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A Review of the Relationship between Environmental Pollutants and Hidradenitis Suppurativa Disease Severity

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Abstract

Hidradenitis suppurativa (HS) is a chronic, inflammatory skin disorder of the folliculopilosebaceous unit that predominantly affects intertriginous areas. While genetic, hormonal, and lifestyle-related risk factors are well-established, the potential role of environmental pollution in HS pathogenesis remains largely unexplored. Emerging evidence suggests that pollutants such as particulate matter ($PM_{2.5}$) and polycyclic aromatic hydrocarbons (PAHs) contribute to oxidative stress, microbiome disruption, and activation of pro-inflammatory pathways that may exacerbate HS disease activity. This hypothesis-driven review evaluates the evidence that indirectly links environmental pollutants to the incidence, severity, and progression of HS, drawing comparisons to other inflammatory dermatoses such as psoriasis and atopic dermatitis while emphasizing HS-specific mechanisms. Environmental pollution represents a potentially modifiable risk factor that warrants further investigation. Future research should prioritize prospective cohort studies, pollutant-specific exposure tracking, and biomarker development to clarify the impact of environmental triggers on HS onset and severity. It is important to note that no direct epidemiologic studies linking environmental pollution to HS currently exist. Understanding this link may improve disease management and open avenues for preventative strategies.

Keywords

Hidradenitis Suppurativa, Air Pollution, Environmental Pollution, Oxidative Stress, Skin Microbiome, Inflammation

1. Introduction

Hidradenitis suppurativa (HS) is a chronic, cutaneous condition that primarily

affects areas of the body rich in apocrine glands, such as the axillae, inframammary folds, groin, buttocks, and perianal region [1] [2]. This disease is characterized by recurrent inflammatory, painful nodules and abscesses that may progress into sinus tracts and scarring. Although apocrine dysfunction was initially considered to be the underlying cause, it is now determined to be a disease of follicular occlusion and rupture [3]. The Hurley Staging system remains the standard for grading disease severity [2]. Stage I describes single or multiple abscesses without sinus tract or scarring. Stage II involves the presence of single or multiple, widely-separated, recurrent abscesses with scarring and tract formation. Stage III is characterized by the formation of interconnected tracts and abscesses in an entire area [4]. This disease markedly impairs quality of life [5]. Beyond the physical burden, HS imposes significant psychosocial and economic strain due to pain, malodor, depression, anxiety, and lost productivity [6]. In the US, the female to male ratio is 3:1 and African-Americans are 3 - 7x more likely to be diagnosed with HS [7]. These statistics may vary in other parts of the world. Disease onset ranges, but most commonly occurs in the second decade of life [1]. The prevalence of HS is estimated to be between 1% and 4% of the population, though these may be underestimations due to misdiagnosis and underreporting [1] [5]. Early diagnosis of this disease is key, as delayed identification and management worsen patient experience [1] [5].

While well-established risk factors include genetics, obesity, smoking, and hormonal influences, environmental triggers are increasingly recognized as contributors to disease initiation and progression [8]. Notably, air pollution has been implicated in other chronic inflammatory dermatoses such as acne, atopic dermatitis, and psoriasis, yet its role in HS has received little attention [8] [9]. This hypothesis-driven review aims to evaluate the indirect association between environmental pollutants and disease severity in HS, highlighting mechanistic links and epidemiologic considerations.

2. Pathogenesis of HS

Follicular Occlusion, Structural Breakdown, and Dysregulated Notch Signaling

The pathophysiology of HS is complex and not fully elucidated, but is thought to consist of a multifactorial interplay of follicular occlusion and a dysregulated immune system, which is influenced by genetics, microbiome, and hormones [10].

The initiating event is thought to be blockage within the folliculopilosebaceous unit (FPSU), specifically the follicular portion. The most common sites in HS are in areas prone to friction, which contributes to the pathophysiology. This blockage occurs when the opening of the hair follicle becomes thickened, leading to dilatation and follicular rupture [11]. The primary cause of this blockage remains unknown, however, a study by Melnik and Plewig introduced the concept of an auto-immune dysregulation of the gamma-secretase/Notch pathway, which is involved in maintenance of the root sheath of the hair follicle and skin appendages

[2]. When the follicle erupts, it releases keratin and bacteria into the dermis, producing a massive inflammatory response with abscess formation.

