***Review Article***

**Psoriasis in Nigeria: The Critical Role of Genetic and Genomic Tools in Addressing a Predominantly Genetic Condition**

**ABSTRACT**

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| Psoriasis, a chronic inflammatory skin condition, is known to affect about 125 million people worldwide but is widely underreported in Africa, particularly Nigeria. Published studies report a low prevalence of psoriasis in Nigeria, estimating only about 0.33% of sufferers. However, recent research suggests an increasing incidence, contrasting to older data indicating a lower prevalence. This review highlights a need for comprehensive Nigerian population-based studies to accurately assess the condition's prevalence and impact in Nigeria.  High costs, limited availability of effective treatments, and side effects associated with existing therapies pose significant challenges to managing psoriasis in Nigeria. Due to the expensive nature and inaccessibility of the known psoriasis management strategies- topical agents, systemic therapies, and phototherapy- psoriasis therapy and management in Nigeria remains an arduous task for sufferers. Developing genomic tools and research offer promising advancements for more effective and personalized treatment options. Genetic research, particularly through genome-wide association studies (GWAS), is beginning to identify new therapeutic targets and potential treatments. However, integrating genomic tools into clinical practice in Nigeria faces barriers such as limited funding, shortage of skilled personnel, inadequate infrastructure, and ethical concerns related to genetic research.  This review highlights the challenges and steps towards addressing them. It emphasizes the need for increased investment in genomic research, capacity building for researchers and healthcare professionals, and improved infrastructure in Nigeria. Additionally, raising public awareness about psoriasis and fostering collaboration between government, research institutes, and non-governmental organizations are essential for advancing psoriasis research and management in Nigeria. |

*Keywords: Psoriasis, Nigeria, Genetics, Genome-wide association studies, Dermatology.*

**1. INTRODUCTION**

Psoriasis is a common auto-immune skin disorder associated with a complex interplay of multiple genetic risk foci, environmental risk factors, and excessive immunological abnormalities [1]. However, genetics largely determines an individual’s susceptibility, accounting for 70% of disease risk. According to Rendon and Schäkel [2], the disease shows dermatological manifestations in the forms of chronic plaque lesions (psoriasis vulgaris, which accounts for 90% of disease cases), acute and usually self-limiting guttate type eruptions, seborrhoeic psoriasis, pustular lesion [3].

Aside from the physical effects, Ferreira et al. [4] reported other resulting psychological effects, such as anxiety, stigmatization, and embarrassment. Beyond both the dermatological and psychological impact, other disorders such as joint disease, as seen in at least 10% of patients, and cardiovascular and metabolic abnormalities may contribute to the morbidity and disability caused by the disease.

A systematic review by Parisi et al. [5] highlighted that epidemiological data on the incidence of psoriasis are limited, and considerable gaps exist in the geographical areas that reported this information, particularly from low and middle-income countries, with studies conducted mainly in Europe and North America. Epidemiological data from Nigeria is limited. A study on patients attending an outpatient dermatology clinic in Enugu in Southeast Nigeria [6] reported that 0.4% were diagnosed with psoriasis. In comparison, 1.1% of the patients attending a similar clinic in Lagos, Southwest Nigeria, had psoriasis [7].

Various reports from sub-Saharan Africa have indicated that psoriasis is not as common as in other parts of the world. However, Parisi et al. [5] acknowledge that the figures published in their study might be underestimates of the true prevalence of the disease because most of the data came from studies using databases that only reflect people with psoriasis who sought healthcare and might not reflect the underdiagnosed population.

2. GENETIC BASIS OF PSORIASIS

Mahil et al. [8] reported more than 40 genome regions associated with psoriasis; 26 were discovered using GWAS, and a further 15 psoriasis susceptibility loci were identified in the Immunochip study. Each region spans many genes; however, specific genes have been highlighted within each locus, contributing to increased individual susceptibility [8]. Harden et al. [9] reported that these genes span an array of functions that involve [antigen presentation](https://www.sciencedirect.com/topics/immunology-and-microbiology/antigen-presentation) (HLA-Cw6, [ERAP1](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/aminopeptidase), [ERAP2](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/erap2), MICA), the IL-23 axis (IL12Bp40, IL23Ap19, IL23R, [JAK2](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/janus-kinase), TYK2), T-cell development and T-cells polarisation ([RUNX1](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/runx1), [RUNX3](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/runx3), [STAT3](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/stat3), [TAGAP](https://www.sciencedirect.com/topics/medicine-and-dentistry/t-cell-activation), IL4, IL13), [innate immunity](https://www.sciencedirect.com/topics/immunology-and-microbiology/innate-immune-system) (CARD14, c-REL, TRAF3IP2, DDX58, IFIH1), and negative regulators of immune responses ([TNIP1](https://www.sciencedirect.com/topics/medicine-and-dentistry/calphobindin-ii), TNFAIP3, NFKBIA, ZC3H12C, IL36RN, [SOCS1](https://www.sciencedirect.com/topics/biochemistry-genetics-and-molecular-biology/suppressor-of-cytokine-signaling-1)).

