Psoriasis: Targets and Therapy

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ORIGINAL RESEARCH

Neurofunctional Reversibility in Psoriasis Vulgaris After IL-17A Inhibition: A Resting-State fMRI and Neurocognitive Analysis

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Purpose: Interleukin-17A (IL-17A) drives psoriasis and central nervous system neuroinflammation, but clinical research on whether IL-17A-targeted biotherapy modulates brain activity to improve neuropsychiatric outcomes in psoriasis is lacking. This study aims to investigate brain functional changes, cognitive impairment, and the effects of IL-17A monoclonal antibody therapy in psoriasis vulgaris.

Patients and methods: Regular secukinumab treatment. Meanwhile, 20 healthy controls (HCs) matched in age and gender were enrolled. The patients underwent functional magnetic resonance imaging (fMRI) before treatment and 48 weeks after treatment. The healthy controls also had fMRI scans. It assessed clinical data, cognition/neuropsych status (MoCA, SDS, SAS), disease severity/quality of Life (PASI/DLQI), and brain function via rs-fMRI (ALFF/ReHo).

Results: Compared to healthy controls, the psoriasis vulgaris patients showed increased ALFF in the frontal lobe, as well as increased frontal ReHo. The treatment group showed signal recovery in some brain regions. Patients before treatment had lower MoCA scores vs controls ($P < 0.001$)) and higher SAS/SDS scores (SAS, $P < 0.0001$; SDS, $P < 0.05$). Patients after treatment showed higher MoCA scores vs before treated ($P < 0.05$), similar to controls, with lower SAS scores ($P < 0.0001$) and reduced PASI/DLQI ($P < 0.0001$).

Conclusion: Psoriasis is associated with brain dysfunction and neuropsychiatric symptoms. IL-17A antibody therapy improves skin symptoms, restores brain function, and alleviates neuropsychiatric issues vs untreated patients, supporting multidimensional treatment.

Keywords: psoriasis vulgaris, rs-fMRI, brain function, cognitive impairment, IL-17A monoclonal antibody

Introduction

Psoriasis vulgaris (PV) is a chronic, immune-mediated inflammatory skin disease characterized by aberrant activation of the IL-23/IL-17 axis, with cutaneous manifestations often reflecting deeper systemic immune dysregulation.[1] Once considered a condition limited to the skin and joints, PV is now widely recognized as a systemic disease associated with a broad spectrum of comorbidities—including metabolic syndrome, cardiovascular disease, and increasingly, neuropsychiatric dysfunction.[2,3] Recent translational and clinical evidence suggests that chronic peripheral inflammation may exert pathogenic effects on the central nervous system (CNS), contributing to cognitive deficits, mood disorders, and alterations in brain functional connectivity among patients with psoriasis.[4]

Resting-state functional magnetic resonance imaging (rs-fMRI), through indices such as amplitude of low-frequency fluctuations (ALFF) and regional homogeneity (ReHo), has enabled noninvasive quantification of spontaneous neuronal activity and functional coherence in the brain.[5] These neuroimaging biomarkers have been instrumental in identifying altered brain activity patterns in patients with neuropsychiatric conditions, and have recently been particularly applied in cognitive neuroscience research.[6] Emerging studies have reported abnormalities in the frontal cortex, limbic system, and

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default mode network among individuals with psoriasis, implicating inflammation-driven neurofunctional changes as a plausible mechanism underlying the cognitive and affective disturbances observed in this population.[4]

The IL-17A cytokine plays a central role in psoriasis pathogenesis and has been implicated in neuroinflammatory cascades across various CNS disorders.[7–9] Preclinical models have demonstrated that elevated IL-17A levels disrupt the blood-brain barrier, activate microglia, and impair synaptic plasticity, leading to cognitive dysfunction.[10] However, the extent to which IL-17A-targeted biologic therapy can modulate CNS activity and improve neuropsychiatric outcomes in patients with PV remains largely unexplored in the clinical setting.

