Antioxidant status in a group of institutionalised elderly people with chronic obstructive pulmonary disease

Elena Rodríguez-Rodríguez[1,2]*, Rosa M. Ortega[2,3], Pedro Andrés[1,2], Aránzazu Aparicio[2,3], Liliana G. González-Rodríguez[2,4], Ana M. López-Sobaler[2,3], Beatriz Navia[2,3], José M. Perea[2,4] and Paula Rodríguez-Rodríguez[5]

[1]*Sección Departamental de Química Analítica, Facultad de Farmacia, Universidad Complutense de Madrid, 28040 Madrid, Spain*
[2]*UCM Research Group, VALORNUT-920030, Spain*
[3]*Departamento de Nutrición, Facultad de Farmacia, Universidad Complutense de Madrid, 28040 Madrid, Spain*
[4]*Departamento de Nutrición Humana y Dietética, Facultad de Ciencias de la Salud, Universidad Alfonso X El Sabio, Villanueva de la Cañada, 28691 Madrid, Spain*
[5]*Servicio de Neumología, Hospital Fundación Jiménez Díaz, 28040 Madrid, Spain*

Abstract

Chronic obstructive pulmonary disease (COPD) is one of the most important and prevalent diseases suffered by the elderly. Evidence exists that its onset and severity might be conditioned by antioxidant status. The aim of the present study was to investigate the relationship between antioxidant status and COPD in institutionalised elderly people. In all, 183 elderly people aged >65 years (twenty-one had COPD and 160 healthy controls) were studied. The subjects' diets were investigated via the use of precise individual weighing for 7 d. Body weight, height, and biceps and triceps skinfold thickness were measured, and body fat (kg) and BMI (kg/m^2) were calculated. Serum retinol, α-tocopherol, β-carotene and vitamin C levels were determined. Subjects with COPD ate less fruits than healthy controls (117 (sd 52) *v.* 192 (sd 161) g/d), their coverage of the recommended intake of vitamin C was smaller (150 (sd 45) *v.* 191 (sd 88) %; note that both exceeded 100 %) and their diets had a lower antioxidant capacity (6558 (sd 2381) *v.* 9328 (sd 5367) mmol trolox equivalent/d). Those with COPD had lower serum vitamin C and α-tocopherol concentrations than healthy controls (32·4 (sd 15·3) *v.* 41·5 (sd 14·8) µmol/l and 12·1 (sd 3·2) *v.* 13·9 (sd 2·8) µmol/l, respectively). In addition, subjects with α-tocopherol <14·1 µmol/l (50th percentile) were at 6·43 times greater risk of having COPD than those subjects with ≥14·1 µmol/l (OR 6·43; 95 % CI 1·17, 35·24; *P* < 0·05), taking sex, age, use of tobacco, body fat and vitamin E intake as covariables. Subjects with COPD had diets of poorer antioxidant quality, especially with respect to vitamins C and E, compared with healthy controls.

Key words: Chronic obstructive pulmonary disease: Antioxidants: Ageing

Chronic obstructive pulmonary disease (COPD) is a chronic, inflammatory ailment of the lungs, in which there is a progressive and irreversible loss of airflow[1,2]. At present, there are 210 million people with COPD, and it has been estimated to be the third leading cause of death worldwide by 2030[3]. Many authors suggest COPD to be very prevalent among the elderly[1,4]. In Spain, there are some 21·4 million people between 40 and 80 years of age, among whom 2 185 764 are affected by the disease[5]. Although these figures come from one of the largest studies undertaken in this field, its age limit of 80 years should not go unnoticed.

Among the factors that might contribute towards the development of COPD are genetic background (especially those types leading to a deficiency in α-1-antitrypsin), exposure to oxidising agents (e.g. those in atmospheric pollution and tobacco smoke) and reduced antioxidant capacity[6–8]. Certainly, people with COPD experience increased oxidative stress, which reduces airflow. This could lead to a greater demand for antioxidant nutrients such as vitamins C and E and carotenoids, which might leave COPD patients more vulnerable to deficiencies[9–11]. For example, in a group of 218 patients diagnosed with asthma and/or COPD, serum β-cryptoxanthin, lutein/zeaxanthin and retinol concentrations were found to be significantly and positively associated with the forced expiratory volume in 1 s (FEV$_1$) – the most important measure of airflow.

