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Review

Diet and Asthma: A Narrative Review

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Abstract: Asthma is a chronic respiratory disease that impacts millions of people worldwide. Recent studies suggest that diet may play a role in asthma pathophysiology. Several dietary factors have been recognized as potential contributors to the development and severity of asthma for its inflammatory and oxidative effects. Some food groups such as fruits and vegetables, whole grains, and healthy fats appear to exert positive effects on asthma disease. On the other hand, a high consumption of dietary salt, saturated fats, and trans-fat seems to have the opposite effect. Nonetheless, as foods are not consumed separately, more research is warranted on the topic of dietary patterns. The mechanisms underlying these associations are not yet fully understood, but it is thought that diet can modulate both the immune system and inflammation, two key factors in asthma development and exacerbation. The purpose of this review is to examine how common food groups and dietary patterns are associated with asthma. In general, this research demonstrated that fruits and vegetables, fiber, healthy fats, and dietary patterns considered of high quality appear to be beneficial to asthma disease. Nonetheless, additional research is needed to better understand the interrelation between diet and asthma, and to determine the most effective dietary interventions for asthma prevention and management. Currently, there is no established dietary pattern for asthma management and prevention, and the nuances of certain food groups in relation to this disease require further investigation.

Keywords: asthma; airway inflammation; allergy; diet; dietary patterns; obesity; Th2

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1. Introduction

Asthma is a major global health issue. The ongoing global rise of asthma cases, affecting both children and adults, is particularly prominent in low to middle-income countries, while it appears to be subsiding in some high-income countries [1]. Asthma is a serious illness that can endanger a person's life and greatly impact their everyday living [2]. The development of asthma is influenced by both genetic and environmental factors, which can interact to cause disease [3]. It has been suggested that this trend in the disease increase in prevalence relates to lifestyle and environmental changes [4].

A central factor could be changes in dietary patterns characterized by an augmented consumption of highly processed and refined foods, and a lower consumption of vegetables

and fruits [4]. Diets with these characteristics are likely to lead to obesity, which is a pressing public health problem, being simultaneously a disease modifier and a risk factor for asthma [4]. Obese individuals have a higher risk of asthma, and they also experience more frequent and severe symptoms and exacerbations, as well as a lower quality of life and a reduced response to asthma medications [3,5]. Although diet has a clearly established role in some human diseases (e.g., cardiovascular and metabolic disorders), it isn't generally determined as a primary causal factor for asthma development. Nevertheless, it may still play a role in the onset and management of the condition [3]. Most studies on diet and asthma associations focus upon specific foods. Nonetheless, foods are ingested as complex combinations, which include nutrients, bioactive components, and their specific effects in the food matrix. These substances and effects interact with each other synergistically, thereby influencing metabolism and health according to the different dietary patterns [3,4]. In this context, it is relevant to study not only the effects of individual foods, but also the effects of food groups and dietary patterns.

This review aims to explore how common food groups and dietary patterns are associated with asthma.

2. Asthma Phenotypes and Endotypes

Asthma is defined as "(. . .) a heterogeneous disease, usually characterized by chronic airway inflammation. It is defined by the history of respiratory symptoms such as wheeze, shortness of breath, chest tightness and cough that vary over time and in intensity, together with variable expiratory airflow limitation." [2]. Episodes usually resolve either spontaneously or with medication [2,6], but episodic flare-ups can still occur and be life-threatening, thereby holding a significant burden to the patient's life [2].

Even though genetic determinants play a role in the initiation of various asthma pathways, several research work have identified the role of environmental changes in the ongoing asthma epidemic [7]. This disease etiology is attributed to the interactions of environmental exposures, host factors, and genetic susceptibility. Environmental factors include weather, air pollution, molds, pollens, and other aeroallergens; host factors comprise obesity, diet and nutritional factors, allergic sensitization, and the susceptibility to infections; while genetic factors encompasses a large number of asthma susceptibility loci [1,8].

Asthma has different phenotypes and endotypes [7,9]. Consequently, there are variations in its clinical severity and treatment response, and these differences may reflect their pathobiological pathways [9]. Patients may respond very differently to the same therapeutic interventions despite having similar clinical symptoms [7,9]. A long-standing clinical approach to asthma has been to categorize patients into phenotypes based on observable combinations of biological, clinical, and physiological characteristics [9]. However, the strategy to link molecular mechanisms to specific phenotypes has been evolving [9], and endotypes of asthma describe these distinct pathophysiologic pathways at the cellular and molecular levels [9].

The common description of asthma is one of excessive T-helper type 2 (Th-2) cell responses and specific immunoglobulin E (IgE) production, leading to allergic sensitization and airway hyperresponsiveness [9,10]. However, the term "asthma" is now considered a heterogenous definition, representing a collection of several distinctly disordered pathways; when referring to endotypes, the more broadly used are type 2-high (T2H) and type 2-low (T2L) asthma [7,9]. A visual representation of T2H and T2L asthma and the involved lymphocytes and cytokines are presented in Figure 1.

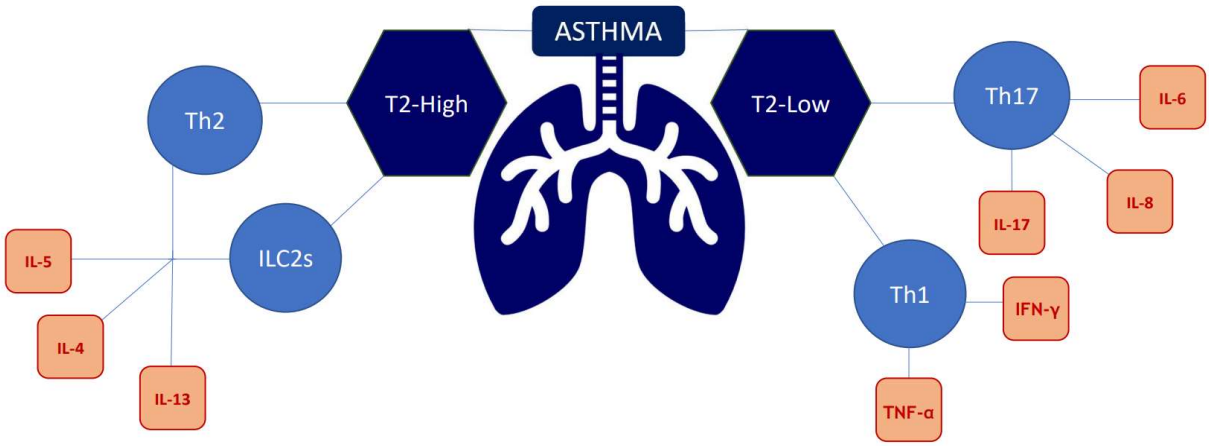

Figure 1. T2-high and T2-low asthma and their involved lymphocytes and cytokines. Th, T helper lymphocytes; ILCs, innate lymphoid cells; and IL, interleukin.

T2H is characterized by airway eosinophilic inflammation [9,11,12] that is stimulated by the adaptive, and, or the innate immune systems: Th-2 cells and type-2 innate lymphoid cells (ILC2s), respectively [7]. This response primarily releases the interleukin (IL) cytokines IL-4, IL-5, and IL-13, respectively [7,12]. IL-4 mediates the differentiation of T cells into Th2 cells [7], and also contributes to the production of IgE by B cells and mast cell degranulation [7,13]. IL-5 has a preponderant role in the recruitment, survival, and maturation of eosinophils [7,13], and IL-13 contributes to the hyperresponsiveness of airway smooth muscle cells and goblet cell hyperplasia [7]. The production of other mediators and chemokines by eosinophils further adds to tissue remodeling [7]. Type 2 immunological responses are linked to atopic diseases and allergy asthma [12].

T2L is sometimes referred in a simplistic way as non-eosinophilic asthma [11,14]. However, not much is known about the pathobiology of T2L asthma [7] other than it is mostly characterized by neutrophilic or paucigranulocitic inflammation [9,11,14], with the latter being defined as patients that do not present eosinophilic or neutrophilic airway inflammation [11,14] but have confirmed airway hyper-responsiveness (AHR) and experience persistent symptoms [11]. Within non-eosinophilic asthma, Th-1 and Th-17 pathways appear to be implicated in neutrophilic asthma [7,13]. The principal cytokines involved in this endotype are IL-6, IL-8, and IL-17 [11], mainly produced by the Th-17 response [7], causing neutrophilic infiltration and AHR [14]. The Th-1 pathway is responsible for elevated interferon-γ (IFN-γ) and tumor necrosis alpha (TNF-α) levels [7,14]. A visual representation of T2H and T2L asthma and the involved cytokines are presented in Figure 1.

Within T2H and T2L endotypes there are also varying phenotypes. Inside the T2H endotype: atopic, late onset, and aspirin-exacerbated asthma; Inside the T2L endotype: non-atopic, smoke-related, obesity-related, and elderly asthma. All of these phenotypes manifest with typical symptoms ranging from wheezing and shortness of breath, to coughing and chest tightness, and are accompanied by variable airflow obstruction [9]. Patients with T2L asthma typically respond poorly to corticosteroids and may therefore be challenging to treat [15].

A substantial body of evidence has documented the co-occurrence of asthma and obesity, implying that the pathobiology of these conditions may be linked [5,16–21]. Obesity and asthma have increased dramatically over the last decades [5,19–22], and have become two of the most common and challenging health issues worldwide [19,21]. According to recent reports of the Global Burden of Disease Study, a high body mass index (BMI) is believed to be one of the most important risk factors for asthma [23,24]. Obesity-mediated asthma has a more severe phenotype [7,25], with obesity seeming to increase asthma prevalence and incidence as a disease modifier, thereby making it more challenging to control [5,25–27]. Individuals with this phenotype present worse asthma symptoms and

a lower quality of life [5,7]. Obesity-mediated asthma appears to be mediated by non-T2 mechanisms, with lower levels of fractional exhaled nitric oxide (FeNO) and normal blood eosinophils [7]. Changes in DNA methylation may influence the Th-1 polarization seen in obesity-mediated asthma, suggesting that environmental factors may contribute to the disease phenotype [17]. It was observed that obese mice presented a higher airway hyperresponsiveness compared to non-obese mice [28].

