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Systematic Review

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Abbreviations:
AMPK, AMP-activated protein kinase; b.w., body weight; CAT, catalase; GSH-Px, glutathione peroxidase; MDA, malondialdehyde; Nrf2, nuclear-related factor-2; RCT, randomised controlled trial; ROS, reactive oxygen species; SOD, superoxide dismutase; TAC, total antioxidant capacity; TAS, total antioxidative status; TBARS, thiobarbituric acid reactive substances; TOS, total oxidative status

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Effects of black chokeberry *(Aronia melanocarpa)* supplementation on oxidative stress, inflammation and gut microbiota: a systematic review of human and animal studies

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Abstract

The scientific literature indicates that chokeberry is widely used as a supplement to support the maintenance of the body's homeostasis by reducing inflammation and oxidative stress. In recent years, positive effects of chokeberry on intestinal parameters have also been observed. Oxidative stress, inflammation and, according to recent reports, also the gut microbiome are closely related to the overall well-being and health of the population. This study, therefore, attempts to summarise all the health benefits of black chokeberry supplementation. This study was registered in PROSPERO (International Prospective Register of Systematic Reviews) under registration number CRD42023395969. Additionally, the systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) method. Electronic databases were searched in Web of Science, PubMed, Scopus and EBSCO using the following combination of the words 'chokeberry or aronia' and 'inflammation or oxidative stress or microbiota or microbiome or permeability or gut'. Ultimately, fifty-seven studies were summarised in the review. Data analysis showed that black chokeberry has a positive effect on the reduction of inflammation, oxidative stress and intestinal microflora, but the size of the changes varies and depends on many variables. Therefore, the researchers concluded that the compounds found in black chokeberry play a pivotal role in maintaining the overall balance within the system. This is a crucial consideration given the tendency for disturbances in organismal homeostasis to accompany disease processes and various disorders. However, further research is necessary to elucidate the mechanisms and optimise its use fully.

In the early 20th century, the prevailing trend was to study foods rich in phytochemicals and evaluate their bioactivity. Among all the berries, black chokeberry, *Aronia melanocarpa*, gained the most popularity as it is one of the richest plant sources of polyphenols[1]. Polyphenols, particularly anthocyanins, exhibit strong antioxidant properties. The literature indicates that the antioxidant capacity of chokeberry juice is four times higher than in cranberry, blueberry or red grape juice[2].

Within the bioavailability framework, contemporary research has elucidated that the constituents of chokeberry are detectable in systemic circulation and excreted in urine at nanomolar concentrations. Furthermore, studies have identified glucuronidation and methylation as primary metabolic pathways in the biotransformation of chokeberry-derived anthocyanins. It is noteworthy that the bioavailability of these anthocyanins is significantly modulated by extensive metabolism conducted by gut microbiota in the colon. This complex metabolic process produces a diverse spectrum of low molecular weight phenolic metabolites, which exhibit enhanced bioavailability and superior absorption rates[3].

According to the literature, black chokeberry *Aronia melanocarpa* exhibits strong antioxidant properties. The mechanisms underlying these properties are multifaceted, involving the enhancement and protection of enzymes such as paraoxonase, superoxide dismutase (SOD) and glutathione. Additionally, chokeberry inhibits the activity of several key enzymes involved in oxidative and inflammatory processes. Specifically, it reduces the activity of inducible nitric oxide synthase, which is responsible for producing nitric oxide in response to inflammatory stimuli and can contribute to oxidative stress and tissue damage. It also inhibits NADPH oxidase, an enzyme complex that generates reactive oxygen species (ROS) and plays a crucial role in the body's defense mechanism and pathology of various diseases. Furthermore, chokeberry suppresses lipoxygenase, an enzyme involved in PUFA metabolism to form pro-inflammatory leukotrienes. By inhibiting these enzymes, chokeberry helps to mitigate oxidative stress and inflammation, contributing to its overall protective effects against various diseases[2,4]. As other research shows, chokeberry anthocyanins also demonstrate protective effects against

the oxidation of α- and γ-tocopherol[5]. Research has shown that *Aronia melanocarpa* constituents can accumulate at the lipid bilayer-aqueous phase interface in erythrocyte membranes. Their localisation within the hydrophilic region of the membrane creates a protective barrier against free radicals, thereby enhancing the effectiveness and safety of these antioxidants[6].

Anthocyanins in black chokeberry also play a significant role in modulating inflammation. This regulatory effect is attributed to their ability to bind iron and regulate various immune system components involved in inflammatory processes[7]. The anti-inflammatory properties of black chokeberry are intricately linked to the enhancement of the human immune response. This involves the suppression of pro-inflammatory cytokines and the release of anti-inflammatory cytokines. The antiviral and antimicrobial properties of chokeberry additionally contribute to its anti-inflammatory effects[8].

Scientific evidence supports aronia supplementation's impact on clinical and sporting populations. This is mainly related to the activity of anthocyanin compounds, which have been shown to have strong antioxidant, anti-inflammatory and cardioprotective properties[1,9]. In individuals with metabolic disorders, such as diabetes, obesity and CVD, oxidative stress and chronic inflammation are critical factors contributing to the progression of these conditions. Research indicates that aronia supplementation can mitigate these pathological processes by reducing oxidative stress markers and inflammatory cytokines, thereby improving metabolic parameters such as lipid profiles, blood glucose levels and insulin sensitivity[10–12]. Moreover, in healthy individuals, particularly athletes, the high-intensity physical exertion associated with training and competition leads to an increased production of ROS, which can result in oxidative damage to cells and tissues. The antioxidant capacity of aronia can help neutralise these ROS, thereby reducing exercise-induced oxidative stress. This can potentially enhance recovery, reduce muscle damage and improve overall athletic performance. The anti-inflammatory effects of aronia may also contribute to faster recovery times and reduced post-exercise soreness[6,13].

Recent studies have established a connection between dysbiosis of the human gut and a variety of diseases, including obesity, diabetes, depression and irritable bowel syndrome. The gut microbiome plays a critical role in maintaining the integrity of the intestinal epithelial barrier, which is essential for the functional maturation of the gut immune system. Disruption of homeostasis in the gut can lead to systemic effects due to the leakage of the epithelial wall, allowing endotoxins and bacteria to enter systemic circulation and trigger endotoxemia and inflammatory responses. Polyphenols have been identified as modulators of the microbiome composition[14]. There likely exists a bi-directional relationship between the human gut microbiome and polyphenols, mirroring the interaction between polyphenols and the microbial population in the root systems of plants[15]. *Aronia melanocarpa*, a rich source of polyphenols, may have consequently a significant impact on the human gut microbiome, promoting gut health and potentially mitigating the risks associated with gut dysbiosis.

Therefore, this study focuses on a detailed investigation through literature, including all available articles from databases, to explore the impact of black chokeberry on oxidative stress, inflammation and intestinal parameters in both animal models and human subjects. Recognising the existence of prior research exploring the impacts of black chokeberry on these physiological factors, our systematic review distinguishes itself as the initial effort to amalgamate and integrate findings across these realms, providing a comprehensive elucidation of the biological effects of black chokeberry on living organisms. Furthermore, this comprehensive aggregation of research articles enables a detailed analysis of results attained to date, thereby identifying areas warranting further exploration and analysis.

This systematic review will address whether and how black chokeberry affects biomarkers of oxidative stress, inflammation and intestinal parameters and whether interactions exist between these biomarkers. Although there is ample evidence supporting the positive effects of chokeberry on oxidative stress, inflammation and intestinal parameters, the hypothesis carries some uncertainty. It should be noted that studies are conducted on different cohorts (animals, healthy individuals, patients, athletes) and the supplement comes in various forms (extract, juice, fresh fruit) and contains different concentrations of active compounds (anthocyanins, polyphenols). Additionally, the dose and duration of supplementation vary, and chokeberry is expected to affect various physiological and pathological states (high oxidative stress, inflammation, post-exercise recovery), each characterised by distinct mechanisms of action. Therefore, it is important to bear these limitations in mind when interpreting the results.

Materials and methods

Search strategy

This systematic literature review focused on the health benefits of black chokeberry *(Aronia melanocarpa)* supplementation. Due to the relatively low number of randomised controlled trials (RCT), animal model studies were also included in the review. For better interpretation and clarity, human and animal data were presented separately. The systematic review was conducted according to PRISMA (Preferred Reporting Items for Systematic Reviews and Meta-Analyses) principles. This study was registered with PROSPERO (International Prospective Register of Systematic Reviews) under registration number CRD42023395969. Electronic database searches were performed on the Web of Science, PubMed, Scopus and EBSCO. The search strategy for RCT and *in vivo* studies was the same by combining the words 'aronia *or* chokeberry' *and* 'inflammation *or* oxidative stress *or* microbiota *or* microbiome *or* gut permeability *or* gut'. The reference list of retrieved literature reviews was then manually searched to find potential articles that could be included in the systematic review. The study used a protocol involving simultaneous searches and separate presentation of results. This was similarly performed by Groulx *et al.*[16]. No restrictions on publication date or study type were applied to any search strategy. The search included original papers in English published before 01·05·2024.

Inclusion and exclusion criteria

The inclusion criteria used in the study:

Articles in English language

Participants: people of all ages, animals only *in vivo*

Supplementation with black chokeberry in any form (i.e. juice, extract, diet) and any dose (if the exact dose used in the study is given)

Published in full in a peer-reviewed journal

RCT and clinical trial concerns

Only the level 1 Oxford Centre for Evidence-Based Medicine scale (Table 1) concerns RCT

Table 1. The Oxford 2011 levels of evidence[17]

Evidence level (treatment benefits)
Level 1 – Systematic review of randomised trials or *n*-of-1 trials
Level 2 – Randomised trial or observational study with dramatic effect
Level 3 – Non-randomised controlled cohort/follow-up study
Level 4 – Case series, case–control study or historically controlled study
Level 5 – Mechanism-based reasoning

The exclusion criteria used in the study:

In vitro testing
The use of another kind of chokeberry (e.g. chokeberry 'Viking')
Observational studies, meta-analysis, systematic review,
Articles in not English language

Data extraction

Data were first evaluated by two investigators (S. K. and H. Dz.) and then was checked independently by two other supervisors (A. S-S. and A. K.). All articles were searched using the keywords. After that, all replicas were removed, and article abstracts were analysed based on the eligibility criteria. Finally, full texts of articles that met the eligibility criteria were analysed. Each publication selected for the review was critically evaluated. All articles' full texts were available online. Independently extracted data from studies were entered into two tables (for the RCT and the animal model).

