REVIEW ARTICLE

The impact of biological treatments in dermatology on the risk of cardiovascular disease: a systematic review and single-arm meta-analysis

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ABSTRACT

Chronic inflammatory skin diseases, including psoriasis and atopic dermatitis, are associated with an increased risk of cardiovascular disease (CVD), largely due to systemic inflammation. Biologic therapies that target key inflammatory cytokines have shown promise not only in improving skin outcomes but also in potentially modifying cardiovascular risk. This study aims to evaluate the impact of biologic treatments used in dermatology on CVD risk through a systematic review and single-arm meta-analysis. Databases including PubMed, Google Scholar, Web of Science, Medline, Scopus, Wiley, EBSCO, and ScienceDirect were searched for studies reporting cardiovascular outcomes in patients receiving biologics for dermatologic conditions. Eligible studies included randomized controlled trials (RCTs), observational cohorts, and case series. Cardiovascular outcomes assessed included major adverse cardiovascular events, myocardial infarction, stroke, and changes in cardiovascular risk factors. A total of 21 studies were included. The pooled proportion of patients experiencing cardiovascular outcomes after biologic therapy was 7.82% (95% confidence intervals: 5.31%-11.37%) under the random-effects model. A modest but significant correlation ($r = 0.2051$, $p = 0.0126$) between biologics and cardiovascular benefit was observed. Sensitivity analyses supported the robustness of findings. Risk of bias ranged from low to moderate. Biologic therapies in dermatology, particularly tumor necrosis factor-α, IL-17, and IL-23 inhibitors, may reduce cardiovascular risk through systemic inflammation suppression. However, heterogeneity, publication bias, and a predominance of observational data limit the strength of conclusions. Further RCTs are needed to confirm these findings.

Keywords: Psoriasis, biologic therapy, cardiovascular disease, meta-analysis, MACE, TNF inhibitors.

Introduction

Plaque psoriasis is a chronic immune-mediated condition marked by cutaneousU and/or articular symptoms and systemic inflammation [1]. This inflammation has been identified as an independent contributor to the development of cardiovascular disease (CVD) [2]. The prevalence of CVD risk factors has been seen to be higher in patients with psoriasis, including hypertension, diabetes, dyslipidemia, obesity, and metabolic syndrome [3]. Research suggests that treating the underlying psoriasis may help reduce cardiovascular risk as systemic inflammation appears to play a central role in both conditions [4]. Major adverse cardiovascular events (MACEs), including myocardial infarction (MI), stroke, and cardiovascular death, have been reported more frequently in patients with moderate-to-severe psoriasis [5].

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Recent therapeutic advancements have introduced biologic agents specifically targeting cytokines involved in the inflammatory cascade, such as tumor necrosis factor (TNF)-α, interleukin (IL)-23, and IL-17. Specifically, TNF-α inhibitors have been foundational in demonstrating that reducing systemic inflammation can decrease endothelial adhesion molecules. Furthermore, IL-17 and IL-23 inhibitors have recently transformed dermatologic care; IL-17 inhibitors directly target the effector cytokine responsible for both plaque formation and vascular inflammation, while IL-23 inhibitors block the upstream activation of Th17 cells. These therapies have proven to be highly effective for psoriasis management and represent emerging treatment strategies that may have profound systemic implications, extending beyond the skin to potentially halt premature atherosclerosis. By mitigating systemic inflammation, these agents may potentially improve cardiovascular outcomes, reduce insulin resistance, and ameliorate metabolic abnormalities linked to atherosclerosis [6,7].

Despite these findings, the literature remains inconsistent regarding the extent of cardiovascular benefit provided by biologic therapies. Existing studies vary significantly in design, population, and outcomes, creating a critical research gap. There is a need to synthesize available evidence to determine if the systemic anti-inflammatory effects of biologics translate into tangible cardiovascular risk reduction. This systematic review and meta-analysis aims to address this gap by evaluating the overall impact of biologic treatments used in dermatology on CVD risk.

Despite these findings, the literature remains inconsistent regarding the extent of cardiovascular benefit provided by biologic therapies. Existing studies vary significantly in design, population, and outcomes, creating a critical research gap. Specifically, there is an absence of a comprehensive quantitative synthesis that aggregates single-arm event rates across various biologic classes to establish a clear, updated baseline cardiovascular risk profile for this demographic, strongly justifying the urgent need for this study. There is a need to synthesize available evidence to determine if the systemic anti-inflammatory effects of biologics translate into tangible cardiovascular risk reduction. This systematic review and meta-analysis aim to address this gap by evaluating the overall impact of biologic treatments used in dermatology on CVD risk.

Materials and Methods

Study design and population

This systematic review focused on studies that assessed the impact of biologic treatments on the risk of CVD in patients with immune-mediated dermatologic conditions, including psoriasis, atopic dermatitis, hidradenitis suppurativa, and alopecia areata. This review was prospectively registered with the International Prospective Register of Systematic Reviews, registration (ID: CRD420251004067).

Search strategy

This systematic review was conducted in accordance with the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines [8]. A comprehensive literature search was conducted across multiple databases, including PubMed, Google Scholar, Web of Science, Medline, Scopus, Wiley, EBSCO, and ScienceDirect. The search terms combined keywords related to biologic therapies, dermatologic conditions, and CVD: (“biologic therapy” OR “biologics” OR “biological treatment” OR “monoclonal antibodies” OR “TNF inhibitors” OR “IL-17 inhibitors” OR “IL-23 inhibitors” OR “JAK inhibitors”) AND (“psoriasis” OR “atopic dermatitis” OR “hidradenitis suppurativa” OR “alopecia areata” OR “chronic inflammatory skin disease”) AND (“CVD” OR “ MI” OR “stroke” OR “hypertension” OR “atherosclerosis” OR “dyslipidemia” OR “heart failure”). Search terms were tailored for each database to ensure optimal retrieval of relevant studies. No restrictions were applied to publication dates, and randomized controlled trials (RCTs), observational studies, and case reports/series were considered eligible for inclusion.

Study selection

Inclusion criteria encompassed RCTs, observational studies, case series with a minimum of five participants, and case reports. Eligible populations included patients with psoriasis, atopic dermatitis, hidradenitis suppurativa, or alopecia areata receiving biologic therapies. Relevant outcomes included cardiovascular risk measures such as MACE, MI, stroke, hypertension, atherosclerosis, dyslipidemia, arterial stiffness, and endothelial dysfunction. Studies such as reviews, editorials, letters, studies with high risk of bias (RoB), non-dermatologic populations, studies without biologic therapies, and those not reporting cardiovascular outcomes were excluded.

Screening and data management

All search results were imported into Mendeley reference management software to facilitate the screening and selection process. Two researchers independently reviewed titles and abstracts to identify potentially eligible studies. Full-text articles were then assessed against inclusion and exclusion criteria. Any discrepancies in study selection were resolved through discussion or consultation with a third reviewer. The reference lists of included studies and relevant review articles were manually screened to identify additional studies that may have been missed during the database searches.

Data extraction

Data were extracted using a standardized form, capturing study characteristics (design, location, year of publication, sample size, and follow-up duration), patient demographics (age, gender, dermatologic diagnosis, disease duration and severity, and baseline CVD risk factors), intervention details (biologic class, specific agent, dosage, and treatment duration), comparator treatments (type and specific drug names),

and cardiovascular outcomes (MACE, MI, stroke, hypertension, and related vascular measures).

Quality assessment

The quality of the included studies was assessed using the Cochrane RoB 2 Tool for RCTs and the ROBINS-I tool for nonrandomized comparative studies [9,10].