The diverse mechanisms underlying the asthma-obesity phenotype may be linked to mechanical impediment, hormonal influence, inflammation [5,14,29], diet [5,30], metabolic dysregulation, and genetic and immune factors [5,29].

Deposition of adipose tissue in the area of the abdomen and chest seems to raise abdominal pressure resulting in an increase of airway resistance and residual capacity over time and in a decrease in tidal volume and airway caliber [14,29,31]. These mechanical changes appear to be contributing to bronchial remodeling, increasing airway obstruction and hyperresponsiveness, and thus the risk of asthma [29].

Adipose tissue is an endocrine organ that produces hormones that can influence both inflammation and metabolism. Fat accumulation results in elevated serum concentrations of leptin and decreased levels of adiponectin [14,27]. The increase in leptin production by the adipose tissue may have a proinflammatory effect by inducing neutrophil chemotaxis, the production of reactive oxygen species (ROS), the activation of natural killer cells and macrophages, and the production of Th-1 cytokines such as IL-6 and IFN-γ [14]. On the other hand, adiponectin inhibits the effects of proinflammatory cytokines on endothelial and other cell types, such as TNF-α and IL-6, while also inducing the expression of anti-inflammatory cytokines (IL-10 and IL-1 receptor antagonists) [32].

Obesity-promoting diets, such as the Western diet pattern, are typically high in saturated fatty acids and low in dietary fiber and antioxidants. There is a growing body of evidence on the negative effects of these unhealthy diets on asthma [3,5]. Adhering to a western diet has been shown to increase the risk of having current asthma and doctor-diagnosed asthma in school-aged children [33].

Another hypothesis implicates gut microbiota changes in the development of many diseases, including asthma and obesity. Changes in the microbiota during childhood are thought to play a role in the development of asthma and obesity [14]. Obesity has been linked to a decrease in the diversity of gut bacteria in humans [34,35], and there is evidence that a diet-induced weight loss promotes a Firmicutes to Bacteroidetes ratio decrease [36]. Fiber can modulate the gut microbiota, leading to the production of short-chain fatty acids (SCFAs), including butyrate, which has immunomodulatory and anti-inflammatory effects [37]. A high-fat diet modifies the composition of the gut microbiota, more specifically causes the reduction of protective bacteria, the expansion and colonization of invasive bacteria, and the reduction of SCFA concentrations, implying a potential role in inflammation and the immune response [3]. These changes in microbiota could play an additional role in obesity-related asthma through changes in the production of bacterial-derived or modified metabolites, e.g., lowering the production of SCFAs that can induce anti-inflammatory effects [38].

Additionally, it has been documented that individuals with obesity may present higher circulating concentrations of many inflammatory markers as a consequence of the disease [39], and GINA guidelines recommend weight loss as part of the strategy of asthma management in obese patients [2]. A systematic review found that, for obese asthmatic adults, the more effective dietary intervention is energy restriction [40].

3. Airway Inflammation

Asthma is associated with airway inflammation, hyperresponsiveness, and remodeling of the airways [9,13,41–43].

Inflammation is a biological mechanism that eliminates detrimental stimuli as irritants and pathogens, and damages cells with the purpose of beginning the healing process [43]. Inflammation can be acute or chronic. Chronic inflammation is associated with adverse

effects, such as damage and remodeling of healthy tissues, as it occurs in asthma [43]. A combination of genetic predisposition, environmental exposures, and potential changes in the microbiota and metabolites are the main factors that may lead to inflammation in the lower airways [15].

Pathogens, toxins, pollutants, irritants, and allergens are recognized by toll-like receptors (TLRs) that activate inflammatory cells such as nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB), which then release growth factors and pro-inflammatory cytokines to begin the resolution process [43]. Activated cells begin wound healing in damaged epithelial cells to repair them. This process is called tissue remodeling [43].

A variety of inflammatory changes at the cellular level affect the airways that cause tissue remodeling in asthma [7,15,43]. Pathologic changes of this process include epithelial damage and dysfunction, goblet cell hyperplasia, increased thickness of reticular basement membrane, angiogenesis, increased subepithelial myofibroblasts and fibrocytes, and hypertrophy of airway smooth muscle cells [7,13]. These pathological changes lead to airway wall thickening, luminal narrowing, and mucus plugging, with small airway obliteration [13], resulting in airway hyperresponsiveness and a progressive obstruction of the airflow [7].

Exhaled nitric oxide is a marker of airway inflammation, more specifically of eosinophilic inflammation [44,45]. Nitric oxide (NO) is a reactive, free radical gas that forms in the airways when l-arginine is oxidized to l-citrulline [44], It is produced by two enzymes: constitutive nitric oxide synthase (cNOS), which produces small amounts of NO, and epithelial inducible NOS (iNOS), which is activated by various inflammatory cytokines [44,45]. In allergic asthma, airway inflammation is caused by the activation of a Th-2-mediated pro-inflammatory cytokine mechanism that promotes the expression of epithelial iNOS [44,45]. However, high FeNO values are not always associated with eosinophilic asthma, as elevated levels can also be associated with eosinophilic bronchitis, allergic rhinitis, and viral or allergen exposure [44,45].

4. Diet and Asthma

Asthma is associated with inflammation and oxidative stress [46], as previously mentioned, and the antioxidant and anti-inflammatory properties of several food groups and nutrients may have an influence on asthma and airway inflammation [47,48] (Figure 2).

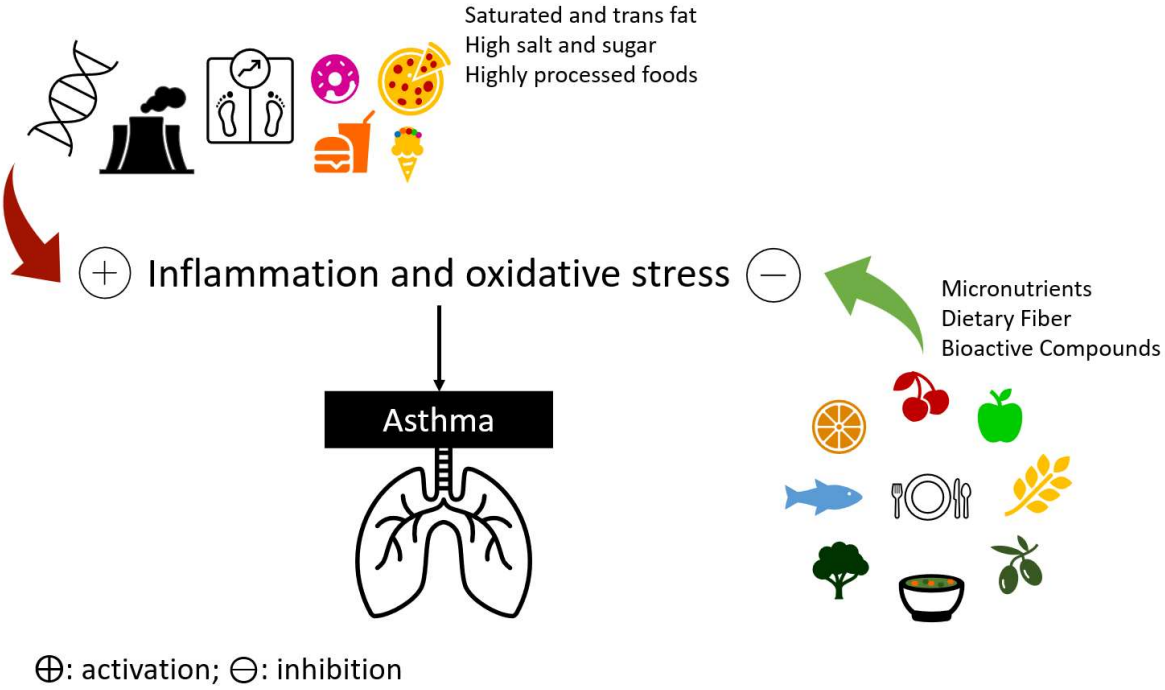

Figure 2. Deleterious or protective effects of diet on asthma genetic, environmental, and host factors.

Recent research is focusing on how epigenetic mechanisms may elucidate the connection between gene regulation and environmental factors in asthma [49]. Epigenetic mechanisms help genes adapt to changes in the environment, and these alterations can contribute to the development of disease phenotypes, being identified as one of the potential causes for the onset of diseases in susceptible individuals [50,51]. Recently, it has been demonstrated that elevated dietary acid loads may modulate asthma-related miRNAs among school-aged children [52,53].

To conduct this review, the inclusion of high-quality studies, such as meta-analyzes, systematic reviews, RCTs, and cohort studies, were prioritized to ensure the reliability of our findings, particularly when examining the impact of specific foods or food groups that were widely explored in this matter. However, to shed light on the less explored areas, we also incorporated cross-sectional studies that delved into more specific aspects of interest. By considering a range of study designs, we aimed to provide comprehensive insights into the relationship between food intake and its potential effects on various asthma outcomes.

4.1. Fruits and Vegetables

Fruits and vegetables are known as a fundamental component of a healthy diet and are recommended to make up half of one's plate at each meal. They offer a variety of nutrients and bioactive compounds that can promote good health and are able to modulate inflammation and prevent chronic diseases [47].

In asthmatic children, fruit and vegetable intake was found to be negatively associated in nasal lavage with IL-8, a marker of inflammation [54]. A study suggested an inverse significant relationship between salad consumption (a source of antioxidants) and FeNO, in children [55]. It was also observed that a greater daily vegetable diversity intake was negatively associated with having self-reported asthma with an odds ratio (OR) = 0.67; 95% CI 0.47, 0.95, whereas a vegetable diversity ingestion superior to three items per day was negatively associated with levels of FeNO $\geq$ 35 ppb (OR = 0.38; 95% CI 0.16, 0.88) and breathing difficulties (OR = 0.39; 95% CI 0.16, 0.97) in school-aged children [56].

A longitudinal study on children up to 5 years observed that the risk of asthma was not associated with the consumption of all fruits and vegetables together, but inverse associations were seen between all leafy vegetables and asthma (Hazard Ratio (HR) = 0.87, 95% CI 0.77; 0.99), and unprocessed vegetables and non-atopic asthma (HR = 0.90, 95% CI 0.81; 0.98), respectively [57].

