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Commentary

An analysis of racial disparities in systemic treatments for psoriasis and their pharmacologic consequences

Rini Manoj Desai, B.S.[1,*], Mariana McCune, B.S.[1], Rebecca Olsen, D.O.[2], Liliana Tong, B.S.[1], Mitchell Sparling Davis, M.D.[1]

[1]University of Arizona College of Medicine-Phoenix, Phoenix, AZ, USA

[2]CHS Northwest Transitional Year Residency, Tucson, AZ, USA

*Author for correspondence: Email: rinidesai@arizona.edu

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Abstract

Psoriasis is a systemic inflammatory skin disease associated with significant comorbidities, yet access to effective systemic treatments remains disparate across racial and ethnic groups. Building upon recent findings of disparities in systemic medication prescriptions and hospitalization outcomes among patients with psoriasis, this commentary explores the pharmacologic, toxicologic, and health equity implications of these inequities. We discuss how prolonged undertreatment in marginalized populations exacerbates systemic inflammation, increases disease burden, and perpetuates poor health outcomes. Addressing these disparities requires a comprehensive, multidisciplinary approach aimed at ensuring all patients with psoriasis can benefit from advances in systemic therapies.

Keywords: Psoriasis, Racial disparities, Biologics, Systemic therapy, Health equity, Treatment access, Pharmacogenomics

Introduction

Psoriasis is a chronic, systemic inflammatory condition that extends beyond dermatologic symptoms to include increased risks for cardiovascular disease, diabetes, and other comorbidities [1]. Systemic therapies–particularly biologics–have transformed care for moderate-to-severe psoriasis, offering powerful tools to control systemic inflammation. However, despite their growing popularity and availability, equal access to these medications remains uneven, disproportionately affecting historically marginalized populations.

In our recent study, we examined the intersection of race, hospitalization outcomes, and systemic medication prescription among patients with psoriasis using the 2020 Healthcare Cost and Utilization Project (HCUP) National Inpatient Sample (NIS) [2]. We discovered that Hispanic, Native American, and Asian or Pacific Islander patients were significantly less likely to receive systemic treatments compared to white patients, and that all non-white racial groups experience longer hospital stays. These findings raise critical concerns about pharmacological access, treatment delays, and their potential toxicologic consequences–particularly the extended systemic inflammatory burden faced by patients who do not receive timely systemic therapy. This commentary seeks to elucidate the pharmacologic and toxicologic ramifications of these disparities.

Systemic Psoriasis Medications: Efficacy and Safety

Traditional systemic treatments for moderate-to-severe psoriasis include methotrexate, cyclosporine, and acitretin [3]. Methotrexate, a dihydrofolate reductase inhibitor, works by decreasing the proliferation of lymphoid cells which leads to an immunosuppressive effect that is beneficial in the treatment of psoriasis [3]. Its efficacy in the treatment of psoriasis is well-described, with clinical trials demonstrating a significant reduction in the Psoriasis Area and Severity Index (PASI) after 16 weeks of treatment in comparison to placebo [4]. Given its widespread use for several decades, the safety profile

of methotrexate is well-characterized. Common side effects include gastrointestinal disturbances, including nausea, vomiting, abdominal pain, and mouth ulcerations [5]. Elevated hepatic transaminases are also observed in over 10% of patients [4]. Although rare, serious infections have been reported as adverse events [4,5]. Yan *et al.* demonstrated a higher incidence of adverse events in patients with psoriatic arthritis as opposed to patients with psoriasis [6].

Cyclosporine is another immunosuppressant commonly used for the treatment of moderate-to-severe psoriasis that functions as a calcineurin inhibitor, which blocks proinflammatory signaling and reduces T-cell activation [7]. Clinical trials have shown high efficacy in rapid clinical improvement of psoriasis, with comparative studies showing greater efficacy of cyclosporine compared with methotrexate [3,8-9]. The most common adverse effects secondary to cyclosporine use are nephrotoxicity and hypertension, with reversible renal impairment occurring in approximately 19-24% of patients during short-term treatment [3]. Long-term use of cyclosporine can increase the risk of irreversible kidney damage via oxidative stress and renal arteriole vasoconstriction [10,11].

