AKT2 deficiency impairs formation of the BCR signalosome

Zuochen Du
Di Yang
Yongjie Zhang
Xingtian Xuan
Han Li
Leling Hu
Changshun Ruan
Liling Li
Anwei Chen
Liang Deng
Yan Chen
Jingwen Xie
Lisa S. Westerberg
Lu Huang
Chaohong Liu

Video Byte

Keywords: AKT, AKT2, B cell, kinase, insulin, cancer, mouse model, knockout, B cell receptor, BCR, signalosome, cell signaling, Cell Communication and Signaling, CD19, actin, antibody, humoral, immune response

Posted Date: June 23rd, 2020

DOI: <https://doi.org/10.21203/rs.3.rs-37678/v1>

License: This work is licensed under a Creative Commons Attribution 4.0 International License.

[Read Full License](#)

Abstract

Some proteins are central to many cell signaling processes. One of these key molecules is AKT2. An important kinase involved in cell survival, growth, and metabolism, it has ties to insulin-induced signaling and cancer. AKT2 has a critical role in immune cells such as neutrophils and macrophages; however, although AKT2 is expressed in antibody-producing immune cells called B cells, its function in B cells isn't clear. In a recent study, researchers sought to understand the role of AKT2 in B cells using AKT2-deficient mice. They found that mice lacking AKT2 had impaired B-cell differentiation. B cells from these mice were not able to form a cluster of molecules called a signalosome in response to B-cell receptor (BCR) signaling, resulting in poor BCR signaling and impaired B cell activation and spreading. These results suggest that as a central orchestrator of signaling, AKT2 function is critical for proper BCR signaling and B cell development, ensuring a functional antibody-mediated immune response.