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xinying xiao

YYBM1X2Y@OUTLOOK.COM

Puning Public Health Center

Sibao Chen

Puning People's Hospital

Zhouyang Zhu

The Second Affiliated Hospital of Jiaxing University

Systematic Review

Keywords: Psoriasis, Cardiovascular Diseases, Drug Interactions, Systematic Review, Drug Repositioning, Risk Assessment

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Bidirectional Pharmacological Interplay between Psoriasis and Cardiovascular Disease: A Systematic Review

Title Page

Authors: Xinying Xiao1†*, Sibao Chen2†, Zhouyang Zhu3*

Affiliations:

1 Department of Dermatology, Puning Public Health Center, Jieyang, Guangdong, China; 2 Department of Cardiology, Puning People's Hospital, Jieyang, Guangdong, China; 3 Department of Cardiology, The Second Affiliated Hospital of Jiaxing University, Jiaxing, Zhejiang, China

† These authors contributed equally to this work and share first authorship. * Corresponding authors.

Corresponding authors: Xinying Xiao, Department of Dermatology, Puning Public Health Center, Jieyang, Guangdong, China. Email: YYBM1X2Y@OUTLOOK.COM; Zhouyang Zhu, Department of Cardiology, The Second Affiliated Hospital of Jiaxing University, Jiaxing, Zhejiang, China. Email: Zyz387@outlook.com

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Abstract

Psoriasis and cardiovascular disease share inflammatory pathways, yet the bidirectional pharmacological interactions between their respective therapies remain incompletely characterised. This systematic review, conducted in accordance with PRISMA 2020 (OSF: osf.io/25uf9), synthesised evidence from 556 studies (5,378 records screened) on the effects of cardiovascular drugs on psoriasis and psoriasis therapies on cardiovascular outcomes. Dual independent screening, formal risk-of-bias assessment (RoB 2/ROBINS-I/AMSTAR 2), and GRADE certainty grading were employed. Observational studies linked beta-blockers (ROR 8.95), ACE inhibitors (OR 1.67), and thiazides (OR 1.23) to psoriasis onset; Mendelian randomisation estimates were near-null, compatible with confounding by indication. GLP-1 receptor agonists, statins, and PDE5 inhibitors carried anti-psoriatic signals. TNF-alpha inhibitors and methotrexate were associated with fewer major adverse cardiovascular events; IL-17 and IL-23 inhibitors showed neutral cardiovascular safety profiles. The synthesised evidence informed a risk-stratified treatment algorithm. GLP-1 receptor agonists, low-dose colchicine, and PDE5 inhibitors emerge as dual-target repurposing candidates meriting prospective evaluation.

Introduction

Psoriasis affects 2–3% of the global population and carries an approximately 50% higher relative risk of major adverse cardiovascular events (MACE) compared with the general population, a differential

most pronounced in severe disease [1–3]. Earlier reviews of the psoriasis-cardiovascular interface [4,5] predate several therapeutic developments that have since altered the landscape: The CANTOS trial demonstrated that anti-inflammatory therapy can reduce cardiovascular events independently of lipid lowering [31]; the SELECT trial showed that GLP-1 receptor agonism reduces MACE in obesity without diabetes [68]; and IL-17 and IL-23 inhibitors have expanded the psoriasis armamentarium. Prior systematic reviews examined either cardiovascular risk in psoriasis or psoriatic adverse effects of cardiovascular drugs, no synthesis has integrated both directions within a common evidence framework.

EULAR and AAD/NPF guidelines now recommend cardiovascular risk assessment for patients with psoriasis [6,7], and the 2021 ESC prevention guidelines recognise chronic inflammatory disorders as cardiovascular risk modifiers [8]. Conventional calculators — the Framingham Risk Score, SCORE2, and the ASCVD Risk Estimator — systematically underestimate risk because they omit inflammatory burden [9,53,54]. Despite converging guideline recommendations and a shared inflammatory substrate, no integrated framework exists to guide simultaneous cardiovascular and dermatological drug selection.

This systematic review synthesises the bidirectional pharmacology of the skin-heart axis: the effects of cardiovascular drugs on psoriasis, and the effects of psoriasis therapies on cardiovascular outcomes. From this synthesis we derived a risk-stratified treatment-selection algorithm and identify dual-target drug repurposing candidates graded by evidence strength [10,11].

Methods

Protocol and Registration

The review followed the PRISMA 2020 statement [12]. The protocol was registered prospectively in the Open Science Framework registry (OSF; https://osf.io/25uf9); the completed PRISMA checklist is provided in Supplementary Appendix S1.

