DENTAL MEDICINE

Chronic apical periodontitis beyond infection: the impact of diet and lifestyle factors

Antonia Boca[1,2], Bianca Adina Boșca[3], Sanda Cîmpean[1], Cosmina Ioana Bondor[4], Anida-Maria Băbțan[2], Anca Ionel[2], Claudia Nicoleta Feurdean[2], Willi Andrei Uriciuc[2], SALIVAGES Project Group, Anne-Marie Constantin[3], Georgeta Victoria Inceu[5], Gabriela Roman[6], Mihaela Băciuț[7], Aranka Ilea[2]

1) Department of Conservative Dentistry, Faculty of Dentistry, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

2) Department of Oral Rehabilitation, Faculty of Dentistry, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

3) Department of Histology, Faculty of Dentistry, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

4) Department of Medical Informatics and Biostatistics, Faculty of Medicine, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

5) Department of Diabetes and Nutrition Diseases, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

6) Department of Diabetes, Nutrition and Metabolic Diseases, Emergency Clinical County Hospital, Cluj-Napoca, Romania

7) Department of Maxillofacial Surgery and Implantology, Faculty of Dentistry, Iuliu Hațieganu University of Medicine and Pharmacy, Cluj-Napoca, Romania

Abstract

Aim. This study aimed to investigate whether chronic apical periodontitis (CAP) is influenced not only by microbial infection but also by systemic biomarkers, lifestyle, and dietary patterns.

Methods. An observational cross-sectional design was applied on a cohort of adult patients, who underwent radiographic diagnosis, blood and saliva collection for oxidative stress and advanced glycation end products (AGEs) analysis, and also completed structured dietary and lifestyle questionnaires.

Results. Biochemical analyses revealed no significant differences in oxidative stress or advanced glycation end products between CAP and non-CAP groups, suggesting that systemic markers may not adequately reflect localized periapical inflammation. Similarly, smoking habits and physical activity did not show relevant associations with disease presence.

By contrast, diet emerged as a significant determinant. Higher consumption of fried potatoes, refined products such as croissants and white rice, and acidic fruits like oranges was associated with increased risk of CAP. Conversely, regular consumption of breakfast cereals demonstrated a protective effect, reducing disease likelihood. These associations were confirmed in multivariate analysis, highlighting the independent role of specific dietary patterns.

Conclusion. CAP appears to be shaped not only by infection but also by everyday nutrition, with fried and acidic foods favoring disease, while wholegrain cereals provide resilience. These findings emphasize the importance of integrating nutritional guidance into dental care to complement conventional endodontic therapy.

Keywords: periodontitis, lifestyle, diet, advanced glycation end products

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Address for correspondence:
Sanda Cîmpean
sandacimpean@gmail.com

Introduction

Chronic apical periodontits (CAP) is one of the most common inflammatory conditions of the oral cavity, typically arising as a consequence of microbial infection of the root canal system. Despite advances in endodontic therapy, CAP remains highly prevalent worldwide, with epidemiological studies reporting rates ranging from 30% to 85% depending on the studied population [1]. Its persistence is explained not only by the complex microbiota associated with infected root canals but also by the host immune response and the factors that influence periapical healing.

In recent years, research has expanded beyond the traditional view of CAP as a purely infection-driven disease. Increasing attention has been given to systemic and lifestyle-related factors that may influence disease risk, progression and healing outcomes. Among these, oxidative stress and advanced glycation end products (AGEs) have been recognized as important contributors to chronic inflammation and tissue damage in a variety of systemic conditions, including diabetes, cardiovascular disease and periodontitis [2,3]. However, their role in CAP is less well understood, with studies offering mixed results regarding their relevance as systemic biomarkers for periapical pathology [4].

Dietary habits represent another important but often overlooked factor. Diets high in refined carbohydrates, saturated fats, or acidic foods can alter the oral microbiome, promote systemic inflmmation and impair homeostastis [5,6]. Conversely, diets rich in fiber, whole grain, and micronutrients support microbial balance and a healthier inflammatory profile [7–10]. Understanding how specific dietary patterns relate to CAP could provide new insights into preventive strategies.

Against this background, the present study was designed to investigate the potential associations between CAP, systemic markers of oxidative stress and AGEs, lifestyle habits, and dietary patterns. By integrating clinical, biochemical, and behavioral data, we aimed to explore whether CAP is influenced not only by microbial infection but also by health-related and lifestyle-driven determinants.

Methods

In the study conducted by Băbțan et al. [11] an analytical, observational, and cross-sectional design was applied, involving the recruitment of 200 patients from the Department of Oral Rehabilitation and Prosthodontics, Faculty of Dentistry, as well as from the Regional Diabetes Center in Cluj-Napoca, Romania, between 2018 and 2019. All participants provided written informed consent, and the study received ethical approval from the Ethics Committee for the human SALIVAGES cohort (Approval no. 93/8.03.2017). The inclusion criteria were the following: patients over eighteen years of age, patients with recommendation of oral examination associated or not with treatment, patients that were clinically healthy or with general illnesses with or without medication. The exclusion criterion was the age under eighteen years [12].

The diagnosis of chronic apical periodontitis (CAP) was established using orthopantomographic images, independently assessed by two evaluators (A.B. and S.C.) who had been previously calibrated to ensure diagnostic consistency. A total of 89 patients were diagnosed with CAP. In cases of disagreement regarding the number of affected teeth, the radiographs were jointly reviewed. The diagnostic criteria for CAP included the presence of periapical radiolucency, discontinuity of the lamina dura, and widening of the periodontal ligament space. Patients showing no pathological changes (intact lamina dura, absence of alveolar bone demineralization, and no pathological relation to anatomical structures such as the maxillary sinus, mandibular canal, or nasal fossae), as well as edentulous patients, were excluded.

Venous blood samples were collected from the upper arm and centrifuged at 4000 rpm for 15 minutes, with plasma aliquoted into sterile cryotubes (100, 200, and 500 μl). The following parameters were assessed: complete blood count, erythrocyte sedimentation rate (ESR), fasting blood glucose, high-sensitivity C-reactive protein (hsCRP), and lipid profile.

Dietary habits were assessed using a structured questionnaire including questions on meal frequency, snacking, late-night eating, beverage preferences, added sugar intake, and eating behaviors. The dietary questionnaire had been previously validated in an earlier study by Roman et al. [13].

Stimulated saliva samples were obtained 30 minutes after tooth brushing, by instructing participants to chew sterile cotton swabs soaked in citric acid for 2 minutes (Salivette® Sarstedt AG & Co., Germany). The samples were centrifuged at 1450 rpm for 7 minutes and aliquoted into sterile cryotubes (100, 200, and 500 μl). All biological fluids (saliva and plasma) were stored at –20 °C until analysis.

Assessment of nitro-oxidative stress

Nitro-oxidative stress markers were evaluated in both plasma and saliva. Saliva samples were collected using Salivette® Sarstedt devices after tooth brushing. The following markers were measured: nitric oxide (μmol nitrites/L), total oxidative status (μmol H_2O_2 equiv./L), total antioxidant capacity (mmol Trolox equiv./L), oxidative stress index (arbitrary units), malondialdehyde (nmol/ml), and total thiols (mmol/ml).

Assessment of nitric oxide levels (NO)

The Griess reaction was employed to indirectly determine NO synthesis by quantifying nitrites (NO_2^-) and nitrates (NO_3^-). Plasma NOx concentrations were calculated using a sodium nitrite-based calibration curve and expressed as nitrite μmol/L.

Assessment of total oxidative status (TOS)

Total oxidative status (TOS) in plasma was determined using a colorimetric assay, with results expressed in μmol H_2O_2 equiv./L.

