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# Influence of Smoking and Alcohol Consumption on Total Antioxidant Status in Patients with Psoriasis*

## Wpływ palenia tytoniu i picia alkoholu na całkowity potencjał antyoksydacyjny u pacjentów chorych na łuszczycę*

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## Abstract

**Background.** Psoriasis is regarded as a multifactorial disease in which interaction between genetic and environmental factors seems to play a causative role. Evidence is now accumulating that there is an association between cigarette smoking, alcohol consumption, and psoriasis, but the detailed mechanisms are not yet known. Many of the adverse effects of smoking and alcohol consumption may result from oxidative damage to critical biological substances. Deficiency in any of the antioxidant defense systems can cause a reduction in the total antioxidant status (TAS) of an individual.
**Objectives.** Determining whether alcohol consumption and tobacco smoking may effect total antioxidant status in psoriatic patients.
**Material and Methods.** Sixty patients with psoriasis vulgaris (33 women and 27 men; mean age: 36.7 years, range: 19–67 years) were examined. Sixty healthy people (33 women and 27 men, mean age: 32.8 years, range: 19–62 years) served as the control group. Information on lifetime smoking and alcohol consumption was obtained by questionnaire and included age of onset, average and maximum daily consumption, kind of consumption, and periods of abstinence. TAS was measured in plasma using a kit manufactured by Randox Laboratories, Ltd.
**Results.** The authors found that TAS was significantly lower in the study group of patients than in healthy controls. TAS was significantly lower in the study group of smokers (smoking more than 20 cigarettes a day) than in non-smokers. From the group of people examined the authors separated out those who drank more than 150 g of pure ethanol per week. In this group, TAS was found to be lower than in the group not consuming that much alcohol, but the results reached significance only for women. The authors found negative correlation between TAS and the number of cigarettes smoked per day and the amount of alcohol consumed.
**Conclusions.** This is the first report on the influence of smoking and alcohol consumption on total blood antioxidant status in psoriasis. The study clearly shows that smoking and alcohol drinking lower the total antioxidant status in patients with psoriasis. The antioxidant system of these patients is possibly in a depressed state or overloaded due to the cutaneous infection and oxidative stress caused by smoking and alcohol drinking (**Adv Clin Exp Med 2006, 15, 3, 463–469**).

**Key words:** psoriasis, smoking, alcohol consumption, total antioxidant status.

## Streszczenie

**Wprowadzenie.** Etiopatogenetyczne aspekty łuszczycy są szczególnie złożone i tylko częściowo poznane. Badania epidemiologiczne zwracają uwagę na związek między paleniem tytoniu i spożywaniem alkoholu a łuszczycą, mechanizmy działania nie są jednak dokładnie określone. Za szkodliwe działanie tych używek najprawdopodobniej są odpowiedzialne reakcje wolnorodnikowe, a w rezultacie stres oksydacyjny. Całkowity potencjał antyoksydacyjny mierzy zdolność przeciwutleniającą osocza, na którą składają się zarówno enzymy antyoksydacyjne, jak również wiele antyoksydantów niskocząsteczkowych.
**Cel pracy.** Zbadanie wpływu palenia tytoniu i picia alkoholu na całkowity potencjał antyoksydacyjny osocza (TAS) u pacjentów chorych na łuszczycę zwykłą.
**Materiał i metody.** Badania przeprowadzono u 60 dorosłych osób, chorych na łuszczycę zwykłą (33 kobiet i 27 mężczyzn, w wieku 19–68 lat, średnia 37,8 lat). W grupie kontrolnej było 60 osób zdrowych (33 kobiet i 27 męż-

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czyzn w wieku 19–62 lat, średnia 32,8 lat). Dane dotyczące ilości spożywanego alkoholu i częstości palenia tytoniu zebrano, przeprowadzając szczegółowy wywiad. Całkowity potencjał antyoksydacyjny osocza zmierzono za pomocą zestawu odczynników firmy Randox.

