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The Connection Between Emotional Regulation and Skin Conditions

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Abstract: Psychological stress has long been associated with inflammatory skin conditions such as acne and eczema, yet the biological mechanisms underlying this relationship are not always clearly explained. This narrative review examines evidence suggesting that emotional regulation in the brain may influence skin health through dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and related neuroimmune pathways. Rather than broadly surveying psychodermatological research, this paper focuses on how stress processing in emotion-regulating brain regions can lead to altered hormonal signaling, immune activation, and impaired skin barrier function. By narrowing the scope to this pathway, the review highlights both the strengths and limitations of current evidence and identifies areas where further research is needed to better understand the brain–skin connection.

Key words: Emotional regulation, skin, inflammation, acne, eczema, stress.

1. Introduction

Stress-related flare-ups of acne and eczema are commonly reported by patients and frequently observed in clinical settings. Periods of sustained emotional strain often coincide with increased inflammation, delayed wound healing, and worsening disease severity. Although stress is widely recognized as a trigger for these conditions, explanations for how emotional experiences translate into dermatological symptoms are often broad and lack clear biological detail.

The skin is closely integrated with the nervous, endocrine, and immune systems, allowing psychological stress to influence cutaneous physiology. One of the most extensively studied mediators of this interaction is the hypothalamic–pituitary–adrenal (HPA) axis, which coordinates the body's hormonal response to stress. Dysregulation of this system has been linked to immune imbalance, altered inflammatory signaling, and impairment of the skin barrier **(Chen and Lyga, 2014; Alexopoulos & Chrousos, 2016)**. However, many reviews discuss stress and skin disease without clearly examining how emotional regulation in the brain contributes to HPA axis activation.

In this review, I focus on emotional regulation as an upstream factor that shapes stress physiology and inflammatory skin disease. By emphasizing a single, biologically plausible pathway—emotional regulation → HPA axis activation → skin immune and barrier responses—this paper aims to move beyond superficial synthesis and toward deeper critical analysis.

2. Methods: Literature Selection

This paper was conducted as a narrative literature review. Relevant studies were identified through searches of PubMed and Google Scholar. Search terms included combinations of *emotional regulation*, *psychological stress*, *HPA axis*, *cortisol*, *neuroimmune signaling*, *acne*, *eczema*, *atopic dermatitis*, and *psychodermatology*.

Studies were included if they examined at least two of the following:

1) emotional stress or emotional regulation,
2) neuroendocrine or immune stress responses, and
3) outcomes related to inflammatory skin conditions or skin barrier function.

Original research articles were prioritized when discussing mechanisms, while review articles were used for contextual background. Due to variability in study design, outcomes were synthesized qualitatively rather than through meta-analysis.

3. Emotional Regulation and Stress Processing in the Brain

Emotional regulation refers to the ability to modulate emotional responses through cognitive and neural control. This process depends on interactions among brain regions such as the prefrontal cortex, amygdala, and hypothalamus. Under chronic stress, regulation of emotional responses may become impaired, leading to prolonged physiological arousal and sustained activation of stress-response systems.

Research in psychodermatology suggests that emotional dysregulation is associated with heightened stress responses that extend beyond psychological experience **(Mento *et al.*, 2020)**. Although many studies do not directly measure emotional regulation at the neural level, consistent associations between negative emotional states—such as anxiety, depression, and anger—and worsening skin disease support a role for emotional processing in disease severity.

The hypothalamus plays a central role in translating emotional stress into endocrine output. Emotional stress signals processed by limbic and cortical regions converge on the hypothalamus, initiating activation of the HPA axis. This pathway provides a biologically plausible mechanism by which emotional dysregulation may influence skin inflammation and barrier function **(Chen & Lyga, 2014; Mar & Rivers, 2023)**.

4. HPA Axis Dysregulation as a Link to Skin Inflammation

Activation of the HPA axis is a normal and adaptive response to acute stress. Cortisol release helps regulate metabolism and immune activity. However, chronic stress can lead to prolonged cortisol exposure, which may disrupt immune balance and impair tissue repair **(Alexopoulos & Chrousos, 2016)**.

In the skin, cortisol influences keratinocyte differentiation, lipid synthesis, and barrier integrity. Experimental and clinical studies indicate that sustained stress weakens the epidermal barrier and increases susceptibility to irritation and inflammation **(Chen & Lyga, 2014 and Zhang, 2024)**. These effects are particularly relevant to eczema, which is characterized by chronic inflammation and barrier dysfunction.

