Serum Paraoxonase-1 Activities and Oxidative Status in Patients With Plaque-Type Psoriasis With/Without Metabolic Syndrome

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Objective: Psoriasis is a chronic immune-mediated inflammatory skin disease associated with metabolic syndrome, which is made up of a cluster of disorders, including obesity, diabetes mellitus, dyslipidemia, and cardiovascular disease. The aim of this study was to investigate serum paraoxonase-1 activities and oxidative status parameters in patients with plaque-type psoriasis with or without metabolic syndrome. **Methods:** In this study, patients with plaque-type psoriasis with ($n = 25$) or without ($n = 27$) metabolic syndrome, according to the criteria of the International Diabetes Federation (IDF), were matched for age and sex to an equally sized control group ($n = 25$). **Results:** In patients without metabolic syndrome, serum paraoxonase and arylesterase activities showed mean decreases of 29 and 6%, respectively, whereas in patients with metabolic syndrome, the mean decreases in the

enzymes' activities were 35 and 11%, respectively, compared with those in the control group. Serum total antioxidant capacity and total oxidant status were not statistically significant in any of the three groups. Multiple linear regression analysis revealed that HDL cholesterol and log-transformed triglyceride were independent variables for serum arylesterase activity and that fasting glucose and diastolic blood pressure were independent variables for serum paraoxonase activity. **Conclusions:** These results suggest that according to the criteria of the IDF, the significant decrease observed in serum paraoxonase activity was independent of the metabolic syndrome in patients with mild-to-moderate plaque-type psoriasis, whereas the significant decrease in serum arylesterase activity was associated with the metabolic syndrome. *J. Clin. Lab. Anal.* 25:289–295, 2011. © 2011 Wiley-Liss, Inc.

Key words: metabolic syndrome; oxidative stress; paraoxonase-1; psoriasis

INTRODUCTION

Psoriasis is a chronic inflammatory skin disease with a genetic basis characterized by epidermal hyperproliferation, abnormal keratinocyte differentiation, T-lymphocyte infiltration, and increased expression of cytokines, which results in the formation of inflamed plaques (1,2). Increased production of reactive oxygen species (ROS) and inflammatory cytokines, such as tumour necrosis factor- α and interleukin-1 play important roles in the development and severity of psoriatic lesions (3,4). Chronic inflammation and ROS are thought to be responsible for the increased prevalence of systemic

metabolic disorders, such as central obesity, diabetes, dyslipidemia, hypertension, and cardiovascular disease.

Several bodies have put forward different definitions of metabolic syndrome, a cluster of metabolic and cardiovascular disorders. The criteria of the World

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Health Organization (WHO), National Cholesterol Education Program Adult Treatment Panel III (NCEP ATP III), and the International Diabetes Federation (IDF) are widely accepted. They include insulin resistance, central obesity, dyslipidemia, and hypertension as major components of the cluster of risk factors (5–7). Several studies of the prevalence of metabolic syndrome in patients with psoriasis have established that psoriasis is an independent risk factor for this syndrome (8–13).

Human serum paraoxonase-1 (PON1) is a calcium-dependent esterase that hydrolyses aromatic carboxylic acid esters, organophosphates, and carbamates, and is associated with high-density lipoprotein (HDL) (14,15). Several studies have suggested antioxidant role of PON1 on general prevention of peroxidative damage to cell membranes and oxidation of biomolecules, such as low-density lipoprotein (LDL) (16,17). PON1 exhibits paraoxonase, arylesterase, and diazoxonase activities in addition with genetic polymorphisms at positions 55 and 192 are the main determinants of its expression and the enzymatic activity (18–20). Recent studies showed that decreased PON1 activity with increased oxidative stress and metabolic syndrome severity were associated with progressive worsening of the oxidant–antioxidant balance (21,22).

The purpose of this study was to investigate the relationship between changes in serum PON1 activities, which were paraoxonase and arylesterase, and the serum oxidant–antioxidant status in patients with plaque-type psoriasis with or without metabolic syndrome according to the definition of the IDF.

