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PKB/Akt-dependent regulation of inflammation in cancer

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Abstract

Chronic inflammation is a major cause of human cancer. Clinical cancer therapies against inflammatory risk factors are strategically determined. To rationally guide a novel drug development, an improved mechanistic understanding on the pathological connection between inflammation and carcinogenesis is essential. PI3K-PKB signaling axis has been extensively studied and shown to be one of the key oncogenic drivers in most types of cancer. Pharmacological inhibition of the components along this signaling axis is of great interest for developing novel therapies. Interestingly,