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Nutraceutical Potential of Vitexin: A Flavone Glycoside

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ABSTRACT

Vitexin is an apigenin flavone glycoside and it is extensively present in numerous edible and medicinal plants. It is considered as important as other flavonoids. It possessed a variety of biological properties, including anti-oxidation, anti-inflammation, anticancer, neuron-protective, cardio-protective, hepato-pancreatic protective effects. Other reported health relevant effects, includes fat reduction and glucose metabolism. The possible mechanism of protective effect through activation or inhibition of the different signaling pathways like AMPKa, Nrf-2, mTOR, PUMA, MMP, PARP, JNK, p38, Bcl-2/Bax ratio etc. The molecular targets of vitexin are very critical to utilize its potential effect as emerging chemo-preventive and chemotherapeutic agent.

Keywords: Vitexin, ROS, Antioxidant, Anti-inflammatory, Anti-microbial, Anti-cancer.

INTRODUCTION

Vitexin is an apigenin flavone glucoside (Apigenin-8-C-glucoside) has received great attention from scientists due to its diverse metabolic activities, like antioxidant, anti-microbial, anti-inflammatory, neuron-protective, anticancer and myocardial-protective effects [1-9]. Currently, it has been shown to exhibit several novel beneficial effects related to glucose homeostasis, fat metabolism, liver protective, antibacterial and antiviral [10-14]. Vitexins are present in various plants, including hawthorn [15], mung bean [16], beetroot [17], passion flowers [18, 19], otter flowers [20], bamboo [13], and gaillardia [21]. From ancient era, these plants have been utilized as food and medicine in Asia [22]. Still today it is widely utilized for the treatment of digestive related disorders, detoxification, dermal diseases, sun-stroke, inflammation and various other diseases and disorders [22]. Because of C-8 glucoside, vitexin causes a reduction of its bond dissociation enthalpy as compared to aglyconeapigenin, it has shown better free radical scavenger and antioxidant activity than apigenin, It has some derivatives too, such as isovitexin, rhamno-pyranosyl-vitexin, methyl-vitexin (isoembigenin), vitexin-2-O-rhamnoside, and vitexin-2-O-xyloside [17, 23]. The current literature, attempt has been made to discuss the nutraceutical potential of vitexin flavonoids.

Nature source of vitexin

Scientifically, Vitexin known as apigenin-8-C-*β*-D-glycopyranoside or 5, 7, 4-trihydroxyflavone-8-glucoside. They are found in many plants, including *Vaccinium bracteatum* Thunb (Sea bilberry) **[24]**, *Passiflora spp.* (Passion flower) **[25, 26]**, *Pennisetum millet* (Candle millet) **[27]**, *Vitex spp* (Chaste tree or Chaste berry) **[28]**, *Bambusa vulgaris* (Bamboo leaves) **[29]**, *Vignar adiata* (Mungbean) **[30]**, *Triticum aestivum* (Wheat leaves) **[31]**, *Mimosa pudica* (Mimosa) **[32]**, *Crataegus spp* (Hawthorn) **[33]** among others.

Delivery of vitexin

Vitexin can be consumed through edible plants, such as beetroot, or bamboo, mung beans and delivered to the target tissues at sufficiently high levels. Isolation of Vitexin can be from above mentioned plants as it is an integral part of that plants and transformed it into a bio-active constituent that can be incorporated into functional foods, supplements, or drugs. Molecular weight of Vitexin is about 432 Da, it's a small polar molecule. It can be delivered orally through foods, supplements, or pharmaceuticals in a number of ways. About solubility, it can be easily dissolved in aqueous-based foods, drinks and different pharma-based preparation. Again it could be converted into a powdered form that can then be incorporated into capsules or pills, tablets for supplement or pharmaceutical applications. The bioavailability and bioactivity of many plant based chemicals has been shown to be extended by controlling food matrix properties or by using colloidal delivery systems or [34-36].

Pharmacokinetics of vitexin

Vitexin is not easily absorbed in the upper gastrointestinal tract. It has very low oral bioavailability due to early removal from blood. It is largely resistant to molecular transformations [37-39].The metabolic transformation of this flavonoid glycoside occurs in the colon, where the micro-biota play principle roles in its degradation [40]. As the metabolic activity starts the Vitexin probably de-glycosylated initially and converted to 3-(4-hydroxyphenyl) propionic acid in the end. It is rapidly and widely distributed into various tissues but poor bioavailability. It is excreted mostly in the urine and bile [41, 42].

