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Research Meta-anaylsis

ACE Inhibitors versus Angiotensin Receptor Blockers for Cardiovascular Protection in Hypertension: A Metanalysis of Major Cardiovascular Endpoints

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Abstract

Angiotensin-converting enzyme inhibitors (ACEi) and angiotensin receptor blockers (ARBs) are both first-line agents for the pharmacological treatment of hypertension and are recommended interchangeably by most major guidelines. Despite acting on the same renin-angiotensin-aldosterone system (RAAS) pathway, these two drug classes are not pharmacologically identical, and a body of trial and meta-analytic evidence has raised the question of whether they confer equivalent protection against hard cardiovascular endpoints — cardiovascular (CV) death, all-cause mortality, non-fatal stroke, and heart failure. This review summarizes the mechanistic rationale, synthesizes evidence from head-to-head and placebo-controlled trials, and situates the findings within current hypertension guidelines to provide a practical framework for clinical decision-making.

1. Introduction

Hypertension remains the leading modifiable risk factor for cardiovascular disease worldwide. Blockade of the RAAS is central to hypertension management because angiotensin II drives vasoconstriction, sodium retention, sympathetic activation, vascular remodeling, and pro-inflammatory and pro-fibrotic signalling — all of which contribute to target-organ damage. ACE inhibitors and ARBs both interrupt this pathway, but at different points, and this pharmacological distinction has been proposed as a possible explanation for differences observed in some outcome trials.

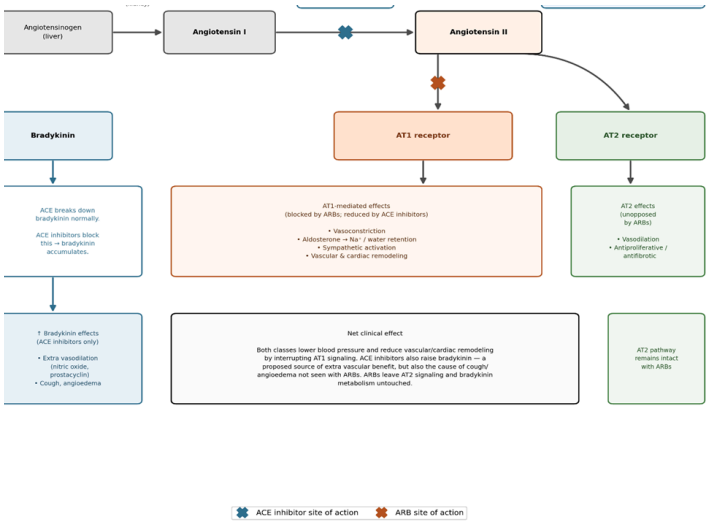
2. Mechanistic Differences

ACE inhibitors block the enzyme that converts angiotensin I to angiotensin II. Because ACE is identical to kininase II, ACE inhibition also prevents the breakdown of bradykinin. Elevated bradykinin contributes to the class-specific side effects of cough and angioedema, but it is also hypothesized to contribute additional vasodilatory, anti-thrombotic, and endothelium-protective effects (nitric oxide and prostacyclin release) that are not shared by ARBs.

ARBs selectively block the angiotensin II type 1 (AT1) receptor, leaving angiotensin II levels unchanged or elevated. Unopposed angiotensin II can still stimulate the AT2 receptor, which some believe carries vasodilatory and antiproliferative properties, but ARBs do not augment bradykinin activity.

This "bradykinin hypothesis" is the most commonly cited biological explanation for any divergence in hard outcomes between the two classes, although it remains incompletely proven in humans.

Picture 1: Mecahnsim of Action: ACE Inhibitors Vs Angiotensin Receptor Blockers ARBs



3. Evidence from Placebo-Controlled Trials

3.1 ACE Inhibitor Trials

The HOPE trial (Heart Outcomes Prevention Evaluation) [1], which enrolled high-risk patients with vascular disease or diabetes plus one additional risk factor, showed that ramipril significantly reduced the composite of CV death, myocardial infarction (MI), and stroke, as well as all-cause mortality, compared with placebo — effects that exceeded what would be predicted from blood pressure lowering alone.

3.2 ARB Trials

Placebo-controlled ARB trials in comparable high-risk or heart failure populations (e.g., CHARM, VALIANT, TRANSCEND [16]) have generally demonstrated reductions in heart failure hospitalization and, in some cases, stroke, but have been less consistent in showing reductions in all-cause or cardiovascular mortality.

