



Microbiome, mechanics, and morphology: Rethinking the etiopathogenesis of pilonidal sinus disease

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Dear Editor,

We read with great interest the article by Maak et al. (1) titled “Intergluteal fold depth has no influence on pilonidal sinus disease development”. This research investigates the long-standing relationship between intergluteal fold (IGF) depth and the onset of primary pilonidal sinus disease (PSD) and determined that IGF depth did not serve as an independent risk factor for PSD. This result deserves additional scrutiny as it directly refutes established surgical assumptions and guideline conclusions.

Akinci et al. (2) initially emphasized the importance of natal cleft morphology in the development of PSD, stating that individuals with PSD exhibit deeper natal clefts, thus justifying the use of cleft lift treatments such as the Karydak and Bascom techniques. These surgical techniques, which were supported by guidelines including the German Society of Coloproctology and the European Society of Coloproctology, aim to reduce recurrence by flattening the natal cleft (3,4). This practice is assumed to relieve the mechanical forces that trap the hairs. However, the results of Maak et al. (1) urge a reevaluation of this paradigm. Maak et al. (1) utilised a standardised, minimally compressive measurement technique. The authors found that the greatest IGF depth occurred distally in the anus, a region rarely affected by PSD. However, the disease is most often seen in the cranial third, where the IGF depth is least. This discrepancy suggests that IGF depth alone may not be sufficient to explain the onset of PSD. This result is consistent with more recent studies emphasizing the role of hair shaft biomechanics rather than static anatomic measurements.

Bosche et al. (5) have shown that sharp, rootless occipital hairs can embed into the skin under friction forces independent of IGF depth. Furthermore, 3D modelling studies now suggest that soft tissue architecture and motion vectors, rather than depth, may create environments conducive to hair emplacement (6). Pilonidal sinus tracts have shown distinct microbial signatures compared to adjacent healthy skin, with a predominance of anaerobic and biofilm-forming bacteria such as *Prevotella* and *Finnegoldia* species (7). Recent studies using 16S rRNA sequencing suggest that microbiome dysregulation may contribute to the pathogenesis and chronicity of PSD (8). These findings suggest that the local microbiota may play a role in maintaining chronic inflammation, impeding wound healing, and possibly initiating sinus formation. Targeted antimicrobial therapies, biofilm-disrupting agents, and microbiome-based interventions may complement surgical management, particularly in recurrent or refractory cases. Incorporating microbiome profiling into future PSD studies may thus unlock new diagnostic and therapeutic strategies.

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Effective management of PSD increasingly demands a shift from a purely morphological focus to a multifactorial, precision-based approach. Strategies should integrate patient-specific biomechanical variables such as hair orientation and anchoring, IGF microclimate, and the effects of posture and gluteal compression during prolonged sitting.

In conclusion, multicenter studies with a patient-centered approach integrating biomechanical modeling, microbiome profiling, and hair flow dynamics are needed to provide a deeper and more holistic understanding of PSD pathogenesis and recurrence. Future studies with such integrated approaches may form the basis for PSD guideline updates.

Footnotes

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