Medical Science Journal for Advance Research

Copyright © The Author(s)
Vol. 7, No. 3, September 2026
e-ISSN: 2774-4892

Retinoid X Receptor-α and Vitamin D Receptor in Chronic Plaque Psoriasis: An Exploratory Patient–Control Study

Sara Ali Mutashar
College of Medicine, Ibn Sina University of Medical and Pharmaceutical Sciences, Baghdad, Iraq.
Corresponding Author Email: sara.ali1995@ibnsina.edu.iq

ABSTRACT

Background: *Retinoid X receptor-α (RXR-α) and vitamin D receptor (VDR) regulate epidermal differentiation and inflammatory signaling. Their clinical significance in chronic plaque psoriasis remains incompletely characterized.* ***Objective:*** *To compare reported RXR-α and VDR levels between patients with chronic plaque psoriasis and controls, characterize associated clinical factors, and assess the feasibility of evaluating receptor–severity relationships.* ***Methods:*** *We performed a retrospective exploratory, observational, patient–control study. The clinical analysis comprised 41 patients and 40 controls once one participant was excluded for inconsistent group allocation. Serving as a test statistic for age and body mass index (BMI) comparison, Welch's t test was used, while Fisher's exact test used for categorical variables. Age, sex, BMI and smoking related to psoriasis status in multivariable logistic regression analysis. Sensitivity analyses performed on post hoc alternate group classification along with an adult-only subpopulation.* ***Results:*** *28.56 ± 6.09 years (range 16-78) in patients and 34.41 ± 4.145 years (range.10.5–55.5) in controls Mean age was 31.39 ± 16.08 years (range 13–78) in patients and 32.75 ± 13.30 (Range 19.8-80.1) years in controls. In this research, a family history of psoriasis was reported by 53.7% of patients and no controls (P < 0.001). Reported RXRα and VDR means were 4.182, 2.168 and 1.068, 0.31, in patients and controls, respectively. Average PASI of patients was 17.19. No clinical covariate had a statistically significant independent association with psoriasis status; all adjusted 95% confidence intervals included unity.*
Conclusions: *Higher reported RXR-α and VDR levels were observed in the patient group, but receptor differences remain descriptive. The study does not establish either receptor as a severity biomarker. Verified individual measurements, standardized assay documentation, and validated clinical severity assessments are required for further evaluation.*

Keywords: **Chronic Plaque Psoriasis; Retinoid X Receptor-Alpha; Vitamin D Receptor; PASI; Patient–Control Study; Logistic Regression.**

Article Information
Received: July 31, 2026; Revised: August 25, 2026; Online: September 2026y

1.INTRODUCTION

A chronic immune-mediated disease, psoriasis, is marked by epidermal hyperproliferation, abnormal keratinocyte differentiation, and chronic inflammation. Psoriasis itself may have involvement in joints, cardiometabolic morbidity, psychological distress, and loss of quality of life in addition to skin manifestations. Although identifying biologically relevant markers may provide insights into disease mechanisms, its clinical relevance requires evidence beyond a simple distinction between a specific group of patients and controls. [1] [2]

Vitamin D participation in epidermal homeostasis and cutaneous immunity. Keratinocytes and various populations of immune cells within the skin express VDR. Interacting with RXR (including RXR-a), the activated ligand-bound VDR regulates transcription via vitamin D response elements. Using immunohistochemical and biochemical methods, scientists have found these receptors in healthy and psoriatic skin (receptors varying based on differentiation state in experimental studies). Receptor abundance and functional transcriptional activity are biological endpoints that are related but remain distinct. [3] [4] [5] [6]

https://doi.org/10.46966/msjar.v7i3.475
This work is licensed under a Creative Commons Attribution 4.0 International License
Published by: READ INSTITUTE

This pathway has therapeutic implications, both based on clinical presentation and experimental evidence of principle. Clinical success was associated with the topical administration of calcitriol in which the expression of VDR messenger RNA was induced. Calcipotriol and calcitriol also affect epidermal proliferation, differentiation, and selected inflammatory markers in psoriatic plaques. The use of topical maxacalcitol for plaque psoriasis demonstrated efficacy in a controlled clinical trial. collectively these observations infer ligand responsive signaling in the absence of a specific receptor mean deficiency (or excess) in Psoriasis. [7] [10] [8] [9] Most of the Published studies in the literature have revealed heterogenous patterns of receptor-expression wherelesional skin on average has less normalized RXR-α messenger RNA and lower lesional VDR expression has also been reported before narrowband ultraviolet B phototherapy. Other reports have noted increased VDR staining or not relevant of differences in patients with involved skin compared to healthy controls. Unably, these data underscore the significant heterogeneity between tissue protein expression, messenger RNA abundance, circulating-cell expression, and vitamin D metabolites. [3] [5] [13] [12] [11]