The table for the RCT studies included study information such as first author and year of publication, Oxford Centre for Evidence-Based Medicine (OCEBM) level, research and control group characteristics and size, type of supplement, dose, duration of supplementation and direction of change in inflammatory, oxidative stress or gut microbiota. The table for *in vivo* studies included information such as first author and year of publication, characteristics and size of the model and control group, type of supplement, dose, duration of supplementation and direction of change in inflammatory, oxidative stress or gut microbiota.

Quality assessment

Following the analyses described above, the level of evidence in the RCT was assessed by four independent reviewers (H. Dz., A. K., S. K., M. P.) using the 2011 Oxford Centre for Evidence-Based Medicine method, developed by an international group of researchers involving clinicians, patients and investigators (Table 1). The Oxford Centre for Evidence-Based Medicine method enables the rapid identification of the best evidence, encouraging clinicians, researchers and patients to autonomously evaluate the evidence.

Subsequently, a bias analysis of human studies was performed by two investigators (H. Dz., M. P.) using the latest version of the Cochrane collaboration risk-of-bias tool (Table 2), which is used in randomised trials[18]. Studies were screened in five areas: bias due to the randomisation process, bias due to deviations from the intended innervation, bias due to missing outcome data, bias in the outcome measure and bias in the choice of reported outcomes. This tool allows the investigator to classify each domain as high risk, of some concern, or low risk. Some concern was found mainly in the randomisation process.

Analysis of bias of animal studies using the SYRCLE's ROB tool (Table 3) was performed by two investigators (M.P., S.K.). The SYRCLE tool is based on the Cochrane Collaboration Tool but contains ten entries. These entries are related to six types of bias: selection bias, performance bias, detection bias, attrition bias, reporting bias and other biases[19].

Statistical analyses

The quantitative data were presented in tables, without further statistics. The studies presented data in different formats and/or with different structures. Because of low heterogeneity in the studies (different cohorts, physiological states, supplement forms, doses and supplementation times), it was not possible to extract data for meta-analysis for statistical comparison. The summary tables contain data extracted from included studies containing participants' characteristics, doses, duration of supplementation and outcomes.

Results

The literature searches identified 960 potential articles. After removing 536 duplicates, 424 records were subjected to article title and abstract screening. Subsequently, the full texts of 120 articles were meticulously examined, including fifty-seven articles in the review (Fig. 1). These articles have been categorised into two tables: Table 4 comprises studies conducted utilising animal models, while Table 5 encompasses studies involving human subjects.

Studies conducted on an animal model

In Table 4, the total number of manuscripts is forty-five. The size of the groups is less than fifty in twenty-one articles[21–23,25,27,29,31,34–36,40–43,45,46,50,53,56,57,62,63]. The group size in the range of 50–100 is fourteen articles[20,24,26,28,30,33,38,39,47,49,52,55,58,71]. More than a group size of more than 100 participants is found in seven articles[32,37,48,54,59–61]. Moreover, twelve articles were about females[23,30,32,37,39,41,52,54,55,57,60,61]. Four articles[31,50,59,63] lack clearly defined sex of animals, while two articles discuss both males and females[35,62]. In other papers, the research was performed with males. Markers of oxidative stress were analysed in thirty-six articles[20,20,23–35,37–39,41–43,45,48–50,52–56,58–61,63,71], while indicators of inflammation were determined in seventeen studies[20,24,26,28,29,33,36,38–41,43,44,51,57,58,71]. Changes in gut microbiota were studied in eight articles[20,22,39,41,46,47,56,62].

Studies conducted with humans

In Table 5, the total number of articles is twelve. The group size is less than fifty in nine articles[6,7,11,13,64–67,69,70]. A group size of more than fifty participants occurs in three articles[12,68,69]. Five articles involved only men[6,7,13,67,68], and the remaining six manuscripts involved both sexes[11,12,64–66,69]. One article focused on women[70]. In five articles, the participants were athletes[6,7,13,65,67], the other two manuscripts were about patients[11,64] and the remaining articles refer to healthy people[12,66,68–70]. Markers of oxidative stress were analysed in nine articles[6,7,11,13,64–67,69]. Indicators of inflammation were analysed in five manuscripts[6,7,13,66,69], while intestinal parameters were indicated in only three[12,68,70].

Table 2. Cochrane collaboration risk-of-bias tool. Symbols used: +, low risk; ?, unclear risk; -, high risk

	Randomisation process	Deviation from the intended intervention	Measurement of the outcome	Selection of the reposted results	Overall
Duchnowicz (2012)	?	-	-	+	+
Broncel (2010)	+	+	-	+	?
Stankiewicz (2021)	+	+	-	+	+
Petrovic (2016)	+	+	+	+	+
Skarpańska-Stejnborn (2014)	+	+	+	+	+
Xie (2016)	+	-	+	+	+
Pilaczyńska-Szczęśniak (2005)	+	-	+	+	+
Istas (2019)	+	+	+	+	+
Le Sayec (2022)	+	+	+	+	+
Chung (2023)	?	?	?	+	+
Lacker (2024)	+	?	+	+	+
Stankiewicz (2023)	+	+	+	+	+

Effect of chokeberry on prooxidant–antioxidant balance parameters – human

Of the human studies, eight analysed markers were related to prooxidant–antioxidant balance, that is, thiobarbituric acid reactive substances (TBARS), GSH, SOD, catalase (CAT), total antioxidant capacity (TAC), glutathione peroxidase (GSH-Px), GSSG and malondialdehyde (MDA). Reduced levels of lipid peroxidation expressed as TBARS were observed in five studies[7,11,64,65,67]. Only one experiment showed no change in TBARS concentration[7]. One manuscript analysed GSH levels and showed a decrease in concentration[69]. The value of SOD was analysed in four studies[64,66,67,69]. In two manuscripts, no changes in SOD levels were observed[66,69]. One article showed an increase or no change depending on the length of supplementation[64], and one manuscript showed a decrease in SOD levels[67]. As for CAT, in one paper, there was a decrease in CAT concentration[64]. No change in CAT values was observed in two articles[66,69]. Only three manuscripts analysed the level of TAC values[6,7,13]. Two exhibits showed an increase in TAC values[6,13], and the other showed no change[7]. The GSH-Px index was analysed in three papers[66,67,69]. Two manuscripts showed a decrease in levels[67,69]. One paper showed no change in values. The GSSG parameter was analysed in only one article, where a reduction in values was observed[69]. Similarly, the biomarker MDA was analysed in only one manuscript where no changes were shown[69].

Effect of chokeberry on inflammation – human

Inflammation was analysed in five papers. IL-6 levels were analysed in five papers[6,7,13,66,69]. In three manuscripts, no changes were observed[6,7,66], while one experiment showed an increase in IL-6 levels[69], and one study showed a decrease[13]. TNF-α levels were analysed in two manuscripts[6,66]. One paper showed no change[66], and one paper observed a decrease in levels[6]. Myoglobin levels were analysed in only one manuscript, where no changes were shown[7]. IL-1β levels were studied in one study, which showed no change[66]. The C-reactive protein biomarker was analysed in one manuscript, and no changes were observed[66].

Effect of chokeberry on gut health – human

There were only three articles on humans considering gut health[12,68,70]. Nevertheless, α and β diversity remained unchanged[12,68], *Bacteroides* and its representative *Bacteroides xylanisolvens* increased[12,68] and *Haemophilus parainfluenzae* decreased in one study[12].

Effect of chokeberry on prooxidant–antioxidant balance parameters – an animal model

In thirty-six studies conducted on animal models, the effects of chokeberry on markers of antioxidation–peroxidation balance were measured, that is, GSH, GSH-Px, SOD, MDA, O_2, H_2O_2, HO^{-1}, TBARS, CAT, glutathione reductase, total antioxidative status (TAS), ROS, TAC, nuclear-related factor-2 (Nrf2) and TOS. The GSH index was analysed in nineteen manuscripts[20,23–25,28,30,35,41,49,50,53,55,60,61,63]. Eight articles showed an increase in the biomarker[20,23,24,28,31,35,49,50], while the rest showed no change[25,41,53,63]. In the seven studies, the change varied at different time points[30,32,52,54,55,60,61]. The GSH-Px parameter was analysed in twenty-three studies, and in fourteen studies observed an increase in its concentration[26,27,29,31–33,37,39,48,50,52,54,56,71]. Most of the six studies showed no change[25,34,42,45,60,63]. In the three studies, the change varied at different time points[30,55,61]. The SOD biomarker was analysed in twenty-nine papers. In eleven articles, an increase was observed[20,24,26–28,33,37,46,48,59,71]. Eight manuscripts showed no change[23,29,39,41,42,45,56,63], while three articles showed a decrease in SOD levels[25,34,50]. In the seven studies, the change varied at different time points[30,32,52,54,55,60,61]. The MDA index was determined in seventeen manuscripts, with thirteen articles showing a decrease[20,24,27,31,33,37,41,43,46,56,59,71] and the rest showing no change[26,38,45,53]. The TBARS biomarker was determined in seven manuscripts. Its reduction was observed in five articles[23–25,29,34], while no change was observed in two[49,63]. The parameters H_2O_2 were determined in six articles. A reduction was observed in three articles[23,32,48]. In the three studies, the change varied at different time points[52,60,61]. In one study, an increase in the concentration of HO^{-1}[46] was observed, and in another, a decrease[58]. The CAT biomarker was

