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# Dietary Factors Influencing the Intensity of Low-Grade Inflammation in Obesity

**Weronika Fic [1] and Ewelina Polak-Szczybyło [2,*]**

[1] Student Scientific Club of Human Nutrition, Faculty of Health Sciences and Psychology, Collegium Medicum, University of Rzeszów, ul. Warzywna 1a, 35-959 Rzeszow, Poland; wf130811@stud.ur.edu.pl
[2] Faculty of Health Sciences and Psychology, Collegium Medicum, University of Rzeszów, ul. Warzywna 1a, 35-959 Rzeszow, Poland
* Correspondence: ewpolak@ur.edu.pl

**Abstract:** According to WHO (World Health Organization), in 2022, 43% of adults worldwide were overweight and 16% suffered from obesity. Overweight and obesity is a condition in which the body, due to the excess of consumed calories, accumulates it in the form of adipose tissue. However, this tissue is not only an energy store but also secretes numerous adipokines, mainly with pro-inflammatory effects. The mobilization of the immune system due to the accumulation of adipose tissue is called low-grade inflammation (LGI) and is a mediating factor between excess body weight and diseases such as cardiovascular disease, insulin resistance, type 2 diabetes, neurodegenerative diseases, sleep apnea, and even cancer. The aim of the review is to update reports related to dietary factors influencing the severity or alleviation of low-grade inflammation in obese people. The review used studies from PubMed and Google Scholar from the last 10 years. The results indicate that the Western diet, rich in processed foods, high levels of saturated fatty acids, simple sugars, salt, and low fiber and nutrient content (vitamins and minerals), leads to increased low-grade inflammation. On the other hand, calorie restrictions and an appropriate balance of macronutrients, fatty acids, and antioxidant or anti-inflammatory nutrients (e.g., polyphenols, vitamins A, C, and E, and selenium) may reduce the severity of LGI, reducing the risk of obesity-related diseases.

**Keywords:** anti-inflammatory diet; low-grade inflammation; metabolic disease; obesity; western diet



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## 1. Introduction

Obesity is a disease that affects many individuals around the world. The World Health Organization (WHO) reports that since 1990, obesity has more than doubled among adults and quadrupled among adolescents. In 2022, 890 million adults as well as 160 million children over 5 years of age and adolescents living with obesity were recorded [1]. Obesity is a pathological condition that involves the storage of excessive amounts of fat tissue in the body. Factors influencing the etiology of obesity include genetics, a western diet based on highly processed and high-calorie foods that are not rich in nutrients, a sedentary lifestyle [2], and endocrine disorders [3]. Many diseases are correlated with obesity and low-grade inflammation (LGI). A high BMI disrupts the homeostasis of adipokines in adipose tissue, contributing to the spread of pro-inflammatory cytokines from adipocytes and macrophages. As a result of this activity, immune cells penetrate adipose tissue, giving rise to LGI [4]. The occurrence of this condition in people with obesity increases the likelihood of developing diseases such as nonalcoholic fatty liver disease, cancer, type 2 diabetes, insulin

resistance, cardiometabolic diseases, asthma, depression, autoimmune disorders, and obstructive sleep apnea syndrome [5]. It should be noted that the mechanism of low-grade inflammation has not yet been fully understood. The complicated nature of inflammatory substances in obesity may be due to the recent report on the anti-inflammatory action of Angiotensin 1–7 (Ang 1–7), which is a component of the renin-angiotensin system (RAS). However, the mechanism of its action remains unclear. Ang 1–7 directly acts on WAT, attenuating obesity-induced inflammation [6].

Despite excessive body weight, patients with obesity often suffer from micronutrient malnutrition [7], which can lead to LGI. Therefore, diet plays such an important role in shaping the course of low-grade inflammation. The relationship between diet and LGI results from the presence or absence of bioactive substances contained in food. Products rich in active chemical compounds effectively reduce markers of inflammation, while products with a low content of bioactive compounds and a high content of pro-inflammatory substances and micronutrient deficiencies increase the concentrations of markers of inflammation. Appropriate dietary intervention can reduce LGI and prevent diseases associated with it [8]. The aim of this article is to collect the latest knowledge on dietary factors influencing changes in low-grade inflammation in obese people. Dietary modifications can prevent numerous diseases accompanying excessive body weight.

## 2. Materials and Methods

The presented article is narrative and does not include a systematic review of the literature. It includes knowledge based on meta-analyses, observational, and experimental studies on LGI. PubMed and Google Scholar databases were thoroughly searched. The revision period was December 2024. Several words in English were used to search for articles: low-grade inflammation in obesity, pro-inflammatory diets, anti-inflammatory diets, pro-inflammatory food groups, anti-inflammatory food groups, pro-inflammatory nutrients, anti-inflammatory nutrients, and pro-inflammatory markers. Articles published in peer-reviewed journals were included if they were published between 2014 and 2024. Publications published before 2014 were also reviewed if they were considered crucial to providing basic knowledge. The search included only literature in English.

