Baxdrostat:

A Novel Aldosterone Synthase Inhibitor for Treatment Resistant Hypertension

Phase 2 Trial of Baxdrostat for Treatment Resistant Hypertension

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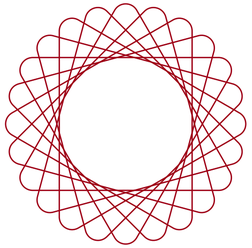
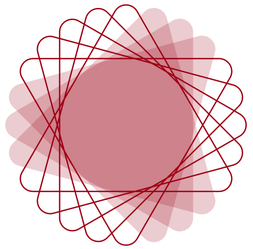
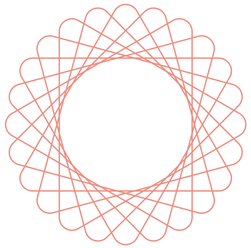
Outline



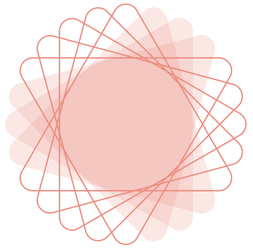
Background



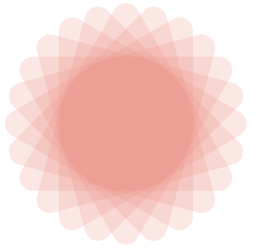
Method&Results



Discussion



Conclusion



Appraisal

Background

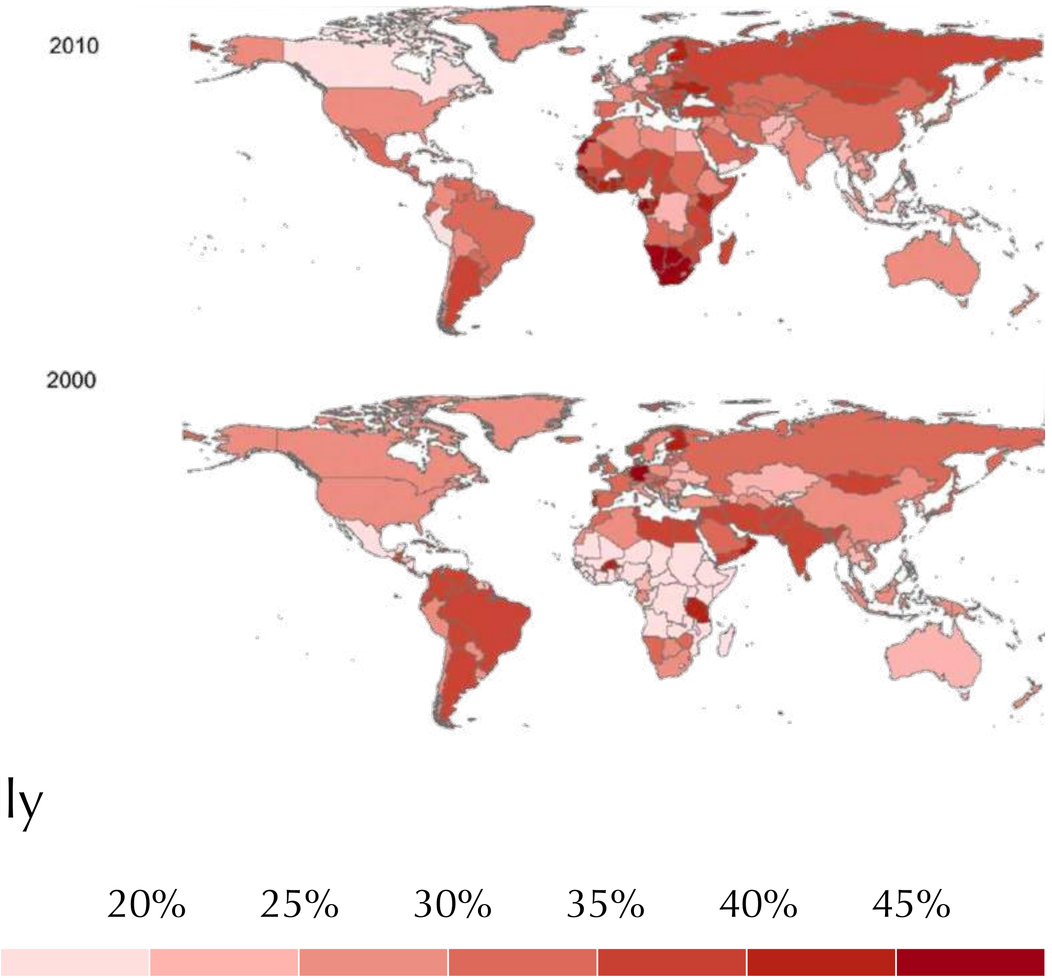
Aldosterone synthase controls the synthesis of aldosterone and has been a pharmacologic target for the treatment of hypertension for several decades.

Selective inhibition of aldosterone synthase is essential but difficult to achieve because cortisol synthesis is catalyzed by another enzyme that shares 93% sequence similarity with aldosterone synthase.

In preclinical and phase 1 studies, baxdrostat had 100:1 selectivity for enzyme inhibition, and baxdrostat at several dose levels reduced plasma aldosterone levels but not cortisol levels.

Background

Approximately **1.13 billion** people worldwide have hypertension.



Health impact of hypertension

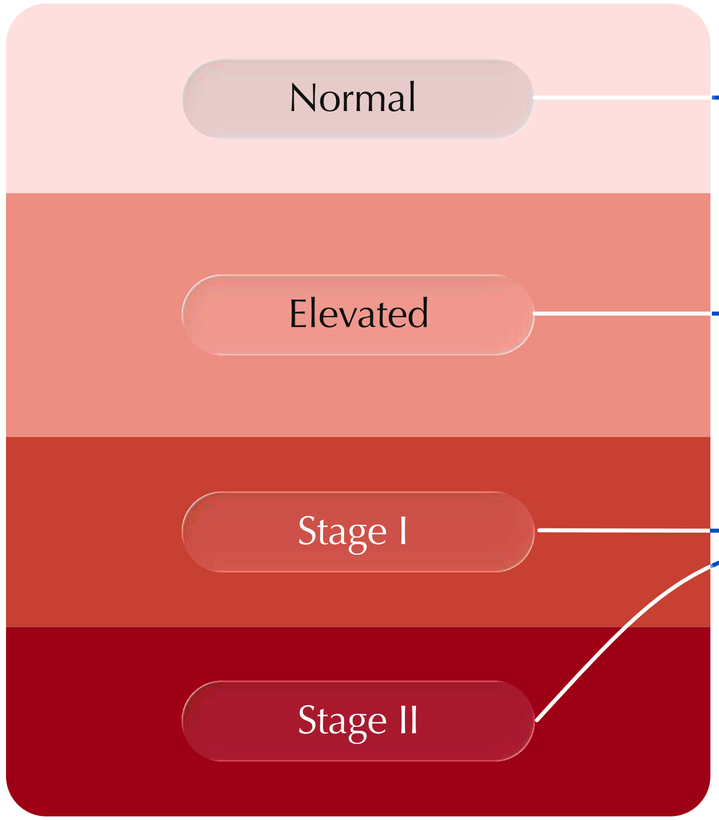
-Major risk factor for cardiovascular disease