Table 3. SYRCLE's risk-of-bias tool for animal studies. (1) sequence generation; (2) baseline characteristics; (3) allocation concealment; (4) random housing; (5) blinding of experimentalists; (6) random for outcome assessment; (7) blinding of outcome assessors; (8) incomplete outcome data; (9) selective outcome reporting; (10) other biases. Symbols used: +, low risk; ?, unclear risk; -, high risk

	1	2	3	4	5	6	7	8	9	10
Zhao (2022)	+	+	?	?	?	?	?	?	?	?
X Liu (2021)	+	?	?	?	?	?	?	?	?	+
Zhu (2020)	+	+	+	?	?	?	?	+	?	?
Rudic (2022)	?	?	?	?	?	?	?	?	?	?
Li (2021)	+	?	?	?	+	?	+	?	?	?
Kujawska (2010)	+	?	?	?	?	?	?	?	?	?
Wei (2017)	+	+	?	?	?	+	?	?	+	?
Ma (2022)	+	+	+	?	?	?	?	?	?	?
Yang (2020)	+	+	+	?	?	?	?	?	?	?
Piotrowska–Kempisty (2020)	+	+	?	?	+	?	?	?	?	?
Dąbrowski (2020)	+	+	+	?	?	?	?	+	?	?
Wang (2020)	+	?	?	?	?	?	?	?	?	?
Ciocoiu (2013)	-	+	?	?	?	?	?	?	?	?
Mężyńska (2019)	+	+	+	?	?	?	?	+	?	?
Wang (2019)	+	+	?	?	?	?	?	?	?	?
Cujića (2018)	+	+	?	?	?	?	?	+	?	?
Pavlova (2024)	+	+	?	?	?	?	?	?	?	?
Jiao (2021)	+	?	?	?	?	?	?	+	?	?
Jing (2022)	+	+	+	+	?	+	?	+	?	?
Valcheva-Kuzmanowa (2014)	-	+	?	?	?	?	?	?	?	?
Liu (2021)	-	+	?	?	?	?	?	+	?	?
Yu (2021)	+	?	?	?	?	?	?	?	?	?
Ren (2022)	+	+	-	?	?	?	?	+	?	?
Kim (2013)	+	?	?	?	?	?	?	?	?	?
Jeong (2017)	+	+	?	?	?	?	?	?	?	?
Ohgami (2005)	-	+	?	?	?	?	?	?	?	?
Song (2018)	+	+	?	?	?	?	?	?	?	?
Zhao (2021)	+	+	?	?	?	?	?	?	?	?
Zhu (2022)	+	+	?	?	?	?	?	?	?	?
Brzóska (2016)	+	+	?	?	?	?	?	?	?	?
Faff and Frankiewicz-Jóźko (2003)	+	+	?	?	?	?	?	?	?	?
Lipińska (2017)	+	?	?	?	?	?	?	?	?	?
Gajic (2020)	-	?	?	?	?	?	?	?	?	?
Onopiuk (2021)	+	+	?	?	?	?	?	+	?	?
Valcheva-Kuzmanowa (2005)	+	?	?	?	?	?	?	?	?	?
Mężyńska (2018)	+	+	+	?	?	?	?	+	?	?
Dąbrowska (2019)	+	+	+	?	?	?	?	+	?	?
Xing (2023)	+	+	+	?	?	?	?	?	?	?
Liu (2023)	+	+	?	?	?	?	?	?	?	?
Wei (2023)	+	+	?	?	?	?	?	?	?	?
Wang (2023)	+	+	+	?	?	+	?	?	?	?
Smereczański (2023)	+	?	?	?	?	?	?	?	?	?

(*Continued*)

Table 3. (*Continued*)

	1	2	3	4	5	6	7	8	9	10
Ruczaj (2024)	+	+	?	?	?	?	?	?	?	?
Wilson (2023)	–	?	?	?	?	?	?	?	?	?
Doma (2023)	+	+	?	?	?	?	?	?	?	?

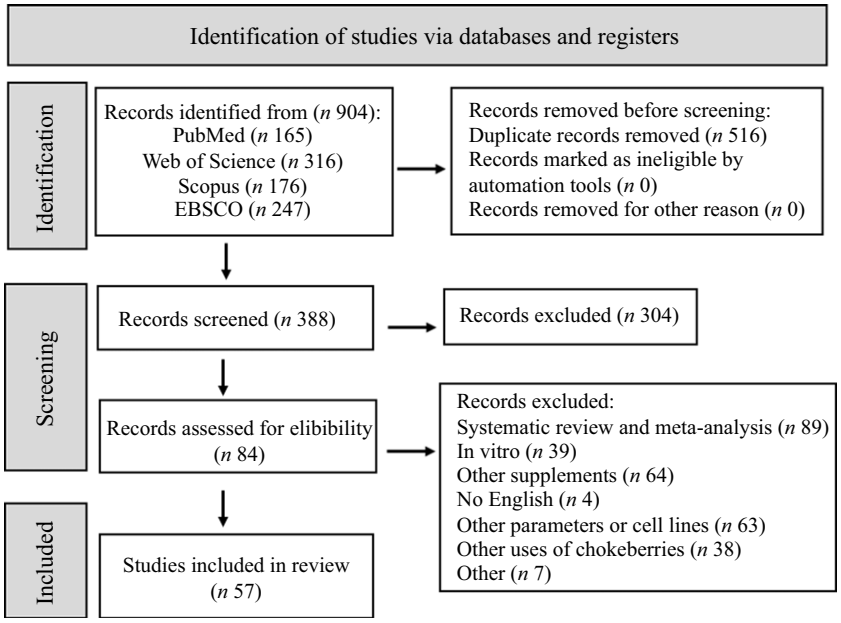

Figure 1. PRISMA flow diagram illustrating the search and selection of studies.

studied in twenty-three manuscripts. Eleven articles showed an increase in concentration[23,24,29,30,33,42,46,48,59,61,71], seven studies showed no change[25,34,39,41,45,56,63], and in five articles, the change varied at different time points[32,52,54,55,60]. The glutathione reductase biomarker was analysed in eight manuscripts. No changes were observed in three studies[25,29,63]. The level was lower in one manuscript[48], and in four studies, the change varied at different time points[32,54,60,61]. The TOS and TAS index were determined in four studies with the change varying at different points in time[48,52,60,61]. The TAC biomarker was analysed in four manuscripts. Three papers showed an increased concentration[31,37,41], while two more showed no change[39]. Nrf2 was determined in four studies, and an increase in its concentration was observed in all of them[27,37,46,59].

Effect of chokeberry on inflammation – an animal model

Indicators of inflammation were determined in seventeen manuscripts. TNF-α was the most frequently analysed biomarker; in eight out of ten articles, a reduction was observed[28,33,36,40,41,44,57,58], while one study showed no change[29]. One study observed no change before renal injury and a reduction after renal injury[24]. IL-6 was the second most frequently analysed biomarker, with eight manuscripts showing a reduction[28,33,36,38,41,57,58,71]. One study showed no change before kidney injury and a reduction after kidney injury[24]. IL-1β levels were determined in five manuscripts. Four papers showed its reduction[33,36,41,57], while one paper observed no change before kidney injury and a reduction after kidney injury[24]. NF-kB factor was also analysed in four studies. In each study, the results indicated a decrease in concentration[36,40,43,46]. IL-10 was determined in three articles, with two papers showing a reduction[33,41] and one showing no change[38]. IL-1 was determined in three manuscripts, and reductions were observed[26,28,58]. The COX2 index in the two articles was reduced[26,28]. Similarly, the transforming growth factor-β1 index was determined in two articles[26,29].

Effect of chokeberry on gut health – an animal model

Eight research teams checked gut health in an animal model[20–22,41,46,47,56,62]. A diversity was raised in three studies[39,47,62] and remained unchanged in three studies[22,41,47]; in one study, this indicator decreased[20]. Four studies found an increase in *Bacteroides* and *Bacteroidetes*[22,41,46,47]. The *Firmicutes*: *Bacteroides* ratio is inconclusive; it decreased in two studies[22,47]. Phyla *Verrucomicrobia* increased in two studies[22,47]; also, *Prevotella* increased in three studies[22,41,47].

Discussion

Effects of black chokeberry on prooxidant–antioxidant balance parameters

After a thorough review of the available research, it can be concluded that compounds derived from chokeberry offer significant efficacy in reducing oxidative stress. This reduction is associated with the scavenging of free radicals, inhibition of lipid peroxidation and modulation of both enzymatic and non-enzymatic antioxidant activities[20,24,27,71]. Nevertheless, the interplay among specific biomarkers fluctuates across studies conducted on diverse cohorts, employing varying supplementation

Table 4. Summary of studies on the effects of chokeberry on inflammation, oxidative stress and intestinal parameters – an animal model

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Zhao *et al.* (2022)[20]	Male ICR mice (4–6 weeks old), *n* 50, Model group (thioacetamide-induced liver fibrosis), *n* 10 Aronia (AMP-L), *n* 10 Aronia (AMP-H), *n* 10	Chokeberry extract	200 or 400 mg/kg b.w./d	4 weeks		↑ GSH, ↑ SOD, ↓ MDA	↑ Shannon index, ↑ Simpson index, ↑ PCoA, ↑ The ratio of *Bacteroidetes* to *Proteobacteria*
X Liu *et al.* (2021)[21]	Male mice (10 weeks old), *n* 24, Control group, *n* 6 Aronia berry (AB, *n* 6 Aronia extract (AE), *n* 6	AB and aronia extract	AB = 6300 mg/kg b.w./d	One week			Whole groups: ↑ Shannon index, ↑ PCoA, Aronia berry-supplemented diet: ↑ *Erysipelotrichia* (marker of diet) ↓ *Bacilli*, Aronia extract-supplemented diet: *Proteobacteria* and *Deltaproteobacteria* (marker of diet)
Zhu *et al.* (2020)[22]	Male rats (aged 6 weeks) *n* 38, HF group (high-fat diet) *n* 8 Aronia (continually fed with high-fat diet (HFD) + AM) *n* 10	Chokeberry extract	1000 mg/kg b.w./d	40 d			↔ ACE, ↔ Chao1 index, ↔ Shannon index, ← Simpson index, ≠ PCoA ↓ F/B ratio, ↑ *Bacteroidetes*, ↓ *Firmicutes*, ↑ *Verrucomicrobia*, ↑ *Bacteroides*, ↑ *Romboutsia*, ↑ *Prevotella*, ↑ *Akkermansia*, ↑ *Marvinbryantia*, ↑ *Anaerofilum*, ↓ *Clostridium*, ↓ *Ruminococcaceae_UCG- 013*, ↓ *Ruminococcaceae_NK4A214_group*, ↓ *Roseburia*, ↓ *Desulfovibrio*, ↓ *Ruminiclostridium_6*, ↓ *Eubacterium_coprostanoligenes _group* Group markers: *Bacteroides*, *Prevotella* and *Ruminococcus_1*
Rudic *et al.* (2022)[23]	Model adult female rats with PCOS, *n* 30 Model group (P), *n* 6 Aronia group (P + A), *n* 6	Standardised *Aronia melanocarpa* extract	0·45 ml/kg b.w./d	28 d		↓ O_2, ↓ H_2O_2, ↓ TBARS, ↔ nitrites, ↔ SOD, ↑ GSH, ↑ CAT.	

