Review

Behavioral Feedback Loops and Kinesiophobia in Hidradenitis Suppurativa: A Macroscopic Network

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Behavioral Feedback Loops and Kinesiophobia in Hidradenitis Suppurativa: A Macroscopic Network

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Abstract

Hidradenitis suppurativa (HS) is a severe inflammatory dermatosis characterized by profound localized pain. Current pathophysiological models of HS focus primarily on microscopic molecular networks and microbiological dysbiosis. Although the psychosocial and behavioral burdens of the disease are individually well-documented, these factors have not yet been integrated into a single macroscopic feedback model. This self-sustaining system operates across three interacting domains: (1) a biomechanical-metabolic loop, where sustained immobility accelerates the accumulation of visceral adiposity and insulin resistance; (2) a psychosocial-physiological loop, where pain-induced sleep disruption and chronic stress drive neuroendocrine dysregulation and maladaptive coping behaviors; and (3) a socioeconomic loop, where economic instability decreases healthcare security. Consequently, these behavioral, psychological, and socioeconomic burdens feed back into the systemic inflammatory core, perpetuating disease chronicity. Moreover, this review explores kinesiophobia (the anticipatory fear of movement) as a potentially critical and overlooked component of the biomechanical-metabolic feedback loop. Currently, there is a notable absence of primary psychometric data quantifying kinesiophobia in the HS population. Future research should aim to quantify this phenomenon to better establish its prevalence and clinical significance. On a macroscopic level, clinicians should aim to systematically break the broader interconnected behavioral feedback loops through multidisciplinary interventions, including cognitive-behavioral therapy and structured patient education. Ultimately, dismantling these psychological and behavioral barriers may be a critical step to attenuate systemic inflammatory amplification and improve long-term clinical outcomes.

Keywords: hidradenitis suppurativa; kinesiophobia; positive feedback loop; systemic inflammation; fear-avoidance model; insulin resistance; lifestyle; sleep; maladaptive coping; socioeconomic burden; psychological burden

1. Introduction

Hidradenitis suppurativa (HS) is a chronic inflammatory dermatosis characterized by deep-seated nodules and abscesses [1,2]. Among its clinical manifestations, pain is consistently identified as the most frequent and debilitating symptom, reported by over 95% of patients [2]. Consequently, the disease imposes a profound impairment on patient quality of life, yielding Dermatology Life Quality Index (DLQI) scores significantly higher than those reported for other severe skin diseases, such as psoriasis and acne [3].

The severe morbidity of HS is closely linked to the anatomical localization of these lesions. The disease predominantly affects intertriginous zones, specifically the axillary, inguinal, gluteal, and perineal regions [1]. Crucially, these areas involve key anatomical joints and skin folds that are continuously engaged during basic human locomotion. Because these dynamic regions must continuously stretch and compress, ordinary daily movement generates friction and shearing forces across the inflamed tissue.

Furthermore, the pain generated by this mechanical stress is frequently reported by patients as particularly severe. HS pain is not exclusively nociceptive; chronic inflammation induces peripheral

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neuroplastic changes and central sensitization, resulting in a profound neuropathic pain profile [2]. Clinical literature confirms that patients experience mechanical allodynia and hyperalgesia, frequently describing the pain as a burning, cutting, or pressing soreness [3]. In this neuropathic state, even minor skin tension from normal activities like walking or reaching can be processed by the central nervous system as significant pain stimuli.

This biomechanical reality can significantly alter patient behavior. To prevent mechanically triggered allodynia, the initial adaptive response is often to reduce movement. However, as the disease progresses, this protective response may lead patients to associate normal locomotion with severe pain, potentially establishing a significant psychological barrier to movement.

2. Translating the Fear-Avoidance Model to Hidradenitis Suppurativa

In musculoskeletal rehabilitation, the transition from acute protective reflexes to chronic disability is explained by the fear-avoidance model [4]. In this context, the model posits that a maladaptive cognitive appraisal of pain triggers kinesiophobia, traditionally defined as an excessive fear of movement due to a perceived vulnerability to exacerbating the pain or injury [4]. Kinesiophobia is a highly specific and measurable clinical entity, robustly quantified in chronic pain populations using the validated Tampa Scale for Kinesiophobia (TSK) [5].