A cohort study demonstrated that a higher fruit intake at 8 years of age was associated with a tendency to lower the odds of prevalent asthma (T3 vs. T1, OR 0.78; 95% CI 0.60–1.01, *p*-trend 0.083), with reduced odds of incident asthma, and increased odds of remittent asthma (OR = 0.76; 95% CI 0.58–0.99 and OR = 1.60; 95% CI 1.05–2.42, respectively) for up to 24 years [58].

Another study conducted in adults revealed that reducing the intake of antioxidant-rich foods worsens asthma control and lung function. Furthermore, it was demonstrated that an increase in airway neutrophils, resulting from a low-antioxidant diet, can be reversed using lycopene-rich treatments (such as tomato juice) that lead to a decrease in airway neutrophil influx [59]. Wood et al. [60] compared the outcomes of a high-antioxidant diet to those of a low-antioxidant diet, with and without lycopene supplementation, in asthmatic patients, over the course of a 16-week period. It was observed that participants on the high fruit and vegetable intervention arm (high-antioxidant diet) had fewer asthma exacerbations. Several participants in the low fruit and vegetable arm (low-antioxidant diet) were given an antioxidant supplement, but this did not reduce the risk of asthma exacerbations. As improvements were only observed after increasing fruit and vegetable consumption, whole-food interventions seem to be more effective, implying that the whole food dietary component is more relevant than antioxidant supplementation [60].

Two meta-analyzes found that a higher intake of fruit and vegetables was associated with a lower risk of having asthma [61,62]. One of the meta-analyzes found that vegetable intake was negatively associated with the risk of prevalent asthma (OR = 0.95; 95% CI

0.92; 0.98) and that fruit intake was negatively associated with the risk of prevalent wheeze (OR = 0.94; 95% CI 0.91; 0.97) and asthma severity (OR = 0.61; 95% CI 0.44; 0.87) [62]. Six of the seven studies found that fruit and vegetable intake in asthmatics had a protective effect against either airway or systemic inflammation [62]. The other meta-analysis also found protective effects of the total intake of vegetables and fruits in asthmatic children with a relative risk (RR) of 0.57 (95% CI 0.42; 0.77). Fruit consumption was found to reduce the risk of wheeze and asthma (RR = 0.81, 95% CI 0.74; 0.88, and RR = 0.90, 95% CI 0.86; 0.94, respectively). Similarly, the intake of vegetables reduced the risk of wheeze and asthma (RR = 0.88, 95% CI 0.79; 0.97, and RR = 0.91, 95% CI 0.82; 1.00, respectively) [61].

Diets that have a high intake of fruits and vegetable are widely recommended for their health-promoting properties due to their mineral, vitamin, dietary fiber, and bioactive content [47].

Table 1 summarizes the results of different research studies on fruit and vegetable intake and asthma.

Table 1. Studies evaluating the effects of fruit and vegetable intake on asthma and asthma-related outcomes.

Fruits and Vegetables			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Romieu et al., 2009, Cross-sectional [54]	A total of 208 children (median age (years)) (quartiles (Q)25, Q75)), comprising 158 asthmatic children, 9.6 (7.9, 11.0 y), and fifty non-asthmatic children, 9.3 (7.9, 11.5 y).	Dietary intake assessed through a 108-item food frequency questionnaire (FFQ): fruit and vegetable index (FVI); Mediterranean diet index (MDI) were constructed.	Higher FVI: (+) inflammation. Higher MDI: (+) lung function.
Cardinale et al., 2007, Cross-sectional [55]	A total of 130 children with mild-to-moderate asthma were included (mean age: N/R).	Dietary intake assessed by 4-point FFQ: 8 food items (margarine, butter, milk, fresh fruit, tomatoes, salad, cooked vegetables and nuts).	Higher consumption of salad: (+) FENO levels.
Mendes et al., 2021, Cross-sectional [56]	In total, 647 children, with 44 asthmatics, were included. mean age = 8.81 ± 0.80 y.	Vegetable intake and fruit intake were assessed by a single self-reported 24-h recall questionnaire. A diversity score for fruits and vegetables was built.	Higher daily vegetable diversity intake: (+) asthma prevalence; (+) airway inflammation; (+) breathing difficulties.
Metsälä et al., 2023, Cohort study [57]	In total, 3053 children, with 184 incidents with asthma. Children were evaluated up to 5 years of age.	Child's food consumption were assessed by 3-day food records at the age of 3 and 6 months, and at 1, 2, 3, 4, and 5 years of age. Consumption of processed and unprocessed fruits and vegetables was calculated.	All fruits and vegetables intake: (=) asthma prevalence. Leafy vegetables and unprocessed vegetables intake: (+) asthma prevalence.
Wood et al., 2008, Randomized controlled trial [59]	Thirty-two asthmatic adults, mean age ± SEM = 52.1 ± 2.4 y.	Follow-up of a low-antioxidant diet during 10 days, then commence of a randomized, cross-over trial involving 3 × 7-day treatment arms (placebo, tomato extract (45 mg lycopene/day) and tomato juice (45 mg lycopene/day).	Lower intake of antioxidant-rich foods: (−) asthma control; (−) lung function. Lycopene-rich treatments (tomato juice): (+) airway inflammation.

Table 1. *Cont.*

Fruits and Vegetables			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Wood et al., 2012, Randomized controlled trial [60]	In total, 137 asthmatics adults were included, mean age: high-antioxidant diet: 54 ± 14 y; and low-antioxidant diet: 58 ± 15 y.	Individuals were assigned to a high-antioxidant diet (n = 46) or a low-antioxidant diet (n = 91) for 14days and then subjects on the high-antioxidant diet received placebo and subjects on the low-antioxidant diet received placebo or tomato extract (45 mg lycopene/d).	High-antioxidant diet: (+) FEV1, and (+) FVC. Antioxidant supplementation: (=) asthma exacerbations (improvements were only observed after increasing fruit and vegetable consumption).
Seyedrezazadeh et al., 2014, Meta-analysis [61]	Adult and children, papers included in the meta-analysis (n = 42): cohort studies (n = 12); case-control studies (n = 4); cross-sectional studies (n = 26).	Fruit and vegetable intake.	High intake of fruit and vegetables: (+) asthma prevalence; (+) wheezing prevalence.
Hosseini et al., 2017, Meta-analysis [62]	Adult and children, papers included in the meta-analysis (n = 58): cross-sectional (n = 30); cohort studies (n = 13); case-control studies (n = 8); experimental designs (n = 7).	Fruit and vegetable intake.	Vegetable intake: (+) asthma prevalence. Fruit intake: (+) wheeze prevalence; (+) asthma severity prevalence. Fruit and vegetable intake: (+) airway/systemic inflammation.

Notes: Age is described as the mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; Q25, the 25th percentile; Q75, the 75th percentile; IL, interleukin; FVI, fruit and vegetable index; MDI, Mediterranean diet index; FEV1, forced expiratory volume in one second; FENO, fractional exhaled nitric oxide; SEM, standard error of the mean; and ppb, parts per billion. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.2. Fiber

Some of the protective effects observed with fruit and vegetable consumption may also be attributed to the fiber content of these food groups [47]. Whole grains, beans, fruits, and vegetables are all sources of dietary fiber [63]. SCFAs are produced by gut bacteria following the fermentation of soluble fiber and are the principal fuel of colonocytes [64,65]. Butyrate has been demonstrated to activate the peroxisome proliferator-activated receptor alpha, (PPAR) which inhibits the NF-kB activity, a pro-inflammatory transcription factor [66]. Roduit et al. observed that oral administration of SCFAs to mice significantly reduced the severity of allergic airway inflammation [67]. Dietary fiber and SCFAs have also shown anti-inflammatory effects by activating free fatty acid receptors such as G protein-coupled receptors 41 and 43 (GPR41 and GPR43) in animal models [64,65]. GPR43 and GPR41 activation can influence the functions of immune cells, such as neutrophils and dendritic cells. This regulation can lead to a decrease in the release of pro-inflammatory mediators. GPR43 and GPR41 activate the Gi/o proteins that reduce the levels of the secondary messenger cAMP, thereby counteracting the pro-inflammatory effects of the cAMP-mediated signaling pathways [64,65].

One study assessed the acute effects of a single meal rich in soluble fiber (175 g yogurt with 3.5 g inulin and probiotics) compared with a simple carbohydrate meal (200 g of mashed potato) on asthmatic airway inflammation, including changes in free fatty acid receptor gene expression. The studied outcomes were analyzed 4 h after the meal intake. It was observed that airway inflammation biomarkers, such as sputum total cell count, macrophages, neutrophils, sputum IL-8, lymphocytes, and eNO were significantly lower in

the soluble fiber group and this corresponded to increased GPR41 and GPR43 sputum gene expression and improved lung function [37].

The consumption of whole grain products, which are a source of dietary fiber, was found to be inversely associated with asthma in a study assessing 598 Dutch children [68]. For current asthma, the adjusted OR was 0.46 (95% CI 0.19; 1.10), whereas for atopic asthma with bronchial hyperresponsiveness was 0.28 (95% CI 0.08 to 0.99) [68]. Additionally, a high consumption of whole grains was associated with lower odds of asthma (OR = 0.52, 95% CI 0.33; 0.82) [69]. However, as previously observed, other nutrients found in fiber-rich foods, such as fruits and vegetables, cereals, and legumes, may also be contributing to the inverse association found between dietary fiber consumption and airway inflammation. Many fiber-rich foods contain antioxidants and other micronutrients, such as polyphenolic compounds and flavonoids, which may help to reduce the oxidative stress in asthma patients [48].

Table 2 summarizes the results of research on fiber and asthma.

Table 2. Studies evaluating effects of fiber intake on asthma and asthma-related outcomes.