Search Strategy

PubMed, Ovid MEDLINE, Web of Science Core Collection, and the Cochrane Central Register of Controlled Trials (CENTRAL) were searched for records published from 1 January 2015 to 31 July 2026 (CENTRAL searched on 20 August 2026). The search window opens with the regulatory approval of the first IL-17 inhibitor (secukinumab, January 2015) and encompasses the published results of all major cardiovascular outcome trials of contemporary therapies. Search strategies, developed with the PRESS 2015 checklist [17], combined psoriasis vocabulary (psoriasis, psoriatic, psoriatic arthritis) with cardiovascular terms (cardiovascular disease, myocardial infarction, stroke, heart failure, atherosclerosis, coronary artery disease) and pharmacological terms (drug therapy, adverse effects, drug interactions, drug repurposing). Full electronic search strategies for all four databases and the total number of records retrieved per database are documented in Supplementary Appendix S2.

Eligibility Criteria

Eligible designs comprised randomised controlled trials (RCTs), cohort studies (n ≥ 500 for observational designs), nested case-control studies, meta-analyses, systematic reviews, and Mendelian randomisation (MR) studies. Populations comprised adults (≥18 years) with psoriasis, cardiovascular disease, or both. Eligible interventions included cardiovascular drugs affecting psoriasis, and psoriasis therapies affecting cardiovascular outcomes. Comparators were placebo,

active comparators, or no treatment. Primary outcomes were (i) incident or exacerbated psoriasis following cardiovascular drug exposure, and (ii) MACE, cardiovascular mortality, and surrogate markers (coronary artery calcium, carotid intima-media thickness, vascular inflammation on FDG-PET/CT, perivascular fat attenuation index) following psoriasis therapy. Secondary outcomes comprised changes in psoriasis severity scores (PASI, BSA, PGA), inflammatory biomarkers (hs-CRP, GlycA, IL-6), and individual cardiovascular endpoints. Non-English records, case reports, animal studies, conference abstracts without full text, and studies with fewer than 20 participants were excluded.

Selection, Extraction, Bias Assessment, and Synthesis

Two reviewers screened records independently using Rayyan [18], extracted data on a piloted standardised form, and resolved disagreements by consensus or third-reviewer adjudication. Risk of bias was assessed with RoB 2 [19] for RCTs, ROBINS-I [20] for observational studies, and AMSTAR 2 [21] for systematic reviews. Certainty of evidence for 24 prespecified outcomes — spanning the effects of eight cardiovascular drug classes and seven psoriasis therapy classes on the primary outcomes — was graded with GRADE [22]. Heterogeneity in interventions, populations, outcomes, study designs, and follow-up durations precluded quantitative synthesis; evidence was therefore integrated by structured narrative synthesis in accordance with PRISMA 2020 guidance, organised by drug class within a direction-of-effect framework.

Ethical Statement

This systematic review synthesised only previously published studies; no participants were recruited, no individual-level patient data were collected, and no biological samples were used. Ethical approval and informed consent were therefore not required.

Study Selection and Quality

The four databases yielded 5,378 records (PubMed 1,107; Ovid MEDLINE 1,064; Web of Science Core Collection 2,774; CENTRAL 433). After removal of 2,214 duplicates, 3,164 records were screened, 2,006 were excluded at title and abstract review, and 1,158 reports were sought for retrieval (Figure 1). Of these, 587 reports were not obtainable (560 full texts unavailable; 15 non-English; 12 conference abstracts only), and seven were excluded after full-text assessment (three with inadequate sample size, three with incompatible design, one with inappropriate population). Ultimately, 556 studies (564 reports) met the inclusion criteria. Among the 56 core evidence studies that directly addressed prespecified outcomes, 33 (59%) were judged at low risk of bias, 18 (32%) at moderate risk, and 5 (9%) at high risk. GRADE certainty ranged from very low to moderate; no outcome reached high certainty. All screening decisions and exclusion reasons are documented in a complete audit trail.

Shared Pathogenic Mechanisms

Chronic cutaneous inflammation propagates a systemic inflammatory state that accelerates atherogenesis. In two independent cohorts, GlycA — a nuclear magnetic resonance biomarker of systemic inflammation — mediated the association between psoriasis severity and cardiovascular outcomes [25], demonstrating that skin severity indirectly tracks vascular risk.

The cytokine core of psoriasis (TNF-α, IL-23, IL-17A, IL-6) operates in atherosclerosis as well, producing divergent effects in the two tissue compartments. IL-17A illustrates this compartment-specific behaviour: it drives keratinocyte hyperproliferation and neutrophil recruitment in skin, while in

the plaque it promotes matrix metalloproteinase-mediated cap thinning and stimulates smooth-muscle-cell collagen synthesis, which stabilises the fibrous cap [26–29]. The CANTOSâ CIRT comparison clarifies the mechanistic picture: canakinumab (anti-IL-1β) reduced MACE by 15% without lipid lowering, whereas low-dose methotrexate — a broad anti-inflammatory — showed no cardiovascular benefit, pointing to NLRP3-IL-1β-IL-6 pathway inhibition, not broad immunosuppression, as the operative mechanism [31,86]. These findings converge on a shared therapeutic node: pro-inflammatory cytokines impair endothelial nitric oxide synthase activity [32,33], reducing NO bioavailability; agents that restore NO/cGMP signalling may therefore benefit both vascular function and keratinocyte differentiation [34].