This psychological framework translates well to the biomechanical realities observed in HS. Qualitative studies demonstrate that HS patients spend extensive cognitive and physical energy anticipating and compensating for pain [6]. Because ordinary movements such as reaching, walking, or typing exert mechanical stress on the lesions, patients often actively avoid even minor postural changes [6]. While initially an acute biological necessity during unpredictable disease flares, this anticipatory fear of movement may evolve into a chronic psychological barrier contributing to a sedentary lifestyle and driving a pro-inflammatory state. Recent epidemiological literature explicitly recognizes this phenomenon, hypothesizing a clinical cycle of pain-induced immobility, kinesiophobia, and severe physical deconditioning in patients with HS [7].

Adjacent medical specialties increasingly recognize kinesiophobia as a critical amplifier of pain and disability in systemic inflammatory conditions like spondyloarthritis [8] and psoriatic arthritis, where kinesiophobia afflicts nearly half of patients, contributing to central sensitization and functional impairment independent of inflammatory disease activity [9]. While HS is among the dermatological conditions most strongly associated with mobility-related pain, standard tools like the DLQI and Patient Health Questionnaire-9 (PHQ-9) do not currently account for kinesiophobia [6]. Kinesiophobia remains a critically unmeasured metric in the HS patient population. If actively identified, kinesiophobia could be mitigated using established musculoskeletal interventions, such as graded exposure therapy and cognitive-behavioral rehabilitation [4]. However, left unchecked, this psychological avoidance and fear of movement may predispose the patient to sustained immobility, initiating a secondary cascade of physical deconditioning and metabolic dysfunction [4,6].

3. Behavior and Lifestyle Positive Feedback Loops: A Macroscopic Framework

HS pathophysiology involves a complex network of positive feedback loops that frequently interact to amplify systemic inflammation, a primary biological driver of the disease [10,11]. To date, dermatological literature has predominantly characterized these feedback loops at the microscopic levels. For instance, existing models define a localized vicious cycle of chronic inflammation driven by innate immune hyperactivation, aberrant antimicrobial peptide expression, and local microbiome dysbiosis [12–14]. Specifically, molecular models have mapped self-amplifying cycles where tumor necrosis factor-α (TNF-α) induces the production of lipocalin-2 in neutrophils, which in turn stimulates further TNF-α release and neutrophil chemotaxis, maintaining the local tissue in a state of chronic suppuration [13].

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We propose that this established pathophysiological paradigm can be expanded macroscopically. While the profound psychosocial and socioeconomic burdens of HS are individually well-documented, these downstream consequences are rarely integrated into a single model as active contributors to disease progression. By combining known behavioral maladaptations with kinesiophobia, we construct a comprehensive macroscopic feedback network reflecting the burdens on the HS patient population. Rather than replacing localized microbiological models, this network approach complements them by illustrating how the behavioral, psychological, and socioeconomic adaptations to the disease can feed back into the systemic inflammatory core, functioning as interconnected positive feedback loops.

As illustrated in Figure 1, we hypothesize that this self-sustaining cycle operates through three broad, interacting domains: (1) a biomechanical-metabolic loop, where pain-driven inactivity accelerates visceral adiposity and systemic metabolic dysfunction, including insulin resistance; (2) a psychosocial and physiological loop, where severe stigma, loss of intimacy, and sleep fragmentation collectively drive chronic stress, hypothalamic-pituitary-adrenal (HPA)-axis dysregulation, and maladaptive coping; and (3) a socioeconomic loop, where disease-related absenteeism leads to sustained healthcare insecurity.

