Join us for an informal science talk

Monday, March 30th at 4:00 pm
Jacobs School of Medicine and Biomedical Sciences
955 Main St., Room 2220A

“A novel model for psoriasis within an evolutionary medicine framework”

One of the most noticeably unique human traits is our skin: it is thicker, oilier, and less hairy than that of our closest primate relatives. We hypothesize that human-specific use of fire, clothing, agriculture, and other technological buffers have reflexively impacted skin evolution, leading to further expression of these traits. From this, human skin microbiome composition and epidemiology differ most greatly within recent primate evolutionary history. Because we share more than 99% of our genomic sequences with our primate relatives, several studies have shown that the majority of traits underlying human uniqueness have evolved through regulatory rewiring of gene expression, rather than changes in protein structure and function.

Within this evolutionary context, we found that genes showing significant variation in expression in the human skin as compared to non-human great apes are also involved in psoriasis, a genetically and environmentally-linked immune-mediated skin disorder. Our results address a fundamental question in epidemiology: why do genetic variants that confer susceptibility to immune-mediated diseases remain in the population? This study identifies dozens of genes that are associated with skin barrier function as novel targets for downstream analysis, both for investigating functional mechanisms leading to psoriatic phenotypes, as well as understanding primate skin immunity evolution. Collectively, our results build on an evolutionary medicine paradigm, where recent human evolution leads to susceptibility to disease within the context of immune-mediated skin disorders.

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