Abbreviations: BF, body fat; COPD, chronic obstructive pulmonary disease; ORAC, oxygen radical absorbance capacity; RI, recommended intakes; TAC, total antioxidant capacity.

* Corresponding author: E. Rodríguez-Rodríguez, fax +34 91 394 17 54, email elerodri@ucm.es; grupo920030@gmail.com

The antioxidant status of patients with COPD may therefore be important in terms of the progression of the disease[10].

Although COPD is one of the most common diseases affecting the elderly, a few studies on antioxidant status in this population have been performed[10,12]. The aim of the present study was, therefore, to determine the antioxidant status of a group of institutionalised elderly people.

Methods

Study subjects

This case–control study included elderly people over 65 years of age, residing in four homes for the elderly in the Region of Madrid, Spain. The health personnel at the different homes selected the subjects after checking their medical records, their cognitive functional and affective capacity scores, and prescribed medication. This study was part of a larger study[13], aimed at selecting groups of subjects with similar nutritional, cognitive and affective characteristics in order to include them in a nutritional intervention trial. The starting number for each group was about fifty subjects. Centres with less than sixty possible participants were excluded.

All subjects were informed of the characteristics of the study, and their signed consent to be included was requested. Those who did not provide signed consent were excluded, as well as those with any condition that might affect the digestion, absorption or utilisation of nutrients (neoplasms, cirrhosis, abnormal liver function, poor intestinal absorption, etc.) and those showing signs of manifest cognitive decline (i.e. with a Lobo & Ezquerra test score of <24[14]). Individuals for whom alcohol made up >10% of their daily energy intake were also excluded. A total of 183 elderly people (sixty-three men and 120 women) completed the study.

The present study followed the principles of the Declaration of Helsinki and was approved by the Human Research Review Committee of the Pharmacy Faculty, Complutense University of Madrid. Signed informed consent was obtained from all participants.

Dietary study

The subjects' consumption of food and intake of nutrients were determined by the 'precise individual weighing' method over a period of 7 consecutive days (including a weekend to take into account any change in the normal daily diet)[15]. The individual portions consumed at each of the main meals (breakfast, lunch, early evening snack, evening meal) were weighed, as were the amounts left on the plate after eating. The subjects could choose their meals as the homes offered a range of menus. Thus, the foods recorded over this 7-d period likely reflect the normal food choices made by the subjects. All foods consumed outside established mealtimes, as well as outside of the homes, were reported by the subjects themselves.

The dietary data collected were treated using DIAL software[16] in order to determine the amounts of the different food groups consumed, the nutrient and energy intakes, the total antioxidant capacity (TAC) of the diet and the adequacy of the intake of different dietary components, especially vitamins A, C and E, with respect to recommended intakes (RI).

TAC can be determined in different ways including the ferric-reducing antioxidant power (FRAP) method, which determines the capacity to reduce a ferric complex[17], the radical trapping antioxidant parameter (TRAP) method, based on the thermal decomposition of azo-hydrosoluble compounds that produce peroxyl radicals[18], the trolox equivalent antioxidant capacity (TEAC) method, which measures the capacity to reduce the ABTS+ (2,2'-azinobis(3-ethylbenzothiazoline-6-suslfonic acid) radical under lipophilic and hydrophilic conditions[19], and the oxygen radical absorbance capacity (ORAC) method, which measures the oxidative degradation of a fluorescent molecule subjected to a flow of peroxyl radicals[20]. In the present study, the TAC of the diets was determined using values recorded in the *Spanish Food Composition Tables*[21], drawn up by collecting values published by different authors. The database used contained 483 measurements made by the FRAP method (mmol/100 g), 140 by the TRAP method (trolox equivalent (TE) mmol/100 g), 158 by the TEAC method (TE mmol/100 g) and 225 by the ORAC method (TE mmol/100 g).

