

Systematic Review

Green Tea (*Camellia sinensis*) Consumption and Human Immune Function: A Systematic Review of Immunomodulatory and Anti-Inflammatory Effects

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Abstract

Background: Green tea (*Camellia sinensis*) is widely consumed around the world, particularly in East Asia, and has traditionally been used to support cardiovascular and metabolic health. Rich in catechins—most notably epigallocatechin-3-gallate (EGCG)—green tea is recognized for its potential immunomodulatory and anti-inflammatory effects. However, evidence from human studies remains inconsistent, and the distinction between functional immune outcomes and systemic inflammatory markers has not been systematically evaluated. **Methods:** We conducted a systematic review in accordance with PRISMA guidelines, searching PubMed, Google Scholar, Cochrane Library, and EBSCO from inception to the most recent date. Eligible studies included human investigations evaluating green tea or catechin intake and reporting either immune response outcomes (e.g., natural killer cell activity, T cell responses) or systemic inflammatory markers (e.g., C-reactive protein, cytokines). Randomized controlled trials (RCTs), non-randomized interventional studies, and observational analyses were included. Data were synthesized narratively due to heterogeneity. **Results:** Nineteen studies (≈ 6800 participants) were included, comprising 12 RCTs, three non-randomized interventions, and five observational studies. Eight studies assessed immune responses and consistently demonstrated enhanced immune activity, including increased natural killer cell function and augmented $\gamma\delta$ T cell responses. Eleven studies evaluated inflammatory markers and showed heterogeneous results: several trials reported reductions in C-reactive protein and related biomarkers, particularly in high-risk populations, whereas others found no significant changes in cytokines such as interleukin-6 or tumor necrosis factor- α . Observational studies generally reported inverse associations between tea consumption and inflammation. Green tea was well tolerated across studies. **Conclusions:** Green tea consumption appears to enhance functional immune responses while exerting modest, context-dependent anti-inflammatory effects. These findings support a dual immunomodulatory role and highlight the importance of distinguishing between immune function and systemic inflammation in nutritional research.

Keywords: green tea; functional foods; immune modulation; epigallocatechin gallate; evidence-based medicine



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1. Introduction

Tea derived from *Camellia sinensis*, particularly green tea, is among the most widely consumed beverages worldwide and is rich in bioactive polyphenols, notably catechins such as epigallocatechin-3-gallate (EGCG) [1–6]. These compounds have been extensively studied for their antioxidant, anti-inflammatory, and immunomodulatory properties, with potential implications for the prevention of chronic diseases and infections [7–15]. Green tea is traditionally recognized for a range of medicinal indications, including cardiovascular protection, metabolic regulation, anticancer activity, and antimicrobial effects, and it is among the most widely consumed beverages for health-promoting purposes worldwide, with China, Japan, and India representing the largest consumer populations [3,16,17]. Beyond its antioxidant, anti-inflammatory, and immunomodulatory properties, green tea and its catechins have also demonstrated antimicrobial, anticancer, neuroprotective, and cardioprotective pharmacological activities, further supporting its broad biological relevance [10,18].

The immune system plays a central role in maintaining homeostasis and protecting against pathogens, while dysregulated inflammation contributes to the pathogenesis of a wide range of conditions, including cardiovascular disease, metabolic syndrome, and cancer [19–24]. Nutritional interventions that can modulate immune function and inflammatory pathways are, therefore, of considerable clinical and public health interest [25–28]. Experimental studies have suggested that green tea catechins can enhance innate and adaptive immune responses—such as natural killer (NK) cell activity and T cell function—while also attenuating pro-inflammatory signaling pathways, including nuclear factor- κ B-mediated cytokine production [29].

Despite these mechanistic insights, evidence from human studies remains inconsistent. Some clinical trials have reported enhanced immune responses and reduced infection risk associated with green tea consumption, whereas others have shown limited or no effects on circulating inflammatory markers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor- α (TNF- α) [30–32]. Furthermore, findings appear to vary according to population characteristics, including baseline inflammatory status, metabolic health, and age [30,33]. Observational studies have generally suggested inverse associations between habitual tea consumption and inflammatory biomarkers, but these designs are inherently susceptible to confounding.

To date, no synthesis has systematically distinguished between functional immune outcomes and systemic inflammatory biomarkers in evaluating the effects of green tea in humans. This distinction is critical, as immune enhancement and anti-inflammatory effects may represent complementary but biologically distinct processes.

Therefore, we conducted a systematic review of human studies to evaluate the effects of green tea consumption and its bioactive components on (1) immune responses and (2) systemic inflammatory markers. By integrating evidence across randomized trials, interventional studies, and observational analyses, we aimed to clarify the extent to which green tea acts as an immunomodulatory agent in humans. Accordingly, the objective of this review was to evaluate the effects of green tea consumption on human immune responses and systemic inflammatory markers.

2. Materials and Methods

2.1. Study Design and Reporting Standards

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) statement (Supplementary Table S1) and was registered in the International Prospective Register of Systematic Reviews (PROSPERO registration number CRD420261450564).

2.2. Search Strategy and Data Sources

A comprehensive literature search was performed in PubMed, Google Scholar, Cochrane Library, and EBSCO from database inception to the most recent search date. The database searches were completed in 21 May 2026.

Search terms included combinations of keywords and Medical Subject Headings (MeSH) related to: “green tea”, “*Camellia sinensis*”, “tea catechins”, “epigallocatechin gallate (EGCG).”

“immune response”, “natural killer cells”, “T cells”, “immunoglobulins”, “inflammation”, “C-reactive protein”, “cytokines”, “oxidative stress”, “clinical trial”. Boolean operators (“AND”, “OR”) were used to combine search terms. Reference lists of included articles were manually screened to identify additional relevant studies.

2.3. Eligibility Criteria

Studies were included if they met the following PICOS (population, intervention, comparisons, outcomes, and study design) criteria: eligible participants were human subjects, including either healthy individuals or clinical populations; the intervention or exposure of interest was green tea consumption or green tea-derived compounds (e.g., catechins, EGCG), administered as beverages or supplements, compared with placebo or no exposure. Eligible outcomes comprised functional immune outcomes (e.g., NK cell activity, T cell responses, infection outcomes) or systemic inflammatory markers (e.g., CRP, IL-6, TNF- α , oxidative stress markers), assessed in randomized controlled trials, non-randomized interventional studies, or observational studies published in English. Studies were excluded if they used in vitro or ex vivo models only, were animal studies, or were reviews, editorials, or conference abstracts without primary data. A detailed description of study search and eligibility is provided in Supplementary Table S2.

2.4. Study Selection

Two reviewers independently screened titles and abstracts for eligibility. Full-text articles were retrieved for potentially relevant studies and assessed against the inclusion criteria. Discrepancies were resolved by consensus. Our systematic search yielded a total of 1277 records, and 1078 records were rapidly excluded because they were duplicates or irrelevant. The abstracts of 199 records were then screened, and 151 of them were excluded as they were Case reports, Review articles, or Preclinical studies. The full text of 48 articles was rigorously evaluated according to the study’s inclusion and exclusion criteria, and ultimately, 19 studies were included in this systematic review. The outline of the literature search, screening, and study selection is detailed in the PRISMA flow diagram (Figure 1).

