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Dietary intake in patients with peripheral arterial disease and concomitant periodontal disease

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Abstract

Nutrition plays a crucial role in the pathophysiology and management of peripheral arterial disease (PAD) and periodontal disease (PD). As PD can have profound effects on an individual's functional ability to eat and can affect nutrient intake, we aimed to evaluate the role of PD severity on dietary intake (DI) and quality in PAD patients and compare it with current dietary recommendations for CVD. PD stages of 421 consecutive PAD patients were determined according to a standardised basic periodontal examination (Periodontal Screening and Recording Index) ('healthy', 'gingivitis', 'moderate periodontitis' and 'severe periodontitis'). Dietary intake (24-h recall), dietary quality (food frequency index (FFI)) and anthropometrical data were assessed. Nutritional intake was stratified according to the severity of PD. No significant differences in DI of macronutrients, nutrients relevant for CVD and FFI were seen between the PD stages. Only median alcohol intake was significantly different between gingivitis and severe periodontitis ($P = 0{\cdot}001$), and positively correlated with PD severity ($P = 0{\cdot}001$; $r\,0{\cdot}159$). PD severity and the patient's number of teeth showed no correlation with investigated nutritional parameters and FFI. Few subjects met the recommended daily intakes for fibre (5 %), SFA (10 %), Na (40 %) and sugar (26 %). Macronutrient intake differed from reference values. In our sample of patients with PAD and concomitant PD, we found no differences in DI of macronutrients, nutrients relevant for CVD and diet quality depending on PD severity. The patients' nutrition was, however, poor, deviating seriously from dietary guidelines and recommendations.

Key words: **Dietary intake: Diet quality: Peripheral arterial disease: Periodontal disease: CVD: Food frequency questionnaires**

There is an epidemiological association between peripheral arterial disease (PAD) and periodontal disease (PD)[1–3], both of which are aggravated by inflammatory processes[4,5]. Nutritional factors influence this significant inflammatory burden in affected individuals[6]. PAD patients experience a high risk of ischaemic events [5] and so need adequate secondary prevention strategies. These include modification of dietary habits[7], since a growing body of evidence emphasises the role of nutrition in the pathophysiology and management of both PAD[8–18] and PD[19–21]. PAD patients in particular are poorly nourished, with an insufficient intake of fibre[8,15], vitamin E[8–10], folic acid[8,10] and *n*-3 PUFA[10,15] and high intakes of Na[8,15], cholesterol[8,15] and saturated fat[8,15].

Poor oral health is a common condition, especially among the elderly, and is associated with an increased risk for atherosclerotic CVD[3]. PD causes chewing discomfort, impairs the

Abbreviations: ACC, American College of Cardiology; AHA, American Heart Association; CHO, carbohydrate; DI, dietary intake; ESC, European Society of Cardiology; FFI, food frequency index; PAD, peripheral arterial disease; PD, periodontal disease; PSR, Periodontal Screening and Recording; TEI, total daily energy intake.

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functional ability to eat and negatively affects diet quality and the intake of essential nutrients[22,23]. Such patients show a reduced intake of vitamin C, folic acid, Mg and fibre as compared with healthy controls[24]. Individuals with impaired oral health avoid foods that are difficult or painful to chew[23,25]. These foods include fresh vegetables and fruit, nuts, seeds and whole grain products, among others, which are sources of essential nutrients and dietary fibre[23].

Since the impact of PD severity on dietary intake (DI) and quality in patients with CVD has not been elucidated, the present study aimed (1) to evaluate the effect of PD severity on diet quality and DI of nutrients relevant for CVD in patients with PAD and (2) to compare DI of this patient population with the current dietary recommendations for CVD. We hypothesised that the severity of PD in PAD patients influences DI and that the consumption of nutrients relevant to CVD will differ from current dietary guidelines.

Methods

Study population and design

The present study evaluated data from a single-centre, prospective, randomised, open trial conducted at the Division of Angiology, Medical University of Graz, Austria, investigating the influence of periodontal therapy on vascular inflammation and function in patients with PAD (PeriPAD trial). PeriPAD was registered as a randomised controlled trial at the Deutsches Register Klinischer Studien (https://drks-neu.uniklinik-freiburg.de/drks_web/) ID: 00004554. The present study was conducted according to the guidelines laid down in the Declaration of Helsinki, and all procedures involving human subjects/patients were approved by the Ethics Committee of the Medical University of Graz, Austria (EK-Nr. 24-456 ex 11/12). Written informed consent was obtained from all subjects/patients. Recruitment took place between March 2013 and January 2015.