Presence of chronic obstructive pulmonary disease

The medical histories of the subjects were examined in search of information on/diagnoses of COPD. When this information was insufficient to identify a subject as having this problem, the doctor who attended to the subjects at each home was asked for assistance. COPD was deemed present when, following the inhalation of bronchodilators, the FEV_1/forced vital capacity multiplied by 100 was <70%[22,23].

Of the 183 subjects who took part in the present study, twenty-one were deemed to have COPD.

Other covariables

Information was also collected on risk factors for COPD, such as age (years), smoking habits (non-smoker, ex-smoker, smoker), alcohol consumption and use of multivitamin supplements.

Anthropometric study

Body weight was determined using a Seca Alpha digital balance (range 0·1–150 kg; accuracy 100 g; Seca Alpha, GmbH & Co.), with the subjects wearing only underwear and standing on the centre of the horizontal plate. Body weight of subjects who found it difficult to stand was determined using an Alpha 6868 medical balance equipped with a seat (range 1–360 kg).

Height was determined using a Harpenden digital stadiometer (range 70–205 cm; accuracy 1 mm; Pfifter). Subjects were measured while standing, holding the back as straight as possible and with the arms parallel to the body, heals together and the head in the Frankfort horizontal plane. This measurement was taken only for those subjects who could maintain a standing position. For subjects with problems of balance or conditions affecting the vertebral column (kyphosis, sclerosis),

height was estimated from the knee–heel distance using the formula of Chumlea[24]:

$$Men: 64\cdot2 - (0\cdot04\,age\,(years))$$
$$+ (2\cdot02\,distance\,knee - heel\,(cm)),$$
$$Women: 89\cdot4 - (0\cdot24\,age\,(years))$$
$$+ (1\cdot83\,distance\,knee - heel\,(cm)).$$

BMI was then calculated from the height and body weight measurements[25]:

$$BMI\,(kg\,/\,m^2) = \frac{weight\,(kg)}{height^2\,(m^2)}.$$

Skinfolds (biceps and triceps) were measured using a Holtain skinfold caliper (range 0–40 mm; accuracy 0·1 mm Holtain Ltd), applying a constant pressure of 10 g/mm^2 over the contact surface. Measurements were taken on the non-dominant side of the body. All readings were taken after some 4 s with the caliper in place to reduce the variation associated with skin compressibility[26]. The biceps skinfold thickness was measured over the cubital fossa with the arm relaxed, at the mid-point between the acromion and the olecranon. The triceps skinfold thickness was measured with the arm relaxed, also at the mid-point between the acromion and the olecranon.

The percentage of body fat (BF%) was calculated using the equation of Siri[27]:

$$BF\,\% = \left(\frac{495}{D}\right) - 450,$$

where D is the density term calculated using the equation of Durnin & Womersley[28]:

$$D = c$$
$$- (m \times \log\,(sum\,of\,the\,biceps\,and\,triceps\,skinfold\,thicknesses)),$$

in which $c = 1\cdot1185$ for men and $1\cdot1226$ for women, and $m = 0\cdot0683$ for men and $0\cdot0710$ for women.

Once BF% was known, BF mass (kg) was determined using the equation of Méndez & Lukaski[29]:

$$BF\,(kg) = \frac{(BF\,\% \times weight\,(kg))}{100}.$$

Blood sample collection

Blood samples were collected by venepuncture between 08.00 and 09.00 hours in the morning after 12 h of fasting. Adherence to the fasting period was checked by nurses before blood was collected. All sample collections were performed at the respective centres.

Biochemical study

Retinol and α-tocopherol levels were determined together by reversed-phase HPLC following the method of Driskel *et al.*[30]. The mobile phase was a 95:5 mixture of methanol–water, the flow rate was 1·0 ml/min and the column was a Supelcosil LC-18 model (4·2 × 150 mm; 5 µm particle size; Sigma-Aldrich Co. LLC). All determinations were made using a Varian 5000 apparatus equipped with a Varian variable wavelength UV detector (Varian BV). Vitamin A detection was performed at 325 nm and vitamin E detection at 294 mm 3 min later. Retinyl acetate and tocopheryl acetate were used as internal standards (CV = 2·4 % for retinol and 2·8 % for tocopherol).