In this context, the present study aims to systematically evaluate brain function and neuropsychiatric status in patients with moderate-to-severe PV, before and after long-term treatment with the IL-17A monoclonal antibody secukinumab. By integrating cognitive assessments, emotional symptom scales, and rs-fMRI imaging biomarkers, this study seeks to elucidate the CNS effects of systemic IL-17A inhibition and to provide a more comprehensive understanding of the neuroimmunological dimensions of psoriasis. In doing so, we aim to support a more holistic paradigm of PV management that addresses both cutaneous and neuropsychiatric disease burdens.

Methods

Study Population

This hospital-based controlled trial recruited a total of 40 participants. We enrolled 20 patients with moderate-to-severe PV who had not received systematic treatment within 8 weeks. These patients were given regular secukinumab treatment. Specific treatment regimen: The initial dose was 300 mg administered via subcutaneous injection; subsequent doses of 300 mg were given at Weeks 2, 4, 6, and 8, followed by maintenance treatment with 300 mg every 4 weeks thereafter, for a total duration of 48 weeks. Functional MRI (fMRI) examinations were performed before treatment and 48 weeks after treatment, respectively. In addition, 20 healthy controls (HCs group) matched in age and gender were recruited, and they also underwent fMRI examinations. The project started in May 2024 and ended in June 2025.

Inclusion criteria were:

(1) diagnosis of PV according to established clinical guidelines;
(2) PASI score ≥3;
(3) Age 18–60 years;
(4) No use of systemic corticosteroids, immunosuppressants, or retinoids within the past 8 weeks;
(5) No topical therapy within 2 weeks;
(6) No recent phototherapy;
(7) Provision of informed consent.

Exclusion criteria included:

(1) Immunocompromised status;
(2) Pregnancy or lactation;
(3) Presence of severe systemic diseases (eg, cardiovascular, hepatic, renal);
(4) Active infections or active neurological/systemic infections;
(5) Left-handedness;
(6) Contraindications to MRI (eg, metal implants, claustrophobia);
(7) Presence of other dermatologic disorders;
(8) Use of neuropsychiatric medications within the past 12 weeks;
(9) History of sleep disorders, smoking, or alcohol abuse that could affect cognitive function;
(10) Presence of suicidal tendency or unstable psychiatric conditions;
(11) Poor compliance or inability to complete assessments.

The study protocol was approved by the Ethics Committee of the Affiliated Hospital of Shandong Second Medical University (approval number: wyfy-2024-ky-257) and conducted in accordance with the Declaration of Helsinki.

Clinical Data Collection

Demographic, clinical, laboratory, and treatment data were collected using standardized forms. PV diagnosis was confirmed by clinical presentation and/or histopathological examination. Disease severity was assessed using the Psoriasis Area and Severity Index (PASI).

Cognitive and Neuropsychiatric Assessments

Participants' cognitive status was evaluated using the Montreal Cognitive Assessment (MoCA). Neuropsychiatric status was assessed with the Self-Rating Depression Scale (SDS), the Self-Rating Anxiety Scale (SAS), and the Dermatology Life Quality Index (DLQI). All assessments were administered on the same day as the rs-fMRI scan by trained clinicians blinded to group allocation.

Resting-State fMRI Acquisition

Imaging was performed using a Philips Ingenia Elition S 3.0T MRI scanner. High-resolution T1-weighted structural images were acquired using a 3D fast spoiled gradient echo (FSPGR) sequence in the sagittal plane with the following parameters: field of view (FOV) = 240×240 mm^2, flip angle = 8°, matrix = 240 × 240, repetition time (TR) = 6.6 ms, echo time (TE) = 3.0 ms, slice thickness = 1.0 mm, number of slices = 360.

Resting-state BOLD-fMRI data were collected using a single-shot gradient-echo echo planar imaging (EPI) sequence with 240 time points: TR = 2000 ms, TE = 30 ms, slice thickness = 3.5 mm, matrix = 64 × 63, 30 slices. Participants were instructed to relax, keep their eyes closed, remain awake, and avoid deliberate cognitive activity during the scan.