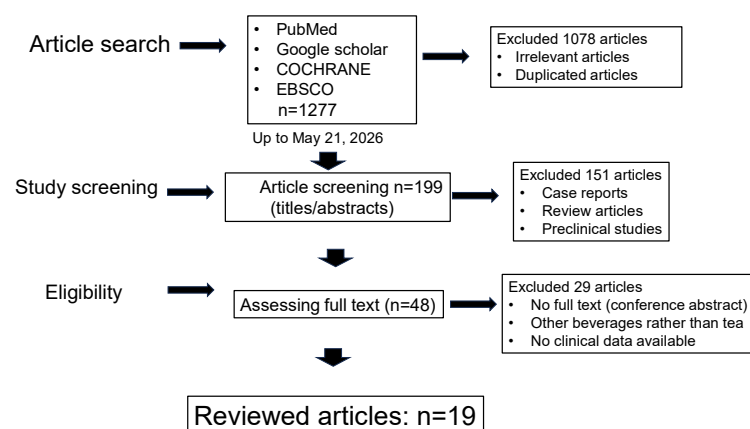


Figure 1. The PRISMA diagram with the article search strategy and study selection process.

2.5. Data Extraction

Data were extracted independently using a standardized form. Extracted variables included: study identification (author, year, country), study design and population characteristics (sample size, age, health status), intervention details (form, dose, frequency, duration), comparator (placebo, control beverage, baseline), outcomes measured (immune or inflammatory), main findings (effect size, direction, statistical significance), adjustment for confounders (for observational studies), adverse events. Studies were categorized into two predefined domains: Immune response studies (functional immune outcomes) and Systemic inflammatory marker studies.

2.6. Risk of Bias and Quality Assessment

Risk of bias was assessed at the study level using tools appropriate to each study design. Randomized controlled trials (RCTs) were evaluated with the Cochrane Risk of Bias 2 (RoB 2) tool. Non-randomized intervention studies were assessed using the ROBINS-I tool. Observational (primarily cross-sectional) studies were evaluated with the Newcastle-Ottawa Scale, adapted for cross-sectional designs and informed by JBI critical appraisal principles. For RCTs, the five RoB 2 domains were assessed: (1) randomization process, (2) deviations from intended interventions, (3) missing outcome data, (4) measurement of the outcome, and (5) selection of reported results. For non-randomized intervention studies, the seven ROBINS-I domains were evaluated: (1) confounding, (2) selection of participants, (3) classification of interventions, (4) deviations from intended interventions, (5) missing data, (6) measurement of the outcome, and (7) selection of reported results. For observational studies, the Newcastle-Ottawa Scale assessed three main domains—selection (representativeness of the sample, sample size, non-respondents, and exposure ascertainment), comparability (adjustment for age/sex and other confounders), and outcome (adequacy of outcome assessment and statistical analysis). Signaling questions, domain-level judgments, and supporting justifications for all assessments are provided in the Supplementary Materials.

Although multiple studies examined the same nominal biomarkers (e.g., CRP, IL-6, and NK cell activity), quantitative pooling was deemed inappropriate due to substantial clinical and methodological heterogeneity. The studies varied simultaneously across several key factors known to influence effect size: intervention form (beverage versus extract or capsule), catechin dose (roughly 300 mg to >900 mg per day), comparator type (placebo, active control, or no-treatment), intervention duration (2 weeks to 6 months), and—most critically—the baseline inflammatory or metabolic status of the participants (healthy versus clinical populations). Given that green tea's anti-inflammatory effects are context-dependent and appear strongest in individuals with elevated baseline inflammation, combining data from healthy and clinical populations would obscure this important effect modification. A single pooled estimate would therefore misrepresent the true effects in both subgroups rather than illuminate them. Compounding this issue, outcome reporting was inconsistent: studies presented results as absolute mean differences, percentage changes, fold-changes, or odds ratios, and many failed to report the variance estimates necessary for valid standardization or meta-analytic pooling. For these reasons, a narrative synthesis stratified by outcome domain and population type (healthy versus clinical) was judged more appropriate and informative than a quantitative meta-analysis.

2.7. Data Synthesis

A narrative synthesis was conducted, structured according to outcome domain (immune response vs inflammatory markers). Findings were summarized qualitatively, with

emphasis on consistency of effects, magnitude, and direction of associations across study designs and populations.

3. Results

3.1. Study Selection and Characteristics

A total of nineteen studies met inclusion criteria, comprising approximately 6800 participants. Sample sizes ranged from small pilot trials ($n \approx 12$ –20) to large observational cohorts ($n > 4000$), with mean participant ages spanning 30–70 years. Among the 19 studies, 12 were RCTs: Steptoe 2007 [34]; Rowe 2007 [35]; Basu 2011 [36]; Ayuso 2014 [37]; Bazyar 2021 [38]; Hadi 2020 [39]; Bahorun 2010 [40]; Gostner 2015 [41]; Dower 2015 [42]; Wang 2011 [43]; Tanaka 2021 [44]; Zeng 2024 [45], four were non-randomized trials: Kamath 2003 [46]; Lowe 2015 [47]; Iketani 2019 [48]; D'Arena [49], whereas three were observational analyses: Hsieh 2021 [50]; De Bacquer 2006 [51]; Rebello 2011 [52]. Eleven studies (Steptoe 2007 [34]; Rowe 2007 [35]; Lowe 2015 [47]; Iketani 2019 [48]; Tanaka 2021 [44]; Gostner 2015 [41]; Dower 2015 [42]; Rebello 2011 [52]; Hsieh 2021 [50]; Zeng 2024 (healthy subgroup) [45]; Kamath 2003 [46]) enrolled healthy individuals, while eight studies Basu 2011 (metabolic syndrome) [36]; Ayuso 2014 (obesity) [37]; Bazyar 2021 and Hadi 2020 (type 2 diabetes mellitus) [38,39]; Bahorun 2010 (cardiovascular risk) [40]; D'Arena 2013 (chronic lymphocytic leukemia) [49]; Wang 2011 (acute inflammatory condition—pharyngitis) [43]; Zeng 2024 (metabolic syndrome subgroup) [45]; and De Bacquer 2006 (cardiovascular risk population) [51]) included clinical populations. Interventions consisted of green or black tea beverages (typically 3–6 cups/day) or catechin-rich extracts (~300–900 mg/day), administered over 2 weeks to 6 months.

Among the included studies, five used black tea interventions (Steptoe 2007 [34]; Rowe 2007 [35]; Bahorun 2010 [40]; Gostner 2015 [41]; Tanaka 2021 [44]), while the remaining 14 used green tea or green tea-derived extracts. Black tea, while fermented, shares key catechin-derived polyphenols with green tea and was included because the immunomodulatory outcomes of interest were directly comparable, and excluding these studies would omit important evidence on tea polyphenols more broadly.

