Gwang Hun Park et al., Altern Integ Med 2014, 3:3
http://dx.doi.org/10.4172/2327-5162.S1.008

OMICS Group Conferences
Accelerating Scientific Discovery

2nd International Conference and Exhibition on
Traditional & Alternative Medicine
August 25-26, 2014 DoubleTree by Hilton Beijing, China

Extracts from the branch of *Abeliophyllum distichum* nakai induces cyclin D1 proteasomal degradation through threonine-286 phosphorylation

Gwang Hun Park[1], Jae Ho Park[2], Hun Ji Eo[1], Hun Min Song[1], Jeong Rak Lee[3], Man Hyo Lee[3] and **Jin Boo Jeong[1]**
[1]Andong National University, Korea
[2]Jungwon University, Korea
[3]Gyeongbuk Institute for Bio-industry, Korea

Abeliophyllum distichum Nakai (*A distichum*) has been reported to exert the inhibitory effect on angiotensin converting enzyme and aldose reductase. Recently, our group found that branch extracts from ethyl acetate fraction from branch of *A. distichum* (EAFAD-B) induces apoptosis through ATF3 activation in human colorectal cancer cells. However, anti-cancer reagents exert their activity through the regulation of various molecular targets. Therefore, the elucidation of potential mechanisms of EAFAD-B for anti-cancer activity may be necessary. To elucidate the potential mechanism of EAFAD-B for anti-cancer activity, we evaluated the regulation of cyclin D1 in human colorectal cancer cells. EAFAD-B decreased cellular accumulation of exogenously-induced cyclin D1 protein. However, cyclin D1 mRNA was not changed by EAFAD-B. 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 EAFAD-B. In addition, EAFAD-B induced threonine-286 phosphorylation of cyclin D1 and EAFAD-B-mediated cyclin D1 proteasomal degradation was attenuated by a point mutation of threonine-286 to alanine. Inhibitions of both ERK1/2 by PD98059 and NF-κB by a selective inhibitor, BAY 11-7082 suppressed cyclin D1 downregulation by EAFAD-B.

Conclusion: From these results, we suggest that EAFAD-B-mediated cyclin D1 downregulation may result from proteasomal degradation through its threonine-286 phosphorylation via ERK1/2-dependent NF-κB activation. The current study provides new mechanistic link between EAFAD-B and anti-cancer activity in human colorectal cancer cells.

enter0230@hanmail.net