Patients who fulfilled the following key inclusion criteria were invited to enrol in the study: written informed consent; current or previously diagnosed symptomatic PAD (Rutherford classification 2–4 (intermittent claudication or rest pain)) and documented luminal stenosis >70 % on ultrasound or angiography or a history of endovascular or surgical revascularisation; periodontal disease determined by the Periodontal Screening and Recording (PSR) Index; signed informed consent form.

The exclusion criteria were defined as PAD Rutherford category 5 and 6 (tissue damage/loss); no natural teeth present; life expectancy <6 months; unstable cerebrovascular disease and CVD; clinically apparent infectious disease (e.g. pneumonia and symptomatic urinary tract infection); systemic inflammatory disease (e.g. chronic inflammatory bowel disease, rheumatoid arthritis, vasculitis by clinical assessment); periodontal treatment within 6 months prior to the study; mouth infection other than periodontitis; uncontrolled diabetes; pregnancy; age <18 years.

Clinical examination/testing

A cohort of 421 consecutive patients was screened for inclusion and exclusion criteria. The study visit was performed in the morning after an overnight fast at the Outpatient Clinic for Preventive Vascular Medicine at the Division of Angiology.

A general medical history was taken. Demographic data, anthropometric data, data on CVD risk factors, medication and pertinent vascular examination records were collected. Blood was sampled to determine circulating biomarkers and blood pressure was measured. The ankle brachial index was measured by trained clinical staff according to current standards and guidelines[26]. Symptomatic PAD was defined as ankle brachial index <0·9 and intermittent claudication or a history of endovascular or surgical revascularisation.

If PAD was diagnosed and at least one original tooth was present, patients were referred to the Division of Prosthodontics and Periodontology, Dental School, Medical University of Graz, to determine dental inclusion criteria. Patients who met the inclusion criteria were enrolled in the study after providing informed consent.

Blood sampling and biochemical analysis

A venous blood sample was drawn from an antecubital vein in the morning after an overnight fast. Analysis was performed on campus by the university's certified laboratory to guarantee rapid processing. Laboratory methods or values were not changed during the study. Automated analysers (Cobas® 8000, Roche; ARCHITECT®, Abbott GmbH & Co. KG; XE 5000®, Sysmex; BNA II nephelometer analyser, Siemens,; IDS-iSYS immunoassay system, IDS) were used to measure serum concentrations of TAG, HDL-cholesterol, total cholesterol, glucose, C-reactive protein, cholesterol/HDL ratio and IL-6. The Friedewald formula was used to determine LDL-cholesterol concentrations. The laboratories were certified according to ISO 9001:2008.

Periodontal examination

The periodontal screening examination included the assessment of the general oral and dental history, determination of the PSR Index and an orthopantomogram. All dental parameters were assessed by experienced periodontists. PSR is an internationally accepted method to examine the periodontium and to detect periodontal diseases[27,28]. Patients were classified according to their PSR stage: 'healthy' (PSR stage 0), 'gingivitis' (PSR stage 1 + 2), 'moderate periodontitis' (PSR stage 3) and 'severe periodontitis' (PSR stage 4).

Dietary assessment

DI was assessed by 24-h food recall (24-h recall) and a thirty-six-item self-administered qualitative FFQ. Participants were asked to report their usual consumption frequency of the thirty-six listed food items and categories during the previous 3 months with frequency options ranging from 'almost never or <1/month' to '>3 times/d'. This recently validated and published, self-administered FFQ has proved to be a good measure of dietary quality in a population of people aged 55 years and older[29]. The information derived from our FFQ was used to calculate the food frequency index (FFI). The development of this score has been described elsewhere[29].

Since elderly patients sometimes have difficulty filling out questionnaires by themselves, a trained dietitian was available for support, checked the FFQ for completeness and undertook the structured 24-h recall together with the patients.