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Table 4. (*Continued*)

Li *et al.* (2021)[24]	Male mice (10–12 weeks old), *n* 52, *n* 13 AC (anthocyanin mixture), *n* 13 C-3-A (cyanidin-3 Arabinoside), *n* 13 C-3-GA (cyanidin-3-galactoside), *n* 13 C-3-GL (cyanidin-3-glucoside), *n* 13	Anthocyanin mixture	50 mg/g b.w./twice daily corresponding to 25 ml/g b.w./twice daily	14 d	Before the renal IR injury: ↔ IL-1β, ↔ IL-6, ↔TNF-α, ↔ MCP-1 After renal IR injury: ↓ IL-1β, ↓TNF-α, ↓MCP-1, ↓IL-6	↑ SOD, ↑ CAT, ↑ GSH, ↓ MDA, ↓ TBARS
Kujawska *et al.* (2010)[25]	Male rats, *n* 48, Group I (control), *n* 8 Group II (aronia juice), *n* 8	Chokeberry juice	10 ml/kg b.w./d	28 d		↓ TBARS, ↓ SOD, ↔ GSH, ↔ CAT, ↔ GSH-Px, ↔ GR
Wei *et al.* (2017)[26]	Male mice (6–8 weeks) *n* 60, Aged model group, *n* 12 Anthocyanins low-dose group, *n* 12 Anthocyanins high-dose group, *n* 12	Chokeberry extract	Low dose: 15 mg/kg b.w./d High dose: 30 mg/kg b.w./d	8 weeks	↓ IL-1, ↓ COX-2, ↓ TGF-β1	In serum: ↑ GSH-Px, ↑ T-SOD, ↔ MDA
Ma *et al.* (2022)[27]	The male mouse model of pulmonary fibrosis, *n* 40, Group 3 (SP group), *n* 8 Groups 4 C3G (100 mg/kg), *n* 8 Group 5 C3G (200 mg/kg), *n* 8	Chokeberry extract	100 and 200 mg/kg b.w. in distilled water/d	56 d		In lung tissue: ↑ SOD, ↑ GSH-Px ↓ MDA
Yang *et al.* (2020)[28]	Hepatic fibrosis male mice (6 weeks old), *n* 50, Model group, *n* 10 AMA high-dose group (CCl4 + AMA-40), *n* 10 AMA low-dose group (CCl4 + AMA-20), *n* 10	Chokeberry fruits	High-dose group: 40 mg/kg Low-dose group: 20 mg/kg	8 weeks	↓ IL-1, ↓ IL-6, ↓ COX-2, ↓ TNF-α	↑ SOD, ↑ GSH

(*Continued*)

Table 4. (*Continued*)

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Piotrowska-Kempisty *et al.* (2020)[29]	Liver fibrosis rats model, *n* 48, Model group, *n* 8 Aronia, *n* 8	Chokeberry juice	100 ml/kg b.w./d	6 weeks	↔ TGFβ, ↔ TNFα	↑ CAT, ↑ GSH-Px, ↓ GST, ↓ TBARS, ↔ SOD, ↔ GR	
Dąbrowski *et al.* (2020)[30]	Rat female model of Cd toxicity (3–4 weeks old), *n* 96, Model group CD_1 i CD_5, *n* 16 Aronia group (AME + CD_1), *n* 16 group, Aronia group (AME + CD_5), *n* 16	Chokeberry extract	0·1 % water solution of extract of polyphenols, which means no less than 51·7–104·6 mg/kg b.w./d	3 and 10 months		↔GSH-Px (after 3 months), ↑ GSH-Px (after 10 months), ↑ CAT (after 3, 10 months), ↑ GSH (after 3, 10 months), ↑ SOD (after 3, 10 months), ↑ TAS (after 3, 10 months)	
Wang *et al.* (2020)[71] numerals in parentheses	Mice male model of alcohol liver injury (8 weeks old), *n* 50, Model group *n* 10 AM group 0·5 g, *n* 10 Am group 2 g, *n* 10	Chokeberry fruit	500 or 2000 mg/kg b.w./d	6 weeks	↓ IL-2, ↓ IL-4, ↓ IL-6	↓ MDA, ↓ ROS, ↑ CAT, ↑ SOD, ↑ GSH-Px	
Ciocoiu *et al.* (2013)[31]	Model of arterial hypertension rats, *n* 48, Model Group AHT (L-NAME 40 mg/kg b.w./d), *n* 12 Aronia group (AHT + P), *n* 12	Chokeberry extract	5 mg/kg b.w./every 2 d	8 weeks		↓ MDA, ↑ GSH-Px, ↑ GSH, ↑ TAC	
Mężyńska *et al.* (2019)[32]	Rat female model of Cd toxicity (3–4 weeks old), *n* 192 Model group CD_1 i CD_5, *n* 32 Aronia group (AME + CD_1), *n* 32 group, Aronia group (AME + CD_5), *n* 32	Chokeberry extract	63·1–159·1 mg/kg b.w./d	3, 10, 17, 24 months		↔ SOD (after 3, 10 months), ↑ SOD (after 17, 24 months), ↔ CAT (after 3, 10, 17 months), ↑ CAT (after 24 months), ↑ GSH-Px (after 3, 10, 17, 24 months), ↑ GR (after 3 and 24 months), ↔ GR (after 10, 17 months), ↔ GSH (after 3 months) ↑ GSH (after 10, 17, 24 months), ↓ GSSG (after 3, 10, 17, 24 months),	

Table 4. (*Continued*)

						↓ H_2O_2 (after 3, 10, 17, 24 months)
Wang *et al.* (2019)[33]	Male rats (8 weeks old), *n* 60, Model group (group receiving colchicine), *n* 12 Aronia group, *n* 12	Chokeberry fruit	500 mg/kg b.w./d or 2000 mg/kg b.w./d	8 d	↓IL-1β, ↓IL-6, ↓IL-10, ↓TNF-α	↓ MDA, ↑ SOD, ↑ CAT, ↑ GSH-Px
Ćujića *et al.* (2018)[34]	Rat male model of hypertension (6 months old), *n* 19, Model group, *n* 10 Aronia group, *n* 10	Chokeberry extract	50 mg/kg b.w./d	4 weeks (28 d)		↓ TBARS, ↓ SOD, ↔ GSH-Px, ↔ CAT
Pavlova *et al.* (2024)[35]	Mice male and female (aged 3 months), *n* 24 Model group (DOX-treated), *n* 6 Aronia group (DOX+ aronia), *n* 6	Aronia extract	20 % water solution	28 d		↑ GSH
Jiao *et al.* (2021)[36]	Male rats (5 weeks old), *n* 30, Model group (HFD), *n* 24 Aronia group low dose, *n* 6 Aronia group high dose, *n* 6	Powder (cyanidin-3-O-galactoside) from *Aronia melanocarpa*	100 and 200 mg/kg b.w./d)	8 weeks	↓TNF-α, ↓ IL-6, ↓ IL-1β, ↓ NF-κB	
Jing *et al.* (2022)[37]	Hens, *n* 300, Control group *n* 60 Aronia 1 % group, *n* 60 Aronia 4 % group, *n* 60 Aronia 7 % group, *n* 60	Chokeberry powder	Diet supplemented with (1, 4 and 7 %) concentrations of AM dried fruit powder of AM)	8 weeks		↓ MDA, ↑ SOD, ↑ GSH-Px, ↑ TAC, ↑ Nrf2
Valcheva-Kuzmanova *et al.* (2014)[38]	Male rats (age 4 months), *n* 96, Control group, *n* 24 Aronia 5 ml group, *n* 24 Aronia 10 ml group, *n* 24	Chokeberry juice	5 ml/kg and 10 ml/kg		↓ IL-6, ↔ IL-10	↔ MDA
Liu *et al.* (2021)[39]	Pigs at weaned stage, *n* 96, Control group, *n* 24 Aronia 0·5 ‰ group, *n* 24 Aronia 1 ‰ group, *n* 24 Aronia 2 ‰ group, *n* 24	*Aronia melanocarpa* Pomace	0·5, 1 and 2 ‰ Aronia into the basal diet	28 d	↔ IgA, ↔ IgG, ↔ IgM	↑ GSH-Px, ↔ SOD, ↔ CAT, ↔ TAC

(*Continued*)

Table 4. (*Continued*)