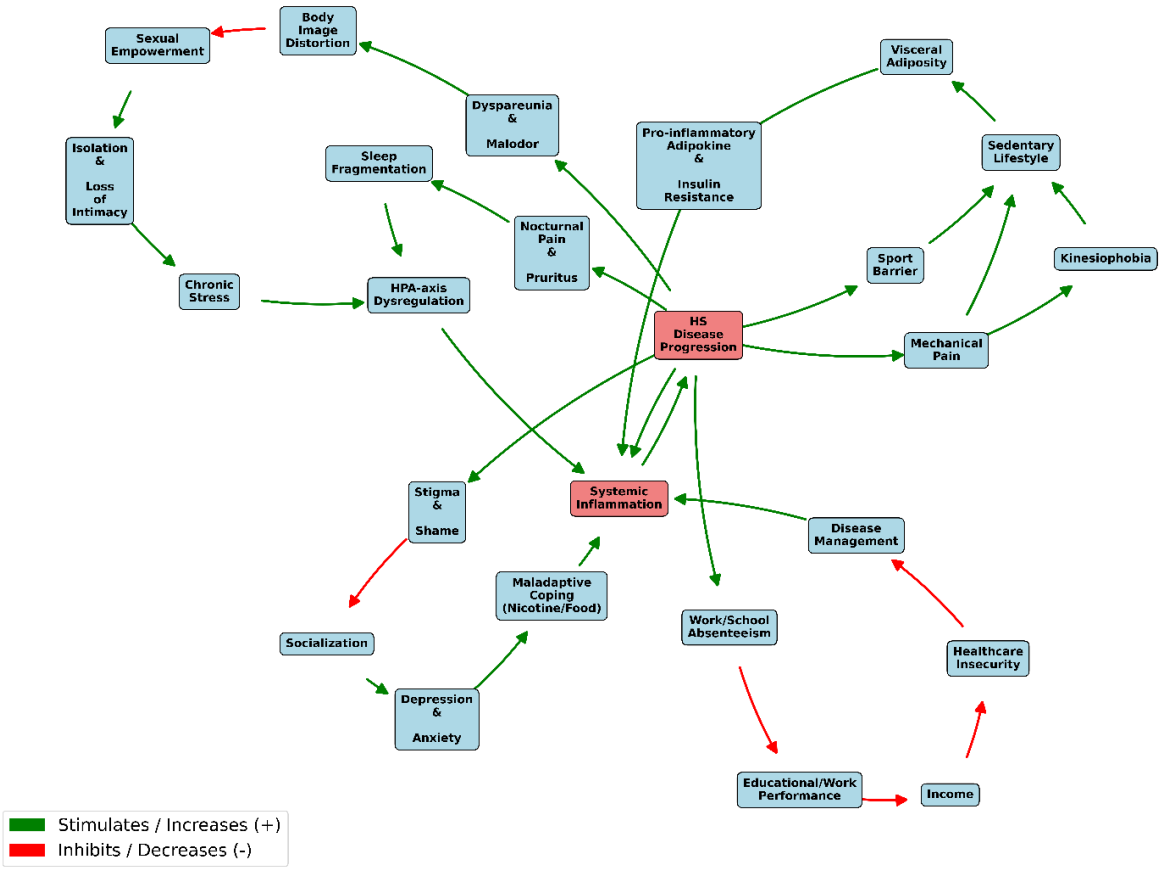

Figure 1. Network of behavioral positive feedback loops in hidradenitis suppurativa. This directed network graph illustrates the interconnected biomechanical, neuroendocrine, and socioeconomic pathways that contribute to disease chronicity. The core disease engine is represented by the central nodes (light coral), demonstrating the bidirectional stimulation between systemic inflammation and active disease progression. Intermediate behavioral and physiological mediators (light blue) illustrate how the downstream consequences of the disease structurally feed back into the inflammatory core. This figure is available as high-resolution file in the supplementary materials.

