BEYOND THE SKIN: EXPLORING THE MIND-SKIN CONNECTION IN PSYCODERMATOLOGY- A REVIEW

PSYCHODERMATOLOGY

Dr.Senthil Rajan.D

Professor & HOD, Department of Pharmaceutics

Swamy Vivekanandha college of pharmacy, Namakkal, Tamilnadu, India.

Email Id: senthilrajan@vivekanandha.ac.in

Ms.Arthi.K

B.Pharm Final year, Department of Pharmaceutics

Swamy Vivekanandha college of pharmacy, Namakkal, Tamilnadu, India.

Email ID: arthikottaimuthu@gmail.com

Ms.Hemavathi.N

B.Pharm Final year, Department of Pharmaceutics

Swamy Vivekanandha college of pharmacy, Namakkal, Tamilnadu, India.

Email Id: nhemavathi172005@gmail.com

Ms.Dhivyadharshini.M

B.Pharm Final year, Department of Pharmaceutics

Swamy Vivekanandha college of pharmacy, Namakkal, Tamilnadu, India.

Email Id: svsv2175@gmail.com

Corresponding Author:

Mrs Sivasankari.M M.Pharm., Assistant Professor, Department of Pharmaceutics

Swamy Vivekanandha college of pharmacy, Namakkal, Tamilnadu, India.

Mail ID: anusankari85@gmail.com

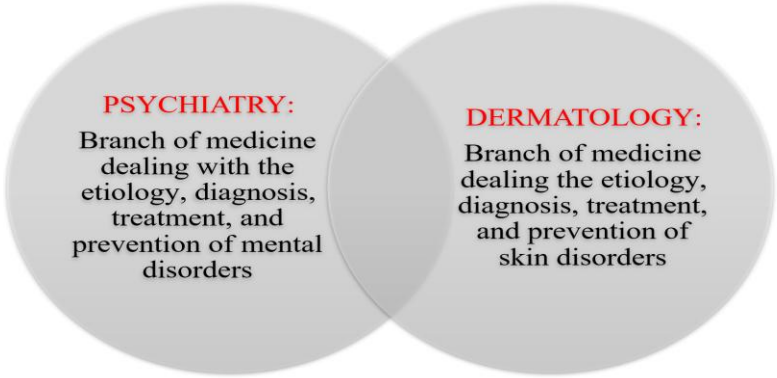
ABSTRACT:
Psychodermatology is an emerging interdisciplinary field that highlights the complex and bidirectional relationship between the skin and the mind. The skin and nervous system share a common embryological origin, and increasing scientific evidence demonstrates the involvement of neuro-immuno-endocrine pathways in several dermatological disorders. Psychological stress can activate the hypothalamic–pituitary–adrenal (HPA) axis, alter immune responses, and impair skin barrier function, thereby triggering or exacerbating inflammatory skin diseases. In dermatological patients, psychiatric comorbidities such as anxiety, depression, obsessive–compulsive disorder, and body dysmorphic disorder are frequently observed. Conversely, chronic and visible skin conditions including psoriasis, acne, vitiligo, atopic dermatitis, and alopecia can significantly affect emotional well-being and overall quality of life. Recent concepts such as the neuro-immuno-cutaneous-endocrine (NICE) model and the gut–brain–skin axis provide deeper insight into the mechanisms underlying this interaction. Therefore, effective management of psychodermatological conditions requires a holistic biopsychosocial approach that integrates dermatological treatment with psychological evaluation, behavioral therapy, and appropriate psychopharmacological interventions. Understanding the mind–skin connection is crucial for improving patient outcomes and enhancing overall well-being.

Keywords: Psychodermatology; Neuro-immuno-cutaneous-endocrine (NICE) model; Skin Barrier Function; Biopsychosocial Approach; Psychodermatological Therapy.

INTRODUCTION:

The medical specialty of psychodermatology is relatively recent. It includes the relationship between the skin and the psyche. Recently, there has been increased interest in the involvement of psychoneuroimmunology in the etiology of psychocutaneous diseases and psychosocial aspects of skin disease. The goals of treating psychodermatological illnesses include enhancing function, lowering physical discomfort, identifying and treating anxiety and depression related to skin conditions, controlling social isolation, and raising the patient's sense of self-worth. Psychocutaneous disorders are treated with a combination of psychosocial and pharmaceutical therapies. Psychodermatology is becoming more and more popular worldwide, and numerous organizations are having regular meetings.[1]. The autonomic nervous system, which is influenced by psychological signs, regulates many of the complex systems that make up the skin, including blood vessels, neurons, muscle components, and glands. They can influence the skin and its parts, causing various skin problems, and they can cause autonomic stimulation. According to several dermatology research, psychological morbidity might reach 60% in inpatients and 30% to 40% in outpatients. Common issues include social dissatisfaction adjustment difficulties, anxiety, and depression. [2] patients in this new speciality have one of the following: 1) a primary mental illness that brings them to a dermatologist (dermatitis artefacta); 2) a primary dermatological disorder with secondary psychosocial comorbidities (acne with body dysmorphic disorder); 3) people who need psychosocial support for their skin disease (rosacea and low self-esteem); or 4) people who have a skin condition that is related to their psychotropic medication (psoriasis may be linked to lithium), or those who Deve Patients with delusional infestations, dermatitis artefacta, trichotillomania, dysaesthesias (including vulvodynia and peno-scrotodynia),

body dysmorphic disorder, social anxiety disorder, depression, and suicidal thoughts are among the most prevalent conditions seen in psychodermatology clinics.Psychodermatology has the following synonyms: Skin and mind (or mind and skin) medicine; psychocutaneous medicine; Cutaneo-somatic dermatology (or medicine), psycho-somatic dermatology (or medicine), and sensoryneuronal dermatology.[3]. Most dermatologists refer to this subspecialty of dermatology as psychodermatology or psycho-cutaneous medicine. There is a debate about whether naming the specialty 'psychodermatology' or that the very prefix 'psycho' is stigmatizing for patients. Whilst most dermatologists are respectful of maximizing patient engagement and minimizing patient stigmatization, most will hold to the term 'psychodermatology' or psycho-cutaneous medicine' as that clearly and uniformly delineates the nature of the specialty [4]



CASE STUDY:

A study of Picardi , showed a prevalence of psychiatric disorders of 25% in outpatients with vitiligo, 26% for psoriasis, 32% for acne, 35% for alopecia, 27% for parasitosis, and 34% for urticaria. A study by Gupta and Gupta found a 5.6% prevalence of suicidal ideation among patients with acne and a prevalence of 5.5% among more severely affected patients with psoriasis, whereas the prevalence in the general medical population is 2.4–3.3%. The most com mon psychiatric disorders observed in dermatology are major depressive disorder, obsessive-compulsive disorders and body dysmorphic disorder. The interrelationship between mind and skin has been investigated also at a cellular level. The neuro-immuno cutaneous-endocrine (NICE) model is a construct featuring four organ systems intimately involved in the bridge between body and mind. [5,6,7]

OVERVIEW OF HISTORY, CONCEPTS AND CURRENT STATUS:

Psychodermatology is an interdisciplinary subspecialty combining dermatology, psychiatry, and psychology, addressing the interactions between skin disorders and psychological factors. Although the concept is ancient—traced back to 1700 BCE with reports of stress-induced psoriasis in Persia and philosophical observations by Aristotle and Hippocrates—the field has gained formal recognition mainly in the last two decades [8]. Modern understanding emphasizes

the mind-skin connection, as both originate from the ectoderm, and the skin is regulated by the autonomic nervous system, which can be influenced by psychological stimuli [9]. Psychiatric comorbidities are common in dermatology, with studies showing 30%–40% prevalence in outpatients and up to 60% in inpatients. Common issues include anxiety, depression, adjustment disorders, and social withdrawal. Surveys indicate that many dermatology patients require psychological support, and skin disorders can exacerbate or trigger psychiatric conditions [10] Psychodermatology has now evolved into a structured, globally recognized specialty. Dedicated societies and working groups exist in Europe, Latin America, and Japan. International collaborations, training programs, and congresses have strengthened its scientific foundation. Studies also emphasize the importance of routine psychosomatic assessment in dermatology clinics, showing that many patients have unrecognized psychological distress contributing to their skin disease [11].Current focus areas include stress-related skin diseases, psychiatric comorbidities in dermatology, and integrated therapeutic approaches combining dermatologic and psychological care [12]

EVOLVING SUBSPECIALITIES IN PSYCHODERMATOLOGY:

1. Psychoneuroimmunodermatology

Psychological stress activates neuroendocrine and immune pathways that directly affect the skin. The skin itself produces stress mediators and responds to them, leading to changes in inflammation and barrier function. This mind–skin–immune interaction can trigger or worsen conditions like psoriasis, eczema, and urticaria.[13]

2. Aesthetic Psychodermatology

Psychodermatology of Aesthetics Since psychological stress can worsen skin disorders, psychodermatology takes a holistic, mind-skin approach. By incorporating this viewpoint into cosmetic dermatology, patient happiness and perceived results may be enhanced.[14]