Assessment of total antioxidant capacity (TAC)

The total antioxidant response (TAR) was measured in plasma through a colorimetric assay, with results expressed as mmol Trolox equiv./L. The oxidative stress index (OSI) was calculated as the ratio of TOS to TAC, serving as an indicator of oxidative stress intensity:

OSI (arbitrary units) = TOS (μmol H_2O_2 equiv./L) ÷ TAC (mmol Trolox equiv./L).

Assessment of malondialdehyde (MDA)

Malondialdehyde (MDA) was used as a marker of lipid peroxidation, determined by the thiobarbituric acid method. Absorbance of the supernatant was measured at 532 nm. A standard calibration curve was constructed using 1,1,3,3-tetraethoxypropane (0.3–10 nmol/ml). Plasma MDA concentrations were expressed in nmol/ml.

Assessment of total thiols (tSH)

Total thiols (tSH) were quantified using Ellman's reagent. Plasma concentrations were expressed in mmol/ml.

Assessment of advanced glycation end-products (AGEs) in biofluids

Key glycation agents, including CML, FruLys, Pyr, MG-H1, CEL, Lys, and Arg, were identified and characterized, emphasizing their roles in protein modification and potential implications in metabolic disorders.

The concentration of AGEs as free glycated amino acids in saliva was determined according to the method described by Manig et al. Briefly, 500 µl of saliva was mixed with 10 µl of an internal standard solution and 490 µl of ice-cold acetonitrile/methanol (70/30, v/v). After 10 minutes of incubation at 4 °C, the samples were centrifuged (10,000 g, 10 minutes). The supernatant was evaporated to dryness under a nitrogen stream, and the residue was reconstituted in 90 µl of 20 mM NFPA. Double-distilled water was used to prepare blanks.

For plasma analysis, 10 µl of the internal standard solution, 25 µl of plasma, and 400 µl of ice-cold acetonitrile/methanol (70/30, v/v) were mixed for deproteinization. Following overnight incubation at –18 °C, samples were centrifuged, and a defined volume of the supernatant was evaporated to dryness under nitrogen. The residue was reconstituted in 20 mM NFPA prepared with double-distilled water. Samples were then analyzed by liquid chromatography coupled with mass spectrometry (LC-MS/MS) [12,14,15].

Data analysis

All the data from the study were analyzed using IBM SPSS Statistics 25 and illustrated using Microsoft Office Excel/Word 2024. Qualitative variables were written as counts or percentages and were tested between groups using Fisher's Exact Test. Z-tests with Bonferroni correction were used to further detail the results obtained in the contingency tables.

Quantitative variables were written as means with standard deviations or medians with interquartile ranges. Normality of the quantitative variables was assessed using the Shapiro-Wilk Test. Quantitative variables with non-parametric distribution were tested between groups using the Mann-Whitney U Test. Quantitative variables with normal distribution were tested between groups using the Student T-Test (after checking for equality of variances using Levene's Test).

Univariate and multivariate logistic regression models were used to predict the odds of having chronic apical periodontitis. Models were tested for significance and goodness-of-fit. Performance of the prediction was quantified using odds ratios with 95% confidence intervals. A multivariateble model was obtained using the standard enter approach in which significant parameters observed in the univariate models were introduced in the model. The threshold considered for the significance level for all tests was considered to be α = 0.05.

Results

The study population consisted of 89 patients recruited between 2018 and 2019, all meeting the eligibility criteria and undergoing comprehensive clinical, radiographic, and biochemical assessments to explore the factors associated with the development and progression of chronic apical periodontitis, which was present in 83.1% of the patients, while 16.9% showed no signs of the disease.

Biochemical parameters: AGEs and oxidative stress

The biochemical analysis included both plasma and salivary samples. Advanced glycation end products (AGEs) were measured to assess systemic and local metabolic alterations.

Table I. Description of the plasma and salivary AGEs parameters analyzed in the study.

Plasma AGEs (N=79)				
Parameter	**Mean ± SD**	**Median (IQR)**	**Min**	**Max**
FruLys	373.62 ± 245.66	332.4 (161.6-465.9)	83.8	1299.8
Pyr	25.53 ± 8.35	27.19 (24.81-28.99)	5.2	43.83
MG-H1	34.42 ± 5.81	33.11 (31.51-36.79)	9.2	52.59
CEL	12.66 ± 2.22	13.27 (12.95-13.53)	6.2	14.85
CML	50 ± 5.09	49.87 (46.71-52.81)	37.02	65.83
Arg	5.32 ± 4.33	3.84 (2.46-7.4)	0.46	20.38
Lys	6.74 ± 2.76	6.15 (5.26-7.28)	1.08	16.07
Salivary AGEs (N=75)				
FruLys	23.96 ± 4.56	22.41 (20.91-26.15)	20.35	49.37
Pyr	1.77 ± 0.24	1.7 (1.69-1.74)	1.4	2.73
MG-H1	3.69 ± 1.53	3.05 (2.48-4.38)	2.21	8.52
CEL	1.34 ± 0.42	1.24 (1.05-1.42)	0.95	3.06
CML	4.28 ± 1.62	3.95 (2.97-5.03)	2.52	12.8
Arg	0.6 ± 0.51	0.53 (0.16-0.87)	0.033	2.391
Lys	0.78 ± 0.79	0.54 (0.18-1.12)	0.033	3.975

In addition, oxidative stress markers were quantified, including total antioxidant capacity (TAC), total oxidative status (TOS), oxidative stress index (OSI), nitric oxide (NO), malondialdehyde (MDA), and total thiols, providing an overview of systemic and salivary redox balance.

Data from table I show the description of the plasma and salivary AGEs parameters analyzed in the study. For plasmatic AGEs the values are the following:

Table II. Description of the plasma and salivary oxidative stress parameters analyzed in the study.

Plasma oxidative stress (N=78)				
Parameter	**Mean ± SD**	**Median (IQR)**	**Min**	**Max**
TAC	23.62 ± 27.85	1.11 (1.09-53.57)	1.088	77.21
TOS	47.22 ± 18.08	44.57 (33.6-58.83)	10.78	87.58
OSI	26.5 ± 24.25	28.99 (1.09-47.33)	1.088	72.81
NO	44.27 ± 17.26	39.89 (32.54-55.27)	9.86	92.08
MDA	175.1 ± 209.78	7.54 (4.65-390.5)	2.28	573
Total tiols	255.14 ± 244.58	304 (3.82-439)	2.05	896
Salivary oxidative stress (N=61)				
TAC	8.37 ± 10.53	1.1 (1.09-16.97)	1.084	37.34
TOS	8.22 ± 3.39	7.53 (6.06-9.46)	3.17	19.78
OSI	5 ± 3.75	5.69 (1.09-7.27)	1.091	18.08
NO	12.06 ± 7.76	10.27 (6.92-19.03)	0	32.21
MDA	24.12 ± 31.53	3.3 (2.08-43)	0.73	113
Total tiols	71.91 ± 61.53	79 (2.68-113.31)	1.11	205

Lifestyle-related factors

Only 86 participants completed the smoking-related section of the questionnaire, while the remaining participants chose not to provide an answer to this item. Smoking habits were first assessed, given their well-established role in oral health. More than 40% of participants reported active smoking.

Data from table II show the description of the plasma and salivary oxidative stress parameters analyzed in the study.

Table III. Distribution of the patients according to the existence of smoking.

Smoking (N=86)	No.	Percentage
Absent	40	46.5%
Present	46	53.5%

Data from table III show the distribution of the patients according to the existence of smoking. 53.5% of the patients were active smokers.

General dietary habits

The survey collected detailed information on dietary behavior, including meal frequency, snack consumption, food categories, and eating-related habits. These results illustrate the overall lifestyle context of the studied population.

