A Perspective on Psoriasis and Depression: Uncovering a Common Inflammatory Pathway

Psoríase e Depressão em Perspetiva: Uma Via Inflamatória Comum Ainda por Explorar

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Abstract:

Psoriasis and depression are very prevalent diseases that often coexist. In fact, the prevalence of depression in patients with psoriasis is higher than that observed in the general population and increases with disease severity. At the same time, it is now known that inflammation plays a central role in both diseases, with high levels of pro-inflammatory cytokines corroborating these assumptions. Assuming that dysregulation of the immune system may be a possible precipitating event for both conditions, therapeutic strategies have been highlighted for various inflammatory pathways, with promising effects on both diseases, including anti-TNF-α and anti-IL-17A therapies, which further corroborate the association between them.

Thus, with this perspective article, the authors intent to review not only the pathophysiological mechanisms behind these entities, focusing on the role of inflammation, but to understand the immune-mediated interaction that appears to be shared by them. The aim, therefore, is not only to provide greater clarification regarding existing knowledge, but also to highlight gaps that still exist, aspiring the development of large-scale studies that can fill them. Furthermore, we propose to expose the pathophysiological similarities of both diseases, aiming to transfer transversal knowledge between both, with an impact on the approach, therapy and prognosis.

Keywords: Depressive Disorder; Inflammation; Psoriasis.

Resumo:

A psoríase e a depressão são doenças muito prevalentes que frequentemente coexistem. Com efeito, a prevalência de depressão em doentes com psoríase é superior à observada na população em geral, com frequência tanto maior quanto mais grave o quadro psoriático. Paralelamente, sabe-se hoje que, em ambas as doenças, a inflamação apresenta um papel central, com níveis elevados de citocinas pró-inflamatórias a corroborar estas hipóteses. Tendo como premissa que a desregulação do sistema imunitário se assume como possível evento precipitante de ambas as condições, tem sido dado destaque às estratégias terapêuticas a várias vias inflamatórias, com efeitos promissores em ambas as doenças e de onde se realçam as terapias anti-TNF-α e anti-IL-17A, o que corrobora ainda mais a associação entre as mesmas.

Assim, o presente trabalho propõe-se, sob a forma de artigo de perspetiva, a rever não só os mecanismos fisiopatológicos por detrás destas entidades, com foco no papel da inflamação, mas a compreender a interação imunomediada que parece ser partilhada pelas mesmas. Objetiva-se, assim, não só o maior esclarecimento quanto ao conhecimento existente, como evidenciar lacunas ainda existentes, perspetivando-se a realização de estudos de larga escala que as consigam colmatar. Mais ainda, os autores propõem-se a expor as semelhanças fisiopatológicas de ambas as doenças, visando com isso transladar o conhecimento transversal entre ambas, com impacto na abordagem, terapêutica e prognóstico.

Palavras-chave: Inflamação; Perturbações Depressivas; Psoríase.

Introduction

Psoriasis is a chronic inflammatory skin disease with heterogeneous clinical manifestations and a prevalence ranging from approximately 0.5% to 4% in adult populations worldwide. On the other hand, depression is considered a public health problem, with significant associated morbidity and mortality and an estimated global prevalence of 12%. Epidemiological data indicate that its prevalence is higher in high-income countries compared with resource-limited settings (18% and 9%, respectively).[1] A growing body of evidence supports a bidirectional association between psoriasis and depression, suggesting shared biological and psychosocial mechanisms.[2] Despite extensive research, the precise factors that predispose certain individuals to depression and psoriasis remain not fully understood. Emerging scientific evidence indicates that inflammation may play a crucial role in the onset of these diseases, thereby providing a compelling rationale for this review. Through a critical evaluation of the current literature, we aim to highlight existing knowledge gaps and discuss potential implications for the development of targeted therapeutic strategies.

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Methods

A reproducible search strategy was implemented, as follows: PubMed and Web of Science electronic searches for peer-reviewed literature published between January 1, 2000, and August 31, 2025. The search was last updated on September 1, 2025. The search strings were as follows:

- "((psoriasis[MeSH Terms]) AND (depression[MeSH Terms])) AND (inflammation[MeSH Terms])"
- "((depressive disorder, major[MeSH Terms]) AND (fatty acids[MeSH Terms]))"
- "((antidepressant drugs[MeSH Terms]) AND (psoriasis[MeSH Terms]))"

Articles were considered eligible if they were: original research articles, reviews, clinical trials, cohort, case-control or cross-sectional studies; included human subjects or translational evidence directly relevant to human pathophysiology; and were published in English. Studies were excluded if they were published outside the predefined time frame.