Genetic linkage studies have identified a minimum of 12 distinct loci suspected to contain genes related to psoriasis susceptibility (PSORS). However, most of these findings could not be replicated, highlighting the limitations of genetic linkage [9,10]

Preliminary genetic studies for psoriasis were conducted using linkage analysis in familial psoriasis. In a study to analyze three suggested psoriasis susceptibility loci, Enlund et al. [11] confirmed linkage to chromosome 6p (HLA region) and to 17q but not to 4q using a large Swedish set of families. The major histocompatibility complex (MHC) that primarily encodes genes involved in antigen presentation is found on chromosome 6p21.3 and contains the strongest susceptibility locus for psoriasis (PSORS1), which accounts for approximately 35%–50% of the genetic risk for psoriasis [12,13,109]. Dand et al. [10] further opined that PSORS1 contains the candidate gene corneodesmosin (CDSN), which encodes a desmosomal protein involved in keratinocyte cohesion and desquamation.

Confirming the association of HLA-Cw6 and psoriasis proved challenging as at least ten other genes were mapped to the just telomeric neighborhood to HLA-C [14]. Several of these genes, including CDSN, HCR, and PSORS1C3, are expressed in skin cells and made very likely candidates for the PSORS1 gene as explained by the existence of multiple associated genes, which is a consequence of [linkage disequilibrium](https://www.sciencedirect.com/topics/neuroscience/linkage-disequilibrium) — the probability of particular alleles at two or more loci to be inherited together more often than would usually be predicted by chance [10]. Thus, the genes in this region are all associated with psoriasis because they are all in linkage disequilibrium. HLA-C\*06:02 is now confidently considered the most likely causal susceptibility allele since single nucleotide polymorphisms (SNPs) that tag this allele have generated the most significant association signals in subsequent case-control studies [10].

Within the PSORS-2 locus, on chromosome 17q-25, the CARD14 gene, which has been reported to be the most likely susceptibility gene, is found [15]. The CARD14 is a CARD-containing, membrane-associated guanylate kinase-like domain-containing protein (CARMA) family of scaffolding proteins that play a critical role in activating the NF-κB signaling pathway and recruiting IKK proteins [16]. PSORS-4 also contains the late cornified envelope (LCE) genes, which encode stratum corneum proteins involved in terminal epidermal differentiation. This locus has been implicated in psoriasis susceptibility in genome-wide association studies of both European and Chinese populations [17,18]. Mutations of the CARD14 gene are associated with rare and common forms of psoriasis- plaque psoriasis, psoriatic arthritis, and pustular psoriasis.

While the only successfully validated linkage results are the PSORS-1, PSORS-2 and PSORS-4 loci, He et al. [19] reported that SC4MOL is situated within the psoriasis susceptibility locus PSORS-9 and may be a genetic risk factor for common skin conditions.

# 3. CLINICAL PRESENTATION OF PSORIASIS

Psoriasis can occur at any age, but the average patient is diagnosed at 33 [3]. Studies have shown that Type I psoriasis and Type II predominantly occur at different age ranges, with the former occurring before 40 and the latter occurring between 55 and 60 [20].

The severity of the clinical features varies depending on the psoriasis variant, from cutaneous patches to severe exfoliation [21,114]. Different variants could coexist in a patient, including the signs and symptoms. Still, a dominant variant at a particular time during the disease course always expresses the most signs and symptoms—the main clinical features of psoriasis are scaly, thick, and erythematous lesions with sharply demarcated margins. The lesions are usually itchy and hemorrhagic [22,23]. Pinpoint bleeding occurs when the outer scales are peeled off from psoriatic plaques. This characteristic sign is called the 'Auspitz sign.'

Usually, psoriasis is a symmetrical eruption. 30% of psoriatic patients have their joints affected (Scarpa *et al.,* 2006). About 33.3% have severe disease that involves cutaneous lesions spreading over 20% of the patient's body surface area (BSA), moderate disease with lesions extending to about 10-20% BSA, and 66.7% of patients have mild disease with lesions in less than 10% BSA [24].

Sometimes, psoriasis can develop through the Koebner phenomenon, which posits that psoriatic lesions can occur 1-2 weeks after different kinds of skin trauma, such as injury or skin disorder caused by microbial agents [24,25]. Different variants of psoriasis include Plaque psoriasis, guttate psoriasis, erythrodermic psoriasis, pustular psoriasis, acral psoriasis, seborrheic psoriasis, and flexural psoriasis.

