A Brief Overview of Nutrient Anti-Inflammatory Molecules and their In Vitro and In Vivo Activity

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Abstract

Anti-inflammatory nutrients are components of normal dietary intake with reported anti-inflammatory properties. In comparison to traditional pharmacological anti-inflammatory drugs, anti-inflammatory nutrients may have less side effects. However, one limitation of anti-inflammatory nutrients is their inefficiency compared to current pharmacological anti-inflammatory drugs, as higher nutrient concentrations may be required to provide similar pharmacological effect. A large range of anti-inflammatory nutrients are currently under investigation for their therapeutic use and efficacy: these include amino acids, fats, vitamins and flavonoids. This review provides a brief overall perspective of the existing literature on anti-inflammatory nutrients and their proposed mechanisms of action.

Introduction

Inflammatory responses are the basic mechanisms of the immune system against microbial invasion as well as promoting healing. Inflammation is described as the local response to cellular injury that is marked by capillary dilatation, leukocytic infiltration, redness, heat, pain, swelling and sometimes loss of function due to pro-inflammatory mediators [1]. Although inflammation is part of a normal response to tissue injury, when uncontrolled, it results in earlier development of chronic diseases such as asthma, rheumatoid arthritis, multiple sclerosis, hepatitis, ulcerative colitis and Crohn’s disease [2]. Chronic inflammation also contributes to the development of cancer, cardiovascular diseases and neurodegenerative diseases [3-5]. Chronic diseases are the leading causes of fatal burden of disease, disability and death in Australia, accounting for up to 90% of all deaths in 2011 [6]. Current pharmacological therapies to control chronic inflammatory disease have the potential for undesirable side effects. Therefore, there is potential for new therapies to be used long-term with minimal negative effects to control chronic inflammatory disease.

Anti-inflammatory nutrients are components of normal dietary intake with reported anti-inflammatory properties. Compared to traditional pharmacological anti-inflammatory drugs, anti-inflammatory nutrients may have less side effects [7]. There is a large range of anti-inflammatory nutrients that are currently beginning to be investigated for their therapeutic use and efficacy in a range of diseases including chronic inflammatory disease. The most prominent nutrient molecules with reported in vitro and/or in vivo anti-inflammatory properties include; amino acids, fats, vitamins and flavonoids. The aim of this review is to provide a brief overall perspective of the existing literature on these reported anti-inflammatory nutrients and their proposed mechanisms of action with a focus of these agents in gut inflammation.

Inflammatory Pathways

There are many biological mediators involved in the inflammatory cascade and anti-inflammatory agents act by inhibiting synthesis or action of these mediators. The inflammatory pathway that is stimulated is often dependent on the type of pathogen introduced to the system. For example, bacterial pathogens are recognised by toll-like receptors (TLRs) and trigger a distinctly different response to viral infections that trigger type 1 interferons (IFN) through a TLR independent mechanism [8].

However, the nuclear factor (NF)-κB pathway unites inflammatory and metabolic responses and directs the body’s inflammatory response by controlling the expression of genes involved in inflammation. In the (NF)-κB canonical pathway, triggered by TLRs and pro-inflammatory cytokines, ligand-receptor binding promotes IκB kinase (Iκκ) phosphorylation, which in turn promotes IκB degradation and release of NF-κB [9]. The free NF-κB dimer complexes are then translocated to the nucleus to bind to specific DNA sequences to promote transcription of inflammatory mediators [10]. The NF-κB pathway is central to inflammation and therefore is generally the target of a number of anti-inflammatory therapies.

Peroxisome proliferator-activator receptors (PPARs) play an important role in regulating inflammatory response through transactivation and transrepression of NF-κB and AP1 signalling pathways, suppressing expression of pro-inflammatory mediators including inducible nitric oxide synthase (iNOS) and interleukins [11], therefore are also key to anti-inflammatory responses.

Amino Acids

Amino acids are organic compounds that are biologically essential for life and are required for synthesis of proteins and other biomolecules. The two main types of amino acids in human nutrition...
are essential and non-essential amino acids and are classified according to whether the body is able to synthesize them from other compounds for normal growth. Some amino acids have been found to exhibit anti-inflammatory properties through suppression of inflammatory mediators in the inflammatory cascade [12-15].