Table IV. Distribution of the patients according to survey items.

Question (Affirmative answer - Nr., %) / (Mean ± SD)	
Do you exert intense physical effort lasting more than 10 minutes in your profession? (N=84)	26 (31%)
How many days a week do you do intense physical activity in your profession? (N=79)	1.87 ± 2.6
Do you perform moderate physical activity lasting more than 10 minutes in your profession? (N=79)	47 (59.5%)
How many days a week do you do moderate physical activity in your profession? (N=77)	3.26 ± 2.94
Do you do high-intensity physical activities continuously more than 10 min in free time? (N=79)	15 (19%)
How many days do you practice intense physical activities mentioned above? (N=77)	0.93 ± 2.13
Do you do moderate intensity physical activities for at least 10 minutes in free time? (N=82)	30 (36.6%)
How many days do you practice the moderate physical activities mentioned above? (N=77)	2.05 ± 2.63
How much time do you spend on a typical day sitting or lying down? (hours) (N=81)	2.75 ± 2.53
Do you currently smoke? (N=86)	46 (53.5%)
Have you quit smoking in the last 2 years? (N=45)	6 (13.3%)
Do you consume 3 meals / day? (N=87)	
Daily	35 (40.2%)
Most times	14 (16.1%)
Rarely	38 (43.7%)
Do you eat snacks? (N=86)	
Daily	27 (31.4%)
Most times	19 (22.1%)
Rarely	40 (46.5%)
What is the most consistent meal? (N=84)	
Breakfast	22 (26.2%)
Lunch	47 (56%)
Dinner	15 (17.9%)
Do you eat breakfast every day? (N=87)	49 (56.3%)
Do you eat consistently after 9 pm? (N=85)	
Daily	38 (44.7%)
Most times	9 (10.6%)
Rarely	38 (44.7%)

Table IV. Distribution of the patients according to survey items (continuation).

Question (Affirmative answer - Nr., %) / (Mean ± SD)									
Do you wake up at night to eat? (N=87)							13 (14.9%)		
If YES, how many times a week? (N=13)							2.69 ± 2.28		
Do you happen to eat large amounts of food in < 2 hours beyond the feeling of fullness? (N=86)							6 (7%)		
If YES, do you feel guilty for eating such large amounts of food? (N=6)							4 (66.7%)		
Do you eat while watching TV, computer or reading? (N=84)							29 (34.5%)		
Consumption frequency (Nr. / %)									
Eating habit	**<1/mo**	**1-3/mo**	**1/wk**	**2-4/wk**	**5-6/wk**	**1/day**	**2-3/day**	**4-5/day**	**≥6/day**
Carbonated drink& sweetener (N=86)	**49/57**	8/9.3	7/8.1	7/8.1	2/2.3	5/5.8	5/5.8	1/1.2	2/2.3
Carbonated drink with sugar (N=87)	**41/47.1**	9/10.3	8/9.2	12/13.8	3/3.4	5/5.7	5/5.7	1/1.1	3/3.4
Non-carbonated drink &sugar (N=86)	**43/50**	12/14	9/10.5	11/12.8	2/2.3	1/1.2	6/7	1/1.2	1/1.2
Regular beer (N=87)	**58/66.7**	10/11.5	10/11.5	6/6.9	-	3/3.4	-	-	-
Light beer (N=87)	**76/87.4**	4/4.6	3/3.4	4/4.6	-	-	-	-	-
Red wine (N=87)	**73/83.9**	7/8	3/3.4	4/4.6	-	-	-	-	-
White wine (N=87)	**78/89.7**	5/5.7	2/2.3	-	1/1.1	-	-	1/1.1	-
Liquer, spirits (N=87)	**69/79.3**	8/9.2	1/1.1	2/2.3	-	-	2/2.3	5/5.7	-
Water (N=87)	12/13.8	-	3/3.4	3/3.4	2/2.3	5/5.7	14/16.1	**28/32.2**	20/23
Cereal for breakfast (N=87)	**55/63.2**	10/11.5	4/4.6	7/8	-	6/6.9	4/4.6	1/1.1	-
White bread/white flour prod (N=86).	18/20.9	2/2.3	1/1.2	7/8.1	5/5.8	14/16.3	**34/39.5**	3/3.5	2/2.3
Wholemeal bread (N=87)	**49/56.3**	5/5.7	7/8	3/3.4	2/2.3	8/9.2	11/12.6	2/2.3	-
Croissants, muffins or biscuits (N=87)	**40/46**	17/19.5	7/8	10/11.5	3/3.4	5/5.7	4/4.6	1/1.1	-
Pancakes/waffles (N=86)	**38/44.2**	29/33.7	12/14	7/8.1	-	-	-	-	-
Black, brown rice (N=85)	**65/76.5**	8/9.4	8/9.4	4/4.7	-	-	-	-	-
White rice (N=87)	25/28.7	**30/34.5**	22/25.3	9/10.3	1/1.1	-	-	-	-
Pasta (N=87)	16/18.4	24/27.6	**29/33.3**	14/16.1	2/2.3	2/2.3	-	-	-
Fried potatoes (N=87)	21/24.1	**22/25.3**	21/24.1	17/19.5	3/3.4	2/2.3	1/1.1	-	-
Baked, boiled potatoes (N=87)	12/13.8	15/17.2	26/29.9	**29/33.3**	3/3.4	1/1.1	1/1.1	-	-
Pizza (N=87)	**43/49.4**	20/23	9/10.3	11/12.6	4/4.6	-	-	-	-
Whole eggs (N=87)	25/28.7	9/10.3	19/21.8	**28/32.2**	4/4.6	2/2.3	-	-	-
Pork or beef hot dog (N=87)	**65/74.7**	13/14.9	2/2.3	3/3.4	2/2.3	2/2.3	-	-	-
Hot dogs or chciken sausages (N=86)	**64/74.4**	7/8.1	8/9.3	5/5.8	1/1.2	1/1.2	-	-	-
Chicken/turkey meat (skin) (N=86)	**33/38.4**	9/10.5	14/16.3	26/30.2	1/1.2	-	-	1/1.2	2/2.3
Chicken/turkey meat (no skin) (N=87)	21/24.1	12/13.8	14/16.1	**33/37.9**	-	7/8	-	-	-
Bacon, kaiser (N=87)	**30/34.5**	17/19.5	14/16.1	19/21.8	3/3.4	2/2.3	2/2.3	-	-
Salami or other meat products (N=87(	**30/34.5**	15/17.2	14/16.1	20/23	2/2.3	6/6.9	-	-	-
Sausages / other minced meat (N=87)	**33/37.9**	14/16.1	24/27.6	13/14.9	1/1.1	1/1.1	-	-	1/1.1
Hamburger (N=86)	**66/76.7**	7/8.1	5/5.8	7/8.1	1/1.2	-	-	-	-
Pork (N=87)	23/26.4	15/17.2	15/17.2	**29/33.3**	3/3.4	2/2.3	-	-	-
Beef, lamb (N=87)	**45/51.7**	21/24.1	11/12.6	9/10.3	1/1.1	-	-	-	-
Canned tuna (N=87)	**54/62.1**	12/13.8	16/18.4	4/4.6	1/1.1	-	-	-	-
Semi-prepared breaded fish (N=86)	**60/69.8**	12/14	7/8.1	6/7	1/1.2	-	-	-	-
Prawns, clams, lobster (N=86)	**76/88.4**	2/2.3	5/5.8	3/3.5	-	-	-	-	-
Tuna, salmon, sardines (N=87)	**58/66.7**	14/16.1	11/12.6	3/3.4	1/1.1	-	-	-	-
Cod, haddock, halibut (N=86)	**72/83.7**	7/8.1	6/7	1/1.2	-	-	-	-	-
Skimmed milk (N=82)	**58/70.7**	4/4.9	5/6.1	4/4.9	3/3.7	7/8.5	1/1.2	-	-
Milk 1-2% fat (N=85)	**43/50.6**	7/8.2	7/8.2	14/16.5	3/3.5	10/11.8	1/1.2	-	-
Whole milk (N=84)	**61/72.6**	3/3.6	5/6	3/3.6	2/2.4	8/9.4	2/2.4	-	-
Soy milk (N=82)	**77/93.9**	2/2.4	1/1.2	-	-	2/2.4	-	-	-
Cream (N=87)	**69/79.3**	8/9.2	6/6.9	2/2.3	2/2.3	-	-	-	-
Full-fat yogurt (N=87)	**34/39.1**	10/11.5	13/14.9	18/20.7	2/2.3	9/10.3	1/1.1	-	-
Fruit yogurt (N=86)	**53/61.6**	8/9.3	13/15.1	11/12.8	1/1.2	-	-	-	-
Cottage cheese (N=87)	**36/41.4**	20/23	14/16.1	15/17.2	1/1.1	1/1.1	-	-	-
Cream cheese (N=86)	**37/43**	14/16.3	15/17.4	18/20.9	1/1.2	1/1.2	-	-	-
Cheese, cheddar (N=86)	**37/43**	12/14	14/16.3	20/23.3	2/2.3	1/1.2	-	-	-
Milk chocolate (N=87)	**49/56.3**	11/12.6	10/11.5	11/12.6	1/1.1	3/3.4	1/1.1	-	1/1.1
Dark chocolate (N=86)	**50/58.1**	14/16.3	9/10.5	8/9.3	-	5/5.8	-	-	-