Table 1. Patient characteristics (*n* 412)
(Mean values and standard deviations; numbers and percentages; median and interquartile range (IQR))

	Mean	SD	*n*	%
Age (years)	63	10		
Height (cm)	171	9		
Weight (kg)	80	14		
BMI (kg/m^2)	27	4		
Males			297	70
Smoking status				
Never			69	17
Former			195	47
Current			148	36
Hypertension			332	81
Obesity			98	24
Diabetes			114	28
Systolic blood pressure (mmHg))	151	20		
Diastolic blood pressure (mmHg))	85	11		
C-reactive protein (mmol/l)	2·6	4·3		
IL-6 (pg/ml)	3·8	3·0		
Glucose (mmol/l)	111	37		
TAG (mmol/l)	120	81		
Cholesterol (mmol/l)	180	47		
HDL-cholesterol (mmol/l)	52	17		
LDL-cholesterol (mmol/l)	99	38		
Neurological events (haemorrhagic and ischaemic stroke)			44	10
CVD (MCI, PCI, CABG)			92	22
Vascular intervention (EVR/bypass)			306	73
Ankle brachial index				
Median	0·63			
IQR	0·5			
Statins			315	75
Antithrombotic agent or anticoagulation			43	10
Angiotensin-converting enzyme/angiotensin receptor blockers			271	64
Ca receptor antagonists			121	29
Marital status				
Single			48	11
Married			249	58
Compulsory schooling			286	68
Completion of secondary education			58	14
University degree/further education			30	7

MCI, myocardial infarction; PCI, percutaneous coronary intervention; CABG, coronary artery bypass graft; EVR, endovascular revascularisation.

Nutrient intake from 24-h recall was subsequently calculated using nut.s software (nutritional.software, dato Denkwerkzeuge). Our results were compared with the D-A-CH reference values for nutrient supply (German Nutrition Society (DGE), the Austrian Nutrition Society and the Swiss Society for Nutritional Research)[30] and recommendations for CVD prevention of the European Society of Cardiology (ESC)[31] and American Heart Association/American College of Cardiology (AHA/ACC)[32–34].

Statistical analysis

Statistical analyses were as follows: For descriptive statistics, means and standard deviations were calculated for normally distributed data; otherwise, medians and interquartile ranges (25th–75th percentiles) were calculated (Table 1). Normal distribution of data was tested by the Kolmogorov–Smirnov test. The Kruskal–Wallis test was used to determine differences in nutrient intake (a *P* value of <0·007 was considered statistically significant after Bonferroni correction[35] to account for multiple testing) (Table 2) and in macronutrient consumption (Table 3) between the three periodontal disease groups. Spearman's correlation coefficient was used in correlation analysis. All analyses were completed with SPSS for Windows version 23 (SSCP Inc.).

Recommendations for the intake of specific nutrients were obtained from the guidelines for CVD prevention by the ESC[31] and AHA/ACC[32].

Results

A total of 807 patients were screened for study inclusion, and 386 patients were not eligible due to various reasons (see Fig. 1). Finally, 421 patients were included for analysis. Nine patients had to be excluded for analysis due to screening failure. Twelve patients had to be excluded due to missing dental data, leaving 400 participants for analysis.

Patients' characteristics are summarised in Table 1. A flow chart of the screening process is shown in Fig. 1.

Impact of periodontal disease severity on dietary intake and quality

We found a very high prevalence of PD among our PAD patients (99·8 %), of whom 53·9 % (*n* 216) were diagnosed with severe periodontitis. Only one of the patients was classified as dentally healthy.

Nutritional intake was stratified according to the severity of PD. No difference in DI of total fibre, cholesterol, *n*-3 PUFA, SFA, sugar and Na was seen among PAD patients with gingivitis, moderate periodontitis and severe periodontitis. Alcohol consumption differed between PD stages and showed a positive correlation with PD severity ($P = 0{\cdot}001$; r 0·159) (Table 2). *Post hoc* testing revealed a significant difference in median alcohol intake between patients with gingivitis and severe periodontitis ($P = 0{\cdot}001$).

PD severity and patients' median number of teeth[18,10–24] did not correlate with other investigated nutritional parameters and FFI. The FFI indicated no difference in diet quality among the PD stages.

Impact of periodontal disease severity and number of teeth on macronutrient composition

The non-parametric Kruskal–Wallis test showed no difference in macronutrient intake between the three PD stages. However, daily macronutrient intake (in %) differed from D-A-CH reference values for nutrient supply[30]. On average, the subjects consumed fewer than the daily reference value of 9204 kJ/d for men 51 years of age and older at a physical activity level of 1·4[30]. Dietary fat intake was well above the recommended 30 % of total daily energy intake (TEI)[30] (Table 3), which 80 % of the participants exceeded. Median daily carbohydrate (CHO) consumption of all subjects was below the recommended intake value (D-A-CH: 50–60 % of TEI[30]) with 41·5 % of TEI. Protein intake of this patient population was found to be adequate (Table 3).