STUDY SELECTION AND DATA SYNTHESIS:

CS and RR independently screened and selected studies, extracted key data using a standardized form, and synthesized findings thematically according to shared inflammatory pathways and clinical implications.

Discussion

Although the association between psoriasis and depression has been described in previous reviews, the present perspective seeks to advance the field by emphasizing the shared inflammatory pathways that may operate independently of psychosocial burden and by exploring their translational relevance for therapeutic decision-making.

EPIDEMIOLOGICAL ASSOCIATION BETWEEN PSORIASIS AND DEPRESSION

The prevalence of major depression in psoriatic patients is 12%-13%, reaching as far as 28%-62%, depending on screening methods.[3,4] Interestingly, a cohort study focused on the incidence of psychiatric pathology in patients with psoriasis determined that, for depression, there is a higher adjusted hazard ratio (HR) in severe psoriasis than in mild psoriasis [1.72 and 1.38, respectively].[5,6] Regarding psoriatic arthritis, depression is estimated to be present in around 20%-40%.[7]

Patients with psoriasis have an increased risk of suicide compared to the general population, with an odds ratio (OR) of 1.94. However, the diagnosis of depression in individuals with psoriasis remains insufficient.[8]

Taken together, available epidemiological data support a robust association between psoriasis and depression. However, the relative contribution of shared biological mechanisms—particularly inflammation—versus psychosocial determinants remains insufficiently characterized.

PATHOPHYSIOLOGICAL INTERSECTIONS BETWEEN DEPRESSION AND PSORIASIS

1. Proposed pathophysiological bases

Although social stigma related to the visible skin lesions caused by psoriasis is a known risk factor for depression, existing evidence suggests that an independent, overlapping biological mechanism underlies the interaction between these two conditions.[9] Fig. 1 is intended as

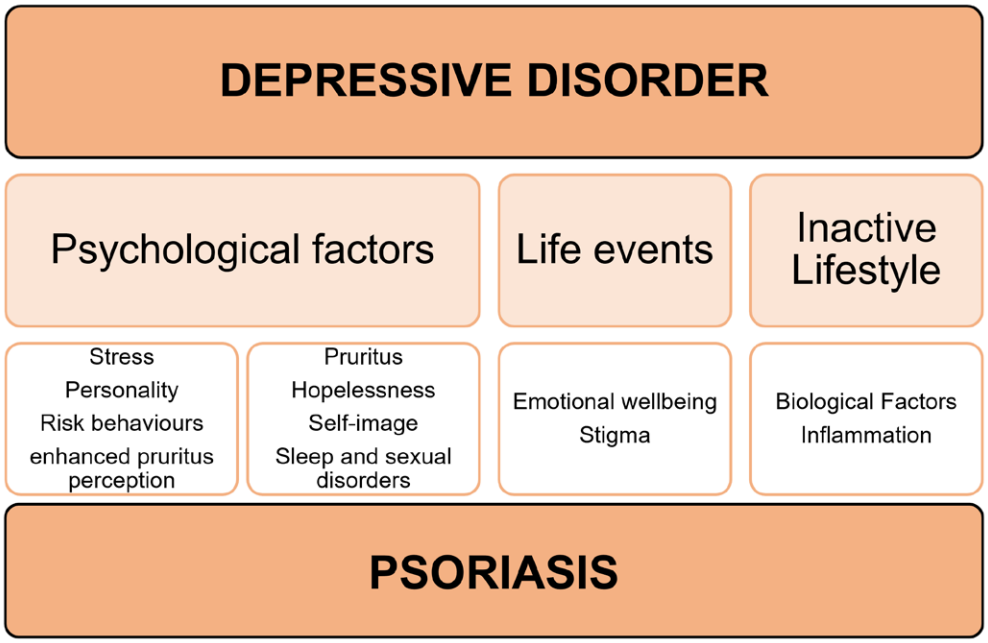

Figure 1: Psoriasis and depression interaction - pathophysiological mechanisms.