Table IV. Distribution of the patients according to survey items (continuation).

Consumption frequency (Nr. / %)									
Eating habit	<1/mo	1-3/mo	1/wk	2-4/wk	5-6/wk	1/day	2-3/day	4-5/day	≥6/day
Chocolate bars (N=86)	**53/61.6**	12/14	7/8.1	9/10.5	-	5/5.8	-	-	-
Donuts (N=86)	**40/46.5**	26/30.2	11/12.8	5/5.8	1/1.2	3/3.5	-	-	-
Cakes (N=87)	**35/40.2**	22/25.3	17/19.5	9/10.3	-	3/3.4	-	1/1.1	-
Pies, puddings (N=87)	**39/44.8**	16/18.4	20/23	8/9.2	-	4/4.6	-	-	-
Jam and honey (N=87)	**40/46**	19/21.8	13/14.9	6/6.9	1/1.1	8/9.2	-	-	-
Popcorn fat free (N=86)	**63/73.3**	12/14	7/8.1	1/1.2	-	3/3.5	-	-	-
Popcorn with butter (N=86)	**65/75.6**	13/15.1	5/5.8	1/1.2	-	2/2.3	-	-	-
Pretzels (N=86)	**53/61.6**	11/12.8	12/14	7/8.1	1/1.2	2/2.3	-	-	-
Hazelnuts (N=86)	**54/62.8**	18/20.9	9/10.5	3/3.5	-	2/2.3	-	-	-
Dried fruit (N=86)	**70/81.4**	7/8.1	5/5.8	3/3.5	1/1.2	-	-	-	-
Regular mayonnaise (N=86)	**50/58.1**	18/20.9	8/9.3	7/8.1	1/1.2	1/1.2	1/1.2	-	-
Salad dressing (N=86)	**62/72.1**	9/10.5	8/9.3	6/7	1/1.2	-	-	-	-
Ice cream (N=87)	**27/31**	22/25.3	16/18.4	12/13.8	2/2.3	8/9.2	-	-	-
Tomatoes (N=87)	14/16.1	10/11.5	14/16.1	17/19.5	4/4.6	**24/27.6**	4/4.6	-	-
Beans, lentils (N=87)	21/24.1	**24/27.6**	19/21.8	17/19.5	3/3.4	2/2.3	1/1.1	-	-
Peas (N=87)	**31/35.6**	24/27.6	17/19.5	12/13.8	-	3/3.4	-	-	-
Broccoli (N=86)	**53/61.6**	15/17.4	6/7	9/10.5	2/2.3	1/1.2	-	-	-
Cauliflower (N=87)	22/25.3	**31/35.6**	17/19.5	13/14.9	2/2.3	2/2.3	-	-	-
Cabbage (N=87)	13/14.9	21/24.1	**31/35.6**	16/18.4	3/3.4	3/3.4	-	-	-
Carrots (N=87)	13/14.9	10/11.5	20/23	**23/26.4**	9/10.3	11/12.6	1/1.1	-	-
Mixed vegetable stir fry (N=86)	22/25.6	12/14	19/22.1	**23/26.7**	6/7	4/4.7	-	-	-
Eggplant, zucchini (N=87)	12/13.8	23/26.4	**33/37.9**	16/18.4	1/1.1	2/2.3	-	-	-
Cooked spinach (N=87)	20/23	22/25.3	**22/25.3**	17/19.5	2/2.3	4/4.6	-	-	-
Peppers (N=87)	21/24.1	12/13.8	**22/25.3**	17/19.5	6/6.9	7/8	2/2.3	-	-
Onion (N=87)	13/14.9	7/8	**25/28.7**	14/16.1	5/5.7	22/25.3	1/1.1	-	-
Raisins, grapes (N=86)	**42/48.8**	13/15.1	14/16.3	9/10.5	-	7/8.1	1/1.2	-	-
Fresh or dried plums (N=86)	**36/41.9**	17/19.8	11/12.8	10/11.6	3/3.5	9/10.5	-	-	-
Apples or pears (N=87)	**19/21.8**	14/16.1	15/17.2	15/17.2	6/6.9	16/18.4	2/2.3	-	-
Bananas (N=86)	17/19.8	14/16.3	16/18.6	**17/19.8**	7/8.1	14/16.3	-	1/1.2	-
Oranges (N=86)	**25/29.1**	15/17.4	20/23.3	18/20.9	2/2.3	6/7	-	-	-
Orange juice (N=85)	**47/55.3**	11/12.9	8/9.4	14/16.5	1/1.2	3/3.5	1/1.2	-	-
Grapefruit (N=87)	**41/47.1**	12/13.8	14/16.1	14/16.1	2/2.3	4/4.6	-	-	-
Strawberries (N=87)	**42/48.3**	19/21.8	12/13.8	6/6.9	1/1.1	7/8	-	-	-
Blueberries (N=86)	**62/72.1**	8/9.3	6/7	7/8.1	-	3/3.5	-	-	-
Peaches (N=87)	**32/36.8**	21/24.1	19/21.8	7/8	1/1.1	7/8	-	-	-
Apricots (N=87)	**30/34.5**	22/25.3	18/20.7	9/10.3	2/2.3	5/5.7	1/1.1	-	-

Question (Affirmative answer - Nr., %)	
How often do you eat fried foods? (N=81)	
< 1 time/week	28 (34.6%)
1-2 times/week	32 (39.5%)
4-6 times/week	14 (17.3%)
Daily	7 (8.6%)
What type of fat do you use for cooking / frying / baking? (Total) (N=81)	
Butter	7 (8.6%)
Margerine	3 (3.7%)
Olive oil	10 (12.3%)
Vegetable cooking oil	69 (85.2%)
Lard	7 (8.6%)
How often do you eat fast food? (N=79)	
< 1 time/week	52 (65.8%)
1-2 times/week	21 (26.6%)
4-6 times/week	5 (6.3%)
Daily	1 (1.3%)

Data from table IV refer to the entire study sample, describing the general distribution of lifestyle and dietary habits among all recruited participants, without stratification by CAP status.