Statistical analysis

Data analysis was performed to evaluate the pooled proportion of cardiovascular outcomes. A random-effects model was employed to account for between-study variability. Heterogeneity among studies was assessed using the I^2 statistic, where I^2 values of 25%, 50%, and 75% represented low, moderate, and high heterogeneity, respectively. Effect sizes were calculated as pooled proportions with 95% confidence intervals (CIs). Sensitivity analyses were conducted to test the robustness of the results by excluding potential outliers. Subgroup analyses were planned based on biologic class and disease severity, subject to data availability. Meta-analysis was performed using Comprehensive Meta-Analysis software (Version 3.0, Biostat, Englewood, NJ) [11].

Results

Study selection

A total of 485 records were identified through database searches. After removing 263 duplicates, 222 records were screened by title and abstract. Of these, 183 were excluded for not meeting the inclusion criteria. The remaining 39 full-text articles were assessed for

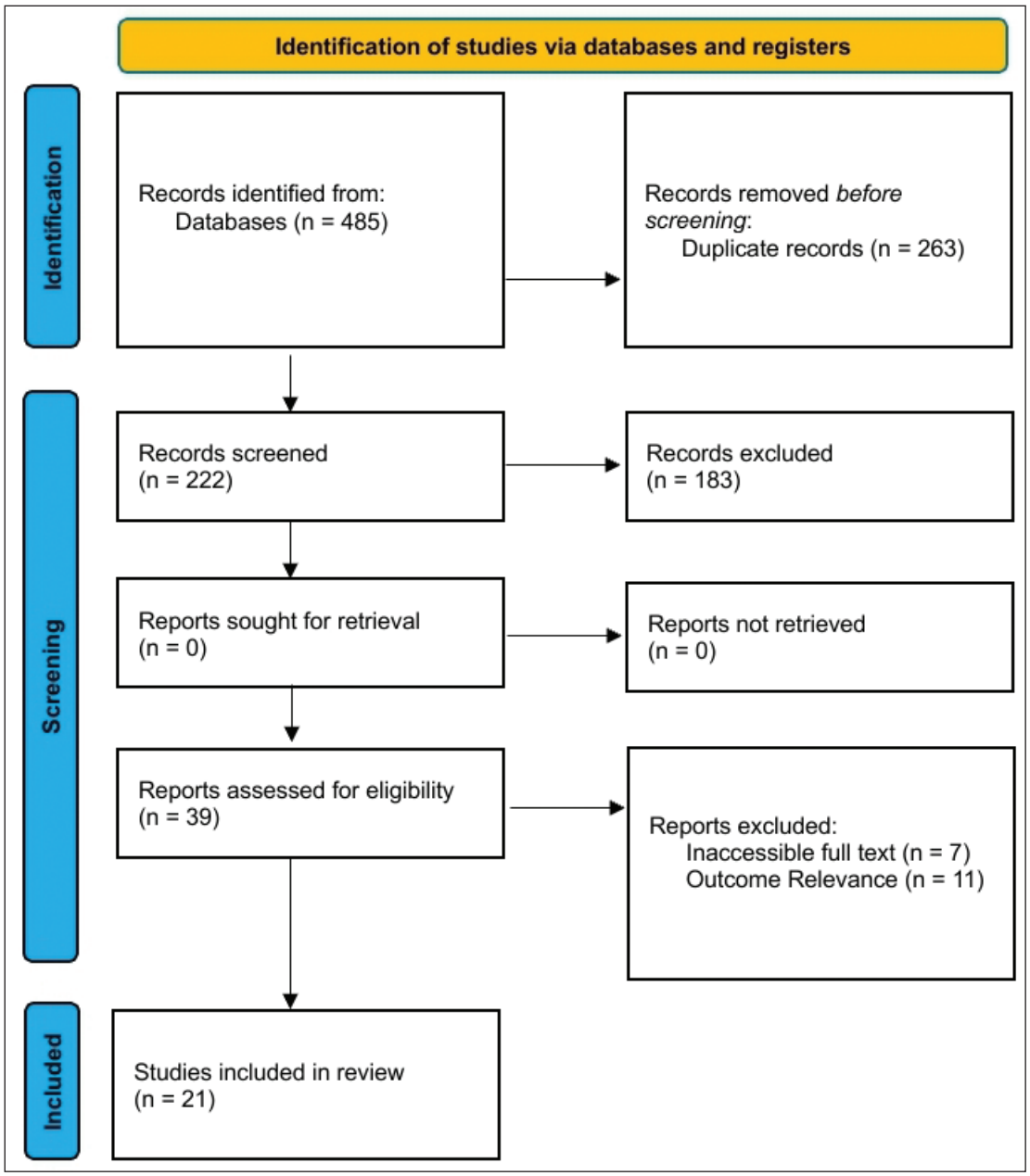

Figure 1. *PRISMA flow diagram of study selection.*

eligibility. Ultimately, 21 studies were included in the quantitative synthesis (meta-analysis), focusing on the single-arm estimation of cardiovascular outcomes in patients treated with biologic therapies for dermatologic conditions. Figure 1 presents the PRISMA flow diagram of the study selection process.

Characteristics of included studies

The 21 included studies were published between 2011 and 2025, originating from various countries, including the USA [12-18], South Korea [19-21], Australia [22], Germany [1,23,24], Canada [25-27], Spain [28], Japan [29], Denmark [30], and Kuwait [7]. Most studies were retrospective or prospective observational designs, with a few RCTs and single-arm clinical reports. Sample sizes varied widely, ranging from small cohorts to large population-based analyses with over 250,000 participants. Patient ages ranged from 21 to 73 years.

The primary outcomes of interest were cardiovascular events such as MI, stroke, MACE, and heart failure. Additional cardiovascular risk factors evaluated included changes in lipid profiles, blood pressure, inflammatory markers [e.g., neutrophil-to-lymphocyte ratio (NLR)], smoking status, obesity, and diabetes. Several studies also utilized formal cardiovascular risk tools, including the Framingham Risk Score and ASCVD risk calculators, although many did not report these numerically.

The included studies varied in design, setting, and sample size. Most studies were either retrospective or prospective observational in nature, with a few RCTs. Table 1 summarizes the general characteristics of the included studies.

Patient demographics, including comorbidities and baseline characteristics such as hypertension, obesity, or dyslipidemia, are presented in Table 2.

Details of biologic treatments—such as agent class (TNF-α, IL-17, IL-23 inhibitors), dosages, and treatment durations—are provided in Table 3.

Adverse events were inconsistently reported, with some studies noting atrial fibrillation, fractures, and treatment-related withdrawals, while others reported no significant safety concerns. A summary of CVD outcomes and adverse events observed across studies is provided in Tables 4 and 5.

RoB and methodological quality

The quality of the included studies was assessed using the Cochrane RoBS 2 Tool for RCTs and the ROBINS-I tool for nonrandomized comparative studies [9,10]. Case reports and series were assessed for causality and reporting rigor, and cross-sectional studies using

Table 1. *General studies characteristics.*

Study ID	Study design	Year of publication	Country of study	Number of patients included (on biological treatment)
Abuabara et al. [12]	Observational cohort study	2011	USA	12,224
Dey et al. [13]	Observational cohort study	2020	USA	316
Bissonnette et al. [25]	RCTs	2013	Canada	30
Cho et al. [19].	Nationwide population-based cohort study	2024	South Korea	2,886
Gelfand et al. [14].	RCTs, double-blinded, placebo-controlled trial	2021	United States	91
Genre et al. [28].	Cohort study	2023	Spain	29
Gulliver et al. [26].	Retrospective cohort study	2016	Canada	139
Hagino et al. [29].	Retrospective study	2023	Japan	165
Hjuler et al. [30].	Single-center prospective, controlled study	2016	Denmark	28
Hoffmann et al. [23].	Retrospective study	2021	Germany	143
Hong et al. [20].	Nationwide population-based cohort study	2021	South Korea	1,817
Kim et al. [21].	Case–control study	2023	Korea	251,813
Kridin et al. [15].	Retrospective cohort with propensity matching	2025	USA, Germany	16,780
Kridin et al. [24].	Cohort study	2025	Germany	8,410
Lee et al. [16].	Cohort study	2019	USA	60,028
Levesque et al. [27].	Retrospective cohort study	2013	Canada (Quebec)	506
Shaaban et al. [7].	Retrospective study	2018	Kuwait	4,762
Smith et al. [22].	Retrospective Study	2025	Australia	39
von Stebut et al. [1].	Randomized, double-blind, exploratory trial	2019	Germany	151
Wu et al. [17].	Retrospective cohort study	2012	United States	8,845
Wu et al. [18].	Observational retrospective cohort study	2018	United States	11,410

the AXIS tool were judged to have overall adequate quality despite some weaknesses in missing data reporting and addressing non-responders. The results of the RoB assessment are summarized in Tables 6 and 7.

