Energy deprivation-induced AMPK activation inhibits milk synthesis via PrlR and PGC-1α

Zhihui Wu
Qihui Li
Siwang Yang
Tenghui Zheng
Jiayuan Shao
Wutai Guan
Fang Chen
Shihai Zhang

Video Byte

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Abstract

In lactating mammals, the mammary glands are responsible for milk production and secretion. Low energy levels in mammary gland cells are known to reduce milk synthesis by activating AMPK, an energy-sensing protein. However, the exact mechanism by which energy status affects lactation efficiency isn't clear. To learn more, researchers recently examined AMPK's role in milk synthesis in mouse mammary epithelial cells. Energy (glucose) deficiency indeed activated AMPK, which reduced milk fat and protein synthesis in the cells. AMPK inhibited milk production partly by inhibiting the classic mTORC1 signaling pathway, but it primarily reduced milk protein synthesis by blocking the signaling pathway of the lactation hormone prolactin. Specifically, AMPK triggered degradation of the prolactin receptor (PrlR) in lysosomes to halt prolactin signaling. In addition, AMPK decreased milk fat production by inhibiting the synthesis of new fatty acids and by promoting the breakdown of existing fatty acids via effects on the protein PGC-1α. Although the findings need to be verified, this study identifies PrlR and PGC-1α as potential targets for milk synthesis regulation and clarifies how cellular energy supply might affect a mother's milk supply.