Silibinina como antiproliferativo e quimiopreventivo no câncer de próstata

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Sylibum marianum – Milk thistle

A cancer chemopreventive agent silibinin, targets mitogenic and survival signaling in prostate cancer.


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Abstract

There are many epigenetic variables that affect the biological responses of autocrine, paracrine and endocrine regulatory molecules, which determine the growth and development of different cancers including prostate cancer (PCA). One of the focuses of the current cancer chemoprevention studies is the search for non-toxic chemopreventive agents that inhibit mitogenic and cell survival signaling in cancer cells. In general, advanced stage cancer cells harbor many constitutively active mitogenic signaling and anti-apoptotic mechanisms, which make them less dependent on external growth factors as well as resistant to chemotherapeutic agents. In this regard, silibinin (a naturally occurring flavanone) has shown the pleiotropic anticancer effects in different cancer cells. Our extensive studies with PCA have shown that inhibition of mitogenic and cell survival signaling, such as epidermal growth factor receptor, insulin-like growth factor receptor type I and nuclear factor kappa B signaling are the most likely molecular targets of silibinin’s efficacy in PCA. We have observed that silibinin inhibits prostate tumor growth in animal models without any apparent signs of toxicity. At the same time, silibinin is also physiologically available in different organs of the body including plasma and prostate, which is generally required for the pharmacological dosing and translational mechanistic studies of the compound. There are substantial amount of data to support the inhibitory effect of silibinin on mitogenic and cell survival signaling in PCA, which are reviewed in the present communication.

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