The 19 included studies exhibited considerable diversity in design, scale, and population characteristics. Study designs encompassed 12 randomized controlled trials (RCTs), three non-randomized interventional studies, and four observational cross-sectional analyses. Sample sizes ranged from small pilot investigations ($n \approx 12$ –20 participants) to large population-based cohorts ($n > 4000$), with a total of approximately 6800 participants across all studies. The mean age of participants spanned from 30 to 70 years, reflecting inclusion of working-age adults, elderly populations, and middle-aged clinical cohorts.

Populations studied were heterogeneous across two broad categories: Eleven studies enrolled healthy individuals without significant comorbidities, including community-dwelling adults, elderly volunteers, and physically active participants. The remaining eight studies focused on clinical populations with elevated inflammatory or metabolic burden, including individuals with type 2 diabetes mellitus, metabolic syndrome, obesity, cardiovascular risk, chronic lymphocytic leukemia, and acute pharyngitis. This diversity enabled assessment of green tea effects across the spectrum of baseline immune and inflammatory status.

Interventions varied in formulation, dose, and duration: Tea was administered either as a beverage (typically 3–6 cups per day) or as a catechin-rich extract or supplement (approximately 300–900 mg/day of catechins or purified EGCG). Intervention periods ranged from 2 weeks to 6 months, with most studies employing a duration of 8–12 weeks. Comparator conditions included placebo capsules, control beverages (caffeine-matched or flavonoid-free), or baseline measurements in single-arm designs.

Outcomes were categorized into two predefined domains: Eight studies assessed functional immune outcomes, including natural killer (NK) cell activity, $\gamma\delta$ T cell responses, cytokine production (e.g., interferon- γ), mucosal immunity (secretory immunoglobulin A), and infection-related clinical endpoints. Eleven studies evaluated systemic inflammatory markers, including C-reactive protein (CRP), high-sensitivity CRP, interleukin-6 (IL-6), interleukin-1 β (IL-1 β), tumor necrosis factor- α (TNF- α), serum amyloid A (SAA), and endotoxin levels. This dual-outcome framework allowed systematic distinction between immune enhancement and anti-inflammatory effects, a differentiation that has not been consistently applied in prior syntheses.

For data synthesis, studies were grouped by outcome into immune response studies and systemic inflammatory marker studies.

3.2. Effects on Immune Response

Eight studies assessed functional immune outcomes. Across these, green tea consumption was consistently associated with enhanced immune activity. In elderly participants, catechin-rich green tea increased natural killer (NK) cell activity by a mean of 17.1 percentage points (95% CI 9.7–24.5), with improvements observed in 85% of individuals [48]. Similarly, in an RCT, black tea consumption increased NK cell activity ($p = 0.031$) and salivary IgA, which was associated with a reduced incidence of upper respiratory tract infections (odds ratio 0.17, 95% CI 0.04–0.68) [44].

Adaptive immune responses were also enhanced. Tea consumption increased interferon- γ (IFN- γ) production by $\gamma\delta$ T cells by 2–3-fold compared with controls [46], while a RCT reported increases in $\gamma\delta$ T cell proliferation (+28%, $p = 0.017$) and IFN- γ secretion (+26%, $p = 0.046$), accompanied by a 32.1% reduction in cold and influenza symptoms [35].

Additional studies reported increased leukocyte functional activity, including enhanced myeloperoxidase and lactoferrin release following green tea extract supplementation. Clinical benefits were observed in disease-specific settings, including improved symptom resolution in pharyngitis and modulation of regulatory T cell populations in chronic lymphocytic leukemia. Overall, findings were consistent in showing enhanced innate and adaptive immune responses, with associated reductions in infection-related outcomes (Table 1).

Table 1. Immune Response Studies (Functional Immunity).

Study Design, Population	Green Tea Exposure and Comparator	Immune Outcomes Measured	Main Results, Adjustment/Confounders, Adverse Events	References
Controlled intervention; $n = 21$ healthy adults	Black tea (5–6 cups/day) vs. coffee; 2–4 weeks	$\gamma\delta$ T-cell; IFN- γ	2–3 $\times$ $\uparrow$ IFN- γ ; significant responder increase ($p < 0.01$); No adverse events reported	Kamath et al., 2003 [46]
RCT; healthy adults (18–70)	Green tea capsules vs. placebo; 3 months	$\gamma\delta$ T-cell; IFN- γ ; infection outcomes	$\downarrow$ cold/flu incidence (−32.1%); $\uparrow$ $\gamma\delta$ T-cell proliferation (+28%); $\uparrow$ IFN- γ ($p < 0.05$) No adverse events reported	Rowe et al., 2007 [35]
Open-label; $n \approx 20$ healthy	GTE 300 mg/day; 14 days	Leukocyte function (MPO, lactoferrin)	$\uparrow$ leukocyte activity ($p < 0.05$); $\uparrow$ antioxidant status No adverse events reported	Lowe et al., 2015 [47]

Table 1. Cont.

Study Design, Population	Green Tea Exposure and Comparator	Immune Outcomes Measured	Main Results, Adjustment/Confounders, Adverse Events	References
Single-arm pilot; $n = 20$ elderly	Catechin-rich beverage (~540 mg/day); 2 weeks	NK cell activity; cytokines	↑ NK activity (+17.1%, $p < 0.001$); no CRP/IL-6 change No adverse events reported	Iketani et al., 2019 [48]
RCT; $n \approx 70$ adults	Black tea 3 cups/day vs. placebo; 12 weeks	NK activity; SIgA; infection outcomes	↓ respiratory infections (OR 0.17); ↑ NK activity ($p = 0.031$); ↑ SIgA No adverse events reported	Tanaka et al., 2021 [44]
RCT; $n = 60$ pharyngitis patients	Green tea polyphenols vs. control	Clinical immune outcome (infection symptoms)	Significant symptom improvement; 96.7% efficacy; No adverse events reported	Wang et al., 2011 [43]
Open-label; $n = 12$ CLL patients	Green tea extract; 6 months	Tregs; IL-10; TGF- β	↓ Tregs; ↓ IL-10/TGF- β ; clinical modulation of immune state No adverse events reported	D’Arena et al., 2013 [49]
RCT; $n \approx 94$ healthy	Black tea 3 cups/day; 6 months	KYN/TRP; neopterin	↑ kynurenine ($p = 0.016$); immune activation marker No adverse events reported	Gostner et al., 2015 [41]

Footnote: Studies are categorized by primary outcome domain as defined in the manuscript. Some studies reported outcomes in both domains but are listed according to their primary focus. The table summarizes the seven studies assessing functional immune outcomes (e.g., NK cell activity, $\gamma\delta$ T cell responses, mucosal immunity), including study design, population, green tea/black tea exposure and comparator, immune outcomes measured, and main results with adjustment for confounders and adverse events, ordered by study identifier as listed in the References column. Abbreviations: CLL, chronic lymphocytic leukemia; CRP, C-reactive protein; GTE, green tea extract; IFN- γ , interferon- γ ; IL-6, interleukin-6; IL-10, interleukin-10; KYN/TRP, kynurenine-to-tryptophan ratio; MPO, myeloperoxidase; NK, natural killer; OR, odds ratio; RCT, randomized controlled trial; SIgA, secretory immunoglobulin A; TGF- β , transforming growth factor- β ; Tregs, regulatory T cells. Note: (↑) indicates increase; (↓) indicates decrease.

