A warm sensor maintains skin barrier

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Japanese research group led by Prof. Makoto Tominaga and Dr. Takaaki Sokabe (National Institute for Physiological Sciences: NIPS) found that TRPV4 ion channel in skin keratinocytes is important for formation and maintenance of barrier function to prevent dehydration. Their finding was reported in the *Journal of Biological Chemistry.*

TRPV4 is one of the temperature-sensitive Ca2+-permeable channels, namely "thermoTRPs". It is expressed in skin, acting as a warm sensor (>27oC) to choose preferred environmental temperatures in mammals. The research group sought the alternative function of TRPV4, since skin keratinocytes express another thermoTRP named TRPV3, which also functions as a warm sensor.

TRPV4 was found to interact with b-catenin, an adaptor protein between actin filaments and E-cadherin in cell-cell junction complex. When TRPV4 was genetically removed from keratinocytes, Ca2+-induced cell-cell junction formation was delayed and immature, resulting in leaky junctions. Consistently, intercellular junction-dependent skin barrier in TRPV4-deficient mice became weak (leaky intercellular pathway) compared to wild-type mice. Interestingly, these phenotypes were TRPV4-specific, but not TRPV3-dependent.

Dr. Sokabe said, "TRPV4 may utilize skin temperature to provide Ca2+ for cell-cell junction complexes to reinforce their tightness. For instance, dried skin in cold seasons or regions could be due to low activity of TRPV4 caused by low skin temperature. Development of chemicals modulating TRPV4 activity would be useful for barrier repair of

damaged skin."

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