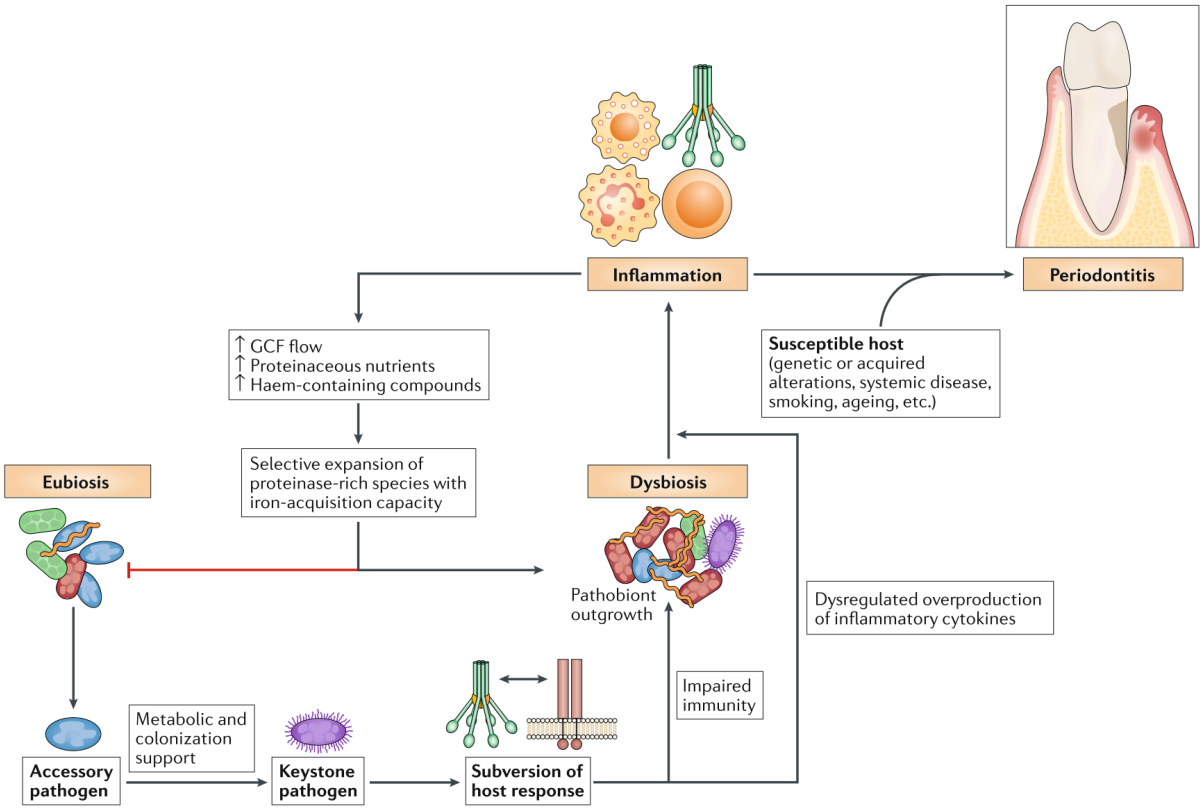
口腔微生物群：动态群落和宿主的相互作用

The oral microbiota: dynamic communities and host interactions【5】



微生物群生态失调和炎症之间促进双方之间的相互作用驱动慢性牙周炎

关键病原体（例如 *Porphyromonas gingivalis*）在辅助病原体（例如 *Streptococcus gordonii*）的帮助下发生定植，导致宿主固有防御系统受损和炎症发展（例如，通过破坏中性粒细胞和其他骨髓细胞中的补体—Toll 样受体(TLR) 串扰(crosstalk) ）[1-4]这些改变促进了微生物生态失调的发生（牙周微生物群在数量和组成上发生改变）。炎症组织遭到破坏后，龈沟液（GCF）的流量增加，导致降解的胶原蛋白和含有血红素的化合物带入牙龈缝隙。具有铁获取能力的蛋白水解 (proteolytic) 细菌和酵解糖 (asaccharolytic) 细菌（病原菌）能够选择性地利用这些化合物分子。相比之下，与健康相关的 (eubiotic) 物种无法利用新的环境条件从而在与病原菌的竞争中失败。这种不平衡会导致微生物群的生态失调，进一步加剧炎症，最终导致易感个体的牙周炎。炎症和微生物群的生态失调在自我持续的正反馈循环中促进双方之间的相互作用可能导致牙周炎的慢性发展。

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7. Maekawa, T. et al. Porphyromonas gingivalis manipulates complement and TLR signaling to uncouple bacterial clearance from inflammation and promote dysbiosis. Cell Host Microbe 15, 768–778 (2014). This study shows

that a keystone periodontal pathogen manipulates complement–TLR crosstalk to block bactericidal mechanisms while fostering a nutritionally favourable inflammatory response; this uncoupling of immune bacterial clearance from inflammation promotes dysbiosis and periodontitis.

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