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Yu *et al.* (2021)[40]	Mice, *n* 35, Control group (high-fat (HF)/high-sucrose (HS), *n* 9 Aronia group (HF/HS + AM), *n* 10	Aronia berry extract powder	0·2 % ARN powder in experimental diets	14 weeks	↓ NF-KB p65, ↓ TNF-α		
Ren *et al.* (2022)[41]	Growing pigs, *n* 27 Control group, (CON group), *n* 9 Aronia (AMP 4 %) group, *n* 9 Aronia (AMP 8 %) group, *n* 9	Chokeberry juice	4 or 8 % Aronia of basic diet	7 weeks	↑ IgA, ↑ IgG, ↑ IgM, Jejunal 4 % Aronia: ↓ IL-1β, ↓IL-6, ↓IL-8, ↓IL-10, ↓TNF-α, Jejunal 8 % AMP: ↓ IL-6, ↓IL-8, ↓IL-10, ↓TNF-α, ↔ IL-1β	Jejunum: ↑ TAC, ↔ SOD, ↔ CAT, ↔ GSH, ↓ MDA Serum without changes	↔ ACE, Chao1, Shannon or Simpson phyla, ↓ *Proteobacteria* 4 % AMP significantly increased the: ↑ *Bacteroidetes* ↑ *Lachnospira*, ↑ *Solobacterium*, ↑ *Prevotella,* ↑ *Romboutsia*, ↑ *Butyrivibrio,* ↓ *Escherichia-Shigella,* ↓ *Pseudoscardovia,* ↑ Zonulin 1, ↑Claudin-1, ↑ Occludin, The 8 % AMP: ↑ *Robinsoniella*, ↑ *Prevotella_9*, ↓ *Escherichia- Shigella*, ↓ *Pseudoscardovia*, ↑ Zonulin 1, ↑ Claudin-1, ↔ Occludin
Kim *et al.* (2013)[42]	Mice (8-week-old), *n* 20, Control group, *n* 10 Aronia 0·05 % group, *n* 10 Aronia 0·005 % group, *n* 10	Chokeberry extract	0·005 % or 0·05 % Aronia of basic diet	4 weeks		↑ PON-1, ↑ CAT, ↔ SOD, ↔ GSH-Px	
Jeong *et al.* (2017)[43]	The mouse model of D-galactose-induced aging, *n* 35, Model group, *n* 7 Aronia group, *n* 7	Chokeberry powder	0·5 or 1 % Aronia of basic diet	8 weeks	↓ NF-κB	↓ MDA, ↓ AGA, ↑ Nrf2	
Ohgami *et al.* (2005)[44]	Rat male (8-week-old) Model (LPS induced uveitis) group, *n* 8 Aronia 1 mg group, *n* 8 Aronia 10 mg group, *n* 8	Chokeberry extract	1 mg, 10 mg or 100 mg	Administered once intravenously	↓ TNF-α, ↓ NO		

Table 4. (*Continued*)

	Aronia 100 mg group, *n* 8						
Song *et al.* (2018)[45]	Mouse male aging model (8-week-old), *n* 35, Model group, *n* 7 Aronia group, *n* 7	Chokeberry fruits	1 % chokeberry of basic diet	8 weeks		↔ MDA (in serum), ↔ CAT, ↔ SOD, ↔ GSH-Px	
Zhao *et al.* (2021)[46]	The aging mice male model (6–8 weeks old), *n* 40 Model group, *n* 10 Aronia 100 mg group, *n* 10 Aronia 200 mg group, *n* 10		100 and 200 mg/kg b.w./d	6 weeks	↓ NF-κB, ↓ IκB-α	↓ MDA, ↑ SOD, ↑ CAT, ↑ Nrf2, ↑ HO-1	↑ *Bacteroides*, ↓ *Firmicutes*
Zhu *et al.* (2022)[47]	The rat model of obesity *n* 28 Model (HFD) group, *n* 8 Aronia group, *n* 10	Chokeberry extract	1000 mg/kg b.w./d	40 d			↔ ACE, Chao1, Shannon and Simpson ↓ F/B *Phylum* ↑ *Bacteroidetes*, ↑ *Verrucomicrobia* ↓ *Firmicutes* Genus level, ↑ *Akkermansia*, ↑ *Bacteroides*, ↑ *Romboutsia*, ↓ *Lachnospiraceae*, ↑ *Prevotella*, ↓ *Clostridium*, ↓ *Desulfovibrio*, ↓ *Lachnoclostridium*
Brzóska *et al.* (2016)[48]	Cd toxicity in rat model, *n* 192 Model group CD_1 i CD_5, *n* 32 Aronia group (AME + CD_1), *n* 32 Aronia group (AME + CD_5), *n* 32	Aronia powder	0·1 % aqueous solution	3, 10, 17 and 24 months		In the serum: ↑TAS (after 3, 10, 17, 24 months), ↓ TOS (after 3, 10, 17, 24 months). In the bone: ↑ TAS (after 3, 10 months), ↔ TAS (after 17, 24 months), ↔ TOS (after 3, 10 months) ↓ TOS (after 17, 24 months), ↑ GSH-Px (after 3, 10, 17, 24 months), ↓ GR (after 3, 10, 17, 24) ↑ SOD (after 3, 10, 17, 24 months),	

(*Continued*)

Table 4. (*Continued*)

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
						↑ CAT (after 3, 10, 17, 24 months), ↓ H_2O_2 (after 3, 10, 24 months)	
Faff and Frankiewicz-Jóźko (2003)[49]	Male rats exercise model *n* 60, control-exercise group, *n* 15 Aronia (AM-fed-exercise (AE)), *n* 15	Chokeberry extract	0·7 mg × kg^{-1} body mass 1:10 water solution	4 d		↔ TBARS ↑ GSH in the heart and the red portion of the gastrocnemius muscle	
Lipińska *et al.* (2017)[50]	Polish Merino lambs *n* 48, Control group, *n* 16 Aronia 150 g group, *n* 16 Aronia 300 g group, *n* 16	Chokeberry pomace	150 g or 300 g of chokeberry pomace/kg of the complete compound feed/d	90 d		↓ SOD, ↑ GSH-Px, ↑ GSH	
Gajic *et al.* (2020)[51]	Mice male (2 months old) *n* 21, Control group, *n* 7 Aronia 50 mg group, *n* 7 Aronia 200 mg group, *n* 7	Chokeberry Extract	200 mg/kg b.w. or 50 mg/kg b.w./d	7 d	↑ IFN-γ, ↔ IL-17		
Onopiuk *et al.* (2021)[52]	Cd toxicity in rat female model (3–4 week-old) *n* 96, Model group CD_1 i CD_5, *n* 16 Aronia group (AME + CD_1), *n* 16 Aronia group (AME + CD_5), *n* 16	Chokeberry Extract	0·1 % aqueous extract from chokeberries ranged from 78·70 mg/kg b.w. to 153·82 mg/kg b.w./d	3, 10 months		↔ CAT, ↑ TAS (after 3, 10 months), ↓ TOS (after 3, 10 months in AME + CD5), ↔ TOS (after 3 months in AME + CD_1) ↑ SOD (after 3, 10 months in AME + CD_5), ↔ SOD (after 3 months in AME + CD_1) ↑ CAT (after 3, 10 months in AME + CD_5), ↔ CAT (after 10 months in AME + CD_1) ↑ GSH-Px (after 3, 10 months)	

Table 4. (*Continued*)

					↑ GSH (after 3, 10 months in AME + CD_5), ↔ GSH (after 3 months in AME + CD_1) ↔ H_2O_2 (after 3 months) ↓ H_2O_2 (after 10 months)
Valcheva-Kuzmanova *et al.* (2005)[53]	Rat model of gastric mucosal damage, *n* 48, Model group, *n* 6 Aronia 5 ml group, *n* 6 Aronia 10 ml group, *n* 6 Aronia 20 ml group, *n* 6	Chokeberry juice	5, 10 or 20 ml/kg b.w.	1 dose	↔ MDA, ↔ GSH, ↔ GSSG
Mężyńska *et al.* (2018)[54]	Rat female model of Cd toxicity (3–4 weeks old), *n* 192, Model group CD_1 i CD_5, *n* 32 Aronia group (AME + CD_1), *n* 32 Aronia group (AME + CD_5), *n* 32	Chokeberry extract	63·1–159·1 mg/kg b.w./d	3, 10, 17 and 24 months	↔ SOD (after 3, 10 months), ↑ SOD (after 17, 24 months), ↔ CAT (after 3, 10, 17 months), ↑ CAT (after 24 months), ↑ GSH-Px (after 3, 10, 17, 24 months), ↑ GR (after 3, 24 months), ↔ GR (10, 17 months), ↑ GSH (after 10, 17, 24 months), ↔ GSH (after 3 months), ↓ GSSH (after 3, 10, 17, 24 months).
Dąbrowska *et al.* (2019)[55]	Cd model of toxicity (3–4 weeks old) *n* 96, Rat female model of Cd toxicity (3–4 weeks old), *n* 96, Model group CD_1 i CD_5, *n* 16 Aronia group (AME + CD_1), *n* 16, Aronia group (AME + CD_5), *n* 16	Chokeberry extract	51·7–104·6 mg/kg b.w./d	3 and 10 months	↔ TAS (after 3 months ↑ TAS (after 10 months) ↑ CAT (after 3, 10 months) ↑ SOD (after 3, 10 months) ↑ GSH-Px (after 3, 10 months) ↔ GSH (after 3, 10 months)

(*Continued*)

Table 4. (*Continued*)