3. Psychopharmacologic Dermatology

Psychodermatology subspecialty must include management of primary-psychiatric disorders manifesting as skin symptoms — dermatologists may need to prescribe psychotropic (psychiatric) medications when patients present with conditions such as Trichotillomania, Excoriation disorder (skin-picking), Onychophagia (nail-biting), Delusional parasitosis or Body Dysmorphic Disorder — because many of these patients "refuse to see psychiatrists," making dermatology the only point of care. [15]

4. Geriatric Psychodermatology

Among elderly patients with skin diseases, a high proportion exhibit psychiatric morbidity — in one study, nearly half had depression, and many reported anxiety and high stress; furthermore, chronic dermatoses significantly reduce quality of life. These observations suggest that skin disease in the aged is not only a dermatologic concern but also a mental-health and psychosocial concern. Hence, there is a need to conceptualize a 'geriatric psychodermatology' — a subspecialty or integrated approach dealing with both dermatologic and psychological aspects in older adults."[3]

5. pediatic psychodermatology

Pediatic psychodermatology is increasingly recognized as an evolving subspeciality. This is supported by the two recent comprehensive reviews on pediatric psychodermatologic conditions

(Part I and Part II, 2021), which highlight that these disorders span both primary psychiatric conditions with self-induced cutaneous manifestations as well as dermatologic diseases with significant psychological comorbidity. The authors emphasize that effective management requires a multidisciplinary model integrating dermatology and mental-health care, indicating the need for a distinct paediatric psychodermatology service. This expanding clinical scope and the demand for specialized, combined care underscore its development as an emerging subspeciality within dermatology."[16][17]

6. Trauma Focused Psychodermatology:

Empirical evidence links trauma exposure and post-traumatic stress symptoms (PTS) with increased burden of skin disease: nearly half of individuals reporting skin disease symptoms in the study also endorsed clinically significant PTS symptoms — and higher PTS symptom burden was associated with worse skin-related quality of life, independently of skin-symptom severity.[18]

STRESS AND BARRIER FUNCTION OF SKIN:

The organ that serves as a partition between the body's internal and external environments is the skin. Thus, it is subject to both internal endogenous stimuli and a wide range of external physical, chemical, and thermal damages.In recent years, stress—once an abstract psychological phenomenon—has become a major focus of research. Nowadays, the "mind-body connection" is more of a complex physiological mechanism that facilitates bilateral communication between the body and the brain than an esoteric New Age word. Stress has long ben recognised by dermatologists and patients as having an impact on the skin and having the potential to cause, worsen, or prolong a number of skin conditions. Understanding the epidermal barrier's susceptibility to psychological stress is essential since it may play a significant role in the development of some common skin conditions. Deterioration of Psychological Stress. [19].

By stimulating the hypothalamic-pituitary-adrenal axis and 11β-Hydroxysteroid Dehydrogenase 1 and the HPA Axis, psychological stress (PS) raises endogenous glucocorticoid (GC). GC has been shown to have detrimental impacts on skin barrier function under PS. However, when 11β-hydroxysteroid dehydrogenase type I (11ß-HSD1) in the peripheral tissue transforms cortisone (inactive form) into cortisol (active form), endogenous GC can also be active.[20] Here, we assessed how PS affected the 11ß-HSD1 and barrier function. Deteriorated barrier function and elevated cortisol in the stratum corneum were associated with elevated 11ß-HSD1 in the oral mucosa. The oral mucosa's and the epidermal keratinocytes' expression of 11ß-HSD1 was connected. In individuals with anxiety, we also looked into whether barrier function improved when PS was reduced using a selective serotonin reuptake inhibitor (SSRI). SSRI therapy was associated with enhanced barrier function and decreased 11ß-HSD1. According to the combined results, higher 11ß-HSD1 under PS raises cutaneous GC levels and ultimately compromises barrier function. PS-alleviating medications, such SSRIs, may be used to treat PS-aggravated skin disease. PS activates the hypothalamic-pituitary-adrenal axis, which raises endogenous glucocorticoid (GC). [21] It is commonly known that GC impairs the function of the epidermal barrier under PS. However, when 11β-hydroxysteroid dehydrogenase type I (11ß-HSD1) in the peripheral tissue converts cortisone (inactive form) to cortisol (active form), endogenous GC might also be active. Here, we assessed the changes in 11ß-HSD1 and barrier function under PS. Deteriorated barrier function and elevated cortisol in the stratum corneum were

associated with elevated 11ß-HSD1 in the oral mucosa. The oral mucosa's and the epidermal keratinocytes' expression of 11ß-HSD1 was connected. In individuals with anxiety, we also looked into whether barrier function improved when PS was reduced using a selective serotonin reuptake inhibitor (SSRI). SSRI therapy was associated with enhanced barrier function and decreased 11ß-HSD1. According to the combined results, higher 11ß-HSD1 under PS raises cutaneous GC levels and ultimately compromises barrier function. SSRIs and other PS-alleviating medications may be useful in treating skin conditions that are exacerbated by PS. [22] The epidermis, the outermost layer of the skin, is one significant permeability barrier. It constantly renews itself, necessitating stringent control over differentiation and proliferation. The stratum corneum (SC), tight junctions, and immunologic surveillance by Langerhans cells are only a few of the many factors that significantly influence the function of the skin barrier. [23] Dr. Alfred Marchionini coined the phrase "acid mantle" in 1928 to refer to the SC's naturally acidic composition. Under physiological settings, the interior body maintains a pH that is close to neutral, but the human skin has an acidic pH due to the extremely thin layer of acid covering it. [24], Several studies now show that short- and long-term psychological stress delays wound healing and that psychological stress may exacerbate certain dermatological conditions. Psychological stress also has similar negative effects on skin barrier recovery after disruption. In a medical student sample, approximately 30% skin barrier recovery was achieved at 3 hours after skin disruption during academic examinations, compared with 45% barrier recovery during the end of winter and spring vacation (20). In another study in healthy adults, skin barrier recovery was slowed by an inter-view stressor (approximately 55% 3 hours after disruption), similar to the laboratory stressor used in this study, compared with skin barrier recovery during a no stressful baseline the previous day (approximately 69%) (21). Notably, these studies used the same dermatological procedure employed in this study. Currently unknown is whether social support speeds [19]

GUT –BRAIN-SKIN INTERACTION AS PART OF PSYCHONEUROENDOCRINE IMMUNOLOGY:

The immune system, which may be the body's second most complicated organ after the brain, is essential to comprehend in psychiatry. It must be intelligent and agile enough to ruthlessly attack a wide range of diseases while preserving as many healthy bodily cells as feasible. Tissue-resident cells identify pathogens or tissue damage, triggering an immune response at the site of infection or injury. then they produce pro-inflammatory cytokines, such as interferons, interleukin-6 (IL-6), IL-1β, and tumor necrosis factor-α (TNF-α), which draw and activate additional immune cells from all throughout the body. [25] In the gut–brain–skin axis, cytokines, neurohormones, neuropeptides, and other messengers play a part in cellular signaling exchange. The gut and skin can be regarded as essential components of neuroendocrine and immunological organs. The idea that the stomach, psychological stress, and mental processes, as well as the skin, are related through gut-brain-skin contact was investigated. Here, mental illness is associated with a decrease in gut bifid and lactobacilli bacteria. An increased absorption of pro-inflammatory mediators from the gut lumen is caused by a malfunction in the gut barrier, which can be brought on by psychological stress through glucocorticoids. Mast cells, which are important cells in a number of psychodermatologic disorders, particularly psychophysiological

disorders, are involved in this process..[26] Neuropeptides/neurohormones, endocrine hormones, immunological cytokines, and connections to other bodily organs and systems are all secreted by the intestinal and cutaneous mucosa. As a result, both the skin and the intestine seem to be in a state of physiological inflammation, which is a natural occurrence. An important physiological process, inflammation is brought on by the continual exposure of the skin and intestines to germs and antigenic charges. Disruptions to the epithelial barrier, changes in the immune system, and disturbances in homeostasis can cause low-grade chronic inflammation, which in turn can cause disease development. Skin microbial alterations and a decline in physiological immunological capability are linked to dermatoses such as vitiligo, acne vulgaris, and atopic dermatitis. Drawing from the concept of psychoneuroendocrine immunology, Lotti et al. presented a novel strategy for treating both acute and chronic cutaneous inflammatory conditions by employing low dosage cytokine treatment.[8]

1.The role of the Micro biota in the regulation of the Gut–Brain Axis:

The two dominant microorganisms, Firmicutes and Bacteroidetes, make up 75–80% of the intestinal flora, which is composed of 10^14 units of microbial cells. A comparatively smaller percentage of intestinal microbes belong to the phyla Protobacteria, Acinetobacteria, Fusobacteria, and Verrucomicrobia. These organisms interact with the immunological and neurological systems and play a significant role in human health. The microbiota actively participates in the digestive processes by encouraging the absorption of nutrients through the intestinal epithelial membranes. In a state of health, they are in harmony with the activity of the host cells, guaranteeing the physiological homeostasis of the gut–brain axis. The microbiota, which controls cellular metabolism and immune response development, ensures the latter's integrity. Modifications to the blood-intestinal barrier's regulation of permeability can trigger innate immunity processes, encouraging systemic and cerebral inflammation and increasing vulnerability to stress and mental disorders. [27]. Actually, the commensal flora controls the production of antimicrobial proteins, the suppression of inflammatory reactions in the epithelium, the production of mucus to protect the epithelium surface, and the recovery of intestinal tissue damage. Numerous factors, including host genetics, toxins, infectious agents, age, location, diet, and drug use, can affect a microbiota's ability to operate and remain healthy. The newborn's microbiota at birth is also influenced by the mother's microbiome throughout lactation and delivery. Numerous neurotransmitters may be produced by the gut bacteria, according to recent study. For example, γ-aminobutyric acid (GABA), which is produced by Bifidobacterium and Lactobacillus, strengthens the inhibitory pathway in brain networks. In the meantime, Oscillibacter and Lactobacillus boost the expression of the tryptophan synthase gene, which raises the synthesis of serotonin. This synthetic pathway accounts for about 95% of 5-HT. This neurotransmitter production is influenced by a variety of circumstances, including immune system activity and vagus nerve activation. Stimulation of the afferent vagal pathways changes the activity of monocytes, macrophages, neutrophils, and dendritic cells and affects T cell recruitment and differentiation through G-protein-coupled receptors or histone deacetylases. [28].

2. Gut microbiota in psychiatric diseases:

Micro biome–Gut–Brain Axis (MGBA) in Depression:

The exact mechanisms of the gut–brain axis are not yet fully understood, although research highlights the crucial role of hormonal, metabolic, immunological, and neural signals—involving the central, autonomic, and enteric nervous systems the gut–brain relationship is considered bidirectional. On one hand, the gut micro-biota (closely linked to diet) influences the brain by modulating gene expression, affecting neurotransmitter pathways (serotonin, dopamine, glutamate, and GABA), regulating neu-roinflammation, and producing IGF-1.[29]

Schizophrenia (SCZ):

Schizophrenia (SCZ): Schizophrenia is a complex illness that affects cognitive, emotional, and occupational functioning. Adults with SCZ are at risk of dying young because of infectious, metabolic, and cardiovascular disorders. For people with SCZ living in the United States. It is projected that 28.5 years of life could be lost on average. Owen claims that SCZ showed three distinct dimensions: cognitive impairment (reduced efficiency in comparison to controls), negative symptoms (decreased desire and withdrawal), and positive symptoms (hallucinations and lack of contact with reality)[30]

The etiopathogenesis of SCZ is also attempted to be explained by the findings of biochemical and neuroimaging investigations. Some dependencies in the systems of specific neurotransmitters have been identified thus far. It could, at least in part, account for the emergence of SCZ clinical signs. The most important appears to be dopamine's involvement, while recent findings suggest that dopamine only indirectly contributes to this pathogenesis. The connections between other neurotransmitters should also be considered sources of SCZ. Kozłowska drew attention to the link between the aetiology of SCZ and immune/inflammatory processes. The development of numerous infection-induced or sterile inflammatory illnesses is caused by the activation of signalling pathways by host peptides/proteins known as alarmins. An increasing quantity of evidence suggests that the glutamatergic system plays a crucial role. This mostly relates to the G72 and G30 genes on chromosome 13q33, the first of which functions as an activator of amino acid oxidase ID-serine amino acid oxidase Inhibitor-DAOA, and the neuregulin 1 gene on chromosome 8p12, a chemical that stimulates NMDA receptors. The neurodevelopmental theory of SCZ and the function of the glutamatergic system in this process are supported by the identification of these genes. [31]

NEUROIMMUNODERMATOLOGY:

Individuals with higher post-traumatic stress (PTS) symptoms reported significantly poorer skin-related quality of life, indicating that psychological trauma and stress can worsen the perceived burden of skin disease.[32] Neuropeptides released by sensory nerves in the skin — such as Substance P, Calcitonin gene-related peptide (CGRP), Vasoactive intestinal peptide (VIP), somatostatin etc. — can directly modulate functions of skin cells (keratinocytes, Langerhans cells, mast cells, endothelial cells) and immune cells. Through this modulation (of cell proliferation, cytokine production, antigen presentation), these neuropeptides participate in a complex, interdependent network that regulates skin inflammation, immunity, wound-healing and overall skin immune responses. [33] Sensory neurons in the skin do more than mediating sensations like itch or pain — they directly regulate immune cell function. Activation of peripheral sensory

nerves can lead to release of neuropeptides (e.g. CGRP, Substance P), which modulate inflammation, immune-cell recruitment, epidermal changes, and host-defense responses. Hence, skin should be viewed as a true neuro-immune organ. [34] Skin-innervating sensory neurons do more than detect itch or pain — they actively regulate both innate and adaptive immune responses in the skin. On activation (by microbes, allergens, tissue-damage, cytokines, etc.), these neurons release neuropeptides that influence immune-cell behavior (like dendritic cell activation/migration, T-cell priming), thereby shaping barrier immunity, inflammation, and overall skin immune responses. [35] Recent evidence indicates that the skin's peripheral nervous system serves not only sensory functions but also as a dynamic regulator of innate immunity. In inflammatory skin diseases, activation of sensory neurons by immune-mediators leads to neuropeptide/neurotransmitter release, which in turn modulates immune cell activity, cytokine production, barrier integrity and inflammation — supporting the concept of skin as a neuro-immune organ rather than a passive barrier. [36].

IMPACT ON PSYCHOLOGICAL FACTORS ON SKIN DISEASE:

Human health is characterized by an intimate psycho-physiological unity, wherein psychological stress can manifest as a wide range of somatic disorders, including those affecting the skin. As the primary interface between the internal and external environments, the skin is uniquely vulnerable to both endogenous and exogenous stressors. Dermatology therefore occupies a distinct position among clinical specialties due to the visibility of skin diseases, which often amplifies their psychological and social impact.[37] Epidemiological studies from five European countries have reported a high lifetime prevalence of skin diseases, including eczema (14.2%), atopic dermatitis (7.9%), psoriasis (5.2%), and vitiligo (1.9%). With progressive environmental deterioration and increasing psychosocial pressures associated with modern lifestyles, the incidence of dermatological disorders has risen in recent years, establishing skin disease as a significant global public health concern.[38] Recent advances in psychodermatology have highlighted the presence of a bidirectional hypothalamic–pituitary–adrenal (HPA) axis within the skin. This peripheral stress response system plays a crucial role in regulating cutaneous inflammation, atopic responses, epidermal barrier function, dermal integrity, wound healing, and melanogenesis. These findings provide mechanistic insight into how psychological stress can directly influence skin physiology and pathology. In parallel, emerging research on the cutaneous microbiome has introduced the concept of stress-induced dysbiosis mediated through the "gut–brain–skin" axis. Alterations in microbial composition under chronic stress conditions may further compromise skin barrier function and immune regulation. Lifestyle factors such as diet, sleep patterns, and personal care practices have been shown to modulate these pathways and may either mitigate or exacerbate the cutaneous effects of psychological stress.[39] Many dermatological conditions are now understood within a biopsychosocial framework that integrates genetic susceptibility, personality traits, environmental triggers, and psychosocial stressors. In addition, several psychotropic medications have been observed to influence the course of dermatological diseases, underscoring the complex interplay between psychological health and skin function.[40].Psychological stress is thus recognized as a critical factor in the initiation and exacerbation of numerous skin disorders. Conversely, the visible nature of dermatological diseases often leads to social stigma and emotional distress, further intensifying psychological

burden and creating a self-perpetuating vicious cycle. A comprehensive understanding of the stress–skin interaction is therefore essential for effective prevention and management strategies in dermatological practice.[41]

PSYCHIATRIC DISORDERS WITH DERMATALOGIC SYMPTOMS:

1.Mind–Skin Relationship

Psychiatric disorders have a strong association with dermatologic symptoms, and the interaction between the mind and skin is bidirectional. Psychological conditions such as anxiety, depression, and stress can manifest as dermatologic symptoms like itching, skin picking, hair pulling, and exacerbation of existing skin diseases. At the same time, chronic dermatological conditions can lead to significant psychological distress, further worsening the skin symptoms. [42].

2. Prevalence of Psychiatric Comorbidity

Psychodermatology demonstrates that a significant proportion of dermatology patients (≈30–40%) have underlying psychiatric comorbidities, and the interplay between psychiatric disorders and skin conditions can be bidirectional — where mental health issues can contribute to or worsen dermatologic symptoms (e.g., anxiety increasing pruritus or self-inflicted skin lesions), and chronic visible skin disease can in turn lead to psychiatric disorders such as depression, anxiety, or body image disturbance.[43]

3. Mechanisms of Psychiatric Influence on Skin

Psychiatric illness can worsen skin symptoms through mechanisms such as stress-induced exacerbation, mechanical trauma, and medication effects, and dermatologic conditions can in turn lead to significant psychiatric morbidity. Emotional states and psychological stress influence skin diseases, while psychiatric disorders manifest as or worsen cutaneous symptoms.[44].