Additional molecular bridges link the two conditions. Neutrophil extracellular traps (NETs), driven by the IL-23/IL-17 axis, activate macrophage inflammation through STING/NF-κB signalling in psoriatic skin [35]; circulating NET burden independently correlates with coronary stenosis severity [36]. Gut dysbiosis links both conditions via trimethylamine N-oxide, which is elevated in psoriasis and independently predicts cardiovascular events [37,38]. Single-cell RNA sequencing has identified $TREM2^{+}$ lipid-associated macrophages enriched in atherosclerotic plaques [39] with analogues in inflamed skin, while $TCF7^{+}$ memory T cells and $CXCL13^{+}$ peripheral helper T cells appear in both psoriatic dermis and coronary perivascular adipose tissue [40].

MR studies probe the directionality of these associations. Ramessur et al. found that genetic liability to coronary artery disease and stroke increased psoriasis risk (inverse-variance weighted OR 1.07, 95% CI 1.04–1.10 and 1.22, 1.05–1.41, respectively), whereas psoriasis liability showed no effect on coronary artery disease (OR 0.99, 0.96–1.02) [41]. These estimates suggest that cardiovascular disease may drive psoriasis incidence more strongly than the reverse, potentially through shared genetic architecture and endothelial dysfunction that precedes skin manifestations [42,43].

Cardiovascular Risk Burden and Assessment

Psoriasis confers an approximately 50% higher relative risk of MACE [1,44–47]. Meta-analyses report coronary artery disease relative risks of 1.51 (95% CI 1.04–2.18) in Europeans and 1.91 (1.62–2.25) in East Asians, and myocardial infarction relative risks of 1.23 and 2.17 in the respective populations [42,48,49]. Atherosclerosis accelerates before clinical events become apparent: patients show increased carotid intima-media thickness correlating with PASI, greater non-calcified coronary plaque burden, and increased vascular ^{18}F-FDG uptake on PET/CT [50–52]. Conventional risk calculators underestimate this burden [9,53,54]; the 1.5-fold multiplier adopted for rheumatoid arthritis has been proposed for psoriasis [55], and perivascular fat attenuation index provides a non-invasive imaging metric of coronary inflammation [56] that may refine risk reclassification.

Cardiovascular Drugs and Psoriasis

Cardiovascular drugs exhibit divergent dermatological profiles, with some classes triggering or exacerbating psoriasis and others carrying signals of improvement (Table 1).

Beta-blockers. The French national pharmacovigilance database recorded 225 psoriatic events after beta-blocker exposure (ROR 8.95, 95% CI 7.75–10.33), with improvement after discontinuation in 68% of cases [57]; a network meta-analysis returned a pooled OR of 1.40 (1.20–1.63) [23]. Three complementary study designs inform this association: a cross-sectional analysis of US claims (OR 1.10 for cardioselective agents vs. 2.65 for non-cardioselective agents) [58]; two case reports of erythrodermic psoriasis following beta-blocker exposure [59,60]; and MR analyses yielding near-null genetic estimates (OR ≈ 1.00) [61]. The disparity between strong pharmacovigilance signals and null

MR results is compatible with confounding by indication at the population level, with an idiosyncratic triggering effect confined to susceptible individuals.

ACE inhibitors and other antihypertensives. ACE inhibitors carry the highest psoriasis risk among antihypertensive classes (pooled OR 1.67, 95% CI 1.31–2.13), plausibly through bradykinin and substance P accumulation [23], with supporting MR evidence [61]. Calcium channel blockers showed a class-level signal (OR 1.53, 1.23–1.89) with subtype heterogeneity, and thiazide diuretics showed modestly elevated risk (OR 1.23).

Statins exert pleiotropic anti-inflammatory effects [62]. A meta-analysis of ten randomised trials found reduced psoriasis severity (standardised mean difference for PASI −0.36 overall; −0.82 when combined with topical therapy) [24]. The EADV Psoriasis Task Force recommends systematic cardiovascular risk assessment and statin consideration in psoriasis [63].

SGLT2 inhibitors reduced heart failure hospitalisation and cardiovascular death in landmark trials [13,64,65]. In a target trial emulation matching 8,799 SGLT2 inhibitor users to DPP-4 inhibitor users with psoriasis and type 2 diabetes, SGLT2 inhibition was associated with lower all-cause mortality and cardiovascular events, though psoriasis-specific outcomes were not reported [66].