Meta-analysis findings

Pooled proportion of cardiovascular risk outcomes

A single-arm meta-analysis was conducted to estimate the pooled proportion of patients experiencing cardiovascular

Table 2. *Patient characteristics.*

Study ID	Age (Mean ± SD)	Age range	Comorbidities	Baseline CV risk	Dermatologic condition	Severity
Abuabara et al. [12].	42.2 ± 11.6	NR	PsA (42%), depression (15%), HTN (25%), DM (11%), DLP (33%), obesity (11%), smoking (12%)	HTN: 25%, DM: 11%, DLP: 33%, Obesity: 11%	Psoriasis	Moderate-to-severe
Dey et al. [13].	47.9 ± 12.6	NR	Excluded DM, CKD, HTN, BMI ≥35	Framingham score: 2.0 (0.6-5.7)	Psoriasis	Very severe
Bissonnette et al. [25].	56.1 ± 11.0	18-80 years	Atherosclerosis, HTN, DM, DLP, obesity	Chol: 4.60 ± 0.97, LDL: 2.74 ± 0.79, HDL: 1.08 ± 0.32, TG: 1.69 ± 0.80, hs-CRP: 4.22	Plaque psoriasis	Moderate-to-severe
Cho et al. [19].	46.0 ± 12.7	NR	DLP (51.1%), HTN (23.0%), DM (13.6%)	Higher DLP in TNF-α users	Psoriasis/PsA	Severe
Gelfand et al. [14].	47.4 ± 13.7	NR	CAD (5.5%), DM (5.5%), DLP (20.9%), HTN (29.7%), PsA (29.7%)	Elevated CV risk	Plaque psoriasis	Moderate-to-severe
Genre et al. [28].	37.4 ± 9.9	NR	Smoking (34.5%), obesity (24.1%), DLP (44.8%)	Smoking: 34.5%, Obesity: 24.1%	Psoriasis	Moderate-to-severe
Gulliver et al. [26].	51.4 ± 11.6	20-80 years	NR	NR	Plaque Psoriasis	Moderate-to-severe
Hagino et al. [29].	56.0 ± NR	43.5-75.5 years	Arthritis, DM (20%), HTN (43%), DLP (25%), hyperuricemia (25%), CVD (6%), smoking (98%)	Elevated CV risk	Psoriasis	Moderate-to-severe
Hjuler et al. [30].	49.2 ± 10.2	30-70 years	DM (7%), DLP (18%), HTN (25%), FHx CAD (39%)	Chol: 208.5; LDL: 127.4	Psoriasis	Moderate-to-severe
Hoffmann et al. [23].	47.3 ± 12.0	NR	CVD, depression, PsA (45%)	Elevated NLR	Psoriasis	Moderate-to-severe
Hong et al. [20].	46.3 ± 16.1	20-71+ years	HTN (23.6%), DM (15.6%), DLP (35.5%), ESRD (0.2%)	HTN: 24.6%-29.0%	Psoriasis	Moderate-to-severe
Kim et al. [21].	61.8 ± 12.8	≥20 years	DM (23.8%), HTN (45.5%), DLP (30.4%)	NR	Psoriasis	NR
Kridin et al. [15].	42.1 ± 24.2	NR	Smoking (7.3%), FHx CAD (2.6%), CKD (3.5%), Cancer (30%)	NR	Atopic dermatitis	Moderate-to-severe
Kridin et al. [24].	49.7 ± 23.6	NR	Smoking (6.7%), HTN (2.2%), DLP (3.6%), DM (2.4%)	NR	Atopic dermatitis	Moderate-to-severe
Lee et al. [16].	46.0 ± 12.6	NR	DM (12.9%), DLP (33.9%), HTN (31.3%), HF (1.2%), CAD (4.4%)	NR	Psoriasis	NR
Levesque et al. [27].	52.8 ± NR	≥20 years	DM, DLP, HTN (higher in psoriasis groups)	Higher in psoriasis versus controls	Psoriasis	Mild to severe
Shaaban et al. [7].	49.6 ± NR	NR	HTN, DM, DLP, TIA	Increased CV risk	Psoriasis	Moderate-to-severe
Smith et al. [22].	51.0 ± 16.9	26.5-55 years	Hyper-TG, DLP, DM	TG: 35.1%, LDL: 25%, low HDL: 50%, HbA1c: 52.6%	Psoriasis	Moderate-to-severe
von Stebut et al. [1].	44.2 ± 12.9	NR	PsA (25%), DM (8.3%), HTN (27.1%)	NR	Plaque psoriasis	Moderate-to-severe
Wu et al. [17].	52.8 ± NR	NR	DM (14.5%), HTN (21.0%), DLP (23.1%), CAD (4.0%), CKD (2.5%)	Increased CV risk	Psoriasis	Moderate-to-severe
Wu et al. [18].	49.3 ± 13.8	NR	HTN (29.3%), DLP (26.1%), DM (15.4%), Obesity (11.6%), Smoking (16.5%)	Higher CV risk in biologics users	Psoriasis	Moderate-to-severe

HTN = Hypertension, DM = Diabetes Mellitus, DLP = Dyslipidemia, TG = Triglycerides, LDL = Low-Density Lipoprotein, HDL = High-Density Lipoprotein, hs-CRP = High-Sensitivity C-Reactive Protein, PsA = Psoriatic Arthritis, CAD = Coronary Artery Disease, CKD = Chronic Kidney Disease, CV = Cardiovascular, CVD = Cardiovascular Disease, ESRD = End-Stage Renal Disease, HF = Heart Failure, TIA = Transient Ischemic Attack, NLR = Neutrophil-to-Lymphocyte Ratio, FHx = Family History, NR = Not Reported.