## 3. Nutritional Factors Modifying the Intensity of Low-Grade Inflammation

### *3.1. Diet Paterns*

#### 3.1.1. Dietary Inflammatory Index

The dietary inflammatory index (DII) is used to assess the pro- or anti-inflammatory effects of a diet [9]. The assessment of the diet is based on the impact of 45 elements on 6 pro-inflammatory biomarkers such as C-reactive protein (CRP), tumor necrosis factor alpha (TNFα), interleukin-10 (IL-10), interleukin-4 (IL-4), interleukin-6 (IL-6), and interleukin-1 beta (IL-1β) [10]. The higher the DII score, the greater the inflammatory potential of the diet and the greater the likelihood of obesity [9,11]. Studies show a correlation between a high DII score and high levels of TNF-α and IL-6 in healthy women and men. As well as a low DII score and lower CRP levels in people with obesity [12].

#### 3.1.2. Western Diet

A typical diet plan for highly developed countries is based on highly processed and high-energy food rich in large amounts of trans fatty acids and saturated, refined carbohydrates, sugar, red meat, and food additives. It is also characterized by low content of vitamins, minerals, unsaturated fatty acids, and fiber [7]. Many systematic reviews have shown a relationship between excessive consumption of ultra-processed food in the group

of adults and the likelihood of developing obesity, overweight, cancer, metabolic, and cardiovascular diseases. Lopes et al. in their ELSA-Brasil study covering women and men. They show that frequent consumption of ultra-processed food causes an increase in the concentration of (CRP) by 14% in women [13]. After adjusting the data for Body Mass Index (BMI), the results lost their significance. The Melbourne Collaborative Cohort study found that higher consumption of processed foods was associated with a 4% increase in high-sensitivity C-reactive protein (hsCRP) in men despite BMI. A cross-sectional study by Martins et al. on adolescents showed a correlation between high consumption of ultra-processed foods and increased plasma leptin [14]. Review studies show that a Western diet increases the production of lipopolysaccharide (LPS) and toll-like receptor 4 (TLR4), which stimulates the generation of (TNF-α), (IL-6), and (IL-1β) [10,15,16]. Advanced glycation end products (AGEs) formed as a result of the specific processing of the Western diet at very high temperatures bind to the receptor for advanced glycation end-products (RAGE), causing an increase in pro-inflammatory cytokines [14]. Additionally, it stimulates the expansion of mitogen-activated protein kinase (MAPK), which initiates the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB) pathway and, as a result, increases the expansion of TNF-α, IL-6, inducible nitric oxide synthase (iNOS), and monocyte chemoattractant protein-1 (MCP1) [16].

#### 3.1.3. Mediterranean Diet

The Mediterranean diet originates from the Mediterranean region. It is based on a high consumption of vegetables, fruits, olive oil, nuts, fish, seafood, and whole grain products and on limiting the consumption of meat and high-fat dairy products. There is a correlation between this eating style and the prevention of many diseases, including reducing the severity of LGI [9,17]. This diet is rich in many anti-inflammatory nutrients such as omega-3 fatty acids, vitamins, minerals, polyphenols, and fiber, thanks to which it effectively reduces the level of pro-inflammatory biomarkers [18]. Observations conducted on obese and overweight adults without diabetes confirm that the Mediterranean diet limits the amount of pro-inflammatory cytokines IL-6 and TNF-α and the level of CPR. In people following a Mediterranean diet, a decrease in the activity of the NF-κβ signaling pathway was observed, and consequently a lower amount of IL-1β and interleukin-15 (IL-15) was produced, which are involved in the inflammatory cascade [8,9]. The effect of this eating style on increasing the anti-inflammatory activity of the cytokine interleukin-10 (IL-10) and adiponectin was also observed [9,17]. Adherence to the principles of the Mediterranean diet is associated with a lower likelihood of obesity or overweight and LGI [10].

#### 3.1.4. Vegetarian and Vegan Diet

A vegan diet is based solely on plant products, such as vegetables, fruits, cereals, and nuts, excluding all animal and animal products [19]. A vegetarian diet, on the other hand, allows for animal products such as eggs or milk and dairy products. Many studies indicate the anti-inflammatory effect of these diets due to the increased consumption of saturated fats and cholesterol, as well as higher amounts of antioxidant micro-nutrients in the diet, such as vitamins C and E, dietary fiber, and phytochemicals. Observations conducted on overweight people on a vegan diet show a decrease in hsCRP levels [20]. In turn, a secondary study showed an association between a vegan diet and a decrease in CRP levels [21]. A systematic review and meta-analysis of 21 studies related to a vegetarian and vegan diet in comparison with a control group of omnivores should be cited. A significant decrease in CRP levels was shown in people on a vegan diet compared to omnivores. This correlation was not as strong when comparing this parameter in vegetarians and

in the control group. No significant effects were observed for all other biomarkers of inflammation, such as IL-6, IL-18, IL-1 RZS, or TNF-α [22].

