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ORIGINAL CONTRIBUTION

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Relationship between dietary intake, antioxidant status and smoking habits in female Austrian smokers

Summary *Background* Previous studies have shown that cigarette smoke contains many oxidants and free radicals, which can increase lipid peroxidation. *Aim of the study* The association between smoking, food pattern, especially vitamin intake and plasma concentrations of important antioxidants, as well as lipid peroxidation products was assessed in this cross-sectional study. *Subjects and methods* Sixty Austrian women aged 18–40 y were enrolled in the study. Twenty-nine women were allocated to the smoking group; thirty-one women served as nonsmoking controls. Plasma concentrations of α- and γ-tocopherol, α- and β-carotene, lycopene, cryptoxanthin, retinol, ascorbate and malondialdehyde were determined by HPLC; dietary intake and food pattern had been assessed by four 24-h dietary intake recalls and one food frequency questionnaire. *Results* Generally, food intake patterns were not different between smoking and nonsmoking women. But, a significantly higher intake of alcohol was observed in the smoking group ($P < 0.05$). Plasma ascorbic acid concentration of the smoking group did not differ from the nonsmoking women. Despite the increased utilization because of the oxidative stress in smokers, this result might be explained by the high dietary intake of vitamin C in our smoking group. Significantly lower plasma concentrations of α-, β-carotene and lycopene have been partly ascribed to the enhanced metabolic turnover resulting from smoking-induced oxidative stress. Our results confirm that smoking had no effects on plasma tocopherol and plasma retinol concentrations. *Conclusions* The poor supply with the carotenoids α-, β-carotene and lycopene may result from the increased metabolism of antioxidants caused by oxidative stress and may be responsible for significantly higher levels of lipid peroxidation products in smokers compared to nonsmokers ($P < 0.05$).

Key words Smoking – Antioxidants – Dietary intake – Lipid peroxidation

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Introduction

Tobacco smoking correlates directly or indirectly with many severe diseases. Smoking favors cardiovascular risk and chronic pulmonary diseases as well as increases the chances of cancer. Altered blood coagulation, damage to the arterial wall and changes in blood lipid and lipoprotein concentrations are some possible explanations for the increased risk of coronary heart disease of cigarette smokers. The increased risk for cancer may be a result of lipid peroxidation products causing oxidative damage to DNA. Inflammatory reactions in the lung may result in pulmonary disease [1].

In industrialized countries, the consumption of tobacco is responsible for about two million deaths per year. The cause of the high mortality is the higher load of free radicals and other reactive species in the smoke of cigarettes. With each inhalation smokers take up 10^{14} free radicals. Consequently, the oxidant/antioxidant balance of smokers becomes perturbed and leads to oxidative stress. Smokers therefore have an additional re-

EJN 329

quirement of protective antioxidants. Supplementation with antioxidative vitamins must be viewed carefully because of the harmful effects observed in the ABTC study [2]. Dietary habits and lifestyle put smokers on a higher risk of disease as well. It is likely that the risk of disease is elevated in smokers because of complex interactions among the above mentioned factors [3].

The aim of this study was to investigate the association between smoking, food pattern, vitamin intake, and plasma concentrations of important antioxidants in female Austrian smokers compared to nonsmokers.

Subjects and methods

Subjects

Sixty women aged 18 to 40 years (26.3 ± 4.1 y) were enrolled in this cross-sectional study. Twenty-nine women were allocated to the group of the smokers; 31 women served as nonsmoking controls. The two groups had equivalent levels of education (smokers: 51.6 % students, 37.9 % employees and 10.3 % workers; nonsmokers: 58 % students, 32.3 % employees and 9.7 % workers). Smoking women must have smoked more than 5 cigarettes per day for a minimum of three years. Of the smoking women, 38 % were considered to be light smokers (< 10 cigarettes/d), 31 % as medium (10–20 cigarettes/d) and 31 % as strong smokers (> 20 cigarettes/d). Inclusion criteria of the nonsmoking group were never smoking or nonsmoking for a minimum of three years. More inclusion criteria for the smoking and nonsmoking group were an age between 18 and 40 years and the women had to be metabolic healthy. Women with metabolic disorders, pregnant and lactating women as well as women taking drugs, except oral contraceptives, were excluded.

Methods

The evaluation of the nutrient intake was made by four 24-h dietary intake recalls by each study subject. 24-h dietary intake recalls included one weekend day and the four days were pooled for a mean intake per day. Based on the 24-h dietary intake recall, data for total energy and nutrient intakes were calculated using the EWP 3.2 (ernährungswissenschaftliches Programm of Dato-Denkwerkzeuge) based on the national German/Austrian food composition data base BLS 2.1 [4]. Nutrition habits were investigated by a food frequency questionnaire. Food frequency questionnaire contained qualitative and quantitative questions (How often do you…?) about the average consumption of special foods and about the use of vitamin supplements.