Biological effects of vitexin

Antioxidant effect

Oxidative stress causes excessive generation of reactive oxygen species (ROS), which causes damage to DNA, RNA and proteins of cells [43]. Chronic oxidative stress accelerates age-related dysfunctions, and play a major role in number of chronic disorders/diseases [9, 44, 45]. Vitexin has been reported as a potential antioxidant against free radicals [46]. It increases cell viability and reducing cellular injury by enhancing resistance against oxidative stress inducers. The capability to alleviate cellular reactive oxygen species (ROS) and malondialdehyde (MDA) levels has been revealed for vitexin [47-50]. This reduction in ROS and MDA by vitexin is attributed by increase of antioxidant enzyme activities, such as superoxide dismutase (SOD), heme-oxygenase, glutathione etc. More-over the up-regulation of antioxidant response proteins like nuclear factor erythroid 2-related factor 2 or nuclear factor erythroid-derived 2-like 2(NFE2L2) and 5'AMP-activated protein kinase/AMPK/5' adenosine monophosphate-activated protein kinase by vitexin play a major role against oxidative stress [47-50]. Out of above mentioned antioxidant proteins, nuclear factor erythroid 2 may regulate the expression of antioxidant proteins by which it protect against oxidative damage initiated by inflammation and cellular injury [51].

AMPK is an enzyme that plays a significant role in cellular energy homeostasis, largely to activate glucose and fatty acid uptake and oxidation. The main effect of AMPK activation is stimulation of liver and skeletal muscle fatty acid oxidation, keto-genesis, glucose uptake, inhibition of cholesterol bio-synthesis, triglyceride synthesis, adipocyte lipogenesis and modulation of insulin secretion by pancreatic B-cells [52]. In summary, vitexin can be considered as a potent antioxidant that prevent Reactive oxygen species induced degenerative damages such as hepatocytic injury, cardiac injury, neuro-degenerative disorder and insulin resistance both in vivo and in vitro [47-50].

Anti-inflammatory effect

The anti-inflammatory properties of vitexin has gained major interest in recent research studies [13, 53-60]. Vitexin has been found to ameliorate lipopolysaccharide (LPS) induced inflammatory reactions [13, 55-61]. Actually, vitexin regulate the release of number of important inflammatory cytokines like tumor necrosis factor-Alpha (TNF-a,) Interleukin 1-b (IL-1b), and Interleukin-6 (IL-6) and enzymes (iNOS, MMP-1, MMP-3, and MMP-13) [56, 62-65]. Immuno-stimulating cytokines or bacterial pathogens activate inducible nitric oxide synthase (iNOS) and produced high concentrations of Nitric Oxide (NO) through the activation of inducible nuclear factors, including *nuclear factor kappa B* (NF-κB). Essentially, all members of the matrix metallo-proteases (MMP) family have been linked to disease development, notably to tumour metastasis, chronic inflammatory reactions and the ensuing connective tissue damage as well as to nervous system disorders [66].

Immunoglobulin E (IgE) is important biomarker for allergic disorders [67]. Vitexin can inhibit the release of IgE into serum by inhibiting calcium release-activated calcium currents and b-hexosaminidase, which resulted in the amelioration of ovalbumin-induced allergic asthma [54]. In summary, vitexin can be considered to ameliorate organ injury, peritonitis, lung edema, inflammatory osteolysisetc [53, 56, 60, 68].

Anti-cancer effect

Vitexin has also been shown to be a potent natural active ingredient in the inhibition of tumour cell genesis and tumor growth [69]. It exerts anti-neoplastic effects on cancer in various tissues, including the hepatic, intestinal, breast, dermal, lung, transisnal epithelial tiisue (urinary) etc [69]. In recent years, there have been numerous studies mainly focusing on the inhibitory effects of vitexin on carcinogenesis by in-vitro route. There are different mechanism have been explored, it induced inhibition of cell proliferation by the prolongation of G2 cell-cycle arrest, cell apoptosis, the reduction of the Bcl-2/Bax ratio and the down-regulation of caspases [26, 70-75].