Rs-fMRI Preprocessing

Preprocessing was conducted using the DPABI toolbox based on SPM12.[11] Steps included:

(1) Removal of the first 10 volumes to ensure magnetization equilibrium,
(2) Slice-timing correction,
(3) Realignment for head motion correction,
(4) Normalization to the Montreal Neurological Institute (MNI) template with resampling to 3×3×3 mm^3 voxels,
(5) Regression of nuisance signals (Friston 24-parameter motion, global signal, white matter, and cerebrospinal fluid),
(6) Linear detrending, and
(7) Temporal band-pass filtering (0.01–0.08 Hz).

Participants exhibiting head motion >3 mm or rotation >3° were excluded.

ALFF Analysis

Preprocessed time series were smoothed using a 4 mm full-width at half maximum (FWHM) Gaussian kernel and transformed into the frequency domain via fast Fourier transform. The ALFF was calculated as the square root of the power spectrum within the 0.01–0.08 Hz range. Individual ALFF maps were normalized by z-transformation (subtracting the global mean and dividing by the standard deviation).

ReHo Analysis

ReHo was calculated using Kendall's coefficient of concordance (KCC) among a voxel and its 26 neighboring voxels. ReHo maps were smoothed with a 4 mm FWHM Gaussian kernel and standardized using z-transformation as described for ALFF.

Statistical Analysis

Statistical Package for the Social Sciences (SPSS) version 26.0 and DPABI were used for statistical analyses. Between-group comparisons of continuous variables were conducted using independent-sample *t*-tests or Mann–Whitney *U*-tests, as appropriate. Categorical data were analyzed using χ^2-tests. Group differences in ALFF and ReHo maps were evaluated using two-sample *t*-tests (voxel-level threshold: $P < 0.001$, cluster-level family-wise error corrected: $P_{FWE} < 0.05$). Correlation analyses between neuroimaging metrics and clinical/neuropsychiatric scores were performed using Pearson or Spearman correlation coefficients based on data distribution.

Results

Clinical Assessments

Participant demographics and clinical characteristics are summarized in Table 1 and Figure 1. The previous treatment history of the 20 patients was as follows: 12 patients had used topical medications, including corticosteroid ointments and vitamin D3 derivatives; 8 patients had received narrowband ultraviolet B phototherapy (NB-UVB); 5 patients had received methotrexate and/or acitretin. 48 weeks after receiving secukinumab treatment, the PASI scores and DLQI scores of PV patients decreased significantly (both $P < 0.0001$). Cognitive function, as measured by the MoCA, was significantly lower in the PV patients compared to HCs ($P < 0.001$). Notably, PV patients exhibited improved MoCA scores after treatment ($P < 0.05$), with no significant difference from healthy controls. Regarding neuropsychiatric status, the pre-treatment SAS and SDS scores of PV patients were higher than those of healthy controls (SAS, $P < 0.0001$; SDS, $P < 0.05$). After treatment, the SAS scores decreased significantly ($P < 0.0001$), while the SDS scores did not show a statistically significant downward trend.

ALFF Differences

Compared with the HCs group, the ALFF values in the frontal lobe of PV patients were significantly elevated. After receiving secukinumab treatment, the frontal lobe ALFF values of these patients decreased significantly and returned to a level comparable to the ALFF values of the control group. The above results are visually presented in Figure 2, with relevant statistical data detailed in Table 2. Clinical indicators did not demonstrate a significant correlation with ALFF values.

ReHo Differences

Compared with the HCs group, the ReHo of the frontal cortex in PV patients was increased. After receiving secukinumab treatment, the ReHo of the frontal region in these patients decreased significantly and returned to a level comparable to the ReHo of the control group. The above results are visually presented in Figure 3, with relevant statistical data detailed in Table 3. The ReHo value of the frontal lobe was significantly positively correlated with the PASI score ($R = 0.541$, $P = 0.014$; Figure 4). Other clinical scores did not demonstrate a significant correlation with ReHo values.