Glutamine

Glutamine is a non-essential amino acid under normal physiological conditions that is used in the biosynthesis of proteins. Glutamine is the most abundant amino acid in the body and plasma [16]. Glutamine is a precursor for purines, pyrimidines and amino acid synthesis and acts as a nitrogen shuttle between tissues [17,18]. The rate of skeletal muscle protein synthesis is directly related to the intracellular glutamine concentration [19]. Additionally, glutamine is an energy source for some cell types such as enterocytes and lymphocytes [18]. Glutamine exerts an anti-inflammatory effect through attenuating pro-inflammatory cytokine and chemokine production [12]. This is achieved with higher nutrient concentrations of glutamine leading to increasing suppression of IκB activity and reduction of IκB degradation [12]. Glutamine is reported to block phosphorylation of NF-κB signalling components as well as the signalling components in P38 mitogen-activated protein kinase (MAPK) pathway in TNF-α exposed HT-29 cells [12,20].

Arginine

Arginine is a non-essential amino acid under normal physiological conditions. Arginine acts as a substrate for protein, creatinine and polyamine synthesis as well as production of nitric oxide through the deiminase pathway [21]. Arginine has similar anti-inflammatory properties to glutamine in that it attenuates chemokine response of IL-8 production in TNF-α exposed HT-29 cells and decreases IκB activity [12]. In TNF-α exposed HT-29 cells, arginine inhibits phosphorylation of signalling components of the NF-kB and MAPK pathways [12,20].

Cysteine

Cysteine is a non-essential amino acid for adults but may be essential in infants and the elderly or in individuals with certain metabolic diseases or malabsorption syndromes [22]. Although cysteine is one of the least abundant amino acids in the body, it is required in functionally important sites of proteins including the catalytic, regulatory and cofactor binding sites [23]. Cysteine has been observed to inhibit chemokine IL-8 response in the human monocytic leukemia cell line, THP-1 cells and peripheral blood mononuclear cells (PCMC) stimulated with TNF-α [13]. Cysteine also reduces activation of NF-xB in THP-1 cells stimulated with TNF-α [13].

Histidine

Histidine is considered an essential amino acid required in protein biosynthesis for growth and tissue repair. Histidine is metabolized to histamine, which has been described as an anti-inflammatory agent [24]. Histidine exerts its anti-inflammatory properties through affecting chemokine IL-8 response in THP-1 cells and PCMC stimulated with TNF-α [13]. In addition, histidine also inhibits production of TNF-α and IL-6 in lipopolysaccharide (LPS)-induced mouse peritoneal macrophages in a concentration-dependent manner. This activity is specific to the L-histidine isomer as D-histidine and the histidine metabolite carnosine had no such anti-inflammatory effect [14]. Histidine also reduces NF-xB activation in THP-1 cells stimulated with TNF-α [13]. Histidine has been shown to inhibit LPS-induced NF-xB activation in macrophages [14]. Histidine also inhibits expression of intracellular adhesion molecule-1 (ICAM-1, CD54) in THP-1 cells and PBMCs [13].

Glycine

Glycine is a non-essential amino acid and is an important component and precursor for many macromolecules in the cells. Glycine is found primarily in gelatine and silk fibroin and used therapeutically as a nutrient. Glycine has been shown to report anti-inflammatory properties through reduced activation of NF-xB in THP-1 cells stimulated with TNF-α [13]. Dietary glycine has been shown to significantly inhibit the increase of IL-1β and TNF-α in two experimental colitis models (2,4,6-trinitrobenzene sulfonic acid (TNBS) and dextran sulfate sodium (DSS) rat models) [15]. Glycine also abrogates TNBS induction of cytokine-induced neutrophil chemoattractant and macrophage inflammatory protein (MIP)-2 in the colonic tissue [15].