**Wyniki.** U osób chorujących na łuszczycę TAS jest niższy w porównaniu z grupą kontrolną. Wartość TAS jest również znacząco niższa w grupie palaczy (palący powyżej 20 papierosów dziennie) w porównaniu z osobami niepalącymi. Wśród osób spożywających więcej niż 150 g czystego alkoholu tygodniowo TAS jest również niższy w porównaniu do osób niepijących lub pijących mniej, różnice te są jednak istotne statystycznie tylko w grupie kobiet. Wykazano nieistotną statystycznie negatywną korelację między wartością TAS a liczbą wypalanych papierosów i ilością spożywanego alkoholu.

**Wnioski.** Palenie tytoniu i spożywanie alkoholu obniża całkowity potencjał antyoksydacyjny osocza u pacjentów chorych na łuszczycę. Układ antyoksydacyjny pacjentów jest najprawdopodobniej mniej wydolny lub przeładowany w związku ze stanem zapalnym oraz stresem oksydacyjnym spowodowanym przez palenie tytoniu i picie alkoholu (**Adv Clin Exp Med 2006, 15, 3, 463–469**).

**Słowa kluczowe:** łuszczyca, palenie tytoniu, alkohol, całkowity potencjał antyoksydacyjny osocza.

Psoriasis is a common inflammatory and proliferative skin disease of unclear etiology and is known to affect about 2–4.8% of the global population [1]. Typical psoriatic lesions are erythomatous papules which form plaques characterized by sharp borders and increased scaling. Research on the etiology of psoriasis has concentrated on epidermal proliferation and differentiation, inflammatory processes, and the dermal vasculature. According to various research groups, each of these broad areas holds the answer, but there is still no single concept that explains psoriatic features. Genetic and environmental factors have been suggested to play an etiological role in the disease. Both the onset and, above all, the progress of psoriasis seem to be related to interactions between endogenous (genetic) and exogenous (mostly environmental) factors. Among the environmental factors, systemic infections, metabolic disturbances, medications, stress, trauma (physical, chemical, thermal, surgical), alcohol consumption, and tobacco smoking are the most common.

Oxidative stress is now considered to be important in the pathogenesis of psoriasis. Significant abnormalities of antioxidant mechanisms have been demonstrated in the blood and plaques of psoriatic patients [2, 3]. An insufficient antioxidant system, together with increased levels of reactive oxygen species (ROS), has been suggested to be important in the pathogenesis of this disease.

Smoking and alcohol over-consumption are the most significant preventable contemporary health problems of our time. Evidence is now accumulating that there is an association between cigarette smoking, alcohol consumption, and psoriasis [4–7]. Smoking appears to be an important factor in the onset of psoriasis. Alcohol consumption appears to exacerbate pre-existing disease and may be a true risk factor for psoriasis, especially in men [8, 9]. Cigarette smoking could influence psoriasis via a variety of mechanisms. The detailed mechanisms are not yet known, but oxidative stress could play a key role [10–12]. Under normal physiological conditions, free-radical-induced oxidative stress is combated by a complex antioxidant defense system. A major contribution to the total antioxidant capacity comes from antioxidant molecules in plasma. The relative contribution of each antioxidant *in vivo* depends not only on its efficacy, but also on its concentration in biological fluids. Determining total antioxidant status (TAS) provides an index of the sum of the activities of all antioxidants.

No studies on oxidative stress have been performed in relation to smoking and alcohol consumption in patients with psoriasis. Therefore authors' aim was to determine blood antioxidant status of patients with psoriasis and its relation to tobacco smoking and alcohol consumption.

# Material and Methods

## Patients and Controls

Blood samples were obtained from 60 patients with psoriasis vulgaris (33 women and 27 men, mean age: 36.7 years, range: 19–67 years). The control group included 33 women and 27 men (mean age: 32.8 years, range: 19–62 years). The duration of the disease ranged from 4 to 25 years (average: 9.4 years). All of the patients were in the eruptive stage of psoriasis and had only been treated topically. Neither photochemotherapy nor oral medication were used. Each case underwent thorough clinical examination to establish the Psoriasis Area and Severity Index (PASI) [13]. The PASI varied from 5.4 to 9.7 (mean: 6.3).