GLP-1 receptor agonists reduced MACE by 13–26% in diabetes trials [14,67] and by 20% in obesity without diabetes (SELECT: HR 0.80, 0.72–0.90) [68]. Weight loss of 5–15%, suppression of pro-inflammatory cytokines, and CRP lowering [69] position GLP-1 receptor agonists as dual-target candidates along the metabolic-inflammatory axis [70].

PCSK9 inhibitors reduced cardiovascular events in FOURIER and ODYSSEY OUTCOMES [15,71]; PCSK9 is upregulated in psoriatic keratinocytes and macrophages [72], but anti-psoriatic effects have not been tested.

Low-dose colchicine reduced MACE by 23% after myocardial infarction (COLCOT: HR 0.77, 0.61–0.96) [16] and by 31% in chronic coronary disease (LoDoCo2: HR 0.69, 0.57–0.83) [73]. Tubulin binding suppresses NLRP3 inflammasome assembly and IL-1β/IL-18 release [74], mechanisms implicated in psoriasis pathogenesis.

Psoriasis Therapies and Cardiovascular Outcomes

TNF-α inhibitors carry the most consistent observational evidence of reduced cardiovascular events among biologics, with lower pericoronary fat attenuation index and slowed non-calcified plaque progression [75–77].

IL-17 inhibitors (secukinumab, ixekizumab, brodalumab, bimekizumab) show no MACE signal, with improvements in endothelial function and coronary microcirculation [78,79]. The VIP-S randomised trial found no change in aortic vascular inflammation (change −0.053, 95% CI −0.169 to 0.063, p = 0.37) [80]; the CARIMA trial reported improved endothelial function with secukinumab (change in flow-mediated dilation +2.0%, p = 0.01) [81].

IL-23 inhibitors showed no MACE signal in registration and long-term extension studies [82]. Ustekinumab reduced aortic vascular inflammation by 18.65% (95% CI 7.85–29.45%) in the VIP-U crossover trial [83], whereas briakinumab was withdrawn after a MACE signal during development, raising the possibility of target-specific rather than class-level differences [84,85].

Among conventional agents, methotrexate is associated with reduced MACE and slowed carotid intima-media thickness progression in observational studies, although the neutral CIRT trial in non-psoriatic populations constrains causal interpretation [86]. Ciclosporin A induces dose-dependent

hypertension, dyslipidaemia, and impaired glucose tolerance; systemic glucocorticoids elevate blood pressure and glycaemia. Apremilast improved endothelial glycocalyx integrity and left ventricular myocardial function in one imaging study [87].

JAK inhibitors: tofacitinib showed MACE and thromboembolism rates comparable to placebo and adalimumab in psoriatic arthritis (pooled MACE incidence 0.25 per 100 patient-years) [88], but the ORAL Surveillance signals of MACE, venous thromboembolism, and malignancy in rheumatoid arthritis mandate class-wide caution [89]. The TYK2-selective inhibitor deucravacitinib has not been associated with cardiovascular signals to date [90].

Dual-Target Drug Repurposing

Candidates span both directions of the axis (Supplementary Table S5). Among cardiovascular agents with anti-psoriatic potential, PDE5 inhibitors stand out: in a TriNetX analysis of 509,788 men, PDE5 inhibition was associated with lower all-cause mortality (RR 0.66 for tadalafil, 0.76 for sildenafil) independently of erectile function [93,94]; psoriasis was not a specific endpoint, but erectile dysfunction is threefold more prevalent in psoriasis [95], creating a clinical niche. Metformin inhibits NLRP3 inflammasome activation in keratinocytes [97] and reduced psoriasis severity in a meta-analysis [98]. Computational repurposing screens have identified antihypertensive candidates with anti-psoriatic cytokine profiles [99].## Discussion

This systematic review of 556 studies demonstrates that pharmacological effects along the skin-heart axis are bidirectional. Cardiovascular drugs can precipitate psoriasis in susceptible individuals (beta-blockers, ACE inhibitors, thiazides) or confer benefit (statins, GLP-1 receptor agonists, SGLT2 inhibitors, colchicine). Psoriasis therapies carry divergent cardiovascular profiles: TNF inhibitors and methotrexate are associated with reduced MACE, IL-17 and IL-23 inhibitors show neutral cardiovascular safety, and ciclosporin A and systemic glucocorticoids increase cardiovascular risk. These findings favour a joint dermatologicalâ cardiovascular approach to drug selection over sequential specialty-specific decision-making.

The beta-blocker controversy demonstrates how multi-design evidence adjudication can resolve an apparent contradiction. A pharmacovigilance ROR of 8.95 coexisted with a near-null MR estimate; confounding by indication at the population level, with an idiosyncratic triggering effect restricted to susceptible individuals, is the most coherent explanation of this divergence. For clinical practice, non-cardioselective beta-blockers are best avoided when alternatives exist for patients with active psoriasis; patients can be counselled on the approximately 1.4-fold observational risk; and beta-blockers should not be withheld after myocardial infarction based solely on psoriasis history. The triangulation of pharmacovigilance, observational studies, and MR provides a generalisable approach for evaluating other contested drug-disease associations.