4. Severe Psychiatric Illness and Dermatologic Manifestations

In a comparative study of psychiatric patients and controls, there was a high incidence of severe psychiatric disorders such as schizophrenia (≈46.9%) and mood disorders (≈21.1%) among individuals presenting with dermatologic symptoms, highlighting that major psychiatric illnesses frequently coexist with or influence cutaneous manifestations.[45].

5. Psychological Impact of Chronic Dermatological Conditions

Chronic dermatological conditions are significantly linked with psychological morbidity, including anxiety, depression, social withdrawal, and stigmatization, which frequently accompany visible skin diseases. Mental health issues can both result from and amplify dermatologic symptoms, necessitating routine psychiatric screening and multidisciplinary treatment.[46]

6. Need for Integrated Psychodermatological Approach

Failure to recognize and address the underlying psychiatric component often results in poor treatment response and reduced quality of life. Therefore, an integrated psychodermatological approach, emphasizing a biopsychosocial evaluation and collaboration between dermatologists and mental health professionals, is essential for effective management.[47]

DISORDERS AND THEIR TREATMENT:

Treatment of Psoriasis:

Psoriasis is a chronic immune-mediated inflammatory disease, and its management focuses on long-term disease control rather than cure. Treatment selection is based on disease severity, extent of skin involvement, and impact on quality of life. Mild psoriasis is primarily managed with topical therapies, including corticosteroids and vitamin D analogues, which reduce inflammation and normalize keratinocyte proliferation. Moderate disease often requires phototherapy, particularly narrowband ultraviolet B (NB-UVB), which suppresses pathogenic T-cell activity. Moderate to severe psoriasis is treated with systemic agents such as methotrexate, cyclosporine, and acitretin. In patients with severe or refractory disease, biologic therapies targeting key inflammatory cytokines, including tumor necrosis factor-α and interleukins (IL-17 and IL-23), have shown high efficacy and sustained disease control. A holistic, individualized treatment approach is essential to optimize clinical outcomes and improve patient quality of life.[48] .

Treatment of Atopic Dermatitis:

The management of atopic dermatitis is based on restoring the skin barrier, reducing inflammation, and controlling pruritus. First-line treatment includes regular use of emollients combined with topical anti-inflammatory agents, mainly topical corticosteroids and topical calcineurin inhibitors. In patients with moderate to severe disease, treatment may be escalated to phototherapy or systemic immunomodulatory agents, while emerging targeted biologic therapies offer effective options for refractory cases.[49]

Treatment of Acne Excoriée:

Management of acne excoriée requires a combined dermatological and psychological approach, with emphasis on behavioral modification and treatment of underlying psychiatric comorbidities, alongside gentle acne therapy to prevent further skin damage and scarring.[50]

Treatment of Hyperhidrosis:

Effective management of hyperhidrosis depends on differentiating primary from secondary hyperhidrosis, as secondary forms require treatment of the underlying cause, while primary focal hyperhidrosis is managed with stepwise symptomatic therapies such as topical agents, systemic medications, or procedural interventions. [51]

Treatment of Urticaria:

According to international guidelines, the first-line treatment of urticaria is second-generation non-sedating H1 antihistamines, with dose escalation up to fourfold in patients with inadequate symptom control, followed by advanced therapies in refractory cases .[52]

Treatment of Herpes Simplex Virus Infection

Treatment of herpes simplex virus infection is centered on systemic antiviral therapy, which reduces symptom severity, shortens lesion duration, and decreases the frequency of recurrences when used as suppressive therapy in patients with frequent outbreaks[53]

Treatment of Seborrheic Dermatitis:

Topical therapies are the mainstay of treatment for facial seborrheic dermatitis. Low- to moderate-potency corticosteroids, antifungal agents such as ketoconazole or ciclopirox, and combination formulations can reduce inflammation and control Malassezia colonization. Regular

use of gentle cleansers and emollients helps maintain skin barrier function and prevent flare-ups.[54]

Treatment of Aphthous Ulcers:

Management of recurrent aphthous stomatitis focuses on reducing pain, accelerating healing, and preventing recurrences. Topical corticosteroids are the first-line therapy to control inflammation, while anesthetic gels or mouth rinses provide symptomatic relief. Additional options include topical immunomodulators, sucralfate, and antiseptic mouthwashes. Severe or frequent episodes may require systemic therapy under clinician supervision. Identifying and managing triggers, such as nutritional deficiencies or stress, is also recommended.[55]

Treatment of Rosacea:

Management of rosacea aims to control inflammation, reduce flushing, and prevent disease progression. Lifestyle modifications, including trigger avoidance (sun exposure, spicy foods, alcohol, heat) and gentle skincare, form the foundation of therapy. Topical treatments such as metronidazole, azelaic acid, and ivermectin are commonly used for inflammatory lesions, while vasoconstrictive agents like brimonidine target persistent erythema. Moderate to severe cases may require oral antibiotics with anti-inflammatory properties. Procedural options, including laser and light therapies, are effective for persistent telangiectasia or redness. [56]

Treatment of Chronic Pruritus:

Management of chronic pruritus requires a multimodal approach targeting both peripheral and central mechanisms. In addition to treating the underlying cause and optimizing skin barrier function with regular emollients, systemic therapies such as antihistamines, gabapentinoids, antidepressants, and opioid receptor modulators may be required. Phototherapy and centrally acting agents are particularly useful in patients with chronic, refractory pruritus. [57]

Treatment of Dermatitis Artefacta:

Management of dermatitis artefacta requires a multidisciplinary approach focused on both physical and psychological aspects. Establishing a non-confrontational, empathetic doctor–patient relationship is essential to encourage adherence and reduce further self-inflicted lesions. Symptomatic care of skin lesions includes wound care, topical antibiotics if necessary, and protective dressings. Referral to mental health professionals for psychological assessment and counseling is recommended to address underlying psychiatric conditions, and in selected cases, psychotropic medications such as SSRIs may be used to manage associated mood or impulse-control issues. [58]

Treatment of Delusional Parasitosis:

Management of delusional parasitosis requires a multidisciplinary approach involving dermatologists and mental health professionals. Establishing a non-confrontational, empathetic doctor–patient relationship is crucial to gain patient trust. Pharmacological therapy primarily involves antipsychotic medications, with second-generation agents such as risperidone or olanzapine preferred for their efficacy and safety profile. Regular follow-up and psychological support improve adherence and outcomes, while addressing self-inflicted skin lesions symptomatically with wound care prevents complications. [59]

Treatment of Trichotillomania:

Management of trichotillomania focuses on reducing hair-pulling behaviors and improving psychosocial functioning. Behavioral therapies, particularly habit reversal training (HRT) and other cognitive-behavioral techniques, are first-line treatments. Pharmacologic interventions, including selective serotonin reuptake inhibitors (SSRIs) or N-acetylcysteine, may be considered in cases with significant distress or co-morbid psychiatric conditions. Supportive strategies such as stress management, psychoeducation, and engagement in alternative behaviors are useful adjuncts. [60]

Treatment of Obsessive-Compulsive Disorder:

Management of OCD involves a combination of psychotherapy, pharmacotherapy, and supportive interventions. Cognitive-behavioral therapy (CBT), particularly Exposure and Response Prevention (ERP), is the most effective evidence-based psychotherapy, helping patients confront obsessions without performing compulsions. Selective serotonin reuptake inhibitors (SSRIs) such as fluoxetine, sertraline, and fluvoxamine are first-line medications, while clomipramine may be used in resistant cases. Many patients benefit from combined therapy (CBT + medication). Long-term adherence and regular follow-up are essential to maintain improvement and reduce relapse. [61]

Treatment of Phobic States:

Management of phobic states focuses on reducing fear, anxiety, and avoidance behaviors. Exposure-based cognitive behavioral therapy (CBT) is the mainstay of treatment, involving gradual, controlled exposure to the feared object or situation to desensitize the patient. Relaxation techniques, breathing exercises, and coping strategies are taught alongside exposure to manage acute anxiety. In selected cases, short-term pharmacologic interventions such as benzodiazepines or beta-blockers may help during anxiety-provoking situations. Emerging approaches, including virtual reality exposure therapy, offer additional options when real-life exposure is impractical.[62]

Treatment of Dysmorphophobia (BDD):

Cognitive Behavioral Therapy (CBT) is the first-line treatment for dysmorphophobia, effectively reducing preoccupations with perceived physical flaws and associated compulsive behaviors. CBT involves identifying and challenging distorted thoughts about appearance, reducing repetitive checking behaviors, and gradually exposing patients to avoided situations. Therapy also incorporates strategies to improve coping skills, reduce anxiety, and enhance overall psychosocial functioning. [63]

Treatment of Eating Disorders:

Management of eating disorders requires a multidisciplinary approach involving medical, nutritional, and psychological care. Psychotherapy, including cognitive behavioral therapy (CBT) and family-based therapy (FBT), is central to treatment and helps patients modify disordered eating patterns and address underlying psychological issues. Nutritional rehabilitation is essential for restoring healthy weight and correcting malnutrition. Pharmacologic interventions, such as selective serotonin reuptake inhibitors (SSRIs), may help reduce bingeing, purging, or co-morbid mood and anxiety symptoms. Ongoing monitoring, support, and early intervention are crucial to improving outcomes and preventing relapse. [64]

Treatment of Neurotic Excoriations (Skin-Picking Disorder):

Management of neurotic excoriations involves a combination of behavioral and pharmacologic approaches. Behavioral therapies, particularly habit reversal training (HRT) and cognitive-behavioral therapy (CBT), are first-line treatments and help patients identify triggers, resist urges, and adopt alternative behaviors. Pharmacotherapy, including selective serotonin reuptake inhibitors (SSRIs), may be employed for moderate-to-severe cases or when comorbid psychiatric conditions exist. Supportive measures such as stress management, mindfulness, and wound care further improve outcomes. [65]

Treatment of Psychogenic Pruritus:

Management of psychogenic pruritus involves a multidisciplinary approach combining psychological and medical strategies. Cognitive-behavioral therapy (CBT) is the mainstay of treatment, helping patients identify stressors, modify maladaptive coping, and reduce the itch–anxiety cycle. Pharmacologic interventions, including selective serotonin reuptake inhibitors (SSRIs) or other psychotropic agents, may be used when psychiatric comorbidities are present or symptoms are severe. Supportive measures, such as stress management, relaxation techniques, and strong physician–patient communication, enhance treatment adherence and overall outcomes. [66]

Treatment of alopecia areata:

Management of alopecia areata involves both topical and systemic approaches depending on disease severity. Topical and intralesional corticosteroids are commonly used for limited patchy hair loss, while topical immunotherapy and minoxidil support hair regrowth in more extensive cases. Systemic therapies, including corticosteroids, immunosuppressants, and Janus kinase (JAK) inhibitors, have shown effectiveness in moderate-to-severe or refractory disease. Treatment selection is guided by disease extent, patient age, comorbidities, and response to prior therapies, with ongoing monitoring to optimize outcomes. [67]

Treatment of Vitiligo:

Management of vitiligo focuses on halting disease progression and promoting repigmentation. Topical corticosteroids and calcineurin inhibitors are first-line therapies for localized lesions, reducing inflammation and stimulating melanocyte activity. Phototherapy, particularly narrowband UVB (NB-UVB), is effective for widespread vitiligo, enhancing melanocyte proliferation and migration. In stable, treatment-resistant cases, surgical interventions such as skin grafting or melanocyte transplantation may be considered. Adjunctive measures, including cosmetic camouflage, patient education, and psychological support, are important for improving quality of life. [68].

Treatment of Generalized Pustular Psoriasis:

Generalized pustular psoriasis is a severe and potentially life-threatening form of psoriasis requiring prompt and effective treatment. The therapeutic approach typically involves systemic therapies rather than topical-only treatments due to the extensive skin involvement and frequent systemic symptoms. First-line systemic agents include oral retinoids such as acitretin, immunosuppressants like methotrexate and cyclosporine, and biologic therapies such as infliximab — all of which have been shown to help control widespread pustules and inflammation. These medications are chosen based on disease severity, patient comorbidities, and treatment response, often with rapid initiation to reduce systemic symptoms and prevent

complications. Biological agents targeting inflammatory pathways are increasingly utilized, and newer targeted therapies such as IL-36 receptor antagonists are under investigation, offering additional options for refractory cases. [69]

Treatment of Chronic Eczema:

Effective management of chronic eczema requires a stepwise and long-term treatment approach aimed at restoring the skin barrier and controlling underlying inflammation. Regular and liberal use of emollients is emphasized as the foundation of therapy to reduce transepidermal water loss and prevent disease flares. During active inflammation, topical corticosteroids remain the first-line treatment, while topical calcineurin inhibitors are recommended for sensitive areas and for maintenance therapy to minimize steroid-related adverse effects. In patients with moderate to severe or refractory disease, systemic immunomodulatory therapies and targeted biologics are indicated to achieve sustained disease control.[70]

Treatment of Ichthyosiform Syndromes:

The management of ichthyosiform disorders, including syndromic forms, is primarily symptomatic and focused on long-term skin barrier repair. Regular application of emollients remains the cornerstone of therapy to reduce scaling and improve skin hydration, while keratolytic agents such as urea or lactic acid are useful in controlling hyperkeratosis. In severe or refractory cases, systemic retinoids may be considered to normalize epidermal differentiation, with careful monitoring for adverse effects. [71]

Treatment of Rhinophyma:

Rhinophyma represents a phymatous subtype of rosacea, in which medical therapy alone is often insufficient in advanced stages. Early intervention targeting rosacea-associated inflammation, including anti-inflammatory and sebosuppressive treatments, may help slow disease progression; however, established rhinophyma requires surgical or procedural management to remove excess tissue and restore normal nasal contour.[72]

Treatment of Neurofibroma:

Patients with neurofibromatosis type 1 (NF1) require regular clinical surveillance of neurofibromas, as a subset—particularly plexiform neurofibromas—carry a risk of transformation into malignant peripheral nerve sheath tumors (MPNSTs). Therefore, early surgical excision of symptomatic or rapidly enlarging lesions, along with prompt evaluation of pain, neurological deficit, or sudden growth, is essential to reduce morbidity and improve outcomes[73]

Treatment of Albinism:

The increasing genetic heterogeneity of albinism underscores that no curative therapy is currently available, and management remains supportive and preventive. Early diagnosis allows timely implementation of photoprotection measures and regular ophthalmologic care, which are essential to reduce cutaneous complications and optimize visual function despite underlying genetic defects. [74]

Treatment of Cutaneous Sensory Syndrome:

Based on the similarities between chronic itch and neuropathic pain described by Ständer and Schmelz, cutaneous sensory syndrome is best managed using neuromodulatory treatment strategies rather than conventional antipruritic therapy. Agents such as gabapentin, pregabalin,

and certain antidepressants are effective in reducing abnormal sensory signaling, supporting the concept that these conditions represent neuropathic dysfunction of cutaneous sensory pathways .[75]

Treatment of Glossodynia:

Effective management of glossodynia involves a multifaceted approach targeting both neuropathic pain and contributing factors. Topical agents such as lidocaine or capsaicin can provide symptomatic relief, while systemic therapies including clonazepam, tricyclic antidepressants, or alpha-lipoic acid may benefit patients with persistent neuropathic pain. Identification and correction of nutritional deficiencies (iron, vitamin B12, folate) and psychological support are also essential components of comprehensive care.[76]

Treatment of Vulvodynia:

Management of vulvodynia should follow a multimodal and individualized approach, addressing both neuropathic pain and pelvic floor dysfunction. First-line therapies include topical anesthetics, estrogen creams, and pelvic floor physical therapy, while oral neuromodulators such as tricyclic antidepressants or gabapentin may be used for persistent pain. Psychological interventions, including cognitive behavioral therapy, are essential to address the chronic pain cycle and improve quality of life. [77]

Treatment of Chronic Scalp Itching:

Chronic scalp itching, particularly when neuropathic or psychogenic in origin, responds better to neuromodulatory therapies rather than conventional anti-itch treatments. Agents such as gabapentin or pregabalin can reduce abnormal nerve signaling, while psychological interventions including stress management and cognitive behavioral therapy help in controlling itch perception and improving quality of life. [78]

Treatment of Psychogenic Purpura Syndrome:

Psychogenic purpura (autoerythrocyte sensitization syndrome) requires a multidisciplinary approach focused on psychological support and stress management, as emotional stress is a key trigger. Cognitive behavioral therapy, counseling, and management of underlying psychiatric conditions are the mainstay of therapy, while symptomatic care with topical analgesics can relieve discomfort during acute bruising episodes.[79]

Treatment of Pseudo-Psychodermatologic Disorders:

Management of pseudo-psychodermatologic conditions should focus on treating the underlying dermatologic or systemic cause while also addressing neuroimmune mechanisms that contribute to itch and pain. Therapies may include topical or systemic agents targeting inflammation or neuropathic pathways, alongside supportive care for associated discomfort. Psychological interventions are reserved for secondary stress or anxiety resulting from chronic symptoms.[80]

Management of Suicide Risk in Dermatology Patients:

Dermatology patients at risk of suicidal ideation benefit from early psychosocial assessment and intervention. Regular screening for depression, anxiety, and suicidal thoughts, combined with referral to mental health professionals, is essential. Integrating psychological support, counseling, and patient education alongside dermatologic care helps reduce emotional distress and suicide risk.[81]

Anxiety in psychodermatology	Depression in psychodermatology	Obsessive-compulsive spectrum in psychodermatology	Psychotic disorders in psychodermatology
• Acute anxiety: • benzodiazepine (e.g.: diazepam, lorazepam, clonazepam); • hydroxyzine • psychotherapy (e.g.: cognitive behavioral therapy; mindfulness therapies, hypnosis). • Chronic anxiety: • selective serotonin reuptake inhibitor; psychotherapy.	• Antidepressant (selective serotonin reuptake inhibitor; serotonin and norepinephrine reuptake inhibitor); • Psychotherapy (e.g.: cognitive behavioral therapy, hypnosis)	• Antidepressant (selective serotonin reuptake inhibitor); • Cognitive behavioral therapy; • N-acetylcysteine; • Antiepileptics can be added in combination with the antidepressant in some cases of skin-picking syndromes.	• Antipsychotic (e.g.: risperidone).