Acitretin, a synthetic retinoid, modulates epidermal differentiation and proliferation without exerting immunosuppressive effects [3]. While less efficacious than methotrexate and cyclosporine in direct comparisons, it may be preferable in patients with a history of chronic infections or malignancies [12]. Despite this, there are several common adverse effects including hyperlipidemia in up to 50% of patients and elevated liver enzymes in over 10% of patients. Rare adverse effects include pancreatitis and pseudotumor cerebri-like symptoms. Acitretin is also contraindicated in pregnancy due to its teratogenicity and long half-life, which requires contraception for three years after discontinuation [3].

Biologic therapies are a newer systemic treatment option for moderate-to-severe psoriasis with TNF-α inhibitors, IL-17 inhibitors, IL-12/23 inhibitors, and IL-23 inhibitors the most commonly used classes. Within the past decade, biologics targeting IL-17 or IL-23 have been of particular interest [13]. Biologics have shown higher efficacy in achieving significant clinical improvement compared to nonbiologic agents including methotrexate and cyclosporine [3,12-15]. While biologics are generally well-tolerated, concerns regarding infection risk remain. Some studies have shown no significant increase in risk of serious infection for biologics compared to non-biologic systemic therapies [16,17]. Another study showed an increase in risk of serious infections when comparing biologics, with adalimumab and infliximab associated with a higher risk of infection compared to etanercept and ustekinumab with a lower risk [18]. Overall, biologics offer highly effective treatment options, though individual safety profiles, particularly regarding infectious complications, warrant careful consideration. Janus kinase (JAK) inhibitors and tyrosine kinase (TYK) inhibitors are the newest classes of small molecule drugs available for the treatment of psoriasis [13,19].

Pharmacogenomic and Pharmacokinetic Considerations

Pharmacogenomic and pharmacokinetic factors play a critical role in the efficacy of biologic therapies for psoriasis. Understanding how these variables change treatment across individuals with diverse backgrounds can optimize clinical outcomes. Despite the advancement of new biologic therapies for psoriasis, gaps remain in their efficacy across different racial populations. One study found that guselkumab was most effective for white patients with 86.8% reaching "clear/almost clear", ixekizumab was the most effective for Asian and Latino patients with 90.7% and 89.4% "clear/almost clear" respectively, and brodalumab was most effective for African American patients with 75.0% reaching "clear/almost clear" [20]. Another study found that while ixekizumab is effective across different racial groups, the response was slower in American Indian and Alaska Native American patients with psoriasis [21]. At the intersection of racial disparities in pharmacological efficacy lies a sobering reality: therapeutic precision is often calibrated to individuals who have historically dominated clinical trials.

Genome-wide association studies (GWAS) have identified many susceptible genomic loci for psoriasis in individuals of European and Asian ancestries, including polymorphism in HLA-C*06:02, IL-23/IL-17 receptors, type I interferons and NF-κB markers [22,23]. However, there are no known large-scale GWAS on psoriasis in individuals of Native American or African ancestries to date.

Clinical pharmacogenomic and pharmacokinetic data also remain limited for individuals of non-european ancestries. Historically, landmark Phase 3 clinical trials for agents such as adalimumab, etanercept, and ustekinumab have substantially underrepresented African, Hispanic, Native American and Asian or Pacific Islander participants [24]. Notably, research on ustekinumab and IL-12/23 blockade has only focused on the HLA-C*06:02 allele, which is more studied among individuals of European ancestry than individuals of African or Asian ancestries [25]. Similarly, milestone investigations into the TNF-blockade response gene TNFAIP3 have demonstrated an association between TNFAIP3 haplotypes and different therapeutic responses to TNF-blockade, but only in a cohort that was exclusively European Caucasian [26]. One recent case control study has identified novel TNFAIP3 haplotypes in African American individuals [27]. However, the clinical significance of these new TNFAIP3 haplotype in the context of psoriasis treatment remains unknown. These disparities underscore the urgent need for studies that not only identify susceptibility alleles in underrepresented populations, but also elucidate their clinical relevance to biologic response and long-term therapeutic efficacy.