Selenocystine

Selenocystine is a non-essential, naturally occurring amino acid found in transfer RNAs and catalytic site of some enzymes. Selenocystine is considered the 21st amino acid as the selenium analogue of cysteine, where selenium replaces the sulphur atom [25]. Selenium must be included in the dietary intake to meet required levels of Selenocystine for normal immune function [26]. Epidemiological studies have suggested an inverse association between selenium levels and inflammatory bowel disease (IBD), which includes Crohn’s disease and ulcerative colitis [27]. Selenium has been shown to down regulate NF-kB dependent thromboxane synthase, microsomal prostaglandin and PGE2 synthase pathways, which catalyse the conversion of PGH2 to PGE2, and also aids in the eicosanoid class-switching phenomenon to differentially regulate inflammatory pathways [28,29]. Selenium and selenoprotein expression in macrophages inhibit the NF-xB pathway through a covalent adduct formation of xB kinase-2 with 15-Deoxy-Δ12,14-prostaglandin J1 (15d-PGJ2) [30]. Further, dietary selenium regulates prostaglandins promoting resolution and reduces reactive oxygen species. The composition of the gut microbiota is also affected by the presence of dietary selenium [27].

Tryptophan

Tryptophan is an essential amino acid required for normal growth in infants and for nitrogen balance in adults. Tryptophan is a precursor of serotonin, a neurotransmitter essential in regulating appetite, mood, sleep and pain [31]. Tryptophan is present in dairy products, meat, brown rice, fish and soybeans. Kynurenic acid, a product of the normal metabolism of tryptophan, targets the N-methyl-d-aspartate receptors of the enteric nervous system [32]. Kynurenic acid is able to suppress TNF-α and IL-6 levels after TNBS administration in a mouse colitis model, and it also decreases myeloperoxidase (MPO) activity significantly in the large bowel [33].

γ-glutamyl

γ-glutamyl is a functional group that can be added to individual amino acid molecules to form dipeptides such as γ-glutamyl cysteine and γ-glutamyl valine through a reaction with γ-glutamyl Cysteine synthetase [34]. γ-glutamyl is able to inhibit TNF-α signalling in intestinal epithelial cells and reduce inflammation in DSS-induced colitis through reduced production of IL-8, IL-6 and IL-1β, and through allosteric activation of the calcium-sensing receptor in the gastrointestinal tract [35].

Fats (Triglycerides)

There are three generic types of triglycerides; unsaturated, saturated and trans. Unsaturated triglycerides are liquid in room temperature and considered to be beneficial in improving cholesterol levels and reducing inflammation [36].

Omega-3 fatty acids

Omega-3 fatty acids, also known as n-3 polyunsaturated fatty acids (PUFAs), includes eicosapentaenoic and docosahexaenoic acids which are mainly found in fish oil [37]. Consumption of fish oils diminishes lymphocyte proliferation, T-cell-mediated cytotoxicity, natural killer cell activity, macrophage-mediated cytotoxicity, monocyte and neutrophil chemotaxis, major histocompatibility class (MHC) II expression and antigen presentation, and production of pro-inflammatory cytokines IL-1, IL-6 and TNF-α [38,39].

Another type of omega-3 fatty acids is α-linolenic acid, which is mainly found in plant oils such as linseed oil and green plant tissues [38]. Diets containing large amounts of α-linolenic acid have been...
shown to reduce lymphocyte proliferation compared to saturated fatty acid or n-6 PUFA-rich diets [38]. Most vegetable oils are rich in n-6 PUFA linoleic acid, the precursor of arachidonic acid, which lead to increased synthesis of pro-inflammatory mediators such as prostaglandins, leukotrienes and thromboxane [38]. However linoleic acid, α-linolenic acid, docosahexaenoic acid (DHA) and eicosapentaenoic acid (EPA) reduce IL-6, IL-1β and TNF-α gene expression in LPS stimulated THP-1 cells [40,41]. Therefore, the anti-inflammatory properties of these types of fats are not immediately clear.

There are at least two mechanistic theories to explain the beneficial effects of n-3 PUFA on inflammation; eicosanoid suppression [38,42] and PPAR activation [43]. N-3 fatty acids from fish oil inhibit cytokine and eicosanoid formation by competing with n-6 fatty acids for incorporation in cell phospholipids and for the binding sites of cyclooxygenase and lipoxygenase [44-47]. Populations consuming a diet low in arachidonic acid (less than 90 mg/day) have been shown to have lower inflammatory rates of rheumatoid arthritis (RA) compared to populations consuming a normal Western diet [48,49]. In addition, patients on low arachidonic acid diet supplemented with fish oil capsules, had the least clinical sign of inflammation [48,50].

