Effect of Natural Functional Foods on Oxidative Stress and the Development of MASLD in HIV-Positive Individuals

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Abstract: The effect of natural functional foods on oxidative stress in HIV-positive patients is summarized in this review, with particular attention to the implications for metabolic dysfunction-associated steatotic liver disease (MASLD). Since chronic liver disease, especially MASLD, is a prominent cause of death among HIV patients, the global prevalence of HIV continues to provide serious public health issues. Insulin resistance, lipid buildup, oxidative stress, and mitochondrial dysfunction are some of the many factors contributing to the pathophysiology of MASLD. In particular, oxidative stress interferes with regular cellular metabolism, resulting in fibrosis, inflammation, and hepatocellular cancer. Antioxidants have demonstrated promise in lowering oxidative stress and enhancing liver health. They can be found in various of functional foods, including salmon, pitaya, and specific plant-based and microbial sources. However, further research is needed to determine the precise therapeutic effects and mechanisms of action of these functional foods. Although there is encouraging evidence that antioxidant-active functional foods can help manage MASLD in persons living with HIV (PLWH), more thorough research is required to confirm their effectiveness and comprehend their underlying mechanisms.

Keywords: Metabolic Dysfunction-Associated Steatotic Liver Disease; Oxidative Stress; Antioxidant; HIV; Functional Foods

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1 Introduction

First identified in 1981, human immunodeficiency virus (HIV) continues to represent one of the most significant global public health challenges, with approximately 39.9 million individuals living with the virus by the end of 2023, according to the World Health Organization (WHO) [1, 2]. In addition to immunological decline, HIV infection predisposes individuals to a wide range of comorbidities, including chronic liver diseases. Among

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these, metabolic dysfunction-associated steatotic liver disease (MASLD), a newly defined entity replacing the traditional concept of non-alcoholic fatty liver disease (NAFLD), has gained attention due to its high prevalence and associated risks in people living with HIV (PLWH) [3]. Unlike NAFLD, which excludes other etiologies of liver disease, MASLD is diagnosed based on positive criteria that emphasize metabolic dysfunction, including obesity, type 2 diabetes, and metabolic dysregulation [4-6]. This shift toward a more inclusive definition reflects the complex pathophysiology underlying MASLD and facilitates a more comprehensive understanding of its etiology, particularly in high-risk populations such as PLWH [7, 8].

Oxidative stress, characterized by an imbalance between reactive oxygen species (ROS) production and antioxidant defenses, is a critical factor in the pathogenesis of MASLD [9, 10] It contributes to lipid peroxidation, mitochondrial dysfunction, inflammation, and hepatic fibrosis, forming a vicious cycle that exacerbation liver damage [11-13]. In HIV-infected individuals, oxidative stress is further amplified due to viral proteins, chronic immune activation, and antiretroviral therapy, leading to an increased prevalence of MASLD compared to the general population [14]. Consequently, addressing oxidative stress offers a promising therapeutic target for managing MASLD in PLWH [15].

Functional foods, which provide health benefits beyond basic nutrition, have emerged as a potential intervention [16-20]. These foods, which include plant-based products, animal-derived components, and microbial sources, are rich in bioactive compounds, such as polyphenols, omega-3 fatty acids, and carotenoids. Preliminary studies suggest these compounds can modulate oxidative stress, improve hepatic metabolism, and alleviate MASLD-related liver damage [21, 22]. However, the therapeutic potential of functional foods in PLWH remains underexplored, and their mechanisms of action are not fully understood.

This review aims to critically evaluate the current evidence on the role of functional foods in mitigating oxidative stress and improving MASLD in HIV-infected populations. By synthesizing findings from experimental and clinical studies, this paper seeks to identify research gaps and propose future directions for developing effective dietary strategies to address this growing public health concern.

2 HIV and Oxidative Stress

Studies have found that HIV infection significantly increases oxidative stress within the body and decreases antioxidant capacity, which leads to oxidative damage to proteins, lipids, and DNA, contributing to the pathogenesis of HIV-related comorbidities, including metabolic-associated fatty liver disease [14]. The envelope protein Gp120 and viral protein R (Vpr) have been implicated in the increased ROS production in HIV-infected individuals. These viral components induce oxidative stress by activating NADPH oxidase and mitochondrial dysfunction, resulting in excessive ROS generation [23-26]. ROS, including superoxide anions, hydrogen peroxide, and hydroxyl radicals, are highly reactive intermediates that disrupt cellular homeostasis and amplify inflammatory pathways [27-30].

The oxidative stress in HIV infection is further compounded by the chronic immune activation and inflammation associated with the disease. Pro-inflammatory cytokines, such as tumor necrosis factor-alpha (TNFα) and interleukin-1 beta (IL-1β), are elevated in HIV-infected individuals, stimulating further ROS production through the activation of NFκB and other signaling pathways [31-34]. Studies have shown a positive correlation between oxidative stress markers and immune dysregulation, as evidenced by elevated malondialdehyde (MDA) levels and reduced antioxidant capacity in HIV patients [14, 35]. However, conflicting findings, such as those reported by Halickoval et al., who found no significant difference in oxidative stress markers between HIV-infected individuals and healthy controls, highlight the complexity of this relationship and the need for further investigation [36].

Oxidative stress also impacts HIV-associated neurological dysfunctions, such as HIV-associated dementia. Lipid peroxidation products, including 4-hydroxynonenal (4-HNE), have been found at elevated levels in the brain tissues of affected individuals, contributing to neuronal apoptosis and mitochondrial dysfunction [37, 38]. Some studies have used antioxidants to treat HIV-associated dementia, finding that antioxidant therapy can halt the progression of the disease. However, potential statistical significance was not observed between the experimental and control groups likely due to insufficient sample sizes [39]. Another study found that Selegiline may exert neuroprotective effects by reducing ROS production and thereby reducing neuronal apoptosis [40].

In addition to its direct effects on tissues, oxidative stress contributes to the suppression of the immune system in HIV-infected individuals. It depletes key nutrients, such as tryptophan, which are critical for maintaining antiviral responses and enhances viral replications [41-46]. Antioxidant-rich interventions, including dietary supplements and functional foods, have shown promise in preclinical studies for mitigating these effects [47-50]. However, the clinical efficacy of such approaches in improving oxidative stress-related outcomes in HIV remains an area requiring robust investigation [51].

3 Oxidtive Stress and MASLD

Oxidative stress plays a central role in the development and progression of metabolic dysfunction-associated steatotic liver disease, particularly in high-risk populations, such as individuals living with HIV [52]. The pathogenesis of MASLD involves the interplay of multiple factors, including insulin resistance, lipid accumulation, inflammation, and mitochondrial dysfunction. Among these, oxidative stress has emerged as a primary driver, contributing to hepatocellular injury, steatosis, and fibrosis [53].

