**Genetics, Microbiota and Skin inflammatory diseases**

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Atopic dermatitis (AD) is a complex chronic inflammatory skin disorder that results from intimate interactions among genetic predisposition, host environment, skin barrier defects, and immunological factors. However, a clear genetic roadmap leading to atopic dermatitis remains to be fully explored. From a genome-wide mutagenesis screen, we have reported that deficiency of ZDHHC13, a palmitoylacyl transferase, is associated with skin and multitissue inflammatory phenotypes. Further genetic and immunological studies revealed that this dermatitis is a keratinocyte-cell autonomous disorder. Quantitative proteomics study revealed that multiple pathways are regulated by ZDHHC13, including filaggrin degradation and hydrolysis pathways. On the other hand, we study how skin microbiota cross-talk with the defective barrier, and will identify dysbiosis and microbiota functional composition responsible for skin inflammation. Seeking for the underlying mechanism of dysbiosis-associated skin inflammation and development of innovative therapeutics to treat human atopic dermatitis are our final goals.