The beta-blocker example also exposes a broader challenge: the evidence base across the axis is predominantly observational, and the risk of confounding must be weighed against the clinical urgency of the question. Methodological strengths of this review include prospective OSF registration, dual independent screening, formal risk-of-bias and GRADE assessment, and the deliberate incorporation of complementary study designs to strengthen causal inference. The 5,378-record screen, 556-study inclusion, and breadth of the reference synthesis make this the most comprehensive evidence appraisal of the skin-heart pharmacological axis to date. Several limitations warrant acknowledgment. No head-to-head cardiovascular outcome trial between psoriasis therapies has been conducted. Heterogeneity in interventions, populations, outcomes, and study designs precluded quantitative synthesis. Grey literature and non-English records were excluded, and publication bias could not be

formally assessed without meta-analysis. Most GRADE ratings ranged from very low to moderate, reflecting the predominance of observational evidence; low-certainty evidence, however, itself identifies research gaps that would otherwise remain uncharted.

Actionable clinical signals have emerged, particularly on the improvement side of the axis. GLP-1 receptor agonists combine established MACE reduction with weight loss, cytokine suppression, and CRP lowering. Obesity is a dose-dependent risk factor for psoriasis, so both direct anti-inflammatory mechanisms and indirect disease-modifying effects through weight reduction are plausible. Low-dose colchicine targets the NLRP3-IL-1β pathway validated by CANTOS, at a fraction of the cost of biologic therapy. PDE5 inhibitors carry a mortality reduction signal but lack dedicated psoriasis endpoints. These converging observations support a clinical decision framework in which psoriasis is treated as a cardiovascular risk enhancer, cardiovascular drugs are selected with consideration of their dermatological profile, psoriasis therapy selection is guided by individual cardiovascular phenotype, and drug repurposing candidates with evidence of dual-compartment benefit are prioritised for prospective evaluation (Figure 4). The risk-stratified treatment algorithm (Table 3) translates this framework into a practical tool for dermatology-cardiology co-management.

Among the repurposing candidates identified, GLP-1 receptor agonists are positioned for testing in a first Phase IIa trial. Their cardiovascular evidence base is mature; weight loss addresses a shared risk factor; and direct anti-inflammatory effects are documented. The hypothesis that GLP-1 receptor agonism modifies psoriasis disease activity awaits prospective evaluation with skin-specific endpoints. Prospective studies with adequate cardiovascular follow-up in psoriasis cohorts — particularly in populations currently underrepresented in the evidence base — remain necessary. Future systematic reviews would benefit from individual-patient-data meta-analysis if sufficient RCT-level data become available, and standardised cardiovascular outcome reporting in psoriasis trials would substantially strengthen the evidence base for treatment decisions at the skin-heart interface.

Declarations

Author Contributions: Xinying Xiao: conceptualization, investigation, data curation, writing of the original draft. Sibao Chen: investigation, data curation, formal analysis, writing (review and editing). Zhouyang Zhu: conceptualization, supervision, validation, writing (review and editing). All authors approved the final manuscript. Xinying Xiao and Sibao Chen contributed equally.

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Data Availability Statement: All data extracted from the included studies are available in the supplementary materials. The review protocol is publicly accessible at the Open Science Framework (https://osf.io/25uf9). No new primary data were generated for this study.

Clinical trial number: not applicable.