MATERIALS AND METHODS

Study Population

This cross-sectional study comprised 52 patients with plaque-type psoriasis and 25 healthy individuals aged 20–55 years. Patients with plaque-type psoriasis who had been followed up at the dermatology outpatient clinic of the Istanbul Education and Research Hospital and first diagnosed with clinical and/or histopathological findings of plaque-type psoriasis at least 1 year duration before enrolment were included in this study. Patients with plaque-type psoriasis were defined as those with metabolic syndrome (Group II, $n = 25$) or without metabolic syndrome (Group I, $n = 27$), as per the IDF criteria (7). The control group consisted of 25 healthy individuals without skin and systemic metabolic diseases. None of the participants were taking any systemic drugs or undergoing phototherapy; however, 48% ($n = 13$) of Group I patients and 56% ($n = 14$) of Group II patients were taking topical steroids.

The study was approved by the local medical ethical committee of the Istanbul Education and Research Hospital, and written informed consent was obtained from all participants.

Exclusion Criteria

Patients with histories and symptoms of inverse, erythrodermic, pustular, or guttate psoriasis, psoriatic arthritis, or other chronic inflammatory skin diseases, and comorbidities associated with psoriasis, such as Crohn's disease, ulcerative colitis, multiple sclerosis, depression, lymphoma, melanoma, or nonmelanoma skin cancer, were excluded. Participants with histories and symptoms of anemia, thalassemia, renal dysfunction, diabetes, cardiovascular disease, autoimmune disease, neoplasms, liver disease, inflammation, hypothyroidism, or hyperthyroidism, who smoked or consumed alcohol and who received any type of drug treatment (e.g. antihypertensives, lipid-lowering agents, antidiabetic agents, or vitamin supplements) for systemic diseases or any type of treatment (e.g. acitretin, cyclosporine, methotrexate, biologics, or phototherapy) for psoriasis at least 1 month before enrolment were also excluded.

Definitions

The homeostasis model assessment index for insulin resistance was calculated as fasting insulin (mU/l) $\times$ fasting glucose (mmol/l) divided by 22.5 (23). The body mass index was calculated as the ratio between weight and height in kilograms per square meter (kg/m^2). The waist circumference was measured on naked skin between the tenth rib and the iliac crest in centimetres. All systolic blood pressure (SBP) and diastolic blood pressure (DBP) measurements were made by a single person using a mercury sphygmomanometer.

The Psoriasis Area and Severity Index (PASI) was used to measure the area of involvement and the severity of disease in patients with plaque-type psoriasis. The original definition of the PASI involves an assessment of four body regions—head [h], trunk [t], upper [u], and lower [l] extremities—of erythema (E), infiltration (I), desquamation (D), and body surface area involvement (A):

$$\text{PASI} : 0.1 (E_h + I_h + D_h)A_h + 0.2 (E_u + I_u + D_u)A_u + 0.3 (E_t + I_t + D_t)A_t + 0.4 (E_l + I_l + D_l)A_l \quad (24).$$

According to the IDF criteria for metabolic syndrome, the condition is defined as the presence of at least three of the following five features: (1) Abdominal obesity (waist circumference ≥ 94 cm in men and ≥ 80 cm in women); (2) hypertriglyceridemia (> 150 mg/dl); (3) low levels of serum HDL cholesterol (< 40 mg/dl in men and < 50 mg/dl in women); (4) high fasting glucose (≥ 100 mg/dl); and

(5) elevated blood pressure (≥ 130 mm Hg systolic or ≥ 85 mm Hg diastolic) (7).

Laboratory Analyses

We obtained venous blood samples from all participants following overnight fasting. Serum samples were stored at -80°C before the collection of measurements. Levels of serum glucose, total cholesterol, HDL cholesterol, and triglyceride were measured using commercially available kits (Abbott Laboratories, Chicago, IL) with an Abbott Aeroset[®] autoanalyzer. LDL cholesterol was calculated using the Friedewald formula (25). Serum insulin levels were measured using commercially available kits (Roche Diagnostics GmbH, Mannheim, Germany) with an Elecsys[®] Systems E170 Modular Analytics autoanalyzer.

Measurements of Total Antioxidant Capacity and Total Oxidant Status

Serum total antioxidant capacity (TAC) and total oxidant status (TOS) were determined using the novel automated measurement methods developed by Erel (26,27). The TAC assay was calibrated with a stable antioxidant standard solution, which is traditionally named the Trolox equivalent, a vitamin E analog. The results were expressed as mmol Trolox Equiv./l. The TOS assay was calibrated using hydrogen peroxide and the data were expressed as $\mu\text{mol H}_2\text{O}_2$ Equiv./l.

