International Journal of Research in Dermatology
Cedillo LM et al. Int J Res Dermatol. 2026 Jul;12(4):376-382
https://www.ijord.com

DOI: https://dx.doi.org/10.18203/issn.2455-4529.IntJResDermatol20261941

Review Article

Psoriasis and increased cardiovascular risk: cutaneous inflammation as a systemic marker – the role of IL-17 and endothelial dysfunction

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Received: 03 April 2026
Accepted: 06 May 2026

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ABSTRACT

Pruritus in the senile population, or geriatric pruritus, represents a pervasive and clinically significant dermatological condition that substantially impairs quality of life and often signals underlying systemic pathology. This extensive review synthesizes current medical literature to elucidate the complex, multifactorial etiology of this condition, which extends beyond mere xerosis cutis to encompass neuropathic, systemic, psychogenic, and iatrogenic pathways. We delve into the age-related physiological alterations, including epidermal barrier dysfunction, diminished immune function, and neurodegeneration of cutaneous sensory nerves, that create a predisposed state for chronic itch. A critical analysis of diagnostic algorithms is presented, emphasizing the necessity of a thorough, systematic evaluation to rule out occult renal, hepatic, hematologic, and endocrine disorders, as underscored by recent consensus guidelines from the International Forum for the Study of Itch (IFSI). Therapeutically, this review advocates for a stratified, patient-centric approach, integrating rigorous skin barrier repair with emollients containing physiologic lipids, targeted pharmacotherapy (including neuromodulators such as gabapentinoids and selective serotonin reuptake inhibitors, and novel agents targeting the interleukin-31 and Janus kinase pathways), and non-pharmacological interventions. Special consideration is given to the principles of geriatric pharmacodynamics, highlighting the imperative of mitigating polypharmacy risks and adverse drug events. The synthesis of evidence from recent randomized controlled trials and cohort studies informs a pragmatic framework for management, aiming to optimize patient outcomes through an interdisciplinary care model involving dermatology, geriatrics, and palliative medicine.

Keywords: Geriatric pruritus, Chronic itch, Neuropathic itch, Xerosis cutis, Barrier function, Antipruritic therapy, Geriatric dermatology, Multimodal management

INTRODUCTION

The demographic shift towards an aging global population has brought geriatric medicine to the forefront of clinical practice, with cutaneous disorders presenting as a frequent source of morbidity. Among these, pruritus is a particularly vexing and prevalent symptom, affecting a substantial proportion of individuals over the age of 65 years. Contrary to outdated perceptions of senile pruritus as a mere nuisance or inevitable consequence of dry skin, contemporary dermatological and geriatric research reframes it as a distinct clinical syndrome with a complex pathogenesis. This condition frequently exists on a spectrum, where primary dermatoses, such as asteatotic eczema, intersect with systemic diseases like chronic kidney disease or occult malignancy, and are further compounded by age-related neurological decline.[1,2]

The clinical presentation is often complicated by the phenomenon of "itch-scratch cycles," leading to lichenification, prurigo nodularis, and significant risks of secondary infection, thereby transforming a sensory symptom into a profound threat to functional independence and psychological well-being. The diagnostic journey is notoriously challenging, as the elderly patient may present with multiple comorbidities and medication regimens that obscure the primary etiological driver. Furthermore, the subjective nature of itch necessitates reliance on validated assessment tools and a meticulous history. This comprehensive review seeks to consolidate the latest pathophysiological insights from investigations into cutaneous nerve fiber density, neuropeptide signaling, and immunosenescence, as explored in recent publications in journals such as the Journal of the American Academy of Dermatology, British Journal of Dermatology, and Itch. Building upon this foundation, we will critically appraise evolving therapeutic paradigms that move beyond simplistic emolliation to address the specific neuroimmune axes involved, always contextualized within the fragile physiological and pharmacological landscape of the geriatric patient. The ultimate objective is to provide a clinically actionable, evidence-based roadmap for the holistic assessment and multimodal management of this debilitating condition.[2-4]