MANAGEMENT OF PSYCHOCUTANEOUS PATIENTS

Psychocutaneous Medicine:

Psychocutaneous medicine, also referred to as psychodermatology, is a subspecialty that lies at the interface of dermatology and psychiatry, focusing on the complex interactions between the skin and the brain. For many patients, dermatological diseases significantly impair quality of life and mental well-being, while in others, psychiatric disorders manifest with prominent cutaneous signs.[82] Psychocutaneous conditions are characterized by disorders in which psychological stress plays a pivotal role in the onset, exacerbation, or recurrence of skin diseases. Chronic dermatoses such as psoriasis and atopic dermatitis commonly show disease flares during periods of stress. There is a well-recognized direct relationship between stress, disease course, and prognosis in several inflammatory skin disorders.[83]

Psychodermatology Broadly Encompasses:

1. Primary dermatological disorders with psychosocial comorbidities, such as patients with psoriasis or acne who develop anxiety, depression, or suicidal ideation.

2. Primary psychiatric disorders presenting with dermatological manifestations, including delusional infestation, body dysmorphic disorder, and dermatitis artefacta.

Patients presenting with primary psychiatric disorders in dermatology clinics are relatively common, while psychosocial comorbidities in individuals with chronic skin diseases are extremely prevalent.[84]

Clinical Experience from a Psychocutaneous Clinic:

A retrospective study conducted at the University of Wisconsin was approved by the Institutional Review Board (IRB) under exempt category 4, as it involved secondary research without the need for patient consent. All patients referred to the Psychocutaneous Clinic between May 2002 and February 2018 were included. Data were collected from 808 referrals, predominantly from dermatologists, with additional referrals from psychiatrists, internists, and other specialties such as surgery and infectious diseases.[82]

Management Principles in Psychocutaneous Disorders

Management of psychocutaneous patients requires additional time, patience, and a multidisciplinary approach. Longer consultation appointments are essential. A quiet consultation room with adequate privacy is crucial, as many patients may feel embarrassed or reluctant to discuss psychological issues in the presence of others. Establishing a strong doctor–patient rapport is fundamental for successful management.[85] Patients with primary psychiatric disorders often lack insight and may resist mental health referral unless facilitated by a dermatologist. This highlights the need for specialized training in psychocutaneous medicine within dermatology departments. The clinical approach must be adapted, as these patients may show poor compliance and limited disclosure compared to other dermatology patients.[86] When psychiatric morbidity arises secondary to a primary dermatological condition, it is beneficial to directly inquire about the patient's mental health. In psychophysiologic disorders, treatment of the underlying skin disease remains the primary objective, as cutaneous symptoms may themselves generate stress and perpetuate a vicious cycle.[83]

Psychogenic Pruritus and Psychiatric Risk

Psychogenic itch is diagnosed in patients with localized or generalized pruritus lasting longer than six weeks, in the absence of an identifiable somatic cause. Psychological stress can lower the itch threshold, leading to secondary dermatological conditions such as nodular prurigo, neurotic excoriations, and frictional amyloidosis, along with psychiatric comorbidities including depression and anxiety.[87] Although dermatological diseases alone rarely cause a marked increase in suicide risk, emotionally vulnerable individuals may experience worsening psychological distress when confronted with severe or disfiguring skin conditions.[88]. Many patients with psychocutaneous disorders resist psychiatric referral and may feel offended when such suggestions are made. In such situations, dermatologists may need to address psychological aspects indirectly or focus primarily on dermatological management while gradually introducing mental health support.[89]

Neuro-Immuno-Cutaneous-Endocrine Interaction:

The intricate relationship between the mind and skin has been extensively studied at both molecular and cellular levels. The brain, nerves, and skin are embryologically derived from the ectodermal neural plate, providing a biological basis for their interaction.[90]. The neuro-immuno-cutaneous-endocrine (NICE) model, proposed by O'Sullivan et al., explains the bidirectional communication between psychological factors and cutaneous inflammation. This

model underpins many inflammatory dermatoses that are triggered or exacerbated by psychological stress.[91]

PHYCHOPHARMACOLOGY IN DERMATOLOGY:

Psychopharmacology in dermatology addresses psychodermatologic disorders, where psychiatric illnesses manifest with prominent cutaneous symptoms. Common presentations include trichotillomania, excoriation disorder, body dysmorphic disorder, onychophagia, and delusions of parasitosis, frequently encountered in dermatologic practice [92,15]. Many patients are reluctant to seek psychiatric care, necessitating that dermatologists initiate psychopharmacologic therapy when appropriate [93,94]. Psychological factors, including stress, anxiety, depression, and compulsive behaviors—can influence the onset, severity, and progression of skin diseases, often triggering, exacerbating, or perpetuating dermatological conditions [98,99,100]. Psychosomatic dermatology explains how these psychological factors can worsen cutaneous symptoms, highlighting the close connection between the skin and the nervous system, where stress can aggravate itching, inflammation, and other manifestations [100]. Effective management involves identifying underlying psychopathology and tailoring treatment with psychotropic agents—such as selective serotonin reuptake inhibitors, atypical antipsychotics, anxiolytics, antidepressants, mood stabilizers, or N-acetylcysteine (NAC)—with psychiatric consultation advised in complex or refractory cases [92-94,15,95–97]. Psychiatric comorbidities affect approximately 25–30% of patients with chronic dermatologic disorders and can significantly impact treatment outcomes [98]. A multidisciplinary approach involving both dermatologists and mental health professionals is essential to optimize outcomes, particularly when psychiatric referral is not feasible, and familiarity with psychotropic medications allows dermatologists to manage patients safely while monitoring for potential cutaneous adverse effects [92-94,98,99]. In addition to traditional psychotropics, glutamatergic modulators such as NAC have shown efficacy in reducing compulsive behaviors associated with trichotillomania, excoriation disorder, and related body-focused repetitive behaviors, as supported by randomized controlled trials and literature reviews [95–97]. Practical psychodermatology emphasizes this integration of dermatologic and psychiatric care, highlighting how psychological factors directly influence disease course and patient outcomes [98,99]

CONCLUSION:

Psychodermatology emphasizes the strong bidirectional relationship between the mind and the skin. Psychological stress and psychiatric disorders can trigger or worsen many dermatological conditions, while chronic skin diseases can significantly affect mental health and quality of life.Therefore, a holistic biopsychosocial approach integrating dermatologic and psychological care is essential for effective management and improved patient outcomes.

REFERENCE:

1.Koo J, Lebwohl A. Psychodermatology: the mind and skin connection. Am Fam Physician. 2001;64(11):1873–1878.

2. Latheef ENA, Hafi BNA. Introduction to psychodermatology. Clin Dermatol Rev. 2023;7(4):303–9. doi:10.4103/cdr.cdr_10_22.

3.Bondade S, Hosthota A, Bindushree R, Raj PR. Psychodermatology: An evolving paradigm. Dermatologica Sinica. 2022;40(2):71–77. doi:10.4103/ds.ds_20_22.

4. Marshall C, Taylor R, Bewley A. Psychodermatology in clinical practice: Main principles. Acta Derm Venereol. 2016;96(Suppl 217):30–34. doi:10.2340/00015555-2370.

5. Picardi A, Abeni D, Melchi CF, Puddu P, Pasquini P. Psychiatric morbidity in dermatological outpatients: an issue to be recognized. Br J Dermatol. 2000;143(5):983–91.

6.Gupta MA, Gupta AK. Depression and suicidal ideation in dermatology patients with acne, alopecia areata, atopic dermatitis and psoriasis. Br J Dermatol. 1998;139(5):846–50.

7.O'Sullivan RL, Lipper G, Lerner EA. The neuro-immuno-cutaneous-endocrine network: relationship of mind and skin. Arch Dermatol. 1998;134(11):1431–35.

8. Munoli RN. Psychodermatology: An overview of history, concept, classification, and status. Indian J Priv Psychiatry. 2020;14(2):85-91.

9.Rodríguez-Cerdeira C, López-Barcenas A, Sánchez-Blanco E, Fabbrocini G, De Rosa M, Abeni D. Psychodermatology: Past, present and future. Int J Dermatol. 2011;50:23-26.

10.Koo J, Lee CS. Psychocutaneous medicine: The mind–skin connection. Dermatol Clin. 2003;21(4):535-544.

11. Picardi A, Pasquini P, Damiano Abeni, Fassone G, Mazzotti E, Fava GA. Psychosomatic assessment of skin diseases in clinical practice. Psychother Psychosom. 2005;74(5):315–322.