Fiber			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Halnes et al., 2017, Randomized controlled trial [37]	Twenty-nine subjects with stable asthma, mean age: soluble fiber group: 42.1 ± 3.4 y, control group: 40.4 ± 4.6 y.	Effect of a single meal rich in soluble fiber (175 g yogurt with 3.5 g inulin and probiotics) compared with a simple carbohydrate meal (200 g of mashed potato) on asthmatic airway inflammation.	Soluble fiber intake: (+) inflammation; (+) lung function.
Tabak et al., 2006, Cross-sectional [68]	A total of 598 children, with 39 asthmatics, mean age: 10.4 ± 1.2 y.	Dietary intake was estimated using a semi-quantitative FFQ.	Whole grain products intake: (+) asthma prevalence.
Berthon BS et al., 2013, Cross-sectional [48]	In total, 202 participants, with 137 stable asthmatics. Mean age: healthy controls: 46.7 ± 17.4 y; intermittent, mild, and moderate persistent asthma: 54.5 ± 15.5 y; severe persistent asthma: 57.8 ± 14.4 y.	Dietary intake was estimated through a 186-item semi-quantitative FFQ.	Less fiber intake: (−) forced volume in 1 s (FEV1); (−) airway eosinophilia.
Han YY, et al., 2015, Case-control [69]	A total of 678 children, with 351 asthmatics. Mean age: controls: 10.5 ± 2.7 y, asthma: 10.0 ± 2.6 y.	Dietary intake was assessed through a 75-item questionnaire regarding the child's food consumption, including wholegrains in the prior week.	High consumption of wholegrains: (+) asthma prevalence.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; FFQ, food frequency questionnaire; GPR, G protein-coupled receptor; and FEV1, forced expiratory volume in 1 s. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.3. Nuts

Nuts are a rich source of nutrients, healthy fats, and fiber, and have been widely studied for their potential health benefits [70]. While the consumption of nuts has been associated with a lower risk of chronic disease [70], some studies suggest that nut consumption may be linked to the exacerbation of asthma symptoms, as nuts are common food allergens that may induce an allergic response in the respiratory system [71]. Nonetheless, in a cross-sectional study, a high dietary intake of nuts appeared to exert a protective role on the prevalence of wheeze in children (OR = 0.46; 95% CI 0.20; 0.98) [72]. Peanuts, another common source of food allergens, even though being a legume, from a nutritional perspective, their contents are similar to tree nuts. Du Toit, G. et al., in a randomized trial,

found no significant differences in risk of asthma events at 5 years of age between infants who were randomly ascribed either to consume peanuts, or to avoid peanut intake from 4 to 60 months old (p-value = 0.230) [73]. Additionally, there were no significant differences found in the severity of asthma events (p-value = 0.839) [73]. Accordingly, Roduilt et al. demonstrated that the introduction of nuts in the first year of life was not associated with asthma at 6 years old in a cohort study (OR = 0.69; 95% CI 0.36; 1.34) [74]. Nevertheless, in the previously referred study, children with higher values of the dietary diversity score in the first year of life had a lower risk of asthma compared with those with lower score values, and there was a significant reduction of 26% for the development of asthma with each additional food item introduced in the first year of life (OR = 0.74; 95% CI 0.61; 0.89). Table 3 summarizes the results of research on nuts and asthma or asthma-related outcomes.

Table 3. Studies evaluating effects of nuts intake on asthma and asthma-related outcomes.

Nuts			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Chatzi, L. et al., 2007, Cross-sectional [72]	A total of 690 children aged 7 to 18 years, mean age: N/R.	Dietary intake was assessed through a 58-item FFQ, including nuts consumption.	High consumption of nuts: (+) wheezing prevalence.
Du Toit, G. et al., 2015, Randomized trial [73]	In total, 640 infants with severe eczema, egg allergy, or both. Mean age: 7.8 ± 1.7 months.	Participants were stratified into two study cohorts based on the results of a skin-prick test for peanut allergy and were then randomly assigned to a group in which dietary peanuts would be consumed, or to a group in which its consumption would be avoided.	Peanut consumption: (=) asthma at 5 y.
Roduit et al., 2014, Cohort study [74]	A total of 856 children (from 3–12 months to 6 years old) Mean age: N/R.	Assessment of introduction of nuts in the first year of life.	Introduction of nuts in the first year of life: (=) asthma at 6 y.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; and FFQ, food frequency questionnaire. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.4. Fats and Fish

Dietary fat may modulate inflammatory responses through a variety of mechanisms [3,48,75]. Depending on the type and source of fat consumed, it may have positive [76] or negative [77] effects on asthma.

On the negative side, a high-fat diet may change the composition of the gut microbiota by altering the expansion and colonization of invasive bacteria, with a decrease in protective bacteria, and a decrease in SCFA concentration [3]. Saturated fatty acids can activate innate immune receptors including toll-like receptors (TLR)-4, resulting in altered transcription factor activity such as NF-kB, consequently stimulating the inflammatory cascade [3]. In addition, omega-6 (n-6) polyunsaturated fatty acids (PUFA) are also associated with pro-inflammatory responses, and are found specially in vegetable oils [78], which are typically linked with the western diet [3].

On the other side, omega-3 (n-3) PUFAs, namely eicosapentaenoic acid (EPA), and docosahexaenoic acid (DHA), that are found in oily fish and fish oil supplements [76], were found to be beneficial in animal models of asthma [76]. EPA and DHA lipid mediators have shown to possess anti-inflammatory and inflammation-resolving properties, such as limiting neutrophil infiltration or inhibiting pro-inflammatory cytokine production [78]. Furthermore, EPA and DHA appears to directly inhibit the production of pro-inflammatory cytokines by inhibiting the activation of the nuclear transcription factor NF-kB [78]. An imbalance in n-6 PUFA intake versus n-3 PUFA intake has been proposed as a contributing

factor to the rise in allergic diseases, that could be a consequence of *n*-6 PUFA's pro-inflammatory activity, which promotes a type 2 helper T lymphocyte immune response [78].

Concordantly, a cross-sectional study of adult asthma patients revealed a positive association between a high *n*-6:*n*-3 PUFA ratio with higher eNO values (β = 0.05, 95% CI 0.02; 0.09) and increased odds for uncontrolled asthma (OR = 3.69, 95% CI 1.37; 9.94) [79].

A study of subjects with severe persistent asthma found that, comparatively to healthy controls, they consumed more fat and less fiber, and these intakes were associated with lower forced expiratory volume in 1 s (FEV_1) and airway eosinophilia in asthmatics [48]. It was also observed that saturated fat intake was positively associated with sputum % eosinophils [48]. Another study observed that a single high-fat/high-energy meal increases neutrophilic airway inflammation and TLR4 mRNA expression in sputum cells and decreases bronchodilator responsiveness in patients with asthma [77]. However, the high-fat meals had a diverse nutritional profile, which included fats of various qualities as well as other macronutrients, which may have had an impact on the inflammatory response [77]. No changes in eNO levels were observed after the high-fat meal. However, as stated before, eNO is a marker of eosinophilic asthma, and as the inflammatory changes observed were increases in airway neutrophils, it is not surprising that eNO levels were left unaltered [77]. In the same study, another group of patients with asthma consumed a high-trans (*n* = 5; 5.2 g trans-fat) or non-trans (*n* = 5, <0.3 g trans-fat) fatty acid meal. After the high-trans fatty acid meal, sputum % neutrophils were found to be significantly higher than after the non-trans meal [77].

Higher ingestion of saturated fatty acid-rich foods, such as butter, appears to be associated with both a higher asthma symptom score and a higher degree of airway inflammation [55].

On the other side, the consumption of olive oil (a monosaturated rich type of fat) in a multi-case study of Italian adults, showed that intakes of oleic acid and of olive oil were associated with a reduced risk of current asthma. Individuals in the highest quartile of oleic acid intake had less than half the chance of having current asthma in comparison to those in the lowest quartile. Moreover, these authors uncovered that when considering olive oil intake as a continuous variable, the risk of having current asthma declined by 20% for an increase of 10 g/day in olive oil consumption [80]. Olive oil is also rich in phenolic compounds and may exert its protective effects through additional anti-inflammatory and antioxidants effects [80].

The consumption of fish (the principal source of *n*-3 PUFA) was found to be inversely associated to asthma in the previously referred study of 598 Dutch children [68], with the adjusted OR for current asthma being 0.34 (95% CI 0.13; 0.85) and for atopic asthma with bronchial hyperresponsiveness, 0.12 (95% CI 0.02; 0.66), respectively [68]. In another study, school children who consumed fish more often had less doctor-diagnosed asthma and less current asthma [81].

However, evidence of fish oil and fish benefits in asthmatic patients are inconsistent [82]. An inverse association between consumption and asthma risk seems to be more evident in children, as their immune system is still under development: a meta-analysis found that infant fish consumption was inversely related to the occurrence of asthma in childhood. When comparing the highest to lowest category of fish consumption, the pooled RR of asthma was 0.76 (95% CI, 0.61; 0.94) [82]. Two different studies from the previously referred meta-analysis examined the relationship between maternal fish consumption during pregnancy and the risk of asthma in offspring. However, as the exposures assessed in these studies were different, they did not combine their results through meta-analysis. In one of the studies, there was no significant association found between maternal fish consumption frequency during pregnancy and the development of asthma in offspring (OR = 1.01, 95% CI 0.85; 1.20) when comparing mothers who consumed fish more than once a week with those who consumed fish less than once a week. Similarly, the other included study reported that maternal plasma long-chain *n*-3 polyunsaturated fatty acid (LCn3PUFA) concentration was not associated with the risk of asthma in offspring [82]. However, the levels of LCn3PUFA

in maternal expressed breast milk (EBM) were found to have a negative association with the occurrence of asthma in offspring. When comparing the group with the highest levels of LCn3PUFA to the group with the lowest levels, there was a combined RR = 0.71 (95% CI, 0.52–0.96). This suggests that higher levels of LCn3PUFA in maternal EBM are associated with a reduced risk of asthma in the children [82]. In the same meta-analysis, no statistically significant associations were found in adults among these studies [82].

A more recent meta-analysis investigating the effects of fish consumption on asthma in children, found that "all fish" (lean and fatty) consumption had an overall beneficial effect on "current asthma" (OR = 0.75, 95% CI 0.60; 0.95) and "current wheeze" (OR = 0.62, 95% CI 0.48; 0.80) in children up to 4.5 years old. In children aged 8 to 14 years old, fatty fish consumption was found to be more protective than no fish consumption (OR = 0.35, 95% CI 0.18; 0.67) [83]. However, another meta-analysis found no significant association between fish intake in infancy and childhood asthma [84].

