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Microbiota control of skin's humoral immunity

The mucocutaneous barrier is home to microbiota composed of trillions of microorganisms. In the gut it is carefully regulated by antibodies. Whether skin commensals engage with humoral responses remains poorly understood. We show that colonization with common human skin commensal *Staphylococcus epidermidis* profoundly modulates cutaneous immunity that results in serum responsiveness with antigen-specific IgG antibodies. IgG2b antibodies are also found on the skin to bind colonizing commensals. Such response is uncoupled from inflammatory signals, occurs in a tissue autonomous manner and is associated with the induction of dermal tertiary lymphoid organs that closely resemble classical germinal centers. Our data also uncover a non-redundant role for the largest antigen presenting cell network composed of Langerhan's cells. While these cells have been associated with both tolerizing and proinflammatory functions, in the context of non-inflammatory skin colonization Langerhan's cells promote tissue intrinsic humoral response. Our work proposes a model by which commensal specific antigens are collected in the hair follicle leading to the generation of T follicular helper cells that further expand the local B cell pool allowing for development of antibody secreting plasma cells. Commensal induced antibodies can protect the host against subsequent infection with the same microbe demonstrating the importance of this line of immune defense in maintaining barrier protection. Collectively, our observations highlight the autonomous potential of skin organizational flexibility and demonstrate that B cells represent an indispensable local cutaneous compartment that actively maintains skin immunity.