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Challenges and Opportunities in Psoriatic Disease: An Integrated View of the Future

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ABSTRACT. Psoriatic disease (PsD), which includes cutaneous psoriasis (PsO) and psoriatic arthritis (PsA), affects 2% of the global population, and results in the development of comorbidities that adversely affect quality of life (QOL) and physical function. Recent advances in the field have allowed for earlier diagnosis of PsD and improved clinical strategies for care, including the use of innovative pathway-specific immune-targeted therapies. Despite these advances, there is no cure for PsD. Ongoing challenges in disease management include the need for adequate treatment response, precision-based care for individual patients, and a better understanding of the interrelationship between the pathogenesis of cutaneous PsO and PsO comorbidities, including PsA. Future progress may arise from integrating clinical disciplines, harnessing artificial intelligence, using molecular dissection to map out the disease pathogenesis of PsA to identify more effective treatment strategies, and exploring the interplay between PsD and comorbidities including cardiovascular disease, obesity, and depression. These developments could lead to personalized treatment approaches and increase the efficacy of therapeutics for PsD, ultimately improving patient outcomes and QOL. This article highlights the presentation of this topic at the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) 2024 annual meeting.

Key Indexing Terms: clinical trial, GRAPPA, inflammation, psoriasis, psoriatic arthritis, pathogenesis

Introduction

Together, psoriasis (PsO) and psoriatic arthritis (PsA) comprise psoriatic disease (PsD) and affect approximately 2% of the global population.[1] PsD is associated with increased disability, reduced quality of life (QOL), and accelerated mortality.[2] PsD is a prototypic immune-mediated inflammatory disease (IMID)[3-5] and—as is typical of such disorders—is associated with substantial comorbidities, particularly of the cardiovascular, metabolic, and neurologic/psychologic systems.[6] Remarkable advances have been made in the last 30 years in the management of PsD, including improved disease recognition and the advent of novel immune-targeted therapeutics such as biologic agents and small molecule inhibitors, offering earlier intervention and more targeted treatment approaches.[7] In this article, we highlight key ideas presented at the Group for Research and Assessment of Psoriasis and Psoriatic Arthritis (GRAPPA) 2024 annual meeting held in Seattle, USA, with a focus on current and future challenges and opportunities in PsD from a rheumatic and skin disease perspective.

Major challenges in PsD

Even though outcomes have improved for a substantial number of patients with PsD, significant unmet clinical needs remain and should be recognized (Figure 1). Persistent disease activity remains common, manifesting as consistent low-level tissue inflammation or as remission/flare cycles that cumulatively impair QOL and confer long-term disability.[8] The manifestations of chronic low-level inflammation in PsD are most evident in the skin (ie, PsO) but also include articular damage and destruction,[9] impaired cardiometabolic function,[10] and significant mental health challenges.[11] There exists a medical need to adequately mitigate clinically evident disease across a range of affected tissues, normalize immune dysregulation, correct tissue damage, and reduce associated accelerated comorbidities.[8] Consequently, a cure, loosely defined as sustained remission without medication beyond 5 years, remains an unattainable goal for the majority. Even remission, defined as completely clear skin or achievement of remission status per composite PsA measures (eg, Psoriatic Arthritis Disease Activity Score [PASDAS] or minimal disease activity [MDA]), is not normally maintained in a drug-free state.[12]

Two problems warrant particular attention. First, international guidelines and recommendations, such as those from GRAPPA or the European Alliance of Associations for

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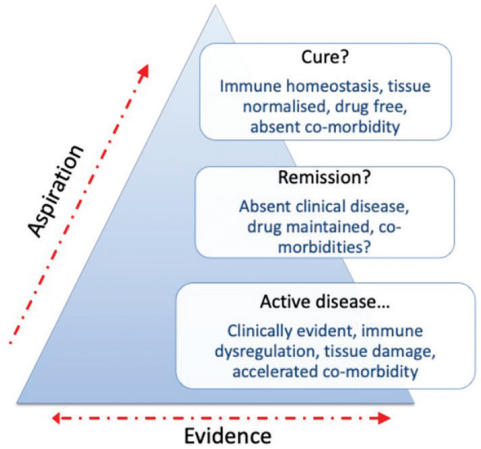

Figure 1. Critical challenges between now and 2030 for psoriatic disease: toward understanding each individual's molecular state of disease for precision medicine, and the current evidence base available for each challenge.

Rheumatology (EULAR),[13,14] facilitate shared decision making between patients and healthcare professionals, leading to consistent therapeutic decisions across populations and healthcare systems. However, these recommendations are based on formal systematic literature reviews, which rely on population-level datasets, making individualization to specific patients challenging.[15] Similarly, datasets submitted for US Food and Drug Administration or European Medicines Agency approval result in generic drug labels that are not entirely useful in the real-world clinical practice, where multimorbidity is common.[16] Moreover, in health systems that require insurance companies to preapprove treatment, patients must fail out of lower-cost therapies before advancing to more expensive systemic and biologic treatments (ie, step therapy), despite the physician and patient often knowing which biologic may provide more immediate and successful results.[17] Consequently, we are far from achieving precision medicine in routine clinical practice, and patient outcomes therefore suffer.