β-Carotene was also determined by reversed-phase HPLC. The target molecule was extracted from 100 µl serum samples via the addition of 120 µl *n*-hexane plus 100 µl of echinenone as an internal standard. The mixture was then centrifuged (500 ***g***, 5 min) and 100 µl of the supernatant was extracted. This was then evaporated in a N atmosphere at 50°C. The lipid residue was then reconstituted in 100 µl of a mobile phase (70 % acetonitrile, 20 % dichloromethane, 10 % methanol). Separation was performed using a Supelcosil LC-18 column (4·6 × 250 nm; 5 µm particle size) at a flow rate of 1·7 ml/min. Detection was performed using a UV detector at 436 nm (CV = 3·3 %)[31].

Vitamin C was determined via ascorbic acid enzymatic oxidation[32] and the formation of quinoxaline to produce a fluorescent compound that was detectable spectrofluorimetrically[33]. The method followed was that of Vuilleumier & Keck[34] without automation (CV = 2·9 %).

Statistical analysis

Values were recorded as means and standard deviations or percentages. The dietary data were adjusted by energy intake using the residuals method. Differences between means were examined using the Student's *t* test or using the Mann–Whitney test when the distribution of the results was not normal. Linear correlation coefficients were determined between some variables. Categorical variables were compared using the χ^2 test. Logistic regression analysis was used to examine the risk factors associated with COPD; these results are presented as OR and 95 % CI. Significance was set at $P < 0\cdot05$. All calculations were performed using RSIGMA BABEL software (Horus Hardware).

Results

In all, twenty-one (11·5 %) subjects had COPD. A high percentage of the population had an inadequate vitamin E and β-carotene status (Table 1). The TAC of the diet, as measured by the ORAC method, was positively and significantly associated with the consumption of fruits (r 0·7357; $P < 0\cdot001$).

Subjects with COPD were more commonly men and smokers, and their diets had a lower TAC compared with control subjects (Table 2). No significant differences were detected in terms of intake of the studied nutrients. However, the RI of vitamin C was better covered among the control subjects (Table 2). In addition, subjects with a vitamin intake <150 % of RI were three times more likely of having COPD than those with higher intakes (OR 2·94; 95 % CI 1·11, 7·75; $P < 0\cdot05$). The main source of vitamin C was fruits (r 0·8091; $P < 0\cdot001$), and consumption of fruits was higher among the control subjects (Table 2). There were no differences between the control and the COPD groups regarding vitamin A intake. However, those with a vitamin A intake <75 % of the RI were 2·69 times more likely of having COPD than those with higher intakes (OR 2·79; 95 % CI 1·01, 7·23; $P < 0\cdot05$).

Table 1. Characteristics of the study population
(Mean values and standard deviations)

	Mean	SD
n	183	
Age (years)	82·4	7·0
% COPD	12·7	
Sex (% men)	34·4	
BMI (kg/m²)	29·2	6·3
BF (kg)	21·3	7·5
Smoking habits (%)		
Never	83·3	
Ex-smoker	1·3	
Smoker	15·3	
Use of supplements (%)		
No	40·7	
Yes	59·3	
Dietary data		
Energy (kJ)	7539	1004
Energy (kcal)	1802	240
Underestimation (%)	− 0·91	16·14
Cereals (g/d)	163	43
Pulses (g/d)	15·9	11·8
Vegetables (g/d)	263	89
Fruits (g/d)	184	155
Dairy products (g/d)	406	118
Meat (g/d)	95·3	39·3
Fish (g/d)	41·5	21·1
Eggs (g/d)	18·6	12·1
Oils (g/d)	33·3	15·2
Vitamin C (mg/d)	112	48
Coverage of RI (%)	187	86
Vitamin E (mg/d)	12·2	5·7
Coverage of RI (%)	115	58
α-Tocopherol (mg/d)	9·90	5·89
Vitamin A (µg/d)	786	236
Coverage of RI (%)	103	36
Retinol (µg/d)	293	68
β-Carotene (µg/d)	2550	1173
ORAC (mmol TE/d)	9034	5199
FRAP (mmol Fe(II)/d)	4·01	1·75
TRAP (mmol TE/d)	4·13	1·65
TEAC (mmol TE/d)	3·12	1·31
Biochemical data		
Vitamin C (µmol/l)	40·3	14·8
% Deficiency (<22·7 µmol/l)	11·8	
Plasma β-carotene (µmol/l)	0·38	0·42
% Deficiency (<0·186 µmol/l)	40·8	
Retinol (µmol/l)	2·05	0·50
% Deficiency (<1·05 µmol/l)	4·48	
Vitamin E (α-tocopherol) (µmol/l)	13·7	2·9
% Deficiency (<11·6 µmol/l)	23·9	
% Low level (11·6–23·2 µmol/l)	76·1	
% Optimum level (>27·9 µmol/l)	0	