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Table 2. Dietary intake (24-h recall) of nutrients relevant for CVD and diet quality in peripheral arterial disease patients with gingivitis, moderate periodontitis and severe periodontitis
(Medians and 25th–75th percentiles)

	Gingivitis: PSR 1 + 2 (*n* 122)		Moderate periodontitis: PSR 3 (*n* 62)		Severe periodontitis: PSR 4 (*n* 216)		
	Median	25th–75th percentiles	Median	25th–75th percentiles	Median	25th–75th percentiles	*P**
Total dietary fibre (g)	17	13–24	17	12–22	16	12–21	0·226
Cholesterol (mg)	234	162–366	291	181–426	287	176–430	0·037
Dietary *n*-3 PUFA (g)	1·1	0·9–1·7	1·3	0·8–1·8	1·2	0·9–1·8	0·617
SFA (g)	35	24–46	36	22–50	35	24–50	0·969
Sugar (g)	77	53–104	74	34–110	66	37–106	0·149
Na (mg)	2466	1728–3194	3105	1834–4290	2535	1935–3760	0·033
Alcohol (g)	0	0–11	0·1	0–18	0·7	0–20	0·006
Food frequency index	30	26–33	28	26–33	29	25–32	0·163

PSR, Periodontal Screening and Recording Index.
* *P* value was calculated from the Kruskal–Wallis test for continuous variables.

Table 3. Comparison of macronutrient intake among peripheral arterial disease patients with gingivitis, moderate periodontitis and severe periodontitis (Medians and 25th–75th percentiles)

		Gingivitis (*n* 122)		Moderate periodontitis (*n* 62)		Severe periodontitis (*n* 216)		
	Reference values	Median	25th–75th percentiles	Median	25th–75th percentiles	Median	25th–75th percentiles	*P*‡
Total daily energy intake (kJ)*	9204	7698	6475–9391	8383	6385–11 335	8072	6444–9971	0·373
% Carbohydrates of daily EI	> 50 %†	42	38–50	41	34–51	41	34–48	0·116
% Fat of daily EI	< 30 %†	41	35–46	38	32–47	39	31–47	0·456
Protein intake (g/kg BW)	0·8 g/kg BW	0·8	0·6–1·1	1	0·7–1·2	0·9	0·7–1·2	0·220
% Protein of daily EI	10–15 %†	15	11–17	15	12–18	15	12–18	0·412

EI, energy intake; BW, body weight.
*D-A-CH reference values for male subjects, 51–65 years, physical activity level = 1·4 was used[30].
† Percentage of daily energy intake.
‡ *P* values were calculated from the non-parametric Kruskal–Wallis test.

Dietary intake relative to recommendations

Table 4 displays the nutritional intake of patients in comparison with established guidelines for CVD prevention.

The recommended daily amount of *n*-3 PUFA[33,34] was consumed by two-thirds of our patients with both PAD and PD, with 1·7 % of the patients reporting the daily use of *n*-3 PUFA supplements. Our subjects showed elevated intake levels of SFA (90 % exceeded the cut-off point of 10 % of TEI, as recommended by the ESC[31]), Na and sugar. An inadequate intake of total dietary fibre was reported (95 % of subjects did not meet the ESC cut-off[31]). Recommendations for alcohol consumption were met by >70 % of this patient population (Table 4).

Discussion

The main finding of this cross-sectional study was that overall DI of nutrients relevant for CVD and diet quality did not differ depending on the severity of PD in patients with PAD. Nevertheless, macronutrient consumption and intake of nutrients relevant for CVD greatly deviated from current dietary guidelines.

Impact of periodontal disease severity on dietary intake and quality

Previous studies have reported an impairment of the functional ability to eat in patients affected with PD and poor oral health. This contributed to poor composition and quality of the patient's diet[23]. In a study by Staudte *et al.*[24], approximately 50 % of the patients with chronic periodontitis experienced oral discomfort while eating. An individual's dental condition and tooth loss, which may be related to PD, can also influence DI of essential nutrients and the selection of foods consumed[23–25]. It could therefore be suspected that patients with a more severe degree of PD would have more trouble chewing and so would not consume enough essential nutrients. Foods difficult to masticate include many with a healthful profile such as fruits, raw vegetables, nuts and whole grain bread, among others[23,25].