ROS, produced during normal metabolic processes, are tightly regulated by antioxidant defense mechanisms, such as superoxide dismutase (SOD), catalase, and glutathione peroxidase [3]. However, when ROS production exceeds the capacity of these defenses, oxidative damage occurs, triggering lipid peroxidation and protein oxidation. In hepatocytes, excessive ROS activates signaling pathways, such as NFκB and MAPKs, leading to the production of pro-inflammatory cytokines, including TNFα, IL-6, and IL-1β [54]. This inflammatory response exacerbates liver injury and promotes fibrosis through the activation of hepatic stellate cells [55, 56].

The Nrf2 pathway, a key regulator of cellular redox homeostasis, plays a protective role by inducing the expression of antioxidant enzymes [57]. However, chronic oxidative stress can impair the Nrf2 pathway, further tipping the balance between ROS accumulation [58]. Conversely, the NFκB pathway, which is activated by oxidative stress, perpetuates the inflammatory cycle by promoting the expression of pro-inflammatory genes [59]. The interplay between Nrf2 and NFκB signaling creates a feedback loop, amplifying oxidative damage and inflammation in MASLD.

In individuals living with HIV, oxidative stress is further amplified by the combined effects of chronic viral infection, antiretroviral therapy (ART), and metabolic disturbances [60]. These factors synergistically exacerbate hepatic oxidative stress, creating a permissive environment for the onset and progression of MASLD. Evidence suggests that the prevalence of MASLD in people living with HIV ranges from 20% to 63% significantly higher than in the general population [61]. This underscores the need for targeted interventions to address oxidative stress as a therapeutic strategy for managing MASLD in this vulnerable group.

Correcting oxidative stress has shown promise in improving liver health and mitigating MASLD [9]. Preclinical studies have demonstrated that antioxidant compounds can reduce hepatic lipid accumulation, inhibit inflammation signaling, and restore mitochondrial function [62-64]. Functional foods rich in bioactive compounds, such as polyphenols, anthocyanins, and omega-3 fatty acids, represent a promising dietary approach for managing oxidative stress-related liver injury [9, 65-67]. However, more rigorous clinical studies are needed to validate their efficacy and establish standardized guidelines for their use.

4 Functional Foods with Anti-HIV Activity

The concept of functional foods was first proposed by Japan in 1984 and subsequently recognized by countries around the world [68, 69]. Over the past decade, research on functional foods has gained widespread attention. Functional foods offer additional health benefits beyond basic nutrition, providing an attractive new option for the prevention and treatment of certain diseases, which has led to their increasing popularity [69]. Functional foods exhibit a wide array of health benefits due to their diverse bioactive components, which commonly include anthocyanins, astaxanthin, lycopene, resveratrol, curcumin, and others [70-74]. These distinct bioactive compounds endow functional foods with varied health-promoting properties, such as reducing blood lipid levels, lowering blood pressure, protecting the heart, combating cancer, and exhibiting anti-inflammatory and anti-diabetic effects [75]. Recent studies on HIV have found that certain functional foods possess anti-HIV effects. For instance, a longitudinal analysis of data from over 60 months of follow-up by Sung et al. revealed that long-term consumption of Korean red ginseng helped maintain CD4+ T-cell counts in HIV patients and delayed the development of drug resistance, thereby improving HIV treatment outcomes and prognosis [76]. Another study indicated that ajoene in garlic can inhibit HIV from attaching to host cells, thus exerting antiviral effects [77]. Furthermore, other antioxidant-rich foods, such as green tea and ganoderma lucidum (Reishi mushroom), have also been recognized for their potential to enhance immunity and combat HIV [78-81]. The anti-HIV effect of ganoderma lucidum is primarily achieved through the inhibition of HIV protease by its constituents, ganoderma B and ganoderic acid B, indicating the potential for use in the development of antiretroviral drugs [82-84]. Green tea, containing epigallocatechin gallate (EGCG), can block HIV-1 from adhering to CD4 molecules through the gp120 protein, significantly reducing the infectivity of HIV-1 [85].

Additionally, certain functional foods possess immunomodulatory properties; for instance, sesamin, a polyphenolic compound derived from sesame, can regulate levels of immune cytokines such as IL-4, IL-5, IL-13, and inhibit the NFκB signaling pathway [86]. NFκB is a crucial molecule regulating HIV replication and is currently a significant potential target in HIV-1 chemotherapeutic research. One major research direction in targeting NFκB for HIV treatment involves the use of antioxidants, such as N-acetyl-L-cysteine (NAC) [87]. Consequently, it is plausible that moderate intake of antioxidant-functional foods could aid in the treatment of HIV.

5 Antioxidant Functional Foods and MASLD

The onset and progression of MASLD are closely related to oxidative stress. Therefore, functional foods rich in antioxidant-active compounds may play an important role in the prevention and treatment of MASLD. It has been confirmed that some functional foods can neutralize free radicals and reduce oxidative stress-induced liver damage, thereby inhibiting hepatic steatosis by providing a variety of active compounds, such as polyphenols and anthocyanins [88]. Based on their origin, these functional foods can be categorized into three major groups: plant-based, animal-based, and microorganism and algae-based, with most currently discovered functional foods being plant-derived [75]. The following section will elaborate on functional foods with antioxidant properties and protective effects against fatty liver disease, classified by their source.

5.1 Plant-based Functional Foods

Plant-based foods are rich in bioactive compounds such as polyphenols, flavonoids, and phytosterols, among others. Previous studies have also shown that certain phenolic compounds found in plants can prevent and treat hepatic steatosis by inhibiting lipogenesis and promoting fat breakdown [89]. These plant compounds not only protect the liver but also improve insulin resistance and inflammation [90]. A summary of plant-based functional foods that can antagonize oxidative stress is provided in Table 1. Since the classification of many plant foods is ambiguous, for example, bitter melon and avocado are considered both vegetables and fruits---have not been subdivided into plant-based foods in the table. It should be noted that some of the research findings listed in the table are controversial. For instance, while some studies suggest that green tea can improve fatty liver disease, others have found that green tea may exacerbate it, whereas black tea effectively prevents and treats the condition [91, 92]. In such cases, we have marked the entries with a symbol (†). The specific details are summarized in Table 1.