COPD, chronic obstructive pulmonary disease; BF, body fat; RI, recommended intake; ORAC, oxygen radical absorbance capacity; TE, trolox equivalent; FRAP, ferric-reducing antioxidant power; TRAP, trapping antioxidant parameter; TEAC, trolox equivalent antioxidant capacity.

Subjects with COPD had lower serum vitamin C and α-tocopherol levels than the control subjects, although no significant differences were seen between the two groups regarding the percentage of people with deficient levels (Table 2).

Logistic regression analysis showed that subjects with a serum vitamin C concentration <42 µmol/l (i.e. below 50th percentile) were 3·59 times more likely of having COPD than those with concentrations ≥42 µmol/l (OR 3·59; 95 % CI 1·13, 11·41; $P < 0.05$). Similarly, subjects with serum α-tocopherol <14·1 µmol/l (i.e. below 50th percentile) were 4·17 times more likely of having COPD than those with higher levels (OR 4·17; 95 % CI 1·11, 15·69; $P < 0.05$). When taking the covariables sex, age, use of tobacco, BF and vitamin C or E (as required) into account, this relationship was lost for vitamin C (OR 4·31; 95 % CI 0·97, 19·30; $P > 0.05$) but was significant for α-tocopherol (OR 6·43; 95 % CI 1·17, 35·24; $P < 0.05$).

Discussion

Oxidative stress appears to play an important role in the pathogenesis of COPD. An adequate intake of antioxidant nutrients, and an adequate status of these nutrients, might therefore be beneficial in the maintenance of pulmonary function in patients with this condition[35]. A study involving 14 120 adults over 17 years of age showed that higher serum concentrations of vitamins A, C, E and β-cryptoxanthine were independently associated with higher FEV_1 scores[9] – a finding in line with the results of the present study.

Although the contribution of vitamin C intake towards covering its RI was indeed high, over 100 %, it was even higher among the control subjects. These results suggest that a coverage >150 % may offer protection against the appearance of COPD. This agrees with that reported by other authors. For example, Ahmadi et al.[36] and Lin et al.[12] reported smaller intakes of vitamin C among patients with COPD compared with healthy controls, and the latter authors also reported greater consumption of fruits and vegetables among control subjects. In addition, in a study of 2917 Finnish men who were followed-up for 20 years, it was noticed that for every 100 g increase in fruit consumption, the risk of developing COPD fell by 24 %[37]. This finding also agrees with the present results: greater fruit consumption – the main source of vitamin C – was seen among the healthy subjects.

As seen at the dietary level, and in agreement with other authors[9,12], higher serum vitamin C concentrations seemed to offer protection against COPD. When use of tobacco was taken into account as a covariable, this effect was lost. Certainly, it has been reported that, in vitro, the plasma concentration of this vitamin falls significantly in the presence of tobacco smoke as a consequence of lipid peroxidation[38]. As the majority of our subjects with COPD were smokers (Table 2), and given that smoking is an important risk factor for low serum vitamin C concentrations, it would seem reasonable to recommend that the intake of this vitamin/the consumption of fruits be increased and that smoking be ceased.

