermore, in this study no distinction was found between CD and UC patients.

Periodontitis and IBD are multifactorial diseases. It is known that both result from an aberrant immune response in a susceptible host that may also be influenced by environmental factors (MacDonald et al. 2000, Bouma & Strober 2003, Kinane & Mark Bartold 2007). Because the key mediators involved in tissue damage in both diseases are common, such as cytokines and matrix metalloproteinases (Pallone & Monteleone 2001, Kinane & Mark Bartold 2007), we hypothesized that patients with IBD could be more susceptible to periodontitis. However, as CD and UC have markedly different features (Bouma & Strober 2003), information is lost when they are combined as IBD and analysed as one disease; hence, they should be evaluated separately.

Therefore, the purpose of this study was to determine the prevalence of periodontitis and the DMFT index of CD and UC patients through a full-mouth examination and compare them with the findings from systemic healthy controls.

## Material and Methods

### Subjects

Ninety-nine CD patients and 80 UC patients participated in the study. The diagnosis of CD or UC had been established previously by clinical, radiological, endoscopic and histological analyses. All these individuals were out-patients attending the inflammatory bowel disease clinics at Pedro Ernesto University Hospital of the Rio de Janeiro State University (HUPE-UERJ) and the Clementino Fraga Filho University Hospital of Federal University of Rio de Janeiro (CFFUH-UFRJ), both in Rio de Janeiro, Brazil.

Seventy-four age-matched systemically healthy individuals who worked at the University Hospitals comprised the control group. The participants of the control group did not show any clinical signs of ongoing systemic disease.

Edentulous individuals, patients requiring antibiotic prophylaxis before the periodontal examination and pregnant women were excluded from this study. Participants enrolled in this study also signed a written informed consent. When the patient was $<18$ years old, one of the parents or the guardians consented. The protocol was reviewed and approved by the Committee on Ethics and Research of the HUPE-UERJ and CFFUH-UFRJ. Eligible participants were recruited from January 2005 to 2006.

### Clinical data

A questionnaire covering demographic data including age, sex, medical history, medication used and smoking habits was administered. The smoking habit was registered as current smokers (those who currently smoke cigarettes daily or occasionally), non-smokers (those who have never smoked cigarettes) and former smokers (those who have smoked cigarettes but who currently do not smoke) (Ojima et al. 2006). In the present study, occasionally refers to the subjects who said that they smoke only during the weekends. Current smokers were asked about cigarette consumption in terms of the number of cigarettes per day.

After the clinical examination, the medical records were reviewed in order to check the information obtained from the patients and to collect information concerning their disease activity. Crohn's Disease Activity Index (CDAI) (Best et al. 1976) was used to assess disease activity in CD patients, whereas Truelove and Witts' index (Truelove & Witts 1955) was used to assess disease activity in UC patients. A CDAI higher than 150 represents active disease in CD patients. The presence of more than six episodes of bloody diarrhoea, fever, anaemia (haemoglobin $<7.5$ g/dl), cardiac frequency $>90$ b.p.m. and erythrocyte sedimentation rate (ESR) $>30$ mm per first hour was considered to indicate active disease in UC patients.

In the CD group, some patients were not taking any medication ($n=9$), those who were used aminosalicylates ($n=26$), immunomodulators ($n=21$), aminosalicylates+immunomodulators ($n=17$), aminosalicylates+corticosteroids ($n=8$), immunomodulators+corticosteroids ($n=9$) and aminosalicylates+immunomodulators+corticosteroids ($n=9$). In the UC group, patients used aminosalicylates ($n=52$), immunomodulators ($n=4$), aminosalicylates+immunomodulators ($n=9$), aminosalicylates+corticosteroids ($n=9$), immunomodulators+corticosteroids ($n=2$) and aminosalicylates+immunomodulators+corticosteroids ($n=4$). In addition, seven CD and three UC patients were using anti-TNFα, four CD and one UC patients were using metronidazol and four CD patients were using ciprofloxacin in combination with the above medication.

### Oral examination

The clinical examination was performed in all present teeth except for the third molars. The following periodontal parameters were measured at six sites (mesio-buccal, buccal, disto-buccal, mesio-lingual, lingual and disto-buccal) for each tooth: presence of plaque, presence of bleeding on probing (BOP), probing pocket depth (PPD) and clinical attachment loss (CAL). PPD (the distance from the gingival margin to the bottom of the pocket) and CAL (the distance from the cementoenamel junction (JCE) to the bottom of the pocket/sulcus) were measured using a conventional periodontal probe (Hu-Friedy®, Chigaco, IL, USA) -15. In the cases where the CEJ was not visible, for example, by a restoration, the position of CEJ was estimated by extrapolating the position of the CEJ from the adjacent teeth. Periodontitis was defined as the presence of at least four sites in different teeth with CAL $\geqslant 3$ mm.