- 31% of the patients exert intense physical effort in their profession with a mean weekly time of 1.87 ± 2.6 days;
- 31% of the patients exert moderate physical effort in their profession with a mean weekly time of 3.26 ± 2.94 days;
- 19% of the patients have high intensity physical activities in their spare time with a mean weekly time of 0.93 ± 2.13 days;
- 36.6% of the patients have moderate intensity physical activities in their spare time with a mean weekly time of 2.05 ± 2.63 days;
- Mean daily time sitting down was 2.75 ± 2.53 hours;
- 53.5% of the patients were smokers, 13.3% of them were trying to quit;
- 50.6% of the patients considered about themselves having a mental condition;
- Most of the patients were rarely consuming 3 meals/day (43.7%), snacks (46.5%) or after 9 pm (44.7%), their most consistent meal was lunch (56%);
- 14.9% of the patients were waking up at night to eat, with a mean weekly frequency of 2.69 ± 2.28 times;
- 7% of the patients were eating large amount of food in less than 2 hours (7%), most of them feeling guilty afterwards (66.7%);
- 34.5% of the patients were eating while watching TV, computer screens or reading;
- Regarding eating habits:
  - o In the drinks category, the most frequently consumed was water (about 4-5 times/day – 32.2%), most of the other products having a very low consumption (less than one time/month);
  - o In the cereals category, the most frequently consumed were white bread/white flour products (2-3 times/day – 39.5%) and white rice (1-3 times/month – 34.5%) most of the other products having a very low consumption (less than one time/month);
  - o In the starch-based fast-food, the most frequently consumed were baked/boiled potatoes (2-4 times/week – 33.3%), pasta (1 time/week -33.3%) or fried potatoes (1-3 times/week – 25.3%), pizza being almost never consumed (49.4%);
  - o In the meat/animal protein category, the most frequently consumed were whole eggs – (2-4 times/week – 32.2%), chicken or turkey meat without skin (2-4 times/week – 37.9%) and pork (2-4 times/week – 33.3%), most of the other products having a very low consumption (less than one time/month);
  - o In the fish category, the most frequently consumed were canned tuna (1 time/week – 18.4%) or fresh tuna (1 time/week – 12.6%), most of the other products having a very low consumption (less than one time/month);
  - o In the diary category, the most frequently consumed were full-fat yogurt (2-4 times/week – 20.7%), cream cheese (2-4 times/week – 20.9%) or regular cheese (2-4 times/week – 23.3%), most of the other products having a very low consumption (less than one time/month);
  - o In the snacks category, the most frequently consumed were pies/puddings (1 time/week – 23%), ice cream (1-3 times/month – 25.3%), donuts (1-3 times/week – 30.2%), cakes (1-3 times/week – 25.3%) and jam or honey (1-3 times/week – 21.8%), most of the other products having a very low consumption (less than one time/month);
  - o In the vegetables category, the most frequently consumed were tomatoes (1 time/day – 27.6%), carrots (2-4 times/week – 26.4%), and mixed vegetable stir fry (2-4 times/week – 26.7%), most of the other products having a low (1 time/week or less) consumption;
  - o In the fruits category, the most frequently consumed were bananas (2-4 times/week – 19.8%), most of the other products having a very low consumption (less than one time/month);
- Regarding fried foods, most of the patients considered eating 1-2 times/week (39.5%) while fast-food consumption was very rare (65.8% - less than one time/week);
- The most frequent type of fat used in cooking was vegetable cooking oil (85.2%), followed by olive oil (12.3%).

For all eating habits, a score for frequency consumption was established, based on the type of response: 0 points for never / less than one time per month, 1 point for 1-3 times/month, 2 points for 1 time/week, 3 points for 2-4 times/week, 4 points for 5-6 times/week, 5 points for 1 time/day, 6 points for 2-3 times/day, 7 points for 4-5 times/day and 8 points for ≥ 6 times/day.

Associations between chronic apical periodontitis (CAP) and patients' lifestyle and dietary habits

To evaluate the relationship between lifestyle, diet, and CAP, comparisons were made between individuals with and without CAP.

Table V. Distribution of the patients according to the existence of chronic apical peridontitis and survey items.

Item (Nr., %)	No CAP	With CAP	p*
Intense effort - Profession	3 (23.1%)	23 (32.4%)	0.746
Moderate effort - Profession	9 (69.2%)	38 (57.6%)	0.544
Intense effort – Spare time	4 (33.3%)	11 (16.4%)	0.227
Moderate effort - Spare time	6 (46.2%)	24 (34.8%)	0.534
Smoking	6 (42.9%)	40 (55.6%)	0.400
Quitting smoking	0 (0%)	6 (15.4%)	0.576
Mental retard	6 (42.9%)	37 (52.1%)	0.571
3 times/day meals			
Daily	5 (35.7%)	30 (41.1%)	0.096
Most times	5 (35.7%)	9 (12.3%)	
Rarely	4 (28.6%)	34 (46.6%)	
Eating snacks			
Daily	3 (21.4%)	24 (33.3%)	**0.032**
Most times	**7 (50%)**	**12 (16.7%)**	
Rarely	4 (28.6%)	36 (50%)	
Most consistent meal			
Breakfast	3 (25%)	19 (26.4%)	0.703
Lunch	8 (66.7%)	39 (54.2%)	
Dinner	1 (8.3%)	14 (19.4%)	
Breakfast every day	7 (50%)	42 (57.5%)	0.770
Eating after 9 pm			
Daily	5 (38.5%)	33 (45.8%)	0.907
Most times	1 (7.7%)	8 (11.1%)	
Rarely	7 (53.8%)	31 (43.1%)	
Waking up at night to eat	2 (14.3%)	11 (15.1%)	1.000
Eating large amounts of food	2 (14.3%)	4 (5.6%)	0.251
Feeling guilty after eating much	1 (50%)	3 (75%)	1.000
Eating while watching TV, computer	5 (35.7%)	24 (34.3%)	1.000
Fried foods consumption			
<1/week	7 (58.3%)	21 (30.4%)	0.228
1-2/week	2 (16.7%)	30 (43.5%)	
4-6/week	2 (16.7%)	12 (17.4%)	
Daily	1 (8.3%)	6 (8.7%)	
Fast foods consumption			
<1/week	10 (90.9%)	42 (61.8%)	0.373
1-2/week	1 (9.1%)	20 (29.4%)	
4-6/week	0 (0%)	5 (7.4%)	
Daily	0 (0%)	1 (1.5%)	
Fat used for cooking			
Butter	1 (8.3%)	6 (8.7%)	1.000
Margarine	1 (8.3%)	2 (2.9%)	0.386
Olive oil	1 (8.3%)	9 (13%)	1.000
Vegetable cooking oil	10 (83.3%)	59 (85.5%)	1.000
Lard	2 (16.7%)	5 (7.2%)	0.276

*Fisher's Exact Test

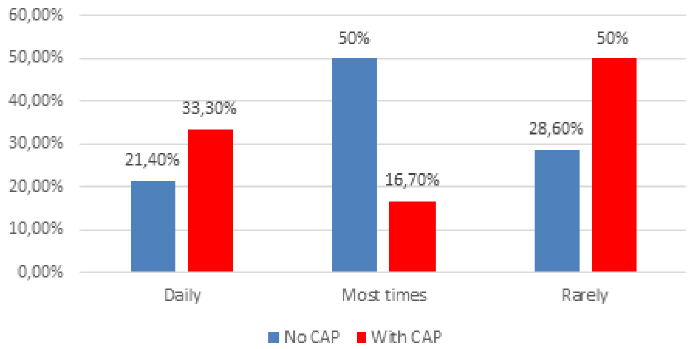

Figure 1. Distribution of the patients according to the existence of chronic apical periodontitis and snacks frequency consumption.