3.3. Effects on Systemic Inflammatory Markers

Twelve studies evaluated systemic inflammatory markers, with heterogeneous effects. Several randomized trials demonstrated reductions in inflammatory biomarkers. Black tea consumption significantly reduced C-reactive protein (CRP), with reductions exceeding 50% in high-risk individuals. In patients with type 2 diabetes, EGCG supplementation reduced high-sensitivity CRP ($p = 0.003$), and other trials reported improvements in serum amyloid A and total antioxidant capacity. However, multiple trials reported no significant changes in CRP, IL-6, or TNF- α , particularly in metabolically stable or obese populations [36–38,45]. In a recent crossover RCT, green tea extract reduced circulating endotoxin ($p = 0.023$) and improved gut permeability, but did not alter systemic inflammatory cytokines [45]. Observational studies showed consistent inverse associations between tea consumption and inflammation [50–52]. Higher intake was associated with lower CRP concentrations (e.g., -12.2% for ≥ 1 cup/day vs. < 1 cup/week, $p = 0.042$) after multivariable adjustment, with similar findings across large cohorts [52].

Interestingly, one RCT reported increased kynurenine-to-tryptophan ratios following tea consumption [41], suggesting activation of immune-related metabolic pathways rather than suppression. Overall, green tea showed modest and context-dependent anti-

inflammatory effects, with greater benefit observed in populations with elevated baseline inflammation (Table 2).

Table 2. Systemic Inflammatory Marker Studies.

Study Design, Population	Green Tea Exposure and Comparator	Inflammatory Outcomes Measured	Main Results, Adjustment/Confounders, Adverse Events	References
RCT; <i>n</i> = 75 healthy men	Black tea vs. placebo; 6 weeks	CRP; platelet activation	↓ CRP (<i>p</i> = 0.05); ↓ platelet activation	Steptoe et al., 2007 [34]
RCT; <i>n</i> = 35 metabolic syndrome	Green tea vs. extract vs. control; 8 weeks	CRP, IL-6, IL-1 β , SAA	No CRP change; ↓ SAA (<i>p</i> < 0.005)	Basu et al., 2011 [36]
RCT; <i>n</i> = 83 obese women	EGCG 300 mg/day; 12 weeks	CRP	No significant effect	Ayuso et al., 2014 [37]
Cross-sectional; <i>n</i> = 2592	Dietary flavonoids/tea	CRP	↓ CRP (OR 0.61, CI 0.44–0.86)	Hsieh et al., 2021 [50]
Cross-sectional; <i>n</i> = 1031	Tea consumption vs. none	CRP, SAA, haptoglobin	Lower inflammatory markers; adjusted	De Bacquer et al., 2006 [51]
RCT; <i>n</i> = 50 T2DM	EGCG 300 mg/day; 2 months	IL-6; TAC	↑ TAC (<i>p</i> = 0.001); no IL-6 change	Bazyar et al., 2021 [38]
RCT; <i>n</i> = 50 T2DM	EGCG 300 mg/day; 8 weeks	hs-CRP	↓ hs-CRP (<i>p</i> = 0.003)	Hadi et al., 2020 [39]
RCT; high-risk adults	Black tea (~3 cups/day); 12 weeks	CRP; uric acid	↓ CRP up to ~50% in high-risk groups	Bahorun et al., 2010 [40]
RCT crossover; <i>n</i> = 37	Flavonoids (epicatechin/quercetin)	IL-1 β ; endothelial markers	↓ IL-1 β (<i>p</i> = 0.009); ↓ inflammation score	Dower et al., 2015 [42]
Cross-sectional; <i>n</i> = 4139	Tea consumption	CRP	↓ CRP (~−12.2%, <i>p</i> = 0.042)	Rebello et al., 2011 [52]
RCT crossover; <i>n</i> = 40	GTE (~890 mg catechins/day); 4 weeks	IL-6; TNF- α ; endotoxin	↓ endotoxin (<i>p</i> = 0.023); no change in IL-6/TNF- α	Zeng et al., 2024 [45]

Footnote: Studies are categorized by primary outcome domain as defined in the manuscript. Some studies reported outcomes in both domains but are listed according to their primary focus. The table summarizes the twelve studies evaluating systemic inflammatory markers (e.g., CRP, IL-6, IL-1 β , TNF- α , SAA, endotoxin), including study design, population, green tea/black tea exposure and comparator, inflammatory outcomes measured, and main results with adjustment for confounders and adverse events, ordered by study identifier as listed in the References column. Abbreviations: CI, confidence interval; CRP, C-reactive protein; EGCG, epigallocatechin-3-gallate; GTE, green tea extract; hs-CRP, high-sensitivity C-reactive protein; IL-1 β , interleukin-1 β ; IL-6, interleukin-6; OR, odds ratio; RCT, randomized controlled trial; SAA, serum amyloid A; T2DM, type 2 diabetes mellitus; TAC, total antioxidant capacity; TNF- α , tumor necrosis factor- α . Note: (↑) indicates increase; (↓) indicates decrease.

3.4. Safety and Tolerability

Green tea and its extracts were well tolerated across studies. No serious adverse events were reported. Mild gastrointestinal symptoms and transient discomfort were occasionally noted but did not lead to discontinuation. Laboratory safety parameters remained within normal ranges.

The evidence indicates a dual immunomodulatory effect of green tea in humans. Functional studies consistently demonstrate enhanced immune responsiveness, particularly in innate and mucosal immunity, whereas biomarker-based studies show variable anti-inflammatory effects. These findings support a model in which green tea acts as a context-dependent immunomodulator, enhancing host defense while exerting selective anti-inflammatory effects in populations with elevated inflammatory burden.

3.5. Risk of Bias and Study Quality

A detailed summary of the risk-of-bias assessments for all 19 included studies is presented in Table 3 and the Supplementary Tables (Table S3–S5). The following narrative synthesizes the key findings.

Table 3. Risk of bias and quality assessment of included human studies.