The PASI is a well established measure of severity and extent of psoriasis by virtue of its application. Nevertheless, the measurement properties of PASI have some inherent limitations, particularly for mild disease. The overall severity may also be influenced by disease extent in clinically relevant sites, body-surface area and patient reported impact. Quality of life and treatment exposure are not interchangeable clinical domains, but they are related. [14] [15] [18] [16] [17] We looked at clinical differences between patients with chronic plaque psoriasis and controls, plausibility of receptor preferences with severity and the reported RXR-α and VDR. Exploratory regression analysis was conducted to identify clinical correlations between psoriasis status and variables.

2. MATERIALS AND METHODS

2.1. Study design

An exploratory observational patient–control comparison was performed. The clinical analysis included patients classified as having chronic plaque psoriasis and participants classified as controls. Independent-group statistical methods were used because a matching structure was not available. Study reporting was organized according to the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement. [19]. Clinical comparisons were based on individual participant information, whereas receptor and PASI results were available as aggregate study summaries.

2.2. Inclusion and exclusion criteria

Eligibility for the clinical analysis required an identifiable patient or control group classification and complete information on age, sex, BMI, smoking, family history of psoriasis, and treatment status. Group allocation was considered valid when the patient and control indicators were mutually exclusive. Sex indicators were required to be internally consistent.

Participants with unresolved group classification or incomplete clinical variables were excluded from the corresponding analysis. One participant was excluded from the primary comparison because both patient and control indicators were positive. No additional exclusions were imposed on the basis of age or treatment exposure. The original clinical recruitment criteria and detailed diagnostic procedures were not documented sufficiently to permit independent assessment; consequently, no additional disease-specific eligibility conditions were assumed.

2.3. Sample collection and receptor assessment

RXR-α and VDR were assessed descriptively using the aggregate receptor results. Specimen type, anatomical sampling site, collection timing, processing, storage conditions, assay platform, calibration, and analytical precision were insufficiently documented to characterize the preanalytical and analytical procedures. The measurement compartment could not be established; the results were therefore not designated as serum concentrations, tissue expression, or circulating-cell expression.

Receptor values were presented with the accompanying reported dispersion terms. These terms were not identified as standard deviations or standard errors because their definitions were unavailable. Measurement units and receptor-

 Copyright © The Author(s)

analysis denominators were also unspecified. Individual RXR-α and VDR measurements were unavailable, precluding independently verified between-group hypothesis tests, adjustment for clinical covariates, and receptor–severity modeling.

2.4. Clinical variables and assessment of disease severity

Age and BMI were analysed as continuous variables. Binary clinical variables were run according to their coding, with 1 defining the positive condition and 0 its negation. Smoking intensity, current versus former smoking, degree of kinship and treatment class or duration could not be verified.

Considering the sample included children and adults, BMI was treated as a continuous variable and not translated into a weight-related terminology. Adult weight-category thresholds were not used for the entire sample; pediatric interpretation requires age- and sex-related reference standards. [20] Because aggregate number of individuals in each weight category could not be tied to each participant, this variable was excluded from categorical analyses.

Disease severity was explained by the reported PASI score that was used. No individual PASI values, assessment date, assessor training, severity thresholds, subgroup denominators were reported. Severity categories could therefore not be extrapolated from the overall PASI mean, and a lack of measured severity was not compensated by using a treatment status variable.

2.5. Statistical analysis

Group classification and completeness of clinical variables were assessed before statistical analysis. The primary clinical sample comprised 81 participants with valid group allocation and complete covariates. Continuous variables were summarized as mean ± standard deviation and median with interquartile range. Mann–Whitney U tests were performed as sensitivity comparisons for age and BMI. Categorical variables were compared using two-sided Fisher's exact tests. The Holm technique was further leveraged to five descriptive comparisons: age, bmi, sex, smoking, and family history. Univariable and multi variable logistic-regression models were fitted with psoriasis status as the outcome. The multivariate model contained age, male sex, BMI, and smoking. Age and BMI were modeled linearly and smoothed to 10-years and 5 kg/m² increments reported as odds ratios (ORs) with Wald 95% CIs. Family history was excluded from conventional logistic modeling because no control had a positive history, resulting in separation. These models evaluated clinical associations with psoriasis status, not receptor levels or severity. Statistical analyses were performed using Python 3.12, pandas 3.1.1, SciPy 1.18.1, and statsmodels 0.15.0. Figures were generated using matplotlib 3.11.2. Tests were two-sided, with a nominal significance threshold of 0.05. Effect estimates and precision were emphasized in accordance with statistical-reporting recommendations. [21]