All of the patients and the healthy controls answered questionnaires. Information regarding socio-demographic factors, alcohol consumption, smoking habits, family history of psoriasis in first- and second-degree relatives, and personal medical history was obtained from psoriatic patients and controls by trained investigators using a structured questionnaire.

The authors computed the average consumption of alcohol assuming the following pure alcohol content for each type of drink: 250 ml of wine = 500 ml of beer = 45 ml of vodka = 18 g of pure ethanol. Positive history of smoking was defined as smoking ≥ 20 cigarette per day for at least 2 years. People who had not smoked at all for at least 2 years were considered non-smokers. A positive drinking history was defined as drinking more than 150 g of pure alcohol per week.

## Collection and Preparation of Blood Samples

Blood samples from the controls and patients with psoriasis were collected after overnight fasting into vacatoner tubes containing the disodium salt of ethylenediaminetetraacetic acid (EDTA) as anticoagulant. The anticoagulated blood was separated into plasma and red blood cells by centrifugation (3000 rpm for 20 min).

### Blood Testing

Total antioxidant status was determined in the psoriatic patients and controls using the Randox Kit, referred to as the "Total Antioxidant Status Kit". This kit allows the measurement of the total amount of antioxidants by inhibition of the transformation of 2,2-azino-di-[3-ethylbenzthiazoline sulfonate] (ABTS) into the radical cation ($ABTS^{\cdot+}$) in the presence of a peroxidase (metamyoglobin) and $H_2O_2$. This cation has a fairly stable blue-green color which can be measured at 600 nm. Antioxidants present in the sample suppress this color formation in proportion to their concentration. A plasma sample volume of 5 µl of plasma is incubated with 200 µl of a reagent containing metamyoglobin and ABTS at 37°C for 15 s, during which two measurements are taken. Then 50 µl of $H_2O_2$ is added and thoroughly mixed, starting the reaction. The reaction is followed for exactly 3 min. The reaction rate of the sample is compared with that of the standard and a blank to determine the concentration of total antioxidants present in the sample and is expressed as mmol/l of plasma. All reagents and calibrator control materials were supplied by Randox Laboratories.

All values are expressed as the mean ± *SD*. The mean values obtained in the different groups were compared by the t-test (parametric) or Mann-Whitney U-test (nonparametric), assuming that there are differences between mean values when statistical comparison resulted in $p \leq 0.05$. Correlation coefficients were calculated using least-square linear regression.

# Results

Total antioxidant status was significantly lower in the study group of patients (TAS, mean concentration: 1.187 ± 0.13 mmol/l) compared with healthy controls (1.339 ± 0.12 mmol/l) (Table 1).

Twenty-four persons in the control group and 36 in the patient group presented positive histories of smoking (as described above). There were only 17 non-smokers in the patient group and 29 in the control group.

TAS was significantly lower in the smokers than in the non-smokers of the group of psoriatic patients (1.064 ± 0.12 mmol/l and 1.243 ± 0.12 mmol/l, respectively) and of the group of controls (1.198 ± 0.12 mmol/l and 1.402 ± 0.13 mmol/l, respectively) (Table 2).

The number of cigarettes smoked daily correlated negatively with TAS in the group of psoriatic patients and controls (correlation coefficients: –0.22 and –0.28, respectively), but without statistical significance.