Measurements of Serum Paraoxonase and Arylesterase Activities of PON1 Enzyme

Serum paraoxonase and arylesterase activities were determined using commercially available kits (Rel Assay Diagnostics[®], Mega Tip, Gaziantep, Turkey). Serum paraoxonase activity toward paraoxon was measured following hydrolysis of paraoxon to yield *p*-nitrophenol and diethyl phosphate in the absence of NaCl (baseline activity). The molar extinction coefficient of *p*-nitrophenol was $17,000\text{ M}^{-1}\text{ cm}^{-1}$ at pH 8; the results were expressed as U/l. Serum arylesterase activity was determined by the presence of phenol following the reaction of phenylacetate. The molar extinction coefficient of phenol was $4,000\text{ M}^{-1}\text{ cm}^{-1}$; the results were expressed as kU/l.

Statistical Analysis

Number Cruncher Statistical System 2004 (NCSS systems, Kaysville, UT) and MedCalc (MedCalc software, Broekstraat, Mariakerke, Belgium) were used for data analysis. Data were presented as mean $\pm$ SD or median (25th and 75th percentiles). Statistical comparisons between groups were performed using one-way

ANOVA analysis for data with Gaussian distributions. The Kruskal–Wallis test was used for data with non-Gaussian distributions. Post hoc analysis was performed using Tukey or Tamhane's T2 test for one-way ANOVA analysis and the Tukey–Kramer method for the Kruskal–Wallis test. Pearson's or Yates' chi-square test was used to compare observed with expected frequencies. Pearson's or Spearman's correlation analysis was used to establish bivariate correlations and presented as the correlation coefficients r or r_s . To determine the independent association of the variables in the presence of other significant risk factors, multiple linear regression analysis was performed. Two-tailed P values less than 0.05 were considered significant.

RESULTS

The clinical and biochemical features of the study population are summarized in Table 1. Age and gender were not significantly different among the three groups ($P = 0.161$ and 0.788 , respectively), and the median values for the PASI score and the disease age were similar in Group I and II. The components of the metabolic syndrome in Group II were significantly different compared with those in the control group and Group I, whereas those in Group I were not significantly different compared with those in the control group (Table 1). Frequencies of metabolic syndrome and its components in the groups according to the IDF definition are shown in Table 2.

The serum paraoxonase and arylesterase activities in the individual groups are shown in Figure 1. Serum paraoxonase activity was 284.6 ± 151.7 U/l in the control group. This activity was significantly decreased ($P = 0.013$) in Group I (201.5 ± 103.9 U/l; mean decrease 29%) and Group II (185.3 ± 112.4 U/l; mean decrease 35%). Serum arylesterase activity was 256.9 ± 38.2 kU/l in the control group and this activity was significantly decreased ($P = 0.042$) in Group I (241.7 ± 40.2 U/l; mean decrease 6%) and Group II (228.6 ± 38.6 U/l; mean decrease 11%). The differences in serum paraoxonase and arylesterase activities between the two patient groups were not statistically significant ($P = 0.885$ and 0.448 , respectively).

Serum TAC and TOS were not statistically different in the three groups ($P = 0.249$ and 0.748 , respectively).

In the control group, there was a positive correlation between arylesterase activity and HDL cholesterol ($r = 0.520$; $P = 0.008$). In Group I, serum paraoxonase activity was correlated significantly with the PASI score ($r_s = -0.425$; $P = 0.027$), and serum arylesterase activity was correlated significantly with total ($r = 0.638$; $P < 0.0001$) and LDL cholesterol ($r = 0.580$; $P = 0.002$).

TABLE 1. Clinical and Biochemical Features of the Study Population ($n = 77$)

