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Circulating adipose fatty acid binding protein is a new link underlying obesity-associated breast/mammary tumor development

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It is clear that obesity increases the risk of many types of cancer, including breast cancer. However, the underlying molecular mechanisms by which obesity is linked to cancer risk remain to be defined. Herein, we report that circulating adipose fatty acid binding protein (A-FABP) promotes obesity-associated breast cancer development. Using clinical samples we demonstrated that circulating A-FABP levels were significantly increased in obese patients with breast cancer in comparison to those without breast cancer. Circulating A-FABP released by adipose tissue directly targeted mammary tumor cells, enhancing tumor stemness and aggressiveness through activation of the IL-6/STAT3/ALDH1 pathway. Importantly, genetic deletion of A-FABP successfully reduced tumor ALHD1 activation and obesity-associated mammary tumor growth and development in different mouse models. Collectively, these data suggest circulating A-FABP as a new link between obesity and breast cancer risk thereby providing A-FABP as a new biomarker and potential therapeutic target for treatment of obesity-associated cancers.

Biography

Bing Li's laboratory is to understand the role of fatty acid binding proteins (FABPs) in chronic inflammation, obesity, and cancer development. FABPs constitute a family of small, highly homologous intracellular lipid chaperones that have been recognized as central regulators of both metabolic and inflammatory pathways. We have shown that adipose FABP (A-FABP) and epidermal FABP (E-FABP) play important roles in many animal disease models. However, the exact mechanisms underlying these observations remain to be determined. Currently, research in my laboratory strives to understand how FABPs, including A-FABP and E-FABP, regulate cellular metabolism and intracellular signal transduction pathways in leukocytes, to determine the mechanisms by which FABPs link metabolism and complex diseases (obesity, skin inflammation, and breast cancer development), and to identify specific small molecular regulators in modification of FABP activities for potential clinical applications.

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