**Table 1.** Total antioxidant status among psoriatic patients and the control group (each value represents mean ± SD)

**Tabela 1.** Całkowity potencjał antyoksydacyjny osocza u chorych na łuszczycę i w grupie kontrolnej (każda wartość oznacza średnią ± odchylenie standardowe)

| | | n | Total antioxidant status (Całkowity potencjał antyoksydacyjny osocza) mmol/l ± SD |
|---|---|---|---|
| Psoriatic patients (Chorzy na łuszczycę) | men (mężczyźni) | 27 | 1.258 ± 0.14 |
| | women (kobiety) | 33 | 1.127 ± 0.12 |
| | total (razem) | 60 | 1.187 ± 0.13* |
| Control group (Grupa kontrolna) | men (mężczyźni) | 27 | 1.368 ± 0.12 |
| | women (kobiety) | 33 | 1.319 ± 0.11 |
| | total (razem) | 60 | 1.339 ± 0.12* |

* statistical significance ($p < 0.05$).

* istotność statystyczna ($p < 0,05$).

**Table 2.** Total antioxidant status in psoriatic patients and in the control group in relation to smoking habits (each value represents mean ± SD)

**Tabela 2.** Całkowity potencjał antyoksydacyjny osocza u chorych na łuszczycę i w grupie kontrolnej w zależności od palenia tytoniu (każda wartość oznacza średnią ± odchylenie standardowe)

| | | Psoriatic patients (Chorzy na łuszczycę) | | Control group (Grupa kontrolna) | |
|---|---|---|---|---|---|
| | | n | TAS mmol/l ± SD | n | TAS mmol/l ± SD |
| Smokers (Palący) | men (mężczyźni) | 19 | 1.083 ± 0.12 | 14 | 1.318 ± 0.12 |
| | women (kobiety) | 17 | 0.982 ± 0.11 | 10 | 1.072 ± 0.11 |
| | all (razem) | 36 | 1.064 ± 0.12* | 24 | 1.198 ± 0.12* |
| Non-smokers (Niepalący) | men (mężczyźni) | 7 | 1.297 ± 0.12 | 11 | 1.446 ± 0.13 |
| | women (kobiety) | 10 | 1.182 ± 0.11 | 18 | 1.381 ± 0.12 |
| | all (razem) | 17 | 1.243 ± 0.12* | 29 | 1.402 ± 0.13* |

* statistical significance ($p < 0.05$).

* istotność statystyczna ($p < 0,05$).

In the group of patients, TAS was lower in those consuming more than 150 g of pure alcohol per week (1.115 ± 0.12 mmol/l) than in those drinking less or non-drinkers (1.203 ± 0.12 mmol/l), but without statistical significance. The difference was significant only for women (Table 3).

Among controls, TAS was also lower in those consuming more than 150 g of pure alcohol per week, but without statistical significance.

Comparing patients with psoriasis and controls in the group consuming more than 150 g of pure alcohol per week, TAS was significantly higher in the healthy women (1.282 ± 0.11 mmol/l), compared with psoriatic women (0.982 ± 0.11 mmol/l). In the whole group with positive history of drinking, the same difference was also recorded (1.115 ± ± 0.12 mmol/l in the study group compared with 1.312 ± 0.12 mmol/l in the whole control group). Also, in the group consuming less than 150 g of pure alcohol per week, the difference between healthy and psoriatic men did not reach statistical significance. There were significant differences between psoriatic patients and controls in the whole groups (1.203 ± 0.12 mmol/l and 1.335 ± 0.13 mmol/l respectively) and between healthy and psoriatic women (1.176 ± 0.11 mmol/l and 1.342 ± ± 0.12 mmol/l, respectively) (Table 3).

Ethanol consumption negatively correlated with TAS in the patients group and controls (correlation coefficients: –0.32 and –0.28, respectively), but without statistical significance ($p = 0.0582$). TAS was higher in males than in females in all the studied groups, also when divided into subgroups according to alcohol consumption and smoking habits.

There were no statistical differences in PASI in all the studied groups of patients.