Variables	Control group ($n = 25$)	Group I ($n = 27$)	Group II ($n = 25$)	<i>P</i>
Age (years)	35 ± 9	36 ± 9	39 ± 9	0.1610
Male gender (%)	40.0	48.1	40.0	0.7880
Period of disease (years)		10 (4–20)	10 (4–19)	
PASI		3.6 (2.7–6.0)	3.7 (3.1–5.8)	
Fasting glucose (mg/dl)	91.8 ± 6.2	92.6 ± 6.2	105.4 ± 21.6 ^{a,b}	<0.0010
Insulin (ng/ml)	7.5 ± 3.3	8.9 ± 3.5	19.7 ± 8.7 ^{g,h}	<0.0001
C-peptide (ng/ml)	1.84 ± 0.46	2.23 ± 0.61 ^a	3.36 ± 1.19 ^{g,h}	<0.0001
Total cholesterol (mg/dl)	177.2 ± 30.4	176.4 ± 36.2	203.7 ± 48.8 ^{a,b}	0.0230
Triglyceride (mg/dl)	74 (61–130)	101 (81–136)	151 (122–249) ^{b,c}	<0.0001
HDL cholesterol (mg/dl)	52.3 ± 9.3	47.3 ± 10.8	39.1 ± 7.4 ^{g,f}	<0.0001
LDL cholesterol (mg/dl)	105.5 ± 26.3	107.2 ± 29.4	125.9 ± 37.5	0.0420
HOMA-IR	1.7 ± 0.7	2.1 ± 0.9	5.2 ± 2.8 ^{g,h}	<0.0001
BMI (kg/m ²)	24.3 ± 3.7	25.6 ± 4.3	31.7 ± 3.6 ^{g,h}	<0.0001
Waist circumference (cm)	81.4 ± 10.7	86.6 ± 8.9	102.3 ± 10.3 ^{g,h}	<0.0001
SBP (mmHg)	111.1 ± 10.2	118.6 ± 12.3	132.4 ± 17.3 ^{g,h}	<0.0001
DBP (mmHg)	73.8 ± 8.3	80.4 ± 9.8	90.2 ± 11.1 ^{g,h}	<0.0001
Paraoxonase activity (U/l)	284.6 ± 151.7	201.5 ± 103.9 ^a	185.3 ± 112.4 ^a	0.0130
Arylesterase activity (kU/l)	256.9 ± 38.2	241.7 ± 40.2	228.6 ± 38.6 ^a	0.0420
TOS (μmol H ₂ O ₂ Equiv./l)	3.57 (3.27–4.63)	3.85 (3.12–5.45)	3.67 (3.42–4.57)	0.7480
TAC (mmol Trolox Equiv./l)	2.27 (2.10–2.34)	2.25 (2.09–2.46)	2.34 (2.19–2.62)	0.2490

^a $P < 0.05$ vs. control group; ^b $P < 0.05$ vs. Group I; ^c $P < 0.01$ vs. control group; ^d $P < 0.01$ vs. Group I; ^e $P < 0.001$ vs. control group; ^f $P < 0.001$ vs. Group I; ^g $P < 0.0001$ vs. control group; ^h $P < 0.0001$ vs. Group I; PASI, Psoriasis Area and Severity Index; BMI, body mass index; SBP, systolic blood pressure; DBP, diastolic blood pressure; TOS, total oxidant status; TAC, total antioxidant capacity; HDL, high-density lipoprotein; LDL, low-density lipoprotein.

TABLE 2. Frequencies of Metabolic Syndrome and its Components in the Groups According to the IDF Definition

	Control group ($n = 25$)	Group I ($n = 27$)	Group II ($n = 25$)
Components ^a (%)			
Fasting glucose	0	7.4	52.0
Waist circumference	28.0	40.7	100
HDL cholesterol	16.0	33.3	84.0
Triglyceride	16.0	11.1	64.0
Blood pressure	0	25.9	76.0
Number of components (%)			
0	60.0	18.5	0
1	28.0	44.4	0
2	12.0	37.0	0
3	0	0	40.0
4	0	0	44.0
5	0	0	16.0

IDF, International Diabetes Federation.

^aThe cut-off values of the components according to the IDF definition were used.

In Group II, there was no statistically significant correlation between these variables.

In patients with plaque-type psoriasis ($n = 52$), serum paraoxonase and arylesterase activities were not associated with the number of metabolic disturbances (0–5) ($r_s = -0.055$; $P = 0.699$ and $r_s = -0.054$; $P = 0.705$, respectively). Patients with plaque-type psoriasis were

divided into two groups; those with low- and high-serum paraoxonase or arylesterase activity, using as cut-off points of the mean–1 SD of the healthy controls. In Group I, 10/27 (37%) patients showed decreased serum paraoxonase activity, and in Group II, 12/25 (48%) patients exhibited a decrease ($P = 0.604$). Likewise, 8/27 (30%) patients in Group I and 8/25 (32%) in Group II exhibited decreased serum arylesterase activity ($P = 1.00$).