Second, the therapeutic revolution, which has been driven by discoveries in PsD pathogenesis, has culminated in the introduction of new cytokine inhibitors in PsO and PsA, including targeted inhibition of tumor necrosis factor, interleukin (IL)-12/23 p40, IL-17A, IL-17 receptor A, and IL-23 p19.[18-21] Nevertheless, a long-standing issue in PsD is the lack of appreciation of discrete immune regulatory networks, which are critical for disease development across the different tissue types involved in PsD.[22] The evolution of host immunological response requires distinctive local regulation of immunity within each tissue, including skin, enthesis, synovium, gastrointestinal mucosa, eye, vasculature, and others.[23] Assuming equivalent pathogenetic pathways across these different tissue sites is overly simplistic. Disparate responses to biologics that inhibit a single cytokine in different tissues (eg, IL-17 in skin vs inflammatory bowel disease [IBD], or IL-23 p19 in skin vs axial skeleton) highlight these immunopathological disparities.[24-27] Incorporating knowledge of tissue-specific differences in disease pathogenesis through identifying new targets and pathways will contribute to the development of new therapeutic treatments—including possibly repurposing already approved drugs—and better treatment approaches for preventing and/or reversing critical components of PsA, such as irreversible destructive arthritis, debilitating enthesitis, and other PsD comorbid conditions, including cardiovascular inflammation.[8,12] This progress, in addition to the identification of biomarkers for disease prediction, treatment response, and/or disease monitoring would represent a significant advance.[28]

Solutions over the next decade

Rapid broad-scoping advances in medical technologies will almost certainly facilitate a revolution in the management of PsD in the next decade. Here we highlight several key exemplary areas that would drive progress.

1. Increasing integration of distinct clinical disciplines (eg, dermatology, rheumatology, gastroenterology) into a core discipline—whimsically termed "IMIDology." This strategy will aggregate knowledge, optimize clinical development and research potential, and significantly advance external advocacy. This unifying approach would facilitate a greater understanding of the shared and discrete pathophysiology of the various IMIDs. The functional alignment of stakeholders across PsD (and IMIDs)[29,30] should be extended to include clinicians in practice, academicians, basic scientists, patient partners, industry partners, and government and policy makers. This in turn can greatly enhance free-flowing and more transparent access to knowledge, data, and advances from different specialties and disciplines. For instance, in gastroenterological clinical practice, a well-established approach is to use therapeutic drug monitoring (TDM)[31-34] to optimize biologic therapy in patients with IBD. TDM is less commonly practiced across rheumatology and dermatology and warrants further exploration in the treatment of IMIDs like PsD. Another interesting example would include a more detailed analysis of clinical trial datasets, including those that did not meet primary or key secondary outcomes; in essence, we should be prepared to learn equally from success and failure in the research domains. This could commence with retrospective evaluation of datasets from failed phase II/III trials over the last decade.

2. This decade of progress will occur concurrently with the increasing application of artificial intelligence (AI) and large language model (LLM)-based methodologies across all areas of PsD investigation and management. Various AI methodologies, such as convolutional neural networks, deep learning models, and machine learning (ML) classifiers are being trained to diagnose PsO through in silico learning, using clinical and dermoscopic images and analyses of images of skin lesions, which can predict severity scores like the Psoriasis Area Severity Index (PASI) and body surface area.[35] AI is also being used to identify potential PsO biomarkers, enhance precision medicine, and revolutionize education strategies.[36] Moreover, through harnessing high-quality data from multiomics, imaging, immunology, and/or electronic health records or collated clinical trial datasets, ML techniques can train models for biomedical prediction tasks such as prognosis or treatment recommendations.[37]

This will accelerate discovery at every level, ranging from disease pathogenesis, epidemiology (eg, using real-time routine data), imaging, and clinical trial data analysis to clinical practice in the real-world setting.