Cell susceptibility to apoptosis can be determined by Bcl-2/Bax ratio [76]. Bax/Bcl-2 ratio can affect tumor progression and aggressiveness through resistance of cancerous cells to apoptosis [76-78]. In other research it has been suggested that vitexin has MMP inhibitor property, which are important regulators of tumor proliferation and invasion [26] and up-regulate the poly (ADP-ribose) polymerase (PARP), an enzyme responsible for a various cellular processes such as damaged DNA repair, genomic stability and apoptosis (programmed cell death) [79].

In addition, vitexin up-regulated tumor suppressors: p53, PUMA (p53 up-regulated modulator of apoptosis) and several serine/threonine kinases [80, 81]. Regarding PUMA, it was identified as a transcriptional target of tumor suppressor (p53) and a potential apoptosis inducer in different neoplastic cells [82-84]. Again it is very interesting that PUMA found to be a critical mediator of p53-dependent and independent apoptosis induced by a wide variety of irritant like, genotoxic stress, deregulated tumour-oncogene expression, toxins, altered redox status, growth factor/cytokine withdrawal and infection [85].

In recent past, different active pharmaceuticals ingredient (API) targeting PUMA for cancer patients are emerging. These PUMA inducers target neoplastic cells, while PUMA inhibitors can be targeted to normal, healthy cells to reduce the unwanted effects of chemo and radiation therapy [85]. It has been also revealed that vitexin exerts it's anti-cancerous effects through inhibiting phosphatidyl-inositol-3-kinase (PI3K) [72, 75]. This pathway has been recently considered as an important mediator to cell proliferation, survival and oncogenesis [72, 75].

The mammalian target of rapamycin (mTOR, officialy known as the mechanistic target rapamycon) regulate various cellular functions. mTOR is basically a protein kinase encoded with MTOR gene. mTOR forms two different protein complex, known as mTORC-1 and mTORC -2. Out of these two forms, mTORC-1 generally regulates cell growth, cell mitotic activity, cell motility, protein synthesis, autophagy, and transcription [86, 87]. The activity of phosphoinositide 3-kinase (PI3K)/ mTOR pathway is dysregulated in

majority of neoplasm and has critical role in tumour development. Vitexin targeting the phosphoinositide 3-kinase (PI3K)/ mTOR signaling pathway so it would be an important therapeutic target agent in preventing carcinognesis. .

Neuron-protective effect

The pathogenesis of neuronal disorders, including brain infarction, hypoxic-ischemic injury, epilepsy seizure, retinal damage, depression, slowness of movement (bradykinesia) and amnesia are closely related with the necrosis of neurons, reduced stress resistance and inflammatory condition in nervous tissues [47-49, 89-96]. Vitexin reduced aforesaid neuron disorders and improved the learning behaviors in laboratory animals mainly by decreasing Reactive Oxygen Substance (ROS) levels, decreasing the release of pro-inflammatory factors and promoting neuron survival [47-49, 89-96].

It also increased the neuron-cell viability by the up-regulation of the Bcl-2/Bax ratio and the down-regulation of cysteine-aspartic proteases *i.e* caspases [47, 90, 91, 94]. Considering the protein oxidation, lipid peroxidation and loss of mitochondrial membrane are typical neuronal cells damage inducer. Vitexin acted by reducing these oxidative stress substance level and thereby preventing injury to nervous [48, 49, 97]. In summary, vitexin has been shown to improve the neurogenesis and memory by alleviating the neuro-degenerative disorder, which lends support to the benefits of vitexin on sleep disorders [92].

Cardio-protective effect

The pathogenesis of cardiovascular disorders, including infarction, atherosclerosis and myocardial cell necrosis are closely linked to inflammatory conditions, oxidative damage and endothelia cell injury [98-102]. Vitexin can protect the vascular endothelial cells from oxidized low density lipoproteins (ox-LDL) by enhancing the cell viability and physical stress resistance via the activation of AMPKa [50]. ox-LDL is the most damaging form of cholesterol that is generated when normal LDL cholesterol is modified by chemical interactions with free radicals particularly by myeloperoxidase (MPO) and reactive nitrogen species (RNS) [103,104].