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Figure Legends

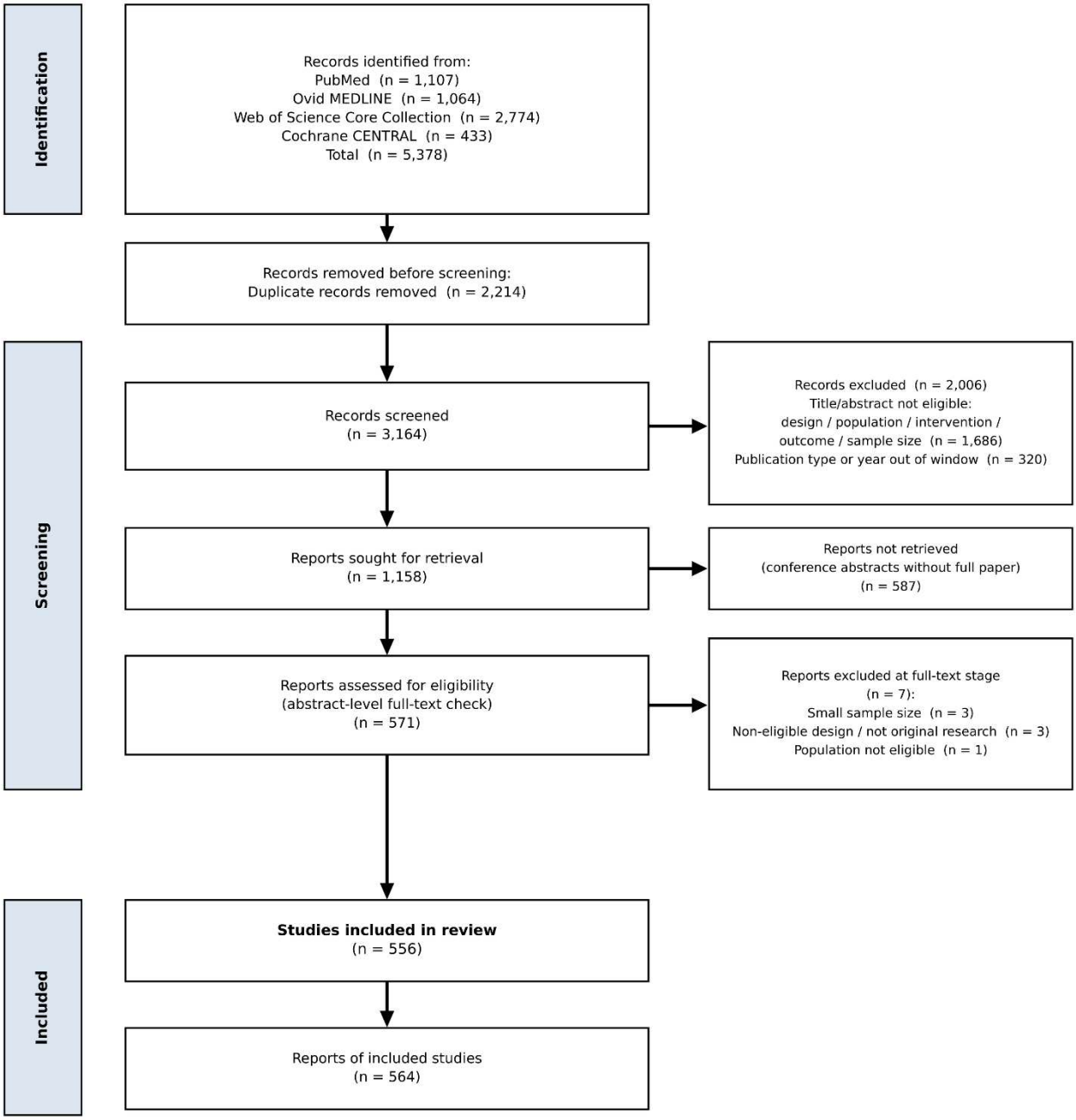
Figure 1. PRISMA 2020 flow diagram of study identification and selection. Four databases yielded 5,378 records (PubMed 1,107; Ovid MEDLINE 1,064; Web of Science Core Collection 2,774; Cochrane CENTRAL 433). After removal of 2,214 duplicates, 3,164 records were screened and 2,006 were excluded at title and abstract review; 1,158 reports were sought for retrieval, of which 587 were not obtainable and 7 were excluded after full-text assessment, leaving 556 studies (564 reports) for inclusion.

Figure 2. The IL-17 paradox in atherosclerosis. IL-17A exerts context-dependent dual effects on plaque stability. Pro-atherogenic arm (red): IL-17A stimulates matrix metalloproteinase (MMP) expression in vascular smooth muscle cells and macrophages, and promotes chemokine-driven (CXCL1/2, IL-6) neutrophil and monocyte recruitment, destabilising the fibrous cap. Atheroprotective arm (blue): IL-17A stimulates smooth-muscle-cell collagen synthesis and promotes a stable plaque phenotype. Modulators: TGF-β favours the atheroprotective arm; IFN-γ and conventional cardiovascular risk factors favour the pro-atherogenic arm.

Figure 3. Forest plot of the beta-blocker-psoriasis association across study designs. Point estimates and 95% CIs from a network meta-analysis of observational studies (Song et al., ref 23: pooled OR