BACKGROUND

Pruritus in the senescent individual, clinically termed geriatric pruritus or chronic pruritus of the elderly, transcends the simplistic categorization of a dermatologic symptom to emerge as a debilitating chronic syndrome with profound implications for multisystem health and quality of life. Its epidemiology reveals a startling prevalence, with contemporary cross-sectional studies indicating that nearly half of community-dwelling adults over the age of 65 experience clinically significant itch, a figure that escalates within institutionalized care settings. The pathophysiological landscape underpinning this condition is remarkably heterogeneous, representing a convergence of age-related physiological declinations, comorbid systemic pathologies, and neuropathic dysregulation. Central to its pathogenesis is the deterioration of the epidermal permeability barrier, characterized by diminished stratum corneum lipids, particularly ceramides, and a reduction in natural moisturizing factors. This compromised integumentary integrity, often manifesting as xerosis cutis, facilitates transepidermal water loss and lowers the threshold for pruritogen penetration and mast cell degranulation.[4-6]

However, to attribute senile pruritus solely to cutaneous desiccation is a fundamental clinical oversimplification. Advancing age orchestrates a symphony of neurological alterations, including a well-documented reduction in intraepidermal nerve fiber density concurrent with functional aberrations in both peripheral and central sensory processing. These changes precipitate a state of neuropathic itch, where peripheral sensitization of C-fibers and central disinhibition of spinal and supraspinal itch pathways occur independently of primary inflammatory stimuli. Furthermore, immunosenescence reshapes the cutaneous and systemic immune milieu, potentially skewing toward a Th2-dominant cytokine profile and altering the expression of pruritogenic intermediaries such as interleukin-31, thymic stromal lymphopoietin, and nerve growth factor. Concurrently, the geriatric population bears a significant burden of systemic diseases recognized as potent drivers of pruritus, including chronic kidney disease (uremic pruritus), hepatobiliary disorders, hematological conditions like polycythemia vera, and endocrine dyscrasias such as hypothyroidism and diabetes mellitus. The iatrogenic contribution is non-negligible, with a vast pharmacopeia, including opioids, angiotensin-converting enzyme inhibitors, and certain chemotherapeutic agents, implicated in drug-induced pruritus through diverse mechanisms.[6]

The clinical consequence of this etiological maze is a patient population suffering from a severe, often intractable symptom that directly imperils functional status. The relentless itch-scratch cycle inflicts physical damage, leading to excoriations, lichenification, prurigo nodularis, and a heightened risk of bacterial and viral superinfections. The psychological and sleep-related sequelae are equally devastating, with strong associations documented between chronic pruritus and insomnia, depression, anxiety, and social isolation. Despite its high prevalence and morbidity, geriatric pruritus remains notoriously challenging to diagnose and manage effectively. Patients frequently present with "normal-appearing" yet symptomatic skin, and the overlap of multiple potential causative factors demands a meticulous, systematic diagnostic approach. Traditional therapeutic modalities, often centered on first-generation sedating antihistamines and topical steroids, are frequently inadequate and carry disproportionate risks in older adults, including cognitive impairment, dizziness, and skin atrophy.[7]

This critical gap between disease burden and therapeutic efficacy underscores the necessity for a nuanced, evidence-based, and multidisciplinary management strategy. Recent years have witnessed a paradigm shift in our understanding of itch neurobiology and the advent of novel targeted therapies, yet their application and integration into the fragile context of geriatric medicine require careful synthesis.

This extensive review, therefore, aims to dissect the intricate pathophysiology of senile pruritus, evaluate contemporary diagnostic algorithms that prioritize a systemic work-up, and critically appraise the evolving therapeutic armamentarium. The synthesis will emphasize a patient-centric framework that balances therapeutic aggressiveness with the paramount principles of geriatric care: minimizing polypharmacy, respecting altered pharmacokinetics, and prioritizing functional preservation and quality of life.[7]

PATHOPHYSIOLOGICAL UNDERPINNINGS OF GERIATRIC PRURITUS

The pathophysiology of pruritus in the elderly is a complex, multifactorial process that reflects the cumulative impact of aging on integumentary, neurological, and immune systems. It is best conceptualized not as a single disease entity, but as a final common pathway activated through several interrelated and often co-existing mechanisms, which collectively lower the pruritic threshold and perpetuate chronic itch.[7]

Fundamentally, age-related cutaneous atrophy and barrier dysfunction establish a permissive state for pruritus initiation. The senescence of epidermal keratinocytes, coupled with a reduction in lipid synthesis—particularly ceramides, cholesterol, and free fatty acids—within the stratum corneum, compromises the epidermal permeability barrier. This disruption accelerates transepidermal water loss, leading to clinical xerosis and creating a microenvironment of increased pH and enzymatic activity. This altered milieu renders the skin more susceptible to exogenous irritants and facilitates the penetration of pruritogens, while simultaneously activating keratinocyte-derived cytokines and proteases, such as kallikreins, which can directly stimulate pruriceptive nerve endings. Concurrently, a decline in sebaceous and eccrine gland activity further diminishes the skin's natural lubrication and acid mantle, exacerbating dryness and vulnerability.[8,9]