3.3 Pooled/Parallel Meta-Analyses

A frequently cited parallel meta-analysis pooling 20 contemporary hypertension trials (n ≈ 159,000 patients) [3] found that, when analyzed separately:

- ACE inhibitors reduced all-cause mortality by approximately 10%, with a trend toward reduced cardiovascular mortality.
- ARBs showed no significant reduction in either all-cause or cardiovascular mortality relative to comparators.

A separate meta-analysis of 10 placebo-controlled trials in high-risk patients (~77,600 patients) [4] reported that ACE inhibitors significantly reduced all-cause mortality, cardiovascular mortality, non-fatal MI, and stroke, whereas ARBs produced only a modest reduction in stroke with no significant effect on mortality or non-fatal MI.

These findings have led some authors to argue that, on mortality grounds, ACE inhibitors should generally be favored as first choice within RAAS-blocker therapy, reserving ARBs primarily for patients who are intolerant of ACE inhibitors (most commonly because of cough or angioedema).

4. Head-to-Head Evidence

4.1 ONTARGET

The landmark ONTARGET trial (Ongoing Telmisartan

Alone and in Combination with Ramipril Global Endpoint Trial) [2] directly compared the ARB telmisartan, the ACE inhibitor ramipril, and their combination in over 25,000 high-risk patients with vascular disease or diabetes with end-organ damage. Telmisartan was non-inferior to ramipril for the composite primary outcome of CV death, MI, stroke, or heart failure hospitalization, with essentially superimposable event curves over more than four years of follow-up. Telmisartan caused significantly less cough and angioedema, while combination therapy increased hypotension, syncope, renal impairment, and hyperkalemia without providing additional cardiovascular benefit [10] — reinforcing current guidance against combining an ACE inhibitor with an ARB.

4.2 Direct Comparative Meta-Analyses in Primary Hypertension

A meta-analysis of nine randomized head-to-head trials (~11,000 patients) [5] specifically in essential hypertension found no significant difference between ACE inhibitors and ARBs for cardiovascular events or total mortality, though ACE inhibitors were associated with modestly higher rates of treatment discontinuation, largely driven by cough.

A broader synthesis comparing ACE inhibitors and ARBs across hypertension and "compelling indication" populations [6,7] similarly found no significant difference in all-cause mortality, cardiovascular mortality, MI, heart failure, stroke, or progression to end-stage renal disease, but noted consistently lower drug-withdrawal rates with ARBs due to their more favorable tolerability profile.

4.3 Recurrent Stroke (Secondary Prevention)

Evidence for secondary stroke prevention — reducing the risk of a recurrent stroke in patients

with a prior stroke or TIA — favors ACE inhibitors more clearly than the general hypertension evidence base does.

The PROGRESS trial (Perindopril Protection Against Recurrent Stroke Study) [11] randomized patients with prior stroke or TIA to a perindopril-based regimen (with or without indapamide) versus placebo. Active treatment produced a 28% relative risk reduction in Taken together, ACE inhibitor-based regimens (particularly in combination with a diuretic) currently have the strongest and most consistent trial evidence for reducing recurrent stroke, and are generally preferred over ARBs for this specific indication, while acknowledging that the PROGRESS benefit was closely tied to the magnitude of blood pressure lowering achieved and that no adequately powered head-to-head ACEi-vs-ARB trial exists in this population.

recurrent stroke, with the benefit concentrated in patients who received combination therapy with a diuretic (43% RRR) and achieving a larger blood pressure reduction (˜12/5 mmHg) versus perindopril alone (˜5/3 mmHg, no significant benefit).

- The PRoFESS trial [12], the corresponding large-scale outcome trial for an ARB in this population, tested telmisartan added to standard secondary-prevention care in over 20,000 patients with recent ischemic stroke. Despite a modest blood pressure reduction, telmisartan failed to significantly reduce recurrent stroke or major cardiovascular events versus placebo.
- Other ARB trials in stroke populations have shown mixed results: candesartan (SCOPE, small subgroup) and eprosartan (MOSES) suggested benefit, but the largest dedicated ARB secondary-prevention trial (PRoFESS) was neutral, whereas the ACE inhibitor-based PROGRESS trial remains the most robust positive trial specifically in recurrent stroke prevention.

Taken together, ACE inhibitor-based regimens (particularly in combination with a diuretic) currently have the strongest and most consistent trial evidence for reducing recurrent stroke, and are generally preferred over ARBs for this specific indication, while acknowledging that the PROGRESS benefit was closely tied to the magnitude of blood pressure lowering achieved and that no adequately powered head-to-head ACEi-vs-ARB trial exists in this population.