a conceptual summary integrating psychosocial and immunological mechanisms discussed throughout the text, rather than as an exhaustive representation of all molecular pathways involved. Accumulating evidence suggests that the interaction between psoriasis and depression is underpinned by shared biological mechanisms that extend beyond psychosocial factors. In particular, both conditions are characterized by a systemic pro-inflammatory state, with elevated levels of key inflammatory mediators, including interleukins (IL) and C-reactive protein (CRP).[3]

For years, most of the theories addressed the role of dysfunctional neurotransmitters, such as monoamines, gamma-aminobutyric acid (GABA), or glutamate in the central nervous system (CNS).[3] Another proposed mechanism involves the overstimulation of corticotropin-releasing hormone (CRH), leading to elevated cortisol levels. This condition manifests depressive symptoms.[6] Immune–brain interactions contribute to depression, as neuroimaging studies demonstrate increased microglial activation and neuroinflammation correlating with symptom severity.[6]

The proposed theories state that in response to stress, the sympathetic nervous system induces myeloid cell proliferation through the release of amines, which leads to an inflammatory state amplified by corticoid resistance.[4]

Proposed mechanisms include increased blood–brain barrier permeability facilitating cytokine entry, cytokine signalling by the blood–brain barrier endothelium, and vagal transmission of peripheral inflammatory signals to the central nervous system.[3] Fig. 2 highlights the principal routes through which peripheral inflammation may influence central nervous system function, providing a visual synthesis of mechanisms described in multiple sections of the manuscript.

Chronic stress is proposed to sustain innate pro-inflammatory cytokine activation through bidirectional neuro–immune signalling, thereby contributing to the onset or exacerbation of psoriatic lesions.

2. Effect of interleukins on the association between psoriasis and depression

The association between depression and inflammation is related first to the serum cytokine levels increased in depressed patients, including key pro-inflammatory mediators such as TNF-α, IL-6, IL-17, and CRP, which are central to both psoriatic inflammation and neuroimmune signaling.[3] Corroborating these findings, an increase in pro-inflammatory cytokines has also been described in around 30% of patients with depression, compared to a healthy population, with IL-1ß, IL-6, tumour necrosis factor (TNF)-α, and CRP standing.[4]