Fasting blood samples had been taken at time of recruitment. Heparinized blood samples were centrifuged at 3000 rpm for 10 min. Plasma was separated and stored at –20 °C. Plasma concentrations of α- and γ-tocopherol, α- and β-carotene, lycopene and retinol were determined by HPLC according to the method of Jakob and Elmadfa [5]. Ascorbate was detected photometrically by a method of Denson and Bowers [6]. Superoxide dismutase and glutathione peroxidase were determined photometrically according to the method of Marklund and Marklund [7] and Beutler et al. [8]. Selenium, copper and zinc were detected by AAS by the method of Speitling et al. [9]. Malondialdehyde (MDA) was determined by HPLC by the method of Wong et al. [10]. Total antioxidative capacity was measured by the photometric method according to Rice-Evans and Miller [11].

Statistical analysis

All values were expressed as mean ± SD. SPSS for windows was used for all statistical procedures. The Mann-Whitney U test was used to compare the two groups.

Results

In our study no significant differences in the consumption of fruit and vegetables were noticed between smokers and nonsmokers. The intake of vitamin C was well above the recommendation of 100 mg vitamin C/d of the D-A-CH [12] in both study groups. Also the postulated additional need of vitamin C in smokers (150 mg/d) was reached by 26 % of the study population. The intake of vitamin A and β-carotene was also in the normal range and showed no differences between smokers and nonsmokers. Sixty-nine percent of the smoking study population did not reach the recommended vitamin E intake (recommendation of the D-A-CH for women: 12 mg/d) [12]. Considering the additional requirement of vitamin E in smokers [13], the intake may be insufficient (Table 1). No significant differences were found between the two study groups in macronutrient intake. Energy intake did not differ between groups, but there was a significantly higher consumption of alcohol among smoking than among nonsmoking women (Fig. 1). Smokers and nonsmokers differed in the way of meeting their energy requirement. The evaluation of the food frequency confirmed the results of the 24-h dietary intake recalls.

Because of the high intake of vitamin C, plasma ascorbic acid concentrations were in the normal range in smokers (smokers: 79.5 ± 17.0 µmol/l, nonsmokers: 73.8 ± 22.7 µmol/l). All smoking women achieved plasma levels above 50 µmol/l, a concentration which guarantees a sufficient supply. Plasma concentrations of vitamin A and tocopherol equivalents adjusted to cho-

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Table 1 Subject characteristics and dietary intake[1]

	Smokers (n=29)	Nonsmokers (n=31)
Age, y	26.3 ± 4.1	25.5 ± 5.3
BMI[2], kg/m^2	22.1 ± 2.8	22.1 ± 2.5
Energy intake, kJ/d	8200 ± 1192	8326 ± 2054
Dietary fat, %energy	37.9 ± 4.9	36.1 ± 7.0
SFA, g/d	37.0 ± 8.0	34.0 ± 15.0
MUFA, g/d	29.0 ± 8.0	29.0 ± 13.0
PUFA, g/d	13.0 ± 5.0	14.0 ± 5.0
Cholesterol, mg/d	295 ± 95	317 ± 182
P/S ratio	0.36	0.50
Protein, %energy	14.4	15
Carbohydrates, %energy	44	46
Fibre, g/d	18.0 ± 5.0	20.0 ± 7.0
Alcohol, g/d	10.7 ± 9.8	6.1 ± 6.7*
Vitamin C, mg/d	138.8 ± 0.3	121.2 ± 48.1
Vitamin E, mg/d	10.6 ± 3.1	11.9 ± 5.0
Vitamin A, mg/d	1.1 ± 0.4	0.9 ± 0.4
β-Carotene, mg/d	2.9 ± 1.6	2.9 ± 1.7

[1] Values are means±SD, * $P < 0.05$
[2] *BMI* body mass index, *SFA* saturated fatty acid, *MUFA* monounsaturated fatty acid, *PUFA* polyunsaturated fatty acid, *P/S ratio* polyunsaturated/saturated ratio

Table 2 Antioxidative status in plasma and erythrocytes of smokers and nonsmokers[1]