12. Gupta MA, Gupta AK. Psychiatric comorbidity in dermatology. J Eur Acad Dermatol Venereol. 1998;10:1–9.

13. Tatu AL, Nwabudike LC. Special Issue "Diagnosis and Management of Skin Diseases, Related Disorders and Their Comorbidities". Diagnostics. 2025;15(7):897.

14. National Institutes of Health. Title of document [Internet]. Bethesda (MD): National Institutes of Health; Year [cited 2025 Dec 05]. Available from: https://share.google/tzJzrmn9Q9AbMXwjC

15. Ha EL, Magid M. Psychopharmacology in dermatology: Five common disorders. Clin Dermatol [Internet]. 2023 Jan-Feb [cited 2025 Dec 05];41(1):112-20. doi:10.1016/j.clindermatol.2023.03.010

16.Cline A, Hogan SR, Pope E. A review of the diagnosis and management of pediatric psychodermatologic conditions: Part I. Pediatric Dermatology. 2021. PMID: 34929757.

17.Cline A, Hogan SR, Pope E. A review of the diagnosis and management of pediatric psychodermatologic conditions: Part II. Pediatric Dermatology. 2021. PMID: 34964183.

18.StrangeTA, Clark HL,Dixon LJ.Potentially traumatic events, posttraumatic stress symptoms, and skin-related quality of life among adults with self-reported skin disease symptoms. Arch Dermatol Res. 2024;317(1):19. doi:10.1007/s00403-024-03451-w

19. Robles TF. Stress, social support, and delayed skin barrier recovery. Psychosom Med. 2007;69(8):807–815. doi:10.1097/PSY.0b013e318157b12e.

20. Slominski A, Wortsman J, Tuckey RC, Paus R.Differential expression of a cutaneous HPA axis homolog.Mol Cell Endocrinol. 2007;265–266:143–149.doi:10.1016/j.mce.2006.12.012.

21.Terao M, Murota H, Kimura A, Kato A, Ishikawa A, Igawa K, et al.11β-Hydroxysteroid dehydrogenase-1 regulates stress-induced skin barrier dysfunction.J Invest Dermatol. 2012;132(6):1682-1690oi:10.1038/jid.2012.35

22. Choe SJ, Kim D, Kim EJ, Ahn JS, Choi EJ, Son ED, Lee TR, Choi EH. Psychological stress deteriorates skin barrier function by activating 11β-hydroxysteroid dehydrogenase 1 and the HPA axis. Sci Rep. 2018;8:6334. doi:10.1038/s41598-018-24653-z.

23. Proksch E, Brandner JM, Jensen JM. The skin: an indispensable barrier. Exp Dermatol. 2008;17(12):1063-72.

24. Baker P, Huang C, Radi R, Moll SB, Jules E, Arbiser JL. Skin barrier function: the interplay of physical, chemical, and immunologic properties. Cells. 2023;12(23):2745. doi:10.3390/cells12232745.

25. Nathan C. Points of control in inflammation. Nature. 2002;420(6917):846-52. Akira S, Uematsu S, Takeuchi O. Pathogen recognition and innate immunity. Cell. 2006;124(4):783-801.

26. Cryan JF, Dinan TG. Mind-altering microorganisms: the impact of the gut microbiota on brain and behaviour. Nat Rev Neurosci. 2012;13(10):701–712.

27. Kelly JR, Minuto C, Cryan JF, Clarke G, Dinan TG.Cross talk: the microbiota and neurodevelopmental disorders. Front Neurosci.2017;11:490.doi:10.3389/fnins.2017.00490.

28. Marano G, Mazza M, Lisci FM, Ciliberto M, Traversi G,Kotzalidis GD, De Berardis D, Laterza L, Sani G, Gasbarrini A, Gaetani E. The microbiota–gut–brain axis: psychoneuroimmunological insights. Nutrients. 2023; 15(6):1496.

29.Diedrick M, Mytych A, Szyller HP, Lasocka M, Augustynowicz G, Szydziak J, Hrapkowicz A, Dyda M, Braksator J, Pytrus T. Gut Microbiota in Psychiatric and Neurological Disorders: Current Insights and Therapeutic Implications. Biomedicines. 2025;13(9):2104

30. Owen MJ, Sawa A, Mortensen PB. Schizophrenia. Lancet. 2016;388(10039):86–97.

31.Góralska-Bińkowska A, Smajda-Krygier D, Kozłowska E. The Microbiota–Gut Brain Axis in Psychiatric Disorders. Int J Mol Sci. 2023;23(19):11245. doi:10.3390/ijms231911245.

32. Darsow U, Ring J. Neuroimmune interactions in the skin. Curr Opin Allergy Clin Immunol. 2001 Oct;1(5):435–9. doi: 10.1097/01.all.0000011057.09816.61.

33. Scholzen T, Armstrong CA, Bunnett NW, Luger TA, Olerud JE, Ansel JC. Neuropeptides in the skin: interactions between the neuroendocrine and the skin immune systems. Exp Dermatol. 1998 Apr-Jun;7(2-3):81–96. doi:10.1111/j.1600-0625.1998.tb00307.x.

34. Trier AM, Mack MR, Kim BS. The neuroimmune axis in skin sensation, inflammation, and immunity. J Immunol. 2019 May 15; 202(10):2829–2835. doi: 10.4049/jimmunol.1801473.

35. Feng X. Sensory neuronal control of skin barrier immunity. Curr Opin Immunol. 2024; 102:ON-line (pii S1471-4906 2024 000668). doi:10.1016/j.coi.2024.000668.

36. Huang X, Li F, Wang F. Neural Regulation of Innate Immunity in Inflammatory Skin Diseases. Pharmaceuticals (Basel). 2023 Feb 6;16(2):246. doi:10.3390/ph16020246.

37. Dalgard FJ, Gieler U, Tomas-Aragones L, Lien L, Poot F, Jemec GB, et al. The psychosocial adaptation of patients with skin disease: a scoping review. BMC Public Health. 2019; 19:1404.

38. Ghosh I, Sinha S. Psychological characteristics of patients suffering from skin disorders. Indones J Health Sci Res Dev. 2023;5(2):43–53. doi:10.36566/ijhsrd/Vol5.Iss2/170.

39. Sun MD, Rieder EA. Psychosocial stress and mechanisms of skin health: a comprehensive update. J Drugs Dermatol. 2021;20(1):62–69. doi:10.36849/JDD.5608.

40. Folks DG, Kinney FC. The role of psychological factors in dermatologic conditions. Psychosomatics. 1992;33(1):45–54. doi:10.1016/S0033-3182(92)72020-9.

41.Zhang H, Wang M, Zhao X, Wang Y, Chen X, Su J. Role of stress in skin diseases: a neuroendocrine-immune interaction view. Brain Behav Immun. 2024;116:286–302. doi: 10.1016/j.bbi.2023.12.005.

42. Connor CJ. Management of the psychological comorbidities of dermatological conditions: practitioners' guidelines. Clin Cosmet Investig Dermatol. 2017 Apr 20; 10:117–132. doi:10.2147/CCID.S111041. PMID:28458571; PMCID: PMC5404497.

43. Ghosh S, Behere RV, Sharma PSVN, Sreejayan K. Psychiatric evaluation in dermatology: An overview. Indian J Dermatol. 2013 Jan–Feb;58(1):39–43. doi:10.4103/0019-5154.105286. PMID:23372211; PMCID: PMC3555371.

44. Kaye AD, Islam RK, Castine AM, Wester GC, Barham WT, Nguyen CG, Plakotaris E, Dorius B, Kim J, Griffin P, Edinoff A, Shekoohi S, Varrassi G. Exploring the relationship between psychiatric illness and dermatological disorders: a narrative review. Cureus. 2025 Feb 8;17(2):e78731. doi:10.7759/cureus.78731. PMID:40065888.

45. Buksh HA, Khan ZU, Fahad M. Enhancing clinical performance through workplace-based assessment in medicine. J Popul Ther Clin Pharmacol. 2024;31(1):3068-3073. doi:10.53555/p9r8qc55.

46. Baskaran N, Arunima A, Shah S, Narang T, Kumaran MS. Psychological morbidity in chronic dermatological disorders: a review. Indian Dermatol Online J. 2025 Apr 17;16(3):381–388. doi: 10.4103/idoj.idoj_458_24. PMID:40395584; PMCID: PMC12088501.

47.Basavaraj KH, Navya MA, Rashmi R. Relevance of psychiatry in dermatology: Present concepts. Indian J Psychiatry. 2010 Jul–Sep;52(3):270–275. doi:10.4103/0019-5545.70992. PMID:21180416; PMCID: PMC2990831.

48. Griffiths CEM, Armstrong AW, Gudjonsson JE, Barker JNWN. Psoriasis. Lancet. 2021;397(10281):1301-15.

49. Langan SM, Irvine AD, Weidinger S. Atopic dermatitis. Lancet. 2020;396(10247):345–60.

50.Gupta MA, Gupta AK. Psychodermatology: An update. J Am Acad Dermatol. 2013;69(2): 207.e1–207.e12.

