

innovative strategies in the ongoing battle against acne, emphasizing a holistic approach to skin and gut health.

GUT MICROBIOTA AND PSORIASIS

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Introduction. Psoriasis is a chronic immune-mediated inflammatory hyperproliferative skin disease affecting approximately 2-3% of the population. Genetic factors, immune system disorders, and environmental factors are considered to be the most important in its etiology. However, it is in relation to psoriasis that the greatest amount of evidence has been accumulated regarding its relationship with digestive tract pathology, most often chronic intestinal diseases.

Materials and methods. Analysis of modern scientific research and literary sources in the field of bacteriology, clinical microbiology, pathophysiology and immunology regarding the study of the relationship between intestinal microflora and the pathogenesis of psoriasis.

Results and their discussion. It has been established that the prevalence of psoriasis in patients with Crohn's disease is significantly higher than in the population - 9.6%. Crohn's disease is a chronic progressive inflammatory disease that affects any part of the digestive tract. Crohn's disease, like psoriasis, has an autoimmune etiology.

Recently, data on the comorbidity of psoriasis and celiac disease (gluten enteropathy) have appeared. Celiac disease is a chronic intestinal disease of immune etiology, associated with a specific inflammatory reaction of the mucous membrane of the small intestine when consuming products containing cereal protein - gluten. An increase in the level of serological markers of celiac disease (antibodies to gliadin, immunoglobulin A) was noted in 14% of patients with psoriasis, in the control group - in 5%.

In addition, their values correlate with the severity of psoriasis. At the same time, in psoriasis, against the background of increased antibodies specific for celiac disease, histological markers of damage to the intestinal mucosa were not always detected. This may indicate a predisposition of some psoriasis patients to the development of gluten intolerance. Data were obtained indicating a decrease in the severity of clinical manifestations of psoriasis and laboratory markers of celiac disease when following a gluten-free diet.

Pathophysiological parallels in the development of psoriasis and chronic intestinal diseases are not limited to a tendency to immune autoaggression. Thus, complications of psoriasis in various diseases of the gastrointestinal tract, accompanied by damage to the intestinal wall, are associated with increased permeability of the

intestinal barrier. Congenital or acquired increased intestinal permeability is considered by some authors to be an important link in the pathogenesis of psoriasis. The intestine, like the skin, is a barrier between the external and internal environment of the body.

Increased intestinal permeability as a result of inflammatory processes or disturbances in the microbiocenosis leads to increased translocation of microbial metabolism products, microbial antigens, toxins into the internal environment of the body. As a result, excessive stimulation of the immune system and the development of local and systemic abnormal inflammatory and allergic reactions. These disorders are noted in both the small and large intestines.

Thus, the primary cause of persistent systemic inflammation in psoriasis may be intestinal pathology.

Disruption of the intestinal barrier function may be the result of a significant change in its microbiota. Thus, a decrease in the diversity of the intestinal microflora composition has been found in patients with psoriasis and psoriatic arthritis. This is also observed in chronic inflammatory bowel diseases.

It has also been proven that psoriasis is a chronic inflammatory skin lesion, in which specific immune cells (Th17) probably have a significant influence. This inflammation leads to uncontrolled proliferation of keratinocytes, which are renewed in 3 days instead of 28, and there are abnormalities in the cells themselves. Moreover, 22% of patients also have joint damage.

Since the gut microbiota appears to regulate the production and activity of these cells, it may be directly involved in the mechanisms of psoriasis, as it is in obesity and some inflammatory bowel diseases. These disorders share similar characteristics with psoriasis, for example, in terms of the type of immune response and the type of inflammatory molecules produced.

To confirm this hypothesis, a team of scientists analyzed the gut microbiota of approximately 30 volunteers, half of whom had psoriasis.

The gut microbiota of psoriasis patients is less diverse and has fewer *Coprococcus*, *Ruminococcus* and *Akkermansia muciniphila* are less represented in patients with psoriatic arthritis, as are patients with chronic inflammatory bowel diseases.

The main differences were related to two bacteria: *Clostridium citoniae*, which was more abundant in patients with psoriasis, and *Akkermansia muciniphila*, which was significantly less abundant. Scientists have also noted this in patients with obesity or chronic bowel disorders.

In healthy adults, the bacterium *Akkermansia muciniphila* is one of the most abundant in the large intestine, accounting for 3% to 5% of all microbial species in the gut microbiota. It may be involved in the barrier function of the gut and may serve as an indicator of individual health. Therefore, it seems that there is another common feature in the mechanisms of psoriasis development with diseases such as obesity or Crohn's disease, which may become a new area of research.

Conclusions. The current studies are trying to determine whether disturbances in the composition of the gut microbiota precede the onset of arthritis, which could help in screening patients at risk and help to understand the cause-and-effect relationship. According to a new study, the development of psoriasis is correlated with a significant decrease in the number of *Akkermansia muciniphila* bacteria in the gut microbiota. This study opens up new opportunities for studying this skin disease.

COTINUS COGGYGRIA SCOP.: A POTENTIAL SOURCE OF BIOLOGICALLY ACTIVE COMPOUNDS WITH ANTIMICROBIAL PROPERTIES AGAINST *ESCHERICHIA COLI* MDR STRAINS

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Introduction. *Cotinus coggygia* Scop. commercial ornamental plant with diverse properties such as: antioxidative, antibacterial, antifungal, antiviral, anticancer, antigenotoxic, hepatoprotective, and anti-inflammatory, belongs to the Anacardiaceae family. Depending on the plant's part, many compounds have been identified and isolated from *C. coggygia*. The active compounds of the plant demonstrated antimicrobial properties against a wide range of microorganisms including *Staphylococcus* spp., *P. aeruginosa*, *Bacillus subtilis*, *Klebsiella* spp., *Escherichia* spp., *Micrococcus*, yeast *Candida albicans* (Matic' et al., 2011). In the last decade, due to the growing selective pressure of antimicrobials excessive use, multidrug-resistant (MDR) strains such as *E. coli* ST131 and ST1193 has developed. MDR clones are linked with acquiring fluoroquinolone resistance, CTX-M β -lactamases, and carbapenemases. (Pitout et al., 2023; Valenza et al., 2019). The present study was designed to investigate antibacterial concentrations of *Cotinus coggygia* leaves ethanol extracts (90%) against *E. coli* MDR strains.

Materials and methods. Twenty *Escherichia coli* strains isolated from urine, infected wounds, bronchoalveolar lavage, feces, and vaginal secretion were used to measure the bacteriostatic (MIC) and bactericidal (MBC) concentrations of the samples used in the study. They included MDR (laboratory collection), non-MDR isolates, and a reference strain of *Escherichia coli* 25922 (ATCC). Microorganisms were identified using "ENTEROtest 23 in the laboratory of bacteriological research of IFNMU and MALDI-TOF mass spectrometry in the bacteriological laboratory of St. George's University of London. The isolated strains of *E. coli* are characterized by the following antibiotic resistance genotypes: extended-spectrum β -lactamases (amoxicillin MIC – 250.0 - 1000.0 μ g/ml), MLS-resistance (macrolides, lincosamides, streptogramin B) (erythromycin MIC – 250.0 - 2000.0 μ g/ml), TET genotype (tetracycline MIC – 250.0 - 1000.0 μ g/ml) and AmpC-resistance (cephalosporins), quinolones resistance phenotype (ofloxacin 50.0 – 100.0 μ g/ml).