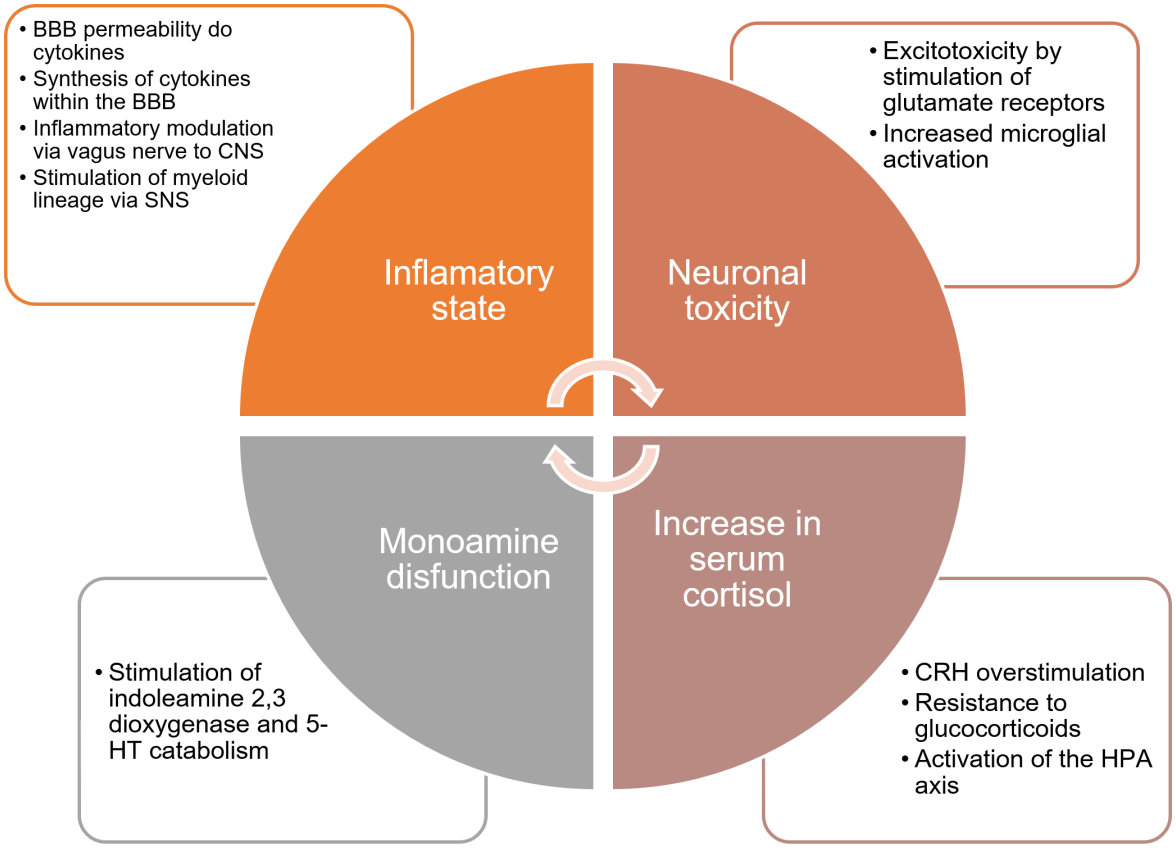

Figure 2: Inflammation-depression interaction.
5-HT - 5-hydroxytryptamine; BHE - blood-brain barrier; CRH - corticotrophic hormone; GC - glucocorticoids; HPA - hypothalamus-pituitary-adrenal.

Accordingly, the association between inflammatory markers and the induction or maintenance of depressive states is corroborated by the depressive state known to follow interferon (INF) therapy.[3]

Additionally, the correlation between IL-18 levels and the severity of depressive symptoms in psoriatic patients raises the hypothesis of possible interference of cytokines in serotonin synthesis.[10] Indeed, the proposed mechanism suggests that the cellular release of INF-α in psoriasis induces the synthesis of IL-6, IL-1ß, and TNF-α, which, by increasing the activity of indoleamine 2,3 dioxygenase, interferes with serotonergic metabolism, leading to a reduction in brain serotonin levels, thus precipitating a depressive mood.[11]

Furthermore, indoleamine 2,3 dioxygenase metabolizes tryptophan, whose catabolites can alter the function of CRH and independently induce depressive symptoms.[6] These cytokines (IL-1, IL-6, TNF-α, and IFN- α), per se, increase CRH, ACTH, and cortisol levels while reducing the expression and activity of glucocorticoid receptors, thus inhibiting negative feedback from the HPA axis and further increasing cortisol synthesis, contributing to depressive symptoms through this additional pathway.

Evidence has established an association between the administration of Th17 cells and an increased susceptibility to depression, suggesting that the Th17 axis may be integral to neuro-immune interactions. Further investigation has revealed that administering IL-17A leads to enhanced NFκB/p38MAPK signalling and the activation of inflammatory mediators in various brain regions, particularly the hippocampus and prefrontal cortex. This cascade of events results in the manifestation of depression-like behaviours. Supporting these findings, the use of inhibitors targeting NFκB, p38MAPK, and anti-IL-17A antibodies has been shown to mitigate these depressive behaviours. This knowledge is particularly relevant since it is in the skin affected by psoriatic lesions that Th17 cells and IL-17A are characteristically increased. Thus, it is assumed that the IL-17A/IL-17RA pathway, activated in the systemic inflammatory process in psoriasis, contributes to the depressive state by the central effects explained.[7]

Collectively, these findings suggest that the Th17/IL-17 axis may represent a critical biological bridge between cutaneous inflammation and central nervous system dysfunction, rather than a disease-specific pathway limited to the skin. If shared inflammatory pathways link psoriasis to depression, therapeutic interventions targeting these pathways may offer a natural experiment to test this hypothesis.

3. Antidepressant effect of anti-inflammatory drugs

Beyond their dermatological efficacy, several immunomodulatory therapies used in psoriasis offer a unique opportunity to explore the causal role of inflammation in depressive symptoms. Although the evidence is still relatively scarce, studies have been carried out using psychometric scales of quality of life in general and depressive symptoms in particular to assess the impact of these therapies. These scales include the Dermatology Quality of Life Index (DLQI), HADS, Beck Depression Inventory (BDI), Hamilton Assessment of Depression Scale (HAM-D), and Zung Self-Rating Depression Scale (SDS). Of these studies, the main drugs that have shown this benefit on depressive symptoms, to date, are etanercept, adalimumab, infliximab, brodalimab, ustekinumab, and secukinumab.6 Importantly, the convergence of antidepressant effects across distinct immunological targets suggests that mood improvement may be linked to global inflammatory burden reduction rather than to blockade of a single cytokine pathway.