Meta-analyzes evaluating the effect of fish oil or *n*-3 PUFA supplementation have not found any effects on asthma incidence or asthma symptoms [85,86]. The lack of effects observed in most studies may be due to fish oil supplementation use, which may have a different health impact than eating the whole fish and all its properties [85]. Table 4 summarizes the results of research on fats and fish intake on asthma and asthma-related outcomes.

Table 4. Studies evaluating effects of fats and fish intake on asthma and asthma-related outcomes.

Fats and Fish			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Berthon BS et al., 2013, Cross-sectional [48]	In total, there were 202 participants, with 137 stable asthmatics, and 65 healthy controls. Mean age: healthy controls: 46.7 ± 17.4 y; intermittent, mild, and moderate persistent asthma: 54.5 ± 15.5 y; severe persistent asthma: 57.8 ± 14.4 y.	Dietary intake was estimated using a 186-item semi-quantitative FFQ.	Higher fat intake: (−) FEV1; (−) airway eosinophilia. Saturated fat: (−) airway inflammation.
Cardinale et al., 2007, Cross-sectional [55]	A total of 130 children with mild to moderate asthma (mean age: N/R).	Dietary intake was estimated using a 4-point food-consumption frequency questionnaire: 8 food items (butter, margarine, milk, tomatoes, fresh fruit, cooked vegetables, salad, and nuts).	Higher butter intake: (−) FENO levels; (−) clinical score severity of asthma.
Tabak et al., 2006, Cross-sectional [68]	In total, there were 598 children, with 39 asthmatics. Mean age: 10.4 ± 1.2 y. Seventy-two adults, with fifty-one asthmatics.	Dietary intake was estimated using a semi-quantitative FFQ.	Fish Intake: (+) asthma prevalence.
Wood LG et al., 2011, Randomized controlled trial [77]	Mean age ± SEMs: Healthy controls high-fat: 49.6 ± 4.6 y. Asthma low-fat/low-energy: 41.7 ± 3.2 y. Asthma-high-fat/high-energy, nonobese: 50.9 ± 4.3 y. Asthma high-fat/high-energy, obese: 56.5 ± 4.3 y.	High-fat/high meal challenge; high-trans (n = 5) or non-trans (n = 5) fatty acid meal challenge.	High-fat/high-energy meal: (−) airway inflammation; (−) bronchodilator responsiveness. High-trans fatty acid meal: (−) airway inflammation.
Cazzoletti L et al., 2019, Cross-sectional [80]	A total of 871 subjects, with 145 asthmatics. Mean age: controls: 51.5 ± 11.5 y. asthmatics: 49.5 ± 11.7 y.	Food intake was collected using an FFQ.	Oleic acid and of olive oil intake: (+) asthma prevalence.

Table 4. *Cont.*

Fats and Fish			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Barros R et al., 2011, Cross-sectional [79]	A total of 174 adult asthmatics. Mean age ± SD: 40 ± 15 years.	Dietary intake was obtained by an FFQ.	High *n*-6:*n*-3 PUFA ratio: (−) airway inflammation; (−) asthma control.
Kim JL et al., 2005, Cross-sectional [81]	In total, there were 1014 children, with 78 asthmatics. Median age: 9 (range 5–14 years).	Current consumption and frequency of fish.	Fish consumption: (+) asthma prevalence.
Yang H et al., 2013, Meta-analysis [82]	Adults and children, papers included in the meta-analysis (*n* = 8), consisting of prospective cohort studies (*n* = 8).	Fish and fish oil intake.	Fish consumption: (+) asthma prevalence in children; (=) asthma prevalence in adults.
Papamichael MM et al., 2018, Meta-analysis [83]	Children, papers included in the meta-analysis (*n* = 23): cross-sectional studies (*n* = 12); case-control studies (*n* = 2); cohort studies (*n* = 9).	Fish intake.	All fish (lean and fatty) consumption: (+) asthma prevalence; (+) wheeze prevalence in children up to 4.5 y. Fatty fish consumption: (+) asthma prevalence in children 8 to 14 y.
Zhang GQ et al., 2017, Meta-analysis [84]	Papers included in the meta-analysis (*n* = 19): cohort studies (*n* = 8) for fish intake in infancy.	Fish intake during pregnancy or infancy.	Fish consumption: (=) asthma prevalence.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; FFQ, food frequency questionnaire; FEV1, forced expiratory volume in 1 s; PUFA, polyunsaturated fatty acids; TLR, toll like receptor; mRNA; and microRNA. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.5. Salt

Dietary sodium having an influence on asthma was first proposed in 1987 based on a correlation observed between asthma mortality and table salt purchases [87]. Dietary changes associated with the development of a different, more rushed, lifestyle have been identified as one of the environmental factors that may have contributed to the rise in the prevalence of asthma, with sodium being identified as a dietary constituent that may be implicated in this phenomenon [88]. Data from a cross-sectional study found a lower potassium and a higher sodium intake in severe asthmatics patients compared to healthy controls [48]. It has been proposed that a high sodium intake may lead to hyperpolarization of the bronchial smooth muscle, causing asthma exacerbation [48], and dietary sodium intake has been shown to be positively associated with airway responsiveness [89]. However, a cross-sectional study did not corroborate these findings, as it found no significant associations between 24 h sodium urinary excretion samples and airway reactivity to methacholine in adults [90].

A meta-analysis of six randomized controlled trials that aimed to assess the effect of a dietary sodium reduction in patients with asthma found no evidence of an effect of salt intake on lung function measures in asthmatic adults [91]. Nonetheless, this systematic review suggested that there is a pattern of a small improvement in airway function and a small reduction in bronchodilator use with low-salt diets [91]. Another meta-analysis aiming to observe the effects of salt on asthma control also found no benefits of salt manipulation on asthma [92]. There was, however, some evidence that a low-sodium diet may improve lung function after exercise and possibly baseline lung function, according to the evidence from exercise-induced asthma studies, but these results were based on

findings obtained from a very small number of participants [92] Table 5 summarizes the results of research on salt intake on asthma and asthma-related outcomes.

Table 5. Studies evaluating effects of salt intake on asthma and asthma- related outcomes.

Salt			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Ardern KD et al., 2004, Systematic Review [91]	Studies included in the systematic review (n = 6). Six RCTs were included in the review.	Salt intake.	Dietary salt reduction: (=) symptoms of allergic asthma.
Pogson Z et al., 2011, Systematic Review [92]	Studies included in the systematic review (n = 9): nine RCTs in relation to sodium manipulation and asthma, of which five were in people with asthma (318 participants), and four in people with exercise-induced asthma (63 participants).	Salt intake.	Dietary salt reduction: (+) lung function; (+) symptoms of asthma.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; and RCTs, Randomized control trial. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.6. Dairy Products

The 2015–2020 Dietary Guidelines for Americans recommend consuming three cups of dairy per day [63]. However, a case-control study compared children in the highest quartiles of dairy intake (Q3–4; OR = 1.72, 95% CI 1.19; 2.49) with children in the lowest quartiles (Q1–2; OR < 1), and found a positive association between frequent dairy consumption and odds of developing asthma (OR = 1.93, 95% CI 1.32; 2.84) [69].

A different study demonstrated that early life exposure to unpasteurized milk seems to exert a protective effect on asthma [93,94], that may be related to its bacterial composition [93] or protein components [94].

A community-based cross-sectional study of Australian young adults found that drinking whole milk was negatively associated with increased odds of doctor-diagnosed asthma (OR = 0.73, 95% CI 0.54; 0.99), while consumption of low-fat cheese was found to be positively associated (OR = 1.50, 95% CI 1.03; 2.19). Similarly, ricotta cheese intake was positively associated with current asthma (OR = 1.94, 95% CI 1.18; 3.19) [95].

Although the mechanisms by which dairy products may influence the development of asthma are unknown, there was a positive association observed between dairy consumption and concentrations of pro-inflammatory IL-17F. The authors suggest the existence of an IL-17F-dependent inflammatory pathway that can play a role in the development of asthma [69].

Table 6 summarizes the results of research on dairy intake on asthma.

Table 6. Studies evaluating effects of dairy intake on asthma.

Dairy			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Han YY, et al., 2015, Case-control [69]	A total of 678 children, with 351 asthmatics. Mean age: controls: 10.5 ± 2.7 y. asthma: 10.0 ± 2.6 y.	Dietary intake was estimated using a 75-item questionnaire on the child's food consumption, including dairy, in the prior week.	Frequent consumption of dairy products: (−) asthma prevalence.

Table 6. *Cont.*

Dairy			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Sozańska B, et al., 2013, Cross-sectional [93]	In total, there were 1700 children, with 78 asthmatics. Mean age: N/R.	Assessment of consumption of unpasteurized milk in the first year of life.	Consumption of unpasteurized milk: (+) asthma prevalence.
Loss G, et al., 2011, Cross-sectional [94]	In total, there were 8334 school-aged children, with 2033 asthmatics. Mean age: N/R.	A comprehensive questionnaire about farm milk consumption was completed by the parents.	Consumption of farm milk: (+) asthma prevalence.
Woods RK, et al., 2003, Cross-sectional [95]	In total, 1601 young adults were included, with 180 asthmatics. mean age: 34.6 ± 7.1 y.	Semiquantitative FFQ, which assessed their habitual food intake over the preceding 12 months.	Whole milk consumption: (+) asthma prevalence. Consumption of low-fat cheese: (−) asthma prevalence. Ricotta cheese intake: (−) asthma prevalence.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; and FFQ, food frequency questionnaire. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

4.7. Dietary Patterns

As reviewed above, diet is an important source of nutrients and other food components that have a variety of properties that may modulate the risk of asthma, as well as other chronic respiratory diseases [96,97].