Table 3. *Biological treatment characteristics.*

Study ID	Type of biological treatment	Dosing regimen	Treatment duration	Administration route	Effectiveness of treatment	Concomitant medications
Abuabara et al. [12].	TNFi: Adalimumab, Infliximab, Etanercept	NR	Ranged from 243 to 591 days	NR	NR	NR
Dey et al. [13].	anti-TNF, anti-IL12/23, anti-IL17	NR	1 year	NR	Baseline 5.6 (2.9-9.3), after 1 year 6.6 (3.1-12.4), *p*-value 0.10	NR
Bissonnette et al. [25].	TNFi: Adalimumab	Every other week for 4 months; loading dose: 80 mg, maintenance dose: 40 mg	4 months	Subcutaneous injection	75% improvement in PASI (PASI 75) at week 16	NR
Cho et al. [19].	TNF-α inhibitors (adalimumab, etanercept, infliximab), IL-12/23 inhibitors (ustekinumab)	NR	Average follow-up: 2.9 ± 1.2 years	NR	TNF-α inhibitor users had higher all-cause mortality, no significant difference in MACE risk	NR
Gelfand et al. [14].	IL-17 inhibitors (Secukinumab)	300 mg weekly for 5 weeks, then every 4 weeks for 52 weeks	12-week double-blind + 40-week open-label	Subcutaneous	PASI 90 response of 74% and 78% at Week 12	NR
Genre et al. [28].	TNF inhibitors: Adalimumab	80 mg at week 0, then 40 mg every other week	6 months	Subcutaneous injections	Baseline PASI: 18.55 ± 7.63; at 6 months: 1.354 ± 2.129	NR
Gulliver et al. [26].	Anti-TNF-α (adalimumab, etanercept, infliximab), Anti-IL-12/23 (ustekinumab)	NR	49 months	NR	NR	NR
Hagino et al. [29].	TNFi, IL-17 inhibitors, IL-23 inhibitors	NR	Over 52 weeks	NR	Significant PASI 75/90/100 achievement with IL-17 and IL-23 inhibitors	NR
Hjuler et al. [30].	TNFi: Adalimumab, Etanercept, Infliximab, IL-23 Inhibitors: Ustekinumab	NR	1 year	NR	Mean PASI reduction: 87.6%	NR
Hoffmann et al. [23].	TNF-alpha inhibitors (adalimumab, etanercept), IL-12/23 antagonist (ustekinumab)	NR	Mean 21 ± 19 months	NR	Median PASI: TNF-α antagonists = 2.9; IL-12/23 antagonists = 3.00	NR
Hong et al. [20].	TNFi, IL-17 inhibitors, IL-23 inhibitors, JAK inhibitors	NR	NR	NR	NR	NR
Kim et al. [21].	TNF-α inhibitor, anti-IL-12/23p40, IL-17A antagonist, or IL-23 antagonist	NR	NR	Injection	NR	NR
Kridin et al. [15].	TNFi, IL-17 inhibitors (IL17i), IL-23 inhibitors (IL23i), JAK inhibitors	Minimum of 2 years of continuous treatment	Minimum 2 years	NR	NR	Classic antipsoriatics excluded
Kridin et al. [24].	Dupilumab	NR	Initial 3 years	NR	Dupilumab reduced risks of HTN (HR = 0.67), T2DM (HR = 0.53), and obesity (HR = 0.70) versus methotrexate/ cyclosporine	NR
Lee et al. [16].	IL-23 Inhibitors: Ustekinumab	NR	NR	NR	NR	NR
Levesque et al. [27].	TNF inhibitors, IL-17 inhibitors, IL-23 inhibitors, JAK inhibitors	NR	NR	NR	Improvement in PASI scores	NR
Shaaban et al. [7].	TNF inhibitors (adalimumab, etanercept, infliximab)	At least three consecutive months	Median follow-up: 3.9 years	Subcutaneous/intravenous	Responders showed reduced MI rates	Statins, beta-blockers, antihypertensives, lipid-lowering drugs

Study ID	Type of biological treatment	Dosing regimen	Treatment duration	Administration route	Effectiveness of treatment	Concomitant medications
Smith et al. [22].	TNF Inhibitors: Adalimumab, Infliximab; IL-17 Inhibitors: Secukinumab, Ixekizumab; IL-23 Inhibitors: Risankizumab, Guselkumab	NR	Continuous treatment for 1 year	NR	PASI decreased from 13.88 to 0.75	Cholesterol-lowering and diabetic medications
von Stebut et al. [1].	Secukinumab (IL-17A inhibitor)	Weekly doses for 5 weeks, then every 4 weeks until Week 48	52 weeks	Subcutaneous injection	≥75% PASI reduction in 81.3% of patients at Week 12	NR
Wu et al. [17].	TNFi (etanercept, infliximab, adalimumab)	Median duration: 685 days	Median follow-up: 4.3 years	NR	Lower MI hazard (adjusted HR = 0.50; 95% CI: 0.32-0.79)	Statins (28.6%), β-blockers (46.5%), methotrexate (20.6%)
Wu et al. [18].	TNFi and phototherapy (UVB/PUVA)	NR	Median: 15.4 months (TNFi), 12.6 months (phototherapy)	NR	TNFi cohort: lower CV event risk (adjusted HR = 0.77; 95% CI: 0.60-0.99)	Adjusted for prior Ps-related medications, statins, antihypertensives, smoking deterrents

TNF = Tumor Necrosis Factor, TNFi = Tumor Necrosis Factor Inhibitor, IL-12/23 = Interleukin-12 and Interleukin-23, IL-17 = Interleukin-17, IL-23 = Interleukin-23, IL-17A = Interleukin-17A, IL23i = IL-23 Inhibitor, IL17i = IL-17 Inhibitor, JAK = Janus Kinase, PASI = Psoriasis Area and Severity Index, HR = Hazard Ratio, HTN = Hypertension, T2DM = Type 2 Diabetes Mellitus, MI = Myocardial Infarction, UVB = Ultraviolet B, PUVA = Psoralen + Ultraviolet A, CI = Confidence Interval, CV = Cardiovascular, NR = Not Reported.

outcomes following biologic therapy. Under the random-effects model, the pooled proportion was 0.0782 (7.82%) with a 95% confidence interval of 0.0531 to 0.1137 (z = 5.06, $p < 0.0001$). This estimate reflects the cumulative incidence of cardiovascular events or risk modulation in dermatologic patients receiving biologics (Figure 2 illustrates the forest plot of the pooled proportions).

By contrast, the fixed-effect model yielded a pooled proportion of 0.0273 (2.73%), highlighting the substantial between-study variability. This variation aligns with observed differences in biologic classes used, underlying dermatologic conditions, and cardiovascular risk profiles of patients.

Heterogeneity and subgroup considerations

The analysis demonstrated extreme heterogeneity with a Tau² of 0.8141, an I^2 of 99.5% (95% CI: 99.4%-99.5%), and a Q-value of 3,871.85 (df = 20, $p < 0.0001$). Such heterogeneity suggests true differences in treatment effects rather than random variation. Potential sources include variations in study design, patient baseline risk (e.g., Framingham scores), and biologic class. Subgroup analyses (e.g., stratification by biologic type, psoriasis severity, or cardiovascular risk scores) were not performed in this analysis due to data limitations and are acknowledged as a limitation. Future analyses should incorporate these to improve interpretability and clinical relevance.

Correlation-based association analysis

A supplementary analysis using Fisher's r-to-z transformation assessed the overall association between biologic therapies and cardiovascular risk modulation. The pooled correlation coefficient was 0.2051 (95% CI: 0.0440 to 0.3661), with a z-score of 2.4951 ($p = 0.0126$), suggesting a modest but statistically significant positive association (Figure 3).

Outlier and sensitivity analysis

Outlier analysis identified Hoffmann et al. [23] and Smith et al. [22] as potential outliers based on studentized residuals > ±3.0381. Cook's distance also flagged Hjuler et al. [30], Hoffmann et al. [23], and Smith et al. [22] as potentially influential studies. Sensitivity analyses excluding these studies were conducted, and results remained consistent with the main findings, supporting the robustness of the pooled estimates.

Publication bias assessment

Visual and statistical evaluation revealed evidence of potential publication bias. Egger's regression intercept was 3.664 ($p < 0.001$), and the Begg and Mazumdar rank correlation was $r = 0.314$ ($p = 0.049$). The Fail-safe N was 1,913 ($p < 0.001$), with zero studies imputed by the trim-and-fill method.

The large fail-safe N suggests statistical robustness, but the observed funnel plot asymmetry implies that negative or null studies may be underrepresented, potentially inflating the apparent benefit of biologics (Figure 4 presents the funnel plot displaying this publication bias).

