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Why does classic scabies spare the adult scalp? A site-specific barrier hypothesis

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Dear Editor,

The relative sparing of the scalp and face in immunocompetent adults with classic scabies is clinically familiar, whereas these sites may be involved in infants, older adults, immunocompromised patients, and crusted scabies.¹ This distribution is commonly used as a diagnostic clue, but its biological basis remains incompletely explained. We propose that the adult scalp is relatively spared not because of a single protective organism or molecule, but because it represents a mature, hair-bearing, sebaceous, frequently groomed, and immunologically competent cutaneous niche that may be less permissive to *Sarcoptes scabiei* burrowing and persistence. The mite lives and lays eggs within the stratum corneum; therefore, local differences in stratum corneum architecture, hydration, lipid composition, temperature, friction, and accessibility may contribute to regional tropism. The proposed contributions of barrier properties, sebum, grooming, and local immunity to adult scalp sparing remain hypothetical and require direct investigation.

This hypothesis may also explain why scalp involvement is more plausible at the extremes of age or in impaired host defense. Neonatal and infant skin is not simply a miniature version of adult skin: it has distinct structural and functional properties, including ongoing maturation of barrier function, surface pH, hydration, permeability, and microbial ecology.² These developmental differences may contribute to infant scalp susceptibility, although their specific role in scabies distribution remains unproven. In older or immunocompromised patients, impaired host defense, reduced scratching or grooming, and diagnostic delay may contribute to cephalic involvement, although their specific roles remain uncertain. Increased mite burden should not be assumed in all such patients, particularly in the absence of crusted scabies.¹ The sebaceous microbiome may represent one component of this model, although it should not be interpreted as a sufficient explanation on its own. A possible association between cutaneous microbiome differences, including *Malassezia*, and facial involvement in scabies was previously suggested by Seok *et al.*, although no direct supporting evidence was provided.³ *Malassezia* species dominate sebaceous sites such as the scalp and face and can influence lipid metabolism, barrier signaling, and local immune responses.⁴ However, *Malassezia* is also present in early life and in seborrheic dermatitis; therefore, it is unlikely to represent an isolated explanation for adult scalp sparing. A microbiome-centered view of scabies is biologically plausible. Experimental work in a porcine model has demonstrated infestation-associated changes in the bacterial skin microbiome and opportunistic bacterial expansion.⁵ These findings do not establish a protective role of the human scalp microbiome. A

direct protective or acarostatic effect of *Malassezia* or the scalp microbiome against *S. scabiei* has not been demonstrated. The proposed contribution of this model is to integrate previously discussed anatomical and sebaceous explanations with behavioral, immunological, and microbial factors into a testable framework. The resulting hypothesis concerns relative scalp sparing rather than established or absolute resistance. Future studies should compare involved adult body sites, spared adult scalp, involved infant scalp, and scalp-involved scabies in older or immunocompromised patients using mite-density assessment, barrier measurements, sebum profiling, bacterial and fungal microbiome sequencing, and lipid metabolomics. We hypothesize that anatomy, sebum, immunity, grooming, and the microbiome may jointly influence mite persistence on the adult scalp; the contribution of each factor and their interactions require direct experimental testing.

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