The protective effect of vitexin in endothelia cells may also be attributed to the increased cell autophagy and up-regulation of relative genes, such as Beclin1 and light chain 3 II (LC3- II), as well as the down-regulation of p62 [50, 105]. Autophagy helped in tumor suppression by clearance of damaged cellular organelles and abnormal proteins. Basically it's a process to maintain cellular balance by the degradation of damaged organelles or misfolded proteins through the phagolysosome pathway. [101]

Hepato-pancreatic protective effects

The pathogenesis of pancreatic gland injury and/or islet tissue damage usually causes insufficiency of beta cell secretion (insulin), which leads to impaired blood glucose absorption and finally increased glucose levels in blood [61,106]. Vitexin has demonstrated protective effect against pancreatic B- cell from toxicity induced by streptozotocin (STZ) or Lipo- polysaccharide (LPS) via the up-regulation of Nrf2 and antioxidases [107], as well as the inhibition of MAPKs (mitogen activated protein kinase or MAP kinase), including JNK (Jun N-terminal kinase) and p38 or p38 mitogen-activated protein kinases [10]. *Nrf2* induces cellular rescue pathways against oxidative injury, abnormal inflammatory and immune responses, as well as apoptosis [33], while MAPKs regulate cell functions like cell proliferation, gene expression, differentiation, mitosis, cell survival, and apoptosis [108] and MAPKs including JNK and p38 are crucial regulators of cellular physiology, cell pathology, and many diseases including cancers [109].

Vitexin also increased glucose transporter type 4 (GLUT4) translocation from the cytoplasm to the cell membrane, suggesting the promotion of glucose uptake (absorption) [110]. GLUT4 is an insulin-regulated glucose transporter, responsible for insulin depended glucose uptake into fat and muscle cells [111]. It also improved diabetes-induced sexual dysfunction and fertility impairments in laboratory animals [112]. In this condition, vitexin therapy increased genital organs weight, reduced testicular pathological structure damage, restored spermatozoa quality, and improved sex hormonal balance [112]. In summary, vitexin have potential health benefits by protection against liver and pancreas injury.

Metabolic (glucose and fat) effect

Vitexin have potential health benefits with respect to hepato-protection, glucose and fat metabolism [10-13, 96, 112]. As in the mechanism of glucose and fat metabolism, AMPKa was a key molecular target on vitexin-mediated fat reduction [11]. It activated AMPKa, a master in controlling fat accumulation [113], and inhibited CCAAT (cytosine-cytosine-adenosine-adenosine thymidine)/Enhancer Binding Protein Alpha (*C*/EBPα) and Fatty Acid Synthase (FAS), two contributors to lipogenesis and adipocyte differentiation [11]. Moreover, vitexin exerted protection against hepatic injury by down-regulating Alanine amino transferaase, Asparted amino Transferase, Alkaline Phosphatase, and Lactate dehydogenase enzymes involved in liver functions [12]. In summary, vitexin have potential health benefits by regulating the homeostasis of glucose and fat metabolism.

Antimicrobial effects

Vitexin has shown inhibitory effects against gram negative bacteria, particularly *Pseudomonas aeruginosa* [68, 114]. *P. aeruginosa* can cause cystic fibrosis and infections in the urinary tract, kidney, and intestine through surface-attached biofilm on animal and human hosts [114]. Vitexin attenuate the formation of *P. aeruginosa* biofilms mainly through inhibiting cell adhesion ability, specifically by decreasing pathogen-swarming motility and down-regulating quorum-sensing regulator proteins [114].

Antiviral effects

Vitexin also have ability to inhibit infection with influenza viruses [115, 116]. Many researches have shown that vitexin acted as an inhibitor for virus release and replication through inhibiting neuraminidase enzyme in influenza virus [115-117].

CONCLUSION

Vitexin is obtained from the food sources. It is used as an active component with herbal supplement. The vitexin has multiple beneficial bioactivities. As a potent antioxidant, it prevent can oxidative stress induced damages to the nervous, heart, liver-pancreas tissues and other systems with possible mechanism on molecular and cellular signaling. It produces different biological effects through activation or inhibition of the signaling pathways like AMPKa, Nrf-2, mTOR, PUMA, MMP, PARP, JNK, p38, Bcl-2/Bax ratio etc. It can enhance stress resistance, inflammatory responses, improvement of

energy homeostasis, inhibiting oncogenesis, detoxification etc. Conclusively, vitexin is a natural molecule with a variety of bioactivities which need further clinical trials to integrate it into effective functional food products.

Conflict of Interest

None declared.

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