#### 3.1.5. Low Glycemic Index Diet

The main assumption of a low glycemic index (GI) diet is the use of products with a low glycemic index, i.e., less than or equal to 55 [23]. The average glycemic index is 56–69, and the high one is above 70. GI applies only to products containing carbohydrates. It indicates the rate at which blood glucose levels increase after their consumption [24]. The aim of this nutritional manipulation is to avoid a rapid increase in insulin and blood glucose [23]. Proper composition of meals by combining carbohydrates with fats and proteins can reduce the GI of the entire meal. The content of fiber in products also affects the value of the index, as well as heating starch products to produce resistant starch (RS), which reduces the GI of the meal [24,25]. It should be mentioned that resistant starch has a significant effect on the intestinal microbiota as a prebiotic and seals the intestinal barrier. A negative correlation has been shown between RS consumption and the level of LPS, IL-2, and IL-4 [26]. The use of a low glycemic index diet by overweight and obese children and adolescents was associated with a decrease in pro-inflammatory biomarkers such as CRP and IL-6 [12,27].

#### 3.1.6. Low-Calorie Ketogenic Diet

A low-calorie ketogenic diet involves minimizing the intake of carbohydrates as much as possible in order to generate ketone bodies by the body [28]. Following this nutritional plan causes increased secretion of adiponectin in people with obesity. Adiponectin stimulates the expression of IL-10 and inhibits the activity of NF-κB, which results in a decrease in the number of TNF-α, IL-1b, and IL-6 [29]. Very low-calorie ketogenic diet (VLCKD) used by obese and overweight people in a clinical study caused a decrease in the amount of interleukin-11 (IL-11), interleukin-12 (IL-12), interleukin-2 (IL-2), matrix metallopeptidase 1 (MMP1), and an increase in interferon gamma (IFN-γ). The decrease in pro-inflammatory cytokines was clearly noticeable in the state of maximal ketosis. [28]. It remains controversial whether the effect of this type of diet results from a reduced amount of calories or a low GI.

#### 3.1.7. DASH Diet

The DASH diet (Dietary Approaches to Stop Hypertension) was created to combat hypertension because its main goal is to reduce sodium intake and increase potassium intake in the diet. It is based on vegetables, fruits, unsaturated fatty acids, low-fat dairy products, poultry, fish, and whole grains. It limits the consumption of highly processed foods rich in saturated fatty acids, sugar, red meat, and refined grains. The pillars of this diet are strongly correlated with an anti-inflammatory diet. Studies have been conducted on young adults to verify the effect of the DASH diet on the immune response. After 8 weeks, obese and overweight individuals showed a decrease in the number of double-positive T helper cells (TCD4+), naive T cells (CD45RA+), memory T cells (CD45RO+), lymphocytes, and B lymphocytes. An increase in granulocytes, general T cells (TCD3+), and TCD4 + CD45RO + memory cells was noted [30]. A study on overweight individuals with periodontitis and a systematic review on nonalcoholic fatty liver disease show a decrease in hs-CRP, CRP, and IL-6 levels after the DASH diet [31,32].

### *3.2. Food Groups Affecting the Intensity of Low-Grade Inflammation in Obesity*

#### 3.2.1. Alcohol

Although some studies report beneficial effects of moderate doses of alcohol, especially in the form of wine and beer [33–35], excessive or chronic alcohol use can promote

inflammation in the body indirectly through tissue damage reaching the cellular level [36]. Alcohol-induced inflammation is one of the main causes of several chronic inflammatory diseases. TNF-α induced by alcohol is the most important pro-inflammatory factor causing liver diseases. The role of alcohol-induced inflammation associated with the cytokine TNF-α in liver cirrhosis has been established in animal model studies, where reduced expression of the cytokine gene protects against alcoholic cirrhosis [37]. Studies have shown that alcohol consumption is associated with increased CPR production and induced increases in IL-6, IL-10, IL-12, and IFN-γ [38]. Additionally, intestinal inflammation or a worse response to infections is associated with a cascade of processes involving monocytes and macrophages and increased ROS production as a result of alcohol consumption. Inflammation resulting from its consumption is also a cause of cancer [39]. Both chronic and acute alcohol consumption in animal and human model studies reduced the intestinal microbiota and increased the level of circulating LPS in the blood [36,40]. It also increased the level of LPS, which initiates the action of TLR4. This is associated with the expansion of Kupffer cells, which generate pro-inflammatory cytokines [41]. The dose that can remain relatively safe depends on many factors, such as age, sex, health, race, and genetic predisposition, as well as the type of alcohol, dose, and frequency of consumption [35,42]. It is worth noting that the World Health Organization considers that there is no safe dose of alcohol [43].