	Smokers (n=29)	Nonsmokers (n=31)
Vitamin C, µmol/L	79.43±16.30	73.81±22.23
Vitamin E, µmol/L	19.55±6.10	20.66±5.54
Vitamin E/Chol, µmol/mmol	4.79±1.57	4.55±1.08
Vitamin A, µmol/L	2.01±0.61	2.07±0.59
β-Carotene, µmol/L	0.38±0.25	0.51±0.25*
α-Carotene, µmol/L	0.13±0.07	0.19±0.09*
Lycopene, µmol/L	0.33±0.24	0.47±0.37*
Cryptoxanthin, µmol/L	0.42±0.30	0.45±0.37
Erythrocyte SOD, U/g Hb	1992±301	2229±446*
Erythrocyte GSH-Px, U/g Hb	30±19	32±8
MDA-TBA-complex, µmol/L	1.52±0.47	1.31±0.55*
TAC, nmol TEAC	1.17±0.41	1.23±0.27

[1] Values are means±SD, * $P < 0.05$
SOD superoxidedismutase, *Hb* hemoglobin, *GSH-Px* glutathione peroxidase, *MDA-TBA-complex* malondialdehyde-thiobarbituric acid-complex, *TAC* total antioxidative capacity, *TEAC* trolox equivalent antioxidative capacity

lesterol were also satisfactory and did not differ between smokers and nonsmokers (Table 2). Despite comparable β-carotene intake of smokers and nonsmokers and no significant differences in the consumption of fruit and vegetables, smokers had significantly lower plasma carotenoid concentrations, except for cryptoxanthin, than nonsmokers ($P < 0.05$). Using a concentration of 0.4 µmol/l of plasma β-carotene as a cut-off point, a concentration thought to be preventive for disease risk [14], we found that 62 % of the smoking women and 26 % of the nonsmoking women were below this concentration.

The activity of erythrocyte superoxid dismutase (SOD) in the smoking group was significantly lower than in nonsmoking women (smokers: 1992 ± 301 U/g hemoglobin (Hb), nonsmokers: 2229 ± 446 U/g Hb; $P < 0.05$). The activity of erythrocyte glutathione peroxidase (GSH-Px) showed no different values in smokers compared to nonsmokers (smokers: 30 ± 19 U/g Hb, nonsmokers: 32 ± 8 U/g Hb). Concentrations of selenium, zinc and copper, important integral parts of SOD and GSH-Px lay in the normal range in smokers as well as in nonsmokers.

Plasma concentrations of the malondialdehyde-thiobarbituric acid-complex (MDA-TBA-complex), one of the markers of lipid peroxidation, were significantly higher in smoking than in nonsmoking women, as the consumption of tobacco leads to increased lipid peroxidation ($P < 0.05$). Total antioxidative capacity in plasma did not differ between smokers and nonsmokers (Table 2).

Discussion

The main purpose of this study was to assess the relationship between smoking, food pattern, vitamin intake, and plasma concentrations of important antioxidants. According to Perkins et al. [15], smoking may either affect food choice directly, or indirectly by altering physiological processes related to smell, taste or appetite. Our data do not confirm results obtained by other authors suggesting that heavy smokers have poorer dietary patterns than nonsmokers. In comparison with nonsmokers, smokers consume fried foods, processed meats, whole milk and sugar more frequently, but fruit, fruit juice, vegetables, salad, skimmed milk and wholegrain

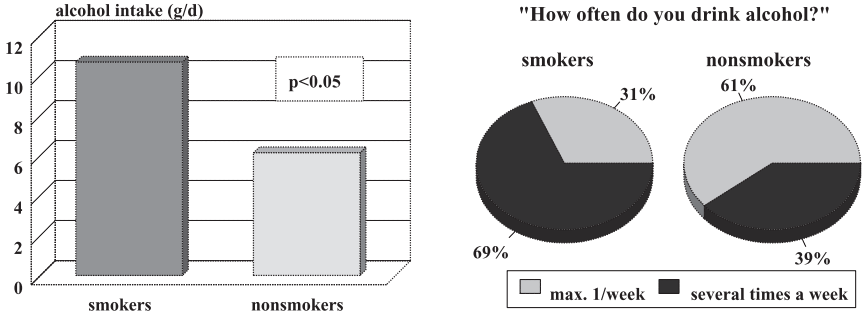

Fig. 1 Alcohol intake in smokers compared to nonsmokers.