In addition to having antioxidant activity, vitamin E exerts anti-inflammatory effects; maintaining an adequate vitamin E status might therefore be beneficial to patients with COPD[39]. In a recent study involving 38 507 women followed-up over 10 years, it was determined that the administration of 400 mg vitamin E reduced the risk of developing COPD by 10 %[40]. In the present study, subjects with COPD not only had less vitamin E intakes compared with healthy controls but also had poorer serum concentrations of the same. Further, serum values below the 50th percentile were associated with four times higher risk of having COPD than higher levels. This relationship held even

Table 2. Characteristics of the study population depending on chronic obstructive pulmonary disease (COPD) status†
(Mean values and standard deviations)

	COPD (n 21)		Normal (n 160)	
	Mean	SD	Mean	SD
Age (years)	82·2	6·2	82·3	7·1
Sex (% men)	57·1*		31·9*	
BMI (kg/m²)	26·5	5·7	29·5	6·3
BF (kg)	17·9*	7·3	21·7*	7·4
Smoking habits (%)				
Never	61·9		38·9	
Ex-smoker	0		1·2	
Smoker	33·3*		9·9*	
Consumption of supplements (%)				
No	38·1		29·0	
Yes	61·9		40·7	
Dietary data				
Energy (kJ)	7339	954	7565	1008
Energy (kcal)	1754	228	1808	241
Underestimation (%)	4·65	11·81	− 1·57	16·49
Cereals (g/d)	150	53	165	42
Pulses (g/d)	16·2	12·3	15·8	11·8
Vegetables (g/d)	280	65	261	92
Fruits (g/d)	117*	52	192*	161
Dairy products (g/d)	370	95	410	120
Meat (g/d)	87·7	39·1	96·2	39·3
Fish (g/d)	43·8	22·4	41·2	21·0
Eggs (g/d)	20·1	16·1	18·4	11·6
Oils (g/d)	30·8	20·0	33·6	14·6
Vitamin C (mg/d)	98·1	32·3	113·8	50·0
Coverage of RI (%)	150**	45	191**	88
Vitamin E (mg/d)	14·32	5·18	11·94	5·73
Coverage of RI (%)	120	52	115	59
α-Tocopherol (mg/d)	11·36	4·65	9·73	5·62
Vitamin A (µg/d)	875	389	776	210
Coverage of RI (%)	104	54	10334	
Retinol (µg/d)	305	52	291	70
β-Carotene (µg/d)	2812	1415	2518	1142
ORAC (mmol TE/d)	6558*	2381	9328*	5367
FRAP (mmol Fe(II)/d)	3·42	0·87	4·08	1·82
TRAP (mmol TE/d)	3·86	1·41	4·16	1·67
TEAC (mmol TE/d)	2·81	0·93	3·16	1·34
Biochemical data				
Vitamin C (µmol/l)	32·4*	15·3	41·5*	14·8
% Deficiency (<22·7 µmol/l)	22·2		10·5	
Plasma β-carotene (µmol/l)	0·31	0·20	0·39	0·44
% Deficiency (<0·186 µmol/l)	30·8		41·8	
Retinol (µmol/l)	21·3	0·49	2·04	0·51
% Deficiency (<1·05 µmol/l)	7·1		4·2	
Vitamin E (α-tocopherol) (µmol/l)	12·1*	3·2*	13·9*	2·8
% Deficiency (<11·6 µmol/l)	35·7		22·6	
% Low level (11·6–23·2 µmol/l)	64·3		77·4	
% Optimum level (>27·9 µmol/l)	0		0	

BF, body fat; RI, recommended intake; ORAC, oxygen radical absorbance capacity; TE, trolox equivalent; FRAP, ferric-reducing antioxidant power; TRAP, trapping antioxidant parameter; TEAC, trolox equivalent antioxidant capacity. Significant correlations:
* $P < 0.05$; ** $P < 0.01$.
† Student's *t* test (or the Mann–Whitney test if the distribution of results was not homogeneous) was used to compare variables between groups. The χ^2 test was used to determine the significance of differences between proportions.

when smoking was taken into account as a covariable. These results are consistent with those reported by Ahmadi *et al.*[36], who reported vitamin E intake to be lower in patients with COPD than in healthy controls. Similarly, Lin *et al.*[12] reported serum vitamin E concentrations to be lower in patients with COPD, and Tug *et al.*[41] observed lower serum concentrations in twenty-four patients with COPD during times of disease exacerbation than during times of stability.