**Conjugated linoleic acid**

Conjugated linoleic acid (CLA) induce both PPAR-γ and δ by modulating PPAR-γ and δ-responsive gene clusters which are involved in lipid metabolism and epithelial cell maturation [51]. CLA also suppresses TNF-α expression and NF-κB activation while inducing the immune-regulatory cytokine transforming growth factor β1 in DSS induced colitis [52]. Removal of the PPAR-γ gene in the colon abrogated the beneficial effects of CLA in DSS colitis thus indicating that CLA ameliorates colitis through a PPAR-γ-dependent mechanism [51].

**Vitamins**

Vitamins are organic components in the diet that are required for growth and maintenance of normal physiological functioning. Vitamins are separated into fat-soluble vitamins such as vitamin A, D, E, and K or water-soluble vitamins such as vitamin B₃, B₆, and C [53].

**Vitamin A**

Vitamin A is essential for proper retinal functioning, differentiation and growth of tissue, bone health, reproduction and the immune response [53]. Low vitamin A levels have been associated with increased chronic bacterial infections, splenomegaly and high neopterin levels indicating a more active inflammatory response [54]. Vitamin A deficient non-colitic rats have been shown to have higher plasma malondialdehyde concentration compared to vitamin A-sufficient and vitamin A-supplemented non-colitic rats, indicating increased oxidative stress in vitamin A deficiency [55]. Vitamin A also inhibits translocation of transcription factor NF-kB and interrupting the secretion of inflammatory cytokines [56]. Vitamin A supplementation in deficient patients have also shown to increase IL-10 and decrease TNF-α levels [57].

**Vitamin D**

Vitamin D, which includes calciferol and cholecalciferol, is found in liver tissue and fish oils and is essential for calcium absorption [58]. Vitamin D serum concentration has been found to be associated with disease severity in IBD [47,59]. The health benefits of 1, 25-dihydroxycholecalciferol seem to be exclusively mediated by vitamin D receptors [59,60]. Calcitriol, the hormonally active form of vitamin D exerts its anti-inflammatory actions through suppression of prostaglandin, inhibition of both p38 MAPK and NF-κB signalling pathway in lymphocytes, fibroblast and peripheral blood monocytes [61-65]. In addition, vitamin D suppresses the production of pro-inflammatory cytokines such as TNF-α [65].

**Vitamin E**

Vitamin E includes tocopherols and tocotrienols, with α and γ-tocopherol being the two major forms found in some fruits, vegetables, plant seeds and nuts [66,67]. Although both exhibit similar anti-inflammatory actions, γ-tocopherols shown to be more potent than α-tocopherol in inhibiting the cyclooxygenase pathway in macrophages, reducing the production of inflammatory mediators such as PGE, as well as suppressing the action of reactive nitrogen species [68-70]. A specific α-tocopherol metabolite, 2,5,7,8-tetramethyl-2-(b-carboxyethyl)-6-hydroxychroman (α-CEHC) has anti-inflammatory properties, mediated through suppression of TNF-α and LPS-stimulated nitrite and PGE₂ production. A further metabolite, 2,7,8-trimethyl-2-(b-carboxyethyl) -6-hydroxychroman only suppresses LPS-stimulated nitrite and PGE₂ production [71]. Vitamin E and C also help to lower free radical plasma concentrations with the combined administration of these vitamins resulting in reduced TNF-α levels [72].

**Flavonoids**

Flavonoids are polyphenolic compounds which includes flavonols, flavones, flavanols, flavans and isoflavans: these are naturally present in vegetables, fruits and plant derivatives such as wine [73].

**Citrus flavonoids**

The anti-inflammatory properties of the citrus flavonoids, hesperidin, and its flavone analogue, diosmin, have been studied. Diosmin and hesperidin inhibit the synthesis and biological activities of different pro-inflammatory mediators, mainly the arachidonic acid derivatives, prostaglandins E2 and F2 and thromboxane A2 [74-76]. Biosynthesis of the inflammatory arachidonic acid-derived mediators involves the actions of phospholipase A2 and key oxidative enzymes, such as cyclooxygenase and lipoxygenase [77]. Flavonoid inhibition of inflammatory reactions catalysed by phospholipase A2, cyclooxygenase and lipoxygenase in vitro, prevents neutrophil activation and the production of reactive oxygen species at inflammation sites [77].

