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# **Psoriasis**

Psoriasis is a long-lasting autoimmune disease which is characterized by patches of abnormal skin.

These skin patches are typically red, itchy, and scaly. They may vary in severity from small and localized to complete body coverage.

Injury to the skin can trigger psoriatic skin changes at that spot, which is known as the Koebner phenomenon.

There are five main types of psoriasis: plaque, guttate, inverse, pustular, and erythrodermic.

Plaque psoriasis, also known as psoriasis Vulgaris, makes up about 90% of cases. It typically presents with red patches with white scales on top. Areas of the body most commonly affected are the back of the forearms, shins, around the navel, and the scalp.

Guttate psoriasis has drop-shaped lesions.

Pustular psoriasis presents with small non-infectious pus-filled blisters.

Inverse psoriasis forms red patches in skin folds.

Erythrodermic psoriasis occurs when the rash becomes very widespread, and can develop from any of the other types. Fingernails and toenails are affected in most people at some point in time. This may include pits in the nails or changes in nail color.

#### **Pathogenesis**

Psoriasis is generally thought to be a genetic disease which is triggered by environmental factors.

Ferroptosis and necroptosis are two recently identified forms of non-apoptotic cell death. Their dysregulation plays a critical role in the development and progression of Psoriasis (PsD) and Atherosclerosis (AS).

Fan et al. explored shared Ferroptosis and necroptosis-related genes and elucidates their molecular mechanisms in PsD and AS through the analysis of public databases.

Methods: Data sets for PsD (GSE30999) and AS (GSE28829) were retrieved from the GEO database. Differential gene expression (DEG) and weighted gene co-expression network analysis (WGCNA) were performed. Machine learning algorithms identified candidate biomarkers, whose diagnostic values were assessed using Receiver Operating Characteristic (ROC) curve analysis. Additionally, the expression levels of these biomarkers in cell models of AS and PsD were quantitatively measured using Western Blot (WB) and real-time quantitative PCR (RT-qPCR). Furthermore, CIBERSORT evaluated immune cell infiltration in PsD and AS tissues, highlighting the correlation between characteristic genes and immune cells. Predictive analysis for candidate drugs targeting characteristic genes was conducted using the DGIdb database, and an IncRNA-miRNA-mRNA network related to these genes was constructed.

They identified 44 differentially expressed ferroptosis-related genes (DE-FRGs) and 30 differentially expressed necroptosis-related genes (DE-NRGs). GO and KEGG enrichment analyses revealed significant enrichment of these genes in immune-related and inflammatory pathways, especially in NOD-like receptor and TNF signaling pathways. Two ferroptosis-related genes (NAMPT, ZFP36) and eight necroptosis-related genes (C7, CARD6, CASP1, CTSD, HMOX1, NOD2, PYCARD, TNFRSF21) showed high sensitivity and specificity in ROC curve analysis. These findings were corroborated in external validation datasets and cell models. Immune infiltration analysis revealed increased levels of T cells gamma delta, Macrophages M0, and Macrophages M2 in PsD and AS samples. Additionally, we identified 43 drugs targeting 5 characteristic genes. Notably, the XIST-miR-93-5p-ZFP36/HMOX1 and NEAT1-miR-93-5p-ZFP36/HMOX1 pathways have been identified as promising RNA regulatory pathways in AS and PsD.

Conclusion: The two ferroptosis-related genes (NAMPT, ZFP36) and eight necroptosis-related genes (C7, CARD6, CASP1, CTSD, HMOX1, NOD2, PYCARD, TNFRSF21) are potential key biomarkers for PsD and AS. These genes significantly influence the pathogenesis of PsD and AS by modulating macrophage activity, participating in immune regulation, and mediating inflammatory responses <sup>1)</sup>.

In twin studies, identical twins are three times more likely to both be affected compared to non-identical twins; this suggests that genetic factors predispose to psoriasis. Symptoms often worsen during winter and with certain medications such as beta blockers or NSAIDs. Infections and psychological stress may also play a role.

Psoriasis is not contagious. The underlying mechanism involves the immune system reacting to skin cells.

There is no cure for psoriasis. However, various treatments can help control the symptoms.

These treatments may include steroid creams, vitamin D3 cream, ultraviolet light, and immune system suppressing medications such as methotrexate.

About 75% of cases can be managed with creams alone.

The disease affects 2-4% of the population.

Men and women are affected with equal frequency.

The disease may begin at any age.

Psoriasis is associated with an increased risk of psoriatic arthritis, lymphomas, cardiovascular disease, Crohn's disease, and depression.

Psoriatic arthritis affects up to 30% of individuals with psoriasis.

Results suggest that psoriasis is associated with an increased risk of herpes zoster (HZ), which involves differences in sex and age. Although systemic therapy may have a major role in the risk of HZ, the intrinsic factors of psoriasis cannot be excluded <sup>2)</sup>.

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### **Diagnosis**

Diagnosis is typically based on the signs and symptoms.

Mean platelet volume (MPV) and red cell distribution width (RDW) could serve as promising predictive diagnostic biomarkers of psoriasis <sup>3)</sup>.

## **Differential diagnosis**

#### Ankylosing spondylitis

1)

Fan J, Zhu T, Tian X, Liu S, Zhang SL. Exploration of ferroptosis and necroptosis-related genes and potential molecular mechanisms in psoriasis and atherosclerosis. Front Immunol. 2024 Jul 12;15:1372303. doi: 10.3389/fimmu.2024.1372303. PMID: 39072329; PMCID: PMC11272566.

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3)

Yi P, Jiang J, Wang Z, Wang X, Zhao M, Wu H, Ding Y. Comparison of mean platelet volume (MPV) and red blood cell distribution width (RDW) between psoriasis patients and controls: A systematic review and meta-analysis. PLoS One. 2022 Feb 25;17(2):e0264504. doi: 10.1371/journal.pone.0264504. PMID: 35213665.

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