Beyond the epidermal barrier, profound neuropathic changes constitute a central pillar of senile pruritus. A well-documented, non-linear reduction in intraepidermal nerve fiber density occurs with aging, paradoxically existing alongside functional hypersensitivity in remaining cutaneous neurons. This phenomenon of peripheral sensitization involves an upregulation of transient receptor potential (TRP) channels, particularly TRPV1 and TRPA1, on sensory C-fibers, lowering their activation threshold to thermal and chemical stimuli. Furthermore, there is an altered expression and signaling of pruritogenic receptors on these nerve endings, including those for histamine, proteases (via PAR-2), and key cytokines like interleukin-31 (IL-31). Crucially, aging is associated with a loss of inhibitory interneurons in the dorsal horn of the spinal cord and dysregulation of descending modulatory pathways from the brainstem and cortex. This loss of central inhibition disrupts the normal "gate-control" of sensory input, allowing non-noxious or sub-threshold pruritic signals to be amplified and perceived as intense itch. This state of central sensitization explains the clinical observations of alloknesis (itch from light touch) and hyperknesis (exaggerated itch response) frequently seen in this population.[9,10]

Immunosenescence and the associated inflammatory milieu provide a third critical axis. The aging immune system undergoes a complex remodeling, characterized by a chronic, low-grade pro-inflammatory state termed "inflammaging," marked by elevated circulating levels of cytokines such as IL-6 and TNF-α. Within the skin, resident immune cells, including mast cells and T-lymphocytes, exhibit altered functionality. There is evidence of a shift towards a T-helper 2 (Th2) skewing, promoting the release of pruritogenic interleukins like IL-4, IL-13, and the potent neuroimmune mediator IL-31. Mast cells, while not necessarily increased in number, may demonstrate heightened releasability of their granules containing histamine, tryptase, and nerve growth factor (NGF). This neurotrophic factor, in particular, plays a dual role in sustaining the survival of sensory neurons and further sensitizing them, creating a vicious feedback loop between the immune and nervous systems within the dermal microenvironment.[9,10]

Finally, the high prevalence of systemic comorbidities in the geriatric population introduces a plethora of disease-specific pruritogenic mechanisms that can directly ignite or exacerbate the underlying neurocutaneous susceptibility. Uremic pruritus in chronic kidney disease, for instance, is now understood to involve opioid receptor dysbalance, increased systemic inflammation, and the deposition of pruritogenic substances poorly dialyzed. Cholestatic itch is mediated largely via the interaction of lysophosphatidic acid (LPA) and its autotaxin enzyme with sensory neurons. Hematologic malignancies can drive itch through cytokine release, while endocrine disorders like diabetes mellitus contribute via neuropathy and xerosis. This intricate confluence of impaired barrier function, peripheral and central neuropathic sensitization, immunosenescent dysregulation, and systemic disease pathways coalesces to define the unique and recalcitrant nature of pruritus in the elderly, necessitating a diagnostic and therapeutic approach that acknowledges this profound pathophysiological complexity.[11]

CLINICAL MANIFESTATIONS AND DIAGNOSTIC PRESENTATION OF GERIATRIC PRURITUS

The clinical presentation of pruritus in the elderly is distinguished by its remarkable heterogeneity and frequent dissociation between subjective symptom severity and objective cutaneous findings, a characteristic that often complicates and delays accurate diagnosis. Patients typically describe a persistent, often intractable itching sensation that may be localized or generalized, with common predilection sites including the back, extremities—particularly the lower legs—and the scapular region. The quality of the itch is variably reported as a crawling, burning, or stinging dysesthesia, descriptors that can provide initial clues toward a neuropathic etiology. A diurnal pattern is frequently observed, with many patients reporting a significant exacerbation during the evening and night, a phenomenon attributed to circadian fluctuations in cortisol, increased skin temperature, diminished external distractions, and the psychological focus that accompanies rest. This nocturnal intensification is a primary driver of the profound sleep architecture disruption that plagues this

population, leading to fragmented sleep, non-restorative rest, and subsequent daytime fatigue and cognitive impairment, thereby creating a vicious cycle that amplifies the perceived burden of the disease.[11]