Table 1 Demographic, Clinical and Cognitive Testing Scores Information of All Subjects

Characteristics	PV (n=20)	Treated	HCs (n=20)	*P* (PV vs Treated)	*P* (PV vs HCs)	*P* (Treated vs HCs)
Age (years)	33 (25.5–47.5)		33.5 (23.5–50.0)	0.96	0.92	0.89
Gender (n, %)				1	1	1
Male	12 (60.0%)		12 (60.0%)			
Female	8 (40.0%)		8 (40.0%)			
Disease duration (years)	6.80 (3.13–12.88)					
PASI	14.45 (11.20–17.03)	0.60 (0.05–0.90)		< 0.0001		
DLQI	10.50 (7.25–15.75)	1.00 (0.00–2.75)		< 0.0001		
MoCA	26.00 (23.25–28.00)	27.00 (26.00–29.00)	28.00 (27.25–29.00)	< 0.05	< 0.001	0.14
SAS	39.50 (33.50–45.00)	31.50 (28.00–33.00)	28.50 (27.00–30.50)	< 0.0001	< 0.0001	0.26
SDS	34.00 (31.00–36.00)	32.00 (30.00–33.00)	31.00 (28.50–33.00)	0.249	< 0.05	0.63

Note: Data were presented as n (%) or median (IQR).

Abbreviations: DLQI, Dermatology Quality of Life Scale; IQR, Interquartile Range; MoCA, Montreal Cognitive Assessment; PASI, Psoriasis Area and Severity Index; SAS, Self-Rating Anxiety Scale; SDS, Self-Rating Depression Scale.