3. RESULTS

3.1. Study population and clinical characteristics

This study population clinically available to analyse included 82 participants. To account for imprecise group allocation, one participant was excluded from the primary comparison, adding up to 41 patients from the index cohort and 40 controls. Among these, 14 were under 18 years of age (nine patients and five controls). Age varied from 10 to 70 years. Table 1 presents clinical characteristics, and Figure 1 depicts age and BMI distributions.

Mean age was 31.39 ± 16.08 years in patients and 32.75 ± 13.30 years in controls. The patient-minus-control mean difference was −1.36 years (95% CI −7.88 to 5.16; P = 0.679). Mean BMI was 26.23 ± 6.22 and 26.62 ± 5.96 kg/m², respectively, with a mean difference of −0.38 kg/m² (95% CI −3.08 to 2.31; P = 0.778). Mann–Whitney sensitivity comparisons showed no statistically significant difference in age (P = 0.444) or BMI (P = 0.783).

Male participants accounted for 56.1% of patients and 45.0% of controls (P = 0.377). Smoking was reported by 24.4% and 20.0%, respectively (P = 0.790). Family history of psoriasis was reported by 22/41 patients (53.7%) and no controls (P < 0.001); this comparison remained significant after Holm adjustment. The remaining four descriptive comparisons had Holm-adjusted P values of 1.000. Treatment exposure was reported in 21/41 patients (51.2%) and no controls.

Copyright © The Author(s)

Table 1. Demographic and clinical characteristics of patients and controls.

Characteristic	Patients, n = 41	Controls, n = 40	P value
Age, years, mean ± SD	31.39 ± 16.08	32.75 ± 13.30	0.679
Age, median [Q1–Q3]	25.0 [18.0–44.0]	31.5 [21.0–42.75]	—
BMI, kg/m², mean ± SD	26.23 ± 6.22	26.62 ± 5.96	0.778
BMI, median [Q1–Q3]	27.4 [22.2–31.1]	27.5 [22.2–31.2]	—
Male sex, n/N (%)	23/41 (56.1)	18/40 (45.0)	0.377
Female sex, n/N (%)	18/41 (43.9)	22/40 (55.0)	—
Smoking, n/N (%)	10/41 (24.4)	8/40 (20.0)	0.790
Family history of psoriasis, n/N (%)	22/41 (53.7)	0/40 (0.0)	<0.001
Treatment exposure, n/N (%)	21/41 (51.2)	0/40 (0.0)	Not tested
Age <18 years, n/N (%)	9/41 (22.0)	5/40 (12.5)	Not tested

Welch's t tests compare mean age and BMI; Fisher's exact tests compare sex, smoking, and family history. Sex was tested once using the male indicator. P values are unadjusted. Binary clinical categories reflect recorded affirmative responses; detailed exposure definitions were unavailable. SD, standard deviation; Q1–Q3, 25th–75th percentiles; BMI, body mass index.