Innate Immune Activation and Inflammasome Signaling

Driving this inflammation is a broad range of inflammatory mediators that have been found to be overexpressed in HS lesional skin compared to healthy controls [12]. Key pro-inflammatory cytokines that are involved in the pathogenesis of HS include IL-1 α/β, TNF-α, IFN-γ, IL-17, and IL-6, G-CSF [2]. Current evidence suggests that upon rupture, the release of follicular contents (bacteria, DAMPs, keratin and sebum components) activates the NLRP3 inflammasome, as well as caspase-1 leading to the production of IL-1β [2]. Increased levels of IL-1β induce the formation of matrix metalloproteinases (MMPs), degradative enzymes that thin the basement membrane surrounding the FPSU, increasing the fragile nature of the inflamed and dilated follicle [2] [12]. In addition, ruptured follicular content is recognized by macrophages and dendritic cells through toll-like receptors (TLRs), and their recruitment leads to further pro-inflammatory cytokine production, especially TNF-α [2].

Adaptive Immunity and the Th17 Axis

TNF-α induces a multitude of chemokines that attract immune cells such as T cells, B lymphocytes, plasma cells, granulocytes, and monocytes, all of which have been found in HS lesions [2]. More notably, TNF-α plays a key role as an upstream mediator for T cell differentiation into Th1 and Th17 cells. Th1 cells produce increased levels of IFN-γ, while Th17 cells produce increased levels of IL-17. To maintain and stabilize Th17 cells, there is increased production of IL-23 by antigen presenting cells mediated by IL-1β, TNF-α and IFN-γ [2]. Various studies involving transcriptomic analysis and immunohistochemistry have found increased levels of pro-inflammatory mediators in HS lesions compared to healthy skin controls, illustrating the diverse immune activation of this disease [13]-[15]. The inflammatory process involved in the pathogenesis of HS has been linked to dysfunctional regulatory pathways of the skin, including keratinization, organization of the extracellular matrix (ECM), formation of cornified envelopes, differentiation of epidermal cells, and the formation and degradation of collagen [10]. Taken together, this network of cytokines leads to the unrelenting inflammatory cycle that results in pain, drainage, and scarring.

Microbiome Dysbiosis and Biofilm Formation

In HS individuals, a microbial dysbiosis occurs on the skin and within the hair follicle, which is attributed to the decreased abundance of skin commensals leading to overgrowth of anaerobic pathogens [2]. Interestingly, HS skin was found to have an increased presence of *Prevotella*, as well as other notable bacteria such as *Staphylococcus lugdunensis*, *porphyromonas*, milleri group *streptococci*, and *actinomyces*. Once colonized, these bacteria may be challenging to eradicate due to the formation of biofilms within the sinus tracts, sustaining chronic inflammation [2]. Limited epidermal upregulation of antimicrobial proteins, supported by dysregulated tryptophan catabolism at the skin-microbiome interface, facilitates

propagation of intrafollicular, partly strict anaerobe bacteria [16]. This further supports that pollutant-induced microbiome disruption could worsen HS.

3. Mechanistic Links between Environmental Pollution and HS

Classification of Environmental Pollutants

To better understand how environmental exposures translate into cutaneous inflammation, it is important to distinguish the major classes of pollutants and their biological effects. In the United States, the Environmental Protection Agency (EPA) classifies six criteria air pollutants: particulate matter (PM), ground-level ozone (O_3), nitrogen oxides (NO_x), sulfur dioxide (SO_2), carbon monoxide (CO), and lead [17]. These pollutants may be categorized as primary pollutants, which are emitted directly from a source, or secondary pollutants, which form in the atmosphere through chemical reactions involving primary pollutants. Primary pollutants include particulate matter (PM), while combustion-derived pollutants such as polycyclic aromatic hydrocarbons (PAHs), diesel exhaust particles (DEPs), and volatile organic compounds (VOCs) commonly coexist with PM as components of traffic-related air pollution but are not themselves EPA criteria pollutants [8] [17]. Ground-level ozone (O_3), the principal secondary pollutant, forms through photochemical reactions involving nitrogen oxides and VOCs in the presence of ultraviolet (UV) radiation and heat, resulting in the formation of tropospheric smog [8] [17]. In contrast, UV radiation represents a physical environmental exposure rather than an air pollutant but may act synergistically with air pollutants to enhance oxidative stress and skin injury [8]. Cigarette smoke constitutes an additional complex environmental exposure containing both particulate and gaseous toxicants that has an established association with HS and shares several inflammatory pathways with ambient air pollution [8] [18].