4.4 Reconciling the Discrepancy

The apparent tension between placebo-controlled meta-analyses (favoring ACE inhibitors) and head-to-head trials/meta-analyses (showing equivalence likely reflects several factors:

- Head-to-head trials such as ONTARGET enrolled patients already established on RAAS therapy and screened for tolerability, which may underrepresent the population in which ACE inhibitor-specific benefits (e.g., bradykinin-mediated effects) would be most apparent.
- Many placebo-controlled ARB trials had shorter follow-up or enrolled lower-risk populations than the ACE inhibitor outcome trials informing the pooled analyses.

Differences in background therapy, population risk profiles, and statistical power across trials included in different meta-analyses limit direct comparability

5. Endpoint-by-Endpoint Summary

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Table 1: Differences in background therapy, population risk profiles, and statistical power across trials included in different meta-analyses limit direct comparability

Endpoint	ACE Inhibitors	ARBs	Head-to-Head Comparison
All-cause mortality	Consistent reduction vs. placebo in high-risk/hypertensive trials (~10%); pooled meta-analyses favor ACE inhibitors	Generally neutral vs. placebo	No significant difference in direct head-to-head trials (e.g., ONTARGET), but indirect/pooled evidence favors ACE inhibitors
Cardiovascular death	Reduction vs. placebo, particularly in high-risk populations; pooled meta-analyses favor ACE inhibitors	Neutral to modest benefit vs. placebo	No significant difference head-to-head, but indirect evidence favors ACE inhibitors
Non-fatal stroke (primary prevention)	Significant reduction vs. placebo	Modest reduction vs. placebo	No significant difference; both classes effective
Recurrent stroke (secondary prevention)	Significant reduction vs. placebo (PROGRESS, 28% RRR) — strongest trial evidence in this indication	No significant reduction vs. placebo in the dedicated trial (PRoFESS)	ACE inhibitors preferred; no direct head-to-head trial exists, but the evidence base clearly favors ACE inhibitor-based (often diuretic-combined) regimens
Heart failure	Reduction vs. placebo, strong evidence base in HFrEF	Reduction vs. placebo, non-inferior to ACE inhibitors in HFrEF trials	Comparable; ARBs are standard alternative for ACE-intolerant HF patients

6. Drug-Specific Trial Evidence (Ramipril, Perindopril, Telmisartan, Olmesartan)

Table 2: Outcome data differ meaningfully by individual agent, not just by class. The table below summarizes the key cardiovascular-outcome trials for two widely used ACE inhibitors (ramipril, perindopril) and two widely used ARBs (telmisartan, olmesartan).

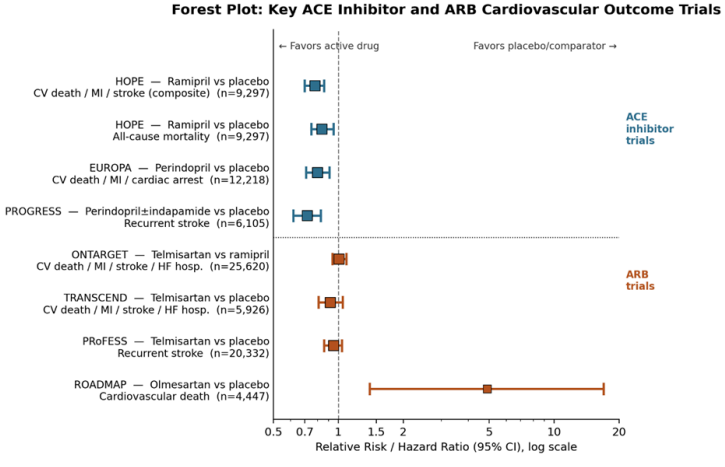
Drug	Class	Key Trial(s)	Population	Reported Cardiovascular Benefit
Ramipril	ACE inhibitor	HOPE [1] (n≈9,300)	High-risk patients with vascular disease or diabetes plus ⩾1 risk factor, without heart failure	Significant reduction in the composite of CV death, MI, and stroke, and in all-cause mortality, versus placebo
Ramipril	ACE inhibitor	ONTARGET [2] (n≈25,600)	High-risk vascular disease/diabetes with end-organ damage	Reference comparator arm; reduced CV death, MI, stroke, and HF hospitalization versus placebo-era risk, and served as the benchmark against which telmisartan was non-inferior
Perindopril	ACE inhibitor	EUROPA [13] (n≈13,655)	Stable coronary artery disease, no heart failure	20% relative risk reduction in the composite of CV death, MI, or cardiac arrest versus placebo
Perindopril (± indapamide)	ACE inhibitor	PROGRESS [11] (n≈6,105)	Prior stroke or TIA (secondary prevention)	28% relative risk reduction in recurrent stroke versus placebo; benefit driven mainly by the perindopril + indapamide combination arm