Of the limited pharmacogenomic studies in individuals of color, it is known that IL17F His161Arg polymorphism, which is more prevalent in the Asian population, leads to elevated serum levels of IL-17F compared to non-carriers [28]. This newly emerging research can be informative in understanding therapeutic efficacy for secukinumab, ixekizumab, and brodalumab in individuals of Asian ancestries. Larger scaled pharmacogenomic studies involving African, Hispanic, Native American and Pacific Islander participants are needed to understand the full effects of this polymorphism. In the age of precision medicine, it is more important than ever to recognize the gap in pharmacogenomic studies for psoriasis in diverse ethnic backgrounds, especially for individuals of African and Native American ancestries.

The Consequences of Undertreated Prolonged Inflammation

Chronic inflammation is known to exacerbate psoriasis, lead to more extensive psoriatic disease involvement, significant post-inflammatory changes, and poorer overall health outcomes [29]. However, the cause of racial disparities in prolonged inflammation is

multifaceted. It may stem from chronic stress exposure, environmental factors, and unequal access to care—which all reinforce a biologic milieu primed for persistent inflammation [30,31].

Individuals of racial minorities often experience higher and prolonged inflammatory burdens than their white counterparts [32]. African American individuals with psoriasis are more likely to have more extensive psoriatic disease involvement and experience higher rates of comorbidities, including diabetes, hypertension, and congestive heart failure compared to their white counterparts, which highlights the underlying inflammatory processes contributing to disease severity [32].

Clinical Trial Exclusion and Biologic Hesitancy

African American, Hispanic, and indigenous patients are frequently underrepresented in psoriasis clinical trials for multiple reasons. Barriers to access such as socioeconomic status may impact transportation and work flexibility, limiting the ability to attend required appointments [33].

There are several factors that may make minority patients hesitant to initiate psoriasis biologic medications. One major factor is unfamiliarity with biologic medications, particularly in African American patients who lack prior biologic experience. The unfamiliarity can cause apprehension predominantly regarding fear of medication side effects and immune suppression [34]. Treatment decisions can also be impacted by cultural expectations and education level [35]. One study demonstrated that patients who do not regularly read books were 79% less likely to receive an anti-TNF-alpha medication, 78% decreased odds of starting anti-IL-12/23 medication, and 74% less likely to initiate anti-IL-7 therapy compared to patients who read books weekly [35].

Patients with a lower education level or salary are less likely to receive biologics particularly newer therapies [35]. Non-English-speaking Hispanic patients face an additional barrier which would be solved by increasing the number of Spanish speaking research coordinators [36]. Lastly, comorbidities often exclude patients from participating in clinical trials [36]. Certain minority groups are disproportionately affected by chronic kidney disease, diabetes mellitus, HIV, heart disease, and many more chronic diseases, which can be disqualifying factors for many clinical trials [37-39]. These exclusion criteria can contribute to underrepresentation of minorities in clinical trial patient populations, and the subsequent lack of diversity can have dramatic impacts on treatment outcomes. There is a disproportionately high risk for treatment response disparities and decreased access to therapy in minority patients compared to the general population. Within 1 year of starting medication, non-white patients have a lower likelihood of responding to psoriasis biologic treatment compared to white patients [36].

Clinical and Public Health Implications

Extended hospital admissions and lack of systemic treatment can exacerbate chronic inflammation in cases where psoriasis causes persistent immune activation and tissue damage. This frequently results in poor long-term outcomes and higher rates of post-discharge complications [40-42]. Prolonged inflammation is associated with an increased risk for development of psoriatic arthritis and other chronic diseases, such as diabetes and cardiovascular disease [43,44]. Delayed treatment of psoriasis can result in increased disease severity and also higher medical costs due to more frequent hospital admissions and the requirement for more intensive and prolonged treatment [44,45]. One key challenge in the use of biologics for treatment of psoriasis is the high cost which can place a high burden on healthcare systems [19].

