Synopsis of Original Research Paper

Altered water barrier function in epidermal-type fatty acid binding protein deficient mice.

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We have generated mutant mice for epidermal type fatty acid binding protein (E-FABP) by the gene targeting technique and examined in detail the phenotype. In spite of lack in the expression of E-FABP mRNA and its protein in the skin and other tissues of the mutant mice, the animals appeared normal in gross and histological examination. Northern blot analysis of other FABPs revealed a distinct elevated gene expression of heart-type FABP (H-FABP) in the skin of the homozygous mice. In analyses of the skin, differences were not observed in contents of major fatty acids, electron microscopic appearances as well as inflammatory responses in ear skin between the mutant and wild type mice. Basal transepidermal water loss (TEWL) of homozygous mice was lower as compared with that of the wild mice. When acetone was applied to the skin for disruption of the water permeability barrier, the recovery in TEWL was delayed, despite maximum TEWL upon acetone treatment was similar between the homozygous and wild type mice in terms of the size and time course. The molecular mechanism by which E-FABP contributes to the water barrier function of the skin remains to be elucidated.