#### 3.2.2. Red Meat

The high content of animal protein, saturated fatty acids, and heme iron in red meat is associated with the occurrence of obesity in people who consume it in large quantities. To prove the relationship between high consumption of red meat and an increase in biomarkers of inflammation, an observation was carried out on 391 healthy women with obesity and overweight. The characteristics of the study group show that women ate an average of 19,660 g/day of red meat. The analysis also showed that regular consumption of this meat increases the levels of plasminogen activator inhibitor-1 (PAL-1), hs-CRP, MCP-1, and N-oxide of trimethylamine (TMAO) [44]. On the other hand, the study on BALB/c mice shows an increase in other pro-inflammatory biomarkers after high consumption of red meat. The mice were divided into 4 groups, of which 1 was the control group, depending on the group they were fed a diet containing 25%, 50%, and 75% of red meat. In the groups fed high concentrations of red meat, an increase in pro-inflammatory biomarkers such as iNOS, cyclooxygenase-2 (COX-2), IL-1β, interleukin-17 (IL-17), IL-6, TNF-α, and nuclear factor kappa-light-chain-enhancer of activated B cells p65 (NF-kB p65) was observed, as well as exacerbation of colonic inflammation [45].

#### 3.2.3. Fish

Fish are rich in unsaturated fatty acids, amino acids, vitamins, and minerals. Due to the nutritional value of fish, it is recommended to consume them twice a week [46]. The ATTICA cross-sectional study conducted on healthy adults illustrates the relationship between regular fish consumption and lower values of CRP, IL-6, and TNF-a [47]. Fish are valuable food due to their fatty acid content and complete protein. It is worth citing an article in which the composition of Atlantic salmon (Salmo salar L.) fillets was examined. It turned out that the concentrations of dioxins, mercury and arsenic were three times higher in wild salmon compared to farmed salmon, but in both cases these amounts did not exceed permissible levels. The protein content was slightly higher in wild salmon, while the fat content in farmed salmon was three times higher than in wild fish, and the share of marine long-chain omega-3 fatty acids was significantly lower (8.9 vs. 24.1%) [48].

#### 3.2.4. Vegetable Oils

A study of 23 people with fatty liver and metabolic syndrome who were given extra virgin olive oil (EVOO) for two months showed a correlation between EVOO consumption and a decrease in plasma levels of IL-6, IL-17A, TNF-α, and IL-1B and an increase in plasma IL-10. Participants also lost weight [49]. Daniela Roxo de Souza et al. administered 4 g/day of fish oil to 32 obese and overweight individuals for 8 weeks. After completing the supplementation, they observed a significant decrease in plasma IL-1β, Il-6, and TNFα [50]. A meta-analysis of randomized, controlled trials shows the anti-inflammatory effect of flaxseed oil, which reduces CRP levels in body fluids in hemodialysis patients [51].

#### 3.2.5. Milk and Dairy Products

Milk and dairy products are full of minerals, vitamins, and macroelements [52]. Aslam et al. in their study on 1338 people did not show any correlation between dairy consumption and immunomodulatory effects [53]. Nestel et al. studied the effect of fermented and unfermented full-fat dairy products and low-fat dairy products in people with obesity. After three weeks, they observed only an increase in IL-6 in the serum of patients consuming full-fat unfermented dairy products [54]. Other pro-inflammatory biomarkers did not show any changes [55]. In a cross-sectional study on 486 women, an association was shown between the consumption of low-fat dairy products and lower levels of CRP, IL-6 and vascular cell adhesion molecule-1 (VCAM-1) [52]. A study conducted on 412 healthy adolescents aimed to assess the correlation between dairy consumption and inflammatory biomarkers. The results of the analysis show that yogurt consumption reduced IL-6 levels only in adolescents with a normal body weight. However, no changes were observed in overweight adolescents. The study also did not detect any relationship between dairy product consumption and plasma CRP, adiponectin, and leptin levels [56]. Analyses conducted on 464 adolescents show a decrease in IL-10, TGFβ-1, IL-1, IL-5, and IL-6 after the consumption of dairy products. However, most cross-sectional analyses show a neutral effect of dairy products on inflammatory biomarkers [5].

#### 3.2.6. Sweetened Beverages and Sweets

Frequent consumption of sweetened beverages and sweets is a factor often mentioned in the literature causing obesity and affecting the intensity of inflammation in the body. At the same time, high intake of simple carbohydrate sources is associated with metabolic diseases. This may be related to LGI. Energy drinks sweetened with sugar or artificial sweeteners have been shown to increase levels of pro-inflammatory TNF-α and IL-6 and reduce levels of anti-inflammatory interleukin-4 (IL-4) and IL-10 [57]. High intake of sweetened beverages promotes an increase in CRP levels [58]. Excessive sugar in the diet stimulates the activity of TLR-4, which initiates the activity of NF-κB, resulting in the expansion of IL-6, IL-1β, TNF-α, and CRP [59].