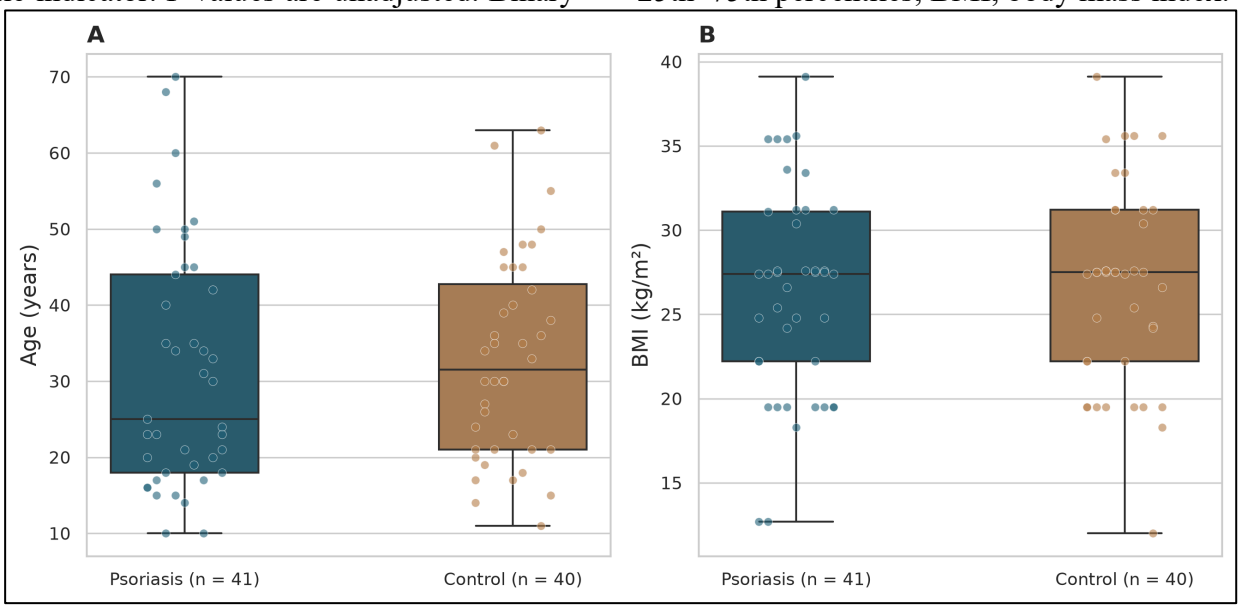


Figure 1. Age and BMI distributions in patients and controls. Panel A shows age and panel B shows BMI. Boxes represent the median and interquartile range; whiskers extend to observations within 1.5 interquartile ranges. Points represent individual measurements with horizontal displacement for visibility.

3.2. RXR-α, VDR, and disease severity

Reported RXR-α and VDR means were higher in the patient group than in the control group (Table 2; Figure 2). RXR-α values were 4.182 ± 1.003 in patients and 2.168 ± 1.328 in controls, corresponding to a difference between reported means of 2.014. VDR values were 1.068 ± 0.421 and 0.31 ± 0.89, respectively, with a difference of 0.758.

Table 2. Reported RXR-α, VDR, and PASI results in the patient and control groups.

Variable	Patients	Controls	Statistical interpretation
RXR-α	4.182 ± 1.003	2.168 ± 1.328	Descriptive comparison
VDR	1.068 ± 0.421	0.31 ± 0.89	Descriptive comparison
PASI	17.19 ± 2.128	Not applicable	Descriptive severity result

Copyright © The Author(s)

Values represent aggregate study results. Units, specimen compartment, dispersion definitions, and analysis denominators were unspecified. The clinical sample sizes in Table 1 were not assumed to be the receptor-analysis denominators. The ± quantities are not identified as SDs or standard errors. RXR-α, retinoid X receptor-alpha; VDR, vitamin D receptor; PASI, Psoriasis Area and Severity Index.