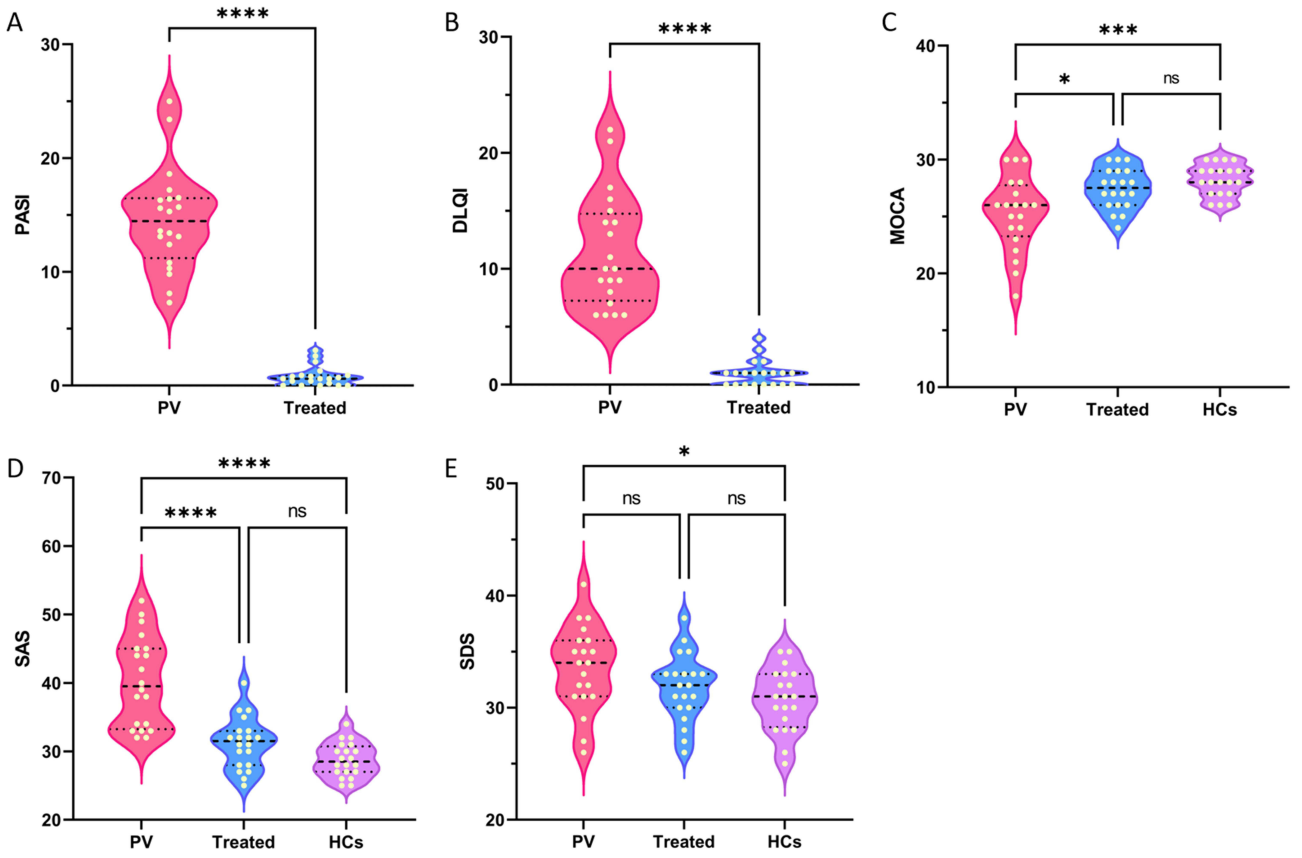

Figure 1 Comparisons of clinical and neuropsychiatric assessment scores among PV patients before treatment, PV patients after 48 weeks of secukinumab treatment, and HCs. (**A**) The PASI scores of PV patients significantly decreased after 48 weeks of secukinumab treatment. (**B**) The DLQI scores of PV patients significantly decreased after 48 weeks of treatment with secukinumab. (**C**) The MoCA scores of PV patients before treatment were significantly lower than those of HCs. After 48 weeks of secukinumab treatment, the scores improved significantly and showed no statistical difference from those of HCs. (**D**) The SAS scores of PV patients before treatment were significantly higher compared with HCs. After 48 weeks of secukinumab treatment, the SAS scores decreased significantly and showed no statistical difference from those of HCs. (**E**) The SDS scores of PV patients before treatment were significantly higher compared with HCs. However, after treatment, the SDS scores did not show a significant decrease and had no statistical difference from those of HCs. *, $P < 0.05$; ***, $P < 0.001$; ****, $P < 0.0001$. The upper and lower dashed lines represent the upper quartile and lower quartile, respectively. The dashed line in the middle represents the median. Each dot represents a sample.

Abbreviations: DLQI, Dermatology Quality of Life Scale; MOCA, Montreal Cognitive Assessment; PASI, Psoriasis Area and Severity Index; SAS, Self-Rating Anxiety Scale; SDS, Self-Rating Depression Scale.

Discussion

This study provides novel clinical evidence that moderate-to-severe PV is associated not only with peripheral inflammatory manifestations but also with functional alterations in the CNS, and that treatment with an IL-17A monoclonal antibody (secukinumab) may ameliorate these changes. By combining resting-state fMRI metrics (ALFF and ReHo) with cognitive and affective assessments, we observed abnormal neural activity in the frontal lobe, alongside decreased cognitive performance and elevated anxiety and depression symptoms in untreated PV patients. 48 weeks after treatment with secukinumab, signal values in some brain regions showed recovery and cognitive and psychiatric measures improved significantly, approaching levels seen in healthy controls. These findings support an emerging view of psoriasis as a systemic neuroimmune disorder with implications that extend beyond dermatologic inflammation.