In our study, the prevalence of moderate and severe periodontitis in screened patients was 69 %, which is similar to what has been reported in the literature[36]. Despite this high prevalence of PD among our PAD patients, the degree of PD did not affect diet quality and intake of nutrients relevant for CVD. Nevertheless, we observed a significant difference in alcohol intake between patients with severe periodontitis and those with gingivitis. Alcohol consumption was the only parameter presenting a significant positive correlation with PD severity and has previously been shown to negatively affect PD[37] and to increase the risk for PAD[17,38].

Number and distribution of natural teeth have also been shown to influence DI, food choice and amount of nutrients consumed, probably due to the reduced ability to chew[22,25], but we did not find any correlation between the number of teeth and PD severity with the nutritional parameters investigated. These contradictory findings might be explained by the fact that the median

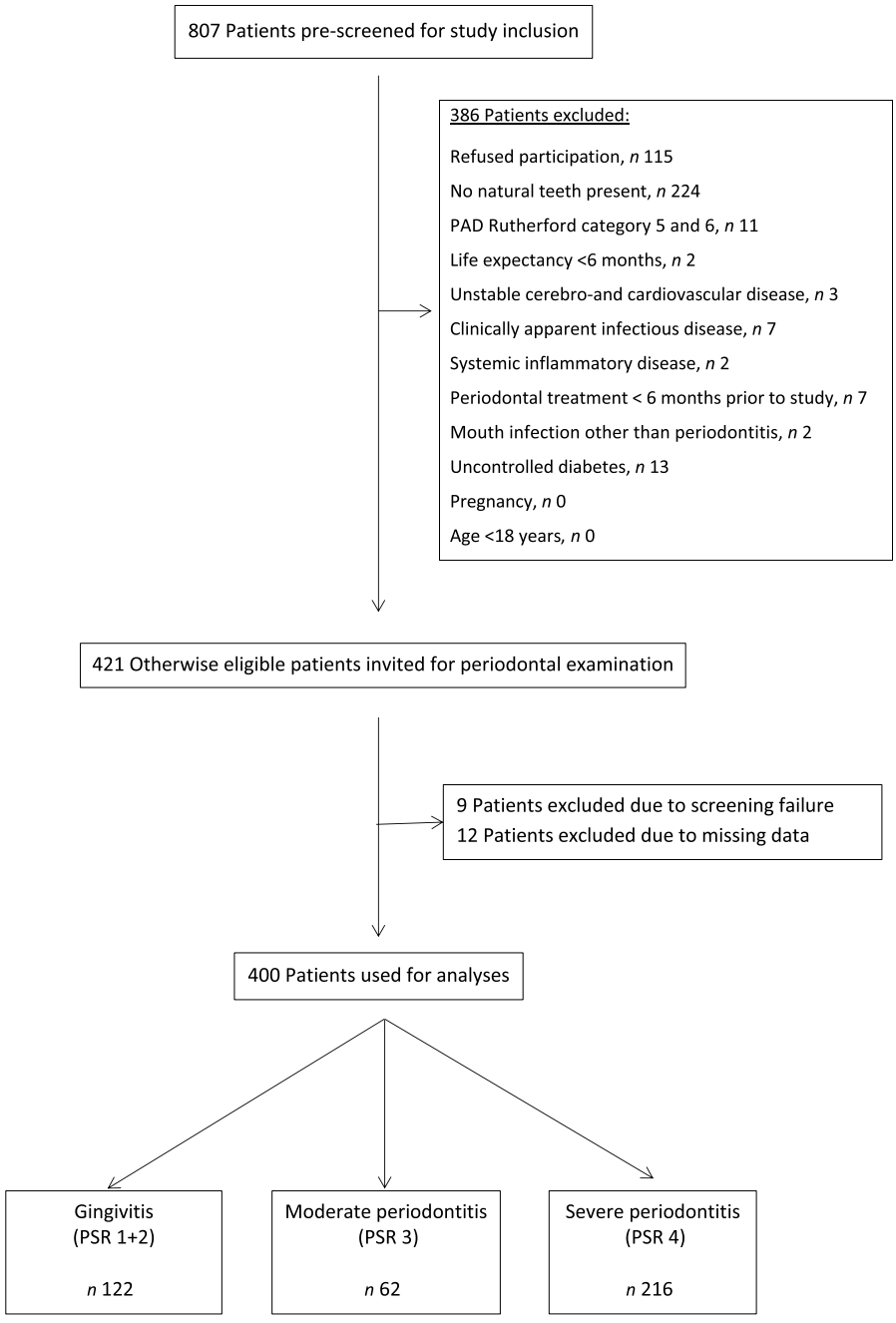

Fig. 1. Flow diagram of the screening process and enrolment of patients. PAD, peripheral arterial disease; PSR, Periodontal Screening and Recording Index.