Design	Key Methodological Strengths	Main Bias Concerns	Overall Judgment	References
Randomized, double-blind, placebo-controlled parallel trial	Washout run-in; matched caffeine placebo; numbered randomization; investigators blinded; objective compliance assessment	Attrition before analysis; modest sample size	Low risk/high quality	Steptoe 2007 [34]
Randomized, double-blind, placebo-controlled trial	Double-blind design; placebo control; good compliance; blinding check reported	Some uncertainty around monthly adherence and symptom self-reporting	Low risk/high quality	Rowe 2007 [35]
Randomized controlled trial, single-blind	Age/sex-matched trios; defined interventions	Participants not blinded; no-treatment comparator; unequal contact intensity; final analysis on incomplete trios	High risk/low quality	Basu 2011 [36]
Randomized, double-blind, placebo-controlled trial	Placebo-controlled; double-blind; liver safety monitored	Outcome of interest secondary; relatively homogeneous obesity-only sample	Low risk/high quality	Ayuso 2014 [37]
Randomized, double-blind, placebo-controlled trial	Placebo control; clinical population; pre/post assessment	Small sample; incomplete reporting of allocation concealment	Some concerns/moderate quality	Baziar 2021 [38]
Randomized, double-blind, placebo-controlled trial	Blinded placebo-controlled design	Small sample; hs-CRP mainly improved versus baseline rather than clearly between groups; limited reporting of concealment	Some concerns/moderate quality	Hadi 2020 [39]
Prospective randomized controlled study	Parallel control group; 12-week intervention plus washout	Blinding not clearly reported; subgroup-driven results; cardiovascular-risk sample may introduce co-intervention effects	Some concerns/moderate quality	Bahorun 2010 [40]
Randomized controlled trial	Flavonoid-free caffeine-matched control; repeated measures at 3 and 6 months	Limited detail on concealment in extracted text; immune biomarkers indirect	Low risk/high quality	Gostner 2015 [41]
Randomized, double-blind, placebo-controlled crossover trial	Crossover design; placebo control; mixed-model repeated-measures analysis	Short intervention periods; biomarkers rather than clinical outcomes	Low risk/high quality	Dower 2015 [42]
Randomized, double-blind, parallel-group comparative trial	Active comparator; blinded parallel-group design; safety reporting	Limited reporting on allocation methods; symptom-score outcomes susceptible to measurement bias	Some concerns/moderate quality	Wang 2011 [43]

Table 3. Cont.

Design	Key Methodological Strengths	Main Bias Concerns	Overall Judgment	References
Randomized, single-blind, placebo-controlled trial	Placebo control; all participants completed intervention; immune outcomes included NK and SIgA	Single-blind only; main signal emerged in stratified/per-protocol analyses; full text unavailable for complete appraisal	Some concerns/moderate quality	Tanaka 2021 [44]
Randomized, double-blind, placebo-controlled crossover trial	Rigorous crossover design; circulating catechins confirmed adherence; clearly defined primary endpoint	Short intervention duration; modest sample for subgroup analyses	Low risk/high quality	Zeng 2024 [45]
Non-randomized intervention	Human in vivo exposure with comparator beverage	Small sample; unclear randomisation; limited participant-characteristic reporting; functional lab outcomes only	Serious risk/low quality	Kamath 2003 [46]
Open-label pre–post intervention	Objective laboratory outcomes; washout reported	No parallel control; no blinding; small sample	Serious risk/low quality	Lowe 2015 [47]
Single-arm pilot study	Defined catechin dose; adherence and adverse events tracked	No control group; no blinding; small sample; authors explicitly acknowledge confounding and placebo effects	Serious risk/low quality	Iketani 2019 [48]
Non-randomised clinical intervention	Disease-specific clinical population; longitudinal immune readouts	Very small sample; attrition; no randomised untreated CLL comparator; sponsor-linked authorship noted	Serious risk/low quality	D’Arena 2013 [49]
Cross-sectional observational study	Large sample; multivariable adjustment for waist circumference, smoking, education, coffee, alcohol, and activity	Cross-sectional design; residual confounding; tea exposure self-reported	Moderate quality	De Bacquer 2006 [51]
Cross-sectional observational study	Large multi-ethnic cohort; extensive adjustment for BMI, activity, smoking, alcohol, hypertension, dyslipidaemia, diet	Cross-sectional design; dietary recall and beverage self-report; reverse causation cannot be excluded	Moderate quality	Rebello 2011 [52]
Cross-sectional observational study	Nationally representative sample; multivariable adjustment including BMI, smoking, diet, and cardiometabolic factors	Cross-sectional design; flavonoid intake estimated from food tables and 24 h recall	Moderate quality	Hsieh 2021 [50]

Abbreviations: BMI, body mass index; CLL, chronic lymphocytic leukemia; hs-CRP, high-sensitivity C-reactive protein; NK, natural killer; SIgA, secretory immunoglobulin A.

Risk of bias varied substantially by study design. Among the 12 RCTs, the most methodologically robust were the double-blind, placebo-controlled studies by Steptoe 2007 [34], Rowe 2007 [35], Ayuso 2014 [37], Gostner 2015 [41], Dower 2015 [42], and Zeng 2024 [45], which reported random allocation, blinded intervention delivery, and prespecified control conditions; Steptoe additionally used a washout period and objective compliance assessment, whereas Zeng used a crossover design with circulating catechin measurements to support adherence. Trials by Bazzyar 2021 [38], Hadi 2020 [39], Bahorun

2010 [40], Wang 2011 [43], and Tanaka 2021 [44] were judged as having some concerns, mainly because of limited reporting of allocation concealment, small sample size, single-blind design, or outcome analyses that relied on per-protocol or subgroup comparisons; Tanaka 2021 [44] was further limited by the lack of accessible full text. Basu 2011 [36] was judged at high risk of bias because participants could not be blinded, the comparator included a no-treatment arm, visit intensity differed across groups, and the final analysis was based on incomplete matched trios rather than the full randomized sample. Domain-level judgments and signaling questions for each RCT are detailed in Supplementary Table S3.

All non-randomized interventional studies were at serious risk of bias. Kamath 2003 used a small non-randomized tea-versus-coffee comparison [46]; Lowe 2015 was open-label and pre-post without a parallel control [47]; Iketani 2019 was explicitly a single-arm pilot [48]; and D'Arena 2013 was a small uncontrolled clinical intervention with attrition and no randomized disease-specific comparator [49]. The ROBINS-I domain-level judgments for these studies are presented in Supplementary Table S4.

The observational studies by De Bacquer 2006 [51], Rebello 2011 [52], and Hsieh 2021 [50] were of moderate quality: each used multivariable adjustment for major confounders, but all were cross-sectional and therefore remained vulnerable to residual confounding and reverse causation. The Newcastle-Ottawa Quality Assessment for these observational studies is provided in Supplementary Table S5.

4. Discussion

In this systematic review of 19 human studies involving approximately 6800 participants, we observed a consistent pattern of enhanced functional immune responses following green tea consumption, alongside heterogeneous effects on systemic inflammatory markers. This divergence represents a central finding with important implications: green tea appears to function as a context-dependent immunomodulator rather than a uniformly anti-inflammatory or immunostimulatory agent. The distinction between these two biological domains—immune competence versus inflammatory burden—has been inadequately addressed in previous syntheses, and our findings suggest that conflating them may obscure meaningful biological effects.