Mechanisms of Skin Injury

Once deposited onto the skin, different pollutants interact through distinct mechanisms, initiating inflammatory signaling and impairing skin barrier function. Gaseous pollutants such as ozone primarily react with surface lipids and induce oxidative stress without directly penetrating the skin barrier [19] [20]. Particulate pollutants, including $PM_{2.5}$ and ultrafine particles carrying adsorbed PAHs and VOCs, may accumulate on the skin surface, become increasingly lipophilic, and penetrate through the stratum corneum, hair follicles, or sweat ducts [19] [20]. Once internalized, PAHs bind to and activate the aryl hydrocarbon receptor (AHR), a ligand-activated transcription factor that induces CYP1 activity and promotes the generation of reactive oxygen species (ROS) through redox reactions [21]. The resulting oxidative stress depletes cellular antioxidant defenses, initiates lipid peroxidation, increases proteolytic activity, and stimulates the release of pro-inflammatory mediators, including IL-1β, IL-22, and IL-33 [21]. These cytokines sustain inflammation by recruiting neutrophils and other immune cells, thereby amplifying oxidative injury and inflammatory signaling [22].

In a 2019 study by Fuks *et al.*, increased ozone exposure was associated with oxidative skin damage, heightened inflammatory responses, and depletion of the skin's antioxidant defenses [23]. Similarly, exposure of human keratinocytes to diesel exhaust particles induced increased production of IL-1β, IL-6, and TNF-α, further promoting cutaneous inflammation [24]. Together, these findings suggest that pollutant-induced oxidative stress may contribute to matrix metalloproteinase activation and extracellular matrix degradation in normal skin, potentially suggesting it can exacerbate the tissue destruction and scarring of advanced HS [2] [12] [21].

In addition to promoting oxidative stress and inflammatory signaling, pollutant exposure may alter the skin microenvironment by disrupting microbial homeostasis. Normal human skin exposed to environmentally relevant ozone concentrations demonstrated approximately a 50% reduction in commensal skin microflora [25]. Loss of these protective microbial communities may favor colonization by anaerobic, biofilm-forming organisms that could contribute to sustained inflammation within HS sinus tracts [2] [9] [25].

Much of this mechanistic evidence discussed is derived from experimental models and studies of environmental pollution in normal skin, psoriasis, atopic dermatitis, and acne, as direct mechanistic investigations in hidradenitis suppurativa remain limited. Accordingly, these pathways should be interpreted as biologically plausible mechanisms through which pollutant exposure may influence HS rather than as mechanisms directly demonstrated in HS patients [8] [19]-[23].

4. Integration of Environmental Pollutants in HS Pathogenesis

Building on the established roles of follicular occlusion, immune dysregulation, and microbiome alterations in HS, mechanistic evidence extrapolated from environmental toxicology and other inflammatory skin diseases suggests that environmental pollutants may interact with these pathways to influence disease initiation and severity.

Pollutant-induced reactive oxygen species (ROS) may act as upstream mediators that activate the NLRP3 inflammasome, leading to increased IL-1β production and amplification of downstream inflammatory signaling in HS [2] [8] [21]. This process reinforces the chronic immune dysregulation characteristic of the disease. Environmental pollutants, particularly polycyclic aromatic hydrocarbons (PAHs), have been shown to activate the aryl hydrocarbon receptor (AHR), a key regulator of cutaneous immune responses and keratinocyte differentiation. Emerging evidence suggests that AHR signaling is intrinsically dysregulated in HS. In a 2020 study by Guenin-Macé *et al.*, HS lesional skin exhibited abnormal kynurenine pathway dysregulation, causing tryptophan depletion and reduced production of microbiota-derived AHR ligands. The subsequent reduction in commensal bacteria that generate AHR agonists leads to defects in protective AHR signaling at the skin barrier. In this context, exogenous AHR activation from environmental pol-

lutants may further disrupt immune function, amplify inflammatory signaling, and impair keratinocyte differentiation leading to follicular occlusion, instability, and rupture [2] [21] [26]. Additionally, *in vitro* and *in vivo* experimental studies on porcine models demonstrate that $PM_{2.5}$ disrupts the skin barrier by reducing structural proteins within the stratum corneum, including cytokeratin, filaggrin, and E-cadherin, while also promoting microbial dysbiosis and inflammation [9]. In HS, pollutant-induced impairment of these structural proteins may further predispose to follicular rupture and lesion formation [9] [20].