age of our participants was lower than in Sheiham *et al.*[25], and the median number of eighteen natural teeth in our patients was still fairly high. Marcenes *et al.*[25] defined acceptable oral health by the presence of >20 natural teeth and stated that subjects with >21 teeth consumed more essential nutrients.

Impact of periodontal disease severity and number of teeth on macronutrient composition

We hypothesised that in PAD patients, concomitant presence of PD alters the composition of the patients' diet. Contrary to expectations, we observed no difference in macronutrient composition among the three PD stages. However, CHO and fat intake of our patients differed from current reference values. D-A-CH reference values for daily intake of CHO, fat and protein are 50–60 %, 25–30 % and 10–15 % of TEI, respectively[30].

Protein intake was adequate in our patients, but CHO intake was far too low in all PD groups. In contrast, 80 % of our patients exceeded the recommended fat intake of 30 % of TEI. This is in agreement with Brostow *et al.*[12], who showed in sixty-four PAD patients that the percentage of energy consumed as fat also exceeded the recommendations.

Our results stand in contrast to Gardner *et al.*[8], who examined the DI of forty-six subjects with PAD and reported a mean

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Table 4. Nutrient intake in peripheral arterial disease patients compared with guidelines for CVD prevention* (Medians and 25th–75th percentiles; percentages and numbers of patients meeting recommendations)

Nutrient	Nutritional guidelines for CVD prevention[31–34]	Dietary intake		Cohort meeting recommendations	
		Median	25th–75th percentiles	%	*n*/*n*†
Dietary *n*-3 PUFA (g)	1 g /d	1·2	0·9–1·7	66	269/142
SFA (% of TEI)	< 10 %	17	13–21	10	39/372
Na (mg)	< 2300 mg	2610	1883–3568	39	160/251
Total dietary fibre (g)	30–45 g/d	16	12–21	5	19/362
Sugar intake (% of TEI)	< 10 %	15	10–22	26	108/303
Alcohol (g)	Male: maximum 20 g/d	0·7	0–20	76	227/70
	Female: maximum 10 g/d	0	0–6	78	90/25

TEI, total daily energy intake; ESC, European Society of Cardiology; AHA, American Heart Association; ACC, American College of Cardiology.
* The Table summarises the subjects' nutritional intake, represented as proportions (%) relative to recommendations for nutrient intake. The values used for comparison were obtained from guidelines for CVD prevention by the ESC[31] and for Na intake by the AHA/ACC[32]. Recommendations for daily dietary *n*-3 PUFA (1 g/d) from the AHA[33,34] were used, as the ESC guidelines do not include specific recommendations for dietary *n*-3 PUFA intake. Dietary *n*-3 PUFA refers to total dietary *n*-3 PUFA (i.e. α-linolenic acid, EPA, docosapentaenoic acid and DHA).
† Number of patients meeting the recommendations/number of patients not meeting recommendations.

macronutrient composition very close to recommendations (17 % protein, 51 % CHO and 30 % fat). Our findings also disagree with Hamasaki *et al.*[39], demonstrating a low intake of dietary fat in patients with advanced PD (23·2 ± 7·1 %). In this specific study the 'weighting capacity technique' was used to determine DI (with this method, food intake was distributed in proportion to family size by dividing by the number of people in the family)[39]. The greater sample size of our investigation and the different methods of dietary assessment might explain the contradictory results. Our representative cross-sectional results might reflect the general DI of the Austrian population[40], with a lower than recommended intake of CHO, but increased intake of dietary fat and adequate intake of protein.