#### 3.2.7. Cereal Products

Cereal products with low fiber content and high glycemic index are considered to be products unfavorable for health. The Kuopio Ischaemic Heart Disease Risk Factory Study (KIHD) was conducted in 1999–2001 on 756 men and women aged 53–73 years with good health status. It showed that high consumption of refined fiber cereal products is associated with increased hs-CRP concentration. The result was independent of the BMI and gender of the observed persons [60]. Refined carbohydrates are associated with higher concentrations of IL-1β, IL-6, CRP, and TNF-α in plasma [61]. Hajihashemi et al. observed that in obese and overweight children and adolescents, consumption of whole grain cereal products was associated with lower hs-CPR, soluble intercellular adhesion molecule (sICAM), and

serum amyloid A (SAA) [62]. In contrast, observations by Han et al. conducted on people with asthma confirmed a decrease in interleukin-17F (IL-17F) after a higher consumption of whole grain products [12,63]. In overweight and obese people consuming 70 g/day of whole grain wheat for 8 weeks, lower levels of TNF-α and higher IL-10 were found [47].

#### 3.2.8. Food Additives

Butler et al. studied the effect of food coloring on health. They observed that large amounts of food coloring caused an increase in interleukin-8 (IL-8), TNF-α, and IL-10 and inhibited the secretion of transforming growth factor-beta (TGF-β) [64]. Ruiz et al. also demonstrated the pro-inflammatory effect of food coloring, which consisted of increasing the permeability of the epithelial membrane and intensifying the generation of IL-1β and interleukin-18 (IL-18) [65]. Another food additive with pro-inflammatory effects is the emulsifier carboxymethylcellulose, which increases the concentrations of TNF-α, MCP-1, IL-6, and IL-8 [66]. The pro-inflammatory effect of artificial sweeteners was also demonstrated in a study conducted on Wistar rats. Rats fed sucralose and steviol glycosides with sucrose significantly increased their weight. Artificial sweeteners stimulated the activity of TLR4, which induced the activation of NFκB, as a result of which the activity of peroxisome proliferator-activated receptor gamma (PPARγ) was stopped, and the secretion of TNFα increased [67].

#### 3.2.9. Vegetables and Fruits

Vegetables and fruits, due to the content of many beneficial ingredients such as fiber, vitamins, minerals, and polyphenols [60], as an addition to the daily diet, cause reductions in pro-inflammatory biomarkers. Studies show a link between higher vegetable consumption and a decrease in hs-CRP in people with obesity and a decrease in CRP in people with nonspecific bowel disease. In people with asthma, it is associated with a decrease in IL-17F. It was also noticed a positive effect of plant food consumption on TNF-α, IL-6, and hs-CRP [5,12]. The rich phytochemical composition of vegetables and fruits is responsible for blocking the TLR-4, NK-kB, LPS, nitric oxide (NO), prostaglandin E2 (PGE2), iNOS, COX-2, and TNF-α pathways, which affects the overall anti-inflammatory effect of this food group [68].

#### 3.2.10. Nuts

The phytochemical composition of nuts includes phytosterols, polyphenols, tocopherols, fiber, minerals such as magnesium, and unsaturated fatty acids. It is thanks to these components that nuts owe their anti-inflammatory effects [69]. A study conducted on teenagers and young adults consuming 56 g of almonds daily for 90 days resulted in a reduction in the concentrations of TNF-α and IL-6. Additionally, higher consumption of peanuts rich in oleic acid causes a decrease in TNF-α in obese and overweight men [46]. An experiment on mice with induced colitis shows a correlation between the consumption of cashew nuts and a decrease in pro-inflammatory markers. In mice that took 100 mg/kg of cashew nuts, there was a decrease in ICAM-1, TNF-α, and IL-1β [70]. It has also been proven that regular consumption of nuts can result in a reduction in the amount of IL-4 [5].

#### 3.2.11. Spices

Many studies prove the anti-inflammatory effects of spices, which are due to their rich phytochemical composition, characterized by high concentrations of phenolic acids, terpenes, and flavonoids [60]. Black cumin has a strong anti-inflammatory effect due to the presence of thymoquinone, which reduces the levels of TNF-α and IL-6 [71]. The anti-inflammatory effects of turmeric have also been proven. Curcumin contained in turmeric inhibits the NF-κB pathway, which results in a decrease in the levels of pro-

inflammatory markers such as TNF-α, MCP-1, IL-12, IL-1, IL-2, IL-8, and IL-6 [72]. Ganjali and Sahebkar, in a randomized, double-blind, crossover study, proved that treatment with curcumin at a dose of 1 g/day with the addition of piperine at a dose of 5 mg/day for a month reduces IL-1β and IL-4 in the plasma of obese people [73]. Ginger consumption also has anti-inflammatory benefits as it minimizes the amount of TNF-α, NO, IL-1β, IL-17, cyclooxygenase-1 (COX-1), COX-2, hs-CRP, and increases the amount of the anti-inflammatory cytokine IL-10 [74]. Studies show that garlic inhibits the NF-κB pathway and induces a decrease in the levels of TNF-α, IL-1β, IL-12, IL-1α, IL-6, IL-8, and IFN-γ, and increases the level of IL-10 [75]. The anti-inflammatory effect of rosemary is based on the reduction in IL-1, TNF-α, and leukocyte expression [60].