-Increases risk of heart attack, stroke, and kidney disease

-Contributes to approximately 10.4 million deaths annually

Background

Blood Pressure Categories

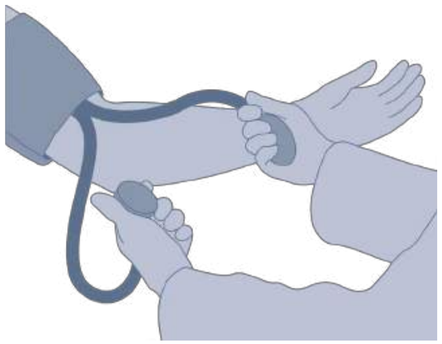


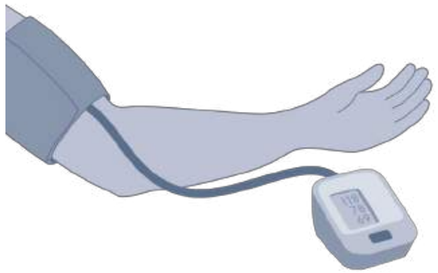
Category

If systolic and diastolic categories are different classification is determined by the higher category.

Office	Home	Daytime	Night	24-hours	
<120/70	<120/70	<120/70	<110/60	<115/65	ESC
120/80 mmHg	120/80	120/80	100/65	115/75	AHA
120/70 I <140/90	120/70 I <135/85	120/70 I <135/85	110/60 I <120/70	115/65 I <130/80	ESC
130/80 mmHg	130/80	130/80	110/65	125/75	AHA
≥140/90	≥135/85	≥135/85	≥120/70	≥130/80	ESC
140/90 mmHg	135/85	135/85	120/70	130/80	AHA
160/100 mmHg	145/90	145/90	140/85	145/90	AHA

Background





Static measurement

Daily activities and sleep

Dynamic conditions

Office BP measurement

- Strong evidence
- Readily available
- Often not standardized
- Poor reproducibility
- Subject to white-coat and masked hypertension effects

Home BP monitoring

- Widely available
- Acceptable by users
- Best method for long-term follow-up of treated patients
- Requires training and medical supervision
- Variable accuracy of devices available on the market
- Possible misreporting of readings by users

Ambulatory BP monitoring

- Multiple readings over 24 h
- Measures BP levels during daily activities and sleep
- Best method for hypertension diagnosis
- Not widely available
- Not accepted by all users, particularly for repeated use

Cuffless wearable BP monitors

- Great potential for BP screening,monitoring and management
- Can provide multiple readings over long periods of time
- No cuff-induced discomfort
- Questionable accuracy
- Unproven clinical usefulness

BP variability

- Long-term
- Visit-to-visit

BP variability

- Mid-term
- Day-to-day

BP variability

- Short-term
- Hour-to-hour

BP variability

- Very short-term, short-term,mid-term and long-term
- Beat-to-beat, hour-to-hour,day-to-day, week-to-week and month-to-month

Background

Primary Hypertension

Definition

Hypertension without an identifiable secondary cause, accounting for approximately 90-95% of all cases of hypertension.

Pathogenesis

Multifactorial etiology with complex interactions between
Genetic predisposition
Environmental factors
Neurohumoral mechanisms

Key physiological systems involved:
Sympathetic nervous system
Renin-angiotensin-aldosterone system
Renal sodium handling
Vascular structure and function

Non-modifiable Risk Factors

Age
Blood pressure increases with age, particularly systolic pressure

Family history
Genetic factors account for approximately 30-50% of cases

Race/ethnicity
Higher prevalence and severity in Black individuals

Modifiable Risk Factors

Obesity
Each 10 kg weight gain associated with 3mmHg rise in systolic BP

Alcohol consumption
More than 1-2 drinks daily raises BP

Physical inactivity
Regular exercise can lower systolic BP by 4-9 mmHg

Dietary sodium
High intake increased blood pressure in salt-senstivite individuals

Low potassium intake
Inadequate potassium can increase BP

Background

Secondary Hypertension

Definition

Hypertension with an identifiable underlying cause, as opposed to primary (essential) hypertension.

Prevalence

Accounts for approximately 5-10% of all hypertension cases
Higher prevalence in certain populations:
Children and young adults
Patients with resistant hypertension
Patients with severe or sudden-onset hypertension

When to Suspect

Onset before age 30 or after age 55
Sudden worsening of previously controlled hypertension
Resistant hypertension (BP ≥130/80 mmHg despite 3+ medications)
Presence of clinical clues suggesting specific causes

Common Causes of Secondary Hypertension

Renal Causes

Renal parenchymal disease
Renovascular disease (renal artery stenosis)
Renin-producing tumors

Endocrine Causes

Primary aldosteronism
Cushing's syndrome
Pheochromocytoma
Thyroid disorders (hypo/hyperthyroidism)
Hyperparathyroidism

Other Causes

Obstructive sleep apnea
Coarctation of the aorta
Pregnancy-related (preeclampsia)
Medications and substances

Medication-Induced Hypertension

NSAIDs
Glucocorticoids
Erythropoietin
Oral contraceptives
Sympathomimetics
Cyclosporine

Background

White Coat Hypertension

Definition

Elevated office BP (≥130/80mmHg) with normal out-of-office BP (<130/80 mmHg) in untreated patients

White Coat Effect

Elevated office BP above goal with normal out-of-office BP at/below goal in treated patients

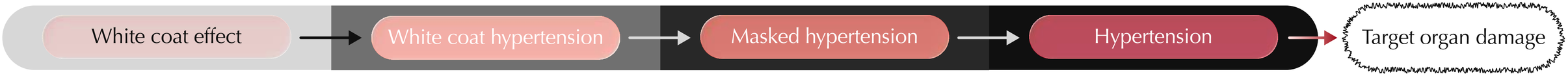
Masked Hypertension

Definition

Normal office BP (<130/80 mmHg) with elevated out-of-office BP (≥130/80 mmHg) in untreated patients

Masked Uncontrolled Hypertension

Normal office BP at/below goal with elevated out-of-office BP above goal in treated patients



Diagnostic Methods

Ambulatory BP Monitoring (ABPM) : Gold standard
Self-Measured BP (SMBP) : 12-14 readings over 1 week

Who to Evaluate

For white coat: Untreated patients with elevated office BP
For masked: Office BP ≤10 mmHg below goal or high CV risk

Background

Stage 1 Hypertension: **No Comorbidities**

Patient Profile

-No history of cardiovascular disease (CVD)
-10-year ASCVD risk <10%

Primary Recommendation:

-Lifestyle Modifications
-Drug therapy is not immediately indicated

Focus on nonpharmacologic interventions

-Weight loss
-DASH diet (rich in fruits, vegetables)
-Sodium reduction (<1500 mg/day)
-Regular physical activity
-Limited alcohol consumption

Stage 1 Hypertension: **With High CV Risk**

Patient Profile

- Established clinical CVD (e.g., coronary artery disease, stroke)
- High 10-year ASCVD risk (≥10%)
- Comorbidities like CKD, DM, HF

Primary Recommendation:

Monotherapy : Initiate treatment with a single antihypertensive agent

First-line choices

-ACE Inhibitors (ACEi) or Angiotensin II Receptor Blockers (ARBs)
-Calcium Channel Blockers (DHP CCBs)
-Thiazide-like Diuretics (e.g., Chlorthalidone)

Goal

Achieve target blood pressure while monitoring for side effects

Background

Stage 2 Hypertension: Initial Treatment

Recommendation:

Combination therapy with two drugs from different classes is recommended
-This approach is more effective at lowering BP and increases the likelihood of reaching the target sooner

Prefer Combination

A ACEi or ARB

+

C CCB(Dihydropyridine)

Alternative Combination

A ACEi or ARB

+

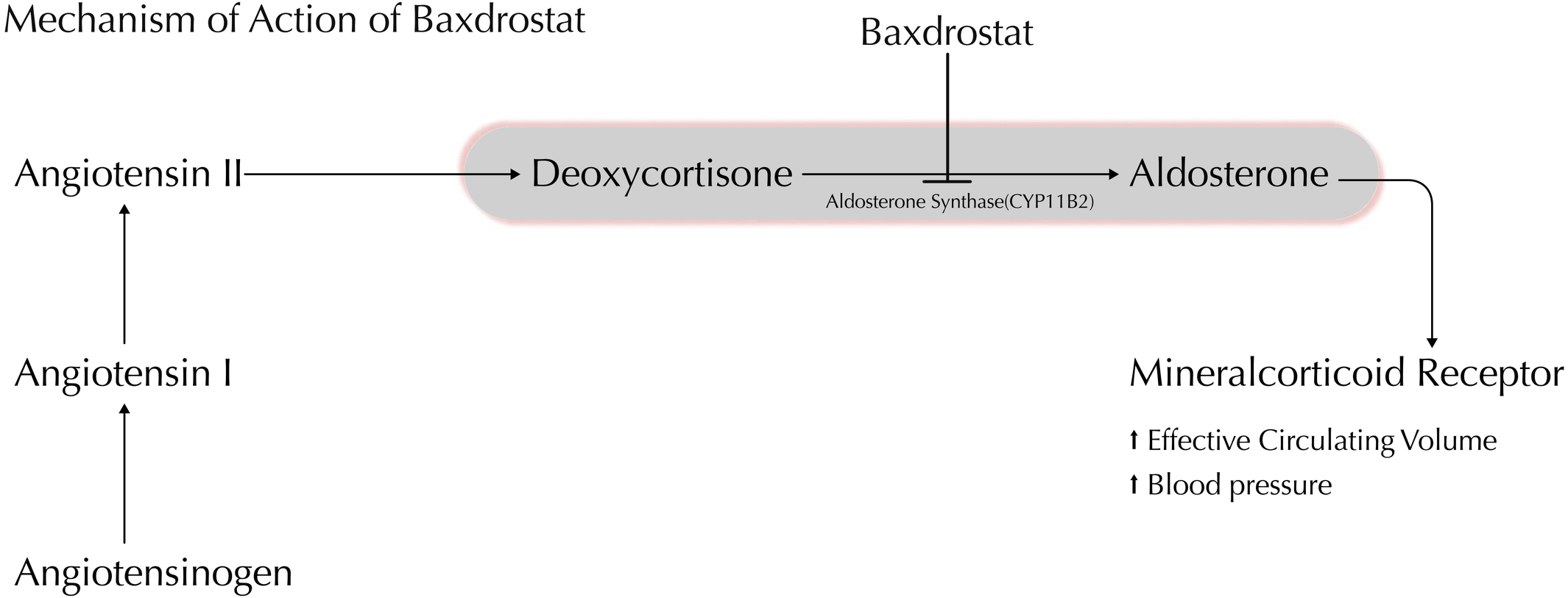
D Diuretics(Thiazid-like)

Best Practice

Use a single-pill combination (SPC) to improve medication adherence

Background

Mechanism of Action of Baxdrostat



Background

Mechanism of Action of Baxdrostat

Target Enzyme

Aldosterone Synthase(CYP11B2)

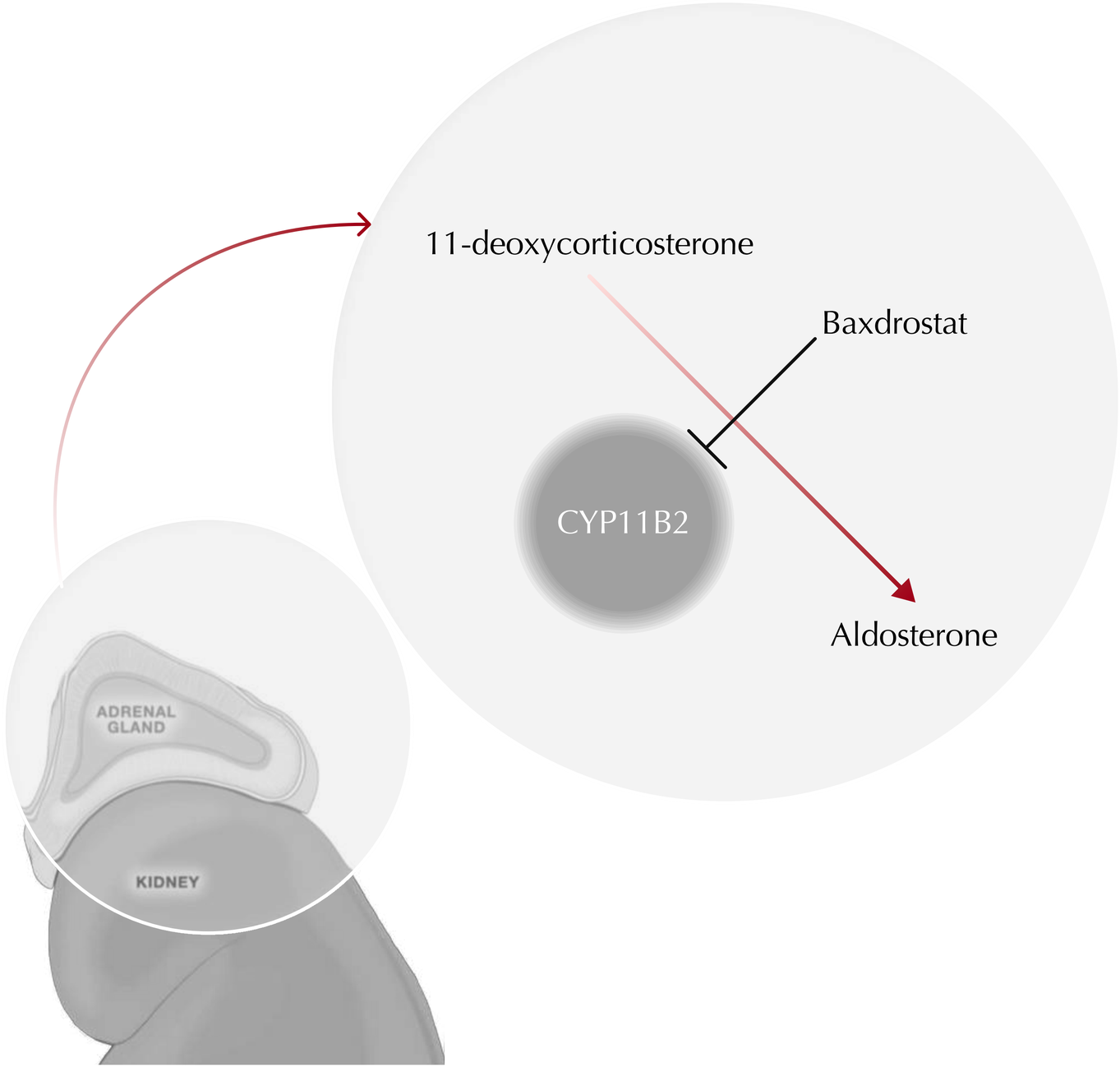
Catalyzes final steps in aldosterone biosynthesis
Located in zona glomerulosa of adrenal cortex