Table 4. *CVD outcomes.*

Study ID	Incident cardiovascular events	Cardiovascular risk factors	Risk assessment tools used	Time frame for outcomes
Abuabara et al. [12].	NR	No significant reductions in cardiometabolic parameters	NR	12 months
Dey et al. [13].	NR	No significant changes in known cardiovascular risk factors	Framingham risk score	1 year
Bissonnette et al. [25].	1 MI	No statistically significant changes among groups for lipid values	NR	4 months
Cho et al. [19].	MACEs included AMI, stroke, heart failure, coronary revascularization, and cardiovascular death	Dyslipidemia, HTN, DM, smoking, and obesity were noted	NR	Up to 5 years (2016-2020)
Gelfand et al. [14].	The study discusses cardiovascular risk but does not specify incident events like heart attacks or strokes	Small increases in total cholesterol, LDL, and LDL particles at Week 12; no changes in inflammation markers	NR	52 weeks
Genre et al. [28].	NR	Increase in cholesterol levels	NR	6 months
Gulliver et al. [26].	MI: 1 in the biologic group, 18 in the control group	NR	NR	49 months
Hagino et al. [29].	NR	Increase in HDL-C with IFX treatment at week 12 and a decrease in HDL-C with IXE treatment at week 52	NR	52 weeks
Hjuler et al. [30].	NR	No significant changes	NR	13 months
Hoffmann et al. [23].	NR	High NLR	NR	3 years
Hong et al. [20].	MACE incidence per 1,000 PYs: Biologic cohort (3.5) versus controls (≥14.5)	NR	NR	Mean 1.4 ± 0.64 years
Kim et al. [21].	NR	NR	NR	1 year
Kridin et al. [15].	Heart attack, stroke, heart failure, cardiac arrest, deep vein thrombosis, pulmonary embolism	HTN, smoking status, obesity (BMI), DM, family history of IHD	Propensity-score matching	Short-term (1 month) to long-term (2-5 years)
Kridin et al. [24].	MACEs (adjusted HR: 0.77; $p = 0.046$)	Not specified	NR	6-24 months
Lee et al. [16].	Stroke incidence: Ustekinumab (7.2/1,000 PYs) versus TNFi (6.3/1,000 PYs); MACE: Ustekinumab (6.2) versus TNFi (6.1)	NR	NR	6 years
Levesque et al. [27].	Heart attack (MI), stroke, heart failure	Changes in cholesterol levels, blood pressure, smoking status, obesity	Framingham/ASCVD risk score (assumed)	Short-term (6 months) to long-term (>3 years)
Shaaban et al. [7].	MI rates: TNF inhibitor (1.79%), MTX (3.03%), topical (3.03%)	Smoking (20.98% TNF cohort), obesity (mean BMI 28.8)	NR	Median 3.9 years
Smith et al. [22].	NR	There were no significant reductions in cardiometabolic parameters	NR	12 months
von Stebut et al. [1].	One case of cerebral infarction after surgery	Cholesterol levels: No significant changes; smoking (~40%); obesity (mean BMI 27.8-30.1 kg/m²)	Framingham risk score	52 weeks
Wu et al. [17].	MI incidence: TNF inhibitors (3.05/1,000 PYs) versus topical (6.73/1,000 PYs)	Smoking (12.8%), obesity (44.2%)	NR	Median 4.3 years
Wu et al. [18].	MACE: TNFi cohort (0.4%-1.4%) versus phototherapy (0.7%-2.7%)	NR	NR	Short-term (6 months) to medium-term (1-3 years)

MI = Myocardial Infarction, *MACE* = Major Adverse Cardiovascular Events, *AMI* = Acute Myocardial Infarction, *CV* = Cardiovascular, *HR* = Hazard Ratio, HTN = Hypertension, DM = Diabetes Mellitus, IHD = Ischemic Heart Diseases, *PYs* = Person-Years, *HDL-C* = High-Density Lipoprotein Cholesterol, *IFX* = Infliximab, *IXE* = Ixekizumab, *NLR* = Neutrophil-to-Lymphocyte Ratio, *BMI* = Body Mass Index, *ASCVD* = Atherosclerotic Cardiovascular Disease, *MTX* = Methotrexate, *TNFi* = Tumor Necrosis Factor Inhibitor, NR = Not Reported.

Narrative summary of study findings

CVD outcomes

Several studies evaluated the incidence of cardiovascular events, such as MI, stroke, and MACE [20,23]. For example, Hong et al. [20] showed a reduction in MACE incidence with a mean follow-up of 1.4 years. In contrast, Hoffmann et al. [23], Joseph et al. [2], Abuabara et al. [12], and Smith et al. [22] did not report any cardiovascular outcomes. Across the studies, while many suggested

Table 5. *Adverse events, follow up, and outcomes.*

Study ID	Incidence of adverse events (cardiovascular events)	Severity of adverse events	Withdrawal due to side effects	Follow-up duration	Loss to follow-up	Long-term effects
Abuabara et al. [12].	NR	NR	NR	12 months	NR	No significant cardiometabolic changes
Dey et al. [13].	NR	NR	NR	1 year	38 lost; 45 with no follow-up	NR
Bissonnette et al. [25].	NR	NR	NR	4 months	0	Adalimumab may reduce vascular inflammation
Cho et al. [19].	No significant difference in MACEs between groups; TNF-α inhibitors had higher all-cause mortality	NR	NR	Up to 5 years (2016-2020)	NR	Higher all-cause mortality in TNFi users
Gelfand et al. [14].	26 AEs (56.5%) in secukinumab group, incl. 2 serious; 16 AEs (35.6%) in placebo group	Rib fracture, upper limb fracture, aortic stenosis (serious AEs)	2 (4.3%) in secukinumab; 3 (6.7%) in placebo	52 weeks	8 (8.8%) discontinued	Secukinumab had neutral effect on aortic inflammation
Genre et al. [28].	NR	NR	NR	6 months	NR	Adalimumab reduced sE-selectin levels
Gulliver et al. [26].	MI: 1 in biologic group versus 18 in control group	NR	NR	49 months	NR	Reduced MI incidence in biologic group
Hagino et al. [29].	NR	NR	NR	52 weeks	NR	TNFi may improve hyperuricemia and dyslipidemia
Hjuler et al. [30].	NR	NR	NR	13 months	2	Medium-term effect on coronary artery disease progression
Hoffmann et al. [23].	NR	NR	NR	3 years	NR	NLR reduction
Hong et al. [20].	MACE: 3.5/1,000 PYs (biologic) versus ≥14.5/1,000 PYs (controls)	Severe	NR	Mean 1.4 ± 0.64 years	NR	MACE risk reduction at 3 years with biologics (HR = 0.46)
Kim et al. [21].	NR	NR	NR	1 year	NR	NR
Kridin et al. [15].	MACE reported; arrhythmias not mentioned	NR	NR	1 month-5 years	NR	Reduced mortality and CV risk with biologics
Kridin et al. [24].	Atrial Fibrillation: Ustekinumab 5.0/1,000 PYs versus TNFi 4.7/1,000 PYs	Severe	NR	6 years	NR	No difference in AF or MACE between ustekinumab and TNFi
Lee et al. [16].	Cardiovascular events noted (e.g., arrhythmias)	Ranges from mild to severe (not explicitly stated)	NR	6 months->3 years	NR	Higher MI risk in psoriatic patients
Levesque et al. [27].	MI: TNFi 1.79% versus MTX 3.03%	NR	NR	Median 3.9 years	NR	TNF responders had lower MI risk
Shaaban et al. [7].	NR	NR	NR	12 months	NR	No significant cardiometabolic change
Smith et al. [22].	1 case cerebral infarction (not related to secukinumab)	NR	11 discontinued, 6 due to AEs	52 weeks	11 discontinued	Improved endothelial function (FMD +2.1%); no change in arterial stiffness
von Stebut et al. [1].	MI: TNFi 3.05/1,000 PYs versus topical 6.73/1,000 PYs	NR	20.6% disenrolled; 4.5% died	Median 4.3 years	20.6% disenrolled; 4.5% died	TNFi reduced MI risk by 50% compared to topicals
Wu et al. [17].	MACE: TNFi 0.4%-1.4% versus phototherapy 0.7%-2.7%	NR	NR	6 months-3 years	NR	NR
Wu et al. [18].	TNFi group had significantly lower incidence of CV events versus phototherapy (HR = 0.77; 95% CI, 0.60-0.99)	Not explicitly graded, CV events only	NR	Mean 3.3 years	NR	TNFi use associated with lower risk of cardiovascular events compared to phototherapy (HR = 0.77)

AE = Adverse Event, AEs = Adverse Events (plural), AF = Atrial Fibrillation, CV = Cardiovascular, FMD = Flow-Mediated Dilation, HUA = Hyperuricemia, HR = Hazard Ratio, LRNC = Lipid-Rich Necrotic Core, MACE = Major Adverse Cardiovascular Event, MI = Myocardial Infarction, MTX = Methotrexate, NLR = Neutrophil-to-Lymphocyte Ratio, PYs = Person-Years, TNFi = Tumor Necrosis Factor alpha inhibitors (TNF-α inhibitors), NR = Not Reported.

