

# Cannabis use and destructive periodontal diseases among adolescents

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## Abstract

**Aim:** The aim of this experiment was to investigate the association between cannabis use and destructive periodontal disease among adolescents.

**Material and methods:** Data from a population screening examination carried out among Chilean high school students from the Province of Santiago were used to determine whether there was an association between the use of cannabis and signs of periodontal diseases as defined by (1) the presence of necrotizing ulcerative gingival (NUG) lesions or (2) the presence of clinical attachment loss (CAL)  $\geq 3$  mm. The cannabis exposures variables considered were “Ever use of cannabis” (yes/no) and “Regular use of cannabis” (yes/no). The associations were investigated using multiple logistic regression analyses adjusted for age, gender, paternal income, paternal education, frequency of tooth-brushing and time since last dental visit.

**Results:** Multiple logistic regression analyses showed that “Ever use of cannabis” was significantly negatively associated with the presence of NUG lesions (OR = 0.47 [0.2;0.9]) among non-smokers only. No significant associations were observed between the presence of CAL  $\geq 3$  mm and cannabis use in either of the smoking groups.

**Conclusions:** There was no evidence to suggest that the use of cannabis is positively associated with periodontal diseases in this adolescent population.

**Key words:** adolescence; cannabis; necrotizing ulcerative gingivitis; periodontal attachment loss; periodontal diseases; smoking; socioeconomic factors

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In a prospective cohort study, Thomson et al. (2008) recently reported that cannabis smoking may be a risk factor for periodontal disease that is independent of the use of tobacco. They attributed their findings to the exposure to deleterious constituents in cannabis similar to those of tobacco (Thomson et al. 2008).

## Conflict of interest and source of funding statement

The authors declare that there are no conflicts of interest in this study.

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If this interpretation is correct, a growing body of evidence would suggest that these deleterious constituents are likely to be found in the combustion products resulting from the burning (smoking) of these substances, rather than in their main active constituents. Interestingly, cannabinoids, which are the main active constituents of cannabis, may suppress important biological pathways related to inflammatory processes whereas nicotine, a key tobacco constituent, promotes them (Melamede 2005a,b). A well-known example of this is the opposite effect of nicotine and cannabinoids on angiogenesis (Galve-Roperh et al. 2000, Heeschen et al. 2001).

The purpose of this study was to investigate the association between cannabis use and destructive periodontal

diseases among adolescents. As most patients with necrotizing ulcerative gingival (NUG) lesions still present without apparent systemic determinants (Rowland 1999), we decided to include two outcomes for destructive periodontal disease in these analyses, clinical attachment loss (CAL) and NUG lesions, respectively.

## Material and Methods

The data used originate in a screening study for signs of periodontitis carried out among high school students from the Province of Santiago, Chile (Lopez et al. 2001, Lopez 2003). The target population included all students attending the four high-school grades covering adolescence in the Province of Santiago.

The full list of high schools in the Province of Santiago was stratified according to governmental funding status (yes/no), and each list was subsequently permuted and merged again to obtain a single random permutation of high schools in which funded schools alternated with private schools. The headmasters of the first 133 high schools of this permuted list were contacted to obtain information on the number of students and the number of classes covering the relevant four grades. A total of 104 schools provided the information and were invited to participate in the study. A total of 98 headmasters consented, and therefore 98 high schools were included in the study.

The second sampling stage was designed to comprise all students in all relevant classes in schools where the number of students was  $\leq 100$ , or where the number of classes was  $\leq 3$ . If the number of students in the last four grades exceeded 100 and the number of classes exceeded three, we randomly selected three classes for inclusion.