Table 1 Functional Foods from Plants

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
SchisandraChinensis bee pollen	Naringenin, rutin, chrysin	anti-inflammatory, antioxidant, hypoglycemic,, inhibiting hepatic lipid accumulation	inhibit SREBP-1c, LXR-α, and FAS, balance gut microbiota	[93]
Black tea (Camellia sinensis)	Black tea extract (Tea flavonoids, theaflavin, troxerutin [TXER])	antioxidan, anti-tumor, anti-inflammatory, improving hepatic steatosis	downregulate iNOS, eliminate NO production, inhibit oxygen free radicals production	[91, 92, 94]
Coccinia grandis (ivy gourd) leaves	alkaloid, cardenolides, flavonoids, terpenoids, saponins, and polyprenols	antioxidant, anti-inflammatory, hypolipidemic, hypoglycemic, analgesic, antimicrobial, antipyretic, sedative, hepatoprotective, anticancer	upregulate PPAR-α & PPAR-γ, downregulate NF-κB, iNOS, TNF-α, upregulate eNOS, inhibit bcl-2 expression	[95]
Bitter gourd	charantins, polypeptide-p, momordin, oleanolic acid 3-o-monodesmoside	anti-inflammatory, antioxidant, anti-diabetic, blood hypolipidemic, improving insulin re-	regulate mtochondrial activity, inhibit lipid accumulation	[96]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
		sistance, alleviating hepatic steatosis		
Olive leaf	Oleuropein	anti-inflammatory, anti-oxidant, hypoglycemic, hypolipidemic, anti-hypertensive, antiviral	scavenging free radicals, inhibit chemical oxidation of LDL	[97]
Broccoli	vitamin C, flavonoids, glucosinolates, sulforaphane, phenethyl isothiocyanate, isothiocyanates	antioxidant, hypolipidemic, reducing hepatic lipid deposition	inhibit CYP2E1, cytochrome P450 1A, cytochrome P450 3A, downregulate TNF, CD68	[98]
Broccoli sprouts	Glucoraphanin	improving insulin resistance, inhibiting oxidative stress	induce Nrf2, protect mitochondria, inhibit the development of NASH	[99]
Asian white radish (Raphanus sativus L.)	4-methylthio-3-butenyl isothiocyanate (MTBITC), anthocyanins, polysaccharides, glucosinolates	anti-inflammatory, anti-tumor, antioxidant, improving insulin resistance, hypolipidemic	reduce hepatic lipid accumulation	[66]
Black radish (Raphanus sativus L. var. niger)	3-(E)-(methylthio)methylene-2-pyrrolidinethione, polyphenols, MTBITC, anthocyanins, polysaccharides, glucosinolates	antioxidant, liver protective, hypolipidemic	enhance Nrf2 expression, eliminate free radicals	[100]
Tomato juice	Lycopene	anti-inflammatory, anti-proliferation, antioxidant, hypolipidemic, reducing hepatic lipid accumulation	upregulate FXR and HNF4A, increase the abundance of intestinal lactobacilli, decrease acetate: propionate ratio	[101]
Spinach	β-carotene, flavonoids, phenolic compounds, lutlein, lycopene, linolenic acid, thylakoids	hypoglycemic, hypolipidemic, anti-obesity, antioxidant, anti-inflammatory	scavenge ROS, induce antioxidant enzymes (catalase, SOD, GPx, and glutathione reductase), upregulate PPAR-γ, downregulate TNF-α and PTX-3	[102]
Orange carrot	carotenoids	anti-inflammatory, antioxidant, improving fatty liver	upregulate PPAR-α, AMPK, and PGC-1α	[103]
Corn	Corn peptide	hypolipidemic, antioxidant, hepatoprotective, hypoglycemic, anti-hypertensive	upregulate SIRT1/PPARα and Nrf2/HO-1	[104]
Pueraria lobata	Puerarin (4’,7-dihydroxy-8-β-D-glucosylisoflavone)	antioxidant, improving insulin resistance, hypolipidemic, hepatoprotective	activate JAK/STAT, upregulate Nrf2 and HO-1	[105]
Quinoa	Quinoa polyphenol	anti-oxidant, hypolipidemic, anti-inflammatory	upregulate AMPK, inhibit SREBP-1c activity, downregulate SREBP-2, HMGCR, IL-1β, IL-6, and TNF-α	[106, 107]
Oat	Beta-glucan	hypolipidemic, hypoglycemic, antioxidant, improving insulin resistance, immunomodulation, anti-infection, anti-tumor	inhibit NFκB, balance gut microbiota	[108, 109]
Djulis (Chenopodium formosanum Koidz)	rutin, Phytochemicals (Polyphenols and catechins)	antioxidant, hepatoprotective, hypolipidemic, hypoglycemic, anti-tumor	activate PPAR-γ, upregulate SIRT1, LXR, pAMPK, PPAR-α, p-ACChe CPT1B	[65]
Mango, anemarrhena	Mangiferin (1,3,6,7-tetrahydroxy-xanthone-C2-β-D-gluoside)	anti-inflammatory, anti-tumor, anti-oxidative stress, hypolipidemic, anti-diabetic, anti-aging, anti-viral, anti-microbial, hepatoprotective	inhibit NFκB, enhance Nrf2 expression, blockTGF-β/Smad pathway, downregulate NLRP3 expression, activate AMPK	[110]
Sesame	Sesame lignans (sesamin, sesamolin, and sesamol)	anti-inflammatory, antioxidant, hypoglycemic, hypolipidemic, improving insulin resistance, improving hepatic steatosis	downregulate phospho-JNK1/2/3, phospho-NF-κB p65, phospho-IRS1 and phospho-IKK α/β, upregulate XBP1, CPT-1A, LXRα, CYP7A1, reductions of TNF-α and IL-6 levels, increasing GSH, 38 vitamin C and Nrf2 levels,	[111-113]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
			decreasing MDA and NO levels, and enhancing SOD, CAT 39 and GSH-Px activities, activate AMPK and PPAR signaling pathways	
Purple sweet potato	anthocyanins	antoxidant, anti-cancer, improving insulin resistance, anti-diabetic, inhibiting liver fat accumulation	inhibit Src/ERK/C/EBPβ	[114]
Purple corncob	peonidin 3-O-glucoside (P3G)	anti-inflammatory, antioxidant, anti-hypertensive, anti-cancer, inhibiting liver fat accumulation	upregulate the lysomal function mediated by transcription factor EB, activate PPARα	[115]
Onion	anthocyanins, flavanols (quercetin and its derivatives)	antioxidant, immunomodulation, antibacterial, hypolipidemic, hypoglycemic, anti-inflammatory, reducing hepatic steatosis	downregulate NFκB, iNOS, TNFα, balance gut microbiota, upregulate Sirt1	[116]
Red cabbage (Brassica oleracea L.)	anthocyanins, flavonoids, ascorbic acid, isothiocyanate (glucosinolates)	antioxidant, anti-inflammatory, hypolipidemic, hepatoprotective	scavenging ROS	[117, 118]
A çaí(Euterpe oleracea Martius)	Anthocyanin (mainly polyphenols), polyunsaturated (PUFA) and monounsaturated (MUFA) fatty acids	antioxidant, improving hepatic steatosis	increase the expression of Gpx1, Gpx4, Sod1	[119]
tomatoes, watermelons, pink guavas, grapefruits, apricots, and papayas	Lycopene (lyco)	antioxidant, anti-inflammatory, anti-cancer, anti-hypertensive, hypolipidemic, anti-proliferation, inhibiting lilver fat accumulation	upregulate NR1H4 and HNF4A, activate SIRT1	[101, 120-122]
Tomato	Saponins (mainly Esculeoside A)	antioxidant, hypolipidemic, hypoglycemic, inhibiting liver fat accumulation	activate AMPK, upregulate Nrf2-Keap1	[123]
Vine tea	Vine tea polyphenol (VTP)	antioxidant, anti-inflammatory, improving insulin resistance, inhibiting liver fat accumulation	upregulate Nrf2, activate AMPKα/PPARα, inhibit the expression of SREBP-1 and FAS	[124]
Green tea†	EGCG	anti-inflammatory, antioxidant, anti-HIV, improving hepatis steatosis	increase NADPH oxidase activity, inhibit TLR4/MyD88 mediated NFκB activation	[80, 81, 85, 125-127]
kombuchas	phenolic compounds, mainly catechins	anti-inflammatory, antioxidant, hypolipidemic, reversing hepatic steatosis, improving glucose tolerance	downregulate SREBP1c and NLR, upregulate CPT-1 and adipo-R2	[128]
Sophora japonica	Troxerutin (TXER)	anti-thrombotic, antioxidant, anti-inflammatory, anti-tumor, neuroprotective	scavenging ROS, inhibit NFκB	[94, 129]
Gardenia jasminoides	Geniposide	anti-inflammatory, antioxidant, lipid-lower, inhibiting liver fat accumulation	upregulate Nrf2/HO-1 and AMPK signaling	[130]