Data from table V and figure 1 show the distribution of the patients according to the existence of chronic apical peridontitis and survey items. The results show that only snack consumption was significantly different between groups (p=0.032), Z-tests with Bonferroni correction showed that patients which eated most times snacks were more rarely associated with chronic apical peridontitis (50% vs. 16.7%), however testing the frequency of CAP between patients with daily snack consumption vs. most times/rarely consumption showed no significant differences (p=0.534), indicating that none of the investigated survey items in table VI were significantly different according to the existence of CAP.

Regarding biochemical markers, no significant differences were observed between CAP and non-CAP groups for AGEs or oxidative stress indices.

Data from table VI show the comparison of AGE and oxidative stress parameters according to the existence of chronic apical periodontitis. According to the results, none of the investigated parameters were significantly different according to the existence of chronic apical periodontitis (p>0.05).

Data from table VII show the comparison of physical activity-related parameters according to the existence of chronic apical periodontitis. According to the results, none of the investigated parameters were significantly different according to the existence of chronic apical periodontitis (p>0.05).

In contrast, several dietary habits showed significant differences between groups, highlighting the potential role of nutrition in CAP occurrence.

Table VI. Comparison of AGE and oxidative stress parameters according to the existence of chronic apical periodontitis.

Parameter (Median (IQR))	No CAP	With CAP	p*
Plasmatic AGE			
FruLys	371.3 (186.3-479.3)	330.8 (157.9-464.1)	0.719
Pyr	27.1 (24.4-29.1)	27.2 (24.8-28.9)	0.939
MG-H1	32.9 (30.7-39.8)	33.2 (30.7-39.8)	0.768
CEL	13.3 (13-13.8)	13.2 (12.9-13.5)	0.301
CML (Mean ± SD)	49.27 ± 5.9	50.16 ± 4.93	0.558**
Arg	3.74 (2.45-7.5)	3.84 (2.56-7.4)	0.847
Lys	6.35 (5.45-7.42)	6.06 (5.26-7.23)	0.362
Salivary AGE			
FruLys	23.3 (20.6-29.2)	22.2 (21-24.2)	0.729
Pyr	1.7 (1.68-2.25)	1.7 (1.69-1.74)	0.633
MG-H1	3.05 (2.43-4.17)	3.05 (2.48-4.72)	0.897
CEL	1.13 (1.03-1.51)	1.26 (1.05-1.41)	0.773
CML	4.42 (2.78-5.48)	3.91 (3.04-5.03)	0.897
Arg	3.91 (3.04-5.03)	0.68 (0.16-1.37)	0.394
Lys	0.81 (0.06-1.93)	0.51 (0.18-0.99)	0.828
Plasmatic oxidative stress parameters			
TAC	1.11 (1.09-53.31)	1.11 (1.09-53.83)	0.956
TOS (Mean ± SD)	39.15 ± 17.89	48.69 ± 17.85	0.093**
OSI	27.13 (1.09-51.57)	28.99 (1.09-47.33)	0.835
NO	34.14 (31.05-52.36)	40.5 (32.81-56.11)	0.218
MDA	9.3 (5.75-360)	7.36	0.547
Total tiols	235.3 (3.3-374)	304 (3.87-440)	0.497
Salivary oxidative stress parameters			
TAC	1.1 (1.09-4.25)	1.1 (1.09-17.26)	0.785
TOS	6.87 (4.39-9.28)	7.55 (6.29-9.56)	0.278
OSI	6.31 (1.1-8.47)	5.63 (1.09-6.98)	0.185
NO	15.63 (3.54-26.58)	9.9 (7.48-15.06)	0.499
MDA	2.94 (1.85-65)	3.67 (2.08-42)	0.922
Total tiols	111.6 (2.94-157)	77 (2.65-113)	0.134

*Mann-Whitney U Test, **Student T-Test

Table VII. Comparison of physical activity-related parameters according to the existence of chronic apical periodontitis.

Parameter (Median (IQR))	No CAP	With CAP	p*
Intense effort – Profession (days/week)	0 (0-2.5)	0 (0-5)	0.251
Moderate effort – Profession (days/week)	3 (0-6)	4 (0-6.75)	0.586
Intense effort – Spare time (days/week)	0 (0-0.75)	0 (0-0)	0.894
Moderate effort – Spare time (days/week)	0 (0-3.5)	0 (0-4.75)	0.665
Time sitting/lying down (hours/day)	2 (1.38-4.25)	2 (1-3)	0.708

*Mann-Whitney U Test

Table VIII. Comparison of food consumption scores according to the existence of chronic apical periodontitis.

Parameter (Median (IQR))	No CAP	With CAP	p*
Carbonated drink & sweetener	0.5 (0-2)	0 (0-3)	0.833
Carbonated drink with sugar	0.5 (0-2.25)	1 (0-3)	0.578
Non-carbonated drink & sugar	0 (0-0.5)	1 (0-3)	**0.046**
Regular beer	0 (0-1.25)	0 (0-1)	0.820
Light beer	0 (0-0)	0 (0-0)	0.897
Red wine	0 (0-0)	0 (0-0)	0.814
White wine	0 (0-0.25)	0 (0-0)	0.140
Liquer, spirits	0 (0-1)	0 (0-0)	0.472
Water	7 (2.25-8)	7 (5-7)	0.717
Cereal for breakfast	1 (0-5)	0 (0-1)	**0.040**
White bread/white flour prod.	5 (0-6)	5 (3-6)	0.155
Wholemeal bread	0 (0-5)	0 (0-4)	1.000
Croissants, muffins or biscuits	0 (0-1)	1 (0-3)	**0.046**
Pancakes/waffles	1 (0-2)	1 (0-1)	0.954
Black, brown rice	0 (0-2)	0 (0-0)	0.088
White rice	0.5 (0-1.25)	1 (0.5-2)	**0.049**
Pasta	1 (1-2.25)	2 (1-2)	0.872
Fried potatoes	1 (0-1.25)	2 (1-3)	**0.007**
Baked, boiled potatoes	2 (0-2.25)	2 (1-3)	0.200
Pizza	0 (0-1)	1 (0-2)	0.196
Whole eggs	2 (0-3)	2 (0-3)	0.688
Pork or beef hot dog	0 (0-0)	0 (0-1)	0.375
Hot dogs or chciken sausages	0 (0-1)	0 (0-0.75)	0.963
Chicken/turkey meat (skin)	1 (0-2.25)	2 (0-3)	0.312
Chicken/turkey meat (no skin)	3 (0-3.5)	2 (1-3)	0.509
Bacon, kaiser	1 (0.75-4.25)	1 (0-3)	0.210
Salami or other meat products	1 (0-3)	1 (0-3)	0.752
Sausages / other minced meat	1 (0-1.25)	2 (0-2)	0.247
Hamburger	0 (0-1.5)	0 (0-0)	0.453
Pork	0 (0-3)	2 (1-3)	0.168
Beef, lamb	1 (0-1)	0 (0-2)	0.860
Canned tuna	0 (0-2)	0 (0-1)	1.000
Semi-prepared breaded fish	0 (0-1)	0 (0-1)	0.587
Prawns, clams, lobster	0 (0-0)	0 (0-0)	0.603
Tuna, salmon, sardines	0 (0-1.25)	0 (0-1)	0.841
Cod, haddock, halibut	0 (0-1)	0 (0-0)	0.184
Skimmed milk	0.5 (0-2.75)	0 (0-1)	0.121
Milk 1-2% fat	0 (0-2.75)	0 (0-1)	0.368
Whole milk	0 (2.75)	0 (0-1)	0.731
Soy milk	0 (0-0)	0 (0-0)	0.343

Table VIII. Comparison of food consumption scores according to the existence of chronic apical periodontitis (continuation).