3. The field is shifting from targeting remission to considering a cure, with a focus on molecular dissection to elaborate pathways driving chronicity. Single-cell sequencing has provided new insight into individual cell biology, resulting in the generation of the Human Cell Atlas.[38,39] Spatial transcriptomics offer an additional layer of molecular understanding by examining individual cell biology within the context of topographical organization of a specific tissue in health and disease.[40,41] Examples of the application of spatial and combined single-cell transcriptional approaches identifying new pathogenic players in PsD are ever-increasing as these technologies become more mainstream. Castillo et al[42] and Ma et al[43] identified CD20+ B cells and fibroblasts as key contributors to disease severity, with significant differences in immune cell distribution and gene expression profiles between mild and severe cases. Further, proteomic analysis revealed the enrichment of diacylglycerol O-acyltransferase 2 (DGAT2) and fibroblast growth factor receptor 3 (FGFR3) in nonlesional skin of patients with severe PsO, suggesting metabolic alterations.[42] Ma et al also identified distinct cell-cell interactions not previously thought of as pathogenic for PsD but now shown to link to the genetic risk factors of disease.[43] Most notably, this technique allowed the identification of discrete synovial neighborhoods in rheumatoid arthritis (RA), with the predominance of each neighborhood varying based on the level of clinical disease activity and phase of disease.[44] The capacity to use these approaches in PsD to study and compare synovium from patients with PsA to those with RA and osteoarthritis (OA) is likely to provide remarkable new insights as to the hierarchies that remain in the immune responses across different tissue sites in PsD and across different types of arthritis. Such 4-D pathway deconvolution (3-D in tissue and over the fourth dimension of time with repeat tissue examination under pressure of an intervention) will identify new therapeutic targets, increase precision in the application of drugs directed at these targets (and potentially existing medicines), and enhance understanding of the molecular state of a patient at a given moment in time.[45] This definition of "state" will move the PsD field—and indeed the IMID field—to more appropriate interventions based on disease stage, which is currently crudely defined by the prior therapeutic exposure (eg, MTX–inadequate responder [IR], biologic-IR). The implications for clinical trials are profound, enabling molecular signature-based patient stratification,[42,46] increased trial success, reduced risk for adverse events, and improvements in cost-time efficiency. Head-to-head studies could potentially determine optimal treatment choices for distinct patient subsets, leveraging deeper molecular resolution to reduce clinical heterogeneity.

4. The relationship of PsD to common comorbidities is increasingly recognized to be mechanistically intertwined (Figure 2). Thus, the next decade should also include investigation of the mechanisms whereby cardiometabolic diseases,[9] obesity,[47] and mental health disorders[11] interact with PsD.

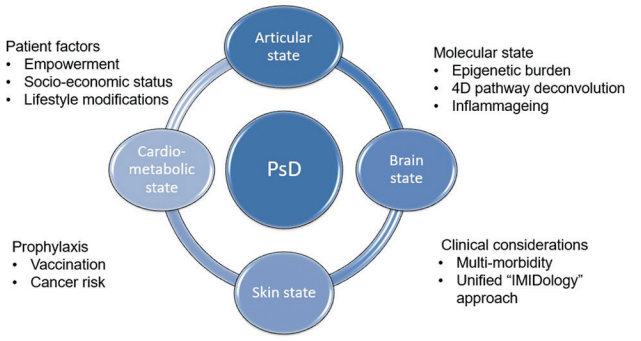

Figure 2. The targets of PsD care and the different considerations for clinical management and research. PsD: psoriatic disease.

Increasing application of functional imaging and tissue biopsy dissection will offer key insights to the multidirectional pathogenesis that drives the poorer clinical outcomes associated with multimorbidity. Future studies evaluating the effect of lifestyle modifications and their interactions with current treatment strategies may provide insight into potential synergy between these.[48] Similarly, the application of fine molecular imaging of the brain will bring a new molecular understanding of the pathways that drive pain,[49] fatigue,[50] depression,[51,52] and cognitive dysfunction[53]—features that are common in PsD. Taken together, the opportunities to generate a molecular map of PsD across different tissue types could be transformative (Figure 2).

5. Potential exists for a more detailed mechanistic understanding of PsD beyond previously described inflammatory pathways. Although elucidating the roles of cytokines and cell signaling pathways led to important and efficacious targeted therapies, a significant number of patients continue to experience pain despite the apparent suppression of known inflammatory pathways.[54] An evolving understanding of the pathologic mechanisms of nociceptive pain may allow more holistic treatment of such patients and higher rates of complete clinical response. Understanding nociceptive pain pathways and their treatment interventions may also help destigmatize patients continuing to experience noninflammatory pain even after having benefited from current targeted therapies.

Conclusion

Various long-standing issues preclude the successful implementation of precision medicine into clinical practice for the ultimate goal of curing PsD. These issues range from a lack of understanding about tissue-specific molecular and immunoregulatory networks to reliance on population-level datasets, which often do not reflect real-world practice, for informing treatment guidelines. Here, we discussed a multifaceted strategy that could propel the next therapeutic revolution in PsD. At a molecular level, single-cell sequencing coupled with spatial transcriptomics is increasingly shedding light on novel mediators of disease, paving the way for stratifying patients according to their molecular state. AI can be harnessed to accelerate multiomics research while assimilating epidemiological data, imaging results, and electronic health records to predict prognosis and manage-

ment recommendations. Moreover, there is a need to establish a more integrated approach to investigating and managing PsD. This could be achieved by encouraging greater free-flowing exchange of data, knowledge, and research advances between the specialties spanning IMIDs; focusing efforts toward uncovering the intertwining pathophysiological processes that link PsD to common comorbidities; and further exploring the mechanisms of nociceptive pain to improve patient QOL.

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ETHICS AND PATIENT CONSENT

Institutional review board approval and patient consent were not required.

PEER REVIEW

As part of the supplement series GRAPPA 2024, this report was reviewed internally and approved by the Guest Editors for integrity, accuracy, and consistency with scientific and ethical standards.

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