Inhibition Mechanism

Competitive inhibition of CYP11B2 active site
Blocks conversion of 11-deoxycorticosterone to aldosterone
Prevents binding of natural substrates

Selectivity Profile

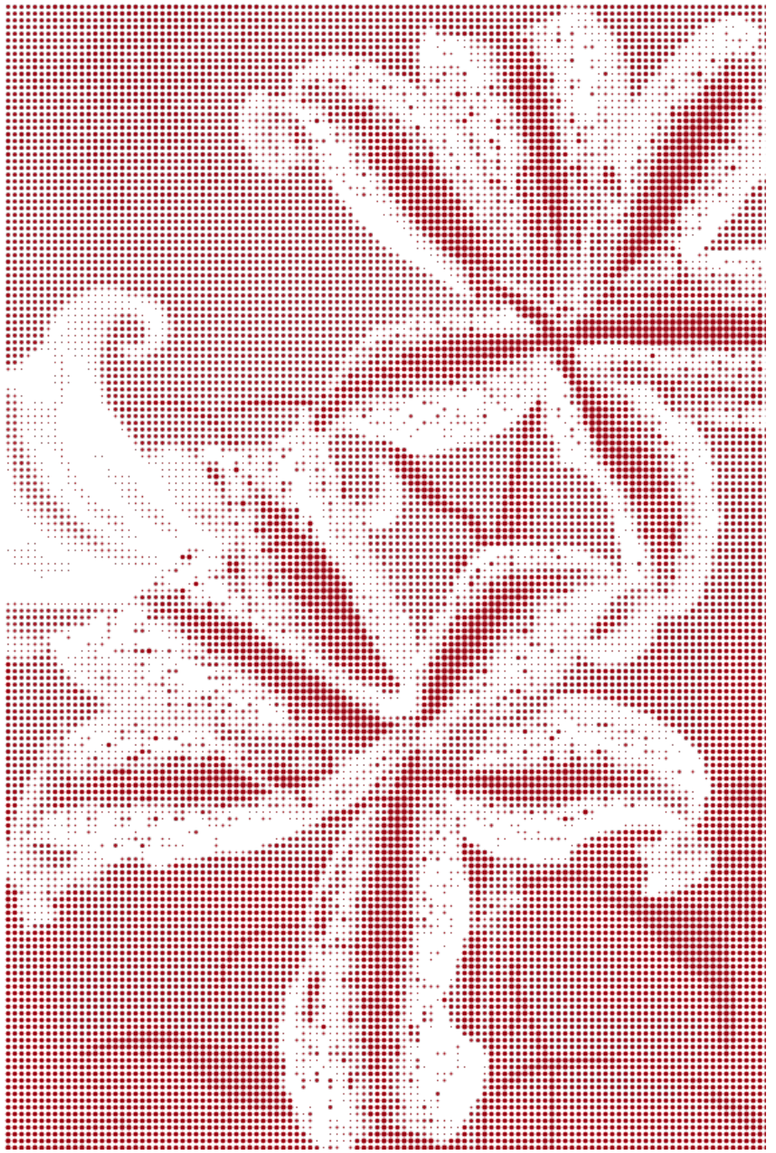
High selectivity for CYP11B2 over CYP11B1 (>100-fold)
Minimal impact on cortisol synthesis pathway
Avoids adrenocortical insufficiency



Method & Results

In this multicenter, placebo-controlled trial, we randomly assigned patients who had treatment-resistant hypertension, with blood pressure of 130/80 mm Hg or higher, and who were receiving stable doses of at least three antihypertensive agents, including a diuretic, to receive baxdrostat (0.5 mg, 1 mg, or 2 mg) once daily for 12 weeks or placebo.

The primary end point was the change in systolic blood pressure from baseline to week 12 in each baxdrostat group as compared with the placebo group.



Method & Results

Phase I Clinical Trial Design

Study Design

Randomized, double-blind, controlled trial

Study Population:

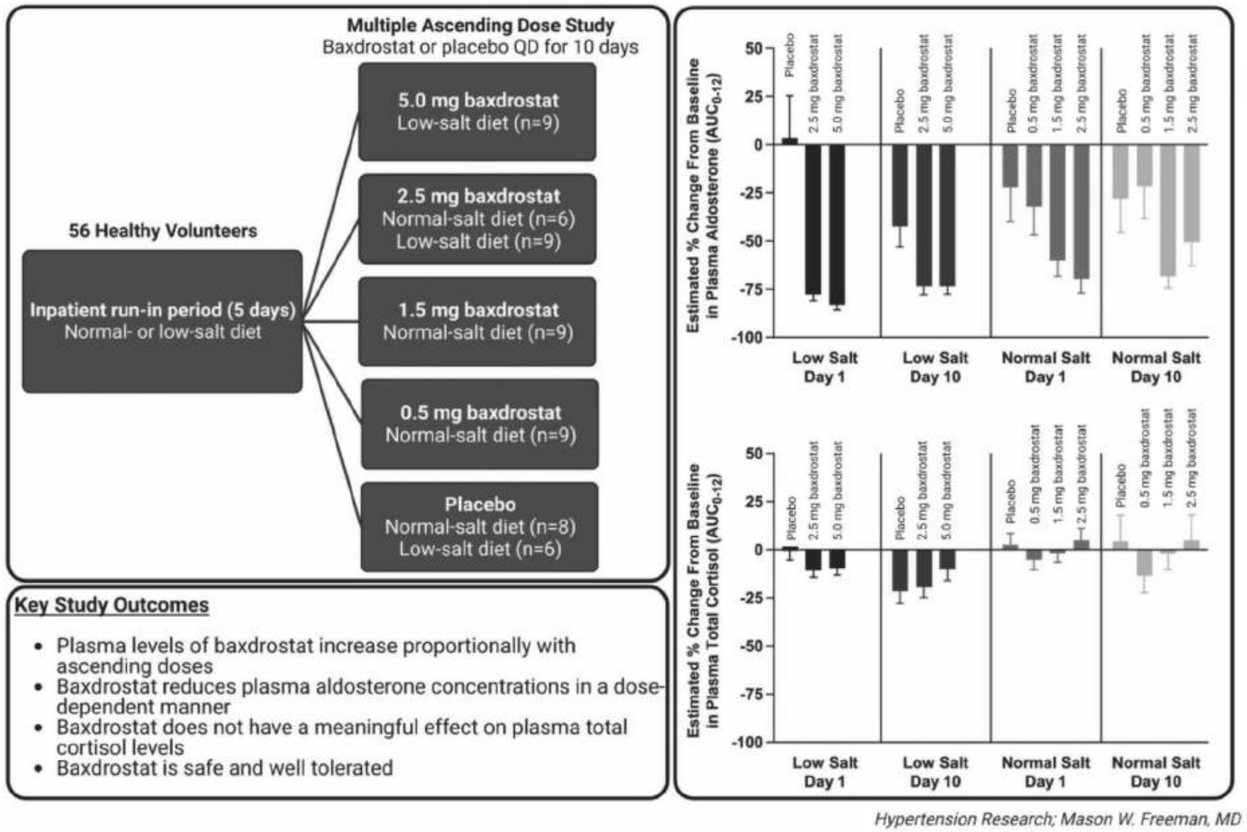
-56 healthy adults
-Ages 18-55
-Divided into normal or low salt diet groups

Dosing

-Varying doses of Baxdrostat (0.5 to 5mg)
-Once daily for 10 days

Primary Objectives

-Assess safety and tolerability of Baxdrostat
-Evaluate potential for blood pressure control
-Determine pharmacokinetic properties



Hypertension Research; Mason W. Freeman, MD

Phase 1 Trial Significance

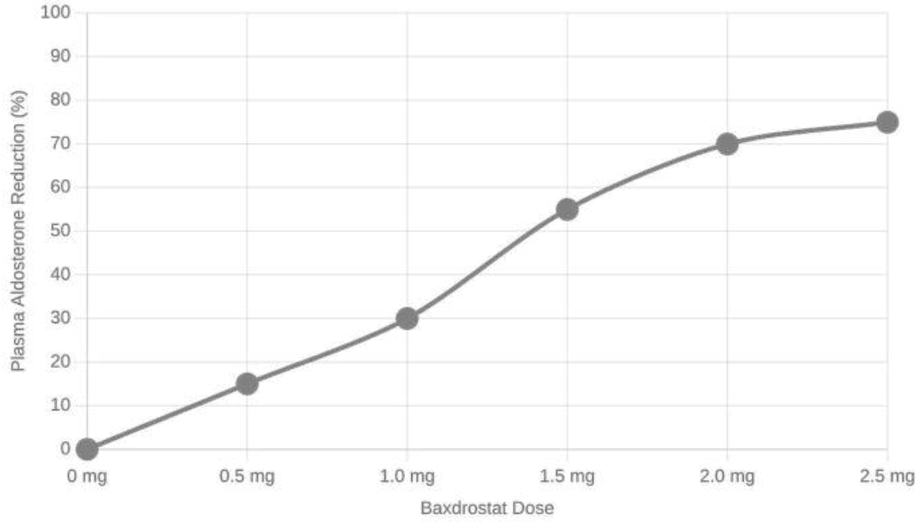
This was the first human trial of Baxdrostat, designed to establish its safety profile and determine the optimal dosing for subsequent Phase 2 trials in patients with treatment-resistant hypertension.

Method & Results

Phase I Clinical Trial Results

Key Findings on Plasma Aldosterone

-Dose-dependent reduction in plasma aldosterone
-Significant reduction observed at doses ≥1.5 mg
-Effect evident from first day of administration
-Persistent aldosterone-lowering effect throughout study



Study Population

56	18-55	10
Healthy Adults	Age Range	Days of Treatment

Safety Profile

No meaningful effect on plasma cortisol or urine free cortisol, demonstrating selectivity for aldosterone synthase

Pharmacokinetics

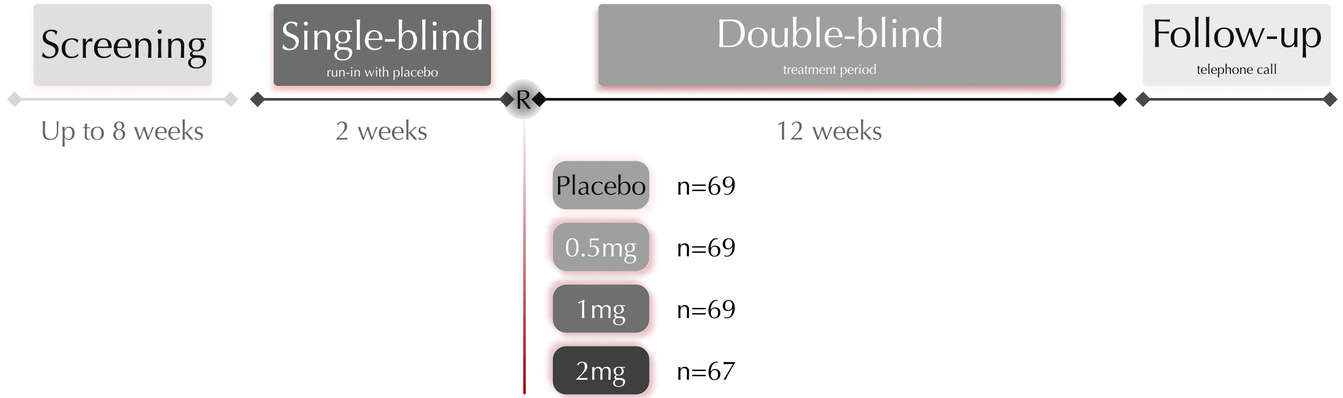
Tmax reached ~4 hours after administration; half-life of 26-31 hours (suitable for once-daily dosing)

Cardiovascular Effects

Decrease in orthostatic blood pressure and moderate increase in orthostatic heart

Method & Results

Phase II Clinical Trial Design



Study Design

Randomized, double-blind, placebo-controlled trial

Study Population:

248 patients with treatment-resistant hypertension

Key Inclusion Criteria

-Patients receiving stable doses of at least 3 antihypertensive medications
-One medication must be a diuretic
-Persistently high blood pressure despite treatment