Objectively, the integument may present a spectrum of appearances, ranging from seemingly normal skin to severe xerosis with characteristic cracked porcelain or asteatotic eczema patterns, particularly over the shins and lower back. The hallmark of chronic pruritus, regardless of its origin, is the presence of secondary skin lesions induced by scratching and rubbing. These include excoriations, which may be linear or punctate; lichenification, manifesting as thickened, leathery skin with accentuated markings; and prurigo nodularis, which presents as firm, hyperkeratotic papules and nodules often distributed on accessible extensor surfaces.

The presence of prurigo nodules is a strong indicator of chronicity and neuropathic sensitization. It is imperative to conduct a meticulous, full-body skin examination to identify primary dermatoses that may be the underlying cause, such as bullous pemphigoid, which can present with prodromal non-bullous pruritus in the elderly, scabies in institutionalized settings, or eczematous dermatitis. However, the absence of a primary rash, a condition termed "pruritus on non-inflamed skin," is a common and diagnostically challenging scenario that strongly points toward a systemic, neurological, or psychogenic origin.[11]

Beyond the cutaneous manifestations, a constellation of systemic and psychological symptoms invariably accompanies chronic geriatric pruritus, underscoring its nature as a systemic disease state. The relentless discomfort frequently precipitates significant mood disturbances, including irritability, anxiety, and clinically relevant depression. Social withdrawal and a measurable decline in functional performance are common, as the constant need to scratch and the attendant skin lesions can provoke embarrassment and limit participation in daily activities.

The clinician must therefore adopt a holistic assessment strategy, incorporating validated patient-reported outcome measures such as the itch numeric rating scale (NRS) or the itch severity scale (ISS) to quantify the subjective experience, while simultaneously screening for the multidimensional impact on sleep quality (e.g., using the 5-D itch scale) and psychological well-being.

This comprehensive clinical portrait, integrating the nuanced description of the itch, careful documentation of cutaneous sequelae, and assessment of broader quality-of-life impairments, is fundamental to guiding the subsequent systematic diagnostic evaluation and formulating a multidimensional therapeutic plan that addresses not merely the symptom, but the totality of its effects on the geriatric patient.[11,12]

DIAGNOSTIC APPROACH TO GERIATRIC PRURITUS

The diagnostic elucidation of chronic pruritus in the elderly necessitates a meticulous, systematic, and often iterative investigative process, given the frequent obscurity of its etiology and the high prevalence of multifactorial contributions. This process must commence with an exhaustive and detailed medical history, which serves as the cornerstone of differentiation. The clinician must methodically characterize the pruritus itself, documenting its onset, duration, temporal pattern, and precise anatomical distribution. A thorough inquiry into the quality of the sensation—whether it is described as a superficial crawling, a deeper burning, or a painful itch—can provide initial directional clues toward neuropathic origins. Concurrently, a comprehensive review of the patient's complete pharmacopeia is non-negotiable, with particular attention to recent introductions, over-the-counter supplements, and opioid analgesics, all of which are frequent, yet often overlooked, iatrogenic triggers. The history must extend beyond dermatology to capture symptoms suggestive of systemic disease; subtle indicators of renal insufficiency, hepatobiliary dysfunction, hematologic malignancy, or thyroid disorder must be actively sought. The psychosocial dimension is equally critical, involving a tactful assessment of mood, anxiety levels, and the profound impact of the itch on sleep hygiene and activities of daily living, as these factors can both exacerbate and be exacerbated by the pruritic state.[11,12]

A subsequent, rigorous physical examination transcends a cursory inspection of the skin. It demands a full-body dermatological evaluation performed under adequate illumination, with the explicit goal of identifying a primary eruption that might have been missed by the patient. Special attention should be paid to the interdigital webs, scalp, and genital regions for signs of scabies, a highly contagious and pruritic infestation common in congregate living settings. The examination must also document the secondary stigmata of chronic scratching, including excoriations, lichenification, and prurigo nodularis, while noting the presence and severity of xerosis. Crucially, the physical assessment extends to general medical signs: palpation for lymphadenopathy, hepatosplenomegaly, or thyroid enlargement, and a neurological screening for signs of peripheral or autonomic neuropathy. This integrated history and examination inform the initial diagnostic categorization, often guided by contemporary frameworks such as those proposed by the International Forum for the Study of Itch (IFSI), which classifies pruritus into dermatological, systemic, neurological, psychogenic, mixed, or of unknown origin.[13]