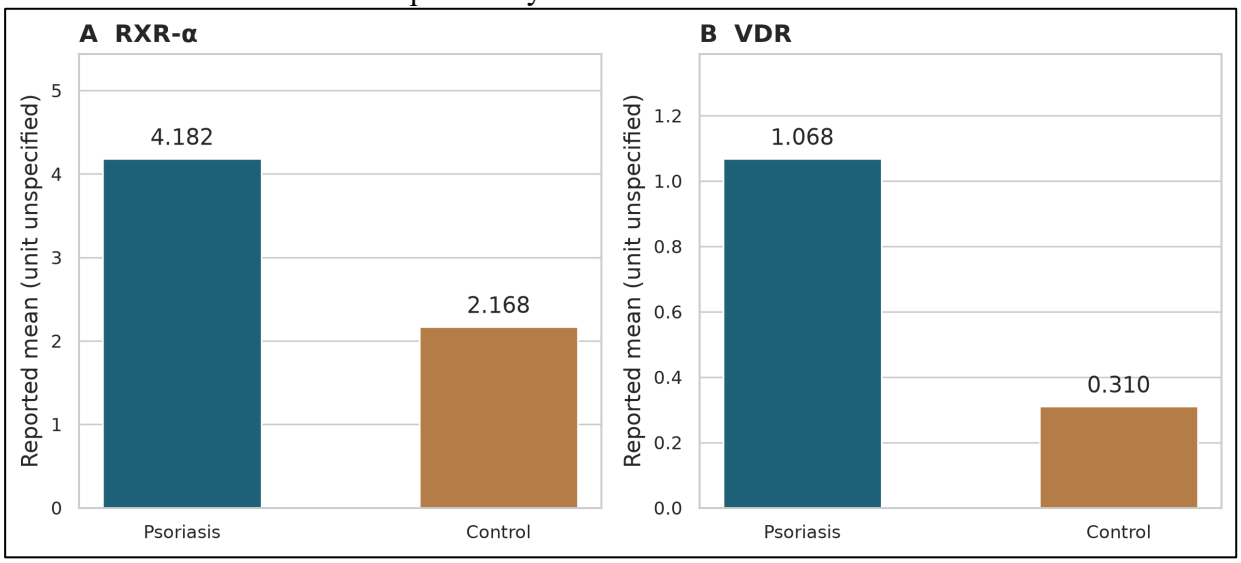


Figure 2. Reported RXR-α and VDR means in patients and controls. *Panel A shows RXR-α and panel B shows VDR. Measurement units and receptor-specific sample sizes were unspecified. Error bars are omitted because the reported dispersion terms were not defined as SDs, standard errors, or CIs.*

The reported overall PASI result was 17.19 ± 2.128. Severity-stratified VDR means were 9.215, 19, and 39 for mild, moderate, and severe disease, respectively (Table S1). Each exceeded the overall patient VDR mean of 1.068. These results are incompatible with an exhaustive partition of the same population on a common scale. Severity-specific comparisons and receptor–PASI associations were therefore not estimated. RXR-α subgroup results were likewise not tested because severity thresholds, subgroup sizes, and individual observations were unavailable.

Table S1. Reported severity-stratified receptor results; descriptive presentation only.

Severity category	RXR-α	VDR
Mild	2.59 ± 0.81	9.215 ± 3.921
Moderate	9.112 ± 2.912	19 ± 3.19
Severe	5.1092 ± 2.005	39 ± 9.219

3.3. Multivariable regression and sensitivity analyses

No covariate demonstrated a statistically significant independent association with psoriasis status in the multivariable model (Table 3; Figure 3). Adjusted ORs were 0.94 per 10-year increase in age (95% CI 0.61–1.45; P = 0.784), 1.70 for male versus female sex (0.63–4.55; P = 0.293), 0.90 per 5-kg/m² increase in BMI (0.52–1.55; P = 0.696), and 1.23 for smoking (0.38–4.01; P = 0.727). All CIs included unity and were sufficiently wide to encompass potentially meaningful associations in either direction.

Table 3. Univariable and multivariable logistic-regression associations with psoriasis status.

Covariate	Unadjusted OR (95% CI)	P value	Adjusted OR (95% CI)	P value
Age, per 10 years	0.94 (0.70–1.27)	0.676	0.94 (0.61–1.45)	0.784
Male versus female	1.56 (0.65–3.75)	0.319	1.70 (0.63–4.55)	0.293
BMI, per 5 kg/m²	0.95 (0.66–1.36)	0.775	0.90 (0.52–1.55)	0.696
Smoking	1.29 (0.45–3.70)	0.635	1.23 (0.38–4.01)	0.727

Copyright © The Author(s)

All four covariates were included in the multivariable model for 81 participants. Wald estimates are used for calculating the confidence intervals and probability values. Smoking is like positive and negative coding.

.