The teeth were examined for caries according to the DMFT index using the World Health Organization (WHO) criteria, and the intra-oral examination for assessment of mucosa lesions was also performed following WHO recommendations (WHO 1997).

*Table 1.* Demographic data of 99 CD patients, 80 UC patients and 74 healthy controls

| | Crohn's disease ($n = 99$) | p1 | Controls ($n = 74$) | p2 | Ulcerative colitis ($n = 80$) | p3 |
|---|---|---|---|---|---|---|
| Age | 39.0 (12.9) | 0.514 (*t*-test) | 40.3 (12.9) | 0.098 (*t*-test) | 43.3 (13.2) | 0.032 (*t*-test) |
| Male | 31 (31.3%) | 0.876 | 24 (32.4%) | 0.259 | 33 (41.3%) | 0.169 |
| Female | 68 (68.7%) | 0.876 | 50 (67.6%) | 0.259 | 47 (58.7%) | 0.169 |
| White | 61 (61.6%) | 0.001 | 27 (36.5%) | 0.005 | 47 (58.7%) | 0.697 |
| Mixed | 32 (32.3%) | 0.100 | 33 (44.6%) | 0.293 | 29 (36.2%) | 0.582 |
| Black | 6 (6.0%) | 0.009 | 14 (18.9%) | 0.007 | 4 (5.0%) | 0.759 |
| Family history | 12 (12.1%) | | – | | 11 (13.7%) | 0.746 |
| Smokers | 12 (12.1%) | 0.993 | 9 (12.2%) | 0.489 | 7 (8.7%) | 0.467 |
| Non-smokers | 63 (63.6%) | 0.043 | 57 (77.0%) | $p < 0.0001$ | 38 (47.5%) | 0.043 |
| Former smokers | 24 (24.3%) | 0.017 | 8 (10.8%) | $p < 0.0001$ | 35 (43.8%) | 0.009 |
| Time of diagnosis (months) | 72 (96) | | – | | 72 (84) | 0.795 (Mann–Whitney) |
| Hypertension | 18 (18.2%) | $p < 0.0001$ | 0 | $p < 0.0001$ | 17 (21.2%) | 0.607 |
| Diabetes | 0 | | 0 | 0.092 | 3 (3.7%) | 0.052 |
| Extra-intestinal manifestations | 50 (50.5%) | | – | | 42 (52.5%) | 0.791 |
| Active disease | 22 (22.2%) | | – | | 19 (23.7%) | 0.809 |

p1: significance of the difference between Crohn's disease (CD) patients and controls, p2: significance of the difference between ulcerative colitis (UC) patients and controls and p3: significance of the difference between CD and UC patients. Test was $\chi^2$ unless otherwise stated.
The number of subjects, and percentage for all the parameters except for age, the data is expressed as mean and standard deviation (SD). Time of diagnosis is expressed as median and quartile range.

Two periodontists involved in the examination achieved substantial inter – examiner reproducibility ($\kappa = 0.775$, $p < 0.001$) for all the variables analysed.

#### Statistical analysis

A post-hoc calculation showed that with a sample size of 253, across the three groups, a one-way analysis of variance would have 99% power to detect, at the 0.05 level, a difference in mean PPD.

Normally distributed data were displayed as mean and standard deviation whereas non-normally distributed data were displayed as median and quartile range. Differences in the frequency of subjects within groups were assessed by the $\chi^2$ test. The *t*-test was used to for parametric data and the Mann–Whitney *U* test was used to compare non-parametric data. The significance level was set at $p < 0.05$. BOP, PPD and CAL were log transformed and analysis test of covariance (ANCOVA) with adjustments for race, gender, age, plaque and smoking habit was performed. Analyses were performed using by software package STATISTICA (StatSoft, Inc. 2005) version 7.1.

## Results

#### Clinical data

The detailed demographic data from UC, CD and controls patients are reported in Table 1. CD patients showed a significantly higher DMFT index ($p = 0.018$), less sites with plaque ($p = 0.017$) and BOP ($p = 0.038$), deeper PPD ($p < 0.0001$), and there were more subjects with periodontitis ($p = 0.03$) when compared with the controls (Table 2A).