All the subjects had low serum α-tocopherol concentrations (below 23·2 µmol/l[42]), yet all had intakes of >100 % of that recommended. Different factors can be related to this fact. First, absorption of vitamin E could be decreased by the use of cholesterol medication. Therefore, the elderly with high cholesterol and cholesterol medication might suffer from inadequate vitamin E intake[43]. Second, a relationship between increased oxidative stress and the most important age-related

pathologies (i.e. CVD and neurodegenerative disease, cancer and diabetes) has been largely demonstrated[44], and antioxidant status has been demonstrated to be compromised in people with chronic diseases[45]. According to these, although we did not find any relationship between the use of anticholesterolaemic drugs and serum α-tocopherol concentrations, we found that elderly people with α-tocopherol serum values below the 50th percentile showed higher percentage of anaemia and ischaemia/heart failure than people with values above the 50th percentile (5·8 *v.* 0%; $P < 0·05$ and 49·3 *v.* 29·0%; $P < 0·05$, respectively). Thus, the condition of these diseases may compromise the status of vitamin E in elderly people and, although in our study all subjects had intakes above the RI, the current intake could be insufficient to maintain an adequate status of this vitamin. For this reason, the vitamin E RI in elderly people could be insufficient and should be revised. At the moment, it may be a good idea for these subjects to further increase their intake. It should also be remembered that low serum α-tocopherol can result in a low retinyl palmitate concentration[46], making the antioxidant status even worse.

Good coverage of vitamin A intake appeared to offer protection against COPD in our subjects. Similar observations have been reported by other authors. For example, Lin *et al.*[12] reported lower serum retinol and β-carotene in patients with COPD compared with healthy controls, and Tug *et al.*[41] indicated serum vitamin A concentrations to be lower during periods of exacerbation of the disease. In the present study, however, no relationship was seen between COPD and serum β-carotene – a circumstance also reported by Walda *et al.*[37] in their large Finnish study.

The TAC (as measured by the ORAC method – the method of choice according to Awika *et al.*[47]) of the diets of the present subjects with COPD was not as great as that of the healthy subjects. Although the literature contains no other reports relating dietary TAC to COPD, some authors report the antioxidant capacity of patients with COPD to be lower. For example, the plasma antioxidant capacity of the eighty patients with COPD, as determined by the FRAP method, was lower than that of the twenty healthy controls (0·936 *v.* 1·110 mmol Fe^{2+}/l)[48]. It was also found to be lower, as determined by the TEAC method, in ninety-five patients with COPD compared with thirty-seven healthy controls (0·81 *v.* 1·31 mmol/l)[49]. As the antioxidant capacities of the plasma and diet are related[50], the results of the present study, and of others studies, suggest that patients with COPD should follow diets rich in components with antioxidant power, especially fruits (note above the significant relationship between fruit intake and the present ORAC results).

Finally, fruit consumption of healthy controls was higher than that of subjects with COPD, which could also be related to a higher intake of phytochemicals such as catechin and flavonoids from this food group. Moreover, in a study carried out in 13 651 adults from three Dutch cities, consumption of solid fruits was inversely associated with the prevalence of one or more COPD symptoms (cough, phlegm and breathlessness). Indeed, catechin intake was independently associated with all three COPD symptoms. Nevertheless, flavonol and flavone intakes were independently associated with chronic cough only[51].

Intake of phytochemicals was not assessed in the present study because of the lack of available data in the Spanish food composition database used. As the present study is a case–control study, it suffers the limitation in that it cannot identify causal relationships. Moreover, the statistical power of the study was low to detect associations for different exposure factors including the deficiency of vitamin C (37·4%), deficiency of β-carotene (14·7%), deficiency of vitamin A (13%) and deficiency of vitamin E (28·1%) owing to the small sample size. Therefore, these results should be interpreted with caution.

In conclusion, the present subjects with COPD had a poorer antioxidant status (especially in terms of vitamins C and E) than the healthy controls, especially those who smoked. It would therefore seem recommendable that the intake of these nutrients, especially via fruit consumption, be increased in such patients.

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