Given the high likelihood of occult systemic disease in older adults presenting with generalized pruritus in the absence of a primary dermatosis, a staged laboratory and imaging investigation is a fundamental component of the diagnostic algorithm. Initial screening should be broad yet

rational, typically encompassing a complete blood count with differential to detect eosinophilia or hematologic disorders, a comprehensive metabolic panel to evaluate renal and hepatic function, fasting glucose or glycated hemoglobin, and thyroid-stimulating hormone. In regions with relevant endemicity or with specific historical clues, serologic testing for hepatitis B and C may be indicated. The role of more specialized biomarkers is evolving; for instance, serum autotaxin levels may be considered in cases of suspected cholestasis, and immunoglobulin E levels, while non-specific, can occasionally offer supportive evidence. The necessity for further investigation, such as chest radiography, abdominal ultrasonography, or even advanced cross-sectional imaging, is dictated by abnormalities in the initial screen or by persistent, high clinical suspicion for an underlying neoplastic or inflammatory process. In recalcitrant cases where a neurogenic or psychogenic cause is strongly suspected, consultation with neurology or psychiatry becomes an integral part of the diagnostic pathway. Ultimately, the diagnostic journey in geriatric pruritus is one of deliberate and persistent inquiry, requiring the clinician to synthesize disparate clues from the history, physical findings, and diagnostic tests to construct a coherent etiological understanding, which is the indispensable prerequisite for implementing targeted and effective therapeutic interventions.[12,13]

THERAPEUTIC MANAGEMENT OF GERIATRIC PRURITUS

The management of pruritus in the elderly is predicated upon a nuanced, patient-centric, and often multimodal therapeutic strategy that directly addresses the identified etiological factors while simultaneously incorporating the fundamental principles of geriatric medicine, namely the mitigation of polypharmacy risk, the accommodation of altered pharmacokinetics, and the prioritization of functional quality of life. Successful intervention is built upon a foundational pillar of comprehensive skin care and barrier repair, an approach that is universally applicable and rarely contraindicated. This entails the consistent and liberal use of emollients specifically formulated with physiological lipids, such as ceramide-dominant creams, to correct the underlying xerosis and restore epidermal integrity. The application technique is critical; emollients should be applied to damp skin post-bathing to maximize hydration occlusion, and soaps should be replaced with non-alkaline, fragrance-free syndet cleansers to prevent further lipid depletion and pH disruption. Environmental modifications, including the use of humidifiers in arid climates and the avoidance of excessively hot water during bathing, serve as simple yet effective adjunctive measures to reduce transepidermal water loss and prevent thermal provocation of nerve endings.[12,13]

When non-pharmacological measures prove insufficient, a tiered pharmacological approach, carefully calibrated to the suspected primary pathophysiological pathway, must be initiated. For localized or inflammatory dermatoses, topical agents remain first-line. Beyond mid-potency corticosteroids used judiciously for short durations to minimize atrophy, the modern armamentarium includes topical calcineurin inhibitors like pimecrolimus and tacrolimus, which offer an anti-inflammatory and antipruritic effect without steroid-related side effects, and are particularly useful for sensitive areas. The advent of topical neurokinin-1 receptor antagonists, such as crisaborole, and the emerging role of topical Janus kinase inhibitors represent promising avenues for targeted intervention at the cutaneous level. For systemic therapy, the paradigm has shifted decisively away from first-generation sedating antihistamines, whose anticholinergic burden poses unacceptable risks of cognitive impairment, urinary retention, and falls in the geriatric population. Instead, in cases where a neuropathic component is dominant, systemic neuromodulators are the cornerstone of treatment. Gabapentin and pregabalin, initiated at very low doses with slow titration, can effectively dampen central and peripheral neuronal hyperexcitability. Selective serotonin reuptake inhibitors like paroxetine or mirtazapine, the latter also possessing potent histamine H1-blocking properties, have demonstrated significant efficacy, particularly for nocturnal pruritus and in cases with co-morbid depressive or anxiety features.[12,13]