**Table 3.** Total antioxidant status among psoriatic patients and in the control group in relation to the amount of alcohol consumed weekly (each value represents mean ± SD)

**Tabela 3.** Całkowity potencjał antyoksydacyjny osocza u chorych na łuszczycę i w grupie kontrolnej w zależności od ilości alkoholu spożywanego tygodniowo (każda wartość oznacza średnią ± odchylenie standardowe)

| | | Psoriatic patients (Chorzy na łuszczycę) | | Control group (Grupa kontrolna) | |
|---|---|---|---|---|---|
| | | n | TAS mmol/l ± SD | n | TAS mmol/l ± SD |
| Group consuming > 150 g pure ethanol/week (Grupa konsumująca > 150 g czystego etanolu tygodniowo) | men (mężczyźni) | 15 | 1.233 ± 0.12 | 13 | 1.328 ± 0.12 |
| | women (kobiety) | 13 | 0.982 ± 0.11* | 11 | 1.282 ± 0.11* |
| | all (razem) | 28 | 1.115 ± 0.12* | 24 | 1.312 ± 0.12* |
| Group consuming < 150 g pure ethanol/ week (Grupa konsumująca < 150 g czystego etanolu tygodniowo) | men (mężczyźni) | 12 | 1.289 ± 0.12 | 14 | 1.385 ± 0.13 |
| | women (kobiety) | 20 | 1.176 ± 0.11* | 22 | 1.342 ± 0.12* |
| | all (razem) | 32 | 1.203 ± 0.12* | 36 | 1.335 ± 0.13* |

* statistical significance ($p < 0.05$).

* istotność statystyczna ($p < 0,05$).

# Discussion

The implication of antioxidant metabolism in the etiopathogenesis of psoriasis has been sustained by several studies [2, 3, 14, 15]. Under normal physiological conditions, free-radical-induced oxidative stress is combated by a complex antioxidant defense system. A major contribution to the total antioxidant capacity comes from antioxidant molecules in plasma. Plasma, however, is not a simple chemical system for combating oxidative stress. The antioxidant defense system has many components. The relative contribution of each antioxidant *in vivo* depends not only on its efficacy, but also on its concentration in biological fluids. In humans there are two main defense systems against oxidative stress. The first involves mineral-dependent enzymes, superoxide dismutase (SOD) and glutathione peroxidase (GSH-Px), that, in association, control the level of reactive oxygen species (superoxide anion, hydroxyl radical, and hydrogen peroxide). The second defense system consists of non-enzymatic substances. Albumin, uric acid, and ascorbic acid account for over 85% of the total antioxidant capacity in human plasma [16]. This predominance is due largely to their high concentrations relative to those of other antioxidants in blood, e.g. α-tocopherol, β-carotene, and bilirubin. Although individual antioxidants play a specific role in the antioxidant defense system, the above antioxidant molecules may act cooperatively *in vivo* to provide synergistic protection against oxidative damage [17]. Measuring the levels of specific antioxidant molecules can yield valuable information, and low levels of such antioxidants provide suggestive, but not definitive, evidence of oxidative stress. However, determining total antioxidant capacity provides an index of the sum of the activities of all antioxidants.

The study demonstrates the influence of smoking and alcohol consumption on TAS in psoriatic patients. Total antioxidant status was significantly lower in psoriatic patients compared with healthy controls. Similar results were obtained by Vanizor Kural et al. [18], who found significant decreases in the levels of antioxidant enzymes activities and total antioxidant status in psoriatic patients compared with control subjects. According to the results of Severin et al. [19], total antioxidative capacity was normal in plasma from psoriasis patients despite elevated bilirubin, tocopherol, and urate levels.

No studies on total antioxidant capacity of plasma have been performed in relation to smoking and alcohol consumption in patients with psoriasis.