Data suggests a significant racial disparity in psoriasis treatment outcomes for non-white as compared to Caucasian patient populations. Minority patients are more likely to have extensive disease involvement, but are less likely to receive biologic treatments that are associated with better clinical outcomes and improved quality of life [48]. As described in the prior section, psoriasis clinical trials have historically lacked participation by diverse patient populations leading to a knowledge gap on the clinical impact of biologic medications on non-Caucasian psoriasis patients. There is less data on the efficacy and safety of biologics in minority populations which can contribute to hesitancy for prescribing by healthcare providers and usage by non-white patients [19].

In 2021, the American Academy of Dermatology shared a 3-year plan to improve diversity, equity, and inclusion in dermatology [47]. The field of dermatology has been making strides in addressing these health inequities by promoting a more diverse workforce, encouraging underrepresented minority dermatologists to take on leadership positions in the field to improve cross-cultural understanding, working to make research more inclusive, expanding residency education, and improving treatment approaches for cutaneous conditions that disproportionately impact non-Caucasian patient populations. It would be beneficial if psoriasis clinical trials included more diverse participants to help develop evidence-based treatments that are effective across multiple racial and ethnic groups which will also improve patient trust and confidence in prescribed therapies [34].

Analysis of Length of Stay

All non-white racial identities in our study had a significantly longer length of stay (LOS) compared to white patients, with Native Americans having the longest LOS that is 1.24 times that of white patients (IRR = 1.24, 95% CI 1.18–1.30). Our results demonstrated an association between systemic treatment and shorter hospital stays.

Admission for a primary diagnosis of psoriasis has been associated with older age, medicare, medicaid, lack of insurance, non-white race and with significantly longer LOS which has been found to vary between types of psoriasis [45]. LOS is increased in erythrodermic and pustular psoriasis and decreased with systemic treatment of psoriatic arthritis. Subtype differences indicate that severity of psoriasis likely influences LOS. Hospital acquired infection was a significant burden for longer length of stay [48]. Inpatient dermatology consultation is associated with decreased LOS in inflammatory skin disease and lack of this service in rural and community settings may increase LOS [49].

Dermatology consultation also optimizes quality of care and treatment effectiveness, being associated with diagnosis change and management adjustment [48]. However, while access to inpatient dermatology may be a disparity in itself, underrepresentation of SOC at all levels of medical training may lead to diagnostic uncertainty in SOC and delayed diagnosis and treatment even with access to dermatologic care [51,52].

LOS disparities may be a marker of racial and socioeconomic disparities in psoriasis burden, which may inform why all minoritized

groups in our study were found to have increased LOS. This may be related to access of outpatient and preventative care, as indicated by insurance status, and higher risk health status attributable to compounding comorbid conditions [53].

Limited English proficiency patients have increased barriers to discharge and appropriate follow-up [54]. Hospital acquired infection increased LOS and cost burden further, which may cause a snowball effect of disease and admission burden in marginalized groups [55].

Conclusion

The observed racial disparities in systemic medication utilization and hospitalization outcomes in psoriasis highlight a critical intersection of clinical pharmacology, toxicology, and health equity. For patients who are systematically undertreated, prolonged inflammation represents not only a pathophysiologic burden but also a marker of structural inequities within healthcare delivery. Equity in psoriasis care demands more than awareness—it necessitates actionable steps: expanding pharmacogenomic research in underrepresented populations, improving diversity in clinical trials, enhancing culturally competent care, and addressing social determinants that limit access to systemic therapies. LOS of stay, as a proxy for systemic barriers, may be a measurable target for intervention. Only through targeted, evidence-based interventions can we hope to mitigate the toxicologic consequences of undertreatment and achieve equitable health outcomes in psoriasis.

Conflicts of Interest

The authors have no conflicts of interest to report.

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