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[관심분야]

Mouse molecular genetics, lung/breast cancer, aging, cancer drug development, Telomerase biology, mouse models for human diseases.

[논문]

1. Y. Song, ···, H. -W. Lee. Dysfunctional Adipocytes Promote Tumor Progression Through YAP/TAZ-dependent Cancer Associated Adipocyte Transformation. Nature Communications in press, 2024.
2. Na, H., Y. Song, H. -W. Lee. Emphasis on adipocyte transformation: anti-Inflammatory agents to prevent the development of cancer-associated adipocytes. Cancers 15(2): 502, Jan. 2023.
3. Jin, Young, ·····, H. -W. Lee. Depletion of adipocyte Becn1 leads to lipodystrophy and metabolic dysregulation. Diabetes 70(1): 182-195, Jan. 2021.
4. Kim, H. J., S. E. Lee, H. Na, J. -S. Roe, J. Roh, H. -W. Lee. Divergence of the PIERCE1 expression between mice and humans as a p53 target gene. PLoS One 15(8):e0236881, Aug 2020.
5. Roh, J., ·····., H. -W. Lee. Impaired AKT and tumorigenesis by PIERCE1 ablation in KRAS mutant non-small cell lung cancer. Oncogene 39(36):5876-5887, Jul 2020.

Dysfunctional adipocytes promote tumor progression through YAP/TAZ-dependent cancer-associated adipocyte transformation

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Obesity has emerged as a prominent risk factor for the development of malignant tumors. However, the existing literature on the role of adipocytes in the tumor microenvironment (TME) to elucidate the correlation between obesity and cancer remains insufficient. Here, we aimed to investigate the formation of cancer-associated adipocytes (CAAs) and their contribution to tumor growth using mouse models harboring dysfunctional adipocytes. Specifically, we employed adipocyte-specific BECN1 KO (BaKO) mice, which exhibit lipodystrophy due to dysfunctional adipocytes. Our results revealed active YAP/TAZ signaling in both CAAs and BECN1-deficient adipocytes, which induced adipocyte dedifferentiation to form a malignant TME for breast and colon cancer progression. Additional deletion of YAP/TAZ from BaKO mice significantly restored the lipodystrophy and inflammatory phenotypes, leading to tumor regression. Furthermore, mice fed a high-fat diet (HFD) exhibited decreased BECN1 and increased YAP/TAZ expression in their adipose tissues. Treatment with the YAP/TAZ inhibitor, verteporfin, suppressed tumor progression in BaKO and HFD-fed mice, highlighting its efficacy against mice with metabolic dysregulation. Overall, our findings provide insights into the key mediators of CAA and their significance in developing a TME, thereby suggesting a viable approach targeting adipocyte homeostasis to suppress cancer growth.