**Apigenin**

Apigenin, found in celery and parsley, has been shown to have anti-inflammatory properties. Apigenin suppresses the expression of IL-8, IL-1β and TNF-α in LPS-stimulated mouse macrophage and human monocytes [78,79]. Apigenin has also been shown to suppress NF-κB activity and inhibit cyclooxygenase-2 and iNOS expression in LPS-stimulated mouse macrophages [80].

**Baicalein**

Baicalein flavone, originally isolated from Scutellaria baicalensis roots is noted to have anti-inflammatory activities. Baicalein inhibits iNOS, cyclooxygenase-2, TNF-α expression and suppresses NF-κB activation in LPS induced RAW264.7 cells [81].

**Genistein**

Genistein, found in soy beans and soy bean-related products, has similar anti-inflammatory properties to other flavonoids. Genistein suppresses the expression of IL-6, IL-1β and TNF-α in LPS stimulated mouse macrophages [82]; It also inhibitsSTAT-1, NF-κB activity, cyclooxygenase-2 and iNOS expression in LPS-stimulated mouse macrophages [80,83].

**Other Nutrients**

**Curcumin**

Curcumin is the main yellow pigment found in turmeric and it is a widely used spice and food colouring agent [84]. Studies have shown that curcumin could play a protective role in ulcerative colitis through regulation of oxidant and anti-oxidant balance as well as controlled release of inflammatory mediators such as TNF-α and nitric oxide [85].

Curcumin significantly attenuates intestinal damage associated with IBD as well as reducing MPO activity and TNF-α release [86].
Curcumin also reduced colonic nitrite levels and induced down-regulation of cyclooxygenase-2 and iNOS expression, as well as a reduction in the activation of p38 MAPK signalling pathway [86]. The inhibition of p38 MAPK signalling pathway by curcumin could result in the reduction in cyclooxygenase-2 and iNOS inflammatory mediators and suppress the colonic mucosa nitrite production, thus reducing the development of chronic colitis in experimental animals [86]. Curcumin has also been shown to activate PPAR-γ and its ligands in TNBS-induced colitis in rats through inhibition of NF-κB translocation [87]. In addition, curcumin has been shown to suppress NF-κB and AP-1 in human glioma cells and inhibit expression of pro-inflammatory cytokines in human lymphocytes and macrophages [88-92].

Pro-inflammatory molecules inhibited by curcumin include phospholipase, lipooxygenase, cyclooxygenase 2, leukotrienes, thromboxane, prostaglandins, nitric oxide, collagenase, elastase, hyaluronidase, monocyte chemotactic protein-1 (MCP-1), interferon-inducible protein, TNF-α, and IL-12 [93]. In addition, curcumin has been shown to suppress NF-κB and AP-1 in human gloma cells and inhibit expression of pro-inflammatory cytokines in murine lymphocytes and macrophages [88-92].

Table 1: Summary of nutrient anti-inflammatory concentrations.