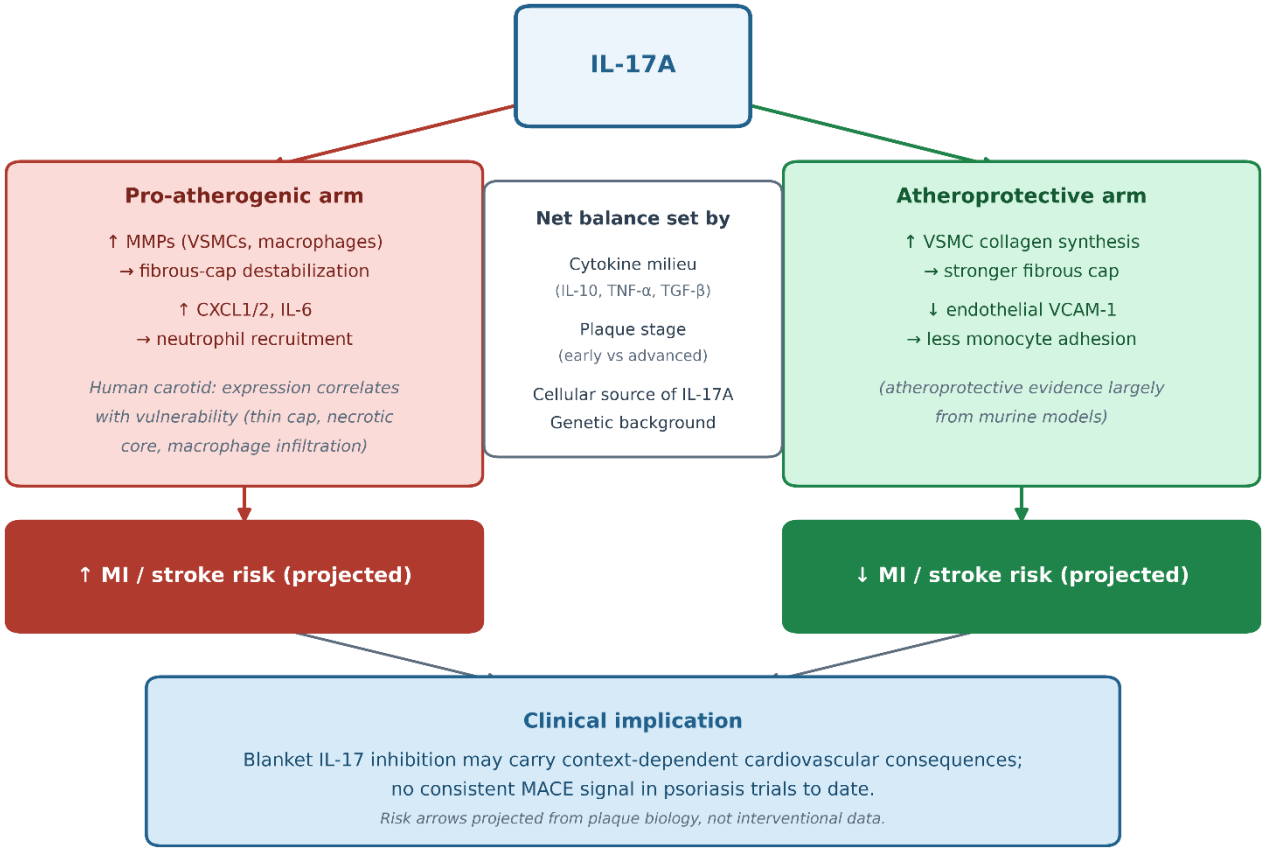
1.40, 95% CI 1.20–1.63), French pharmacovigilance (Azzouz et al., ref 57: ROR 8.95, 95% CI 7.75–10.33), cross-sectional US claims (Zheng et al., ref 58: OR 1.10 cardioselective; OR 2.65 non-cardioselective), and Mendelian randomisation (Fan et al., ref 61: OR ≈ 1.00). Positive observational signals contrast with near-null genetic estimates, consistent with confounding by indication.

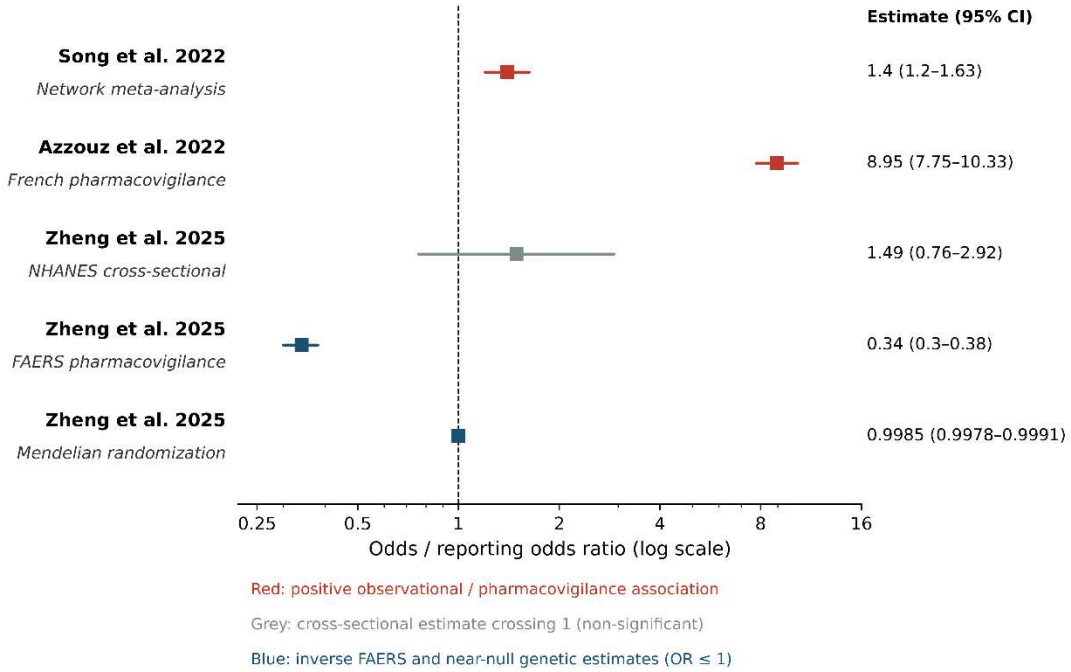
Figure 4. Clinical Decision Framework for the Psoriasis–Cardiovascular Axis. A three-step, evidence-based approach integrating cardiovascular risk assessment (Step 1), phenotype-guided treatment selection for cardiovascular drugs and psoriasis therapies (Step 2), and dual-target repurposing priorities (Step 3). ARB, angiotensin receptor blocker; CCB, calcium channel blocker; CIMT, carotid intima-media thickness; FAI, fat attenuation index; GLP-1 RA, glucagon-like peptide-1 receptor agonist; MACE, major adverse cardiovascular events; PASI, Psoriasis Area and Severity Index; PDE5i, phosphodiesterase-5 inhibitor; SGLT2i, sodium-glucose cotransporter-2 inhibitor.