Table 6. *Cochrane RoB2 assessment for RCTs.*

Study ID	Randomization process	Deviations from intended interventions	Missing outcome data	Measurement of the outcome	Selection of the reported result	Overall RoB s
Bissonnette et al. [25]	Low	Low	Low	Low	Low	Low
Gelfand et al. [14]	Low	Some concerns	Low	Low	Low	Some concerns
von Stebut et al. [1]	Low	Low	Low	Low	Low	Low

Table 7. *ROBINS-I assessment for non-randomized studies (18 studies).*

Study ID	Confounding	Participant selection	Classification of interventions	Deviations from intended interventions	Missing data	Outcome measurement	Selection of reported result	Overall bias
Abuabara et al. [12]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Dey et al. [13]	Low	Low	Low	Low	Low	Low	Low	Low
Cho et al. [19]	Moderate	Low	Low	Low	Low	Moderate	Moderate	Moderate
Genre et al. [28]	Moderate	Low	Low	Low	Low	Moderate	Moderate	Moderate
Gulliver et al. [26]	Moderate	Low	Low	Low	Low	Low	Low	Moderate
Hagino et al. [29]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Hjuler et al. [30]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Hoffmann et al. [23]	Moderate	Low	Low	Low	Low	Moderate	Moderate	Moderate
Hong et al. [20]	Moderate	Low	Low	Low	Low	Low	Low	Moderate
Kim et al. [21]	Serious	Moderate	Low	Low	Low	Serious	Low	Serious
Kridin et al. [15]	Moderate	Low	Low	Low	Low	Moderate	Moderate	Moderate
Kridin et al. [24]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Lee et al. [16]	Moderate	Low	Low	Low	Low	Moderate	Moderate	Moderate
Levesque et al. [27]	Moderate	Low	Low	Low	Low	Low	Low	Moderate
Shaaban et al. [7]	Moderate	Low	Low	Low	Low	Low	Low	Moderate
Smith et al. [22]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Wu et al. [17]	Moderate	Low	Low	Low	Low	Moderate	Low	Moderate
Wu et al. [18]	Moderate	Low	Low	Low	Low	Low	Low	Moderate

Note: “Moderate” indicates acceptable risk, “Serious” flags high bias risk.

a favorable cardiovascular trend, others did not report significant effects.

Cardiovascular risk factors

Several studies assessed changes in lipid profiles, inflammatory markers, and other risk indicators [4,23]. Hoffmann et al. [23] demonstrated an elevation in NLR post-therapy, while Elnabawi et al. [4] reported improvements in total cholesterol and lipid markers. Studies such as Joseph et al. [2] and Smith et al. [22] did not observe significant risk factor modulation.

Risk assessment tools

Only a subset of studies used formal cardiovascular risk calculators. The Framingham Risk Score was applied in studies including Elnabawi et al. [4], von Stebut et al. [1], Dey et al. [13], and Levesque et al. [27].

Adverse events

Adverse events varied widely; for example, Hong et al. [20] and Lee et al. [16] reported cardiovascular complications, including atrial fibrillation. Kridin et al. [15] and Levesque et al. [27] noted MACE events, while Gelfand et al. [14] observed fractures linked to secukinumab. Details are available in Table 5.

Long-term effects

Long-term impacts were inconsistently reported. Hoffmann et al. [23] observed reduced NLR over time, while Lee et al. [16] reported no long-term differences in

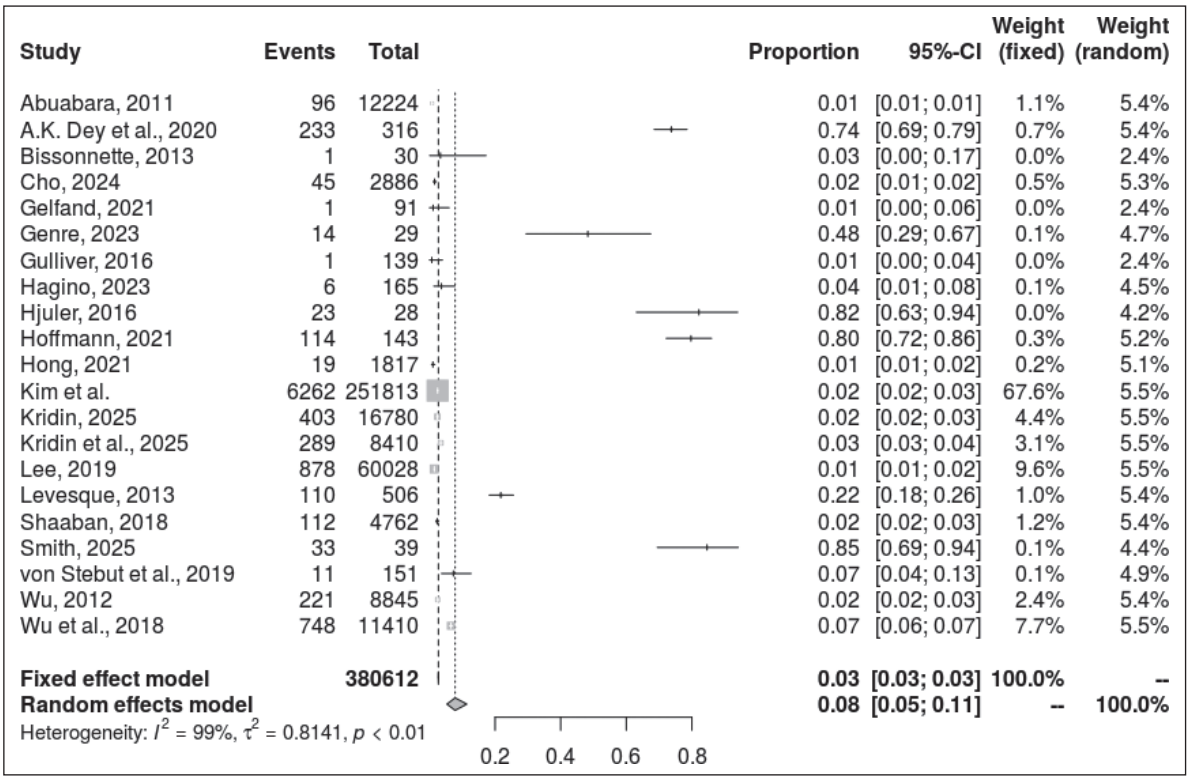

Figure 2. *Forest plot of pooled proportions of cardiovascular outcomes in biologic-treated patients across 21 studies.*