The link between chronic skin inflammation and CNS dysfunction has received increasing attention in recent years. Psoriasis is driven by systemic immune dysregulation, in which IL-17A plays a central pathogenic role. Beyond the skin, IL-17A acts on vascular endothelium, immune cells, and resident CNS cells, including microglia and astrocytes.[4,12–14] Preclinical models of Alzheimer's disease and lipopolysaccharide (LPS)-induced neuroinflammation have shown that IL-17A promotes microglial activation, cytokine release (eg, IL-1β, TNF-α), and synaptic dysfunction, ultimately leading to cognitive impairment and mood disturbances.[10,15] Moreover, IL-17A compromises blood-brain barrier (BBB) integrity,

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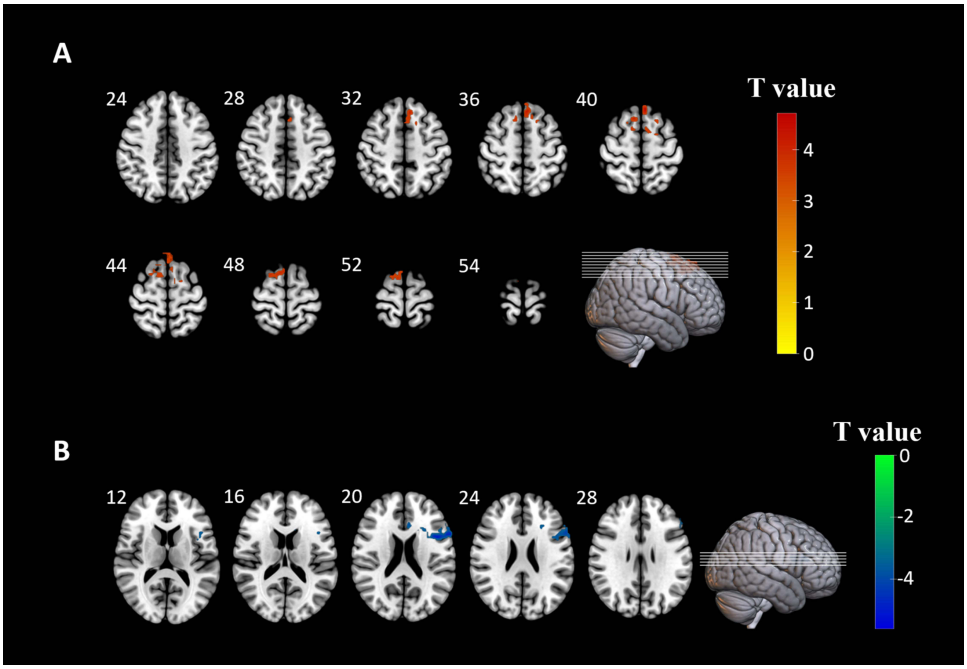

Figure 2 Brain regions with altered ALFF. Axial images are overlaid on transverse sections of MNI-152 standard anatomical images. (**A**) PV patients showed increased local ALFF compared with HCs, with red denoting elevated ALFF. (**B**) After secukinumab treatment, patients showed decreased local ALFF compared with PV patients, with blue denoting reduced ALFF. White lines denote sagittal layers. Numbers in the upper left of transverse sections indicate z-slices. The color scale represents t-values.

facilitating the entry of peripheral immune cells into the CNS.[16] These mechanisms collectively support the plausibility of a psoriasis–brain axis mediated by chronic systemic inflammation, in which IL-17A plays a key effector role.[17]

Our neuroimaging findings are consistent with known functional roles of the frontal lobe in cognition, executive function, and emotional regulation. The left frontal cortex is critical for attention, working memory, and language, and functional abnormalities in this region have been reported in neuroinflammatory conditions.[18,19] The elevated ALFF and ReHo values observed in these regions in PV patients suggest the presence of hyperactivation or abnormal neural activity patterns, possibly reflecting compensatory mechanisms in response to inflammatory stress or impaired neuronal efficiency.

The therapeutic implications of these findings are twofold. First, they suggest that IL-17A monoclonal antibodies may have direct or indirect neuroprotective effects. While secukinumab is primarily indicated for skin clearance, its potential to modulate CNS inflammation may offer added value for patients experiencing cognitive or psychiatric comorbidities. Supporting this, recent studies have shown that IL-17A blockade can improve cognitive outcomes in models of aging and neurodegeneration.[15,20] To our knowledge, this is the first clinical study to demonstrate parallel improvements in both skin and brain outcomes following IL-17A inhibition in PV patients, bridging dermatologic and neurologic domains.