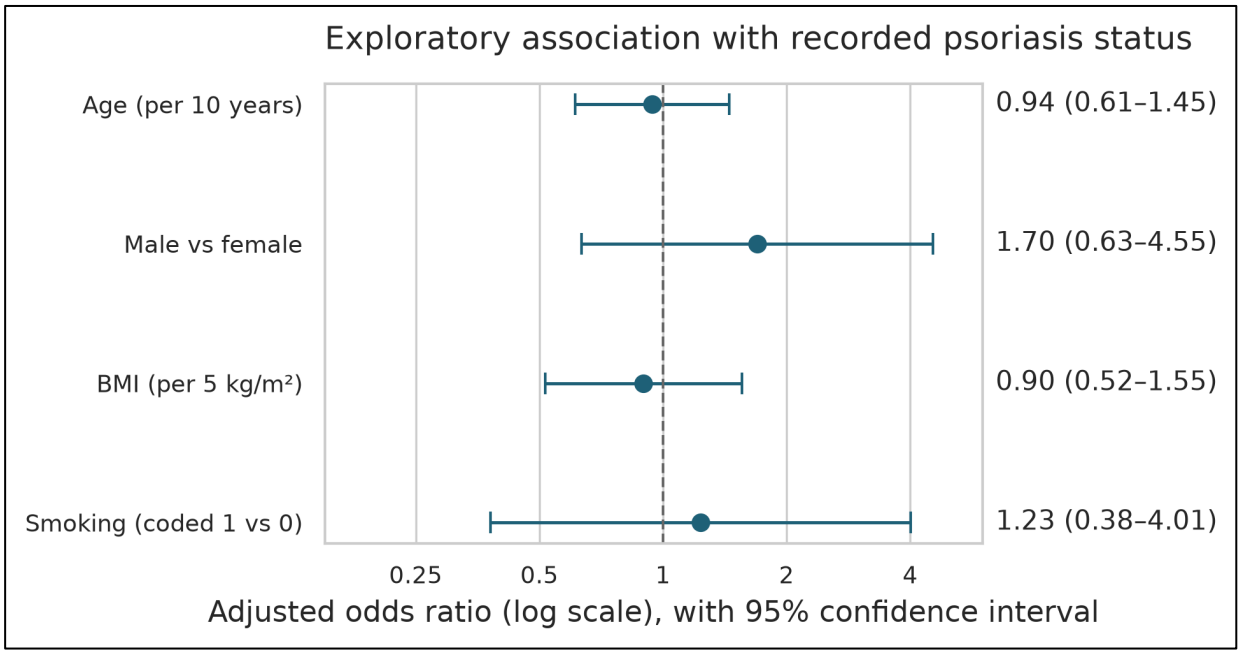


Figure 3. Adjusted clinical associations with psoriasis status. *Points represent adjusted ORs and horizontal bars indicate 95% CIs. The model includes age, sex, BMI, and smoking. The dashed vertical line indicates OR = 1.*

In the combined study population, age and BMI were correlated (Pearson r = 0.713). VIFs ranged from 1.23 to 2.29. The multivariable model converged, and no participants were excluded for statistical influence. Reclassification of the participant with indeterminate group allocation did not materially change the results: all adjusted confidence intervals included unity. The adult-only analysis included 32 patients and 35 controls. The adjusted odds ratios were 1.02 (95% CI 0.65–1.62), 1.96 (0.65–5.94), 0.92 (0.50–1.69), and 1.23 (0.37–4.04) for age, male sex, BMI, and smoking, respectively.

4. DISCUSSION

The present study identified higher reported RxR-α and VDR means in patients with psoriasis chronic plaque versus controls. These findings are descriptive and do not establish statistically significant receptor differences, altered signaling activity, or a relationship with disease severity. Family history of psoriasis was more frequent in patients, but age, sex, BMI and smoking showed no statistically significant independent associations with psoriasis status. The latter estimates were imprecise and thus not an evidence of the absence of a true effect (non-equivalence testing). Interpretation of receptor levels depends on the biological compartment and analytical endpoint. Immunohistochemistry assesses expression and localization within a tissue, messenger RNA quantification reflects transcription, and flow cytometry defines cell populations. Receptor protein concentrations, if measured in a circulating compartment, require separate analytical validation and cannot be automatically considered surrogates for epidermal expression or nuclear activity. Therefore, the present findings do not provide evidence of increased VDR-RXR transcription, altered ligand availability, or compensatory receptor regulation. [5] [11] [13]

Here some of the studies assessing the expression of different receptors in human tissue are often giving heterogeneous type of evidence in regards to psoriasis. Milde and colleagues found lesional VDR staining, however Jensen et colleagues found no locally involved-usual differences in VDR abundance or VDR-RXR DNA binding. Törmä et al noted lower normalized RXR-α transcripts in lesional skin. Elgarhy reported a decrease in VDR staining in

 Copyright © The Author(s)

the lesions in the normal lesional to the normal adjacent healthy skin to the level found ultimately after narrowband ultraviolet B treatment. There may be differences across these contradictory studies related to the comparator tissue, assay endpoint, normalization, or cellular composition. No to higher or lower overall level of receptor expression can be derived from this literature [3] [5] [11] [12].

This evidence of vitamin D signaling is pharmacologically responsive (treatment-response studies). It shows clinical improvement with topical calcitriol, accompanied by raising of VDR messenger RNA, and basal-keratinocyte VDR expression and subsequent modulation of epidermal proliferation and differentiation is induced by calcipotriol treatment. These findings bolster efforts to investigate this pathway, but they are not evidence of a rigidity mechanism as a severity marker. As with 51.2% of the patients in the treatment exposure group, its effect on receptor expression and clinical severity in the absence of the certain drug class, timing, and receptor sampling could not be determined [7] [8] [9] [10].