<table>
<thead>
<tr>
<th>Nutrient</th>
<th>Concentration/ Dose</th>
<th>Delivery method</th>
<th>Cell/ Animal type</th>
<th>Ref.</th>
</tr>
</thead>
<tbody>
<tr>
<td>Amino Acids</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Glutamine</td>
<td>15-240 mM</td>
<td>in vitro</td>
<td>HT29 cells</td>
<td>[12]</td>
</tr>
<tr>
<td>Arginine</td>
<td>2.5-50 mM</td>
<td>in vitro</td>
<td>HT29 cells</td>
<td>[12]</td>
</tr>
<tr>
<td>Cysteine</td>
<td>0.2-20 mM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[13]</td>
</tr>
<tr>
<td>Histidine</td>
<td>20 mM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[13]</td>
</tr>
<tr>
<td>Glycine</td>
<td>20 mM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[13]</td>
</tr>
<tr>
<td>Selenomic/ Selenocysteine</td>
<td>0.4-1.0 ppm</td>
<td>Oral, in vivo</td>
<td>C57BL/6 mice</td>
<td>[29]</td>
</tr>
<tr>
<td>Kynurenic acid</td>
<td>25 mg/kg</td>
<td>IV, in vivo</td>
<td>Sprague-Dawley rats</td>
<td>[33]</td>
</tr>
<tr>
<td>γ-glutamyl cysteine and γ-glutamyl valine</td>
<td>0.5 mM</td>
<td>in vitro</td>
<td>Caco-2 cells</td>
<td>[35]</td>
</tr>
<tr>
<td>FATS</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Eicosapentaenoic acids</td>
<td>100 μM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[41]</td>
</tr>
<tr>
<td>Docosahexaenoic acids</td>
<td>100 μM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[40,41]</td>
</tr>
<tr>
<td>α-linolenic acid</td>
<td>100 μM</td>
<td>in vitro</td>
<td>THP-1 cells</td>
<td>[40]</td>
</tr>
<tr>
<td>Conjugated linoleic acid</td>
<td>45-80 mg/day</td>
<td>Oral, in vivo</td>
<td>C57BL6/J mice</td>
<td>[51]</td>
</tr>
<tr>
<td>Vitamins</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Vitamin A</td>
<td>41 IU/kg/day</td>
<td>Oral, in vivo</td>
<td>Sprague-Dawley rats</td>
<td>[56]</td>
</tr>
<tr>
<td>Vitamin D</td>
<td>50 IU/day</td>
<td>Oral, in vivo</td>
<td>C57BL6 mice</td>
<td>[59]</td>
</tr>
<tr>
<td>Vitamin E</td>
<td>800 mg/d</td>
<td>Oral, in vivo</td>
<td>Human</td>
<td>[65]</td>
</tr>
<tr>
<td>Flavonoids</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Citrus flavonoids (Hesperidin and Diosmin)</td>
<td>500 mg Daflon</td>
<td>in vitro</td>
<td>Endothelial fibroblasts, RBL-2H3 cells</td>
<td>[75,76]</td>
</tr>
<tr>
<td>Apigenin</td>
<td>10-30 μM</td>
<td>in vivo</td>
<td>J773.2 cells, RAW 264.7 cells</td>
<td>[78,79]</td>
</tr>
<tr>
<td>Baicalein</td>
<td>10 μM</td>
<td>in vitro</td>
<td>RAW264.7 cells</td>
<td>[81]</td>
</tr>
<tr>
<td>Genistein</td>
<td>10-100 μM</td>
<td>in vitro</td>
<td>J774 cells</td>
<td>[63]</td>
</tr>
<tr>
<td>Others</td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Curcumin</td>
<td>12.5-30 μmol/L</td>
<td>in vitro</td>
<td>lymphocytes</td>
<td>[89]</td>
</tr>
<tr>
<td>Resveratrol</td>
<td>50 μM</td>
<td>in vitro</td>
<td>J773.2 cells</td>
<td>[78]</td>
</tr>
<tr>
<td>S-allyl cysteine</td>
<td>80 mg/kg</td>
<td>Oral, in vivo</td>
<td>Rats</td>
<td>[101]</td>
</tr>
</tbody>
</table>

S-allyl cysteine (SAC)

S-allyl cysteine (SAC) is the most abundant organosulfur compound in aged garlic extracts (AGE) [101]. SAC exerts anti-inflammatory actions through inhibition of TLR4 and Iκκ activity, and increasing the PPAR-γ expression which has the overall effect of decreasing NF-κB pathway signalling. SAC also results in reduced levels of pro-inflammatory cytokines such as IL-1β [101] (Table 1).

Conclusion

There is a significant volume of literature reporting the anti-inflammatory activities of numerous nutrients. A number of amino acids, and their derivatives, have been reported to exert anti-inflammatory effects through suppression of inflammatory pathways and reduced production of anti-inflammatory mediators. Omega-3 fatty acids such as DHA, EPA as well as linoleic acid and conjugated linoleic acid, reduce inflammation and promote protective effects by suppressing synthesis of pro-inflammatory mediators. Vitamins are important for normal physiological processes in the human body but also appear to be increasingly important in the suppression and control of inflammation. Flavonoids have many anti-inflammatory properties: several are currently being investigated for their therapeutic action as they are able to suppress the production of inflammatory mediators through major inflammatory pathways.

The possibility of using nutrients instead of synthetically derived
anti-inflammatory drugs is very promising however there remains concern around the effective concentrations required for nutrient anti-inflammatory benefits. Understanding of how nutrients exert anti-inflammatory effects will assist in the development of safer and potentially more effective anti-inflammatory therapies. Nevertheless, there appears to be a role for these nutrients in future therapeutic interventions for chronic inflammatory disease.

References


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