Parameter (Median (IQR))	No CAP	With CAP	p*
Cream	0 (0-0.25)	0 (0-0)	0.864
Full-fat yogurt	1.5 (0-5)	1 (0-3)	0.418
Fruit yogurt	0 (0-0.5)	0 (0-2)	0.120
Cottage cheese	1 (0-2)	1 (0-2)	0.753
Cream cheese	2 (0.5-3)	1 (0-2)	0.087
Cheese, cheddar	1 (0-3)	1 (0-2.5)	0.607
Milk chocolate	0 (0-0.25)	0 (0-2)	0.087
Dark chocolate	0 (0-2.5)	0 (0-1.5)	0.887
Chocolate bars	0 (0-0)	0 (0-2)	0.068
Donuts	1 (0-1)	1 (0-1.5)	0.816
Cakes	0 (0-2.25)	1 (0-2)	0.303
Pies, puddings	0 (0-2)	1 (0-2)	0.616
Jam and honey	0 (0-1.5)	1 (0-2)	0.275
Popcorn fat free	0 (0-1)	0 (0-1)	0.625
Popcorn with butter	0 (0-0)	0 (0-1)	0.470
Pretzels	0 (0-1)	0 (0-2)	0.380
Hazelnuts	0 (0-0)	0 (0-1)	0.057
Dried fruit	0 (0-0)	0 (0-0)	0.790
Regular mayonnaise	0 (0-0.5)	0 (0-1)	0.153
Salad dressing	0 (0-2.5)	0 (0-1)	0.243
Ice cream	1.5 (0-2.25)	1 (0-3)	0.775
Tomatoes	3 (0.75-5)	3 (1-5)	0.728
Beans, lentils	1 (0-2)	2 (1-3)	0.071
Peas	1.5 (0-2)	1 (0-2)	0.670
Broccoli	0 (0-2.5)	0 (0-1)	0.323
Cauliflower	1 (0-2.25)	1 (0-2)	0.810
Cabbage	2 (1-2.25)	2 (1-3)	0.645
Carrots	2 (1.5-3.5)	3 (1-3.5)	0.818
Mixed vegetable stir fry	2 (0-3)	2 (1-3)	0.734
Eggplant, zucchini	2 (0.75-2)	2 (1-2)	0.551
Cooked spinach	1 (0-3)	2 (1-3)	0.665
Peppers	2 (0.75-5)	2 (0.5-3)	0.328
Onion	2 (1.5-5)	2 (2-4)	0.785
Raisins, grapes	2 (0-2.5)	0 (0-2)	0.360
Fresh or dried plums	1 (0.5-3)	1 (0-2.5)	0.251
Apples or pears	3 (1.75-5)	2 (0.5-3.5)	0.060
Bananas	3 (0.5-4.5)	2 (1-3)	0.821
Oranges	1 (0-1)	2 (0-3)	**0.006**
Orange juice	0 (0-1)	0 (0-2.75)	0.400
Grapefruit	1 (0-2.25)	1 (0-2)	0.941
Strawberries	0 (0-1.25)	1 (0-2)	0.291
Blueberries	0 (0-1)	0 (0-1)	0.976
Peaches	1 (0-2)	1 (0-2)	0.387
Apricots	1 (0-2)	1 (0-2)	0.290

*Mann-Whitney U Test

Data from table VIII show the comparison of food consumption scores according to the existence of chronic apical periodontitis. According to the results, the following food scores were significant between groups:

- Non-carbonated drink with sugar score, significantly higher in patients with CAP vs. non- CAP (median = 1, IQR = 0-3 vs. median = 0, IQR = 0-0.5, p=0.046);

Table IX. Univariate and multivariate logistic regression models used in the prediction of the existence of chronic apical periodontitis.

Parameter	Univariate		Multivariate*	
	OR (95% C.I.)	p	OR (95% C.I.)	p
Non-carbonated drink & sugar	1.39 (0.89-2.19)	0.145	-	-
Cereal for breakfast	0.74 (0.57-0.95)	**0.022**	0.73 (0.54-0.99)	**0.045**
Croissants, muffins or biscuits	1.43 (0.91-2.23)	0.115	-	-
White rice	1.97 (0.99-3.95)	0.053	-	-
Fried potatoes	2.15 (1.17-3.93)	**0.013**	2.18 (1.07-4.43)	**0.030**
Oranges	2.13 (1.18-3.86)	**0.012**	2.24 (1.13-4.43)	**0.020**

*Multivariable logistic regression model, χ^2 (3) = 19.992, p<0.001, Nagelkerke R^2 = 0.362, Hosmer and Lemeshow Test = 0.693, Sensitivity = 97.3%, Specificity = 23.1%, Overall accuracy = 86%.

- Cereal for breakfast score, significantly lower in patients with CAP vs. non- CAP (median = 0, IQR = 0-1 vs. median = 1, IQR = 0-0.5, p=0.040);
- Croissants, muffins or biscuits score, significantly higher in patients with CAP vs. non- CAP (median = 1, IQR = 0-3 vs. median = 0, IQR = 0-1, p=0.046);
- White rice score, significantly higher in patients with CAP vs. non- CAP (median = 1, IQR = 0.5-2 vs. median = 0.5, IQR = 0-1.25, p=0.049);
- Fried potatoes score, significantly higher in patients with CAP vs. non- CAP (median = 2, IQR = 1-3 vs. median = 1, IQR = 0-1.25, p=0.007);
- Oranges score, significantly higher in patients with CAP vs. non- CAP (median = 2, IQR = 0-3 vs. median = 1, IQR = 0-1, p=0.006).

Data from table IX show the univariate and multivariate logistic regression models used in the prediction of the existence of chronic apical periodontitis. According to the results observed in the multivariable model, each food consumption score which was significant in the univariate models, maintained its significant prediction over the existence of CAP in the multivariate model, indicating that the following variables had a significant and independent impact over the prediction of chronic apical periodontitis:

- An increase of one point in the consumption score in case of cereals for breakfast significantly decreases the odds of having CAP by 1.37 times (95% C.I. = 1.01-1.85) (p=0.045);
- An increase of one point in the consumption score in case of fried potatoes significantly increases the odds of having CAP by 2.18 times (95% C.I. = 1.07-4.43 (p=0.030);
- An increase of one point in the consumption score in case of oranges significantly increases the odds of having CAP by 2.24 times (95% C.I. = 1.13-4.43 (p=0.020).

Discussion

The present study investigated the relationship between CAP, oxidative stress markers, AGEs, lifestyle habits, and dietary patterns. The findings of our study confirm that CAP is a highly prevalent condition, affecting 83.1% of the investigated patients.

Biomarkers of oxidative stress and AGEs

Although both AGEs and oxidative stress markers are widely recognized as contributors to chronic inflammation and tissue damage [16,17], our results did not demonstrate significant differences between CAP and non-CAP groups for plasma or salivary AGEs (Table I) and oxidative stress markers (Table II). This suggests that in this population, these biomarkers may not play a decisive role in differentiating between affected and unaffected patients. CAP is a localized chronic disease, and systemic biomarker levels may not accurately reflect the localized inflammatory activity at the periapical site (Table VI) [18,19].

Lifestyle and behavioral factors

With regard to smoking and physical activity, our results did not reveal significant associations with CAP (Table III). While this seems to contradict the established role of smoking as a major risk factor for impaired periapical and periodontal healing, the lack of significance may be attributed to the sample size. It is also possible that self-reporting bias influenced the accuracy of lifestyle-related data [20].