A total of 9,203 students were present in the selected classes and were invited to participate. All students accepted to fill a questionnaire on oral health related behaviours and conditions (Lopez et al. 2001), but 40 students refused to participate in the clinical examinations, whereby 9,163 students were examined. The examinations were carried out by four examiners who had been extensively trained and calibrated before the commencement of the study. Kappa values for the intra-examiner reliability in the assessment of the presence of  $CAL \geq 3$  mm ranged between 0.32 and 1.0. Corresponding values for inter-examiner reliability were 0.64 and 0.85, respectively (Lopez et al. 2003). Additional information regarding the reliability of the clinical recordings has been provided elsewhere (Lopez et al. 2002, 2003). Students who participated in the clinical examinations also accepted to fill a second questionnaire containing information on socioeconomic factors (Lopez et al. 2006); on the use of drugs (including information on cannabis use) and on oral health related impacts on the quality of life (Lopez & Baelum 2007). Systematic efforts were made during data collection to secure the confidentiality of the information provided by the students in order to safeguard the validity of the self-reports (Swadi 1990). Additional details on sampling strategy and the participation rates can be found elsewhere (Lopez et al. 2001, 2002).

### Clinical examinations

CAL was defined as the distance from the cemento-enamel junction to the base of the clinical pocket, and direct recordings of CAL were obtained at six sites (mesio-buccal, mid-buccal, distobuccal, mesio-lingual/mesio-palatal, mid-lingual/mid-palatal and disto-lingual/disto-palatal) of each of the incisors and all first and second molars. The presence of NUG lesions was recorded if at least one inter-proximal papilla presented with necrotic ulcerated lesions, which had a punched-out appearance and loss of surface tissue. All papillae in the mouth were examined but no attempts were made to count the number of affected papillae or to record the presence of bleeding or pain (Lopez et al. 2002).

Two periodontal disease outcome variables were defined, one being the presence of  $CAL \geq 3$  mm (yes/no), and the other being the presence of NUG (yes/no). Using multiple logistic regression analysis, the association between either of the two outcome variables and cannabis use was explored for each of three tobacco smoking strata, the non-smokers ( $n = 4885$ ), the occasional smokers ( $n = 1997$ ) and the daily smokers ( $n = 2281$ ). Two cannabis exposure variables were considered, one being 'Ever use of cannabis' (yes/no) and the other being 'Regular use of cannabis'. No attempts were made to assess the length of the exposure to cannabis or the amount of cannabis used.

The logistic regression analyses were adjusted for age, gender, paternal income, paternal education, frequency of tooth-brushing and time since last dental visit. These covariates had been found to be statistically significantly associated with the outcome variables in previous analyses (Lopez et al. 2006). Detailed information on the distribution of the covariates in relation to the outcomes considered is found elsewhere (Lopez et al. 2001, 2002, 2006). The option 'robust cluster' for the procedure 'logit' in Stata (College Station, TX, USA) version 10.0 was used to take account of the clustered sampling strat-

egy (Lopez et al. 2001, 2002, 2006, Lopez 2003).

### Results

Table 1 shows the distribution of the study population according to each of the two outcome variables and tobacco smoking status. The prevalence of the outcome presence of  $CAL \geq 3$  mm ranged between 3.9% among occasional smokers and 5.0% among daily tobacco smokers. The prevalence of NUG was slightly higher, ranging between 6.1 and 7.2%, depending on tobacco smoking status (Table 1).

Table 2 shows the prevalence of the exposure variables and the important covariates. The prevalence of 'Ever use of cannabis' ranged from 5.5% among non-smokers to 45.5% among daily tobacco smokers. 'Regular use of cannabis' was reported by 1.3% of the non-smokers and by 16.3% of the daily tobacco smokers (Table 2).

The multiple logistic regression analyses showed no association between 'Ever use of cannabis' and  $CAL \geq 3$  mm, whether non-smokers (OR = 0.95), occasional smokers (OR = 1.15) or daily tobacco smokers (OR = 0.98) were concerned (Table 3). Similarly, there was no evidence for an association between 'Regular cannabis use' and  $CAL \geq 3$  mm, irrespective of the tobacco smoking category considered. The OR estimates observed in the models describing the effect of regular use of cannabis were similar to the estimates presented in Table 3.

All but one OR estimate pointed towards a negative association between cannabis use and the presence of NUG when analyses were adjusted for the effects of the covariates age, gender, paternal income, paternal education, frequency of tooth-brushing and time since last dental visit. However, only one of these negative associations was statistically significant, namely the association between 'Ever use of cannabis' and presence of NUG among non-smokers (OR = 0.47) (Table 4).