Importantly, the skin also contains its own HPA-like system. Skin cells, including keratinocytes, are capable of producing corticotropin-releasing hormone, adrenocorticotropic hormone, and cortisol in response to stress **(Saric-Bosanac *et al.*, 2020)**. This local stress response amplifies inflammatory signaling within the skin and strengthens the connection between central emotional regulation and peripheral skin disease.

5. Neuroimmune Signaling and Cutaneous Immunity

Stress influences skin health not only through hormonal pathways but also through neuroimmune signaling. Psychological stress stimulates peripheral nerve endings to release neuropeptides such as substance P and calcitonin gene-related peptide (CGRP). These molecules interact with immune cells, including mast cells and Langerhans cells, promoting inflammatory responses in the skin **(Madva & Granstein, 2013)**.

Neuroimmune signaling plays a critical role in cutaneous immunity by regulating antigen presentation, immune cell activation, and inflammatory mediator release. Stress-induced alterations in these pathways may increase susceptibility to inflammatory skin conditions and exacerbate existing disease (Madva & Granstein, 2013; Rodriguez-Vallecillo & Woodbury-Fariña, 2014). These findings further support the idea that emotional regulation in the brain can influence skin inflammation through downstream immune mechanisms.

6. Evidence from Acne and Eczema Research

In acne, stress has been associated with increased sebum production and inflammatory signaling. Stress-related hormones and neuropeptides stimulate sebaceous gland activity and promote inflammatory responses within the pilosebaceous unit, contributing to lesion development and disease severity **(Panconesi & Hautmann, 2003; Chen & Lyga, 2014)**. These mechanisms help explain why acne frequently worsens during periods of emotional stress.

Eczema appears to be influenced by stress through somewhat different mechanisms. Chronic stress impairs skin barrier function, allowing allergens and irritants to penetrate more easily and trigger immune responses. Stress has also been shown to bias immune signaling toward pathways associated with allergic inflammation, contributing to itching, redness, and chronic disease flares **(Zhang, 2024; Alexopoulos & Chrousos, 2016)**.

Clinical studies consistently report correlations between emotional stress and eczema severity **(Rodriguez-Vallecillo & Woodbury-Fariña, 2014)**. However, many rely on self-reported stress measures, which introduces subjectivity and limits causal inference. Despite this limitation, the convergence of clinical, neuroendocrine, and immunological evidence supports a meaningful link between emotional regulation and disease expression.

7. Limitations of Current Research

Despite increasing interest in psychodermatology, several limitations remain in the current literature. Many studies rely on subjective measures of stress and emotional state, which vary widely across individuals. Neurobiological studies often involve small sample sizes, reducing generalizability.

Additionally, much of the evidence linking emotional regulation to skin disease is correlational. Experimental studies that directly manipulate emotional regulation or examine its neural correlates alongside dermatological outcomes are relatively rare **(Mar and Rivers, 2023)**. Addressing these gaps will be essential for establishing stronger causal relationships.

8. Implications for Treatment and Future Research

The evidence reviewed suggests that addressing emotional regulation may be beneficial in managing inflammatory skin conditions. Integrating psychological interventions—such as stress-management strategies or cognitive behavioral approaches—into dermatological care may help reduce symptom severity for some patients **(Koblenzer, 1988 and Alexopoulos & Chrousos, 2016)**.

Future research should prioritize longitudinal designs, objective stress measurements, and direct assessment of emotional regulation. Combining neuroimaging, immune biomarkers, and clinical outcomes may help clarify how emotional regulation influences skin disease and guide more targeted treatment approaches.

9. Conclusion

Emotional regulation within the brain appears to influence physiological stress responses that are relevant to inflammatory skin conditions such as acne and eczema. Dysregulation of emotional processing may contribute to sustained HPA axis activation, neuroimmune signaling, and impaired skin barrier function. While current evidence supports a strong association between stress and skin disease, limitations in study design prevent definitive conclusions.

By focusing on a single neuroendocrine pathway, this review highlights both the promise and the gaps in current psychodermatology research. A more rigorous examination of emotional regulation as a biological factor may improve understanding of stress-related skin disease and support more comprehensive, integrative approaches to treatment.

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Biography

Rick Liu is a student at Westlake High School in the Class of 2027. He wrote *The Connection Between Emotional Regulation and Skin Conditions* while participating in the IRIS Research Program, where he explored how emotional regulation in the brain may influence the development and severity of skin conditions such as acne and eczema. His interest in psychodermatology grew from personal experiences with long-term stress and inflammatory skin conditions, which motivated him to better understand how psychological and physiological factors interact in health and disease. Through this research, Rick became especially interested in whole-person approaches to medicine that integrate mental health with traditional medical care. He hopes this paper helps bring attention to psychodermatology as an emerging field and encourages further research into more holistic and integrative models of treatment.