Dietary habits and CAP

We identified a significant association between CAP and higher consumption of fried potatoes, croissants/muffins/biscuits, white rice and oranges, while regular breakfast cereal intake appeared to be protective (Table VIII). The logistic regression analysis (Table IX) showed that people who frequently ate fried potatoes and oranges were more than twice as likely to have CAP, while those who regularly consumed cereals had about a one-third lower risk. Our results are consistent with previous research showing that diet plays an important role in oral inflammatory conditions. Diets rich in refined carbohydrates and unhealthy fats are known to fuel systemic inflammation and disturb the oral microbiota [21]. In particular, fried foods can raise oxidative stress and stimulate the production of inflammatory citokines [22], which may worsen periapical inflammation and slow down healing.

The association with oranges is more complex.

On the one hand, oranges are rich in vitamin C, a nutrient essential for collagen synthesis and periodontal health [23]. On the other hand, their acidic content and frequent consumption can lower oral pH, potentially damaging hard tissue [24]. This dual effect may explain the unexpected positive association with CAP observed in our cohort, though further research is needed to confirm this finding.

By contrast, breakfast cereal consumption was found to be protective. Wholegrain cereals provide dietary fibers, B vitamins, and micronutrients that contribute to methabolic and microbiome stability . Their regular intake may improve satiety, reduce consumption of high-sugar snacks, and support a healthier inflammatory profile, thereby reducing susceptibility to CAP.

Our findings highlight that nutrition may play a meaningful role in both the prevention and management of endodontic and periapical conditions. While CAP has traditionally been understood mainly as the result of microbial infection and the succes or failure of endodontic treatment, our results indicate that diet can also shape disease risk and healing outcomes. This suggests that integrating basic dietary counseling into dental care, especially for patients who struggle with reccurent slow-healing periapical lesions, could be a valuable addition to conventional treatment strategies.

Conclusions

While systemic biomarkers of oxidative stress and AGEs did not differ significantly between groups, dietary habits emerged as strong predictors of CAP. Frequent intake of fried foods and acidic fruits such as oranges increased the risk of CAP, whereas regular breakfast cereal consumption had a protective effect. These results highlight the importance of integrating nutritional education into oral healthcare and suggest that CAP should be understood not only as an infectious disease but also as a condition influenced by systemic and lifestyle-related factors.

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SALIVAGES Project group

Nausica Bianca Petrescu, Ioana Roxana Bordea, Maria Crișan, Elena Alina Pârvu, Ana Uifălean, Anca Ștefania Mesaros, Friederike Mani, Michael Hellwig, Thomas Henle

References

1. Nair PN. Pathogenesis of apical periodontitis and the causes of endodontic failures. Crit Rev Oral Biol Med. 2004;15:348-381.
2. Mukhtar O, Jackson SH. Drug therapies in older adults (part 2). Clin Med (Lond). 2015;15:155-159.
3. Wang Y, Andrukhov O, Rausch-Fan X. Oxidative Stress and Antioxidant System in Periodontitis. Front Physiol. 2017;8:910.
4. Macedo Signorelli NS, Rende SGS, Iorio EL, Ferraz DC, Paranhos LR, Moura CCG. Identification of oxidative stress biomarkers in apical periodontitis: A scoping review with bibliometric analysis. Aust Endod J. 2024;50:742-760.
5. Hujoel P. Dietary carbohydrates and dental-systemic diseases. J Dent Res. 2009;88:490-502.
6. Ghaemmaghami RS, Bayani M, Nakhostin A, Vahid F. A pro-inflammatory diet is associated with an increased odds of periodontitis: finding from a case-control study. BMC Nutr. 2023;9:109.
7. Makki K, Deehan EC, Walter J, Bäckhed F. The impact of dietary fiber on gut microbiota in host health and disease. Cell Host Microbe. 2018;23:705-715.
8. Jefferson A, Adolphus K. The effects of intact cereal grain fibers, including wheat bran on the gut microbiota composition of healthy adults: a systematic review. Front Nutr. 2019;6:33.
9. Milesi G, Rangan A, Grafenauer S. Whole Grain Consumption and Inflammatory Markers: A Systematic Literature Review of Randomized Control Trials. Nutrients. 2022;14:374.
10. Garutti M, Nevola G, Mazzeo R, Cucciniello L, Totaro F, Bertuzzi CA, et al. The Impact of Cereal Grain Composition on the Health and Disease Outcomes. Front Nutr. 2022;9:888974.
11. Băbțan AM, Ilea A, Boșca BA, Crișan M, Petrescu NB, Collino M, et al. Advanced glycation end products as biomarkers in systemic diseases: premises and perspectives of salivary advanced glycation end products. Biomark Med. 2019;13:479-495.
12. Candrea S, Bondor C, Boșca A, Boca A, Muntean A, et al. Behind the scene view of oxidative stress markers and dental caries. Rom J Oral Rehab. 2025;17:857-868.
13. Roman G, Bala C, Creteanu G, Graur M, Morosanu M, Amorin P, et al. Obesity and health-related lifestyle factors in the general population in Romania: A cross sectional study. Choroid Plexus - Pineal Gland Correlations Medical Anthropology - Computed Tomography Studies Intracranial Physiological Calcification. 2015;11:64-71.
14. Boca A, Bondor C, Cimpean S, Bâldea I, Candrea S, Ilea A. Association between advanced glycation end products and inflammation in root canal infections: a cross-sectional observational study. Rom J Oral Rehab. 2025;17:173-186.
15. Boca A, Delean A, Cimpean S, Boșca A, Bondor C, Candrea S, et al. Association between chronic apical periodontitis and oxidative stress or meta-inflammation: a cross-sectional observational study. Rom J Oral Rehab. 2024;16:633-650.
16. Nair PN. Pathogenesis of apical periodontitis and the causes

of endodontic failures. Crit Rev Oral Biol Med. 2004;15:348-381.

17. Baik JE, Kum KY, Yun CH, Lee JK, Lee K, Kim KK, et al. Calcium hydroxide inactivates lipoteichoic acid from Enterococcus faecalis. J Endod. 2008;34:1355-1359.
18. Macedo Signorelli NS, Rende SGS, Iorio EL, Ferraz DC, Paranhos LR, Moura CCG. Identification of oxidative stress biomarkers in apical periodontitis: A scoping review with bibliometric analysis. Aust Endod J. 2024;50:742-760.
19. Barcelos RCS, Rosa HZ, Roversi K, Tibúrcio-Machado CDS, Inchaki PT, Burger ME, et al. Apical periodontitis induces changes on oxidative stress parameters and increases Na+/K+-ATPase activity in adult rats. Arch Oral Biol. 2020;118:104849.
20. Bergström J. Tobacco smoking and chronic destructive periodontal disease. Odontology. 2004;92:1-8.
21. Hujoel P. Dietary carbohydrates and dental-systemic diseases. J Dent Res. 2009;88:490-502.
22. Uribarri J, Woodruff S, Goodman S, Cai W, Chen X, Pyzik R, et al. Advanced glycation end products in foods and a practical guide to their reduction in the diet. J Am Diet Assoc. 2010;110:911-916.e12.
23. Chapple IL, Genco R; working group 2 of the joint EFP/AAP workshop. Diabetes and periodontal diseases: consensus report of the Joint EFP/AAP Workshop on Periodontitis and Systemic Diseases. J Periodontol. 2013;84:S106-112.
24. Imazato S, Walls AW, Kuramoto A, Ebisu S. Penetration of an antibacterial dentine-bonding system into demineralized human root dentine in vitro. Eur J Oral Sci. 2002;110:168-174.