Psoriasis is a chronic recurrent inflammatory disease. Genetic and immunological factors are involved in development of psoriasis. Psoriasis is associated with numerous comorbidities including metabolic syndrome (MetS). Adipocytokines produced by white adipose tissue may be involved in the pathogenesis of psoriasis. Adipocytokines could serve as a missing link in the association between psoriasis and obesity/MetS. The most important adipokines include adiponectin, leptin and resistin. Adiponectin is expressed by adipocytes and has a high anti-inflammatory potential. Leptin is a protein produced in adipose tissue and is an important part in regulating energy metabolism. It has a pro-inflammatory effect. Polypeptide resistin is produced by macrophages and monocytes of the visceral adipose tissue. It was named for its ability to induce insulin resistence. Lipoprotein-associated phospholipase A2 (Lp-PLA2) is also product of macrophages, that can be served as a marker for cardiovascular risk. Increased smoking rates in patients with psoriasis is associated with their reduced quality of life. In addition, smoking of tobacco cigarettes is closely associated with MetS: smokers have an increased risk of MetS. Between psoriasis and smoking has also been demonstrated a direct link. Smoking is a well-recognized cause of elevated neutrophils and activates inflammatory biomarkers, including C-reactive protein (CRP). CRP is one of the most important indicator of inflammation. CRP levels are elevated in smokers and positively correlates with obesity.

The aim of present study was to evaluate chronic inflammation, MetS and incidence of smoking in psoriatic patients. We compared the inflammatory parameters – CRP, leptin, adiponectin, resistin and Lp-PLA2 in patients with psoriasis (PP) and in the control group (CG) with respect to presence of MetS and smoking.

Metabolic syndrome was diagnosed in 60.8 % of psoriatic patients and in 47.7 % subjects from the control group. In the study we confirmed earlier findings that psoriatic patients are more likely to smoke. In the PP group, 40.5 % of the patients smoked and in the control group 30.8 % of the subjects. PASI was higher in patients smokers than in non-smokers patients ($p<0.05$). We measured a statistically significantly higher diastolic blood pressure (DBP) in PP compared to CG ($p<0.05$). The link between psoriasis and obesity confirms a statistically significantly higher values of BMI in psoriatic patients compared to controls ($p<0.05$). Proinflammatory CRP was significantly higher in the patient group compared to the control group ($p<0.001$), and that independently related to smoking. In adiponectin, there was a significant interaction between the disease and MetS ($p<0.001$). Psoriasis had significant impact on levels of adipokines leptin ($p<0.01$) and resistin ($p<0.01$). Level of pro-atherogenic Lp-PLA2 was higher in psoriatic patients compared to controls ($p<0.001$). We measured significantly higher CRP ($p<0.01$), leptin ($p<0.01$), resistin ($p<0.05$) and Lp-PLA2 ($p<0.001$) in PP with MetS compared to CG with MetS. Levels of Lp-PLA2 ($p<0.001$) were also higher in the group of PP without MetS compared to CG without MetS. There was significant elevation of leptin ($p<0.05$) and lower adiponectin ($p<0.001$) in the group of PP with MetS compared to PP without MetS. Related to smoking habits, we detected a significantly higher leptin levels ($p<0.01$) in PP non-smokers compared to PP smokers. PASI ($p<0.05$) was significantly higher in PP smokers compared to PP non-smokers.

In conclusion, psoriasis is an inflammatory state that is certainly associated with obesity, regardless of smoking history. However, smoking makes the severity of psoriasis worse. It is important to treat psoriasis without delay and instruct patients that is necessary to quit smoking and improve their eating habits.