In interpretation, the degree of concordance between overall and severity-stratified VDR means meant that no conclusion about a receptor–severity relationship could be reached. If all subgroups used the same population and scale measurements, a weighted average of exhaustive subgroup means must fall within their range. Mean results overall were lower than the mean of every sub-group, which raises questions of the measurement scale, sub-group composition or transcription. With no additional detail to explain these findings, severity gradient, correlation with PASI levels or adjusted receptor– severity association is not acceptable. The patients in the study had a significantly higher family history which is in line with what we know about the genetics of psoriasis. Not having a positive family history in controls however may have been influenced by selection or differential ascertainment. The study does not separate inherited predisposition from these possible causes. Admittedly, large missing confidence intervals and lack of recruitment strategy hamper of the generalizability of the findings of non-significant associations for age, sex, BMI, and smoking [1] [2].

The mixed-age population requires the same note in terms of interpreting the BMI of the adult population. Fourteen participants were younger than 18 years. Categorical adult weight categories were avoided by employing continuous modelling of age-adjusted with an adult only sensitivity analysis yet did not meet the need for age-and sex specific pediatric assessment. Standardized anthropometric procedures and appropriate BMI-for-age reference measures is required for future studies. [20] Studying clinical psoriasis disease extent has many limitations. Receptor-PASI associations Known to exist, are influenced by body-surface area, clinically relevant sites and patient-reported impact. Accordingly, the measure of treatment exposure is not an exchange for these domains an overall mean PASI cannot classify individual severity levels [14] [15] [16] [17] [18]

4.1. Strengths and limitations

Study strengths included explicit type group-classification checks, complete clinical covariates in the primary analytical sample, effect estimates with CIs, multivariable clinical analyses and sensitivity analyses addressing group allocation and age composition. These approaches increased the reproducibility of clinical comparisons. Major limitations were no individual RXR-α and VDR measurements, incomplete assay documentation, lack of defined receptor units and measures of dispersion, unclear denominators for receptors, and heterogeneity in severity results. Individual PASI data, severity thresholds, and subgroup sizes were also not available. Information related to Recruitment Setting, Study Date, Eligibility Procedures, Matching and Original Sample-Size Justification was not available for reproduction. Independent-group analyses assume no matching structures apply. Small sample size limited the power to assess nonlinearity or interactions. Specific receptor measurements, a distinct biological compartment, analytically characterized assays, standardized severity assessment, and documented therapeutic exposure should be used in future studies. The association between severity and diagnostic differentiation warrants

Copyright © The Author(s)

study in separate outcomes and validated in out-of-sample populations.

5. CONCLUSIONS

Chronic plaque psoriasis patients reported higher mean RXR-α and VDR than controls; however, the receptor comparisons are limited to descriptive data. Family history differed markedly between groups. Clinical associations were imprecise when adjusted . The evaluation of receptor-severity relationships was limited by the lack of individual receptor and PASI measurements and the inconsistent severity-stratified VDR findings. The study finds no biomarkers for psoriasis severity in RXR-α or VDR. Further assessment requires validated clinical severity assessments, standardized assays, and verified measurements.

Declarations

Ethics approval

Ethical approval from Ibn Sina University of Medical and Pharmaceutical Sciences was reported for the study, together with adherence to institutional and national research-ethics guidance and the Declaration of Helsinki.

Availability of data and materials

The data are available from the corresponding author on reasonable request.

Competing interests

The author declares no competing interests.

Funding

None.

Author contributions

Sara Ali Mutashar: conceptualization, methodology, investigation, resources, data curation, formal analysis, validation, visualization, supervision, project administration, and writing—original draft and review and editing.

REFERENCES

1. Boehncke WH, Schön MP. Psoriasis. Lancet. 2015;386(9997):983–994. doi:10.1016/S0140-6736(14)61909-7.

2. Armstrong AW, Read C. Pathophysiology, clinical presentation, and treatment of psoriasis: a review. JAMA. 2020;323(19):1945–1960. doi:10.1001/jama.2020.4006.

3. Milde P, Hauser U, Simon T, Mall G, Ernst V, Haussler MR, et al. Expression of 1,25-dihydroxyvitamin D3 receptors in normal and psoriatic skin. J Invest Dermatol. 1991;97(2):230–239. doi:10.1111/1523-1747.ep12480255.

4. Reichrath J, Münssinger T, Kerber A, Rochette-Egly C, Chambon P, Bahmer FA, et al. In situ detection of retinoid-X receptor expression in normal and psoriatic human skin. Br J Dermatol. 1995;133(2):168–175. doi:10.1111/j.1365-2133.1995.tb02612.x.

5. Jensen T, Sørensen S, Sølvsten H, Kragballe K. The vitamin D3 receptor and retinoid X receptors in psoriatic skin: the receptor levels correlate with the receptor binding to DNA. Br J Dermatol. 1998;138(2):225–228. doi:10.1046/j.1365-2133.1998.02065.x.