## 3.1 Plaque Psoriasis

Plaque psoriasis is called Psoriasis vulgaris, and about 70-90% of psoriatic patients show plaque psoriasis. This thus implies that it is the most common form of psoriasis. Patients show well-demarcated, round-oval discoid plaques covered with silvery white scales. The skin shows an erythematous appearance, with the plaque ranging from white to red, and it might turn greyish-white when left untreated. Lesions can vary from 0.5cm in diameter to vast areas on the skin, usually beginning with erythematous papules extending and merging to form plaques. Wornoff's ring (a white blanching ring) can also be observed on the skin surface surrounding the psoriatic plaque [23,27]. The lesion appears on cutaneous surfaces such as the scalp, knees, elbow, and lumbosacral area. Lesions are always absent on the face.

Over time, plaque psoriasis lesions are steady, and as they relapse, partial or total central clearing produces an annular or polycyclic appearance; this central clearing can occasionally be associated with hypopigmentation [23,24].

## 3.2 Guttate Psoriasis

This is also known as eruptive psoriasis. Guttate was coined from the Greek word 'gutta,' which translates as droplet. Just 2% of psoriasis cases are guttate psoriasis [22,27]. The small confetti-like lesions (2-10mm diameter) usually appear over the trunk and extremities [28]. The number of lesions may vary from a few scattered papules to hundreds.

Guttate psoriasis primarily affects children and young adults, especially those with a history of the disease in their ancestry. This disease may result from streptococcal upper respiratory tract infection [28]. Guttate psoriasis is self-limiting but can sometimes develop into chronic plaque psoriasis [29]. Naldi [26] reported that 10% of psoriatic patients with plaque psoriasis had guttate psoriasis during the disease pathogenesis. According to Martin et al. [30], there is about a 40% likelihood of developing chronic plaque psoriasis after guttate psoriasis. Plaque psoriasis and guttate psoriasis are very similar genetically, with a stringent association with the PSORS1 genetic loci, a significant factor leading to plaque psoriasis [31,32].

## 3.3 Erythrodermic Psoriasis

This is a seldomly reported variant, but it is very severe. Erythroderma psoriasis appears as erythema, scales, pruritus, or exfoliation covering at least 75% of the body and can cover over 90% of the body [33-35]. This condition results in vasodilation, which leads to excessive heat loss with hypothermia, leading to erythema and protein loss [36-38]. The extravasation of proteins may further result in edema of limbs, especially in patients with longstanding erythroderma [33,35,39]. Other symptoms include myalgia, fever and chills, dehydration, lymphadenopathy, arthralgia, insomnia, sweats, diarrhea, constipation, weight changes, allodynia, rarely high output heart failure, and cachexia [33,34,40].

Only about 2-3% of reported psoriatic cases are erythrodermic psoriasis, but this disease is declared a dermatological emergency because it results in electrolyte disturbances and desquamation, which can be life-threatening [22].

Occasionally, erythrodermic psoriasis can lead to plaque psoriasis, and generalized pustular psoriasis may lead to erythrodermic psoriasis when pustule formation ceases. The possible factors that result in erythrodermic psoriasis are sunburns, skin trauma, emotional stress, systemic illness, drug or chemical exposure, and rapid withdrawal from corticosteroids or methotrexate [34,37,38,111]. To confirm the diagnosis, histologic analysis of erythrodermic psoriasis is done, which should show epidermal perivascular infiltrate of lymphocytes and eosinophils, hyperkeratosis, dilated capillaries, parakeratosis, acanthosis, spongiosis, Munro micro-abscesses, and seasonal apoptotic keratinocytes [38,41,42].

## 3.4 Generalized Pustular Psoriasis

Generalized pustular psoriasis (von Zumbusch) is also an uncommon variant. It occurs when tiny sterile pustules and erythema coalesce into pus lakes [43]. It is a very severe and acute variant of psoriasis associated with an interleukin 36 receptor antagonist (IL36RN) sequence variation [22]. It is initiated by abrupt withdrawal from steroids, hypocalcemia, pregnancy, upper respiratory tract infection, and phototherapy [44,45].

Generalized pustular psoriasis (GPP) and acute generalized exanthematous pustules (AGEP) share some features in common [46]. AGEP can be seen as drug-induced pustular psoriasis having contrasting features, treatment courses, and factors that trigger their expression [47].

Pustular psoriasis brings about recurrent waves of fever, which are then accompanied by outbreaks of pustules. More signs include leukocytosis, high erythrocyte sedimentation rate (ESR), weakness, weight loss, fever, neutrophilia, increased C-reactive protein, and liver abnormalities [35,47]. Pustular psoriasis could occur as a localized variant in palms (palmoplantar pustulosis) and sole of feet or as acrodermatitis continua of the Hallopeau variant affecting fingers and toes near nails (27,48].