4.1. Functional Immune Enhancement: A Consistent Finding

The consistency of findings across immune function studies is noteworthy. Eight studies evaluating outcomes such as NK cell activity, $\gamma\delta$ T cell responses, and mucosal immunity demonstrated significant enhancements following green tea consumption. The magnitude of effects was clinically meaningful: NK cell activity increased by a mean of 17.1 percentage points in elderly participants [48], while $\gamma\delta$ T cell proliferation increased by 28% and IFN- γ secretion by 26% in healthy adults [35]. These improvements translated into tangible clinical benefits, including a 32.1% reduction in cold and influenza symptoms [35] and a substantially reduced odds of upper respiratory tract infections (odds ratio 0.17) [44].

The mechanistic basis for these effects is supported by experimental evidence. Green tea catechins, particularly EGCG, have been shown to directly enhance cytotoxic activity of NK cells and $\gamma\delta$ T cells through modulation of signaling pathways including mitogen-activated protein kinase (MAPK) and phosphoinositide 3-kinase (PI3K) [29,53]. Additionally, catechins may promote the differentiation and proliferation of effector T cells while simultaneously modulating regulatory T cell populations, as observed in the chronic lymphocytic leukemia cohort [49,54]. The finding that these effects occurred across diverse populations—including healthy adults, elderly individuals, and clinical populations—suggests that the immunostimulatory properties of green tea are broadly conserved and not limited to specific demographic or health contexts.

The enhancement of mucosal immunity, evidenced by increased secretory immunoglobulin A (SIgA) levels [44] and improved leukocyte functional activity [47], is particularly relevant given that mucosal surfaces represent the primary interface between the host and environmental pathogens. This suggests that green tea consumption may strengthen first-line immune defenses, potentially reducing susceptibility to respiratory and gastrointestinal infections [55,56]. The rapid time course of these effects, with changes observed within 2–4 weeks of intervention, indicates that green tea catechins exert relatively acute immunomodulatory effects that are likely mediated through direct cellular actions rather than through long-term epigenetic or metabolic reprogramming.

4.2. Anti-Inflammatory Effects: Context Dependency and Heterogeneity

In contrast to the consistent immune-enhancing effects, the anti-inflammatory effects of green tea were markedly heterogeneous. Of the twelve studies evaluating systemic inflammatory markers, approximately half reported significant reductions in CRP or related biomarkers, while the remainder found no significant changes. This variability appears to be systematically related to population characteristics, with the most pronounced effects observed in individuals with elevated baseline inflammatory burden.

Several lines of evidence support this context-dependent interpretation. In patients with type 2 diabetes mellitus, EGCG supplementation significantly reduced high-sensitivity CRP ($p = 0.003$) [39], while in individuals at high cardiovascular risk, black tea consumption reduced CRP by up to 50% [40]. Similarly, in the crossover trial by [45], green tea extract reduced circulating endotoxin ($p = 0.023$) and improved gut barrier function, but did not alter systemic IL-6 or TNF- α levels in the overall cohort. The magnitude of anti-inflammatory effects appeared to correlate with baseline inflammatory status, suggesting that green tea may be most effective in populations with pre-existing low-grade inflammation—a pattern that could be conceptually interpreted, albeit speculatively, through the “reserve capacity” framework proposed in nutritional interventions [57].

The absence of consistent effects on inflammatory cytokines such as IL-6 and TNF- α in metabolically stable or healthy populations further supports the threshold hypothesis: green tea catechins may exert anti-inflammatory effects primarily when inflammatory pathways are actively upregulated, with minimal impact under homeostatic conditions. This pattern is biologically plausible, as catechins inhibit NF- κ B signaling and downstream pro-inflammatory cytokine production [58], but these pathways may already be quiescent in healthy individuals. The observation that oxidative stress markers improved in some studies [38,47] while cytokine levels remained unchanged suggests that the antioxidant and anti-inflammatory effects of green tea may operate through partially distinct mechanisms.

The heterogeneity in findings cannot be attributed solely to population differences. Intervention characteristics, including catechin dose, formulation (beverage versus extract), duration, and bioavailability, likely contributed to the observed variability. Studies using higher doses of catechins (≥ 800 mg/day) or longer intervention periods (≥ 12 weeks) tended to show more consistent anti-inflammatory effects, although this pattern was not universal. Bioavailability represents a particularly important consideration: EGCG undergoes extensive first-pass metabolism and exhibits relatively low systemic bioavailability (approximately 0.1–0.5% of ingested dose) [2], suggesting that local effects in the gastrointestinal tract—including modulation of gut microbiota and barrier function—may be equally or more important than systemic actions in mediating anti-inflammatory effects. The demonstration by [45] that green tea extract reduced circulating endotoxin without altering systemic cytokines provides direct support for this gut-centric mechanism.

The gut microbiota is increasingly recognized as a central mediator of green tea's systemic effects. Because most ingested catechins are poorly absorbed in the small intestine, a substantial fraction reaches the colon, where they are metabolized by resident bacteria into smaller phenolic acids and valerolactones that are more bioavailable and biologically active than the parent compounds. This microbial biotransformation is bidirectional: catechins and their metabolites can also reshape microbial community composition, favoring taxa associated with short-chain fatty acid production and barrier maintenance, while suppressing potentially pro-inflammatory taxa. These microbial metabolites and short-chain fatty acids can, in turn, modulate intestinal immune signaling, including regulatory T cell induction and epithelial tight-junction integrity, thereby linking gut barrier function to systemic inflammatory tone. The finding that green tea extract reduced circulating endotoxin without altering systemic cytokines is consistent with this gut-centric pathway, in which effects on microbiota composition and intestinal permeability may precede, or substitute for, detectable changes in circulating inflammatory markers. Future studies incorporating gut microbiome sequencing and metabolite profiling alongside immune and inflammatory endpoints would help clarify the relative contribution of this pathway to the dual immunomodulatory effects described here.

4.3. Bridging the Two Domains: A Dual Immunomodulatory Model

The concurrent findings of enhanced immune function and variable anti-inflammatory effects suggest that green tea acts through a dual immunomodulatory mechanism rather than through a single unified pathway [59]. This conceptual model has important biological and clinical implications. Enhanced immune surveillance, manifested through increased NK cell activity and T cell responses, represents a state of heightened readiness that may improve host defense against pathogens [60]. Simultaneously, the attenuation of inflammatory responses—when present—may reduce the deleterious effects of chronic low-grade inflammation, including endothelial dysfunction, insulin resistance, and tissue damage [61–63].