Most studies have focused on the effects of individual components or nutrients to investigate the impact of diet on asthma [3,4,98]. However, findings linking specific dietary components and eating patterns to altered airway inflammation, asthma, and lung function have rarely been consistent in human studies [76,82,85,86,91,92,99]. Considering that foods are ingested as complex combinations, which include nutrients and bioactive components, which interact with each other and the food matrix, recent efforts have concentrated on researching validated indices that assess the properties of the entire diet influencing metabolic and health effects [3,4,30,53,98].

Scores that aim to classify diet quality, such as the Healthy Eating Indexes (HEI), as well as dietary patterns such as the Mediterranean Diet (MD) and the Dietary Approaches to Stop Hypertension (DASH), mostly highlight the consumption of fruits and vegetables, whole grains, and unsaturated fat sources, recommending a lower intake of saturated fats, sodium, and refined grains [4,100–102]. A study using the HEI-2015, which is a measure to assess diet quality, specifically to which the dietary pattern aligns with the Dietary Guidelines for Americans, comparing dietary patterns between adults with asthma with and without asthma-related emergency room (ER) admissions, found that individuals with asthma had low vegetable and fruit intakes as well as a low HEI-2015 score (mean ± SE 52.6 ± 0.53), and those with asthma-related ER visits consumed less vegetables compared to those without (median 0.61 cup vs. 0.85 cup equivalents) [103].

Ma et al. conducted a pilot randomized controlled trial of a 6-month behavioral intervention promoting the DASH diet, which includes the intake targets for total fat, saturated fat, protein, cholesterol, fiber, magnesium, calcium, sodium, and potassium, in patients with uncontrolled persistent asthma. When compared to usual care, this intervention produced greater improvements in diet quality, asthma control, and asthma-related quality of life [104].

As for children, the evidence from eight observational studies on Mediterranean diet exposure and its association with lower asthma prevalence was examined in a systematic review. The definitions “current wheeze”, “current severe wheeze”, and “asthma ever” were used. When all studies were considered, the results of the meta-analysis revealed

that an higher adherence to the Mediterranean diet during childhood is a protective factor for "current wheeze" and "asthma ever", but not for "severe current wheeze" [105]. Another systematic review reported that increased adherence to the Mediterranean diet was negatively associated with asthma symptoms in children [106]. More recently, a systematic review indicated a protective role of the Mediterranean diet on childhood asthma [107]. On the other side, school-aged children, who follow a strict Western diet high in total and saturated fat and processed foods, seem to be at a higher risk of asthma [33].

Tarazona-Meza et al. [108] found in a cross-sectional study that better diet quality was associated with a lower odds of having asthma (OR = 0.83, 95% CI 0.72; 0.95) in children and adolescents [108]. Lower values in the Revised Brazilian Healthy Eating Index score, representing lower dietary quality, both at 18 and 22 years old, increased the odds of wheezing in the previous year, with an OR = 1.97, 95% CI 1.33; 2.91, and OR = 1.98, 95% CI 1.36; 2.87, respectively, in a longitudinal study. On the other side, remaining on a poor diet from ages 18 to 22 increased the odds of chest wheezing by more than three-fold (OR = 3.28; 95% CI 1.84; 5.84) compared to staying on a high-quality diet [109].

Findings from the PARIS birth cohort demonstrated that children in the higher tertile group of adherence to the Mediterranean diet, considered to have the higher diet quality, compared to children from the lowest tertile group, had a lower risk of having current asthma (aOR = 0.28, 95% CI 0.12; 0.64) [110]. Furthermore, adherence to the Mediterranean diet may modulate the production of several asthma inflammatory mediators: a higher adherence to this diet pattern was associated with lower IL-4 and IL-17 in asthmatic children [111].

Accordingly, a randomized controlled trial revealed that a Mediterranean diet supplemented by two meals of 150 g of cooked fatty fish weekly for six months, compared to the usual diet, reduced airway inflammation as assessed by eNO ($\beta = -14.15$ ppb, 95% CI -27.39; -0.91) in childhood asthma [112].

A study that observed associations between three different dietary scores with asthma symptoms and asthma control in French adults, found that higher dietary scores assessed by the Alternative Healthy Eating Index 2010 (AHEI-2010), the Mediterranean diet based on the literature (MEDI-LITE), and modified *Programme National Nutrition Santé* Guideline Score were associated with a lower asthma symptom score [4]. However, when analyzing the association of a higher diet quality with asthma symptom score according to BMI, some of the statistically significant associations were lost when BMI is $\geq$25 and <30 kg/m^2, and most were lost when BMI is $\geq$30 kg/m^2 [4].

Individuals with obesity may present higher circulating concentrations of many inflammatory markers as a consequence of the disease [39], and GINA guidelines recommend weight loss as part of the strategy of asthma management in obese patients [2]. Knowing that obesity is a strong risk factor for asthma, having a higher diet quality may not be enough to counterbalance the negative effects that are associated with being overweight and obese. A systematic review found that, for obese asthmatic adults, the more effective dietary intervention seems to be energy restriction, regardless of the specific dietary components or dietary pattern [40].

A meta-analysis looked at the link between high adherence to the Mediterranean diet during childhood and the risk of asthma and wheezing in children, and although there is a trend towards high adherence to the Mediterranean diet in childhood to prevent current wheeze in later life, no inverse relationship was found for asthma (OR = 0.87, 95% CI 0.72; 1.04) or severe asthma (OR = 0.97, 95% CI 0.89; 1.06) [113]. Accordingly, the US-based Nurses' Health Study revealed that high AHEI-2010 scores were not significantly associated with a reduced risk of adult-onset asthma [114]. Visser et al. also found no effect of higher diet quality on adult-onset asthma in a cohort study [115]. Another research, in Hispanic adults, with the purpose of seeing whether a pro-inflammatory diet (as measured by the energy-adjusted dietary inflammatory index [E-DII]) or a high dietary quality (as measured by the AHEI-2010) is associated with current asthma, current asthma symptoms, and lung function, observed that a higher E-DII score (representing a more pro-inflammatory diet)

was associated with current asthma (OR for quartile 4 vs. 1: 1.35, 95% CI 0.97; 1.90) and asthma symptoms (OR for quartile 4 vs. 1: 1.42, 95% CI 1.12; 1.81). However, the AHEI-2010 score was not significantly associated with any of the outcomes [116]. E-DII is composed of 45 individual food parameters, mostly micro- and macronutrients, which are scored to characterize their inflammatory potential (but only 29 parameters were available and used in the study by Han et al.). Points are assigned to each of these parameters according to whether they increase, decrease, or have no effect on six biomarkers of inflammation [116]. As for AHEI-2010, this index is a tool used to evaluate the quality of an individual's diet, consisting of eleven components that contribute to the overall classification of diet quality. These components include the consumption of vegetables (excluding potatoes), whole fruits (not including fruit juice), whole grains, sugar-sweetened beverages and fruit juices, nuts and legumes, red and processed meats, trans fats, long-chain omega-3 fats, PUFA, sodium, and alcohol. Each component was assigned a score ranging from 0 (inadequate intake) to 10 (optimal intake). Scores of all components were then summed, resulting in a total score that ranged from 0 to 110. Higher scores indicated healthier eating habits [116].

The E-DII may be a more adequate index to characterize a pro-inflammatory diet, and as asthma is an inflammatory disease, the observed significant association of a higher E-DII with asthma may reflect the capacity of the index to identify components that contribute to asthma risk. Han et al. refer that E-DII may be a better indicator of dietary patterns leading to airway inflammation than the AHEI-2010 [116].

Table 7 summarizes the results of research on dietary patterns on asthma and asthma-related outcomes.

Table 7. Studies evaluating effects of dietary patterns on asthma or asthma-related outcomes.

Dietary Patterns			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Andrianasolo RM, et al., 2018, Cross-sectional [4]	A total of 34,766 adults were included, mean age by tertiles of the Alternate Healthy Eating Index 2010 (AHEI-2010): Women: 1st: 46.7 ± 13.4; 2nd: 53.5 ± 13.1; and 3rd: 57.3 ± 12.4. Men: 1st: 55.8 ± 14.2; 2nd: 60.2 ± 13.0; and 3rd: 62.5 ± 12.0).	Quality of diet was evaluated by three dietary scores: the AHEI-2010, the literature-based adherence score to Mediterranean diet (MEDI-LITE), and the modified Programme National Nutrition Santé Guideline Score (mPNNS-GS) through FFQ.	Higher dietary scores assessed by AHEI-2010, MEDI-LITE, and mPNNS-GS: (+) asthma symptom score.
Patel S, et al., 2014, Cohort study [33]	1252 children. Follow-up age 8, mean age: 7.98 ± 0.17. Follow-up age 11, mean age: 11.5 ± 0.54.	Dietary Patterns were obtained through FFQ: 'Traditional'—mostly fruit and vegetables with meat and oily fish 'Western'—mostly processed foods, which are associated with a modern Western diet (chips, crisps, and pizza) 'Other'—mostly items that are eaten by people following a vegetarian diet (lentils, soya, rice, and nuts) but also contained fried foods, offal, and pastry dishes.	Following a strict western diet: (−) asthma prevalence.
Zhang Y et al., 2021, Cross Sectional [103]	A total of 1681 individuals, mean age: N/R.	2015 Healthy Eating Index (HEI-2015).	Low fruit and vegetable intake and low mean ± SE HEI-2015 score: (−) asthma prevalence.
Ma J et al., 2016, Pilot randomized trial [104]	Ninety adults, mean age: 51.8 ± 12.4 y.	6-month behavioral intervention promoting the DASH diet in patients with uncontrolled persistent asthma.	Adherence to DASH diet: (+) asthma control; (+) asthma-related quality of life.