3.1. The Biomechanical-Metabolic Feedback Loop

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The most direct physical consequence of kinesiophobia is a profound reduction in mobility. While factors such as pain, active suppuration, malodor, and generalized physiological burden inherently inhibit regular exercise [15,16], we postulate that kinesiophobia may also act as the psychological stimulus contributing to infrequent exercise and a chronically sedentary lifestyle in HS patients. This sustained physical inactivity can accelerate the accumulation of visceral adiposity and contribute to a systemic decline in insulin sensitivity [17]. Adipose tissue increases the systemic secretion of pro-inflammatory adipokines while simultaneously downregulating anti-inflammatory mediators [18]. Furthermore, the resulting insulin resistance induces compensatory hyperinsulinemia, a state known to stimulate androgen-mediated epidermal hyperplasia and follicular hyperkeratosis [19]. Because these adipokine and metabolic disruptions upregulate inflammatory pathways implicated in HS lesion formation, the psychological fear of movement may perpetuate disease chronicity by sustaining a pro-inflammatory metabolic state.

3.2. The Psychosocial-Physiological Feedback Loop

This metabolic cascade is accompanied by a concurrent neuroendocrine feedback loop. The physical realities of active HS, including pain, suppuration, dyspareunia, and malodor, can strongly predispose patients to anxiety, body image distortion, social stigma, and the loss of socialization and sexual intimacy [16,20–24]. These factors promote chronic psychological stress, which can dysregulate the HPA axis and elevate baseline systemic inflammatory markers [25].

Moreover, the disease significantly disrupts essential physiological recovery processes, such as sleep. HS profoundly impairs sleep architecture, primarily driven by disease-specific pain and severe pruritus [26–28]. Clinical HS literature establishes pain and pruritus as a primary driver of prolonged sleep latency and frequent nocturnal awakenings [27]. The specific biomechanical mechanisms underlying this disruption likely stem from the fact that deep-seated nodules predominantly afflict intertriginous and weight-bearing regions, where mechanical allodynia [3] severely restricts comfortable resting postures. This underexplored positional pain may also act as a direct, physical barrier to sleep onset and continuity. Consequently, this chronic sleep deprivation functions not only as a clinical symptom but also as an active pathophysiological stressor. Insufficient sleep disrupts circadian homeostasis and upregulates the systemic production of pro-inflammatory cytokines, TNF-α and interleukin-6 (IL-6) [25]. This establishes a positive feedback loop where HS-driven pain and pruritus impair sleep, and the resulting sleep loss exacerbates the systemic inflammation associated with further disease progression [25].

Furthermore, the physiological exhaustion resulting from chronic sleep loss can significantly deplete cognitive reserves, thereby exacerbating baseline emotional distress, anxiety, and depressive symptoms [26]. To mitigate this compounding psychological and physiological fatigue, patients frequently resort to maladaptive coping behaviors, most notably nicotine consumption and high-glycemic emotional eating [11]. These behaviors can act as active vectors for disease progression. Nicotine stimulates specific receptors in the follicular infundibulum, promoting epidermal thickening as well as chemotaxis and neutrophil extravasation [11]. Concurrently, a reliance on high-glycemic diets induces rapid spikes in blood glucose and compensatory hyperinsulinemia [19]. This diet-induced loss of insulin sensitivity can significantly amplify the systemic inflammatory burden and stimulate androgen-driven sebaceous gland overactivity [11,19]. Additionally, insulin resistance and obesity disrupt metabolic homeostasis by promoting the continuous release of pro-inflammatory cytokines from adipose tissue, further sustaining systemic inflammation [18].

3.3. The Socioeconomic Feedback Loop

Simultaneously, the physical and psychological burdens imposed by HS contribute to significant socioeconomic deterioration [29]. The compounded burden of chronic pain, immobility, and generalized physiological exhaustion can significantly impair educational and professional capabilities. Clinical evaluations utilizing validated productivity metrics demonstrate that HS patients experience substantial rates of both absenteeism (missed work) and presenteeism (reduced

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productivity while at work) [22,30]. As inflammatory disease severity increases, overall work productivity and the capacity to perform essential daily activities decline markedly [30].

This chronic professional disruption is closely associated with long-term economic impairment. Compared to healthy demographics, patients with HS experience significantly hindered income trajectories, incur substantial indirect disease-related costs, and suffer disproportionately high rates of both unemployment and reliance on long-term disability support [22,31].