In addition, multiple linear regression analysis was performed with serum paraoxonase or arylesterase activity serving as the dependent variable. Fasting glucose, waist circumference, HDL cholesterol, triglyceride, and SBP and DBP—all components of the criteria for metabolic syndrome—were used as the independent variables (Table 3). In patients with plaque-type psoriasis, HDL cholesterol ($P < 0.001$) and log-transformed triglyceride ($P = 0.029$) for serum arylesterase activity and fasting glucose ($P = 0.016$) and DBP ($P = 0.006$) for serum paraoxonase activity remained as the independent variables in model after stepwise backward elimination analysis.

DISCUSSION

In our study, serum paraoxonase activity decreased significantly in patients with plaque-type psoriasis with or without metabolic syndrome compared with the

Fig. 1. (A) Serum paraoxonase activity and (B) serum arylesterase activity in the control group ($n = 25$), Group I ($n = 27$), and Group II ($n = 25$). Error bars were presented as 95% coefficient interval for mean. $P = 0.047$ for serum paraoxonase activity (Group I vs. control group). $P = 0.016$ for serum paraoxonase activity (Group II vs. control group). $P = 0.003$ for serum arylesterase activity (Group II vs. control group).

TABLE 3. Multiple Linear Regression Analysis Between Serum Paraoxonase or Arylesterase Activity and the Components of Metabolic Syndrome in Patients With Plaque-Type Psoriasis ($n = 52$)

	Serum paraoxonase activity			Serum arylesterase activity		
	β	SE	P	β	SE	P
Constant	160.1	204.0	0.437	65.2	72.5	0.374
Fasting glucose	1.9	1.0	0.058	0.0	0.4	0.950
Waist circumference	1.0	1.6	0.539	0.3	0.6	0.570
HDL cholesterol	1.5	1.6	0.339	1.8	0.6	0.003
Log-transformed triglyceride	21.0	72.2	0.772	59.0	25.7	0.026
SBP	-0.3	1.4	0.858	-0.6	0.5	0.219
DBP	-3.9	2.1	0.079	0.2	0.8	0.824

Serum paraoxonase or arylesterase activity was the dependent variables. Also, the components of metabolic syndrome were independent variables. β , unstandardized regression coefficient; SE, standard error. HDL, high-density lipoprotein; SBP, systolic blood pressure; DBP, diastolic blood pressure.

control group, and was correlated inversely with disease severity according to the PASI in the patients without metabolic syndrome. Serum arylesterase activity in different groups also showed a similar decreasing profile. There was a significant difference, however, in patients with plaque-type psoriasis with metabolic syndrome compared with the control group. These data suggest that the decrease in serum paraoxonase activity in the former was independent of metabolic syndrome and that the decrease in serum arylesterase activity was associated with the syndrome, according to the IDF criteria. Although the association between the activities of PON1 and plaque-type psoriasis remains unclear in the literature, a recent study in contrast to our study reported increases in baseline serum paraoxonase and arylesterase activities in patients with psoriasis (28). Unlike our study, patients with different clinical types of psoriasis, such as vulgar, guttate, palmoplantar, and scalp, were included in the analysis. In addition, prediabetic, diabetic, and

hypertensive states of the patient and control groups in the study were not clear. Investigating serum or plasma PON1 activities in different clinical types of psoriasis would, therefore, prove useful.

As noted earlier, WHO, NCEP ATP III, and IDF use different definitions of metabolic syndrome (5–7). Although the same properties are measured, each organization employs different combinations and cut-off points. For example, insulin resistance and impaired fasting glucose are essential components of the WHO definition, whereas central obesity assessed via waist circumference is crucial, according to the IDF definition. Based on our findings, evaluated according to the IDF criteria, 52% of the patients with metabolic syndrome had impaired fasting glucose, 84% had decreased HDL cholesterol, and 64% had increased triglyceride levels. Therefore, the patients with metabolic syndrome exhibited a more lipidcentric than gluco-centric phenotype.