Table 2 Regional ALFF Differences in PV Patients Before and After Treatment

Contrast	Brain Regions	MNI Peak Coordinate			T Value	Cluster Size
		X	Y	Z		
PV > HCs	Cluster 1 Frontal Lobe	−6	33	60	4.8449	174 139
Treated < PV	Cluster 1 Frontal Lobe	−42	9	21	−5.6091	120 106

Abbreviation: MNI, Montreal Neurological Institute.

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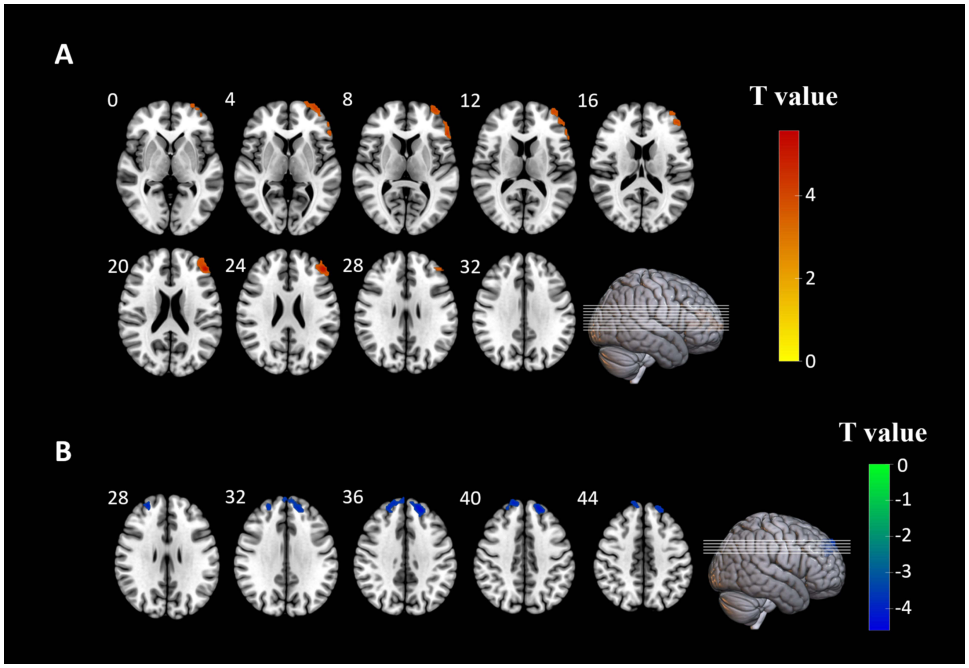

Figure 3 Brain regions with altered ReHo. Axial images are overlaid on transverse sections of MNI-152 standard anatomical images. (**A**) PV patients exhibited local ReHo alterations compared with HCs, with red regions indicating increased ReHo. (**B**) The treatment group exhibited decreased local ReHo compared with PV patients, with blue regions denoting reduced ReHo. Numbers in the upper left of transverse sections indicate z-slices. White lines denote sagittal layers. The color scale represents T-values.

Second, our results highlight the need for routine screening of neuropsychiatric symptoms in patients with moderate-to-severe psoriasis. Although mood disorders and cognitive complaints are frequently underrecognized in dermatologic practice, they can substantially impair quality of life and may be responsive to systemic treatment.9 Our findings reinforce the concept that comprehensive care in psoriasis should encompass mental health assessment and, potentially, neurocognitive testing, particularly in patients with longstanding disease, poor treatment response, or subjective cognitive decline.[21]

The bidirectional interplay between systemic inflammation and CNS dysfunction also raises important questions regarding causality and therapeutic timing. It remains unclear whether the observed brain abnormalities are reversible only at early stages of disease or whether persistent inflammation induces long-term structural damage. Further, while this study focused on secukinumab, it is possible that other biologic agents targeting the IL-23/IL-17 axis (eg, ixekizumab, brodalumab, risankizumab) may exert similar CNS effects. Future head-to-head studies or real-world comparative effectiveness analyses could help clarify the neuropsychiatric impact of different therapeutic strategies.

