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Silymarin induces cyclin D1 proteasomal degradation via its phosphorylation of threonine-286 in human colorectal cancer cells

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Silymarin from milk thistle (*Silybum marianum*) plant has been reported to show anti-cancer, anti-inflammatory, antioxidant and hapatoprotective effects. For anti-cancer activity, silymarin is known to regulate cell cycle progression through cyclin D1 downregulation. However, mechanism of silymarin-mediated cyclin D1 downregulation still remains unanswered. The current study was performed to elucidate the molecular mechanism of cyclin D1 downregulation by silymarin in human colorectal cancer cells. The treatment of silymarin suppressed the cell proliferation in HCT116 and SW480 cells and decreased cellular accumulation of exogenously-induced cyclin D1 protein. However, silymarin did not change the level of cyclin D1 mRNA. Inhibition of proteasomal degradation by MG132 attenuated silymarin-mediated cyclin D1 downregulation and the half-life of cyclin D1 was decreased in the cells treated with silymarin. In addition, silymarin increased phosphorylation of cyclin D1 at threonine-286 and a point mutation of threonine-286 to alanine ameliorated silymarin-mediated cyclin D1 downregulation. Inhibition of NF-κB by a selective inhibitor, BAY 11-7082 suppressed cyclin D1 downregulation by silymarin. From these results, we suggest that silymarin-mediated cyclin D1 downregulation may result from proteasomal degradation through its threonine-286 phosphorylation via NF-κB activation. The current study provides new mechanistic link between silymarin, cyclin D1 downregulation and cell growth in human colorectal cancer cells.

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