## 3.5 Acral Psoriasis

This affects just the palms and soles. It shows characteristic hyperkeratosis, erythema, scales, a fissure in the distal phalanges of the finger, and shortening and tapering of the nails. It is noticeable at the margins of heels where rhagades can be seen [24,49,110].

## 3.6 Seborrheic Psoriasis

This occurs at the same sites where seborrheic dermatitis occurs following the Koebner phenomenon. These sites include the scalp, hair margin, eyebrows, nose, nasolabial folds, and pre-sternal and intertriginous areas. It is very similar to plaque psoriasis [49].

## 3.7 Flexural Psoriasis

Another name for flexural psoriasis is inverse or intertriginous psoriasis. The plaque observed is not like the conventional thick plaque forms. They are very thin and majorly seen under skin folds like axillae, submammary, groin, and natal cleft regions. Usually, very little or no scaling is observed in flexural psoriasis [50,51]. Flexural psoriasis lesions are well demarcated, show erythema, are shiny, most, and occasionally show fissure in the centre. The location of this variant makes it very susceptible to microbial invasion [52].

## 3.8 Clinal Presentation of Psoriasis in the Black Race

In Nigeria, the predominant race of the people is the black race, and the clinical presentation discussed in 3.1 to 3.7 above does more justice to the white race, thus, the need for this section.

Psoriasis presents differently in patients, not of the white race, and more attention should be given to these cases by dermatologists to learn about the clinical presentation and diagnosis [53].

In blacks, psoriasis is presented as violaceous or hyperpigmented plaques covering a large BSA, which is very different from the pink-red scales observed in white patients [54,55]. The violet or purple-like lesions observed in black patients have made it difficult to diagnose psoriasis in people with very dark skin tones. Psoriatic arthritis is not frequently reported in black patients compared to white patients. The black plaques are usually much thicker than those observed in white skin [55-57]. Generally, black patients have a higher psoriasis area and severity index (PASI) score than whites [58]. This might be because black patients do not seek medical attention as soon as the signs and symptoms are observed due to the belief that psoriasis does not affect blacks or general ignorance.

# 4. EPIDEMIOLOGY OF PSORIASIS IN NIGERIA

Approximately 125 million people worldwide suffer from one or more types of Psoriasis [59]. Although common in the West, psoriasis has rarely been reported among Africans and even fewer among Nigerians. As reported in the Global Psoriasis Atlas [60], the prevalence of psoriasis in Western Europe was estimated to be 1.81%, 1.45% in Central Europe, 0.47% in Eastern Europe, and 1.34% in high-income Americas. The value was much lower in Sub-sahara African countries, including Nigeria, with a rate of 0.33%. However, Enigbokan et al. [61] opined that the perceived rarity of psoriasis may be attributed to the lack of data and research on the condition. This is due to reports from recent studies demonstrating an increasing incidence of psoriasis among Nigerians [61]. This rising trend has also been observed with other skin diseases in the country. Akinboro et al. [62] asserted that the prevalence of skin diseases within the country has witnessed a gradual rise and shift from infectious to non-infectious. The authors attributed this shift to the developing economy, industrialization, improved healthcare facilities, and environmental cleanliness. These external factors may have triggered the diseases, contributing to the observed trends.

Several studies have investigated the prevalence and occurrence of different skin conditions in Nigeria. These reports indicated a high occurrence of papulosquamous disorders, eczema, dermatitis, hypersensitivity disorders, and autoimmune connective tissue disorders [62,63]. However, similar reports on psoriasis among the Nigerian population are very limited. It is important to highlight that only a few studies have examined the prevalence of psoriasis among Nigerians, excluding other related skin disorders. Additionally, the studies were conducted using data from patients who presented with psoriasis in dermatology outpatient clinics [64]. This further highlights the gap in psoriasis and the need for population-based research on this disorder to understand its prevalence and how it affects Nigerians.

Two of the studies available were conducted about three decades ago, while the other two are more recent. One such study investigated the prevalence of psoriasis vulgaris in the savannah regions of the country [65]. Although the study dates far back to the 1980s, the results provided valuable insights into the existence and incidence of psoriasis in the country. Obasi [65] reported that psoriasis had an incidence rate of 0.8%, appearing in only 44 of 5250 skin disorders examined. This study further highlighted the low morbidity rate among West Africans compared to Europeans and Americans.