Table 7. *Cont.*

Dietary Patterns			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Garcia-Marcos L et al., 2013, Systematic review and meta-analysis [105]	Children, papers included in the meta-analysis (n = 8), comprising cross-sectional studies (n = 8).	Mediterranean diet.	Higher adherence to the Mediterranean diet during childhood: (+) wheeze prevalence; (+) asthma prevalence; (=) severe current wheeze prevalence.
Papamichael MM et al., 2017, Systematic review [106]	Children Papers included in the systematic review (n = 15): cross-sectional studies (n = 11); intervention trial (n = 1); case-control studies (n = 1); cohort studies (n = 2).	Mediterranean diet.	Following a Mediterranean dietary pattern: (+) asthma prevalence.
Tarazona-Meza CE et al., 2020, Cross-sectional study (nested in a case-control study) [108]	A total of 767 children and adolescents were included (573 with asthma and 194 controls), mean age: 13.8 ± 2.6 y	Diet was assessed using an FFQ, with food groups classified as "healthy" or "unhealthy".	Better diet quality: (+) asthma prevalence.
Menezes AMB et al., 2020, Cohort study [109]	In total, there were 2986 young individuals. 1st evaluation: 18 years. 2nd evaluation: 22 years.	Longitudinal study with follow-up information from 18- and 22-year-olds. Diet quality was measured with a revised version of the Healthy Eating Index (IQD-R) for the Brazilian population at 18 y and 22 y with FFQ referring to the last 12 months.	Higher values in the Revised Brazilian Healthy Eating Index score, both at 18 and 22 y: (+) wheezing prevalence. Remaining on a poor diet from age 18 to 22 y: (−) wheezing prevalence.
Amazouz H et al., 2021, Cross sectional study [110]	In total, 975 school-aged children were included, age of evaluation: 8 y.	Adherence to the MD was assessed with FFQ and based on two scores: the KIDMED index and the Mediterranean Diet Score (MDS).	Higher tertile group of adherences to the Mediterranean diet: (+) asthma prevalence.
Koumpagioti D et al., 2022, Systematic review [107]	Children, papers included in the systematic review (n = 12): cross-sectional studies (n = 7); randomized controlled trial (n = 1); case-control studies (n = 1); cohort studies (n = 1).	Adherence to MD was measured by diet quality indices as KIDMED and MDS.	Adherence to MD: (+) asthma prevalence.
Douros K et al., 2019, Cross-sectional study [111]	Seventy children, mean age: asthmatic: 8.9 ± 2.4 y. Non-asthmatic: 8.6 ± 2.1 y.	Adherence to MD was estimated with the Mediterranean Diet Quality Index for children and adolescents (KIDMED) score	Higher adherence to MD: (+) IL-4 and IL-17 values.
Papamichael et al., 2019, Randomized controlled trial [112]	Seventy-two asthmatic children, mean age: 7.98 ± 2.24 y.	A single-centered, 6-month, parallel randomized controlled trial comparing the consumption of a Mediterranean diet supplemented with two meals of 150 g of cooked fatty fish weekly (intervention) with the usual diet (control).	Mediterranean diet supplemented with two meals of 150 g of cooked fatty fish: (+) airway inflammation.
Zhang Y et al., 2019, A systematic review and meta-analysis [113]	Papers included in the meta-analysis (n = 16): cross-sectional studies (n = 12); case-control studies (n = 1); cohort studies (n = 5). A total of 34,698 controls, with 477 incidents of asthma.	Adherence to the Mediterranean diet	Adherence to MD: (=) asthma prevalence; (=) severe asthma prevalence.
Visser et al., 2023 Cohort study (105)	Mean age: BMI < 25 kg/m^2: cases: 40.4 ± 13.3 y, Controls: 44.3 ± 12.6 y BMI ≥ 25 kg/m^2: cases: 46.8 ± 12.5 y, Controls: 49.1 ± 12.0 y	Diet quality-assessed by the Lifelines Diet Score and Mediterranean Diet Score	Higher diet quality: (=) adult-onset asthma prevalence.

Table 7. *Cont.*

Dietary Patterns			
Author, Year, Study Design	**Sample, Age**	**Dietary Assessment/Intervention**	**Results**
Varraso et al. (2015), Cohort study [114]	A total of 73,228 adults. Mean age according to fifths of AHEI-2010: 1st: 48.6 ± 7.2; 2nd: 49.5 ± 7.2; 3rd: 50.3 ± 7.1; 4th: 51.0 ± 7.0; 5th: 52.1 ± 6.8.	Scores of AHEI-2010, assessed by FFQ.	High AHEI-2010 scores: (=) adult-onset asthma prevalence.
Han et al. (2020), Cross-sectional study [116]	A total of 12,687 adults, with 962 asthmatics included. Mean age: no current asthma: 41.4 ± 0.3 y. current asthma: 43.6 ± 0.8 y.	The energy-adjusted Dietary Inflammatory Index [E-DII]) and AHEI-2010 were calculated based on two 24 h dietary recalls.	Higher E-DII score: (+) asthma prevalence; (+) asthma symptoms. AHEI-2010 score: (=) asthma prevalence; (=) asthma symptoms.

Notes: Age is described as mean age ± standard deviation (SD) in years, unless stated otherwise. N/R, Not reported; AHEI-2010, Alternative Healthy Eating Index-2010; MEDI-LITE, literature-based adherence score to Mediterranean diet; mPNNS-GS, modified Programme National Nutrition Santé Guideline Score; FFQ, food frequency questionnaire; HEI-2015, 2015 Healthy Eating Index; DASH, Dietary Approaches to Stop Hypertension; IQD-R, revised version of the Healthy Eating Index; KIDMED, Mediterranean Diet Quality Index for children and adolescents; MDS, Mediterranean Diet Score; and E-DII, energy-adjusted Dietary Inflammatory Index. (+) stands for significant and positive effects on asthma outcomes; (=) stands for no significant effects on asthma outcomes; and (−) stands for negative effects on asthma-related outcomes.

An overall view of the different dietary component's effects and its interaction in relationship with inflammation are presented in Figure 3.

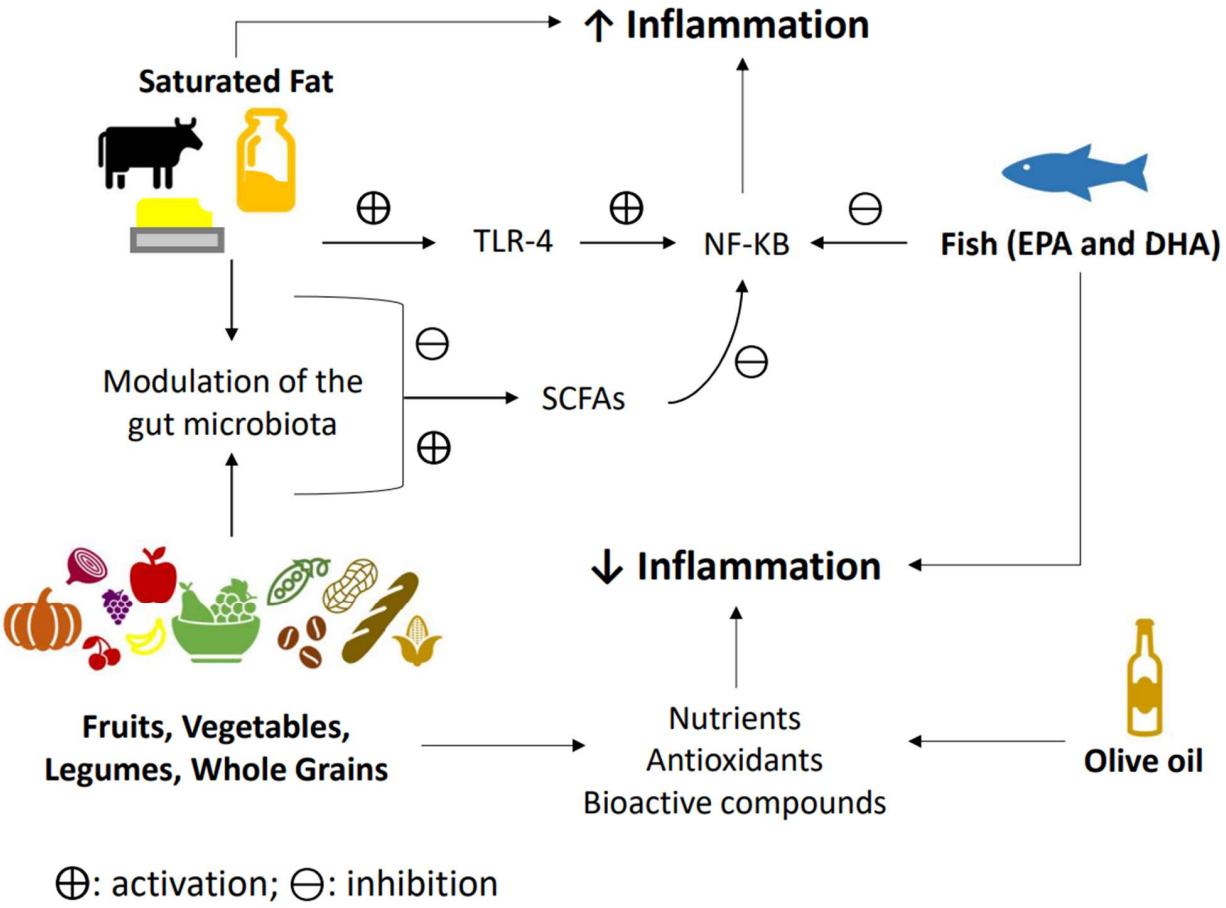

Figure 3. Diet and inflammation. The western dietary pattern promotes a pro-inflammatory environment due to a lack of consumption of antioxidants rich foods such as fruits and vegetables,

leading to an increase in oxidative stress and inflammation. Fruits, whole grains, vegetables, legumes, and olive oil all have antioxidants properties that may lower inflammation. On the other side, a high intake of saturated fatty acids seems to be able to activate the innate immune system through the toll-like receptor-4 (TLR4), consequently stimulating the nuclear factor kappa-light-chain-enhancer of activated B cells (NF-kB) inflammatory cascade, while also dysregulating the microbiota, and consequently leading to a decrease in short-chain fatty acid production, thereby augmenting inflammation. Fruits, vegetables, legumes, and whole grains, on the contrary, are fiber-rich foods that can modulate microbiota, leading to an increase in short chain fatty acids (SCFAs) that seem to inhibit NF-kB function. Eicosapentaenoic acid (EPA) and docosahexaenoic acid (DHA) appear to be able to inhibit the production of proinflammatory cytokines by inhibiting NF-kB activation. ⊕: activation; and ⊖: inhibition.