citrus	flavonoids	antioxidant, hypolipidemic, anti-cancer, anti-cardiovascular disease, improving insulin resistance	activate SIRT1/PGC-1α signaling pathway	[131]
Pimpinella anisum L.	trans-anethole, γ-himachalene, methyl chavicol, cis-isoeugenol, linalool, pseudo-iso-eugenyl-2-methyl butyrate	antioxidant, lower blood lipid, anti-diabetic, hepatoprotective, reducing liver fat accumulation	inhibit TLR-4, decrease TNF-α, IL-6	[132]
Qu Zhi Qiao (Fruit of Citrus Paradisicv. Changshanhuyou)	total Flavonoids (TFCH)	anti-inflammatory, antioxidant, improving hepatic steatosis	upregulate Nrf2-ARE signaling pathway	[133]
Haw (Crataegus aronia)	total flavone of haw leaves (TFHL), polyphenolic antioxi-	antioxidant, hypolipidemic, improving hepatic steatosis	upregulate Nrf2	[134, 135]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
	dants, flavonoids (luteolin-7 glucoside, hyperoside and rutin			
Vitis coignetiae Pulliat leaves	Polyphenols and anthocyanins	antioxidant, reducing liver fat deposition	inhibit CYP2E1	[136]
Crataegus oxyacantha L	flavonoids and triterpene acids	anti-nflammatory, hypolipidemic, reducing hepatic fat accumulattion	increase the activity of GSH and FRAP	[137]
Dragon fruit (Red Pitaya)	betacyanins	antioxidant, anti-inflammatory, hypolipidemic, hypoglycemic, improving insulin resistance, reducing hepatic steatosis	scavenging ROS, downregulate TNF-α, IL-1β, IL-6, upregulate IL-10, balance gut microbiota	[138]
Grape seed extract	proanthocyanidins, resveratrol	anti-inflammatory, antioxidant, anti-cancer, improving hepatic steatosis	upregulate PPARβ/δ, SOD, GPx, inhibit MAPK	[139, 140]
Grape fruit	resveratrol, ellagic acid, pterostibene	antioxidant, anti-inflammatory, anti-cancer, improving hepatic steatosis	inhibit NFκB, upregulate Gstp1, FABP1, Gpx4, Gpx8, Gss, Gps7, Sod1, Nrf2, PPARα, HO-1, downregulate mTORC1, SREBP-1c	[141-143]
Vitis vinifera L. skin	anthocyanidins	anti-inflammatory, antioxidant, improving insulin resistance, improving hepatic steatosis	downregulate lipogenic factor sterol regulatory-element binding protein-1c, upregulate lipolytic pathway (phosphorylated liver kinase B1/phosphorylated adenosine-monophosphate-activated protein kinase	[144]
Black soybean	Isoflavones	antixodiant, improving insulin resistance, hypolipidemic	downregulate SREBP2, HMG-CoA reductase, upregulate PPARα/γ, ABCA1, pAMPK	[145]
Pea	Pea albumin	antioxidant, hypolipidemic, anti-inflammatory, improving insulin resistance, reducing liver fat accumulation	activate AMPKα and ACC, downregulate SREBF1 and FASN, upregulate ATGL and PPARα/γ	[146]
Loquat fruit	Polyphenols	anti-inflammatory, antioxidant, anti-cancer, anti-hypertensive, anti-diabetic	activate PI3K/Akt/GLUT-4, SREBP-1c, ChREBP, Nrf-2, inhibit TLR4/MyD88/TRIF pathway	[147]
Eriobotrya japonica seed	Polyphenols (caffeic and chlorogenic acids)	blood hypolipidemic, antioxidant, inhibiting liver fat accumulation	scavenging ROS	[148]
Eriobotrya japonica	hot water extract of Eriobotrya japonica (EJW)	antioxidant, reducing hepatic lipid accumulation	upregulate AMPK, CPT-1, PPARα	[149]
Loquat leaf	Sesquiterpene glycoside 3 (SG3), total sesquiterpene glycosides, total flavonoids (quercetin and kaemferol)	anti-inflammatory, antioxidant, improving insulin resistance, hypolipidemic	downregulate CYP2E1, p-JNK and NLRP3, upregulate IRs-1and Akt phosphorylation	[150-152]
Coconut oil	polyphenols (ferulic acid, caffeic acid, coumaric acid)	antioxidant, hypolipidemic, improving hepatic steatosis	upregulate Nrf2	[153]
Olive oil	polyphenols, nitro-fatty acids (NO2-FA), oleic acid, monounsaturated fatty acid, tyrosol	anti-inflammatory, antioxidant, improving insulin sensivity, hypolipidemic, improving hepatic steatosis	upregulate Nrf2, AMPK, and PPARs, inhibit the activation of JNK, promote the expression of Bcl2 and Bax, activate CBS and CSE mediated H_2S synthesis	[154-162]
Pine nut	Pinolenic acid	anti-inflammatory, antioxidant, hypolipidemic	downregulate SREBP1c, FASN, SCD1, upregulate AMPK/SIRT1 signaling pathway	[163]
Avocado oil	oleic acid, linolenic acid	antioxidant, hypolipidemic	improve mitochondrial ETC function, upregulate PPAR-δ	[164]
Flaxseed oil	Alpha-lipoic acid	antioxidant, reducing hepatic	activate PPARα, inhibit FAS	[165-167]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
		lipid accumulation	and SREBP1	
Berberis vulgaris	Berberine	antioxidant, blood hypolipidemic, improving glucose tolerance, reducing hepatic lipid accumulation	downregulate SREBP-1c, pERK, TNF-α, JNK	[168]
Sesame oil	sesamol, sesamolin, and sesamin	anti-inflammatory, antioxidant, reducing liver fat accumulation	downregulate TNF-α, TGF-β and leptin, inhibit the activation of NFκB	[169]
Rapeseeds oil	phenolic compounds (sinapic, salicylic, protocatechuic, phydroxybenzoic, gentisic, ferulic, p-coumaric, cinnamic, caffeic, syringic acids), tocopherol, phytosterols	antioxidant, inhibiting lipogenesis	inhibit FAS, ACC, SREBP1 and SREBP, upregulate PPAR-α/γ	[165, 170]
Coffee	Caffeic acid, caffeine	anti-inflammatory, antioxidant, alleviating hepatic steatosis	activate Nrf2, PPARα and CPT1, inhibit FAS and ACC	[171, 172]
Dark chocolate	Polyphenolsv (catechin, epicatechin)	antioxidant, slowing the progression of hepatic steatosis	downregulate Nox2	[173-175]
blackberry	anthocyanins (cyanidin-3-glucoside, total alkaloids), flavonoids, ellagic acid	anti-inflammatory, antioxidant, improving insulin resistance, and reducing hepatic lipid accumulation	inhibit NFκB, downregulate FAS, SREBP-1c and ACC, upregulate CPT-1, improve gut microbiota balance, increase SCFA levels	[176]
Coffeeberry	caffeine, chlorogenic acid, condensed proanthocyanidins, quinic acid, ferulic acid	anti-inflammatory, antioxidant, improving hepatic steatosis	activate CaMKII/CREB/BDNF pathway, upregulate Sirt1, inhibit NFκB, iNOS, COX-2, PARP-1, PI3K/Akt/mTOP/p70S6K signaling	[177]
Orange	quercetin, phytosterols, dietary fibers, mono and poly-unsaturated fatty acids, phenolic compounds	anti-inflammatory, antioxidant	downregulate TGF-β, CTGF, collagen-1α, inhibit NFκB	[178]
Citrus reticulate blanco peel	polymethoxyfalvones (PMFs)	antioxidant, anti-inflammatory, anti-cancer, improving hepatic steatosis, hypolipidemic, improving insulin resistance	activate Nrf2, upregulate CPT1α, ACOX1, PPARα, downregulate IL-6, TNF-α, IL-1β	[34]
Pu-erh tea	theabrownin	anti-inflammatory, antioxidant, improving insulin resistance	upregulate adenylate cyclase activity, increase cAMP levels, activate PKA/AMPK, upregulate PPARs, inhibit NFκB/JNK	[179, 180]
Fuzhuan brick tea†	theabrownin, caffeine	anti-inflammatory, anti-oxidant, blood hypolipidemic, hypoglycemic, improving hepatic steatosis	upreguate PPARs, promote gut microbiota baance, lower ERK phosphorylation, downregulate TGFβ, α-SMA, Col1A1, Col3A1	[181, 182]
Que Zui tea	6-O'-caffeoylarbutin	anti-inflammatory, antioxidant, hypolipidemic, hypoglycemic, reducing liver fat accumulating	upregulate Nrf2, adiponectin and adipoR2, activate AMPK/PPAR-α signaling pathway	[183]
Red wine	flavonoids, resveratrol	anti-inflammatory, antioxidant	inhibit NFκB, activate Nrf2, AP-1	[21, 184]
Lycium barbarum	Lycium barbarum polysaccharides	anti-inflammatory, antioxidant, immunomodulation	downregulate NLRP3/6, NFκB	[185]
Phyllanthus emblica L fruit†	polyphenols (tannins, lignans, flavonoids, alkaloids, mucic acid, gallic acid, ellagic acid, quercetin, geraniin, chebulagic acid, corilagin, phyllanthin)	anti-inflammatory, antioxidant, hypolipidemic, hypoglycemic, anti-cancer, anti-bacterial, hepatoprotective	downregulate SREBP-1c, LXRα, upregulate PPARα and adiponectin	[186]