### *3.3. Nutrients That Affect Low-Grade Inflammation*

#### 3.3.1. Trans Fats, Saturated Fats and n-3 and n-9 Fatty Acids

It was suggested that modulating dietary fat intake can be a potential strategy for mitigating obesity-related inflammation and leptin resistance, highlighting the need for targeted nutritional interventions in obesity and metabolic syndrome management [76]. The majority of trans fats are produced during industrial processing, which involves the hydrogenation of vegetable oils rich in unsaturated fatty acids [77]. Observations show that elaidic acid, which belongs to the group of trans fats, increases membrane permeability and also increases the levels of IL-6, IL1β, intercellular adhesion molecule 1 (ICAM-1), vascular cell adhesion molecule 1 (VCAM-1), and prostaglandin E (PGE) [78]. Observations on healthy men show that the consumption of industrial trans fats causes an increase in plasma CRP. A cross-sectional analysis performed on overweight women shows the relationship between the consumption of products rich in industrial trans fats and the increased production of TNFα, C-C motif chemokine ligand 2 (CCL2), and IL-6. In addition, these fats increase the expression of NF-κB, which induces an increase in the concentrations of TNF-α, CCL2, osteopontin (OPN), and macrophages [77]. Studies also clearly confirm the pro-inflammatory effect of saturated fatty acids. Palmitic acid increases the secretion of p38, c-Jun N-terminal kinases (JNK) and kinases stimulated by mitogen-activated protein kinase (MAPK) and extracellular signal-regulated kinase (ERK). It also increases the expansion of activator protein 1 (AP-1), as well as NF-κB, which induces an increase in COX-2, IL-1β, IL-6, and TNF-α. Stearic acid is also responsible for the activation of NF-κB and an increase in the concentrations of p38, JNK, IL-1β, IL-6, and TNF-α. It stimulates the activity of MCP-1, which stimulates greater production of IL-6, IL-8 [79]. Lauric acid stimulates the activity of NF-κB, which results in the initiation of the secretion of pro-inflammatory cytokines [80]. Omega-3 fatty acids have a completely opposite effect, as they reduce the inflammatory response by reducing the adhesion of cytokines to endothelial cell receptors and the generation of eicosanoids and the expression of genes that limit the production of pro-inflammatory cytokines. In addition, N-3 polyunsaturated fatty acids (N-3 PUFA) containing docosahexaenoic acid (DEHA) and eicosapentaenoic acid (EPA) acids protect against intestinal leakage and have a positive effect on the intestinal microbiota. They can increase the number of LPS-inhibiting bacteria and reduce the number of LPS-producing bacteria [81]. Thanks to these actions, they prevent the inflammatory response caused by the entry of LPS into the main circulation through the leaky gut [82]. EPA and DEHA acids stimulate PPARγ, which inhibits the NF-κB pathway, which results in a reduction in IL-1β and MCP-1. Omega-3 acids also prevent the formation of ROS, which minimizes the level of inflammasomes [83]. Additionally, monounsaturated fatty acids (MUFA) have anti-inflammatory effects. They consist of deactivating the NF-κB chain reaction, reducing the number of cytokines, and intensifying the activity of neutrophils [84]. Moreover, a study on Wistar rats with retinitis showed that omega-9 also significantly minimizes the

development of CPR, leukotriene C4 (LTC4), leukotriene B4 (LTB4), PG E2, leukotriene B4 receptor 1 (BLT-1), prostaglandin E2 receptor 4 (EP-4), prostaglandin E2 receptor 1 (EP-1), and COX-2 [85].

#### 3.3.2. Simple Sugars

A diet rich in sucrose has a pro-inflammatory effect. An experiment conducted on wild-type BALB/c mice shows that increased consumption of simple sugars increases the number of T helper 17 cells (Th17), neutrophils, and macrophages, e.g., lymphocyte antigen 6 complex, locus C (Ly6C). It also increases the concentration of TNFα, IL-6, IL-1β, nitric oxide synthase 2 (Nos2), and IL-17 [80]. On the other hand, excessive consumption of fructose leads to increased penetration of macrophages through MCP-1 and ICAM-1 into adipocytes, which leads to the expansion of TNFα [86], CRP, IL-6, and interleukin-1 receptor antagonist (IL-1RA) [87]. High sugar consumption also leads to increased intestinal permeability, as well as greater generation of CPR [88].