Concerning anti-TNF-α therapies, some have a potential antidepressant effect, in particular etanercept, adalimumab, and infliximab. In fact, in a retrospective cohort study of 980 patients with psoriasis and/or psoriatic arthritis, there was a 47% reduction in initially reported depressive symptoms and insomnia in patients treated with anti-TNF-α drugs over two years.[12] The positive impact of anti-TNF-α drugs on mood and cognition seems to be greater the higher the inflammatory state. Although they are molecules incapable of crossing the blood-brain barrier (BBB), they can produce changes in cytokine expression by acting on the HPA axis, thus improving depressive symptoms independently of their action on psoriatic lesions.[4] This conclusion was corroborated by a placebo-controlled study, which verified this association with etanercept.[13]

Concerning ustekinumab, an anti-IL-12/23 drug, there is also evidence that, by mechanisms that are not yet fully understood, it can improve depressive and anxiety symptoms in patients with moderate-severe psoriasis.[14]

Ixekizumab, an anti-IL17A monoclonal antibody, showed antidepressant effects in psoriasis patients, reducing depressive symptoms and achieving a 40% remission rate at 12 weeks, while also significantly lowering systemic inflammation (high-sensitivity CRP).[15,16]

A prospective study found that fumaric acid, an antipsoriatic agent, improved both psoriasis and associated depressive symptoms. These results are suggested to be not only due to the reduction of psoriatic lesions and their stigma but also to the reduction of pro-inflammatory cytokines by fumaric acid, which seems to have an independent positive effect on depression. Importantly, in experimental in vitro models, this drug has also been shown to inhibit microglial and astrocytic inflammation by suppressing the synthesis of pro-inflammatory cytokines, and it is proposed that similar mechanisms may underlie its effect in humans.[17]

Another line of research has focused on the immunomodulatory role of fatty acids, whose role in the pathophysiology of depression has already been detailed. Of these, PUFA has the greatest immunomodulatory potential, the most potent being EPA and docosahexaenoic acid (DHA). Although still few in number and small in scale, some clinical trials in animals and humans have shown an anti-inflammatory effect of these

fats.[18] In particular, serum PUFA levels seem to predict the response of proinflammatory cytokines to psychosocial stress, corroborating that a change in the omega-6/omega-3 PUFA ratio in major depression may be related to an increase in the production of these cytokines. In this context and importantly, EPA and DHA supplementation has already been shown to be beneficial in the treatment of other diseases, such as rheumatoid arthritis, inflammatory bowel disease, and asthma, by reducing cytokine levels and thereby reducing the systemic inflammation that underlies the pathophysiology of these entities.[19]

4. Evidence of the anti-psoriatic effect of antidepressant drugs

If, on the one hand, anti-psoriatic therapies may help treat depression, the opposite also seems to be true. In this respect, several studies have suggested a relevant role for serotonin in the pathophysiology of psoriasis, as already mentioned above, especially regarding its risk of depression.

The expression of the serotonergic 5-HT receptor is significantly elevated in the epithelium, the target territory of psoriasis. At the same time, the SERT serotonin receptor is also over-expressed in this condition, mainly in dendritic cells (Langerhans cells in the epidermis), mast cells, and lymphocytes. Also, the 5-HT1AR and 5-HT2AR receptors are differentially expressed in psoriasis, with greater expression of the latter in psoriatic skin. In parallel, the 5-HT3R and 5-HT7R receptors are involved in pruritus, a mechanism mediated by serotoninergic action, which appears to play a role in psoriasis. Finally, the role of serotonin in skin inflammation also involves stimulation of keratinocyte proliferation, with a direct influence on CD4+ T lymphocytes and indirectly, via 5-HT2AR-mediated stimulation of CD8+ T cells to produce IL-16, which attracts CD4+ T cells and perpetuates the inflammatory cycle.[20] Even further, antidepressants have been shown to have beneficial effects not only on mood but also, independently, on the resolution of skin lesions. An original study found that escitalopram, in combination with anti-TNF-α drugs, resulted in a significant reduction in psoriatic lesions according to the PASI scale, as well as itching complaints, as compared to anti-TNF-α alone.[21] Similarly, moclobemide (iMAO) has also been evaluated for its potential anti-psoriatic effect, with results pointing to a beneficial and independent effect in the treatment of lesions, also according to the PASI scale.[21]

Hence, antidepressants may improve both depressive symptoms and psoriatic lesions via downregulation of systemic pro-inflammatory cytokines, resulting in lower PASI scores and reduced need for systemic therapy.[20]

Conclusion

Considering the premises explored above, epidemiological evidence favours the hypothesis of an etiological association between psoriasis and depression. Several mechanisms have been proposed to explain the pathophysiological bases that interconnect these pathologies.