The kynurenine pathway findings from [41] are particularly instructive in this regard. The observed increase in circulating kynurenine concentrations following black tea consumption suggests activation of indoleamine 2,3-dioxygenase (IDO), a key enzyme in tryptophan catabolism that plays a central role in immune regulation. IDO activation is associated with both immune activation (through tryptophan depletion and metabolite production) and immune tolerance (through regulatory T cell induction), illustrating the complexity of green tea's immunomodulatory effects. This dual capacity to enhance immune surveillance while limiting excessive inflammation could conceptually, though speculatively, be framed as an evolutionary adaptation; this interpretation is offered as a hypothesis-generating perspective rather than an established mechanism, given that the immune system must maintain vigilance against pathogens while avoiding autoimmunity and chronic inflammatory damage (Figure 2).

The clinical relevance of this dual modulation is supported by the infection-related outcomes observed in several studies. The reduction in upper respiratory tract infections [35,44] and improved symptom resolution in pharyngitis [43] indicate that the immune-enhancing effects translate into meaningful clinical benefits. Conversely, the anti-inflammatory effects in high-risk populations, while more modest, may contribute to long-term risk reduction for cardiovascular disease, metabolic syndrome, and other inflammation-related conditions [30,57].

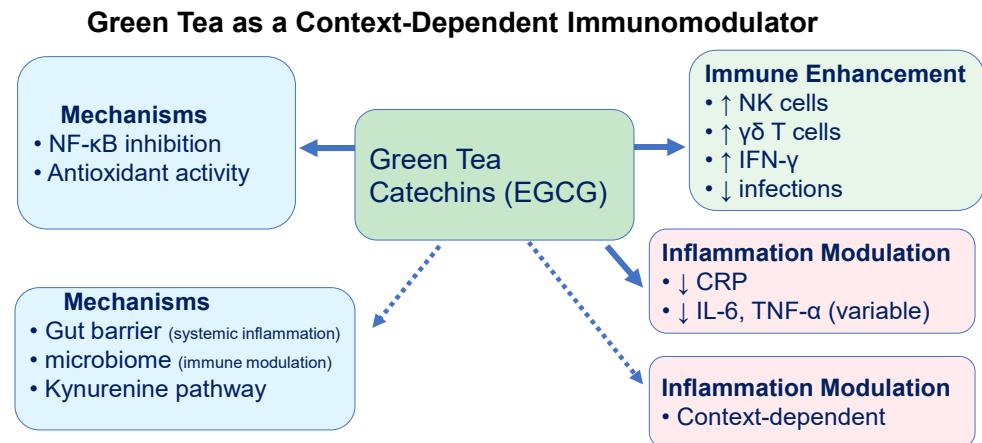


Figure 2. Immunomodulatory effects of green tea. Green tea as a context-dependent immunomodulator in humans. Green tea-derived catechins, particularly epigallocatechin-3-gallate (EGCG), exert coordinated immunomodulatory effects through multiple interconnected biological pathways. At the mechanistic level, catechins inhibit nuclear factor- κ B (NF- κ B) signaling, reduce oxidative stress, and modulate gut barrier integrity and microbiome composition, thereby influencing systemic immune responses. Arrow legend: Solid arrows (—) indicate pathways with well-supported evidence from human or mechanistic studies (e.g., NF- κ B inhibition, NK cell activation, CRP reduction). Dashed arrows (---) indicate hypothetical or emerging relationships that require further investigation (e.g., kynurenine pathway-mediated effects, microbiome–immune crosstalk, context-dependent modulation based on baseline inflammation). The variable anti-inflammatory effects ($\downarrow$ IL-6, TNF- α) are marked with a dashed box to reflect the heterogeneity observed across populations and study designs. These upstream effects translate into enhanced immune function, including increased natural killer (NK) cell activity, augmented $\gamma\delta$ T-cell responses, increased interferon- γ (IFN- γ) production, and improved mucosal immunity (e.g., secretory IgA), collectively contributing to reduced susceptibility to infections. Concurrently, green tea modulates inflammatory pathways, with reductions in circulating biomarkers such as C-reactive protein (CRP) and, in some populations, interleukin-6 (IL-6) and tumor necrosis factor- α (TNF- α), as well as decreased endotoxemia. However, these anti-inflammatory effects are variable and appear to depend on baseline inflammatory status, metabolic health, and duration of exposure. Together, these findings support a dual, context-dependent immunomodulatory role, in which green tea enhances host defense while selectively attenuating excessive inflammation.

4.4. Methodological Considerations Sources of Heterogeneity

Several methodological factors contributed to the heterogeneity observed across studies and warrant systematic consideration.

First, variation in study design introduced different sources of bias. The most methodologically robust RCTs (double-blind, placebo-controlled with objective compliance assessment) tended to show more modest anti-inflammatory effects than observational studies, which consistently reported inverse associations between tea consumption and inflammation. This discrepancy likely reflects both residual confounding in observational designs and the challenges of translating population-level associations to controlled intervention settings.

Second, heterogeneity in tea formulations represents a critical source of variability. Studies used different forms of tea (beverages versus extracts), different tea varieties (green versus black tea), and varying catechin content (from approximately 300 mg to >900 mg/day). Black tea, which undergoes fermentation and contains theaflavins and thearubigins in addition to catechins, may have distinct biological effects from green tea. The absence of standardized dosing and the poor reporting of catechin content in several studies limit the ability to draw dose–response conclusions. These sources of heterogeneity can be summarized along several explicit axes: beverage versus extract formulation, purified EGCG versus whole-tea catechin mixtures, green versus black (fermented) tea,

catechin concentration, and caffeine content, which co-varies with tea intake and may independently influence inflammatory and immune parameters. Because these factors were not systematically stratified across the included studies, their individual contributions to the observed heterogeneity cannot be disentangled with the current evidence, and future trials should explicitly report and control for each of these dimensions.

Third, intervention duration varied widely, from 2 weeks to 6 months. The time course of different biological effects may differ: functional immune changes may occur relatively rapidly through direct cellular actions, whereas changes in systemic inflammatory biomarkers may require longer periods to manifest, particularly in populations without overt inflammation.

Fourth, population diversity contributed to heterogeneity. Populations ranged from healthy young adults to elderly individuals and clinical populations with diverse metabolic and inflammatory profiles. While this diversity enhances generalizability, it complicates interpretation of pooled findings. The apparent context-dependency of anti-inflammatory effects suggests that future studies should stratify populations by baseline inflammatory status and metabolic health.

Fifth, the distinction between green and black tea warrants explicit consideration. While we included black tea studies within the broader framework of tea polyphenols, the fermentation process alters the polyphenolic profile substantially. Subgroup analyses by tea type were not possible due to the limited number of studies, but this represents an important direction for future research.

4.5. Safety and Tolerability

Green tea and its extracts were well tolerated across all included studies. No serious adverse events were reported, and no study reported discontinuations due to treatment-related effects. Mild gastrointestinal symptoms (e.g., nausea, abdominal discomfort) and transient symptoms were occasionally noted but did not lead to study withdrawal. Laboratory safety parameters, including liver function tests, remained within normal ranges in all studies that monitored them.