Similarly, Jacyk [66] asserted that psoriasis is uncommon among Nigerians, with an estimated 0.8% of 9806 (78) skin disorders reported in northern Nigeria between 1977 and 1981. Compared with other African countries, Jacyk [66] reported that psoriasis had a lower incidence and burden rate in Nigeria. In contrast, the Global Psoriasis Atlas [60] statistics reported the same incidence rate in Nigeria and other Sub-Saharan African countries (0.33%). This is an exception to East-Subsaharan Africa, where the prevalence of psoriasis was 0.2%. According to Parisi et al. [5], these regional variations may be attributed to differences in climate conditions, geographical locations, genetic differences, age and sex. Hence, there are differences in incidence rates across different countries and continents.

However, more recent studies confirm the increasing occurrence of psoriasis in Nigeria [7,64]. Ayanlowo and Akingkugbe [7] studied the clinical patterns of Psoriasis and its predisposing factors. The study reported that 124 of 11,015 participants were diagnosed with psoriasis, giving a prevalence rate of 1.13%. However, Husain [64] reported an occurrence of 0.6% in a larger population of 39,037 examined between 2001 and 2021. These differences may be due to the geographical location where the studies were conducted. Ayanlowo and Akinkugbe [7] conducted their study in Southwestern Nigeria, where psoriasis is considered indigenous. Both studies reported that males were more likely to present with psoriasis and other skin conditions than females. However, Husain [64] opined that females have an earlier onset than males, as evidenced by mean onset ages of 27.6 and 32.2 years, respectively. While it is necessary to note the increasing trend in psoriasis cases among the Nigerian population, the available studies are conducted on patients who attend dermatology clinics and are not population-based studies. Therefore, population-based studies are required to accurately ascertain the prevalence and incidence of psoriasis in Nigeria and Africa.

# 5. ADVANCEMENTS IN THE DIAGNOSIS AND THERAPY OF PSORIASIS

Psoriasis is a chronic inflammatory disorder that requires various strategies to accurately diagnose and manage the symptoms [67]. Over the years, an increasing understanding of the pathogenesis of psoriasis has led to advancements in diagnosis and treatment [68]. Being a genetic condition, there are currently no curative therapies available. However, treatment strategies widely used focus on reducing the rapid spread of affected keratinocytes, enhancing the appearance of the condition on the skin and improving patients' quality of life [69]. These strategies comprise topical agents, biological agents, systematic agents, and phototherapy, administered based on the severity of the condition [22]. These therapeutic agents have demonstrated commendable success and comprise the arsenal of treatment options available to patients.

Topical agents have been the main-stay for managing mild psoriasis–scaly lesions that cover 3%-4% of the skin surface [70]. These include topical corticosteroids that reduce inflammation and inflammatory mechanisms and vitamin D analogues that promote keratinocyte differentiation and prevent rapid proliferation [69]. Additionally, topical calcineurin inhibitors block T-cell production of pro-inflammatory cytokines, while topical keratolytics clear up scaly psoriasis plaques [22]. Finally, ultraviolet light phototherapy targets skin cells, preventing their production and subsequent inflammation [71]. However, recent studies have reported advancements in moderate-to-severe psoriasis, which typically do not respond satisfactorily to topical agents [69]. Consequently, research is geared towards biologic agents known to regulate immune cell activation, differentiation and signaling pathways [72]. The aim of this is to provide more effective, precise and tolerable therapeutic options.

As defined by Bernstein [73], biologic agents are medications that block pro-inflammatory cytokines. These include drugs like Guselkumab, Risankumab and Tildrakizumab that inhibit interleukin-23 (IL-23), prevent inflammation and reduce psoriasis plaques on the skin [69]. More options include the recently approved IL-17 inhibitor (bimekizumab) and tumor necrosis factor blockers (deucravacitinib and apremilast) [74,114]. Although several biologic therapies are being introduced into the pipeline, many are limited by side effects and loss of efficacy, patient comorbidities, compliance with the treatment regimen, and resistance development in moderate-to-severe psoriasis [75]. These challenges have necessitated the search for longer-lasting personalized approaches to enhance patient outcomes and quality of life.

Studies have begun to investigate the use of genetic approaches to mitigate psoriasis recurrence and efficacy loss observed with biologic or topical agents [76]. GWAS is instrumental in diagnosing psoriasis and identifying specific genetic targets that are then used to design effective medications [68]. Consequently, Dand et al. [10] conducted a meta-analysis of GWAS to identify susceptibility alleles and potential therapeutic targets. The authors reported targets that have been identified using GWAS and used to produce successful medications. These targets include IL-23A and IL-23B found at the 12q13.3 and 5q33.3 psoriasis susceptibility loci, respectively. They were important in producing the agent that targets interleukin-23 (IL-23). Additionally, identifying the non-functional alleles of TYK2 was instrumental in developing deucravacitinib. Deucravacitinib inhibits the non-functional alleles, thereby increasing resistance to psoriasis. Furthermore, Dand et al. [10] identified genetic variants at the 5’ untranslated region of the interleukin receptor A (IL17RA) gene found on chromosome 22q11.1. This gene could potentially be targeted to reduce the inflammation associated with psoriasis and improve the array of precision-based therapeutics available.