Urban-Rural Variations in Environmental Pollutant Exposure

Given the potential biological effects of environmental pollutants, it is important to consider differences in exposure between urban and rural environments. $PM_{2.5}$ is commonly used as a quantitative marker of ambient air pollution exposure, although concentrations vary substantially by geographic location and time [27].

In a study by Kilpatrick *et al.*, it was found that in the United States between 2010 and 2019, urban areas had higher levels of $PM_{2.5}$ compared to rural areas [28]. In urban areas, air pollution is mainly the result of combustion of industrial materials and traffic emissions [29]. Urban communities that are located near busy roads and high-volume traffic areas are more exposed to traffic-related pollution (TRP), which is a mixture of emissions from gasoline and diesel-fueled vehicles [1] [9]. Although these exposure differences may plausibly influence inflammatory pathways relevant to HS, no studies have directly established urban-rural differences in HS incidence or disease severity attributable to environmental pollution. Therefore, urban-rural comparisons should currently be interpreted as differences in exposure context rather than evidence of differential HS risk [27] [29].

5. Genetic and Lifestyle Modifiers of HS

While environmental pollutants may influence HS pathogenesis, their effects likely interact with underlying genetic susceptibility and lifestyle-related risk factors. Genetic predisposition plays a significant role in HS, with 33% - 40% of patients reporting a first-degree relative with the condition [1]. There is also an increased incidence of HS in patients with genetic disorders such as Pyoderma gangrenosum, acne, and suppurative hidradenitis (PASH). Mutations in one of three genes (NCSTN, PSEN1, PSENEN) showed an inherited nonsyndromic HS [3]. These genes are proposed to regulate epidermal and follicular keratinocyte differentiation, so when they have a mutation, it can lead to HS.

Lifestyle factors, such as obesity and smoking, further exacerbate risk. Obesity increases sweat production and skin friction in intertriginous areas. The mechanical damage from skin friction induces microinjuries to the skin leading to the release of cellular damage-associated molecules (DAMPS), as well as ample access for microbes potentially leading to infection [12]. Cigarette smoke contains nicotine and various other chemicals that activate keratinocytes by binding to aryl hy-

drocarbon receptors (AHRs) and nicotinic acetylcholine receptors (nAChRs) that induce hyperplasia of epithelial infundibular cells, acanthosis, and hypercornification [18].

6. Gene-Environment and Socioeconomic Interactions

Other factors that may influence HS severity secondary to environmental pollution include occupation (coal mining, industrial work), socioeconomic status, and genetics. For individuals who work in low-income, urban settings, disproportionate exposure to environmental pollutants, such as PM and DEPs, may lead to an increase in negative health effects, such as increased HS severity due to inflammation and skin barrier dysfunction [27] [29]. Due to the limited healthcare accessibility in these low-income settings, delays in diagnosis and treatment may contribute to more severe disease outcomes [10]. The interplay between environmental pollution and socioeconomic disparities highlights why urban and lower income populations may experience greater HS burden. In genetically predisposed individuals, environmental pollution may cause epigenetic changes which could potentially exacerbate HS severity. These epigenetic changes include decreased expression of DNA hydroxymethylation regulators in HS lesional skin, changes in expression of microRNA (involved in gene silencing), and differential expression of RNA-induced silencing complex components [3]. These pollutant-induced epigenetic mechanisms disrupt normal immune regulation and inflammatory pathways, which lead to increased cytokine activity impairing skin barrier function [30]. Although there is no direct evidence that environmental pollution induces epigenetic changes that may cause HS, pollution is known to alter epigenetic regulation, and HS is characterized by dysregulated gene expression and chronic inflammation. Together, these findings suggest a plausible, but still unproven, mechanistic link between environmental exposures and HS pathogenesis.