UC patients had significantly fewer teeth ($p = 0.002$), a higher DMFT index ($p < 0.0001$), deeper PPD ($p < 0.0001$), more CAL ($p = 0.004$), more subjects with periodontitis ($p < 0.001$) and more sites with CAL $\geqslant 3$ mm than controls ($p = 0.007$). Furthermore, UC patients had significantly higher CAL ($p = 0.005$) and a significantly higher number of sites with CAL $\geqslant 3$ mm ($p = 0.006$) than CD patients (Table 2A).

After adjustments for race, gender, smoking habit, age and plaque, CD and UC patients still had a significantly higher DMFT index ($p < 0.0001$, $p = 0.003$, respectively) and deeper PPD ($p < 0.0001$, for both) than controls. UC patients had significantly more CAL ($p = 0.02$) than controls. After the same adjustments, when compared with CD, UC patients had a significantly higher DMFT index ($p < 0.0001$) and a significantly deeper PPD ($p < 0.0001$).

Among non-smokers (Table 2B), CD patients showed significantly fewer sites with plaque ($p = 0.033$) and deeper PPD ($p < 0.0001$) when compared with controls. UC patients had a greater DMFT score ($p = 0.037$) and deeper pockets ($p < 0.0001$) compared with controls. After adjustment for race, gender, age and plaque, both IBD groups showed deeper PPD ($p < 0.001$ for both) and greater DMFT scores ($p = 0.023$ for CD and $p = 0.005$ for UC) than controls.

Among smokers (including former smokers), CD patients had a significantly greater DMFT score ($p = 0.031$) and fewer sites with BOP ($p = 0.049$) compared with controls. UC patients had deeper pockets ($p < 0.0001$) than controls and slightly more sites with CAL $\geqslant 3$ mm ($p = 0.076$) than CD patients (Table 2C). After adjustment for race, gender, age and plaque, CD and UC patients had greater DMFT scores ($p = 0.003$ and 0.04) as compared with controls while only UC patients showed deeper pockets ($p < 0.001$).

Periodontitis was more common among smoking patients with UC (95.2% *versus* 70.6%, $p = 0.008$) odds ratio 9.60 (95% confidence interval 1.38–66.82) as compared with smoking controls. Periodontitis was also more common among non-smoking UC patients than controls (84.2% *versus* 66.7%) but the difference did not reach significance ($p = 0.057$).

#### Oral lesions

No difference was apparent between the groups in the number of subjects with oral lesions, and consequently no

*Table 2*. Median (quartile range) for the number of teeth, percentage of sites with plaque, percentage of sites with bleeding on probing (BOP), probing pocket depth (PPD), clinical attachment loss (CAL), subjects with periodontitis and percentage of sites with AL $\geqslant$3 mm, and mean (standard deviation) for the decayed, missing and filled tooth (DMFT) index in CD patients, UC patients and healthy controls

