Synopsis of Original Research Paper

Keratinocytes homeostasis by contact inhibition signaling and its disorders

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Mice deficient in one or both copies of *Mob1a* and carrying a trapped mutation in *Mob1b* developed various cancers. Most frequent cancers in these mice resembled trichilemmal carcinomas. Keratinocyte-specific homozygous null mutations of *Mob1a* and *Mob1b* (kDKO) mice showed hyperplasia of keratinocyte progenitors. kDKO keratinocytes exhibited hyperproliferation, apoptotic resistance, impaired contact inhibition, enhanced progenitor self-renewal, and increased centrosomes. In addition, loss of *Mob1a/b*, or activation of downstream *YAP1* were replicated in human trichilemmal carcinomas.