Crucially, we suggest that this socioeconomic decline establishes a barrier to physiological recovery. A progressive reduction in personal income can restrict a patient's capacity to afford optimal and supplementary healthcare, consequently hindering effective disease management. Reduced financial agency can limit access to the clinical interventions needed to disrupt these interconnected feedback loops, potentially leaving the patient vulnerable to a self-sustaining cycle of systemic inflammation.

4. Future Directions and Conclusions

Building upon the established understanding of localized and systemic inflammation, the conceptualization of hidradenitis suppurativa can be expanded to integrate a complex network of behavioral, physiological, and socioeconomic feedback loops. While advanced biologic therapies and surgical interventions remain the foundational cornerstones of HS management, their overall clinical impact may be attenuated if a patient remains caught in a cycle of psychological, physical, and socioeconomic decline. By recognizing maladaptive behavioral responses as potential contributors to systemic inflammation, clinicians can better understand the persistent chronicity of the disease and implement the appropriate treatments or referrals to address these key aspects of the disease. Consequently, optimal disease management warrants a greater emphasis on multidisciplinary care.

To adequately address kinesiophobia in HS, routine dermatological assessments would benefit from expanding beyond standard quality-of-life indices to incorporate validated musculoskeletal and psychological screening tools, most notably the Tampa Scale for Kinesiophobia (TSK) [5]. Characterizing this anticipatory fear of movement opens an interesting new research frontier and introduces a potential novel therapeutic target for HS management. If validated in the HS population, patients exhibiting high kinesiophobia scores could potentially benefit from structured musculoskeletal rehabilitation programs. Utilizing established interventions such as cognitive-behavioral therapy and graded exposure therapy [4] can effectively dismantle the psychological barrier to movement. Addressing this immobility is a crucial biological target to help mitigate the secondary cascade of visceral adiposity, hyperinsulinemia, and subsequent inflammatory amplification.

To formally establish the clinical role of kinesiophobia in HS, future research should incorporate both prospective observational and interventional studies. Initial efforts could focus on quantifying the prevalence and severity of this phenomenon across diverse patient cohorts, evaluating how these behavioral metrics correlate with systemic inflammatory markers and clinical disease stages. Subsequently, interventional trials would be valuable to determine whether physical rehabilitation and behavioral therapies can help reduce the systemic inflammatory burden and improve overall disease severity.

Ultimately, investigating whether actively disrupting these behavioral feedback loops enhances the clinical efficacy and longevity of existing biologic therapies will support the establishment of comprehensive, disease-modifying treatment protocols for hidradenitis suppurativa. HS patients frequently experience delayed diagnosis, ineffective treatments, undertreatment of psychological and psychiatric comorbidities, as well as a lack of education regarding effective lifestyle modifications, contributing significantly to medical alienation. To improve patient outcomes and satisfaction, greater emphasis should be placed on describing, quantifying, and treating these frequently overlooked dimensions of the disease. From a broader philosophical and clinical perspective, it would be highly valuable to map the additional positive feedback loops in HS and develop an expanded network that combines both microscopic (molecular) and macroscopic (behavioral)

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feedback loops. While the macroscopic framework presented in this study compartmentalizes these burdens into three distinct modules, they are likely far more intricately interconnected in clinical reality. Moreover, it is essential that future research aim to expand upon the macroscopic network proposed in this study. The three distinct behavioral feedback loops identified within this review could be mathematically translated and integrated into existing mathematical models. By undertaking this integration, we can more comprehensively and accurately simulate the profound dynamic complexity of this disease.

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Data Availability: The network map (Figure 1) is available as high-resolution file in the supplementary materials.

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Abbreviations

DLQI, Dermatology Life Quality Index

HPA, hypothalamic-pituitary-adrenal

HS, Hidradenitis suppurativa

IL-6, interleukin-6

PHQ-9, Patient Health Questionnaire-9

TNF-alpha, tumor necrosis factor-alpha

TSK, Tampa Scale for Kinesiophobia

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