Decreased serum PON1 activity and concentrations have previously been reported in patients with coronary artery disease (CAD) and diabetes in the presence of metabolic syndrome, according to the WHO definition, and these conclusions largely held when diabetic patients were excluded from the study (21). In two studies that employed the NCEP ATP III definition of metabolic syndrome, one reported a significant decrease in PON1 activity in the patients with metabolic syndrome (22), whereas the other was unable to draw any conclusion as a result of the small sample size (29). PON1 activity did not decrease dramatically in the patients with metabolic syndrome in either study, and the participants were older than those in our analysis. In our study, the patients with metabolic syndrome did not exhibit clinical findings and symptoms of CAD or diabetes. Both the control and patient groups showed similar features in respect to age and sex, and serum paraoxonase and arylesterase activities showed no significant differences in patients with plaque-type psoriasis with or without metabolic syndrome. In considering these findings, it is important to note that waist circumference, the main component of the IDF definition, showed no independent correlation with either serum paraoxonase or arylesterase activity. In addition, HDL cholesterol and log-transformed triglyceride were independent variables for serum arylesterase activity, and that fasting glucose and DBP were independent variables for serum paraoxonase activity.

In our study, oxidative stress status was evaluated using serum TOS and TAC. The main components of serum TOS were hydrogen peroxide and lipid hydroperoxide; in serum TAC, they were total-SH groups and ascorbic acid. We found that the values of these parameters in the three groups were not statistically different. Several possible factors may explain this finding. First, studies show that antipsoriatic therapy leads to hyperglycemia, impairment of glucose intolerance, and subsequent increased ROS generation (30–32). The patients in our study were not taking any systemic medication or undergoing phototherapy; therefore, excluding the possibility of such treatment influencing oxidative stress. Second, these patients had mild-to-moderate disease, according to the PASI. Only 4% of the patients without metabolic syndrome had PASI values of 10 or more; similar PASI values were measured in 8% of the patients with metabolic syndrome. The patients were followed up at outpatient clinics and could, therefore, be considered to be more stable than inpatients. Moreover, it is not possible to measure the possible calming effect of a blood drawn at outpatient clinics. For example, in a cohort study conducted in Sweden, the researchers reported an increased risk for cardiovascular mortality in relation to oxidative stress in psoriatic inpatients

but not outpatients (33). Third, oxidative damage triggered by inflammatory cytokines in psoriatic skin lesions may not be reflected in serum or plasma. As reported recently, although tissue malondialdehyde levels in psoriatic skin lesions were statistically higher in patients with psoriasis than in the control group, serum levels of this marker did not significantly differ between the two groups (34). Finally, several studies have suggested that some enzymes and molecules involved in antioxidant defence mechanisms against oxidative stress show a decrease in patients with psoriasis but that others increase or remain the same (35,36). In addition, the measurement of serum TAC is affected by the individual antioxidant components of serum, such as total-SH groups, ascorbic acid, uric acid, bilirubin, and vitamin E.

There are two notable limitations in our study. First, patients with plaque-type psoriasis under treatment were excluded due to the possible effects of systemic treatment or phototherapy on oxidant–antioxidant status and PON1 activities. Hence, patients with plaque-type psoriasis in the study groups exhibited only mild-to-moderate disease activity, as defined by the PASI score. Second, because the sample sizes of patient groups, according to the metabolic syndrome definitions of the NCEP ATP III and WHO, were relatively small, we could not evaluate patients with plaque-type psoriasis with metabolic syndrome according to these definitions. This situation might have had an effect on the findings. Elucidating this issue would require larger sample sizes.

Based on our data, serum paraoxonase activity of PON1 enzyme, independent of metabolic syndrome, may be a useful marker related to disease activity in patients with plaque-type psoriasis. Furthermore, it might be supposed that the total measurements of antioxidant capacity and oxidant status instead of measuring separately different antioxidant metabolites and oxidant species do not accurately reflect the serum oxidative status, even in patients with metabolic syndrome.

In conclusion, this cross-sectional study indicated decreased serum paraoxonase and arylesterase activities in patients with mild-to-moderate plaque-type psoriasis with or without metabolic syndrome, according to the IDF criteria. The observed decreases in the activities of these enzymes were not associated with serum TAC and TOS. Among the PON1 activities, only serum paraoxonase activity was associated with psoriatic activity evaluated by the PASI score, and the decrease in the activity of serum paraoxonase was independent of metabolic syndrome. Further studies involving patients with moderate-to-severe plaque-type psoriasis and metabolic syndrome according to definitions other than that of the IDF are required to elucidate the data in this study.

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