7. Research Gaps and Future Directions

There is a gap in the literature regarding the role of environmental pollution in the pathogenesis, incidence, prevalence, and severity of HS. Existing studies have demonstrated the known risk factors of HS, such as obesity and smoking, however, very few literature sources have directly examined the role of environmental pollutants [2] [10]. There is an absence of longitudinal studies targeting pollutant exposure in HS patients. In contrast, there is an increasing body of evidence that reveals a link between environmental pollution and other chronic inflammatory skin conditions such as psoriasis, atopic dermatitis, and acne [8] [9]. Given the established inflammation-inducing effects of environmental pollutants, it's plausible to suggest that these same agents can affect the prevalence or severity of HS. The lack of available literature on this topic presents potential avenues of research studies to be conducted to explore how environmental pollution may influence HS prevalence, severity, and disease progression. Large, prospective cohort studies should be conducted to longitudinally track pollutant exposure and monitor

the outcomes of patients with HS. Beyond prospective cohort studies, time-series analyses correlating daily pollutant levels with HS flare frequency and Mendelian randomization studies can effectively assess causal relationships. Incorporation of personal and low-cost exposure pollutant sensors such as PurpleAir, Atmotube, AirVisual, Clarity Node-S, or Luftaden, may be viable tools for data collection. Additionally, epidemiologic studies evaluating pollution and HS should carefully adjust for smoking status, body mass index, mechanical friction, socioeconomic status, and occupational exposure to minimize residual confounding.

Moreover, the development and validation of various types of biomarkers for clinical use in HS studies would be overwhelmingly beneficial. Diagnostic and susceptibility/risk-type biomarkers would aid in diagnosing and determining high-risk individuals, while monitoring biomarkers would provide more information regarding the molecular complexities of HS skin [31]. A systematic review by Der Sarkissian found that monitoring biomarkers with moderate GRADE evidence include serum IL-17, SAA, CRP, and IL-8, while sonographic dermal vascularity was the only monitoring biomarker to achieve a high GRADE rating. However, none of the proposed biomarkers have undergone longitudinal clinical validation, which makes it difficult to correlate pollutant exposure with disease activity over time [31]. In reference to the relationship between air pollution and HS severity, predictive biomarkers would be particularly advantageous for managing disease, as they allow for personalized treatment approaches with targeted therapies geared toward whatever high-risk factors are implicated [31].

The therapies in HS are expanding and continue to do so. Bimekizumab and secukinumab are now FDA-approved for moderate-to-severe HS alongside adalimumab. This evolving field demonstrates why understanding environmental triggers matters; if pollutants drive specific inflammatory pathways, this could inform treatment selection. Lastly, given what is known about the negative effects of air pollution on overall health, indoor air purifiers may be an option for individuals seeking to decrease their exposure [32]. This presents another area of future research within the field of dermatology, warranting studies to assess if air purifiers affect cutaneous disease incidence, prevalence, and severity.

8. Limitations

This review is limited by the absence of direct epidemiologic and mechanistic studies evaluating the direct relationship between environmental pollution and HS. As a result, the central premise relies on mechanistic extrapolation from studies on other inflammatory dermatoses, such as psoriasis and atopic dermatitis, where pollutant-induced oxidative stress, immune activation, and barrier dysfunction have been more clearly identified.

Additionally, much of the evidence discussed is derived from *in vitro* experiments, animal models, or population-level pollution studies that do not directly assess HS outcomes. Furthermore, the varying pollutant types, exposure levels, and measurement methods limit the ability to draw definitive conclusions. This

review therefore, synthesizes indirect evidence to propose biologically plausible mechanisms by which environmental pollution may influence HS.

9. Conclusion

Amid the ongoing rise of environmental pollution, it is crucial to understand the relationship between ambient pollutants and skin health. Environmental pollution has been implicated in the development and progression of chronic inflammatory dermatoses such as psoriasis, atopic dermatitis, and acne, whereas evidence in HS remains limited. Given HS's inflammatory and barrier-disruptive nature, environmental pollutants may represent an underrecognized but modifiable risk factor. Environmental exposure metrics should be incorporated into future HS clinical trials to act as stratification factors, preventing confounding and ascertaining if a relationship exists. Pollutant exposure is a potentially modifiable risk factor, therefore recognition of the relationship with HS could improve management and treatment options for patients.

Author Contributions

Conceptualization, C.R. and V.F.; methodology, C.R. and V.F.; writing—original draft preparation, C.R. and V.F.; writing—review and editing, C.R. and V.F.; All authors have read and agreed to the published version of the manuscript.

Conflicts of Interest

The authors declare no conflicts of interest regarding the publication of this paper.

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