Telmisartan	ARB	ONTARGET [2] (n≈25,600)	High-risk vascular disease/diabetes with end-organ damage	Non-inferior to ramipril for CV death, MI, stroke, and HF hospitalization; less cough/angioedema, no added benefit from combination with ramipril
Telmisartan	ARB	TRANSCEND [16] (n≈5,926)	High-risk, ACE inhibitor–intolerant patients	Modest, borderline-significant reduction in CV events versus placebo; effect strengthened when pooled with PRoFESS data
Telmisartan	ARB	PRoFESS [12] (n≈20,332)	Recent ischemic stroke (secondary prevention)	No significant reduction in recurrent stroke or major cardiovascular events versus placebo
Olmesartan	ARB	ROADMAP [14] (n≈4,447)	Type 2 diabetes with ⩾1 CV risk factor, normoalbuminuria	Significantly delayed onset of microalbuminuria (primary renal endpoint), but showed a numerically higher rate of fatal cardiovascular events versus placebo (15 vs. 3 CV deaths)
Olmesartan	ARB	ORIENT [15] (n≈566)	Type 2 diabetes with overt nephropathy (Japan/Hong Kong)	No significant benefit on the renal composite endpoint; numerically more cardiovascular deaths versus placebo (10 vs. 3), though event numbers were small

Interpretation: Ramipril and perindopril both have large, well-replicated outcome trials (HOPE [1], ONTARGET [2], EUROPA [13], PROGRESS [11]) demonstrating consistent, significant reductions in hard cardiovascular endpoints across different high-risk populations, including a specific and well-established recurrent-stroke benefit for perindopril. Telmisartan has one of the strongest ARB evidences bases overall (ONTARGET [2] non-inferiority to ramipril, TRANSCEND [16]), but its own dedicated secondary-stroke-prevention trial (PRoFESS [12]) was neutral. Olmesartan's evidence base is comparatively weaker for hard cardiovascular endpoints: its two dedicated placebo-controlled outcome trials (ROADMAP [14], ORIENT [15]) were designed around renal, not cardiovascular, primary endpoints, and both showed a numerical excess of cardiovascular deaths versus placebo in diabetic patients — a signal that prompted an FDA safety review. A subsequent individual-patient-data meta-analysis across the full olmesartan trial program did not confirm an overall excess cardiovascular risk, but no large, dedicated olmesartan cardiovascular-outcome trial comparable to HOPE, EUROPA, or ONTARGET exists, and this remains a relative evidence gap for the drug.

6.1 Forest Plot of Key Trials

The forest plot below summarizes the point estimate (relative risk or hazard ratio) and 95% confidence interval for the primary/key endpoint of each major trial discussed in this review. Squares represent the point estimate (sized loosely by precision); horizontal lines represent the 95% CI. A CI that crosses the vertical reference line at 1.0 indicates no statistically significant difference between arms.



RR/HR values with 95% CI as reported in original trial publications (see reference list). ROADMAP's CI is wide due to few events (15 vs 3 CV deaths); a subsequent pooled individual-patient-data analysis of the full olmesartan program did not confirm an overall excess risk.

Figure 1. Forest plot of relative risk (RR) or hazard ratio (HR) with 95% CI for the primary or key endpoint of each trial. ACE inhibitor trials (HOPE [1], EUROPA [13], PROGRESS [11]) are shown in blue; ARB trials (ONTARGET [2], TRANSCEND [16], PRoFESS [12], ROADMAP [14]) are shown in orange. Note that ONTARGET compares telmisartan against ramipril directly (active-comparator, non-inferiority design), whereas the remaining trials are placebo-controlled. ROADMAP's estimate for cardiovascular death (HR 4.9, 95% CI 1.4–17.0) is imprecise given only 15 vs. 3 events, and a later pooled analysis of the full olmesartan development program did not confirm an overall excess cardiovascular risk — this trial is included for completeness and transparency, not as a definitive verdict on the drug.

7. Tolerability and Its Clinical Relevance

Tolerability is not a secondary consideration — it directly affects long-term adherence, which in turn affects real-world cardiovascular protection. ACE inhibitors are associated with a dry cough in roughly 10–20% of patients and carry a small but serious risk of angioedema, disproportionately affecting Black patients and those with a prior history of angioedema. ARBs largely avoid these bradykinin-mediated effects and show lower overall treatment discontinuation rates in comparative trials, making them a reasonable first-line alternative, not merely a fallback, particularly for patients at elevated angioedema risk.