| | Crohn's disease ($n = 99$) | p1 | Controls ($n = 74$) | p2 | Ulcerative colitis ($n = 80$) | p3 |
|---|---|---|---|---|---|---|
| *(A) All subjects* | | | | | | |
| Number of teeth | 24.0 (9.0) | 0.133 | 25.0 (6.0) | 0.002 | 22.0 (10.0) | 0.086 |
| DMFT index | 15.1 (7.2) | 0.018 | 12.5 (6.8) | <0.0001 | 16.4 (6.6) | 0.229 |
| % of sites with plaque | 38.2 (47.4) | 0.017 | 52.7 (41.6) | 0.479 | 53.7 (60.4) | 0.239 |
| % of sites with BOP | 19.6 (20.5) | 0.038 | 24.4 (29.7) | 0.265 | 21.5 (21.9) | 0.308 |
| PPD (mm) | 2.3 (1.3) | <0.0001 | 1.6 (0.4) | <0.0001 | 2.3 (0.4) | 0.941 |
| CAL (mm) | 0.9 (0.9) | 0.576 | 1.2 (1.0) | 0.004 | 1.3 (1.4) | 0.005 |
| Subjects with periodontitis | 81 (81.8%) | 0.030 | 50 (67.6%) | <0.001 | 72 (90.0%) | 0.122 |
| % sites CAL $\geqslant$3 mm | 13.2 (21.8) | 0.655 | 11.7 (26.5) | 0.007 | 22.8 (39.3) | 0.006 |
| | Crohn's disease ($n = 63$) | p1 | Controls ($n = 57$) | p2 | Ulcerative colitis ($n = 38$) | p3 |
| *(B) Non-smokers* | | | | | | |
| Number of teeth | 25.0 (9.0) | 0.453 | 26.0 (6.0) | 0.124 | 24.0 (10.0) | 0.337 |
| DMFT index | 13.0 (6.7) | 0.483 | 12.1 (7.2) | 0.037 | 15.3 (7.0) | 0.120 |
| % of sites with plaque | 37.5 (47.3) | 0.033 | 51.7 (33.3) | 0.566 | 38.3 (79.2) | 0.516 |
| % of sites with BOP | 20.4 (19.0) | 0.234 | 23.8 (29.2) | 0.670 | 22.0 (19.0) | 0.430 |
| PPD (mm) | 2.4 (1.1) | <0.0001 | 1.5 (0.3) | <0.0001 | 2.3 (0.3) | 0.336 |
| CAL (mm) | 0.7 (0.8) | 0.808 | 0.7 (0.9) | 0.092 | 1.0 (1.4) | 0.108 |
| Subjects with periodontitis | 50 (79.4%) | 0.116 | 38 (66.7%) | 0.057 | 32 (84.2%) | 0.546 |
| % sites CAL $\geqslant$3 mm | 10.0 (18.7) | 0.910 | 8.7 (25.3) | 0.140 | 14.7 (40.9) | 0.162 |
| | Crohn's disease ($n = 36$) | p1 | Controls ($n = 17$) | p2 | Ulcerative colitis ($n = 42$) | p3 |
| *(C) Smokers* | | | | | | |
| Number of teeth | 23.0 (11.0) | 0.306 | 23.0 (5.0) | 0.067 | 19.5 (8.0) | 0.533 |
| DMFT index | 18.7 (6.4) | 0.031 | 13.9 (4.9) | 0.228 | 17.4 (6.2) | 0.396 |
| % of sites with plaque | 53.2 (45.6) | 0.084 | 66.7 (42.5) | 0.183 | 54.2 (50.4) | 0.527 |
| % of sites with BOP | 17.3 (22.2) | 0.049 | 25.0 (26.2) | 0.218 | 20.7 (26.1) | 0.359 |
| PPD (mm) | 2.0 (1.8) | 0.179 | 1.7 (0.5) | <0.0001 | 2.2 (0.5) | 0.249 |
| CAL (mm) | 1.1 (1.1) | 0.804 | 1.4 (1.3) | 0.402 | 1.6 (1.4) | 0.102 |
| Subjects with periodontitis | 31 (86.1%) | 0.177 | 12 (70.6%) | 0.008 | 40 (95.2%) | 0.159 |
| % sites CAL $\geqslant$3 mm | 22.8 (27.6) | 0.826 | 22.4 (39.7) | 0.436 | 31.7 (33.8) | 0.076 |

p1: significance of the difference between Crohn's disease (CD) patients and controls, p2: significance of the difference between ulcerative colitis (UC) patients and controls and p3: significance of the difference between CD and UC patients. Mann–Whitney $U$ test was used to comparisons except for DMFT index and for subjects with periodontitis in which $t$-test and $\chi^2$ test were used, respectively.

relationship was found between disease activity and the higher prevalence of oral lesions either in CD or in UC patients. Altogether, there were 20 patients with candidiasis lesions (CD $n = 8$, UC $n = 8$, controls $n = 4$), three with ulcerous aphtous (CD $n = 2$, UC $n = 1$) and five with lichen planus lesions (CD $n = 1$, UC $n = 3$, controls $n = 1$).

In CD group, the patients who had candidiasis were taking aminosalicylates ($n = 2$), immunomodulators ($n = 2$), aminosalicylates+immunomodulators ($n = 2$), aminosalicylates+corticosteroids+ciprofloxacin ($n = 1$) and aminosalicylates+immunomodulators+corticosteroids ($n = 1$). The patients who had ulcerous aphtous were taking aminosalicylates ($n = 1$) and a combination of aminosalicylates+immunomodulators+corticosteroids ($n = 1$). The patient who had lichen planus was taking aminosalicylates ($n = 1$). In the UC group, all the patients who had candidiasis were taking aminosalicylates ($n = 8$) and also the patient who had ulcerous aphtous ($n = 1$). The patients who had lichen planus were taking aminosalicylates ($n = 1$) and immunomodulators ($n = 2$).