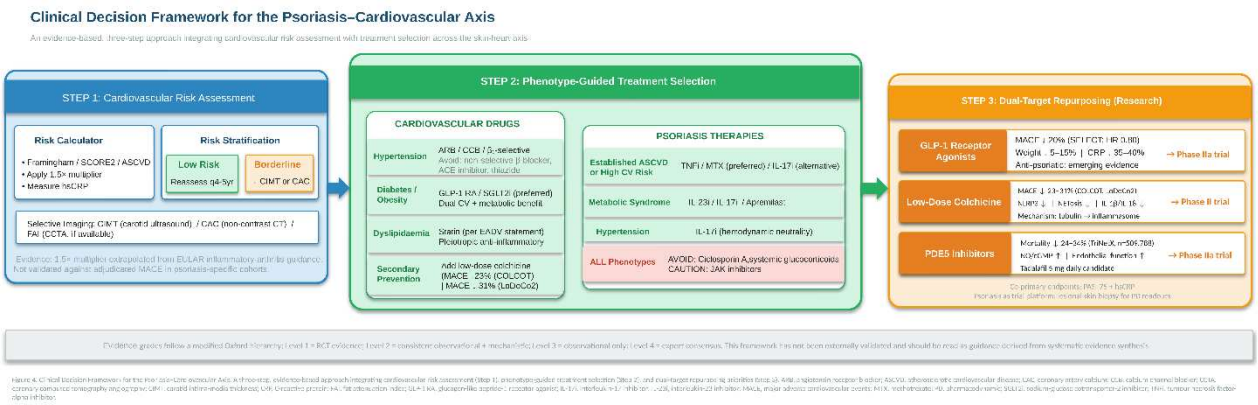

Identification
Records identified from:
PubMed (n = 1,107)
Ovid MEDLINE (n = 1,064)
Web of Science Core Collection (n = 2,774)
Cochrane CENTRAL (n = 433)
Total (n = 5,378)
Records removed before screening:
Duplicate records removed (n = 2,214)
Screening
Records screened
(n = 3,164)
Records excluded (n = 2,006)
Title/abstract not eligible:
design / population / intervention /
outcome / sample size (n = 1,686)
Publication type or year out of window (n = 320)
Reports sought for retrieval
(n = 1,158)
Reports not retrieved
(conference abstracts without full paper)
(n = 587)
Reports assessed for eligibility
(abstract-level full-text check)
(n = 571)
Reports excluded at full-text stage
(n = 7):
Small sample size (n = 3)
Non-eligible design / not original research (n = 3)
Population not eligible (n = 1)
Included
Studies included in review
(n = 556)
Reports of included studies
(n = 564)

The IL-17 paradox in atherosclerosis

Context-dependent dual effects of IL-17A on plaque stability







Tables

Table 1. Cardiovascular Drug Classes and Their Effects on Psoriasis

Table 2. Summary of Psoriasis Therapies and Cardiovascular Outcomes

Table 3. Risk-Stratified Treatment Selection for Psoriasis Based on Cardiovascular Phenotype

Supplementary Material

Supplementary Table S1: study characteristics; S2: risk of bias; S3: GRADE profile; S4: exclusions with reasons; S5: repurposing candidates. Supplementary Appendix S1: PRISMA 2020 checklist; S2: search strategies.

Supplementary Files

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