*Table 1.* Prevalence of the two outcomes considered, presence of  $CAL \geq 3$  mm and presence of NUG, for each of the three tobacco smoking status groups considered

	Non-smokers (%) ( $n = 4,885$ )	Occasional tobacco smokers (%) ( $n = 1,997$ )	Daily tobacco smokers (%) ( $n = 2,281$ )	All subjects (%) (9,163)
$CAL \geq 3$ mm	4.5	3.9	5.0	4.5
NUG	6.8	7.2	6.1	6.7

CAL, clinical attachment loss; NUG, necrotizing ulcerative gingiva.

Table 2. Prevalence distribution of the key exposure variables and important determinants according to tobacco smoking status group

Determinant	Non-smokers (%) (n = 4,885)	Occasional tobacco smokers (%) (n = 1,997)	Daily tobacco smokers (%) (n = 2,281)	All subjects (%) (9,163)
Ever use of cannabis	5.5	21.4	45.5	18.9
Regular use of cannabis	1.3	5.8	16.3	6.0
Age (years)				
12–14	30.1	20.4	8.1	22.5
15–17	64.2	71.2	79.2	69.5
18–21	5.7	8.5	12.7	8.0
Gender				
Boy	55.8	43.7	46.2	50.8
Girl	44.2	56.3	53.8	49.2
Income of father (\$)				
≥500,000 (Chilean pesos)	25.1	21.8	26.4	24.7
300–499,000	14.7	14.5	13.6	14.4
100–299,000	34.2	34.8	31.7	33.7
<100,000	11.7	14.3	12.2	12.4
No income	6.0	6.8	6.5	6.3
Not answered	8.4	7.9	9.5	8.6
Education of father				
Technical/university completed	31.9	27.1	30.3	30.5
High school completed	31.0	32.9	34.5	32.3
Up to primary school completed	32.3	35.1	30.5	32.5
Not answered	4.8	5.0	4.8	4.8
Frequency of tooth brushing				
Less than once a day	3.8	3.7	3.3	3.7
Once a day	26.1	25.7	24.5	25.6
More than once a day	70.1	70.6	72.3	70.7
Last visit to the dentist				
<6 months ago	33.8	34.4	31.4	33.3
6–12 months ago	19.8	19.3	19.4	19.6
>12 months ago	38.9	39.0	42.2	39.8
Never	7.4	7.4	7.1	7.3
Not answered	0.02	0	0	0.01

## Discussion

Our findings do not corroborate the recent results presented by Thomson et al. (2008), who, based on analyses of both prevalence and incidence data, suggested that cannabis use may be an independent risk factor for periodontitis. In the present study, only one statistically significant association was observed between the use of cannabis and the periodontal outcomes considered, and this association was not consistent with a deleterious effect of cannabis use.

Some might surmise that the negative association observed between signs of NUG and cannabis use is no more than a spurious result occurring in a cross-sectional study using a NUG case definition, which yields relatively high prevalence estimates (Lopez et al. 2002). However, we are not ready to dismiss our observation on those grounds, as case definitions based on the fulfilment of additional criteria are

likely to be biased in the direction of estimates that are too low. In the present study, the diagnosis of NUG was based solely on visual inspection of the gingival tissues for the pathognomonic features of NUG, and no attempts were made to assess the presence of gingival bleeding or pain, as is done in several other studies. This may explain the relatively higher prevalence estimate found in the present study compared with other populations (Lopez et al. 2002). However, while it is clear that pain is a critical symptom among patients seeking treatment, and hence come to the attention of dental professionals, it remains unclear whether pain is a key sign among cases identified in population-based studies. Hence, some authors report that subjective complaints are unusual and that the previously described classical symptoms of Vincent's infection, including pain, are present in a surprisingly small number of cases (Grupe & Wilder 1956). Similarly,

Barnes et al. (1973) have reported that 14% of cases of acute NUG (ANUG) had no pain and another 40% suffered only mild pain. Conversely, gingival bleeding is not unique for NUG and can be found in many periodontal conditions (for review, see Lopez et al. (2002)). Clearly, had we used a more complex diagnostic classification, with several inclusion criteria, a lower prevalence estimate would have emerged, but we see no reason to dismiss our observation solely on those grounds.