## Discussion

The current study revealed that patients with UC and CD had a significantly worse oral health than matched controls. Our definition of periodontitis was based on the criteria of the American Academy of Periodontology (Lindhe et al. 1999). In an attempt to avoid false-positive cases, we considered only patients with a minimum of four sites and at least 3 mm of CAL in different teeth as having periodontitis. According to these criteria, the prevalence of periodontitis was significantly higher in both patient groups compared with the controls. This is in line with the study by Flemmig et al. (1991), who showed a slightly higher prevalence of periodontitis in patients with IBD. Grössner-Schreiber et al. (2006) also showed that patients with IBD had slightly more sites with CAL than controls. The high prevalence of periodontitis in our control group is in accordance with an earlier population study in Brazilian adults in which 97.4% of the subjects had CAL $\geqslant$3 mm (Susin et al. 2004).

Smokers (former and current) in all three groups had more periodontitis. Both non-smokers CD and UC patients showed deeper pockets compared with the controls after adjustments for race,

gender, smoking habit, age and plaque. This is in contrast with Grössner-Schreiber et al. (2006), who found deeper pockets in the control group compared with patients with IBD. The median pocket depth and CAL found in our patients were similar to those reported by Flemmig et al. (1991) and Grössner-Schreiber et al. (2006). However, we showed a higher prevalence of disease between the groups studied compared with the controls. A possible explanation could be that we used whole-mouth examination (six sites per tooth in all existing teeth except for the third molars), which is currently considered the gold standard. Beck et al. (2006) showed that the prevalence of periodontitis is underestimated in half-mouth studies, especially in younger populations.

UC patients, both smokers and non-smokers, had a tendency of more CAL and more sites with CAL $\geqslant 3$ mm compared with CD, which suggests that the response to the dental plaque may be different between these groups. Indeed, CD and UC differ regarding their immunopathogenesis, which involves the T helper (Th) cell differentiation; CD is considered to be Th1 disease while UC has characteristics of a Th2 disease (Bouma & Strober 2003). Thus, it may be possible that this difference might also be reflected in the extension of periodontal destruction, but further studies are warranted to confirm this hypothesis. Another possible explanation for the difference between UC and CD regarding CAL is that the CD patients were taking significantly more immunomodulators than UC patients (data not shown). However, it is unknown whether the medications used by these patients have any effect on the peridontium.

Our findings of a higher DMFT in the patient groups are in line with earlier studies (Rooney 1984, Bevenius 1988, Sundh & Emilson 1989, Schütz et al. 2003) but in contrast with the results of Grössner-Schreiber et al. (2006). Grössner-Schreiber et al. (2006) showed no significant difference in the DMF-S index between cases and controls although IBD patients had significantly more plaque. Increased sugar consumption seems to be the main cause of a higher prevalence of caries in CD patients (Sundh & Emilson, 1989, Schütz et al. 2003). We did not take dietary records from the subjects but, as our CD patients had significantly less plaque than controls and there was no difference regarding plaque between UC patients and controls, we speculate that the diet can be the cause of the higher DMFT index. Our study is the first to assess caries in UC patients.

We were unable to find any significant differences in the prevalence of oral lesions between the three groups. Furthermore, other previously described oral manifestations of IBD, such as cobblestone appearance of the mucosa, swelling of the oral and perioral tissues, angular cheilitis and pyostomatitis vegetans (Basu 1976, Plauth et al. 1991, Lisciandrano et al. 1996), were not observed. This may be attributable to the fact that the ongoing medication diminished these oral manifestations.

Overall, CD and UC patients had higher DMFT and prevalence of periodontitis than controls, but smoking was an effect modifier: there was no difference in the prevalence of periodontitis among non-smoking control subjects and non-smoking subjects with CD or UC, but the prevalence of periodontitis was greater among smokers with UC than smokers without UC.

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**Clinical Relevance**

*Scientific rationale for the study:* CD and UC patients may be more susceptible to periodontitis because the mechanisms of tissue destruction are similar. Previously, the oral status of CD patients has been investigated but data are lacking for UC patients.

*Principal findings:* CD and UC patients had a higher prevalence of periodontitis and a higher DMFT index than controls. A higher prevalence of periodontitis was most pronounced in smoking UC patients.

*Practical implications:* CD and UC patients should be enrolled in oral care programme for early diagnosis and preventive care that includes smoking cessation.