8. Guideline Recommendations

Current major guidelines — including the 2023 European Society of Hypertension (ESH) guidelines [8] and ACC/AHA guidance — list ACE inhibitors and ARBs among the core first-line antihypertensive classes (alongside calcium channel blockers and thiazide/thiazide-like diuretics), generally treating them as interchangeable for uncomplicated hypertension, with drug-class selection guided more by compelling indications, comorbidities,

and tolerability than by anticipated differences in hard outcomes. Both guidelines explicitly recommend against combining an ACE inhibitor and an ARB, given the lack of incremental cardiovascular or renal benefit and the increased risk of hyperkalemia, hypotension, and acute kidney injury demonstrated in ONTARGET [2,10] and related trials. In diabetic hypertensive patients and those with chronic kidney disease and albuminuria, both classes remain preferred agents for renal and cardiovascular protection [7], with some guidelines and pooled analyses suggesting a modest edge for ACE inhibitors on renal outcomes specifically.

9. Special Populations

- Chronic kidney disease/albuminuria: Both classes reduce proteinuria and slow CKD progression; some data suggest a possible advantage for ACE inhibitors, but ARBs remain an appropriate alternative, especially when cough limits ACE inhibitor use.
- Diabetes: Both classes are guideline-preferred for their renal and cardiovascular protective effects in diabetic hypertension [7]; note, however, that olmesartan's own diabetic-population outcome trials (ROADMAP [14], ORIENT [15]) showed a numerical excess of cardiovascular deaths, so this general class-level guidance should not be assumed to extend uniformly to every individual ARB.
- Heart failure with reduced ejection fraction (HFrEF): ACE inhibitors have the longer and larger evidence base (SOLVD, CONSENSUS), with ARBs as a well-validated alternative for ACE-intolerant patients. Note that angiotensin receptor-neprilysin inhibitors (ARNIs, e.g., sacubitril/valsartan)have since shown superiority over ACE inhibitors alone in reducing CV death and and heart failure hospitalization in HFrEF [9] and are now preferred over either ACE inhibitors or ARBs alone in eligible patients — a distinct class outside the scope of this comparison.
- Black patients: Higher baseline angioedema risk with ACE inhibitors makes ARBs a reasonable first-choice RAAS blocker in this population when RAAS blockade is otherwise indicated.

10. Clinical Practice Implications

1. For most patients with uncomplicated hypertension, ACE inhibitors and ARBs perform comparably in direct head-to-head trials for CV death, all-cause mortality, non-fatal stroke, and heart failure. However, pooled placebo-controlled evidence consistently favors ACE inhibitors for reducing all-cause mortality and cardiovascular death, and this should carry some weight when the two classes are otherwise equally suitable for a given patient.
2. *For secondary prevention of stroke in patients with a prior stroke or TIA, ACE inhibitors (particularly combined with a diuretic, as in PROGRESS) are preferred over ARBs, given that the corresponding large ARB outcome trial (PRoFESS) did not demonstrate a significant reduction in recurrent stroke.*
3. *ARBs remain the preferred initial choice in patients with a prior history of ACE inhibitor-induced cough or angioedema, and may be reasonably considered first-line in populations at elevated angioedema risk, even though this may come with some trade-off in the mortality and recurrent-stroke advantages associated with ACE inhibitors.*
4. *Combination ACE inhibitor plus ARB therapy should be avoided outside of specific, monitored circumstances due to excess adverse events without added cardiovascular benefit.*

11. Conclusion

ACE inhibitors and ARBs both provide substantial and well-documented cardiovascular protection in hypertensive patients, and in direct head-to-head trials such as ONTARGET they perform comparably across most major endpoints. That said, the weight of evidence points to two areas where ACE inhibitors hold a meaningful advantage: placebo-controlled trial data and pooled meta-analyses consistently favor ACE inhibitors for reducing all-cause mortality and cardiovascular death, plausibly related to bradykinin-mediated vascular effects unique to this class; and for recurrent stroke prevention specifically, ACE inhibitors have the stronger and more consistent trial evidence, with PROGRESS demonstrating a clear benefit that the comparable ARB trial (PRoFESS) did not replicate. ARBs retain a clear role — particularly for patients intolerant of ACE inhibitors because of cough or angioedema — but where either class is otherwise suitable, this evidence supports favoring ACE inhibitors as the preferred RAAS blocker for mortality and secondary stroke prevention, with ARBs positioned as the primary alternative when tolerability concerns arise.

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