It clearly remains a possibility that the negative association between cannabis use and NUG is mediated by unknown confounders or by different combustion products; it is also possible that the association is biologically mediated by exposure to the cannabinoids of the cannabis plant. Hence, an emerging body of scientific evidence suggests that cannabinoids have potent immunomodulatory and anti-inflammatory effects that are relevant for several inflammatory diseases (for review, see Croxford & Yamamura (2005), Centonze et al. (2007), Iversen (2008)). In particular, the findings demonstrating immunomodulating effects of cannabinoids on several human immune cells (Croxford & Yamamura 2005) and the down-regulation of immunologically generated free radicals by the promotion of an anti-inflammatory Th2 immune cytokine profile (Yuan et al. 2002) are interesting for the explanation of a possible biological effect of systemic cannabinoids resulting from marijuana smoking. Interestingly, while low doses of cannabinoids have been reported to stimulate the Th2 response, high doses may inhibit it and change the balance in favour of a Th1 response (Berdyshev et al. 1997). This calls for caution in the interpretation of studies on this association and further evaluation of the dose-response relationship is needed.

When comparing the result of the present study with those of Thomson et al. (2008), it must be borne in mind that the two studies differ in terms of their design. In the present study, the temporal relationship between exposure and disease may be considered less certain, as information on current and previous cannabis exposure and disease status was obtained at the same time, whereas in the New Zealand study the information was obtained prospectively. It follows that the present study population and the New Zealand population studied by Thomson and co-workers

**Table 3.** Multiple logistic regression analyses of the association between having ever used cannabis, and the presence of CAL  $\geq 3$  mm, for each tobacco smoking status groups

Determinant	Non-smokers ( <i>n</i> = 4,885) (OR [95%CI])	Occasional tobacco smokers ( <i>n</i> = 1,997) (OR [95%CI])	Daily tobacco smokers ( <i>n</i> = 2,264) (OR [95%CI])
Ever use of cannabis (ref = No)	0.95 [0.5;1.8]	1.15 [0.7;2.0]	0.98 [0.7;1.4]
Regular use of cannabis (ref = No)	1.03 [0.3;3.4]	1.27 [0.5;3.3]	0.77 [0.4;1.4]
Age (years) (ref = 12–14)	–	–	–
15–17	1.80 [1.1;2.9]	2.50 [1.2;5.4]	0.96 [0.4;2.3]
18–21	5.30 [2.7;10.3]	1.30 [0.4;4.5]	1.67 [0.6;4.5]
Gender (ref = Boy)	–	–	–
Girl	1.04 [0.7;1.4]	1.73 [1.0;3.0]	1.23 [0.8;1.9]
Income of father (\$) (Chilean pesos)	–	–	–
$\geq 500,000$	–	–	–
300–499,000	0.84 [0.4;1.6]	1.18 [0.4;3.1]	2.09 [1.0;4.6]
100–299,000	1.95 [1.1;3.4]	0.90 [0.4;2.0]	3.16 [1.6;6.3]
< 100,000	2.09 [1.1;3.8]	1.49 [0.7;3.4]	3.06 [1.4;6.8]
No income	2.31 [1.2;4.6]	0.29 [0.1;1.3]	1.42 [0.5;4.3]
Education of father	–	–	–
Technical/university completed	–	–	–
High school completed	1.01 [0.6;1.6]	1.00 [0.5;2.0]	0.66 [0.4;1.2]
Up to primary school completed	1.75 [1.1;2.7]	1.78 [0.9;3.5]	1.02 [0.6;1.8]
Frequency of tooth-brushing	–	–	–
Less than once a day	–	–	–
Once a day	0.63 [0.3;1.2]	0.54 [0.2;1.7]	1.82 [0.4;7.7]
More than once a day	0.53 [0.3;1.0]	0.32 [0.1;0.9]	1.54 [0.4;6.3]
Last visit to the dentist	–	–	–
< 6 months ago	–	–	–
6–12 months ago	0.69 [1.1;2.6]	1.18 [0.6;2.5]	0.56 [0.3;1.0]
> 12 months ago	1.57 [1.1;2.3]	1.68 [0.9;3.1]	0.75 [0.5;1.2]
Never	2.32 [1.4;3.9]	1.84 [0.8;4.2]	0.64 [0.3;1.5]