Similarly, Brooks et al. [77] highlighted the importance of genome-based research to identify genetic markers. The identification of genetic markers allows precise therapeutics to be developed, increasing the amount of potent, long-lasting drugs [77]. An emerging gene-based strategy is the use of small interfering RNA (siRNA) technology. siRNA are double-stranded RNA molecules that cleave specific complementary mRNA before translation, inducing sequence-specific gene silencing [78]. Due to the specificity and efficiency of this process, siRNA can silence any target gene. Therefore, Zhao et al. [76] conducted a systematic study highlighting the efficacy of siRNA and novel siRNA-delivery systems as an option for the targeted treatment of psoriasis. The authors suggested that siRNA-mediated gene silencing can potentially prevent the proliferation and abnormal death of keratinocytes. This approach involves silencing genes that contribute to aberrant keratinocyte division, including Fibroblast Growth Factor Receptor 2, Nuclear Factor of Activated T Cells 2, and TRAF3 Interacting Protein 2 genes [76]. Doing this will diminish keratinocyte growth and development, conferring resistance to spreading psoriasis on the skin.

Additionally, Lee et al. [79] asserted that siRNA therapy effectively neutralizes the potency of proinflammatory cytokines and reduces their infiltration. The genes that may be silenced include ubiquitin-specific peptidase 15 and Keratin 17. Silencing these genes will reduce the proliferation and infiltration of cytokines that trigger inflammation. Despite the promising potential of this strategy, several challenges are associated with the development and delivery of siRNA therapy. While the most suitable method of administering siRNA therapy is the topical route due to the ease and reduced risk of adverse side effects, its widespread use and acceptance are limited by its large size and the skin barrier, making cellular uptake difficult [80,81]. Notwithstanding, current ongoing research is focused on developing efficient topical delivery systems to mitigate the challenges of administering siRNA therapy. This includes promising systems such as nanoparticles, ionic liquid combinations, and fractional laser-assisted nanocarriers [76,79-81]. The diagnostics and therapeutics of psoriasis are constantly evolving with advancements in understanding the disorder, its incidence and pathogenesis. This has led to advancements in genomic strategies and precision-based techniques that contribute to improvements in treating and diagnosing psoriasis.

# 6. CURRENT MANAGEMENT PRACTICES IN NIGERIA

Psoriasis is a chronic disease without a cure. Therefore, the management practice has to be life-long. Medications for psoriasis ought to have very high efficacy, availability, and cost-effectiveness. But this has not been the case in Nigeria. The treatment of psoriasis in Nigeria has faced significant limitations due to the high cost and non-availability of most treatment options [82]. These treatment options can be;

TOPICAL – effective for only mild to moderate cases [83]. Foladun and Sabir [82] showed that topicals such as betamethasone cream and calcipotriol posed no improvements in a 43-year-old psoriatic man who had the disease for five years. Further studies showed calcipotriol and betamethasone caused grave itching, thickness of plaque, and scaling in blacks [84,85]. Oil-based vehicles, emollient foams, and lotions are more effective in combating psoriasis [54]. Corticosteroids are also topical therapies for managing psoriasis in Nigeria [82].

SYSTEMIC – according to the Canadian Psoriasis Guidelines Committee [86], this is very effective in cases where topicals prove ineffective. They include phototherapy using psoralen plus UVA, broadband UVA, and narrow-band UVB, acitretin, methotrexate, cyclosporine, retinoids, hydroxyurea, fumarates, and biologic therapy [83,87]. All these are not cost-effective, as an average Nigerian cannot afford them, and they also pose some side effects. Retinoids can increase the risk of abnormalities in birth so pregnant women are advised to avoid this treatment, they also cause hair loss and lip irritation, and ultraviolet light from phototherapy can lead to thinning of the skin and can cause harm to the skin [88], methotrexate causes fatigue, stomach upset, liver damage over time, and reduced blood cells [87].

The current management practices in Nigeria call for the use of genomic tools for therapy, such as topical, oral, and systemic therapy, which are not cost-effective, unavailable, and have side effects.

# 7. BARRIERS AND SOLUTIONS TO IMPLEMENTING GENETIC AND GENOMIC TOOLS IN NIGERIA

Global Genome-Wide Association Studies (GWAS) carried out on psoriasis and other genetic diseases mostly employ sets of individuals and families in the West, excluding many African populations as such, creates a research gap and need for more data on African population genomics to address this gap [88,90]. Parisi et al. [5] corroborated this by highlighting considerable gaps that exist in research from these geographical areas, particularly low and middle-income countries, with studies conducted mainly in Europe and North America.