Mulberry	flavonoids, polyphenol compounds, 1-deoxynojirimycin	hypoglycemic, increasing insulin secretion	upregulate PPARs, inhibit FAS and HMGCoA reductase	[187, 188]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
	(DNJ)			
Cherry	anthocyanins (cyanidin-3-rutinoside), flavones	anti-inflammatory, antioxidant, hypoglycemic, hypolipidemic	inhibit NFKB, upregulate PPAR-γ, Nrf2	[189]
Ginkgo biloba	Ginkgolide B (GB)	anti-inflammatory, antioxidant, inhibiting hepatic fat accumulation	upregulate Nrf2	[190]
Cranberry (Vaccinium oxycoccos L.)	Vit E, Vit C, phenolic polyphenols (proanthocyanidins, anthocyanins, resveratrol)	antioxidant, anti-inflammatory, hypolipidemic, inhibiting hepatic fat accumulation	inhibit SREBP-1c, TLR4/NFκB, downregulate TNFα and IL-6	[191, 192]
Blueberry leaf	anthocyanins, flavonoids (D-catechin, L-epicatechink, rutin, isoquercitrin, iridin and quercetin), phenolic acid (chlorogenic acid), last anthocyanin (cyanidin-3-O-glucoside), catechol	anti-inflammatory, antioxidant, inhibiting hepatic fat accumulation	induce the expression of AMPK/PGC-1α/SIRT3, upregulate ERRα, Nrf1, Nrf2	[193]
blueberry	Flavonoids, polyphenols (anthocyanins), pterostibene	anti-inflammatory, antioxidant, anti-cancer, improving hepatic steatosis	activate SIRT1/PGC-1α, inhibit SREBP-1c and nTORC1, upregulate PPAR-α, Nrf2, HO-1	[143, 194, 195]
Mulberry leaf extracts	chorogenic acid derivative, flavonoid, rutin, quercetin	anti-inflammatory, antioxidant, anti-diabetic, hypolipidemic, reducing liver fat deposition	upregulate PPARα, inhibit FAS, HMGCoA reductase	[109, 188]
Bayberry juice	phenolic acids (caffeic, ferulic, sinapic, salicylic acids), anthocyanins, vitamin C	anti-inflammatory, antioxidant	downregulate TNF-α, IL-8	[196]
White wine	tyrosol	anti-inflammatory, antioxidant, hepatoprotective	activate CBS and CSE mediated H2S biosynthesis	[162]
curcuma longa	curcumin	anti-inflammatory, antioxidant, anti-tumor, hypolipidemic, hepatoprotective	induce Nrf-2-FXR-LXR signaling pathway	[168, 197-200]
Lemon balm	Rosmarinic acid	anti-inflammatory, antioxidant, improving insulin resistance, reducing liver fat accumulation	upregulate Nrf2, AMPK, PGC-1α, PPARα and downregulate SREBP-1c	[201]
Maoberry (antidesma bunius)	Polyphenol (anthocyanin, cyanidin, peonidin, ascorbic acid, gallic acid, (-)-epicatechin(+)-catechin, cyanidin-3-O-glucoside	anti-inflammatory, antioxidant, hypolipidemic, improving hepatic steatosis	downregulate ACC and GPAT-1, TNFα, and IL-6	[202]
Satsuma mandarin	β-Cryptoxanthin	anti-inflammatory, antioxidant, improving insulin resistance, hypolipidemic, reducing liver fat accumulation	inhibit JNK, p38 MAPK, and NFκB, upregulate PPARs	[203, 204]
Beet (Beta vulgaris)	Betaine	anti-inflammatory, antioxidant, hypolipidemic, reducing hepatic steatosis	activate beclin 1, Atg4 and Atg5, induce autophagy, upregulate Akt/mTOR signaling	[205]
raspberry	Ellagic Acid, raspberry ketone, resveratrol	anti-inflammatory, antioxidant, reducing hepatic steatosis	downregulate p47phox, and HIF-α, activate Nrf2, and Akt, downregulate TNFα and IL-6, inhibit TLR4/NFκB	[7, 142, 192, 206]
Pomegranate	Polyphenols, anthocyanidins, tannic acid, gallic acid, and ellagic acid	anti-inflammatory, antioxidant	reduce the expression of TNFα, IL-1β, IL-6, and IL10	[207, 208]
Thymbra spicata L.	Rosmarinic acid, carvacrol	anti-inflammatory, antioxidant, improving hepatic steatosis, hypolipidemic	reducing oxidative/nitrosative stress pathay, reducing the contents of intracellular NO and ROS, inhibit NFκB	[209]
Carob pod	polyphenols	anti-inflammatory, antioxidant, anti-diabetic, reducing liver fat accumulation	upregulate SIRT1, PCG-1α, CTP-1a	[210]
Soybean	Polyenylphosphatidylcholine (PPC), phytic acid, pinitol, soy	anti-inflammatory, antioxidant, hypolipidemic, improving insulin	balance the gut microbiota, reducing TNFα, upregulate	[211-214]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
	protein	resistance	Cyp7b1, FXR, and FGFR4	
Black bean	polyphenols (delphinidin 3-glucoside, petunidin 3-glucoside, and malvidin 3-glucoside), flavonoids (anthocyanins)	antioxidant, hypolipidemic, increasing insulin secretion, reducing hepatic lipid concentration	downregulate SREBP-2, Cyp7A1	[215]
Green coffee bean	chlorogenic acid	hypolipidemic, reducing hepatic fat accumulation	activate AMPK, upregulate CPT-1, GLUT4 and PPARγ, inhibit ACC	[216]
Chickpea (Cicer arietinum)	biochanin A (Bio-A)	hypolipidemic, hypoglycemic, anti-inflammatory, inhibiting hepatic steatosis	activate Sirt3/AMPK/ULK-1 signaling	[217]
Mung bean (Vigna radiata L.)	phenolic acids	hypocholesterolemic, anti-inflammatory, antioxidant, hepatoprotective, inhibiting hepatic steatosis	increase fecal excretion of cholesterol, upregulate CPT1, Bcl2a1a, downregulate SREBP-1, HMG-CoA reductase	[218]
White kidney bean (Phaseolus vulgaris L.)	phaseolin, polyphenols, resistant starch, oligosaccharides	antioxidant, anti-inflammatory, anti-cancer, anti-obesity, cardioprotective, hypolipidemic, hypoglycemic, improve insulin resistance, reducing hepatic fat accumulation	balance gut microbiota, inhibiting α-amylase	[219, 220]
Pigeon pea (Cajanus cajan L)	stilbenes, phytosterol (β-sitosterol, stigmasterol), linoleic acid	hypolipidemic, hypoglycemic	upregulate PPARα, CPT-1, and CYP7A1	[221]
Walnut	Walnut green husk polysaccharide (WGHP), ellagic acid	anti-inflammatory, antioxidant, anti-bacterial, improving glucose intolerance	inhibit CYP2E1, JNK, and p38K, activate AMPK and SIRT1	[222, 223], [206]
Chestnut	tannin, ellagic acid, scoparone	anti-inflammatory, antioxidant, immunomodulation, hypolipidemic, inhibiting hepatic steatosis	inhibit SREBP-1c, downregulate FAS, ACC	[224]
Poria cocos	Poria cocos polysaccharides	anti-inflammatory, antioxidant, immunomodulation, hypolipidemic, balancing gut microbiota, reducing hepatic fat accumulation	inhibit NLRP3	[225]
Ganoderma mushroom	Triterpenoids (lucidenic acids, ganoderic acids)	antioxidant, anti-bacterial, anti-cancer, anti-HIV, anti-hypertensive, anti-diabetic, improving hepatic steatosis	increase the activity of ACC and AMPK, inhibit SREBP1, upregulate CPT-1	[79]
Cinnamon	Cinnamyl alcohol	anti-inflammatory, antioxidant	downregulate SREBP-1c and ACC1	[226]
Lycii (Goji berry, wolfberry)	LFP-a1 (Lycii fructus polysaccharides)	antioxidant, immunomodulation, anti-aging, anti-cancer	inhibit the expression of Bax and C-Myc	[227]
Humulus japonicas	luteolin 7-O-β-D-glucoside (LU)	anti-inflammatory, antioxidant, alleviating hepatic steatosis	upregulate PPARα and SOD1, increase catalase activity	[228]
Korean Red Ginseng	Red ginseng extract	antioxidant, anti-HIV, inhibiting hepatic fat accumulation	upregulate PPAR-α, CPT-1, CYP7A1, AMPK, and ACC, inhibit C/EBPα, SREBP-1c, PPAR-γ, FAS, ACAT2, HMGCR, and ApoB	[76, 229]
Ginger	6-gingerol, 6-shogao1,, alpinetin, citral, zingerone	anti-inflammatory, antioxidant, hypolipidemic, anti-cancer, anti-diabetic, reducing liver fat accumulation, anti-HIV, immune-enhancing	downregulate TNFα and IL-1β, downregulate SREBP-1c, SREBP-2, FAS, SCD1, LXR, ACC1, DGAT-2, and CYP2E1, inhibit NFκB, activate Nrf2, LKB1/ AMPK signaling pathway, activate SIRT3, and Akt, upregulate CPT1α, PPARα/γ, PGC1α	[230-237]