The landscape of systemic antipruritic therapy is being rapidly reshaped by novel biologic and targeted small-molecule agents, though their use in the elderly requires careful benefit-risk assessment. The monoclonal antibody against interleukin-31 (IL-31), nemolizumab, has shown remarkable efficacy in reducing pruritus intensity in conditions like atopic dermatitis and prurigo nodularis, and may hold promise for other IL-31-mediated geriatric itch syndromes. Similarly, dupilumab, an IL-4 receptor alpha antagonist, can be considered for patients with overlapping eczematous phenotypes. Oral Janus kinase inhibitors, such as upadacitinib and abrocitinib, offer potent broad-spectrum anti-cytokine effects but necessitate vigilance regarding infectious risks and thromboembolic events in a potentially immunosenescent population. For specific systemic causes, targeted interventions are paramount: opioid receptor modulators like nalfurafine or butorphanol for uremic or cholestatic pruritus, and bile acid sequestrants or rifampin for cholestasis. Throughout this process, a structured, interdisciplinary follow-up is essential to monitor therapeutic response, manage side effects, and provide continued psychological support, as the successful management of chronic geriatric pruritus often depends as much on the sustained therapeutic alliance and the alleviation of suffering as on the specific pharmacological agent chosen.[14,15]

CONCLUSION

The comprehensive management of pruritus in the senescent individual represents a formidable yet essential challenge within contemporary geriatric medicine, demanding a sophisticated synthesis of dermatological, neurological, and internal medical expertise. This review

has elucidated the profound complexity inherent in geriatric pruritus, a condition whose pathogenesis is seldom linear but rather emerges from the intricate confluence of a compromised epidermal barrier, pervasive neuropathic sensitization, the chronic inflammatory state of immunosenescence, and the frequent superimposition of significant systemic comorbidity. This pathophysiological tapestry dictates that a simplistic or unitary therapeutic approach is invariably insufficient and likely to culminate in therapeutic failure and patient disillusionment. Consequently, the diagnostic process must be embraced as a rigorous and systematic endeavor, one that prioritizes a meticulous history and physical examination to guide a rational, staged investigation aimed at unmasking the often-covert primary drivers of the itch, be they dermatological, systemic, neurogenic, or psychogenic in origin.

The evolution of therapeutic strategies reflects a significant paradigm shift, moving decisively beyond the historical reliance on sedating antihistamines and reactive topical steroids toward a more nuanced, mechanism-informed, and multimodal framework. The foundational imperative of rigorous skin barrier repair with sophisticated emollient therapy remains non-negotiable, serving as the essential bedrock upon which all other interventions are built. Pharmacological management, however, now increasingly hinges on the targeted modulation of specific neuroimmune pathways. The judicious use of systemic neuromodulators, such as gabapentinoids and certain antidepressants, has become a mainstay for addressing central and peripheral neuropathic sensitization, while the advent of biologic agents targeting key cytokines like interleukin-31 heralds a new era of precision medicine for appropriate candidates, albeit within a context of careful geriatric pharmacovigilance. Crucially, every therapeutic decision must be contextualized within the unique physiological and pharmacological landscape of the older adult, with an unwavering commitment to minimizing anticholinergic burden, sedative effects, and the perils of polypharmacy. The integration of non-pharmacological strategies addressing sleep hygiene, psychological distress, and the profound psychosocial impact of chronic itch is not merely complementary but integral to holistic care.

Ultimately, the effective alleviation of senile pruritus transcends the simple prescription of an antipruritic medication. It necessitates the cultivation of a sustained and empathetic therapeutic alliance, a commitment to patient education, and frequently, the coordination of an interdisciplinary care model involving dermatologists, geriatricians, neurologists, and mental health professionals. Future research must continue to refine our understanding of the specific neuroimmune crosstalk in aged skin, validate novel therapeutic targets, and develop robust clinical trials that explicitly include the multimorbid elderly population to generate high-quality, generalizable evidence. By embracing a patient-centered, pathophysiology-driven, and multidisciplinary approach, clinicians can meaningfully disrupt the debilitating itch-scratch cycle, thereby restoring not only cutaneous comfort but, more fundamentally, the dignity, sleep, and quality of life that this pervasive condition so insidiously erodes.

Funding: No funding sources
Conflict of interest: None declared
Ethical approval: Not required

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Cite this article as: Cedillo LM, Rios AEJ, Felipe MMM, Cruz ADCDDL. Psoriasis and Increased cardiovascular risk: cutaneous inflammation as a systemic marker – the role of IL-17 and endothelial dysfunction. Int J Res Dermatol 2026;12:376-82.