5. Discussion

The relationship between diet and asthma is complex and multifactorial. Fruits and vegetables have been shown to exert beneficial effects on asthma and airway inflammation [47,56,62]. It is also worth noting the significance of dietary diversity, especially in terms of consuming a variety of vegetables, as a relevant factor to consider for asthma [56]. The positive effects of fruits and vegetables on asthma is likely attributed to their high content of polyphenols, vitamins, minerals, bioactive compounds, and fiber [47,56,62]. Taking this into consideration, the inclusion of vegetables and fruits in the dietary pattern emerges as a preventive measure against asthma [61,62]. However, it is important to emphasize the significance of incorporating a diverse range of vegetables and fruits to maximize their potential benefits [117]. In addition to their potential protective effects against the development of asthma, these food groups have also shown promise in managing asthmatic symptoms. Studies have indicated that a higher consumption of fruits and vegetables is associated with improved asthma control [59] as well as better lung function parameters [54]. Therefore, advocating for the inclusion of a wide variety of vegetables and fruits in the diet holds significant potential for both asthma prevention and management strategies. Fiber has also been found to exert a protective effect on airway inflammation and on asthma [37,68]. This association is also reinforced as many fiber-rich foods contain antioxidants and bioactive compounds, which may help to reduce oxidative stress and inflammation [48]. Overall, incorporating fiber-rich foods into the diet may be a beneficial dietary intervention for individuals with asthma, potentially reducing airway inflammation and improving lung function. However, further research is needed to fully understand the mechanisms by which fiber exerts its protective effects, and to identify the optimal amount and types of fiber for asthma management.

In contrast, the effects of dietary fats on asthma are more nuanced [76,77]. Saturated and trans fats have been linked to negative health outcomes, including an increased risk of poor asthma disease outcomes [48,77]. On the other hand, monounsaturated and polyunsaturated fats, such as those found in olive oil (oleic acid) and fatty fish (EPA and DHA), have been associated with a decreased risk of asthma and inflammation [79,80]. However, the effects of omega-3 and fish oil supplementation on asthma are less clear. While fatty fish, the primary source of omega-3 fatty acids, has been shown to have a beneficial effect on this disease, the same is not necessarily true for omega-3 or fish oil supplements [85,118]. Whole fish contains a variety of nutrients and bioactive compounds, such as proteins, vitamins, minerals, and omega-3 fatty acids, which work together synergistically to provide health benefits [85,118]. Fish oil supplements typically provide isolated forms of EPA and DHA that lack the complex mixture of nutrients and other bioactive compounds present in whole fish [85]. It is possible that the components of the whole fish work in concert to provide optimal health benefits and optimal absorption of EPA and DHA [119]. Moreover, the context in which fish has been consumed, such as in the Mediterranean diet or other healthy patterns, often includes a variety of other beneficial foods that contribute to overall health. While more research is still needed to investigate the benefits of fish oil supplements, obtaining nutrients through a varied and balanced diet that includes whole fish is generally

considered the preferred approach for maximizing health benefits. Additionally, in terms of maternal fish consumption, two studies found no significant association between the frequency of fish consumption during pregnancy and the risk of asthma in children [82]. However, a different study focusing on the levels of LCn3PUFA in maternal EBM has revealed that higher levels of LCn3PUFA in maternal EBM were significantly associated with a reduced risk of asthma in children [82]. These insights contribute to the growing body of evidence supporting the importance of maternal nutrition in early-life programming and the prevention of childhood asthma. Further research is necessary to better understand the mechanisms underlying these associations, and to develop targeted interventions that promote optimal maternal intake for reducing the risk of childhood asthma.

As for salt intake, it appears that high consumptions of this component can lead to airway inflammation and increased airway hyperresponsiveness [48,89]. Nevertheless, a systematic review and meta-analysis found no evidence of an effect of salt intake on the disease [91,92], although a pattern of a small improvements in airway function and a small reduction in bronchodilator use with low-salt diets was observed [91]. While the evidence on the negative effects of salt intake on asthma is not definitive, there is a lack of evidence to support any positive effects of high salt consumption on asthma. Therefore, reducing salt intake may be a beneficial strategy for individuals with asthma, even though the exact relationship between salt intake and asthma requires further investigation.

Dairy consumption, on the other hand, has been a topic of debate in relation to asthma. Some studies have suggested a positive association between frequent dairy consumption and the development of asthma [69], and a positive association between consumption of low-fat cheese and ricotta cheese and current asthma has also been described [95]. Nevertheless, not all dairy products have a negative impact on asthma. Early life exposure to unpasteurized milk has been shown to have a protective effect on asthma, potentially due to its bacterial composition or protein components [93,94]. Additionally, drinking of whole milk was found to be negatively associated with doctor-diagnosed asthma [95].

The HEI-2015, an index of diet quality, considers dairy as a positive food component of a healthy diet [120]. The 2015–2020 Dietary Guidelines for Americans recommend consuming 3 cups of dairy per day [63]. However, when it comes to asthma, the specific recommendations regarding dairy intake are still under research. Further investigation is needed to better understand the relationship between dairy consumption and its potential impact on asthma prevention and management, as different types of dairy products may exert different effects.

As for dietary patterns, the evidence presented implies that they can play a significant role in the development and management of asthma and airway inflammation. Higher intake of fruits and vegetables, whole grains, healthy fats, and adherence to diets that are rich in the mentioned food groups, such as the high-quality Mediterranean diet, has been associated with a lower prevalence of asthma, airway inflammation, and better asthma control or symptoms [4,100–102,108,109]. Moreover, adherence to the Mediterranean diet modulates the production of several inflammatory mediators known to have a pathogenetic role in asthma [112]. Conversely, diets high in total and saturated fats, and low in dietary fiber, fruits, and vegetables, as typical of current western diets, have been associated with negative asthma-related outcomes [33,121]. It has even been demonstrated that higher dietary acid loads, usually present in a western diet, may be positively associated with asthma [30,53].The additional and synergistic effects of combining the overall nutrient and phytochemical content acquired from various food matrices and overall diet with higher quality, may explain the inverse associations observed between several of the dietary patterns and asthma. These effects may favor the antioxidant and anti-inflammatory activities, which may act through different pathways, for instance by exerting antioxidant properties, suppressing or inducing metabolic gene expression, activating transcription factors that antagonize inflammation, and inhibiting the production of pro-inflammatory cytokines [122]. However, it should be noted that not all dietary indexes may be suitable for characterizing a diet that can effectively prevent or increase the risk of asthma and

airway inflammation [116]. It is possible that there are specific dietary indexes that better capture the anti-inflammatory and antioxidant effects of dietary patterns, which could be followed as preventive measures against asthma. Further research is needed to identify and validate these specific dietary indexes that can provide a more accurate representation of the potential preventive effects of diet on asthma and related airway inflammation.

Additionally, when investigating the intricate relationship between diet and asthma, it is crucial to consider other environmental factors that can influence this interplay. These factors include breastfeeding, duration of breastfeeding, maternal smoking during pregnancy, the child's body mass index, parental education, family income, total energy intake, physical activity levels, and exposure to pollution, etc. Researchers, by adjusting their analyzes for these confounding variables, may provide a more comprehensive understanding of the relationship between diet and asthma.

This review highlights the crucial role of promoting a dietary pattern that prioritizes the consumption of nutrient-rich foods such as fruits, vegetables, and other fiber-rich foods such as whole grains, as well as healthy fats. These foods provide a wide array of vitamins, minerals, antioxidants, and dietary fiber that are essential for inflammation prevention. Additionally, to achieve a healthy dietary pattern, it is also important to limit the intake of saturated fats, trans fats, and sodium. By combining this dietary approach, individuals can improve their nutritional intake and reduce the risk of chronic disease. Nonetheless, there are still significant gaps in the interpretation of the types of foods that the general population should incorporate to improve their respiratory health, and it is still unknown what dietary pattern is more adequate to follow to prevent or manage asthma.

6. Limitations and Strengths

In this narrative review, we aimed to summarize the existing evidence of dietary influences in asthma, and how food groups and dietary patterns are associated with asthma. As such, we selected and described several of the most recent and higher-quality studies within this research line to examine this broad topic. Nonetheless, as this is a narrative review, we only provided a critical consideration and a description of the current knowledge on the subject without conducting a detailed search strategy or objective assessment of the study quality. However, we reported data from systematic reviews and meta-analyzes, which have the highest level of evidence, all while searching for studies on specific food groups or dietary patterns where meta-analyzes and systematic reviews still need to be performed.

7. Conclusions

Overall, the evidence suggests that adherence to a healthier and higher-quality diet pattern can protect against asthma and airway inflammation. This review underlines the importance of promoting a diet that is diverse, rich in fruits, vegetables, whole grains, healthy fats, and overall nutrient and fiber-rich, while at the same time low in saturated fats, trans fats, and sodium. Dietary interventions promoting healthier eating patterns may be a valuable strategy in managing and preventing asthma while focusing on an adequate/moderate consumption of specific food groups.

8. Future Directions

The role of diet in asthma is an area of raising interest and importance. While the evidence suggests that certain dietary patterns, nutrients, and specific food groups may have a protective effect on asthma and asthma outcomes, more research is needed to confirm these findings and determine the underlying mechanisms involved. Additionally, the optimal diet for asthma prevention and management remains to be determined. Further investigations are needed to develop evidence-based dietary recommendations, as there are significant gaps in the interpretation of the types of foods or diets that patients should incorporate to improve their health.

Longitudinal studies are needed to establish the causal relationships between diet and asthma-related outcomes, and randomized controlled trials are required to assess the efficacy of dietary interventions in preventing and managing asthma. The potential interactions between diet and other asthma risk factors, such as environmental exposures, weight, and genetics, should also be explored. Other factors, such as dietary diversity and the potential dietary acid load of a dietary pattern, should also be examined when exploring this complex interplay between asthma and diet.

Understanding the potential effects of food intake on asthma and airway inflammation might help to introduce clinical dietary guidelines and public health recommendations for asthma prevention and management. Identifying modifiable dietary factors that can reduce the risk of asthma or improve asthma-related outcomes could lead to more personalized dietary recommendations and improved health outcomes for individuals with asthma.

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