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
Garlic (Allium sativum)	Garlic-derived S-allylmercaptoccysteine, N-trans-coumaroyloctopamine, N-trans-feruloyloctopamine, allicin ajoene, diallyl disulfide, SAC sulfoxide, S-methylcysteine sulfoxide, allicin	anti-inflammatory, antioxidant, immunomodulation, hypolipidemic, improving insulin resistance, anti-HIV	downregulate CYP2E1, p38MAPK, downregulate TGF-β1 and α-SMA, activate MEK/ERK, inhibit AP-1, NFκB	[238-241]
black rice (Oryza sativa L.)	anthocyanin (Cyanidin-3-glucoside, C3G)	anti-inflammatory, antioxidant, hypolipidemic, hypoglycemic, improving fatty liver	upregulate AMPK, PPAR-α, CPT1A, ACO, and CYP4A10	[242]
Sintanur brown rice	phenolic acids (trans-ferulic, trans-p-coumaric acid), vitamin B3 (niacin)	anti-inflammatory, antioxidant, reducing hepatic lipid accumulation	upregulate SCFAs, regulate FXR and reduce hepatic lipogenesis	[243]
Red rice	phenolic extract, anthocyanins, proanthocyanidins, protocatechuic acid, oryzanol, vitamin E, coenzyme Q10	anti-inflammatory, antioxidant, anti-diabetic, hypolipidemic, reducing hepatic steatosis	downregulate NFκB, Bax/Bcl-2 ratio, iNOS, p47PHOX, ATGL, CD36, LXRα, SREBP-1c, SREBP-2, HMGCR, and hepatic lipase (HL), upregulate ApoA-1, LPL, and CPT1A	[244]
Brown sorghum (Sorghum bicolor L.)	phenolic acids, 3-deoxyanthocyanidins (3-DXA), proanthocyanidins (tannins)	antioxidant, anti-inflammatory, anti-diabetic, anti-cancer, anti-obesity, improving insulin resistance, improving hepatic steatosis	block lipogenic enzymes, increase β-oxidation, upregulate PPARα, downregulate SREBP-1c, increase adipo R2 sensitivity	[245]
whole wheat	leucine-arginine-proline (LRP), leucine-glutamine-proline (LQP)	antioxidant, improving fatty liver	upregulate AMPK, ACC	[246]
Buckwheat (Fagopyrum esculentum Moench)	fagopyrin polyphenols (flavonoids)	anti-inflammatory, antioxidant, hypolipidemic	upregulate ACOX1, PPARα, ACAT2, FXR, SCFAs, downregulate CEBPα, CD36, ACC1, SREBP-1c,	[247]
Tartary buckwheat (Fagopyrum tataricum (L.) Gaertn.)	flavone	anti-inflammatory, antioxidant, hypolipidemic, anti-hypertensive, hypoglycemic	inhibit iNOS, COX2, IL-6, IL-1β, TNF-α, downregulate SREBP1, acetyl-COA carboxylase protein levels, activate AMPK, downregulate MAPK and NFκB, balance gut microbiota	[248]
Khorasan bean	polyphenols, carotenoid, coumarin, and ferulic acid isomer	anti-inflammatory, antioxidant, improving hepatic steatosis, improving insulin resistance	downregulate NFκB, TNF-α and IL-1α	[249]
Purslane seeds (Portulaca oleraceae L.)	Omega-3, α-tocopherol, ascorbic acid, β-carotene, glutathione, phenolic and flavonoids compounds	anti-inflammatory, antioxidant, hepatotective, anti-diabetic	scavenging ROS, increase intracellular glutathione concentration	[67]
Garden cress seeds (Lepidium sativum Linn.)	phenolic compounds, alkaloids, falvonoids, ascorbic acid, tocopherol, polyunsaturated fatty acids	anti-inflammatory, antioxidant, hypolipidemic, hepatotective, anti-diabetic	scavenging ROS, increaseintracellular glutathione concentration	[67]
Adzuki bean (Phaseolus angularis)	anthocyanin, catechin, and adzukisaponin	anti-inflammatory, antioxidant, hypolipidemic	upregulate adiponectin, AMPKα, CPT-1, PPARα, downregulate SREBP-1c, FAS, TNFα and NFκB	[250]