#### 3.3.3. Minerals

Sodium intake above the recommended norm can cause an increase in pro-inflammatory biomarkers. Excess sodium caused an increase in interleukin-17A (IL-17A, interleukin-23 receptor IL-23R), TNF-α, and RORγ in mice, and moreover caused a decrease in anti-inflammatory IL-10 [89]. Muller DN et al. as well as a case-control study analyzing the effect of nutrients on prostate inflammation (PLS). They prove that excess sodium increases the differentiation of CD4+ T lymphocytes into TH17 and inhibits the function of T cells, which results in a disproportion within the regulatory T cell (Treg) subpopulation [90,91]. Another element that, in excess, can demonstrate a pro-inflammatory effect is iron. It turns out that iron intake above the norm for a given population intensifies the expansion of IL-1β, IL-6, IL-8, and TNF-α [92]. In contrast to the action of the above-mentioned elements, it has been shown that an appropriate supply of magnesium, zinc, or selenium has a beneficial anti-inflammatory effect. Magnesium effectively counteracts low-grade inflammation. Mg counteracts the initiation of the NF-κB pathway, which is responsible for the prescription of cytokines such as interleukin-1 alpha (IL-1α). It also inhibits the N-methyl-D-aspartate receptor (NMDA), which secretes substance P, and also minimizes the amount of interleukin-1 (IL-1), IL-6, and TNF-α. It additionally increases anti-inflammatory transmitters such as NO, resolivins, protectins, and lipoxins. Mg transported by Magnesium Transporter 1 (MagT1) to T lymphocytes increases their immunological activity [93]. Zinc, on the other hand, is responsible for the maturation and differentiation of T lymphocytes and the generation of IL-2 and IFN-γ [94]. Zinc activates the activity of the A20 protein, which inhibits the NF-κB pathway, which results in a reduction in the number of IL-6, TNF-α, IL-1β, IL-8, and MCP [94,95]. Selenium, an element with immunomodulatory activity, also blocks the secretion of LPS, which is associated with the inhibition of the NF-κB pathway. As a result, the secretion of thromboxane A2 (TXA2) and PGE2 is also inhibited [96]. It also reduces the secretion of TNF-α, IL-6, TGF-β1, COX-2, p38, TLR4 and JNK [97].

#### 3.3.4. Vitamins

A randomized controlled trial involving the intake of a balanced diet enriched with 500 mL/day of orange juice by people with metabolic syndrome showed that vitamin C intake reduces inflammation biomarkers such as CRP and hsCRP [98]. Ascorbic acid also affects the activity of pro-inflammatory genes, and thanks to its ability to demethylate histones, it generates epigenetic transformations of NF-κB [99]. Vitamin E also has anti-inflammatory properties, as it contains γ-tocopherol (γ-TF) and γ-tocotrienol (γ-TT), which actively counteract LGI. γ-tocopherol and γ-tocotrienol limit the formation of pro-

inflammatory eicosanoids and also stimulate the initiation of Sirtuin 1 (SIRT-1), which, thanks to the deacetylation of p65, stops the action of NF-κB and intensifies the synthesis of the NF-κB suppressant, which is inhibitor kappa B alpha (IκBα). γ-TF reduces the amount of CRP, TNF-α, and IL-1β, and γ-TT reduces the number of IL-8 and IL-6. Vitamin E also minimizes the concentration of Cluster of Differentiation 11c (Cd11c) and MCP-1 [100]. Vitamin A has been shown to inhibit the activity of CD4+ T cells, resulting in a decrease in the concentrations of IFN-γ and IL-17. This vitamin activates the expression of Treg, which induces an increase in the anti-inflammatory cytokines IL-10 and TGF-β [68]. Vitamin D also has an immunomodulatory effect. It increases the expansion of MAP, which stops the NF-kB pathway, which reduces the level of IL-8. It also reduces the level of IL-6 and TNF-α [101], IL-12, and IFN-γ. It reduces the response of the Th1 and Th2 immune systems, as a result reducing the level of IFN-γ and TNF-β produced by Th1, as well as L-4, interleukin-5 (IL-5), IL-10, and interleukin-13 (IL-13) produced by T helper 2 cells (Th2) [102]. Vitamins from the B group also affect the regulation of the immune response. Biotin inhibits the expansion of IL-23, IL-1β, IL-23, IL-1β, IFN-γ, and IL-17 as well as reduces the differentiation of TCD4 + into Th17 and Th1. Thiamine ($B_1$) in optimal amounts reduces the concentrations of IL-6, IL-1, and TNF [66]. In turn, riboflavin ($B_2$) blocks the NF-κB pathway by reducing the secretion of IL-6, TNF-α, and the generation of NO [103]. Moreover, Lee et al. noticed that niacin ($B_3$) is able to reduce CRP levels in people with type 2 diabetes and cardiovascular diseases. Niacin also inhibits the NF-κB pathway, which leads to a reduction in the amount of IL-1β, IL-6, and TNF-α. Although the effect of niacin on leptin is less well studied compared to adiponectin. A meta-analysis showed that niacin increased both the anti-inflammatory adiponectin and the pro-inflammatory leptin [104]. Pyridoxine ($B_6$) consists of pyridoxamine, pyridoxal, pyridoxine, and their 5′-phosphates. This vitamin reduces the levels of TNF-α, IL-6, and IL-4 and increases interleukin-2 (IL-2), IL-8, and IL-10 [105]. A cross-sectional analysis based on information from the PREDIMED study demonstrated an association between the presence of cobalamin ($B_{12}$) and lower levels of CRP and IL-6 in individuals at high cardiovascular risk [106]. Studies have shown that folic acid ($B_9$) has an inhibitory effect on the NF-κB pathway, which results in reduced levels of IL-6, IL-8, and TNF-α [107].