Dosing Groups

0.5mg 1mg 2mg Placebo Once-daily

Method & Results

Phase II Clinical Trial Design

Cardiac Conditions

-Family history of long QT syndrome
-Heart block
-Complex ventricular arrhythmias
-History of sudden death

Baseline Laboratory Abnormalities

-Baseline hyperkalemia (elevated serum potassium)
-Estimated glomerular filtration rate (GFR) < 45 ml/min/1.73 m^2

Blood Pressure Thresholds

-Mean seated systolic BP ≥ 180 mm Hg
-Mean seated diastolic BP ≥ 110 mm Hg

Metabolic Conditions

Uncontrolled diabetes mellitus

Medication Restrictions

-Current use of mineralocorticoid receptor antagonists (MRAs) (e.g., spironolactone, eplerenone)
-Current use of potassium-sparing diuretics
-Patients on MRAs or potassium-sparing diuretics must discontinue for at least 4 weeks before randomization

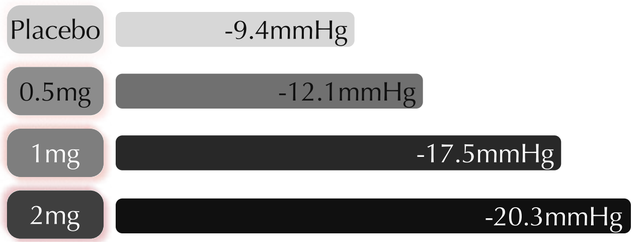
Method & Results

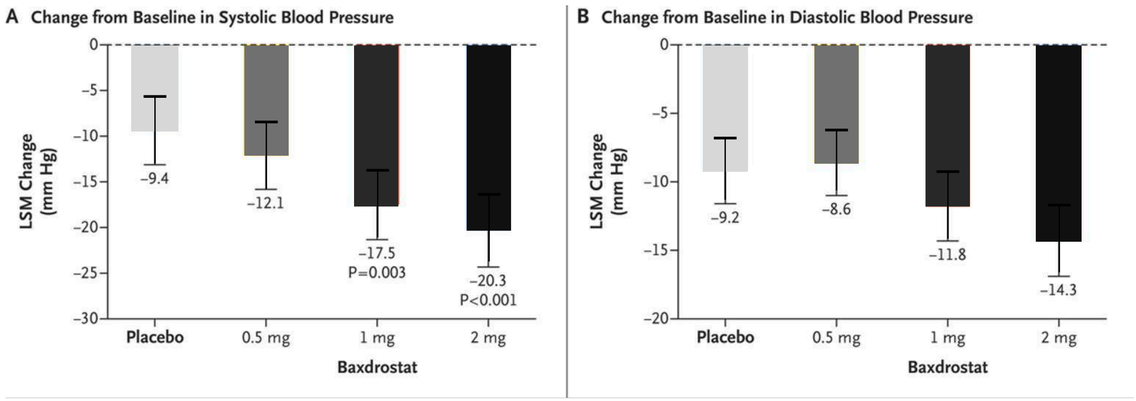
Phase II BrigHTN Trial Results - Efficacy

Primary Endpoint

Change in systolic blood pressure from baseline to week 12

Systolic Blood Pressure Reduction





Secondary Endpoints

-Diastolic BP reduction significant at 2 mg dose
-Dose-dependent decrease in urinary aldosterone
-Dose-dependent increase in plasma renin

Key Conclusion

Baxdrostat demonstrated a dose-dependent reduction in blood pressure in patients with treatment-resistant hypertension, highlighting the role of aldosterone in this condition.

Method & Results

Phase II BrigHTN Trial Results - Biomarkers

Key Biomarker Changes:

The Phase II trial demonstrated significant dose-dependent effects on key biomarkers related to the RAAS pathway.

Urinary Aldosterone:

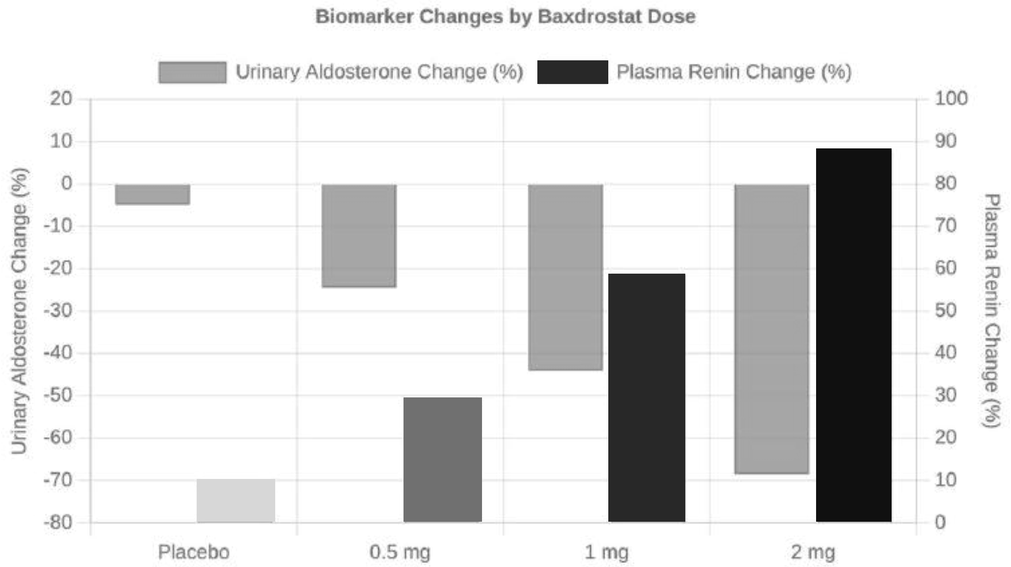
-Dose-dependent decrease across all Baxdrostat groups
-Greatest reduction observed in 2mg group

Plasma Renin:

-Dose-dependent increase across all Baxdrostat groups
-Compensatory response to aldosterone reduction

Implications:

-Confirms aldosterone's role in treatment-resistant hypertension
-Validates Baxdrostat's mechanism of action
-Supports dose selection for Phase 3 trials



Method & Results

Conclusions and Future Directions

Key Findings from Clinical Trials:

Clinical trials have not shown any deaths or serious adverse events associated with Baxdrostat.