5.2 Animal-based Functional Foods

Although the number of animal-based functional foods discovered to have antioxidant activity is relatively limited, some have been proven to improve MASLD significantly. These functional foods mainly include fish, krill, shellfish, dairy products, and honey products, among others. A detailed summary is provided in Table 2.

Table 2 Functional Foods from Animals

Foods	Bioactive ingredients	Primary Benefits	Mechanisms of Action	Ref.
Salmon	Astaxanthin	anti-inflammatory, antioxidant, anti-cancer, immunomodulation, hypolipidemic, anti-diabetic	Block TGF-β/Smad3 signaling, inhibit MAPK and NFκB	[251, 252]
Fish oil†	Eicosapentaenoic (EPA), docosahexaenoic (DHA)	anti-inflammatory, antioxidant, improving hepatic steatosis	activate PPARs	[21, 253-257]
Bluefin tuna	Selenoneine	anti-inflammatory, antioxidant, hepatoprotective	downregulate Gpx1, selenop	[258]
Menhaden	Menhaden oil (C20-22(n-3)PUFA)	anti-inflammatory, hypolipidemic, hepatoprotective	inhibit SREBP1, ACC1, Fasn, NFκB, inhibit hepatic lipogenesis	[259]
Antarctic Krill (Euphausia superba)	phospholipid-protein complex, carotenoid fucoxanthin, astaxanthin	antioxidant, hypolipidemic	downregulat SREBP1c inhibit the expression of IL-1β, IL-6, TNFα	[260]
Freshwater clam (corbicula fluminea)	Campesterol, stigmasterol, unsaturated fatty acids	anti-inflammatory, antioxidant, improving hepatic steatosis	inhibit NLRP3, downregulate CYP3A	[261]
Silkworm pupae	Silkworm pupae peptides, α-linolenic acids	antioxidant, improving fattyliver	upregulate PPARα, CPT-1	[262]
Yogurt	lactic acid, conjugated linoleic acid, folate, S. thermophiles, fructosan, galacto-oligosaccharides, galactose	ant-inflammatory, antioxidant, improving insulin resistance	inhibit NFκB, downregulate the expresson of TNFα and FGF21	[263]
Camel milk	α-Lactalbumin, L-carnitine	antioxidant, anti-inflammatory, antibacterial, immunomodulation, hypolipidemic, alleviating hepatic steatosis	scavenging ROS, inhibit lipid absorption, upregulate PPARα/SREBP1 ratio	[264, 265]

5.3 Microbial and Algal-based Functional Foods

Microbial-based functional foods with antioxidant properties and effects on improving fatty liver disease primarily include probiotics that regulate gut microbiota balance, postbiotics, and fungi used in the production of specific foods. Postbiotics are beneficial substances produced by microbial metabolism or structural components of microorganisms, such as short-chain fatty acids (SCFAs), polysaccharides, peptides, and phospholipids, among others [266]. These substances exert their antioxidant effects and improve fatty liver through various mechanisms. Similarly, algal-based functional foods also demonstrate significant antioxidant properties and can improve fatty liver disease. Common algal functional foods include Spirulina, brown algae, Chlorella, and astaxanthin-rich microalgae, such as Haematomatococcus Pluvialis is rich in anthocyanins, which are potent natural antioxidants that protect liver cells from damage caused by free radicals. Table 3 provides a summary of microbial and algal-based functional foods with antioxidant functions.

