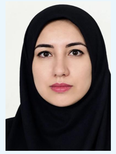
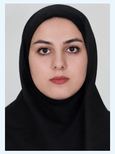
THE FIRST INTERNATIONAL CONGRESS ON HEALTH SERVICES TRANSFORMATION
اولین کنگره بین المللی تحول خدمات سلامت
۲۷ و ۲۸ مهر ماه
ISC 05251-58426 Scopus CIVILICA

Code: 1519

From Occupational Stress to Hair-Cycle Dysregulation: A Narrative Review of Stress-Associated Alopecia

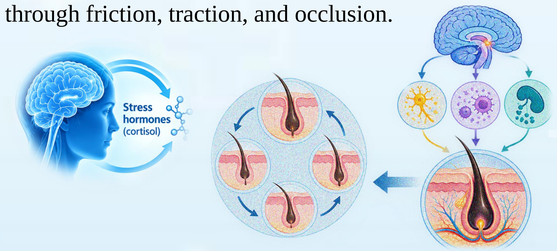
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Backgrounds

Occupational stress may contribute to hair-cycle dysregulation and stress-associated alopecia through neuroendocrine, sympathetic, immune, and follicular pathways. Healthcare workers are exposed to multiple stressors, including high workload, prolonged working hours, shift work, sleep disruption, and inadequate recovery time. Stress may alter normal hair-follicle cycling and contribute particularly to telogen effluvium, while potentially aggravating alopecia areata, traction alopecia, and trichotillomania. Prolonged use of protective headgear may also add mechanical stress through friction, traction, and occlusion.



Methods

A narrative review searched PubMed/MEDLINE, Scopus, Web of Science, Embase, and Google Scholar for studies through June 2026. Key terms included occupational stress, alopecia, hair follicle, and healthcare workers. Evidence was narratively synthesized to assess links between occupational stress, hair-cycle dysregulation, and alopecia.

Findings



- Long working hours
- Heavy workload
- Shift work & lack of rest
- Workplace conflicts
- Prolonged protective headgear

The literature suggests a multifactorial relationship between occupational stress and hair-cycle dysregulation. HPA-axis activation, sympathetic signaling, immune alterations, and disruption of the follicular stem-cell niche may impair normal hair cycling. Sleep disruption, nutritional disturbances, prolonged working hours, inadequate recovery, and prolonged protective headgear use may further aggravate these effects. Collectively, these biological, behavioral, and occupational factors may contribute mainly to telogen effluvium and potentially influence alopecia areata, traction alopecia, and trichotillomania.

Occupational stress → **HPA axis, sympathetic activation** → **Immune & follicular stem-cell changes** → **Sleep, nutrition & mechanical stressors** → **Hair-cycle dysregulation** → **Clinical outcomes**

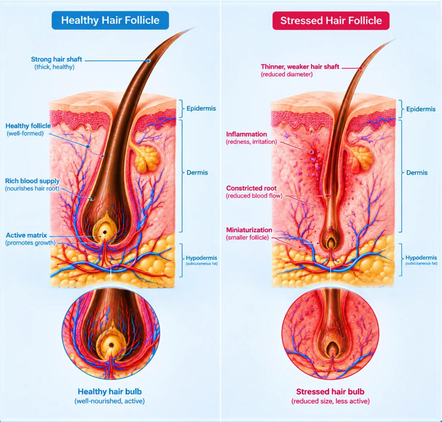
Telogen effluvium | **Alopecia areata** | **Traction alopecia** | **Trichotillomania**



Biological
HPA-axis and sympathetic signaling; immune and stem-cell effects.

Occupational
Workload, long hours, shifts, sleep disruption and headgear.

Clinical
Excessive shedding is especially relevant to telogen effluvium.



Conclusion

Occupational stress–associated hair loss is multifactorial, involving biological, environmental, behavioral, and personal factors. Stress may affect hair-follicle cycling through neuroendocrine, sympathetic, immune, and follicular pathways, while workload, long working hours, shift work, sleep disruption, and protective headgear may contribute. Clinical assessment should consider dermatologic causes, occupational stressors, sleep, nutrition, and mechanical or behavioral exposures. However, direct evidence linking occupational stress to alopecia remains limited. Future longitudinal studies using standardized stress measures and objective biomarkers are needed to clarify these associations and improve prevention and management.

References

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