However, the safety profile of green tea warrants careful consideration in the context of formulation and dose. The studies included in this review used tea beverages (3–6 cups/day) or moderate-dose extracts (300–900 mg/day of catechins), which are generally recognized as safe. By contrast, high-dose EGCG supplements (>800–1000 mg/day) have been associated with rare instances of hepatotoxicity in case reports and clinical trials, particularly when taken on an empty stomach. This dose-dependent safety concern underscores the importance of distinguishing between dietary beverage consumption and high-dose supplement use in public health recommendations. The caffeine content of tea beverages (approximately 30–50 mg per cup) was not associated with adverse cardiovascular or gastrointestinal effects in the included studies, though individuals sensitive to caffeine should exercise caution.

Based on the available evidence, public health messaging should emphasize consumption of green tea through dietary beverages rather than high-dose extracts, until further safety data are available to establish the upper limit of safe catechin intake. Healthcare providers should counsel patients using high-dose green tea supplements about the potential for hepatotoxicity and the importance of taking supplements with food to reduce gastrointestinal irritation.

4.6. Comparison with Previous Studies

Our findings align with and extend previous systematic reviews and meta-analyses. A meta-analysis by [57] reported modest but significant reductions in CRP and IL-6 following

green tea supplementation, particularly in populations with metabolic disorders [57]. Similarly, [30] found that green tea supplementation reduced inflammatory markers in patients with metabolic syndrome, but with considerable heterogeneity [30]. However, these previous syntheses did not systematically distinguish between functional immune outcomes and systemic inflammatory markers, nor did they address the concept of context-dependency in a comprehensive manner.

Our review extends this literature by demonstrating that the enhancement of functional immune responses is more consistent than the anti-inflammatory effects, suggesting that these are distinct biological processes with different determinants. This distinction has important implications for the design of future studies and for the interpretation of existing evidence. It also highlights the need for nutritional immunology research to adopt more nuanced outcome frameworks that distinguish between different aspects of immune function rather than treating inflammation as a proxy for overall immune status.

4.7. Clinical and Public Health Implications

The findings of this systematic review have several implications for clinical practice and public health recommendations. First, the consistent enhancement of functional immune responses supports the use of green tea consumption as a dietary strategy to support immune function, particularly in populations at risk of infections, such as the elderly or those with compromised immunity. The reduction in upper respiratory tract infections observed in two RCTs [35,44] provides direct evidence of clinical benefit, although the effect size and generalizability remain to be established.

Second, the context-dependent anti-inflammatory effects suggest that green tea may be most beneficial in populations with elevated inflammatory burden, such as those with type 2 diabetes, metabolic syndrome, or established cardiovascular risk. In these populations, the anti-inflammatory effects may complement other lifestyle interventions and pharmacological therapies. Conversely, in healthy individuals without evidence of systemic inflammation, the anti-inflammatory effects appear to be limited, suggesting that public health messages should avoid overstating the anti-inflammatory benefits in low-risk populations.

Third, the safety and tolerability profile of green tea was excellent across all studies, with no serious adverse events reported. This favorable safety profile, combined with the low cost and widespread availability of green tea, supports its consideration as a safe dietary intervention. However, the potential for liver toxicity at very high doses (exceeding those used in these studies) should be noted, and public health recommendations should emphasize consumption through dietary beverages rather than high-dose supplements.

4.8. Limitations and Future Directions

Several limitations should be considered. First, substantial heterogeneity in study design, populations, interventions, and outcome measures precluded quantitative meta-analysis. This was not merely due to an insufficient number of studies; several outcomes were reported frequently enough for pooling (e.g., CRP in eleven studies and NK cell activity in a subset of immune-related trials). However, these apparently similar outcomes were not clinically or methodologically comparable. Studies differed simultaneously in intervention form and catechin dose, comparator type, treatment duration, and—most critically—participants' baseline inflammatory or metabolic status. Our findings indicate that baseline status is a key effect modifier, with anti-inflammatory benefits appearing more pronounced in populations with elevated inflammation. Pooling data across such heterogeneous conditions would have produced a summary estimate that is difficult to interpret for any specific population and would have obscured the context-dependent effects that represent one of this review's main findings. This limitation stems primar-

ily from the current evidence base rather than from the review methodology itself. This limitation is inherent to the current evidence base rather than to the review methodology, and it underscores the need for future trials using harmonized dosing, comparator, and outcome-reporting standards to enable a meaningful quantitative synthesis. Second, many trials had modest sample sizes and short durations, limiting statistical power to detect changes in systemic biomarkers, particularly in healthy populations. Third, variation in tea formulation (beverage vs. extract), catechin content, and bioavailability likely influenced outcomes and complicates dose–response interpretation. Fourth, the risk of bias varied considerably, with non-randomized interventions showing serious methodological limitations and observational studies vulnerable to residual confounding. Fifth, most studies were conducted in Western or East Asian populations, limiting generalizability to other ethnic groups and dietary contexts.

Future research should prioritize well-designed, long-term randomized controlled trials with standardized catechin dosing, objective compliance measures (e.g., plasma catechin levels), and clearly defined primary outcomes that distinguish between immune function and inflammatory markers. Mechanistic studies integrating gut microbiome analysis, metabolic profiling, and immune cell phenotyping would help elucidate the pathways underlying the dual immunomodulatory effects observed here. Dose–response studies are needed to establish optimal intake levels, while population-based research should explore effect modification by baseline inflammatory status, age, and metabolic health. Finally, studies evaluating clinically meaningful endpoints—such as infection incidence, disease progression, and quality of life—would strengthen the translational relevance of findings.

5. Conclusions

This systematic review of 19 human studies demonstrates that green tea consumption consistently enhances functional immune responses, including increased NK cell activity, augmented $\gamma\delta$ T cell function, and improved mucosal immunity, while exerting modest, context-dependent anti-inflammatory effects primarily in populations with elevated baseline inflammation. These findings support a dual immunomodulatory role for green tea, with immune-enhancing effects more consistent across populations and anti-inflammatory benefits more pronounced in high-risk groups. The distinction between these two biological domains is clinically meaningful and should inform both future research design and public health recommendations. Based on the available evidence, public health messaging should emphasize that green tea consumption may be most beneficial for infection prevention in healthy individuals (based on consistent immune-enhancing effects) and for inflammation reduction in populations with metabolic syndrome, diabetes, or cardiovascular risk (based on context-dependent anti-inflammatory effects). Green tea appears to be a safe, accessible dietary strategy to support immune function, particularly in populations vulnerable to infections, while its anti-inflammatory utility may be greatest in individuals with elevated baseline inflammatory burden. Consumption through dietary beverages is preferred over high-dose extracts until further safety data are available.

Supplementary Materials: The following supporting information can be downloaded at: <https://www.mdpi.com/article/10.3390/app16157515/s1>, Table S1: PRISMA 2020 Checklist [64]; Table S2: Full Boolean search strings for each database; Table S3: RoB 2 domain-level judgments for randomized controlled trials; Table S4: ROBINS-I domain-level judgments for non-randomized interventional studies; Table S5: Newcastle-Ottawa Quality Assessment for observational studies.

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