Summary

Metabolic syndrome is a chronic metabolic and systemic inflammatory disease that is associated with high morbidity and mortality. The increasing prevalence of metabolic syndrome highlights the need for a deeper understanding of the pathophysiological mechanisms and their interrelationship with common comorbidities, including psoriasis. By analysing changes in the levels of selected parameters of inflammation and cellular damage, the knowledge of the pathogenesis of the inflammatory process of both diseases and their combination can be expanded. The results may contribute to effective secondary prevention and primary care interventions and facilitate modification of therapies.

Data for the doctoral thesis were obtained from three separate studies, for which a set of parameters reflecting metabolic status, inflammatory processes, and cell damage (oxidative DNA/RNA damage, calprotectin, angiopoietin-like protein 8 /ANGPTL8/, clusterin, elafin, chromosomal aberrations, CRP, and vitamin D) were selected. The studies included groups of people with metabolic syndrome, persons with psoriasis, persons with psoriasis and metabolic syndrome, and healthy subjects. A total of 248 subjects participated in the study in three studies (122, 85 and 41 subjects). Laboratory determination of parameters was performed by ELISA, EIA, nephelometry, and microscopic analysis (chromosomal aberration test). The levels of the parameters were determined and compared between the groups. The analysis of relationships (potential dependencies) between selected parameters of groups of people and the analysis of relationships between metabolic syndrome and psoriasis were performed. The nature of inflammation in metabolic syndrome and psoriasis was compared and the level of genotoxic risk associated with chronic inflammation in people with metabolic syndrome and in persons with metabolic syndrome and psoriasis was evaluated.

Significantly higher levels of calprotectin, oxidative DNA/RNA damage, CRP, clusterin, and elafin were found in people with metabolic syndrome and in people with psoriasis (compared to healthy subjects). The levels of parameters in persons with psoriasis were higher than in persons with metabolic syndrome. A similar trend (increase in levels in both diseases with accentuation of the increase in their combination) was observed for the parameters of cellular damage (chromosomal aberrations).

The presented results confirmed that metabolic syndrome and its comorbidity psoriasis are closely associated with damaging inflammation. The combination of both diseases may further

increase the level of health risks by increasing the intensity of inflammation and the degree of genotoxic damage. I would like to emphasize that some of the selected parameters (presented in the thesis) have not yet received adequate attention in the study of the pathophysiology of metabolic syndrome and/or psoriasis. Thus, the presented data expand the knowledge of the pathogenesis of the inflammatory process of both diseases (and their combination) and demonstrate the associated cancer risk. From this perspective, these data are unique, underlining the importance of secondary prevention and primary care. Individuals with metabolic syndrome and psoriasis are at increased risk of health complications and need to be regularly monitored and therapeutic modification is necessary.

In ongoing research, we will focus on other functions (roles) of inflammatory process parameters in the pathophysiological mechanisms of common diseases and on the use of the results for the purpose of interventions in the areas of primary and secondary prevention and primary care.