Table 3 Functional Foods from Microorganisms and algae

Foods	Primary Benefits	Classification	Mechanisms of Action	Ref.
Bifidobacteria	Antioxidant, improving hepatic steatosis	Probiotic	upregulate SIRT1 and PPARα, downregulate SREBP1c	[194]
Lactobacillus casei strain Shirota (LCS)	Anti-inflammatory, anti-oxidant, immunomodulation	Probiotic	downregulate TLR singaling pathway, inhibit SREBP1c and FAS	[267, 268]
Lactobacillus coryniformis subsp. Torquens T3 (T3L)	Anti-inflammatory, anti-oxidant, hypolipidemic, inhibiting liver fat accumulation	Probiotic	upregulate NRF2-NQO-1 pathway, upregulate SCFAs concentration, inhibit the expression of TNF-α and IL-6	[269]
Lactobacillus casei	Anti-inflammatory, anti-oxidant, reducing hepatic steatosis	Probiotic	balance gut microbiota, downregulate NGAL and KIM-1, activate FOXO1/pAMPK	[270]
Saccharomyces cerevisiae probiotic yeast	Anti-inflammatory, antioxidant, anti-diabetic (improving insuln sensitivity), inhibting fatty degeneration of liver	Probiotic	increase SCFAs concentration, immunemodulation, promote lipolysis	[271]
Sugary Kefir Strain Lactobacillus mali	Anti-oxidant, reducing liver fat accumulation	Probiotic	activate AMPK, SIRT-1, PGC-1α and PPAR-γ, inhibit SREBP-1, upregulate	[272]

Foods	Primary Benefits	Classification	Mechanisms of Action	Ref.
APS1			Nrf-2/HO-1	
Sodium butyrate	anti-inflammatory, antioxidant, hypolipidemic, improving hepatis steatosis	Postbiotic	upregulate miR-150, inhibit CXCR4, activate LKB1-AMPK-Insig, upregulate GLP-1	[273, 274]
Monascus	anti-inflammatory, anti-oxidant, anti-diabetic, anti-hypertensive, immunomodulation, anti-tumor, hypolipidemic	Probiotic	activate PPAR-γ/Nrf-2, upregulate FXR, PGC1α and PPAR-α, downregulate TNFα and IL-6, enhance NO synthesis, inhibit JNK, ERK p38 kinase, NPC1L1, HR-LPL, C/EBPβ, and RAGE signalng pathways	[275]
Wakame	anti-inflammatory, antioxidant, hypolipidemic, hypoglycemic, reduing hepatic steatosis	Phaeophyta	upregulate CTP-1 and PPAR-α, downregulate SREBF-1	[210]
Sargassum serratifolium	anti-inflammatory, antioxidant	Brown algae	activate Nrf2, AMPK, PPARα, upregulate NQO1, HO-1, GST, and CPT-1, inhibit SREBP1c and C/EBPβ	[276, 277]
Ulva prolifera	anti-inflammatory, antioxidant, hypolipidemic, improving insulin resistance, alleviating hepatic fat accumulation	green macroalgae	upregulate CPT-1a, ACOX1, and Acadm, inhibit DGAT1 and DGAT2	[278]
Brown seaweeds	anti-inflammatory, antioxidant, anti-cancer, reducing hepatic lipid accumulation	phaeophyta	activate PPARα/γ and Nrf2, downregulate TNFα, IL-1β, IL-6 and MCP-1	[279]
Spirulina	anti-inflammatory, antioxidant, immunomodulation, anti-cancer, anti-diabetic, hypolipidemic	blue-green algae	inhibit NFκB, downregulate TNFα, IL-6, and iNOS, upregulate adiponectin	[280-282]
Haematococcus pluvialis	anti-inflammatory, antioxidant, anti-cancer, immunomodulation, hypolipidemic, anti-diabetic	green microalga	block TGF-β/Smad3 signaling, inhibit MAPK and NFκB	[251]
Chlorella vulgaris	anti-inflammatory, antioxidant, hypolipidemic, immunomodulation, improve hepatic steatosis	microalgae	downregulate NGAL and KIM-1, activate FOXO1/pAMPK	[270]

6 Conclusions

In the absence of universally effective pharmacological treatments for MASLD, functional foods have emerged as a promising, non-toxic alternative for managing this condition, particularly in HIV-infected populations. Functional foods rich in antioxidants offer multiple health benefits, including reducing oxidative stress, mitigating inflammation, and improving hepatic lipid metabolism. These advantages make them attractive as a dietary intervention for managing MASLD. The findings summarized in this review highlight the therapeutic potential of functional foods, such as plant-based polyphenols, animal-derived omega-3 fatty acids, and probiotics. These bioactive compounds act through diverse molecular pathways, including the activation of Nrf2, suppression of NFκB, and modulation of lipid metabolism, to improve liver health. Notably, certain functional foods, such as EGCG, allicin, and ganoderma lucidum, have demonstrated dual benefits by both mitigating MASLD and exhibiting anti-HIV activity [78, 81, 241]. For example, green tea's EGCG not only reduces hepatic steatosis but also inhibits HIV reverse transcriptase, providing a novel approach to addressing comorbidities in PLWH [81].

Despite these promising findings, the current evidence is limited by several factors. Most studies are preclinical or observational, with a paucity of high-quality randomized controlled trials validating the efficacy and safety of functional foods in clinical settings. Moreover, inconsistent results, as seen in studies on green tea and fish oil, suggest that individual variability, dosage differences, and complex metabolic interactions may influence outcomes. Further research is needed to clarify these discrepancies and establish standardized protocols for incorporating functional foods into dietary recommendations for MASLD. Looking forward, the integration of functional foods into clinical practice for MASLD management among PLWH requires a multidisciplinary approach. Collaboration among nutritionists, clinicians, and researchers is essential to design robust clinical trials that account for patient-specific factors, such as HIV status, antiretroviral therapy regimens, and coexisting metabolic disorders. Moreover, advances in nutrigenomics and metabolomics offer exciting opportunities to personalize dietary interventions based on individual genetic and metabolic profiles.

In conclusion, antioxidant-rich functional foods hold significant promise for addressing the dual challenges of oxidative stress and metabolic dysfunction in MASLD among HIV-infected individuals. By bridging the gap between basic research and clinical application, these interventions could offer a sustainable and effective strategy for improving liver health and overall quality of life in this high-risk population. However, a concerted effort is needed to overcome current limitations, validate their benefits, and unlock their full therapeutic potential.

Abbreviations

ART: Antiretroviral Therapy
Bio-a: Biochanin A
DNJ: 1-deoxynojirimycin
EGCG: Epigallocatechin Gallate
EGW: Eriobotrya Japonica
GB: Glinkgolide B
gp120: Glycoprotein 120
HIV: Human Immunedeficiency Virus
IL: Interleukin
LRP: Leucine-arginine-proline
LQP: Leucine-glutamine-proline
MASLD: Metabolic dysfunction-associated Fatty Liver Disease
NAC: N-acetyl-L-cysteine
NAFLD: Non-alcoholic Fatty Liver Disease
P3G: Peonidin 3-O-glucoside
PLWH: People Living with HIV
PMF: Polymethoxyfalvone
PPC: Polyenylphosphatidylcholine
PUFA: Polyunsaturated Fatty Acid
MUFA: Monounsaturated Fatty Acd
ROS: Reacctive Oxygen Species
SCFA: Short Chain Fatty Acid
SG3: Sesquiterpene Glycoside 3
TFCH: Total Flavonoid
TXER: Troxerutin
TNF: Tumor Necrosis Factor
Vpr: Viral Protein
VTP: Vine Tea Polyphenol
WGHP: Walnut Green Husk Polysaccharide
WHO: World Health Organization

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Ethical Approval

Not applicable.

Consent to Participate

Not applicable.

Consent to Publication

Not applicable.

Availability of Data and Materials

Not applicable.

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Conflicts of Interest

The author declares that he has no conflicts of interest.

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