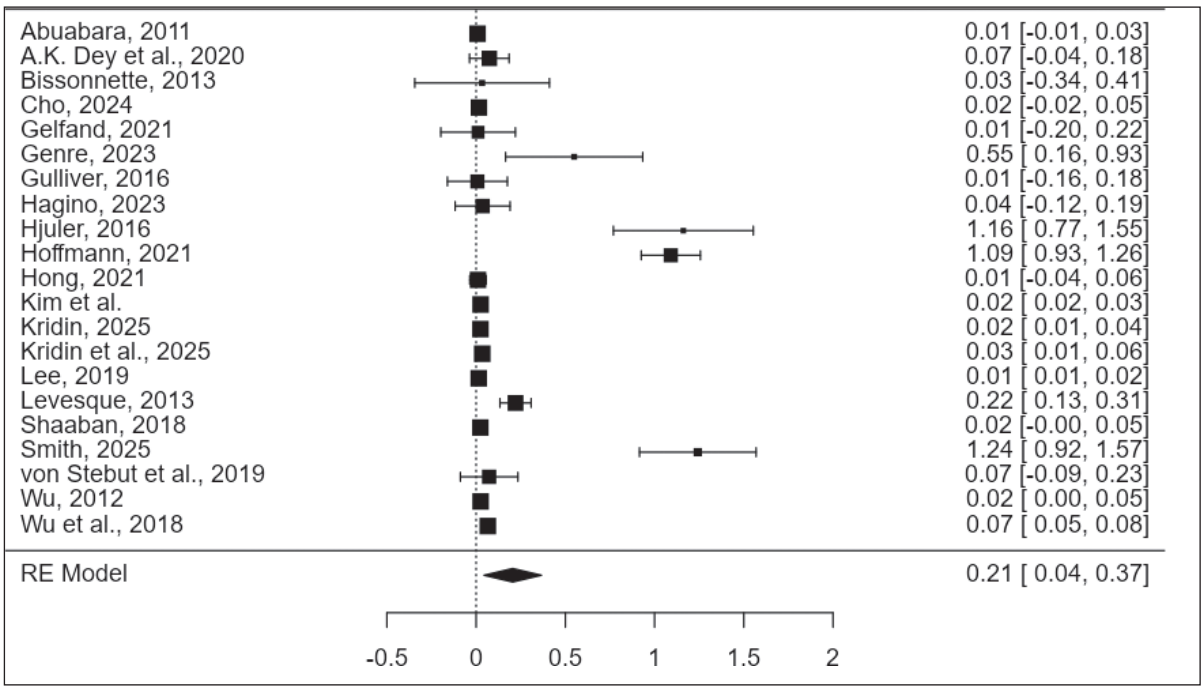

Figure 3. *Forest plot of Fisher r-to-z transformed correlation coefficients across the 21 studies. Importantly, the 95% prediction interval ranged from -0.5302 to 0.9403, indicating that in certain populations, biologic therapy may reduce, increase, or have no effect on cardiovascular risk. This further reinforces the need for stratified analyses in future work.*

atrial fibrillation or stroke. Elnabawi et al. [4] suggested biologics may lower long-term cardiovascular risk. Heterogeneity of long-term outcomes is summarized in Table 5.

Clinical interpretation

The meta-analysis suggests that biologic therapies may be associated with a modest reduction in cardiovascular risk among dermatologic patients, reflected by a pooled event

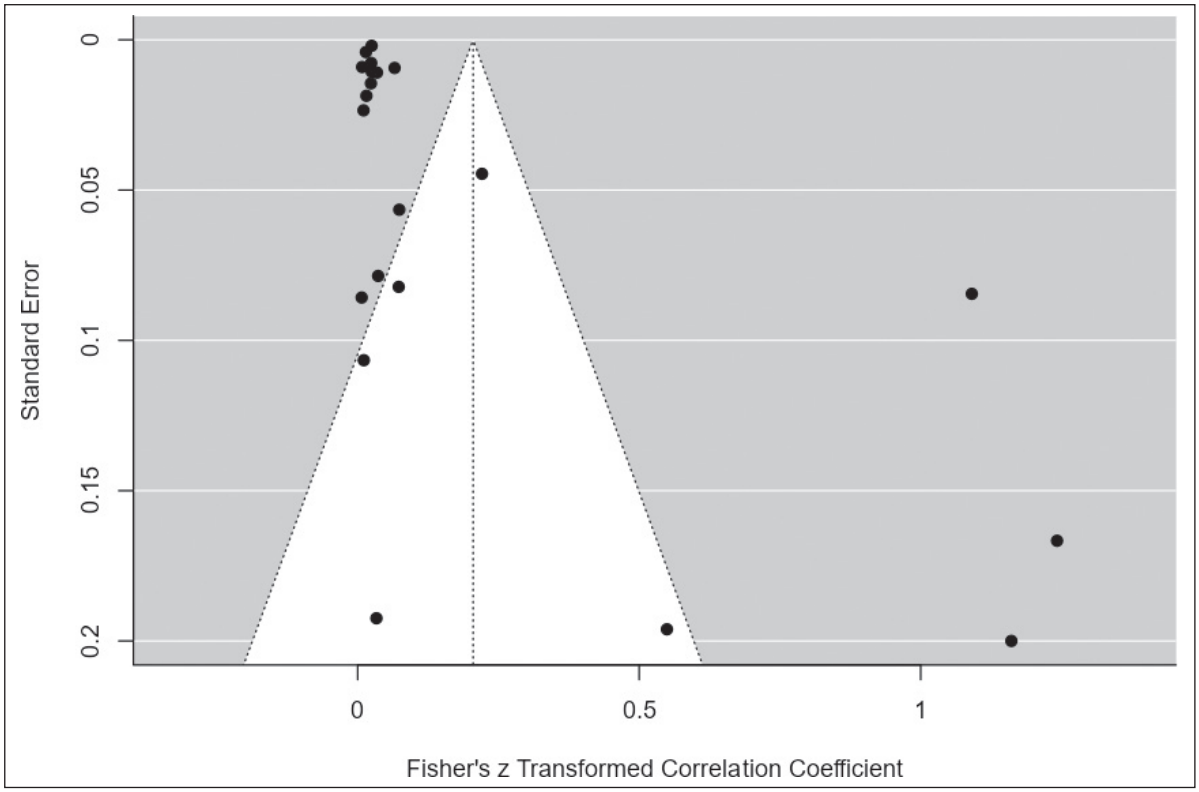

Figure 4. *Funnel plot displaying asymmetry suggestive of publication bias among included studies.*

rate of 7.82% and supported by a small but significant average correlation. However, the extreme heterogeneity (I^2 = 99.5%), influential outliers, and risk of publication bias limit definitive conclusions.

The diversity in reported outcomes—ranging from lipid profile improvements and blood pressure changes to Framingham scores and hard cardiovascular events (e.g., MACE)—highlights the need for standardization in outcome definitions and assessment tools. Future research should prioritize subgroup meta-analyses, stratified by biologic class and baseline cardiovascular risk, and leverage prospective cohort data with uniform endpoints to clarify these relationships and guide clinical practice.

Discussion

This systematic review and single-arm meta-analysis assessed the relation between biologic therapy for dermatologic diseases and CVD outcomes. The key findings of our current study demonstrate that the pooled proportion of patients experiencing cardiovascular outcomes following biologic therapy was 7.82% (95% CI: 5.31%-11.37%), and there was a modest but statistically significant positive correlation (r = 0.2051, p = 0.0126) between biologic therapies and cardiovascular risk modulation. Our results support the concept that these therapies, especially TNF-α, IL-17, and IL-23 inhibitors, may protect against cardiovascular damage by repression of systemic inflammation, a key factor in endothelial dysfunction, atherosclerosis, and raised risk of CVD in patients with chronic inflammatory skin conditions such as psoriasis and hidradenitis suppurativa.