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Xing *et al.* (2023)[56]	Rat male model of age-related macular degeneration, *n* 40 Model group, *n* 10 Aronia (AAE) group, *n* 10	Chokeberry extract	600 mg/kg b.w./d in distilled water was administrated	28 d		↔ CAT, ↔ SOD, ↑ GSH-Px, ↓ MDA	↓ Chao1 index ↓ Simpson index ↔ *Lactobacillus*, ↔ *Alistipes*, ↔ *Parabacteroides*, ↔ *Akkermansia*, ↔ *Escherichia*, ↔ *Parasutterella*
Liu *et al.* (2023)[57]	Mouse female model of PM (particulate matter in the atmosphere), *n* 40 Model group, *n* 8 Aronia group 3 (PM10 + C3G 75), *n* 8 Aronia group 4 (PM10 + C3G150), *n* 8 Aronia Group 5 (PM10 + C3G300)	Cyanidin-3-galactoside extract	75 mg/kg b.w./d or 150 mg/kg b.w./d or 300 mg/kg b.w./d	28 d	↓ IL-1β, ↓ IL-6, ↓ TNF-α		
Wei at al. (2023)[58]	Mice male model of alcoholic liver disease, *n* 80 Model group (42 % alcohol), *n* 10 Aronia (low-dose AMA) group, *n* 10 Aronia (high-dose AMA) group, *n* 10	Chokeberry fruits	20 mg/kg b.w./d or 40 mg/kg b.w./d	8 weeks	↓ IL-1, ↓ TNFα, ↓ IL-6	↑ GSH-Px, ↓ HO-1, ↑ Nrf2	
Wang *et al.* (2023)[59]	1. Crabs long-term ammonia exposure, *n* 600 Aronia (AME1) group *n* 25 Aronia (AME3) group, *n* 25 Aronia (AME5) group, *n* 25 2. Crabs after acute ammonia exposure, *n* 300 Aronia (AME3) group, *n* 25	Chokeberry extract	1, 3 and 5 % Aronia of basic diet	4 weeks		Long-term ammonia exposure: ↓ MDA, ↑ CAT, ↑ SOD, acute ammonia exposure: ↓ MDA, ↑ CAT, ↑ SOD.	
Smereczański *et al.* (2023)[60]	Rat female model of Cd toxicity (3–4 weeks old), *n* 192 Model group CD1 i CD5, *n* 32	Chokeberry extract	0·1 % aqueous solution (1 g of the powdered extract was dissolved in 1 L water)	3, 10, 17 and 24 months		In the kidney: ← TAS (after 3, 10, 17, 24 months), ← TOS (after 3, 10, 17 months)	

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Table 4. (*Continued*)

	Aronia group (AME + CD_1), *n* 32, Aronia group (AME + CD_5), *n* 32				↓ TOS (after 24 months) ↑ CAT (after 3, 17, 24 months) ← CAT (after 10 months) ← SOD (after 3 months) ↑ SOD (after 10, 17, 24 months), ← GSH-Px, ← GR (after 3, 10, 17 months), ↓ GR (after 24 months), ← GSH, ← GSSG, ← H_2O_2
Ruczaj *et al.* (2024)[61]	Rat female model of Cd toxicity (3–4 weeks old), *n* 192 Model group CD1 i CD5, *n* 32 Aronia group (AME + CD1), *n* 32, Aronia group (AME + CD5), *n* 32	Chokeberry extract	0·1 % Aronia into the basal diet	3, 10, 17 and 24 months	↑ CAT (after 3, 10, 17, 24 months), ↔ SOD (after 3 months), ↑ SOD (after 10, 17, 24 months), ↔ GSH-Px (after 3, 10 months), ↑ GSH-Px (after 17, 24 months), ↔ GR (after 3, 10), ↓ GR (after 17, 24 months), ↔ GSH (after 3 months), ↑ GSH (after 10, 17, 24 months), ↔ GSSG (after 3, 10, 17 months), ↓ GSSG (after 24 months), ↔TOS (after 10 months), ↓ TOS (after 3, 17, 24 months), ↔ TAS (after 3, 10 months), ↑ TAS (after 17, 24 months), ↔ H_2O_2 (after 3 months), ↓ H_2O_2 (after 10, 17, 24 months)

(*Continued*)

Table 4. (*Continued*)

Author	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Wilson *et al.* (2023)[62]	Mice *n* 14, Control (sugar-matched juice) CON_{LO}, *n* 3, CON_{HI}, *n* 3. Aronia ARO_{LO}, = 3, ARO_{HI}, *n* 5 $_{LO}$-low inflammation $_{HI}$- high inflammation	Chokeberry juice	150 ml juice/week per cage	8 weeks			↑ Shanon index, ↑ *Eisenbergiella*, ↑ *Faecalibacillus*, ↑ Faecaelbacillus, ↑ *Howardella*, ↑ *Eggerthellaceae*
Doma et al (2023)[63]	Rat *n* 40, Control group, *n* 10, Aronia (CA) group, *n* 10	Chokeberry extract	6 % aqueous extract as drinking water	4 weeks		↔ CAT, ↔ SOD, ↔ TBARS, ↔ GSH-Px ↔ GSH, ↔ GR	

Abbreviations: ↔ *No change;* ↓ decrease; ↑ increase; ≠ not homogeneous; b.w., body weight; ACE, abundance-based coverage estimator; CAT, catalase; COX-2, cyclooxygenase-2; Chao1, total number of species in a sample; CRP, C-reactive protein; GSH-Px, glutathione peroxidase; GR, glutathione reductase; GST, glutathione transferases; IFN-*γ*, interferon-*γ*; H_2O_2, hydrogen peroxide; F/B ratio, *Firmicutes:Bacteroides* ratio; TLR4, toll-like receptor 4; PI3HO-1, haem oxygenase-1; MDA, malondialdehyde; MCP-1, monocyte chemoattractant protein-1; Nrf2, nuclear-related factor-2; PCoA, principal coordinate analysis; ROS, reactive oxygen species; SOD, superoxide dismutase; TAC, total antioxidant capacity; TGF-*β*1, transforming growth factor-*β*1; TAS, total antioxidative status; TBARS, thiobarbituric acid reactive substances; TOS, total oxidative; NO, nitric oxide.

doses and durations. For instance, Zhao *et al.* administered chokeberry extract at doses of 200 and 400 mg/kg b.w./d to mice with thioacetamide-induced liver fibrosis over 4 weeks. Their findings indicated an increase in glutathione (GSH) levels, associated with elevated SOD levels and a decrease in MDA levels (see Table 4)[20]. Li *et al.* also obtained analogous findings, by administering a blend of anthocyanins at a dose of 50 mg/g body weight, equivalent to 25 ml/g body weight, twice a day for 14 d to mice afflicted with kidney ischaemia-reperfusion injury. They noted increased glutathione (GSH), SOD and catalase (CAT) levels, with a reduction in MDA and TBARS (see Table 4). These observations underscore a favourable correlation between enzymatic and non-enzymatic antioxidant activity and lipid peroxidation, confirming the beneficial effects of chokeberry[20,24]. In contrast, Ćujić *et al.* conducted a study where hypertensive rats were administered 50 mg/kg b.w./d of black chokeberry extract for 28 d, which led to a decrease in both TBARS and SOD levels. Ćujić suggests that the reduced SOD activity may be attributed to the chokeberry compounds' capacity to scavenge superoxide anions, consequently lowering the substrate (superoxide anions) required for the SOD dismutation reaction (see Table 4)[34]. In contrast, Rudic *et al.* supplemented female rats with chokeberry extract at a dose of 0·45 ml/kg b.w. per d for 28 d, observing an increase in GSH and CAT levels, no change in SOD levels and a decrease in TBARS levels. The elevation in GSH may account for the inhibition of lipid peroxidation and the subsequent decrease in TBARS; however, the reason for the increased CAT without a concurrent change in SOD remains unclear (see Table 4)[23]. Some researchers propose that an increase in one antioxidant may prompt a compensatory decrease in another due to converse reactions[44].

Liu *et al.* supplemented pigs with a chokeberry-rich diet for 28 d but observed no changes in SOD, CAT and TAC biomarkers. They attribute this discrepancy to the need for polyphenols present in black chokeberry to be hydrolysed by microbes or endogenous enzymes, limiting their bioavailability to monogastric animals (Table 4)[39]. Additionally, discussing the observed positive effects of black chokeberry following long-term supplementation could provide valuable insights. Dabrowski *et al.*, for example, demonstrated the beneficial effects of long-term administration of black chokeberry extract, both alone and in Cd poisoning models, in rats. They noted increased SOD activity and TAS values, along with decreased TOS levels, after 10 months of chokeberry extract administration, affirming the antioxidant properties of the extract (Table 4)[30].

There is considerably less research on humans compared with animal models. Furthermore, comparing studies involving humans is complicated by the diversity of study groups, which include athletes, healthy individuals and patients with various conditions. For example, Duchnowicz *et al.* supplemented patients with hypercholesterolaemia with 300 ml/d of chokeberry extract for 2 months, observing a reduction in TBARS levels (see Table 5)[11]. Broncel *et al.* administered the same dose and duration of chokeberry extract to patients with metabolic syndrome. They reported reductions in TBARS and CAT levels and increases in GSH-Px and SOD indices after 1 month of supplementation. The researchers suggested that anthocyanins could serve as direct substrates for peroxidases, leading to the deactivation of hydrogen peroxide and thereby increasing GSH-Px activity. Additionally, they explained that hydrogen peroxide is deactivated in two reactions catalysed by CAT and GSH-Px; thus, high GSH-Px activity reduces the substrate concentration for CAT, inhibiting CAT activity via

Table 5. Summary of studies on the effects of chokeberry on inflammation, oxidative stress and intestinal parameters – human

