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科目：英文科普文章測驗

共 1 頁

請用中文寫出下面整篇文章的摘要（專有名詞可用英文）

Across skin regions, the density and variety of glands and hair follicles vary considerably, creating a complex physical and chemical landscape of geographically distinct niches for bacterial growth. For example, *Cutibacterium* (formerly *Propionibacterium*) and *Staphylococcus* species dominate sebaceous areas (such as the face and torso), while *Corynebacterium*, *Staphylococcus*, and beta-Proteobacteria are found in moist areas (such as the armpits and the elbow and knee creases).

In broad terms, the chemistry of a skin niche drives its microbiome composition, but unknown microbial and host factors contribute to important species- and strain-level differences in composition. For some species, such as *Cutibacterium acnes*, the same strain tends to colonize multiple body sites of the same individual; others, such as *Staphylococcus epidermidis*, differ among body sites of an individual (but tend to be similar in, for example, the axillae of different individuals). Most metagenomic cataloging of the human microbiome has focused on species composition. However, recent work demonstrates that, even within the same species, different strains can differ markedly in their effects on the host. Strain-level differences have been largely unexplored and remain a frontier for studies of the skin microbiota.

The process of skin microbiota assembly begins during birth and proceeds primarily according to body site over several weeks. The microbiota shifts notably during puberty, with increased predominance of *Corynebacterium* and *Cutibacterium* (formerly *Propionibacterium*) and decreased abundance of Firmicutes (including *Staphylococcus* and *Streptococcus* species). In adulthood, despite the skin's continuous exposure to the environment, the microbial composition remains surprisingly stable over time. This suggests that stabilizing, mutually beneficial interactions exist among commensal microbes and between microbes and the host.

The composition of the skin microbiome can shift markedly during inflammation. It is not yet understood how pathogens and skin inflammation contribute to a vicious cycle, how homeostasis is re-established, or how pathogens interact with the existing commensal population. The critical role of context to the outcome of a microbe–host interaction animates this review. For example, pathogens such as *Staphylococcus aureus* often colonize the skin asymptotically, whereas mutualists such as *S. epidermidis* can at times promote disease.