cereals less frequently [16–18]. Contrary to these study reports, we noticed no significant differences in food intake between smokers and nonsmokers. Some authors also report that meal patterns differ by smoking status, with smokers eating breakfast less frequently than nonsmokers [19]. In our study, smoking women did not eat breakfast regularly as well. Only 48 % of the smokers consumed breakfast every day, but 71 % of the nonsmokers did so ($P < 0.05$). Confirming other results [20–21], we found a significantly higher intake of alcohol in the smoking group compared with nonsmokers ($P < 0.05$). Many authors found that alcoholism is about 10 times higher among smokers than among nonsmokers [22]. The main differences in macronutrient intake between smokers and nonsmokers were observed for alcohol, polyunsaturated and saturated fatty acids and for dietary fiber. Most studies found that smokers consume more saturated fat, sugar and alcohol and less protein, fiber, polyunsaturated fats and antioxidant vitamins [1]. In our study smokers took more energy from alcohol and fat at the expense of carbohydrates. With regard to the type of fat consumed, our smokers also showed a slightly higher intake of saturated fat and a smaller intake of polyunsaturated fat, but these results were only tendentious and not significantly different (Table 1). The intake of fiber did not differ between smokers and nonsmokers and 90 % of the investigated women could not reach the recommendation of the D-A-CH (30 g fiber/d) [12]. Poorer dietary practice could be a reflection of less healthy lifestyles and a lack of dietary knowledge. Many smokers also know about the higher risk through tobacco smoking and may pay less attention to the risk associated with the consumption of an "unhealthy" diet. Lifestyle of our smoking study group was "healthy" and did not influence habits concerning food intake. This result may be dependent on gender. We investigated women, who are generally more interested in food pattern. Results of different smokers (light, moderate, strong) are not uniform. Some studies show that moderate smokers are more similar to nonsmokers in antioxidant as well as total dietary intake [18, 21]; others found significant differences between moderate smokers and nonsmokers [23].

Larger differences in nutrient intakes by smoking status have been observed for micronutrients. Smokers appear to have lower circulating levels of antioxidants. This suggests that smoking affects the metabolic demands for these nutrients. The most likely explanation is the increased load of free radical. Thus, many authors observed reduced ascorbic acid levels in plasma and leukocytes among smokers. It has been shown that if smokers and nonsmokers consume a similar amount of vitamin C, the serum level of ascorbic acid is lower in smokers than in nonsmokers [24]. Many observations suggest that smoking directly lowers plasma ascorbic acid concentrations [24–26]. Contrary to these studies, plasma ascorbic acid concentrations of our smoking group did not differ from the nonsmoking women. This may be explained by the very good dietary intake of vitamin C in our smoking group. Many smokers know about the additional need of vitamin C and eat vitamin C rich food or take vitamin supplements.

The effect of smoking on circulating concentrations of β-carotene generally agrees with the conclusion of other studies [18]; lowered plasma concentrations of β-carotene have been ascribed in part to enhanced metabolic turnover resulting from smoking-induced oxidative stress. In our study we also noticed decreased plasma concentrations of α-carotene and lycopene. We found no significant effect of smoking on plasma cryptoxanthin levels. Our results confirm previous reports of no effect of smoking on plasma tocopherol and retinal concentrations [18, 27–31]. Dietary supplementation with antioxidant vitamins is not without its critics. Two large intervention trials raise the possibility that these supplements (β-carotene, α-tocopherol, retinol) might have harmful as well as beneficial effects, depending on the doses given [2, 32–34].

As parameters of the endogenous protective system the activity of superoxide dismutase and glutathione peroxidase in erythrocytes was determined. In agreement with other authors, we found lowered SOD activities in smokers compared to nonsmokers [35–37]. The cause of the decreased activity of SOD may be excessive oxidative stress leading to the destruction of the enzymatic protective system or the increased load with cadmium which cigarettes contain in large amounts. Enzyme related trace elements as selenium, copper and zinc lay in the normal range in both study groups and were not responsible for the decreased activity of SOD. Therefore, we noticed that the capacity of the enzymatic protective system in smokers was overloaded.

Smoking cigarettes results in altered lipid metabolism and increased inflammation response. The high free radical load and the relatively low antioxidant status in many smokers may result in an imbalance between free radicals and antioxidant defenses that may increase the atherogenicity of LDL. As one marker of lipid peroxidation, thiobarbituric acid-reactive substances (TBA-MDA-complex) in plasma were measured and a significant higher plasma TBA-MDA-complex concentration was observed in the investigated smokers compared to nonsmokers ($P < 0.05$). This result can be explained by the higher generation of free radicals because of tobacco consumption and the significant higher intake of alcohol in the smoking group. The investigated smokers had a sufficient or even higher intake of antioxidants, leading to plasma vitamin concentrations in the normal range; nevertheless lipid peroxidation products occurred in significantly higher concentrations than in nonsmokers. Despite the good supply of antioxidative vitamins, smokers showed a lower total antiox-

idative capacity in plasma compared with nonsmokers. Rahman et al. [38] also observed lower levels of total antioxidative capacity and higher concentrations of lipid peroxidation products in plasma of smokers. Therefore, two recommendations can be made: stop smoking and increase the dietary intake of fruit and vegetables – important sources of carotenoids and other antioxidative vitamins.

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