

An Introduction to Bioactivity of Fucoidan

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ARTICLE INFO

Received: 📅 January 25, 2019

Published: 📅 February 04, 2019

ABSTRACT

Citation: Merve Tutuncu, Zekiye Altun. An Introduction to Bioactivity of Fucoidan. Biomed J Sci & Tech Res 14(1)-2019. BJSTR. MS.ID.002480.

Fucoidan

Fucoidan is a polysaccharide, which is derived from brown algae and some marine invertebrates, consisting mainly of L-fucose and sulfate ester groups [1]. Fucoidan is particularly found in the cell wall of marine brown algae. This polysaccharide is named as fucoidin when it is derived from the first marine brown algae. Kylin gave Fucoidin in 1913. However, this name has been changed to fucoidan according to IUPAC rules [2]. Fucoidan related different studies have performed in the literature [1,2]. It has different bioactivities such as anticoagulant, anti-thrombotic, anti-inflammatory, antitumoral, immunomodulatory, anti-inflammatory, antioxidant, anti-hepatopathy, anti-uropathy, anti-inflammatory. These activities depend on the source of fucoidan samples taken from different species [1,2]. In addition, fucoidan is non-toxic or any adverse effects on the healthy tissues so that it can be used safely. There are many forms of fucoidan but the simplest molecular structure of fucoidan is obtained from *Fucus vesiculosus* consists mainly of 44.1% fucose, 26.3% sulphate and 31.1% ash and a small proportion of aminoglucose [3,4].

Bioactivity of Fucoidan

Anticancer effect of fucoidan on various cancer cells has been showed thus far. The researchers have showed that its' anticancer effect induced by inhibiting angiogenesis, metastasis and invasion and by cell accumulation in sub G0 / G1 phase [5-8]. In a study, examining about the effect of fucoidan on human metastatic PC-3 prostate cancer cells showed that fucoidan increased the levels of p21 / WAF1 / CIP1 in cells and suppressed the E2F transcription factor. In another research related to effect of Fucoidan on Burkitt's lymphoma cells have also been showed to suppress metalloproteinase-9 secretion and migration [9]. Fucoidan induces

apoptosis by extrinsic and intrinsic pathways in cancer cells. It is reported that the activation of caspase-3 due to the permeability of the mitochondrial membrane is reduced [6]. Besides, Fucoidan stimulates ER stress mechanisms by induces Toll-like receptor-4 regulated reactive oxygen species and promotes endoplasmic reticulum stress-mediated apoptosis in lung cancer cell lines and tumors [10]. Consequently, it would be suggested that fucoidan induces apoptosis in cancer cells, but its effect depends upon dose and time. One study has been reported that fucoidan has significantly increased drug efficacy because of synergistic effects with anticancer drugs. Furthermore, the immune response has been shown to increase the activity of T cells and reduce the effect of free oxygen radicals as a protective effect of fucoidan against side effects of chemotherapeutic drugs [9]. Fucoidan coated doxorubicin nanoparticles for the transmission of doxorubicin, shows a significant increase in the cell is also determined in the literature. According to this effect, it is thought to be an important agent in increasing the effectiveness of cancer drugs [11]. Masahide et al. indicated that fucoidan regulated the incidence of fatigue for the period of chemotherapy.

The study outcomes showed that chemotherapy with fucoidan group was endured for a longer period than chemotherapy without fucoidan group. Besides, Fucoidan inhibited toxicity of chemotherapeutic agents. As a result, the survival of patients with fucoidan treatment group was longer than that the patients without fucoidan [12]. Fucoidan is a safely agent both increasing the anti-tumoral effects of chemotherapeutics beside it has no adverse effects on healthy tissues. Fucoidan has the potential to be a high influence drug without damaging normal tissue and by reducing

the side effects of anticancer drugs. However, it is still needed to study understanding the mechanism of fucoidan.

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ISSN: 2574-1241

DOI: 10.26717.BJSTR.2019.14.002480

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