6. Segaert S, Garmyn M, Degreef H, Bouillon R. Suppression of vitamin D receptor and induction of retinoid X receptor α expression during squamous differentiation of cultured keratinocytes. J Invest Dermatol. 2000;114(3):494–501. doi:10.1046/j.1523-1747.2000.00859.x.

7. Chen ML, Perez A, Sanan DK, Heinrich G, Chen TC, Holick MF. Induction of vitamin D receptor mRNA expression in psoriatic plaques correlates with clinical response to 1,25-dihydroxyvitamin D3. J Invest Dermatol. 1996;106(4):637–641. doi:10.1111/1523-1747.ep12345443.

8. Reichrath J, Müller SM, Kerber A, Baum HP, Bahmer FA. Biologic effects of topical calcipotriol (MC 903) treatment in psoriatic skin. J Am Acad Dermatol. 1997;36(1):19–28. doi:10.1016/S0190-9622(97)70320-7.

9. Reichrath J, Perez A, Müller SM, Chen TC, Kerber A, Bahmer FA, et al. Topical calcitriol (1,25-dihydroxyvitamin D3) treatment of psoriasis: an immunohistological evaluation. Acta Derm Venereol. 1997;77(4):268–272. doi:10.2340/0001555577268272.

10. Barker JN, Ashton RE, Marks R, Harris RI, Berth-Jones J. Topical maxacalcitol for the treatment of psoriasis vulgaris: a placebo-

 Copyright © The Author(s)

controlled, double-blind, dose-finding study with active comparator. Br J Dermatol. 1999;141(2):274–278. doi:10.1046/j.1365-2133.1999.02975.x.

11. Törmä H, Karlsson T, Michaëlsson G, Rollman O, Vahlquist A. Decreased mRNA levels of retinoic acid receptor alpha, retinoid X receptor alpha and thyroid hormone receptor alpha in lesional psoriatic skin. Acta Derm Venereol. 2000;80(1):4–9. doi:10.1080/000155500750012405.

12. Elgarhy LH, Eltatawy RA, Rizk O, Ismail M. Vitamin D receptor expression in chronic plaque psoriasis before and after narrowband ultraviolet B phototherapy. J Cutan Aesthet Surg. 2023;16(2):128–133. doi:10.4103/JCAS.JCAS_122_22.

13. Zanghaneh AJ, Elmelid A, Gillstedt M, Ahmic O, Andersson B, Osmancevic A. The expression of vitamin D receptor on peripheral blood mononuclear cells in patients with psoriasis. Int J Mol Sci. 2024;25(19):10677. doi:10.3390/ijms251910677.

14. Fredriksson T, Pettersson U. Severe psoriasis—oral therapy with a new retinoid. Dermatologica. 1978;157(4):238–244. doi:10.1159/000250839.

15. Spuls PI, Lecluse LLA, Poulsen MLNF, Bos JD, Stern RS, Nijsten T. How good are clinical severity and outcome measures for psoriasis? Quantitative evaluation in a systematic review. J Invest Dermatol. 2010;130(4):933–943. doi:10.1038/jid.2009.391.

16. Finlay AY. Current severe psoriasis and the rule of tens. Br J Dermatol. 2005;152(5):861–867. doi:10.1111/j.1365-2133.2005.06502.x.

17. Mrowietz U, Kragballe K, Reich K, Spuls P, Griffiths CEM, Nast A, et al. Definition of treatment goals for moderate to severe psoriasis: a European consensus. Arch Dermatol Res. 2011;303(1):1–10. doi:10.1007/s00403-010-1080-1.

18. Augustin M, Krüger K, Radtke MA, Schwippl I, Reich K. Disease severity, quality of life and health care in plaque-type psoriasis: a multicenter cross-sectional study in Germany. Dermatology. 2008;216(4):366–372. doi:10.1159/000119415.

19. von Elm E, Altman DG, Egger M, Pocock SJ, Gøtzsche PC, Vandenbroucke JP; STROBE Initiative. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) statement: guidelines for reporting observational studies. PLoS Med. 2007;4(10):e296. doi:10.1371/journal.pmed.0040296.

20. Barlow SE; Expert Committee. Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. Pediatrics. 2007;120(Suppl 4):S164–S192. doi:10.1542/peds.2007-2329C.

21. Lang TA, Altman DG. Basic statistical reporting for articles published in biomedical journals: the Statistical Analyses and Methods in the Published Literature or the SAMPL guidelines. Int J Nurs Stud. 2015;52(1):5–9. doi:10.1016/j.ijnurstu.2014.09.006.

Copyright © The Author(s)