According to Dand et al. [10], results from genetics studies are sometimes population-specific due to the heterogeneity of genome composition across human populations and the exclusion of the African population which harbours the highest genetic diversity hinders global health equity. Also, it is expedient for extensive research to be done to develop databases and other platforms for easy access to data on genomic variants and their associated genes and diseases [91,115], especially among African populations as different populations must be sufficiently represented in genomic research [92].

However, efforts of research facilities in Nigeria such as the African Centre of Excellence for Genomics of Infectious Diseases (ACEGID) at the Redeemer’s University, and Covenant Applied Informatics and Communication Africa Centre of Excellence (CApIC-ACE) at Covenant University are currently bridging the genomics research gap. The Human Heredity and Health in Africa (H3Africa Consortium) initiative also corroborates the effort of research facilities in Nigeria by investing in the establishment of high-quality biorepositories in Africa, a bioinformatic network, and a strong training program that has developed skills in genomic data analysis and interpretation among bioinformaticians, wet-lab researchers, and healthcare professionals [93,116].

Therefore, emphasis must be placed on the inclusion of more populations especially those in Africa in genetics research to facilitate the implementation of genomics-based approaches for diagnostic and therapeutic purposes [94].

## 7.1 Paucity of Research

The goal of genomic research is to reveal an individual’s genetic predisposition to diseases, usually by establishing a link between genotype and phenotype through genome-wide association studies (GWAS). Genetics and genomics research in Nigeria is not as widespread as it should be due to a consortium of factors. Funding of genomic research which can be quite expensive in terms of cost, time, and computational resources required is important but only about 0.2 – 0.4% of Nigeria’s gross domestic product (GDP) is allocated to funding research and development projects [95]. Many genetic researchers in Nigeria operate in relatively resource-scarce environments and have to fund these capital-intensive research projects either out of pocket or with reliance on private and international grants. The distressing nature of obtaining research funding results in the abandonment of novel research and this has over time impeded the obtaining of genetic information on the Nigerian population. This hinders the capacity to contribute and compete effectively with their counterparts in larger and better-resourced groups in the analysis of genomics data generation.

Mechanisms that allow government authorities, industrial partners, and/or researchers in different African countries to pool resources together in establishing and strengthening regional research centres and research networks with common interests instead of working in isolation should be encouraged [96,116].

## 7.2 Shortage of Skilled Personnel

The complex nature of sequencing technologies, managing genomic datasets and carrying out genetic research drives a growing need to train a significant mass of African researchers with the essential skills and expertise to be able to contribute to research in this area. Another huge barrier in the implementation of personalized genomic medicine is the knowledge deficit among healthcare professionals as many clinical practitioners are out of touch with the latest trends in genomics technology and many are unable to interpret genetic testing results exhibiting an insufficient capacity to interpret genomic information for their patients [97].

To effectively utilize information obtained from genetics research, a solid foundation of skills, knowledge, and infrastructure to effectively apply genetic information in healthcare are imperative [98]. Focused efforts towards capacity building to create a significant pool of bioinformaticians for biomedical data analysis is important. This includes participatory genomic training programs for healthcare professionals such as mentorships, internships, workshops, hackathons, fellowships, and conferences where interested investigators can interact and learn from experts in genomics and personalized medicine [99].

## 7.3 Infrastructure

Nigeria is faced with a general lack of infrastructure which affects the availability of resources to support research and clinical translation, and this presents a major barrier to the implementation of genetic and genomic tools [93,100]. Investment is required in establishing the framework needed to facilitate comprehensive DNA analysis, DNA sequencing and genotyping facilities, establishment of biobanks for sample storage along with their associated data, and access to technology for researchers. Extensive data infrastructure, information management systems for efficient data generation, storage, and analysis systems, improvement of electronic health records, reliable internet connectivity, and establishment of facilities for clinical action and clinical trials are also essential.

African diversity and the African reference genome

Genetic diversity is largely observed across the Nigerian population, which is the most populous and most diverse country in Africa with 250 ethnic groups and over 500 different native languages [101]. Joshi et al. [102] published a study that described the whole-genome sequencing of 449 Nigerian individuals across 47 unique self-reported ethnolinguistic groups in the country and this emphasizes the need to necessitate an improved understanding of human ancestry and health.