Among these, it is important to highlight the inflammatory pathways resulting from the psoriatic process, which exert a pro-depressive effect independent of the psychosocial burden of the disease derived from the stigma secondary to the skin lesions. As studies indicate, this inflammatory process applies its effect on neuronal pathways, namely through dysfunction in serotonin metabolism, induction of a state of hypercortisolaemia, and neuronal toxicity.

At the same time, there is an association between increased levels of various cytokines, psoriasis, and depression, favouring the hypothesis that cytokines play a role in these entities. Of these interleukins, TNF-α and INF-α stand out as playing a central role in neuronal dysfunction, with IL-17, IL-16, IL-1, and IL-6 also being highlighted.

These findings are corroborated by the antidepressant effects that some biological therapies targeting cytokine pathways used in psoriasis have already been shown to have. Importantly, this effect was shown to be independent of the improvement in a depressive state that directly resulted from the improvement in psoriatic lesions, and positive effects on mood and cognition were identified with these therapies. In addition, antidepressant drugs have been shown to benefit dermatological manifestations of psoriasis, also through immune-mediated pathways.

Despite growing evidence supporting an inflammatory link between psoriasis and depression, significant gaps persist. Prospective studies specifically designed to evaluate depressive outcomes in patients receiving immunomodulatory therapies are lacking, as are trials assessing the impact of antidepressant treatment on psoriatic disease activity. Future research should prioritize integrated dermatological–psychiatric study designs, standardized screening for depressive symptoms in psoriasis cohorts, and biomarker-driven approaches to identify patients most likely to benefit from immune-targeted interventions. ■

Contributorship Statement

CS - Literature review, writing of the manuscript.
RR - Writing and critical review of the manuscript.
PCP, AG - Critical review of the manuscript.
All authors approved the final version to be published.

Declaração de Contribuição

CS – Revisão de literatura e redação do manuscrito.
RR – Redação e revisão crítica do manuscrito.
PCP, AG – Revisão crítica do manuscrito.
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REFERENCES