Author	OCMB	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Duchnowicz *et al.* (2012)[11]	1 level	Patients with hypercholesterolaemia, no pharmacological treatment, *n* 45 (males, females), Aronia, *n* 25 (55·9 ± 7·4 years), Control, *n* 20 (50·3 ± 8·2 years)	*Aronia melanocarpa* extract	300 ml/d (100 mg three times a day)	2 months		↓ TBARS	
Broncel *et al.* (2010)[64]	1 level	Patients with metabolic syndrome, *n* 47 (32 females, 15 males, 42–65 years), Aronia, *n* 25, Control, *n* 22	*Aronia melanocarpa* extract	300 ml/d (100 mg three times a day)	2 months	↔ CRP	↓ TBARS ↑ GSH-Px ↑ SOD (after 1 month) ↔ SOD (after 2 months) ↓ CAT (after 1 month)	
Stankiewicz *et al.* (2021)[7]	1 level	Young football players, *n* 20 (males, 15·8 ± 0·7 years), Aronia, *n* 12, Control, *n* 8	Chokeberry juice	200 ml/d (100 ml twice a day)	7 weeks	↔ IL-6 ↔ myoglobin	↔ 8-OHdG ↔ TAC ↔ TBARS	
Petrovic *et al.* (2016)[65]	1 level	Handball players, *n* 32 (males 18·5 ± 1·06 years; females 17·2 ± 0·93 years), Aronia, *n* 18 (8 males, 10 females, 18·5 ± 1·06 years), Control, *n* 14 (7 males, 7 females)	Chokeberry juice	100 ml/d	4 weeks		Male: ↓ TBARS Female: ↔TBARS	
Skarpańska-Stejnborn *et al.* (2014)[6]	1 level	Elite rowers, *n* 19 (males), Aronia, *n* 10 (20·5 ± 0·97 years), Control, *n* 9 (20·8 ± 1·09 years)	Chokeberry juice	150 ml/d (50 ml three times a day)	8 weeks	↔ IL-6 ↓ TNF-α	↑ TAC	
Xie *et al.* (2016)[66]	1 level	Former smokers, *n* 49 (males, females), Aronia, *n* 25 (32·6 ± 2·6 years), Control, *n* 24 (37·4 ± 3·0 years)	Chokeberry extract	500 mg/d (250 mg twice a day)	12 weeks	↔ IL-6 ↔ IL-1β ↔ TNF-α ↔ CRP ↔ MCP-1	↔ CAT ↔ GSH-Px, ↔ SOD	
Pilaczynska-Szcześniak *et al.* (2005)[67]	1 level	Elite rowers, *n* 19 (males), Aronia, *n* 9 (21 ± 0·8 years), Control, *n* 10 (22 ± 1·7 years).	Chokeberry juice	150 ml/d (50 ml three times a day)	4 weeks		↓ TBARS, ↓ GSH-Px, ↓ SOD	
Istas *et al.* (2019)[68]	1 level	Healthy males, *n* 66, Aronia extract, *n* 23, (24 ± 6·3 years), Aronia whole fruit powder, *n* 23, (24 ± 5·2 years), Control, *n* 20	Aronia extract, Aronia whole fruit powder	500 mg/d	12 weeks			Aronia extract/whole fruit: ↔ α diversity ↔ β diversity Aronia extract: ↑ *Anaerostipes* Aronia whole fruit: ↑ *Bacteroides*

(*Continued*)

Table 5. (*Continued*)

Author	OCMB	Group	Supplement	Dose	Duration of supplementation	Markers of inflammation	Markers of oxidative stress	Changes in gut microbiota
Le Sayec *et al.* (2022)[12]	1 level	Prehypertensive participants, *n* 102 (47 males, 55 females), Aronia, *n* 51 (56·2 ± 8·7 years), Control, *n* 51 (56·2 ± 9 years)	Aronia extract	500 mg/d	12 weeks			↔ Shannon-Wiener index, ↔ Simpson index, ↔ Bray-Curtis, ↑ *Intestinimonas butyriciproducens*, ↑ *Lawsonibacter asaccharolyticus*, ↑ *Butyricimonas faecihominis*, ↑ *Bacteroides xylanisolvens*, ↓ *Senegalimassilia anaerobia*, ↓ *Haemophilus parainfluenzae*
Chung *et al.* (2023)[69]	1 level	Healthy adults, *n* 70, (35 males, 35 females), Aronia, *n* 35 (45·0 ± 1·3 years) Control, *n* 35 (46·5 ± 1·3 years)	Aronia berry extract	300 mg/d (150 mg twice a day)	8 weeks	↑ IL-6 Immediately post-exercise	↓ GSH 30 min post-exercise, ↓ GSH-Px immediately and 30 min post-exercise, ↓ GSSG 30 min post-exercise, ↔ CAT 30 min post-exercise, ↔ SOD 30 min post-exercise, ↔ MDA 30 min post-exercise	
Lackner *et al.* (2024)[70]	1 level	Normal weight females, *n* 40 (18–40 years) Aronia, *n* 20 Placebo, *n* 20	Aronia juice	200 ml/d (100 ml twice a day)	6 weeks			↔ Shannon index
Stankiewicz *et al.* (2023)[13]	1 level	Semi-professional male football players, *n* 22 Aronia, *n* 10 (19·86 ± 0·61 years) Placebo, *n* 12 (20·05 0·52 years)	Chokeberry extract	6 g of lyophilised extract (in capsules)/d	90 d	↓ IL-6 ↑ IL-10	↑ TAC, ↓ 8-OHdG	

Abbreviations: ↔ *No change*, ↓ decrease, ↑ increase; 8-OHdG, 8-hydroxy-2′-deoxyguanosine; CAT, catalase; CRP, C-reactive protein; GSH-Px, glutathione peroxidase; GR, glutathione reductase; MCP-1, monocyte chemoattractant protein-1; MDA, malondialdehyde; SOD, superoxide dismutase; TAC, total antioxidant capacity; TAS, total antioxidative status; TBARS, thiobarbituric acid reactive substances.

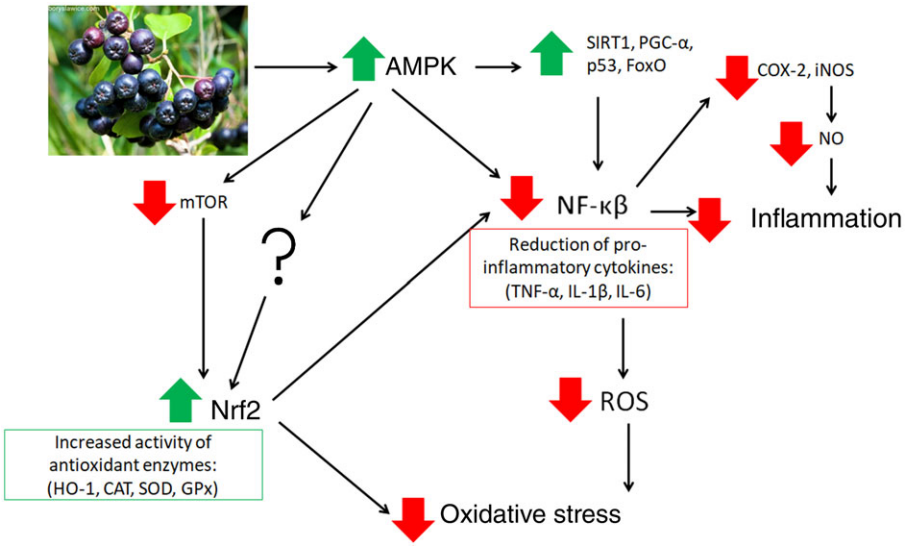

Figure 2. Effects of AMPK pathway activation on NF-κB and Nrf2 pathways. The colour of the black arrow indicates influence. The colour of the red arrow indicates inhibition, and the colour of the green arrow indicates activation. AMPK, AMP-activated protein kinase; Nrf2, nuclear-related factor 2.

negative feedback (see Table 5)[64]. In contrast, Chung *et al.* used 300 mg/d of chokeberry extract for 8 weeks in healthy subjects. The researchers showed a statistically significant increase in GSH and GSH-Px concentrations and no significant changes in CAT, SOD and MDA concentrations. The explanation provided by Chung *et al.* indicates that the glutathione defense system was most sensitive in response to exercise used as a 'factor' to disrupt the prooxidant–antioxidant balance. However, unlike the glutathione defense system, SOD and CAT remained stable in the proposed model. This may indicate that the antioxidant enzyme response is dependent on the 'severity' of oxidative stress (Table 5)[69]. Thus, discrepancies in antioxidant enzyme activity may be, at least partially, attributed to differences in the pathological conditions responsible for oxidation–antioxidation imbalance.

Analysing studies on athletes, in whom oxidative stress arose as a response to an intense physical exertion, we also observed the effectiveness of chokeberry supplements. Researchers often confirm this by demonstrating a reduction in TBARS concentrations[65,67] (see Table 5). However, given the recent systematic review that focused on the effects of chokeberry in a model of exercise[3], we omit further analysis.

In summary, studies conducted on both animal models and human subjects collectively affirm the antioxidant properties of *Aronia melanocarpa*. This is primarily evidenced by alterations in the concentration of enzymes crucial for maintaining the balance between oxidation and antioxidation. However, the biomarkers analysed do not always exhibit expected behaviour. It can be inferred that the impact on antioxidant enzyme levels is contingent upon two factors: first, the severity of oxidative stress, which is influenced by the characteristics of the study population, and second, the content of biologically active compounds that directly mitigate oxidative stress, which is related to the dose, type of supplement administered (e.g. juice, extract, fruit) and the duration of supplementation.

Effects of black chokeberry on markers of inflammation

After analysing the findings from the systematic review, it is evident that the bioactive compounds in black chokeberry exert significant anti-inflammatory effects by modulating cytokine profiles. This modulation involves a complex interplay between various pro-inflammatory and anti-inflammatory mediators within the immune system. In animal models (see Table 4), it has been shown, that these compounds can reduce the levels of pro-inflammatory cytokines such as IL-1β[24,33,36,41] and TNF-α[24,28,33,36,38,41,71], which act as primary inflammatory stimuli during the cell signalling process.

To elucidate the impact of chokeberry on inflammation, oxidative stress and their interactions, it is critical to examine the role of AMP-activated protein kinase (AMPK), which modulates the NF-κB and Nrf2 pathways[43,72]. Nrf2 is a pivotal regulator of various antioxidants and oversees the activity of antioxidant enzymes, including haem oxygenase-1, catalase (CAT), SOD and GSH-Px[43]. According to Zhao *et al.*, black chokeberry influences AMPK activation, leading to the inhibition of the mammalian target of rapamycin activity. This subsequently activates the P-phosphatidylinositol-3-hydroxykinase/Akt/mammalian target of rapamycin pathway through the regulation of Nrf2 nuclear translocation, resulting in enhanced production of antioxidant enzymes[46]. However, the direct phosphorylation effect of AMPK on Nrf2 remains to be clarified[73].