Several studies included in this review indicated that the incidences of MACE, MI, and stroke were lower in patients treated with biologics compared to patients treated with phototherapy or conventional systemic agents [14,19,25]. For example, MACE incidence with biologic treatments is reported to be 3.5 per 1,000 person-years compared to 28.4 per 1,000 PY with cyclosporine and 12.1 per 1,000 PY with methotrexate, respectively. This aligns with previous observational data on TNF-α blockers for chronic plaque psoriasis, which suggests diminished CVD risk [17,20].

The beneficial impact of biologics appears multifactorial. Inflammation plays a central role in the pathophysiology of both psoriasis and atherosclerosis. Inflammatory cytokines such as TNF-α and IL-17 contribute to endothelial dysfunction, insulin resistance, and plaque instability [31]. Therefore, inhibiting these pathways may translate into vascular benefit. This mechanistic hypothesis is supported by imaging studies showing regression in coronary plaque burden and reduced perivascular inflammation in patients treated with biologics [18].

Our review included longitudinal studies showing that sustained biologic treatment leads to improvements in surrogate cardiovascular markers such as carotid intima-media thickness, arterial stiffness, and the NLR [20,30]. For example, one study observed significant reductions in non-calcified coronary plaque after 1 year of biologic therapy [23]. Similarly, patients treated with IL-17 inhibitors demonstrated improvements in endothelial function and reductions in vascular inflammation [45].

Among biologic classes, TNF-α inhibitors have the most evidence supporting cardiovascular benefit [12,16]. IL-17 and IL-23 inhibitors such as secukinumab and ustekinumab also demonstrated potential in reducing lipid abnormalities and inflammatory markers, although not all studies found statistically significant reductions in clinical cardiovascular events [14,27]. Some studies noted that while TNF-α inhibitors reduced MI risk compared to phototherapy, the differential effect across biologic classes was less pronounced, highlighting the need for further head-to-head trials [7].

While most of the studies included in the review considered psoriasis, there is growing yet limited evidence that biologics might also confer cardiovascular risk reduction in hidradenitis suppurativa and atopic dermatitis patients [7,22]. Dupilumab has, for instance, been shown to have favorable cardiovascular outcomes in atopic dermatitis compared to cyclosporine and methotrexate by reducing the risks of hypertension and type 2 diabetes [22].

Despite encouraging trends, cardiovascular improvements have not been consistently shown across studies. Short-term trials or trials with smaller sample sizes were unable to demonstrate changes in lipid profile or blood pressure [22]. Similarly, while some inflammatory markers like C-reactive protein decreased, other studies failed to show a reduction in cardiovascular event incidence [31]. This demonstrates the complexity of translating surrogate markers into clinical outcomes and emphasizes the need for long-term follow-up.

Safety outcomes have been generally positive. Only a handful of studies reported serious cardiovascular adverse events potentially related to biologics. Events such as atrial fibrillation or ischemic complications were rare, largely observed in patients with preexisting cardiovascular risk factors. While there was no increased risk for all-cause mortality in patients undergoing biologic treatment, this safety profile agrees with previous registry data and real-world studies [16].

This study has several limitations, including the predominance of observational data. Even though propensity-score matched analyses were performed in some studies with adjustments for comorbidities, confounding may still be present [13]. Only a handful of RCTs with endpoints related to cardiovascular outcomes have been identified. Additionally, direct comparisons and causal inferences are hindered by heterogeneity in studies with respect to follow-up duration, biologic agent, dosing, and definitions of outcomes. Differences in reporting methodologies, including the inconsistent use of cardiovascular risk calculators (e.g., Framingham, ASCVD), further complicate the synthesis. A second limitation is the underrepresentation of non-psoriatic populations; evidence mostly favors moderate-to-severe plaque psoriasis, while little supports extrapolation into atopic dermatitis, alopecia areata, and hidradenitis suppurativa. Furthermore, newer agents such as JAK inhibitors and IL-36 inhibitors are seldom studied concerning cardiovascular outcomes, representing a gap in the literature. However, this study also has notable strengths, including a comprehensive search strategy and robust methodology that synthesizes available data to provide a clearer picture of the cardiovascular safety profile of dermatologic biologics.

Future research should focus on large, multicenter randomized trials with clearly defined cardiovascular endpoints. Integrating biomarkers, imaging modalities such as coronary computed tomography angiography, and long-term follow-up data will be critical to confirm these findings. Stratification by disease severity, sex, ethnicity, and baseline cardiovascular risk could provide valuable insights into which patients benefit most from biologic therapy.

Clinically, our findings advocate for a multidisciplinary approach. Dermatologists, cardiologists, and primary care physicians should collaborate to assess cardiovascular risk in patients with chronic inflammatory skin conditions. When appropriate, biologic therapies should be considered not only for skin clearance but also for their potential to reduce systemic inflammation and long-term cardiovascular burden.

Conclusion

Biologic therapies used in dermatology, particularly in the management of moderate-to-severe psoriasis, appear to be associated with a reduction in cardiovascular risk markers and events, including MACE, MI, and stroke. These benefits are likely mediated through the suppression of chronic systemic inflammation, a key driver of both dermatologic and cardiovascular pathology.

While these findings support the dual therapeutic role of biologics in improving both skin and cardiovascular health, further high-quality, long-term RCTs are warranted to confirm causality and establish agent-specific effects. Future research should also address diverse patient populations, including those with high baseline cardiovascular risk and non-psoriatic inflammatory skin diseases. Clinicians should consider cardiovascular comorbidity profiles when selecting systemic treatments for dermatologic patients, recognizing biologic agents as potentially beneficial beyond skin clearance alone.

List of Abbreviations

AE	Adverse event
AEs	Adverse events
AF	Atrial fibrillation
AMI	Acute myocardial infarction
ASCVD	Atherosclerotic cardiovascular disease
AXIS	Appraisal tool for cross-sectional studies
BMI	Body mass index
CAD	Coronary artery disease
CI	Confidence interval
CKD	Chronic kidney disease
CV	Cardiovascular
CVD	Cardiovascular disease
DLP	Dyslipidemia
DM	Diabetes mellitus
ESRD	End-stage renal disease
FHx	Family history
FMD	Flow-mediated dilation
HDL	High-density lipoprotein
HDL-C	High-density lipoprotein cholesterol

HF	Heart failure
HR	Hazard ratio
hs-CRP	High-sensitivity C-reactive protein
HTN	Hypertension
IFX	Infliximab
IHD	Ischemic heart disease
IL	Interleukin
IL-12/23	Interleukin-12 and interleukin-23
IL-17	Interleukin-17
IL-17A	Interleukin-17A
IL-17i	Interleukin-17 inhibitor
IL-23	Interleukin-23
IL-23i	Interleukin-23 inhibitor
IXE	Ixekizumab
JAK	Janus kinase
LDL	Low-density lipoprotein
LRNC	Lipid-rich necrotic core
MACE	Major adverse cardiovascular events
MI	Myocardial infarction
MINORS	Methodological index for non-randomized studies
MTX	Methotrexate
NLR	Neutrophil-to-lymphocyte ratio
NR	Not reported
PASI	Psoriasis area and severity index
PRISMA	Preferred Reporting Items for Systematic Reviews and Meta-Analyses
PUVA	Psoralen plus ultraviolet A
PYs	Person-years
RCT	Randomized controlled trial
RoB2	Revised cochrane risk of bias tool
SD	Standard deviation
SE-selectin	Soluble E-selectin
T2DM	Type 2 diabetes mellitus
TG	Triglycerides
TIA	Transient ischemic attack
TNF	Tumor necrosis factor
TNFi	Tumor necrosis factor inhibitor
TNF-α	Tumor necrosis factor alpha
UVB	Ultraviolet B

Conflict of interests

The authors declare that there is no conflict of interest regarding the publication of this article.

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Supplementary content (If any) is available online.

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