

The role of very long chain fatty acid containing ceramides in the epidermal barrier and the underlying molecular mechanisms

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In multicellular organisms, the surface barrier is essential for maintaining the internal environment. In mammals, the barrier is the stratum corneum. Fatty acid transport protein 4 (FATP4) is a key factor involved in forming the stratum corneum barrier. Mice lacking Fatp4 display early neonatal lethality with features such as tight, thick, and shiny skin, and a defective skin barrier. FATP4 is a member of the FATP family that possesses acyl-CoA synthetase activity for very long chain fatty acids. How Fatp4 contributes to skin barrier function, however, remains to be elucidated. In the previous study, we characterized two *Caenorhabditis elegans* genes, *acs-20* and *acs-22*, that are homologous to mammalian FATPs. Animals with mutant *acs-20* exhibited defects in the cuticle barrier, which normally prevents the penetration of small molecules. We demonstrated that the incorporation of exogenous very long chain fatty acids into sphingomyelin was reduced in *acs-20* and *acs-22* mutants. In the present study, we further analyzed the skin barrier of *acs-20* and *acs-22* mutants. We also developed *C. elegans* infection models against human skin flora such as *Propionibacterium* and *Malassezia*.