NF-κB is a transcription factor that triggers the production of pro-inflammatory factors[24]. AMPK activation stimulates factors such as sirtuin 1, PPAR gamma coactivator 1α, tumour protein p53 and FoxO transcription factor, which can inhibit NF-κB signalling, thereby preventing the synthesis of pro-inflammatory factors[72]. Additionally, Wang *et al.* demonstrated that AMPK inhibition of NF-κB signalling reduces the expression of various NAD(P)H oxidase subunits, decreasing the production of ROS and thereby alleviating oxidative stress[74]. Furthermore, Li *et al.* indicated that Nrf2 can inhibit NF-κB signalling, suggesting that Nrf2 activators have anti-inflammatory properties[75] (Fig. 2).

These interactions have been corroborated by other researchers. For instance, Li *et al.* conducted an experiment in which male mice were administered an extract containing cyanidin-3-arabinoside, cyanidin-3-glucoside and cyanidin-3-galactoside at a dosage of 50 mg/g body weight. These anthocyanins are potent antioxidants capable of neutralising ROS, thereby mitigating oxidative stress, which is frequently associated with chronic inflammation. The authors demonstrated a significant reduction in the levels of IL-1β, TNF-α and IL-6, indicating a suppression of the inflammatory response. The study further elucidated that the production of

pro-inflammatory cytokines, particularly TNF-α and IL-1β, is mediated by the transcription factor NF-κB, which is activated through toll-like receptor signalling. Activation of toll-like receptor 4 is positively correlated with inducible nitric oxide synthase, thereby linking inflammation and oxidative stress (Table 4)[24]. This is also confirmed by Wang *et al.* who indicated that it is ROS that activates T lymphocytes, which release inflammatory factors (Table 4)[71]. Furthermore, Ohgami *et al.* also indicate that it is the antioxidant effect of chokeberries that is responsible for inflammatory responses. The authors confirm this with a reduction in nitric oxide production, which led to the inhibition of nitric oxide synthase and, consequently, a reduction in the inflammatory cytokine TNF-α. In addition, the researchers emphasise that, first, the anti-inflammatory effect of chokeberry is dose-dependent. Second, the authors point out that the anti-inflammatory mechanism of anthocyanin compounds consists of several factors. Ohgami *et al.* explain that one of the anti-inflammatory mechanisms of chokeberry is the blocking of cyclooxygenase-2 protein expression. Cyclooxygenase-2 is primarily responsible for increased PGE2 production during inflammation, and PGE2 is generally considered a pro-inflammatory agent (Table 4)[44] (Fig. 2).

Only five studies analysing biomarkers of inflammation in humans were included in the systematic review. Skarpańska-Stejnborn *et al.* reported a statistically significant reduction in TNF-α levels following the administration of 150 ml of chokeberry juice daily for 8 weeks to elite rowers. The authors suggest that anthocyanins in chokeberry juice may attenuate the activity of major inflammatory enzymes and prevent the adhesion of leukocytes to vascular endothelial cells by inactivating TNF-α (Table 5)[6].

In contrast, Xie *et al.* observed no significant changes in inflammatory biomarkers when supplementing 500 mg/d of chokeberry extract for 12 weeks among former smokers. The researchers propose that the anti-inflammatory effects of polyphenols from chokeberry may be more pronounced in populations suffering from chronic inflammation, rather than in relatively healthy individuals (Table 4). This suggests that the efficacy of chokeberry-derived polyphenols might be contingent upon the baseline inflammatory status of the subjects (Table 5)[66].

We hypothesise that the administration of black chokeberry exerts a notable impact on inflammation reduction, intricately linked to the mitigation of ROS and subsequent oxidative stress. However, this reduction appears to be observed in increased and chronic inflammation. A clear discrepancy emerges between studies focusing on inflammation reduction and those targeting oxidative stress, with approximately 70 % fewer studies analysing inflammation biomarkers. This underscores the imperative for future experiments to delve deeper into elucidating the trajectory of alterations and the intricacies involved in mitigating the inflammatory process after chokeberry use. Such endeavours are crucial for comprehensively understanding the mechanisms underlying the anti-inflammatory properties of chokeberry and optimising its therapeutic potential in combating inflammatory conditions.

Effect of black chokeberry on intestinal parameters

Diversity and richness are pivotal parameters characterising the human gut microbiota, with implications extending to various health outcomes[26]. Diminished gene diversity and richness within the intestinal microbiome are frequently observed in individuals afflicted with diverse disorders, such as obesity[76], type 2 diabetes mellitus[76], psychiatric disorders[77], Crohn's disease[78] or even in infants delivered by caesarean section[79]. Clinical interventions (e.g. antibiotics, drug use) and environmental factors (e.g. smoking, diet and physical activity) also affect microbial diversity[80].

Supplementation with *Aronia melanocarpa* has been explored for its potential impact on gut health; however, no effect on α and β diversity has been observed in human studies[12,68,70]. In animal research, results were inconclusive. In three studies, parameters remained at the same level[22,41,47], and in three, they increased[20,21,62]. However, the *Firmicutes:Bacteroides* ratio tended to lower in animal models[22,47]. It is a widely accepted marker that has an essential influence on maintaining normal intestinal homeostasis. An increased or decreased *Firmicutes:Bacteroides* ratio is regarded as dysbiosis, whereby the former is usually observed concerning obesity and the latter to inflammatory bowel disease[81]. Both studies were carried out on high-fat diet models suggesting a potential trend in reducing the *Firmicutes:Bacteroidetes* ratio in obesity following *Aronia melanocarpa* supplementation[22,47]. Moreover, increases in *Bacteroides* and *Bacteroidetes* have been consistently noted across studies[12,20,22,41,47,68]. Notably, *Bacteroides* play a crucial role in supplying nutrients to other microbial residents of the gut, thereby contributing to the overall balance and functioning of the gut microbiota. Furthermore, they serve as a protective barrier against pathogens, helping to maintain intestinal homeostasis and prevent infections. This highlights the potential importance of increasing *Bacteroides* abundance through aronia supplementation to promote gut health and mitigate the risk of various gastrointestinal disorders[82].

We also noticed a rise in *Verrucomicrobia* and *Akkermansia muciniphila*[22,47], which are connected to gut health[83] and are even proposed as a next-generation probiotic[84]. Furthermore, levels of *Prevotella* also increased[22,41,47]. The Western lifestyle is causal in the loss of *Prevotella* diversity, and aronia could reverse this pathway. An increase in *Romboutsia* was also observed[22,41,47], the bacterium is negatively correlated with body weight, insulin and fasting glucose. In the end, the abundance of pathogens in the gut decreased after aronia supplementation: *Proteobacteria*, the microbial signature of dysbiosis[85], was decreased[24]; *Escherichia-Shigella* associated not only with dysbiosis but also with inflammation was also decreased[41], *Haemophilus parainfluenzae*, which may cause infections of soft tissue, central nervous system and endocarditis[86], followed the same pattern[12]. To summarise, supplementation with *Aronia melanocarpa* may positively affect gut health, especially by reducing potential pathogens and increasing the abundance of bacteria positively correlated to gut health, such as *Akkermansia* and *Bacteroidetes*. In our review, only one study on gut permeability was conducted on growing pigs. Aronia significantly increased the jejunal gene expression of tight junction proteins, such as occludin, claudin and zonulin[41], thus improving gut barrier tightness. Another study carried out on a rat model of obesity confirmed aronia's positive impact on gut tightness, but it was not approved for the review process due to the lack of full text[87]. Preliminary animal studies may suggest the possibility of chokeberry influencing the tightness of the intestinal barrier.

The compounds found in black chokeberry play a pivotal role in maintaining the overall balance within the system. This is a crucial consideration given the tendency for disturbances in organismal homeostasis to accompany disease processes and various disorders. Chokeberry not only reduces oxidative stress and

modulates the inflammatory response but also influences the composition of the intestinal microbiome through the growth of bacteria, which are positively correlated with health. This underscores the potential of natural supplements, like chokeberry in restoring systemic equilibrium, as evidenced by numerous studies highlighting their efficacy across a spectrum of physiological processes. However, it is imperative to acknowledge the significant divergence between findings in human and animal studies. While existing research underscores the potential benefits of chokeberry supplementation, particularly in prophylactic contexts aimed at preserving organism harmony and homeostasis, further investigation is warranted to fully elucidate its mechanisms and optimise its application. Moreover, the substantial gap between studies focusing on oxidative stress, inflammation and intestinal parameters underscores the necessity for additional research to comprehensively understand the effects of chokeberry on inflammation and microbiota alterations. Additionally, the observation of fewer studies involving women compared with men highlights an area requiring considerable attention and clarification. Addressing these research gaps would be pivotal in further understanding the potential benefits and optimal applications of black chokeberry supplementation for promoting overall health and mitigating disease risk across diverse populations.

Limitations

The primary limitation of this review is the considerable heterogeneity in the methodologies of the included studies. Variations in supplementation duration, type of supplement (e.g. extract, juice, fruit), dosage and sample sizes among the studies introduce substantial challenges in result comparison. Additionally, the diversity of study populations and the significant discrepancies between human and animal model studies further complicate the synthesis of findings. A notable concern is the predominance of studies from Eastern countries, which raises questions about the generalizability of the results to Western populations. These factors necessitate a cautious interpretation of the review's conclusions. Furthermore, despite employing the PRISMA protocol for article selection, there remains a possibility that some relevant manuscripts were inadvertently omitted. Another critical limitation is the presence of significant sources of bias in many of the studies analysed. This is particularly noticeably true for studies on animal models. This may indicate that scientists are still not very familiar with this tool.

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Data are available from the corresponding author upon reasonable request.

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