## 7.4 Data Generation and Data Sharing

A general apathy toward clinical trials due to ethical and societal apprehensions of Nigerians surrounding the use of genomic technologies presents a problem with obtaining genetic data. Apart from these, poverty and insufficient healthcare funding have been largely responsible for patients presenting at health facilities at later stages of diseases which prevents them from contributing valuable genetic data during the early phases of disease progression [94].

More public enlightenment must be done to encourage more members of the public to volunteer as subjects in genetic studies and concerns should also be properly addressed through adequate education, orientation, and legislation where necessary.

# 8. RECOMMENDATIONS AND CALL TO ACTION

The NCD Alliance [103] asserted that the lack of access to adequate care facilities and resources that cater to skin disorders is a global problem. However, this problem is more pronounced in underdeveloped areas and further compounded by the shortage of healthcare workers specialized in dermatology. Although psoriasis is uncommon in Nigeria, studies have reported an increase in the number of non-infectious skin conditions that present in clinics around the country [64]. Despite this, diagnostic and treatment strategies against these skin disorders are poorly sufficient and unavailable in some regions of Nigeria.

The main limitation to advancing psoriasis treatment is the lack of awareness among the public. Only relatively few people know about the disorder in Nigeria. Additionally, disorders with low prevalence rates like psoriasis are poorlly publicized and tend to be dismissed with more attention focused on other health priorities across the country [104,113]. Further compounding the problem is the presentation of psoriasis, which causes people to think of it as a contagious and infectious disease. Hence, people tend to distance themselves from patients, leading to stigmatization and isolation [105]. Therefore, creating awareness about psoriasis, its causes, comorbidities and types is important. An increased understanding will significantly reduce the stigma associated with the condition [105]. This will result in enhanced knowledge of psoriasis and improved public perception, inclusion and acceptance.

Furthermore, Abdulghani et al. [106], who reviewed the management of psoriasis in Africa and the Middle East, suggested population-based research to provide in-depth data into the incidence and prevalence of psoriasis in African countries. The insights from this data will help to understand differences in how psoriasis affects populations in Africa and relative to the rest of the world. In addition, it will elaborate on factors that contribute to the observed differences and will stimulate the public’s interest in learning more about its occurrence and management strategies [107,110]. The Federal government and non-governmental organizations can collaborate with research institutes and tertiary institutions to conduct population-based studies. These studies will provide results that will inform diagnosis and therapeutics of psoriasis in the country.

On another note, access to healthcare facilities to diagnose and manage psoriasis is limited in Nigeria. Thus, patients with this condition will benefit from capacity building and investments in treatment resources and facilities [108,112]. Moreso, collaboration with national and international organizations will strengthen efforts to sustain public awareness of psoriasis and enhance management strategies. With increase in genetic tools for therapeutic purposes, several recent studies have elucidated the role of genetic and genomic tools to treat psoriasis [109,111]. Therefore, there is a need to invest in research geared towards the use of genetics to target psoriasis and integrate evidence-based genomic tools into healthcare practice in Nigeria. Additionally, healthcare workers may be trained to increase the workforce skilled and knowledgeable about managing psoriasis. These training will include the use of strategies ranging from topical agents to precision/personalized-based genomic tools, as they relate to the Nigerian population. With this, Nigeria will be better positioned to effectively respond to the rising trend of psoriasis incidence across the country.

**9. CONCLUSION**

Psoriasis affects about 2% of the world's population but is gravely underreported in many low and middle-income countries, with Nigeria inclusive. There is an excellent role of genetic predisposition in the course of psoriasis and essential genetic markers that contribute to how susceptible an individual is to the disease. The major clinical presentation of psoriasis is scaly, thick, and erythematous lesions with finely demarcated margins on patients' skin, with severity depending on the variant.

In Nigeria, the prevalence rate of psoriasis is meagre compared to Europe and Africa. Psoriasis is an incurable disease that has been managed over time with topical, oral, and systemic therapies. However, these therapeutic agents cause significant side effects in the patients, especially the blacks; in some cases, they are ineffective, unavailable in Nigeria, or too expensive for the average Nigerian.

Genomic tools pose great potential in finding a long-lasting solution to psoriasis. Still, there are many challenges to the actualization of genomic implementations in Nigeria, such as a lack of skilled personnel in the field of genomics and inadequate funding opportunities for genomic research. In essence, the government of Nigeria should allocate adequate funds to improve genomic research. Nigerians should also be sensitized about non-infectious diseases and the role genetics plays in them, seek appropriate medical attention when a disease is noted in their ancestry, and not resort to self-medication and other untested ethnomedical options.

**DISCLAIMER (ARTIFICIAL INTELLIGENCE**)

The author(s) hereby certify that no generative artificial intelligence (AI) tools such as Scalable Language Models (ChatGPT, COPILOT, etc.) or text-to-image generators were utilized in the authoring or editing of the paper.

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