#### 3.3.5. Polyphenols

Polyphenols are compounds from the phenolic family that occur naturally in plants. Many of them have antioxidant and health-promoting effects. They have strong anti-inflammatory effects. They effectively reduce the concentrations of IL-1β, IL-6, TNF-α, and MCP-1. They also inhibit the NFκB pathway and increase the activity of Adenosine Monophosphate (AMP), which initiates the secretion of protein kinase [108]. Additionally, it has been observed that polyphenols contained in blackberries reduce the levels of IL-6 and interleukin-15 (IL-15) and CRP [109]. And polyphenols contained in grapes effectively reduce the concentrations of NF-κB, TNF-α, IL-6, and leptin, and increase the number of adiponectin [110]. On the other hand, soy isoflavones reduce the amount of MCP-1 and TNF-α [109]. Anthocyanins also have anti-inflammatory effects. People with a BMI of 29.6 to 39.2 were given 250 mg/day of anthocyanins in the form of blood orange juice. After 12 weeks, the researchers observed a decrease in the levels of leptin, adiponectin, CRP, and TNF-α in the study participants. Observations conducted on obese mice (C57BL/6) that were given a high-fat diet enriched with mulberry for 16 weeks showed that bioactive anthocyanins found in mulberry reduced the levels of leptin, TNF-α, NF-κB, and IL-6 [111]. In addition, a randomized, double-blind, crossover study demonstrated that resveratrol supplementation at 150 mg/day reduced pro-inflammatory markers in obese men. Additionally, in a different study, it was observed that in rhesus monkeys with obe-

sity induced by a high-fat diet, resveratrol supplementation blocked the NF-κB pathway, resulting in a decrease in the levels of IL-1β, IL-6, and TNF-α in plasma [73].

#### 3.3.6. Fiber and Probiotics

A positive effect of large amounts of dietary fiber and probiotics on inflammatory markers has been recorded indirectly through a beneficial effect on GALT (gut-associated lymphoid tissue). It was shown in a study among people with type 2 diabetes that the higher the fiber intake, the lower the CPR level [111]. Additionally, a study conducted by Miller et al. on obese Latino and African-American teenagers proved that a higher dietary fiber intake reduces the amount of PAI-1 and resistin [112]. Based on a meta-analysis that analyzed the effect of resistant starch on inflammatory biomarkers, a reduction in serum TNF-α and IL-6 concentration was found [107]. Fiber additionally initiates the expression of short-chain fatty acids (SCFAs), which binds to PPARγ, thus stopping the activity of the NF-kB pathway. This reaction results in a decrease in VCAM-1 and ICAM-1 [84]. Probiotics are "live microorganisms which when administered in adequate amounts confer a health benefit on the host" [113,114]. They reduce intestinal permeability, preventing LPS from entering the bloodstream. They also inhibit the NF-κB pathway, which induces a reduction in the amount of TNF-α, IL-6, IL-8, and IL-1β and enhances the expansion of IL-10 [115]. In a randomized, double-blind, placebo-controlled study on adults with type 2 diabetes, participants in the research group were given *L. paracasei* HII01 for 12 weeks, while those in the placebo group were given corn starch. The researchers noted that in the research group, supplementation with *L. paracasei* HII01 reduced the levels of hsCRP, LPS, TNF-α, and IL-6 in plasma compared to the placebo group [116].

A summary of the above information is presented in Table 1.

**Table 1.** Classification of pro- and anti-inflammatory diets, food groups, and nutrients.

| | **Pro-Inflammatory** | **Anti-Inflammatory** |
|---|---|---|
| Diet-pattern | Western Diet | Mediterranean diet<br>Vegan diet<br>Low glycemic index diet<br>Low calorie ketogenic diet<br>DASH Diet |
| Food group | Alcohol<br>Red meat<br>Fatty dairy products<br>Sweetened beverages<br>Sweets<br>Refined grain products<br>Food additives | Vegetables<br>Fruits<br>Fish<br>Vegetable oils (e.g., olive oil)<br>Low-fat dairy products<br>Nuts<br>Whole grain cereals<br>Spices |
| Nutrients | Trans and saturated fatty acids<br>Simple sugars<br>Excessive iron consumption<br>Excessive sodium Consumption | Omega-3 and omega-9 fatty acids<br>Fiber and probiotics<br>Polyphenols<br>Selenium<br>Zinc<br>Magnesium<br>Vitamins A, from group B, C, D, E |

## 4. Conclusions

Obesity is a growing health problem with a complex etiology, which is why it requires an individual approach to treatment or appropriate prevention. Low-grade inflammation present in obesity is the result of improper nutrition, based on high-fat and high-calorie

products poor in beneficial nutrients. At the same time, LGI is the basis of many diseases. It is worth noting that a properly composed diet can affect not only weight loss but, above all, lower inflammatory markers, reducing the health consequences of the endocrine activity of adipocytes. The diets, food groups, and nutrients described in this review can affect both the creation of appropriate diet therapy and pharmacological intervention.

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