In presented study, TAS in smoking men and women was lower than in non-smokers. In the group of patients and in controls, TAS was lower in women who smoked than in nonsmoking women, although the difference was not statistically significant. Several studies have suggested a role for tobacco smoke in psoriasis. Naldi et al. [7] reported that smoking 15 cigarettes per day is one of the risk factors that induce psoriasis. Poikolainen et al. [5] suggested that smoking, drinking, and life style are related to psoriasis, especially among women. Cigarette smoke contains a large number of oxidants, leading to the hypothesis that many adverse effects of smoking result from oxidative damage. Such damage could result from the oxidants present in cigarette smoke and from the activation of phagocytic cells that generate reactive oxygen species [11]. Chemotaxis in patients who smoke appears higher than in nonsmokers [10]. Thus, this phenomenon might contribute to the causative link between smoking and psoriasis.

TAS was found to be lower in patients consuming more than 150 g of pure alcohol per week compared with those drinking less or non-drinkers. Increased alcohol consumption is a recognized stress response and there has been much debate as to whether increased drinking is a cause or a consequence of psoriasis.

Alcohol can not only trigger psoriasis, but it also influences the course and nature of the disease, heavy drinkers tending to have more severe, extensive, and inflamed disease [9, 20, 21]. According to many studies [5, 22], psoriasis is much more frequent in alcoholics than in controls. The fact that heavy drinkers usually tend to pay less attention to healthy nutrition (rich in natural antioxidants) may be responsible for this [23, 24]. Many antioxidants are present in red wines. Polyphenols possess antioxidant capability due to the hydrogen-donating capacity of their phenolic groups. Unfortunately, nobody in the experimental group drank red wine. The most popular were beer and vodka. None of the above-mentioned studies were aimed at clarifying whether drinking increases the risk of psoriasis or whether it was only an epiphenomenon related to the chronic course and disabling nature of the disease. In presented studies there were no statistical differences in PASI in all the studied groups, which is probably due to the relatively low PASI score in the studied patients. According to many studies, both alcohol consumption and smoking may have effect on PASI [20–22].

There are several theories on how alcohol may affect psoriasis. Most are based on theoretical considerations inferred from our knowledge of the action of alcohol on other organic systems. Most investigators suggest that alcohol affects psoriasis mainly by altering the immune system. Proposed mechanisms have included suppression of cell-

mediated immunity [25], up-regulation of pro-inflammatory cytokines [26], and enhancement of mitogen-driven lymphocyte proliferation [27] caused by alcohol consumption. The mechanisms leading to the decrease in TAS in psoriatic patients consuming alcohol are conflicting. Besides inadequate dietary intake, increased losses or reduced hepatic storage capacity may be involved [24]. This study shows that decreased total antioxidant capacity could be added to the possible mechanisms explaining the interaction between alcohol and psoriasis.

Diet, food, and alcohol are the major factors affecting both the antioxidant system and the production of free radicals and reactive oxygen species [28]. Plasma antioxidants of dietary origin (e.g. tocopherols, ascorbic acid, carotenoids) are influenced directly by nutritional supplements as well as by food and alcohol consumption [29, 30]. In addition, lower levels of plasma antioxidants of dietary origin were found in subjects who smoke cigarettes [31, 32]. Poorer dietary practice associated with smoking, which is observed around the world in various cultures, could be a reflection of the different personality types of smokers and nonsmokers, with less healthy lifestyles and lack of dietary knowledge more common in persons who smoke [33].

In conclusion, this study clearly shows that smoking lowers the total antioxidant capacity of plasma in patients with psoriasis. The antioxidant system of those patients is possibly in a depressed state or is overloaded due to the inflammation and oxidative stress caused by smoking and/or alcohol drinking. Physicians who consult patients with psoriasis should be aware that there is a greater chance they are treating a patient with psoriasis who is also an alcoholic or a smoker. If psoriasis positively influences some people to drink alcohol excessively, then this additional risk factor needs to be considered when designing a treatment strategy. Prevention of excessive alcohol use and smoking among patients could lower the frequency and severity of psoriasis, alleviate their disease, and improve their quality of life.

These findings confirm previous investigations that have suggested that alcohol consumption and, more likely, smoking have negative influence on the antioxidant system in patients with psoriasis.

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