**Table 4.** Multiple logistic regression analyses of the association between having ever used cannabis, or being a regular user of cannabis, and the presence of NUG, for each tobacco smoking status group

Determinant	Non-smokers ( <i>n</i> = 4,885) (OR [95%CI])	Occasional tobacco smokers ( <i>n</i> = 1,997) (OR [95%CI])	Daily tobacco smokers ( <i>n</i> = 2,264) (OR [95%CI])
Ever use of cannabis (ref = No)	0.47 [0.2;0.9]	1.14 [0.8;1.7]	0.76 [0.5;1.1]
Regular use of cannabis (ref = No)	0.45 [0.1;1.8]	0.78 [0.3;1.8]	0.85 [0.5;1.4]

Models are adjusted for the effects of age, gender, paternal income, paternal education, frequency of tooth brushing, and time since last dental visit.

NUG, necrotizing ulcerative gingiva.

also differed in terms of the age groups studied. Most individuals of the present study population were substantially younger, covering the age range between 12 and 21 years, with most subjects being 15–18 years old. This difference has important implications, as the duration of exposure is likely to have been much longer in the New Zealand population; just as the cannabis-exposed New Zealanders may represent a different health behavioural and socio-economic profile (Lubman et al. 2007). Cannabis smoking is typically taken up at a rather young age, but most cannabis users quit their habit relatively early in their adult lives (Sidney 2003). People who remain cannabis smokers at the age of 32 years can therefore be classified as “long-term” users, and as

such they are at risk of the “amotivational syndrome” (Schwartz 1987), characterized, among others, by a lack of concern for personal hygiene and appearance. While one may attempt to control the confounding that may result from the different age profiles of the two studies by introducing covariates such as socioeconomic classification, tobacco use and plaque score in the regression models, it must also be realized that doing so does not constitute a guaranteed protection against residual confounding. Hence, while Thomson and co-workers used the continuous variable “Pack-years of tobacco use” to adjust for the possible confounding effect of tobacco smoking, we chose to perform three analyses; one for “never smokers” and one for each of the

categories “occasional” and “daily” tobacco smokers. Even so, some residual effect of tobacco smoking cannot be excluded in the present study due to the fact that cannabis is usually mixed with tobacco in hand-rolled cigarettes (Cho et al. 2005). Although Thomson et al. (2008) noted that the usual mode of cannabis use among New Zealanders does not involve tobacco, they were open to the suggestion that cannabis use and smoking may have gone hand in hand for some of their users.

The use of self-reported information on cannabis use could be seen as a limitation of the study, but these self-reports have been found to be valid in previous studies on drug use among adolescents (Winters et al. 1990), and are commonly used in epidemiological studies involving the assessment of cannabis exposure (Swadi 1990). Moreover, in the present study, strong efforts were made to ensure the confidentiality of the answers and the privacy of the participating students in the study settings in which the questions were answered. While it cannot be excluded that valuable exposure information might have been gained had we attempted to assess the duration or the cumulative extent of the exposure to cannabis, we refrained from this owing to the young age of our study group.

In conclusion, our study did not confirm the recently reported significant positive association between cannabis smoking and signs of periodontitis. The results might suggest that periodontal effects of a short-term exposure to cannabis could differ from the effects of long-term exposure to the drug, or that it is difficult to control confounding when the exposed groups are short-term *versus* long-term users. However, further studies are needed to address these issues.

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**Clinical Relevance**

*Scientific rationale for the study:* A positive association between the use of cannabis and periodontitis has been recently reported. However, the reported findings require confirmation in other studies.

*Principal findings:* The findings of this study conducted amongst Chilean high school students do not confirm the previously reported positive association between signs of periodontitis and cannabis use in adults.

*Practical implications:* The results of the study suggest that the immunomodulatory effects of exposure to cannabis and their effect on periodontal tissues might warrant further exploration.

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