1. Michalek IM, Loring B, John SM. A systematic review of worldwide epidemiology of psoriasis. J Eur Acad Dermatol Venereol. 2017;31:205-12. doi:10.1111/jdv.13854
2. Kessler RC, Ormel J, Petukhova M, McLaughlin KA, Green JG, Russo LJ, et al. Development of Lifetime Comorbidity in the World Health Organization World Mental Health Surveys. Arch Gen Psychiatry. 2011;68:90-100. doi: 10.1001/archgenpsychiatry.2010.180.
3. Hölsken S, Krefting F, Schedlowski M, Sondermann W. Common Fundamentals of Psoriasis and Depression. Acta Dermatol Venereol. 2021;101:1-9. doi:10.2340/ACTADV.V101.565.
4. González-Parra S, Daudén E. Psoriasis and Depression: The Role of Inflammation. Actas Dermosifiliogr. 2019;110:12-9. doi:10.1016/j.ad.2018.05.009
5. Kurd SK, Troxel AB, Crits-Christoph P, Gelfand JM. The risk of depression, anxiety, and suicidality in patients with psoriasis: A population-based cohort study. Arch Dermatol. 2010;146:891-5. doi:10.1001/archdermatol.2010.186
6. Patel N, Nadkarni A, Cardwell LA, Vera N, Frey C, Patel N, et al. Psoriasis, Depression, and Inflammatory Overlap: A Review. Am J Clin Dermatol. 2017;18:613-20. doi:10.1007/s40257-017-0279-8
7. De Lorenzis E, Natalello G, Bruno D, Tanti G, Magurano MR, Lucchetti D, et al. Psoriatic arthritis and depressive symptoms: does systemic inflammation play a role? Clin Rheumatol. 2021;40:1893-902. doi:10.1007/s10067-020-05417-5
8. Dalgard FJ, Gieler U, Tomas-Aragones L, Lien L, Poot F, Jemec GB, et al. The Psychological Burden of Skin Diseases: A Cross-Sectional Multicenter Study among Dermatological Out-Patients in 13 European Countries. J Invest Dermatol. 2015;135:984-91. doi:10.1038/jid.2014.530
9. Łakuta P, Marcinkiewicz K, Bergler-Czop B, Brzezinska-Wcisło L. How does stigma affect people with psoriasis? Postepy Dermatol Alergol. 2017;34:36-41. doi:10.5114/pdia.2016.62286
10. Pietrzak D, Pietrzak A, Grywalska E, Kicinski P, Rolinski J, Donica H, et al. Serum concentrations of interleukin 18 and 25-hydroxyvitamin D3 correlate with depression severity in men with psoriasis. PLoS One. 2018;13:e0201589. doi:10.1371/journal.pone.0201589
11. Berthold-Losleben M, Himmerich H. The TNF-System: Functional Aspects in Depression, Narcolepsy and Psychopharmacology. Curr Neuropharmacol. 2008;6:193-202. doi: 10.2174/157015908785777238.
12. Wu CY, Chang YT, Juan CK, Shen JL, Lin YP, Shieh JJ, et al. Depression and insomnia in patients with psoriasis and psoriatic arthritis taking tumor necrosis factor antagonists. Medicine. 2016;95:e3816. doi: 10.1097/MD.0000000000003816.
13. Tyring S, Gottlieb A, Papp K, Gordon K, Leonardi C, Wang A, et al. Etanercept and clinical outcomes, fatigue, and depression in psoriasis: double-blind placebo-controlled randomised phase III trial. Lancet. 2006;367:29-35. doi: 10.1016/S0140-6736(05)67763-X.
14. Langley RG, Feldman SR, Han C, Schenkel B, Szapary P, Hsu MC, et al. Ustekinumab significantly improves symptoms of anxiety, depression, and skin-related quality of life in patients with moderate-to-severe psoriasis: Results from a randomized, double-blind, placebo-controlled phase III trial. J Am Acad Dermatol. 2010;63:457-65. doi:10.1016/j.jaad.2009.09.014
15. Griffiths CE, Fava M, Miller AH, Russell J, Ball SG, Xu W, et al. Impact of ixekizumab treatment on depressive symptoms and systemic inflammation in patients with moderate-to-severe psoriasis: an integrated analysis of three phase 3 clinical studies. Psychother Psychosom. 2017;86:260-7. doi:10.1159/000479163
16. Danesh MJ, Kimball AB. Brodalumab and suicidal ideation in the context of a recent economic crisis in the United States. J Am Acad Dermatol. 2016;74:190-2. doi:10.1016/j.jaad.2015.08.057
17. Schmieder A, Poppe M, Hametner C, Meyer-Schraml H, Schaarschmidt ML, Findeisen P, et al. Impact of fumaric acid esters on cardiovascular risk factors and depression in psoriasis: a prospective pilot study. Arch Dermatol Res. 2015;307:413-24. doi:10.1007/s00403-015-1541-7
18. Maes M, Christophe A, Bosmans E, Lin A, Neels H. In humans, serum polyunsaturated fatty acid levels predict the response of proinflammatory cytokines to psychologic stress. Biol Psychiatry. 2000;47:910-20. doi: 10.1016/s0006-3223(99)00268-1.
19. Mocking RJT, Harmsen I, Assies J, Koeter MW, Ruhé HG, Schene AH. Meta-analysis and meta-regression of omega-3 polyunsaturated fatty acid supplementation for major depressive disorder. Transl Psychiatry. 2016;6. doi:10.1038/TP.2016.29
20. Martins AM, Ascenso A, Ribeiro HM, Marto J. Current and future therapies for psoriasis with a focus on serotonergic drugs. Mol Neurobiol. 2020;57:2391-419. doi:10.1007/s12035-020-01889-3
21. Derme AM, Zanieri F, Campolmi E, Santosuosso U, Betti S, Agnoletti AF, et al. Therapeutic implications of adding the psychotropic drug escitalopram in the treatment of patients suffering from moderate-severe psoriasis and psychiatric comorbidity: A retrospective study. J Eur Acad Dermatol Venereol. 2014;28:246-9. doi:10.1111/j.1468-3083.2012.04690.x