This study has several limitations. The sample size was modest, limiting generalizability, and the observational design cannot definitively establish causality. In addition, although exclusion criteria were designed to minimize confounding factors, unmeasured variables such as lifestyle behaviors and subtle neuropsychiatric conditions may still have influenced

Table 3 Regional ReHo Differences in PV Patients Before and After Treatment

Contrast	Brain Regions	MNI Peak Coordinate			T Value	Cluster Size
		X	Y	Z		
PV > HCs	Cluster 1 Frontal Lobe	−45	42	21	5.6215	198 198
Treated < PV	Cluster 1 Frontal Lobe	−18	45	36	−4.5038	169 158

Abbreviation: MNI, Montreal Neurological Institute.

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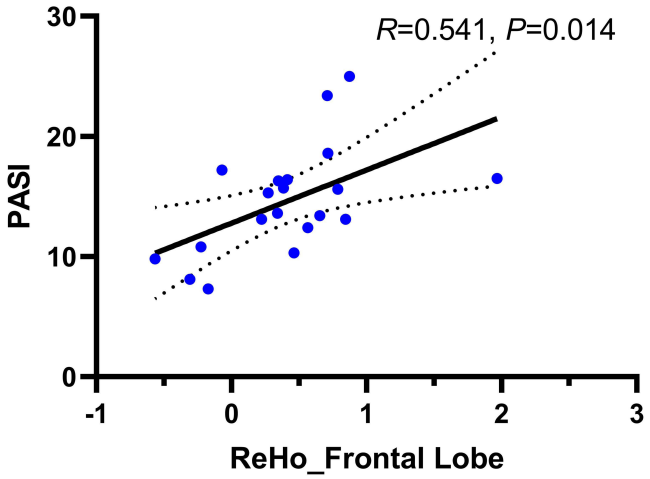

Figure 4 PV patients exhibit a correlation between ReHo alterations and PASI scores, where each blue dot represents a sample. The solid line denotes the regression line, and the smooth curve signifies the fitted 95% confidence interval.

cognitive outcomes. While resting-state fMRI provides valuable insights into brain function, it does not directly capture structural changes or task-specific activation. Future studies should incorporate multimodal neuroimaging (eg, diffusion tensor imaging, task-based fMRI, PET imaging) and longer follow-up periods to assess the durability of CNS improvements. Inclusion of inflammatory biomarkers (eg, IL-17A serum levels, high-sensitivity C-reactive protein) and genetic or epigenetic profiling could further illuminate mechanisms underlying interindividual variability in neuropsychiatric outcomes.

Conclusion

This study provides compelling evidence that moderate-to-severe PV is associated with functional alterations in key brain regions involved in cognition and emotional regulation, and that these changes may be partially reversible following targeted IL-17A blockade. Our findings expand the current understanding of psoriasis beyond its cutaneous manifestations, positioning it as a systemic inflammatory disorder with neuropsychiatric dimensions. By demonstrating parallel improvements in brain function and neurocognitive symptoms following secukinumab treatment, this work underscores the potential for biologic therapies to confer benefits that extend beyond skin clearance.

These insights support a more integrated model of psoriasis management—one that prioritizes early recognition of cognitive and emotional comorbidities, incorporates neurologic assessment into dermatologic care, and leverages anti-inflammatory therapies to restore systemic homeostasis. As research continues to unravel the complex interplay between skin, immune signaling, and the central nervous system, our findings highlight the importance of interdisciplinary approaches that bridge dermatology, neurology, and psychiatry to optimize outcomes for patients with chronic inflammatory skin disease.

Data Sharing Statement

All data used and analyzed to support the findings of this study are available from the two corresponding authors upon reasonable request.

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Disclosure

The authors declare no competing interests in this work.

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