Common Adverse Effects:

-Urinary tract infections

-Hyperkalemia

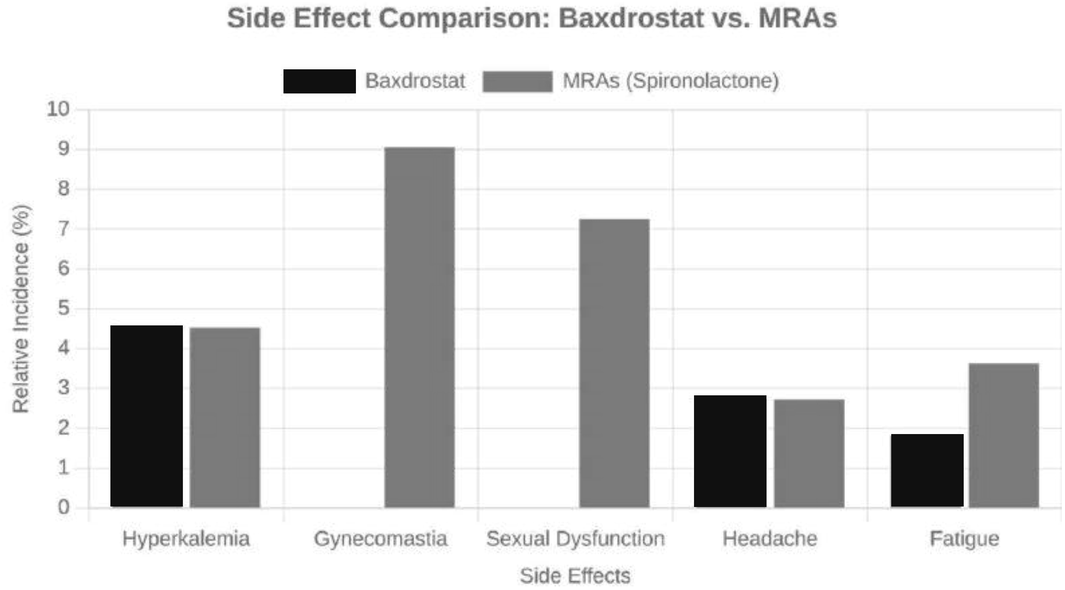
-Headache

-Fatigue

Note: Investigators concluded these findings were unlikely caused by the drug itself.

Hyperkalemia Considerations:

aldosterone inhibition. Patients who developed hyperkalemia did not experience cardiac arrhythmias and were able to resume medications without further complications



Method & Results

The BaxHTN Phase III trial had three key components

Primary Endpoint Assessment

12-week double-blind, placebo-controlled period
796 patients randomized 1:1:1 to receive:

1mg | 2mg | Placebo

Primary efficacy endpoint

Difference in mean change from baseline in seated SBP at Week 12 between baxdrostat groups and placebo

Long-term Safety Assessment

Safety evaluated at the end of 52 weeks

Comparison to standard of care arm

Monitoring for adverse events and long-term effects

Persistence of Efficacy

-Randomized withdrawal period from week 24 to week 32

-SBP at the end of 8 weeks compared between groups

~300 patients on baxdrostat 2mg were re-randomized 2:1 to:
- Continue baxdrostat 2mg
- Switch to placebo

NOTE

This comprehensive trial design allows for evaluation of both short-term efficacy, durability of response, and long-term safety of baxdrostat in patients with uncontrolled or treatment-resistant hypertension.

Discussion

Our trial showed substantial decreases in blood pressure when patients with treatment-resistant hypertension who were receiving stable doses of at least three antihypertensive medications also received the selective aldosterone synthase inhibitor baxdrostat.

The reduction in blood pressure was associated with a decrease in the plasma aldosterone level and a compensatory increase in plasma renin activity, without a reduction in the cortisol level. Baxdrostat generally had an acceptable side-effect profile, and none of the patients discontinued the trial because of hyperkalemia.

Discussion

Study Design Limitations

Combination Therapy Only

Baxdrostat was tested only as an add-on to existing antihypertensive regimens, not as monotherapy

Short Duration

The study measured drug response over only 12 weeks, limiting insights into long-term efficacy and safety

Sample Size

While adequate for a Phase 2 trial, the relatively small sample size (248 patients) limits generalizability

Population Considerations

Limited diversity in study population
Exclusion of patients with severe comorbidities
Unclear efficacy in special populations (elderly, different ethnic groups)

Methodological Considerations

-Primary focus on surrogate endpoint (blood pressure reduction) rather than clinical outcomes
-Limited assessment of drug interactions
-Potential placebo effect influence on results
-Lack of direct comparison with mineralocorticoid receptor antagonists (MRAs)

Discussion

Remaining Questions and Future Research Directions

Long-term Efficacy and Safety

Cardiovascular Outcomes

Does Baxdrostat reduce major adverse cardiovascular events beyond blood pressure reduction?

Sustained Effect

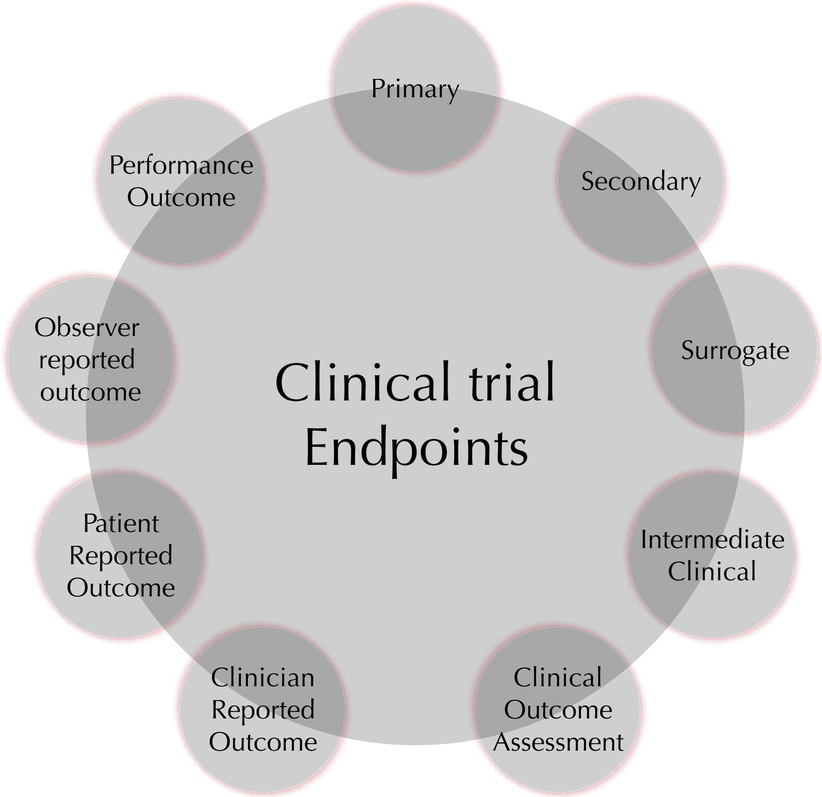
Is the blood pressure-lowering effect maintained over years of treatment?

Safety Profile

What is the long-term safety, particularly regarding hyperkalemia risk and adrenal function?

Optimal Clinical Use

What is the optimal dosing strategy across different patient populations?
Which patients benefit most from Baxdrostat therapy?
How should Baxdrostat be integrated into treatment algorithms?



Comparative Effectiveness

How does Baxdrostat compare to MRAs in efficacy and safety?
Is there a role for combination therapy with MRAs?
Cost-effectiveness compared to existing therapies
Potential applications beyond resistant hypertension (primary aldosteronism, heart failure, CKD)

Apprisal

Apprisal

Question 1: Did the study address a clearly formulated research question?