51.Walling HW. Clinical differentiation of primary from secondary hyperhidrosis. J Am Acad Dermatol. 2011;64(4):690–5

52.Zuberbier T, Aberer W, Asero R, et al. The EAACI/GA²LEN/EDF/WAO guideline for the definition, classification, diagnosis, and management of urticaria. Allergy. 2018;73(7):1393–414.

53. Johnston C, Wald A. Epidemiology, clinical manifestations, and diagnosis of herpes simplex virus infections. 2024.

54.Gupta AK, Versteeg SG. J Drugs Dermatol. 2017;16(5):440–4.

55.Lau CB, Smith GP. Recurrent aphthous stomatitis: a comprehensive review and recommendations on therapeutic options. Dermatol Ther. 2022;35(6):e15500.

56.A Review on Pathophysiology and Current Treatment Trends of Rosacea: Review Article. J Pharma Insights Res. 2025;3(2):185–93).

57.Yosipovitch G, Bernhard JD. Chronic pruritus. N Engl J Med. 2013;368(17):1625–34.

58.Estill MC, Jafferany M. Eur Med J. 2024;9(2):104–115.

59.Ahmed A, Affleck AG, Angus J, et al. Br J Dermatol. 2022;187(4):472–480.

60. Grant JE, Odlaug BL, Chamberlain SR. Expert Rev Neurother. 2012;12(4):379–386.

61. National Institute of Mental Health (NIMH). Obsessive-Compulsive Disorder NIMH. 2024.

62.Samra CK, Torrico TJ, Abdijadid S. Specific Phobia. In: StatPearls [Internet]. Treasure Island (FL): StatPearls Publishing; 2025.

63.Prazeres AM. J Clin Psychiatry. 2013; doi:PMC3589080.

64. Brownstone L, Thibault G. Management of Eating Disorders: A Review. CMAJ. 2018;190:E259–E265. doi:10.1503/cmaj.170973.

65.Stein DJ, Lochner C. Pharmacotherapy for skin picking disorder. Curr Psychiatry Rep. 2015;17:579. doi:10.1007/s11920-015-0579-4.

66. Misery L, Dutray S, Chastaing M, et al. Psychogenic itch. Transl Psychiatry. 2018;8:52. doi:10.1038/s41398-018-00.

67. Tsai CF, et al. Real-world evidence in alopecia areata treatment. Dermatol Ther. 2024.

68. Ezzedine K, Lim HW, Suzuki T, et al. Vitiligo. N Engl J Med. 2015;372:58–69. doi:10.1056/NEJMra1404255.

69. Romitia R, da Silva Hirayama AL, Arnone M, Magalhães RF. Generalized pustular psoriasis (von Zumbusch). An Bras Dermatol. 2022;97(1):63–74. doi:10.1016/j.abd.2021.05.011.

70. Wollenberg A, et al.Atopic dermatitis: disease mechanisms and therapy. Allergy. 2018;73(1):24-34.

71. Traupe H, Fischer J, Oji V. Nonsyndromic types of ichthyoses—an update. J Dtsch Dermatol Ges. 2014;12(2):109-21.

72.Gallo RL, Granstein RD, Kang S, et al. Standard classification and pathophysiology of rosacea. J Am Acad Dermatol. 2018;78(1):148-55.

73. Evans DG, Baser ME, McGaughran J, Sharif S, Howard E, Moran A. Malignant peripheral nerve sheath tumours in neurofibromatosis 1. J Med Genet. 2002;39(5):311-4.

74. Montoliu L, Grønskov K, Wei AH, et al. Increasing the complexity: new genes and new types of albinism. Pigment Cell Melanoma Res. 2014;27(1):11-8.

75. Ständer S, Schmelz M. Chronic itch and pain—similarities and differences. Eur J Pain. 2006;10(5):473-8.

76. López-Jornet P, Camacho-Alonso F. Burning mouth syndrome: an update. Oral Dis. 2010;16(3):223-34.

77.Bornstein J, Goldstein AT, Stockdale CK, et al. 2015 ISSVD, ISSWSH and IPPS consensus terminology and classification of persistent vulvar pain and vulvodynia. J Low Genit Tract Dis. 2016;20(2):126-30.

78. Yosipovitch G, Samuel LS. Neuropathic and psychogenic itch. Dermatol Ther. 2008;21(1):32-41.

79.Gardner FH,DiamondI.LK.Autoerythrocyte sensitization:a form of purpura producing painful bruising following autosensitization.Blood.1955:10(2):675-90.

80.Yosipovitch G,Papoiu AD.Neuroimmune interaction in pruritus:the itch-pain connection.Semin Cutan Med Surg.2008;27(2):106-12.

81.Picardi A, Lega I, Abeni D, et al. Psychosocial predictors of suicidal ideation in dermatologic outpatients. Psychosomatics. 2006;47(4):331-7.

82.Mostaghimi L. Psychocutaneous medicine clinic: Wisconsin experience. J Acad Consult Liaison Psychiatry. 2021;62(5):522–527. doi:10.1016/j.jaclp.2021.04.008.

83.Jafferany M, Ferreira BR, Abdelmaksoud A, Mkhoyan R. Management of psychocutaneous disorders: A practical approach for dermatologists.Dermatol Ther. 2020;33(6):e13969. doi:10.1111/dth.13969.

84.Bewley A, Affleck A, Bundy C, Higgins E, McBride S. Psychodermatology services guidance: the report of the British Association of Dermatologists' Psychodermatology Working Party. Br J Dermatol. 2013;168(6):1149–1150. doi:10.1111/bjd.12330

85.Koo J, Lebwohl A. Psycho-cutaneous medicine. J Am Acad Dermatol. 2001;45(4):625–32.

86.Koblenzer CS. Psychotropic drugs in dermatology. Dermatol Clin. 1996;14(3):471–8.

87 .Ständer S, Weisshaar E, Mettang T, Szepietowski JC, Carstens E, Ikoma A, et al. Clinical classification of itch: A position paper of the International Forum for the Study of Itch. Acta Derm Venereol. 2007;87(4):291–4.

88.Dalgard FJ, Gieler U, Tomas-Aragones L, Lien L, Poot F, Jemec GBE, et al. The psychological burden of skin diseases: A cross-sectional multicenter study among dermatological out-patients in 13 European countries. J Invest Dermatol. 2015;135(4):984–91.

89.Mehta H, Narang T. Evaluation and approach to patients with psychodermatoses. Clin Dermatol Rev. 2023;7(4):310–318. doi:10.4103/cdr.cdr_3_22.

90.Arck PC, Slominski A, Theoharides TC, Peters EMJ, Paus R. Neuroimmunology of stress: Skin takes center stage. J Invest Dermatol. 2006;126(8):1697–704.

91.Yadav S, Narang T, Kumaran MS. Psychodermatology: a comprehensive review. Indian J Dermatol Venereol Leprol. 2013;79(2):176–192. doi:10.4103/0378-6323.107632.

92. Gupta MA, Gupta AK. Psychopharmacology in dermatology: five common disorders. Clin Dermatol. 2023;41(3):368-375. doi:10.1016/j.clindermatol.2023.01.006.

93. Shukla R, Sasseville D. Psychopharmacology in psychodermatology. J Cutan Med Surg. 2008 Nov-Dec;12(6):255–67. doi:10.2310/7750.2008.07062Shukla R, Sasseville D.

Psychopharmacology in psychodermatology. J Cutan Med Surg. 2008 Nov-Dec;12(6):255–67. doi:10.2310/7750.2008.07062.

94. Menon V, Selvakumar N, Faheem A. Selection of psychotropics in dermatologic practice. CosmoDerma. 2021;1:60. doi:10.25259/CSDM_60_2021.

95. Grant JE, Odlaug BL, Kim SW. N-Acetylcysteine, a glutamate modulator, in the treatment of trichotillomania: a double-blind, placebo-controlled study. Arch Gen Psychiatry. 2009;66(7):756-763. PMID: 19581567.

96. Lee DK, Lipner SR. The potential of N-acetylcysteine for treatment of trichotillomania, excoriation disorder, onychophagia, and onychotillomania: an updated literature review. Int J Environ Res Public Health. 2022;19(11):6370. doi:10.3390/ijerph19116370. PMID: 35681955.

97. Adil M, Amin SS, Mohtashim M. N-Acetylcysteine in psychodermatological disorders: pharmacology and clinical use in body-focused repetitive behaviors. Indian J Dermatol Venereol Leprol. 2019;84(6):652-659. PMID: 31444827.

98. Bewley A. Psychodermatology: Where dermatology meets psychiatry. Clin Med (Lond). 2010;10(1):58–60.

99. Bewley A, Taylor R, Reichenberg J, Magid M. Practical psychodermatology. J Eur Acad Dermatol Venereol. 2014;28(4):445–50.

100. Misery L. Psychosomatic dermatology. J Eur Acad Dermatol Venereol. 2005;19(6):629–33.