Dietary intake relative to European Society of Cardiology and American Heart Association/American College of Cardiology recommendations

Strategies for secondary prevention of CVD include the modification of dietary intake and habits. Accordingly, lifestyle and nutritional guidelines for the management of CVD have been developed by various institutions including the ESC[31], AHA and ACC[32–34]. Specific dietary parameters relevant for CVD, and therefore for patients affected with PAD, include *n*-3 fatty acids, SFA, Na, dietary fibre, sugar intake and alcohol consumption[31–34]. The results of the present study demonstrate that the consumption of these nutrients diverged greatly from guidelines.

Several classes of fatty acids have been shown to influence cardiovascular risk factors and outcomes[11]. The prospective Prevención con Dieta Mediterránea (PREDIMED) trial showed that the intake of *n*-3 PUFA may be associated with a reduced risk for developing PAD[41]. Generally, PAD patients appear to have an inadequate intake of vegetable lipids and hence essential fatty acids[9]. *n*-3 Fatty acids have also been suggested to play a role in PD. Using National Health and Nutrition Examination Survey (NHANES) III data, Naqvi *et al.*[42] found an inverse association of *n*-3 fatty acid intake with lower prevalence of periodontitis. In our large cohort of PAD patients suffering from concomitant PD, 66 % reached the AHA/ACC recommendation of at least 1 g/d of *n*-3 PUFA. A similar result was obtained in a recent prospective study by Nosova *et al.*[15], who showed that 59 % of their veteran subjects suffering from PAD reached this recommended cut-off. Another previous study by Lane *et al.*[10] demonstrated the reduced consumption of PUFA among PAD patients and suggested a protective effect of *n*-3 fatty acids against CAD and PAD.

A low intake of SFA, another class of fatty acids, is also of major importance in CVD prevention. Dietary SFA have been associated with an increased risk for PAD[11]. Gardner *et al.*[8] demonstrated that 80 % of their PAD patients exceeded the recommended DI of SFA. These results are confirmed by our study, where more than 90 % exceeded the recommended upper value.

Adequate fibre intake has previously been shown to have an inverse association with PAD[18]. Our data confirm the findings of Nosova *et al.*[15], who showed a low intake of fibre and high intake of Na among their PAD patients. In our cohort poor nutrition was reflected in high intake of Na and sugar, whereas more than 90 % of our subjects did not reach the recommended intake of dietary fibre. This is in accordance with Gardner *et al.*[8], who showed that only 26 % of their PAD patients reached the recommended intake for dietary fibre. It reflects a low intake of high-fibre foods, such as whole grain products, fruits and vegetables, all of which are difficult to chew. Since our patients were also suffering from PD, this might have further influenced fibre intake. Periodontal inflammation and loose teeth make it difficult to eat high-fibre foods. Staudte *et al.*[24] also demonstrated a lower intake of fibre in patients with periodontitis than in healthy controls.

The findings of our study highlight important implications for secondary prevention of CVD and public health, although we could not demonstrate that PD influences DI in PAD patients. Nevertheless, these patients should be encouraged to follow the ESC, AHA/ACC and D-A-CH dietary guidelines. Dietary modification and enhancement of nutritional strategies (including management of alcohol intake) should be considered as key components in secondary prevention to reduce CV risk in this population.

Limitations

There are limitations to this cross-sectional, observational study. The patient population is not representative of a general population since predominately Caucasian PAD patients were

studied. Furthermore, we did not include PAD patients with Fontaine Stage IV (defined by skin necrosis or ulcers), and our patients were not compared with a healthy control group. Another limitation is that DI was recorded via 1-d 24h-recall and a FFQ. A 7-d dietary recall would have been of advantage to evaluate the patients' dietary intake and habits in more detail, also capturing foods that are not eaten daily (e.g. fish), but was not feasible in our patient population due to age, cognitive impairment and other co-morbidities. Nevertheless, the methods we used are widely accepted tools for dietary assessment and can be a reliable indicator of trends in dietary habits within a population. In addition, to avoid potential errors in DI reporting, a trained dietitian was always available to help patients with the questionnaires.

Conclusion

In our sample of patients with PAD and concomitant PD, we did not observe differences in DI of nutrients known to be relevant for CVD and diet quality in relation to the severity of the PD, though macronutrient intake did differ from D-A-CH recommendations. Our most relevant finding is that our patients showed poor nutrition with regard to DI of Na, fibre, sugar, *n*-3 fatty acids and SFA, which differed greatly from current recommendations of the ESC, AHA and ACC. Future studies should focus on nutritional modification and intervention in order to study the effect on the progression of PD and CV risk in PAD patients.

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