■ YES ☐ NO ☐ CAN'T TELL

The study clearly formulated its research question in terms of:

P — Population: Patients with treatment-resistant hypertension

I — Intervention: Baxdrostat (0.5 mg, 1 mg, or 2 mg)

C — Comparator: Placebo

O — Outcome: Change in systolic blood pressure

Assessment

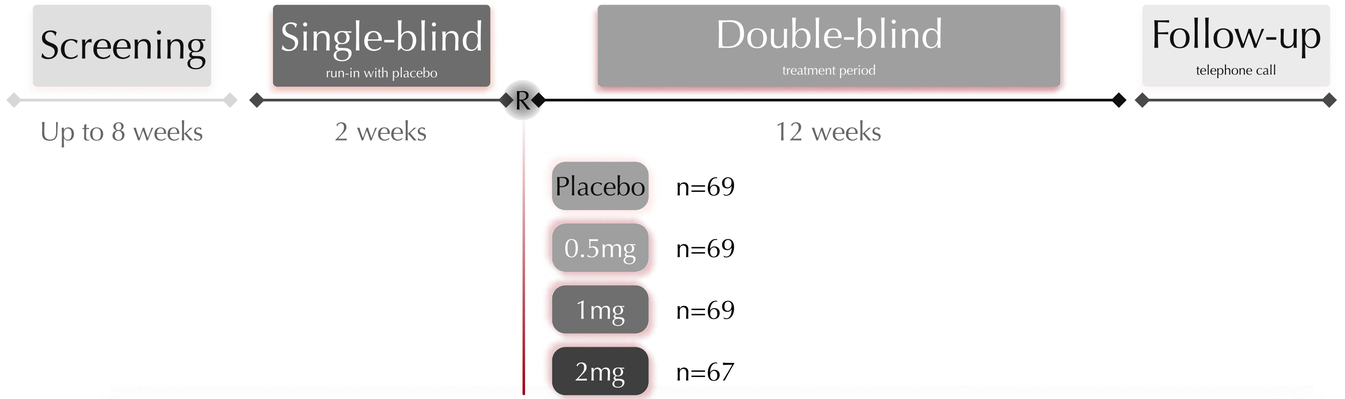
The study aimed to assess a specific intervention in a well-defined population
The primary endpoint was clearly specified

Apprisal

Question 2: Was the assignment of participants to interventions randomised?

■ YES ☐ NO ☐ CAN'T TELL

"In this multicenter, placebo-controlled trial, we **randomly assigned patients who had treatment-resistant hypertension, with blood pressure of 130/80 mm Hg or higher, and who were receiving stable doses of at least three antihypertensive agents, including a diuretic, to receive baxdrostat (0.5 mg, 1 mg, or 2 mg) once daily for 12 weeks or placebo**. The primary end point was the change in systolic blood pressure from baseline to week 12 in each baxdrostat group as compared with the placebo group."



Assessment

-Appropriate randomization method was used
-Stratification helped balance important prognostic factors
-Randomization was sufficient to eliminate systematic bias
-Allocation sequence was concealed from investigators and participants

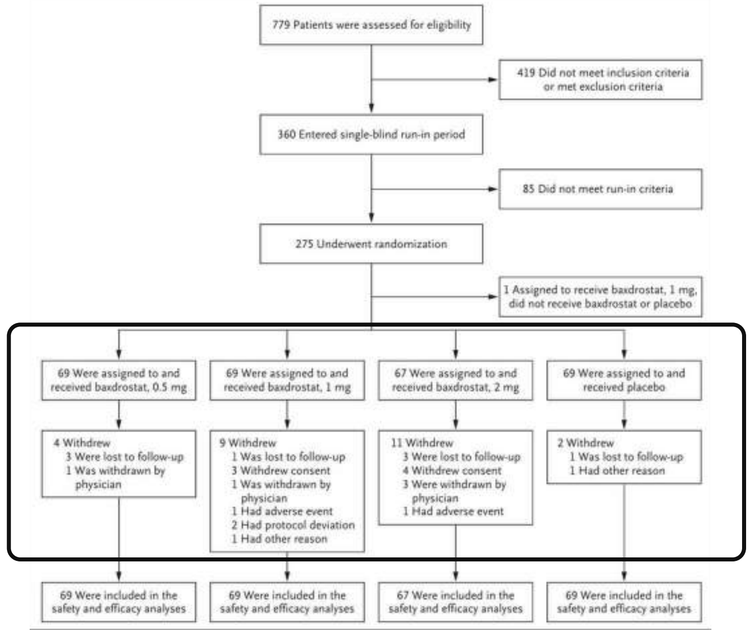
Apprisal

Question 3: Were all participants who entered the study accounted for at its conclusion?

■ YES ☐ NO ☐ CAN'T TELL

275	26	248
Total randomized	WIthdrawals	Completed trial

(4 from 0.5 mg, 9 from 1 mg, 11 from 2 mg, 2 from placebo)



Analysis

Modified intention-to-treat population (all who received at least one dose)

Assessment

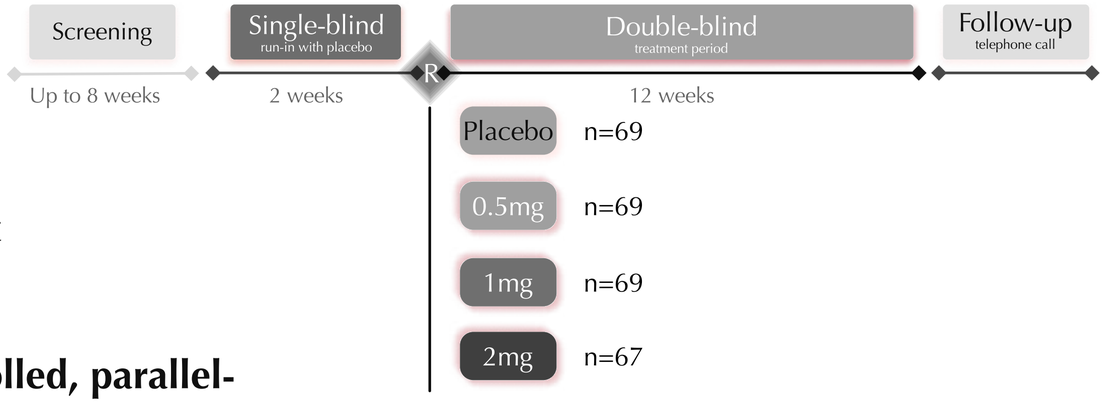
All losses to follow-up and exclusions after randomization were accounted for

The study used a modified intention-to-treat analysis

Apprisal

Question 4: Were participants, investigators, and outcome assessors 'blind' to treatment?

■ YES ☐ NO ☐ CAN'T TELL



Strengths of blinding:

Double-blind design protected against investigator bias in assessment

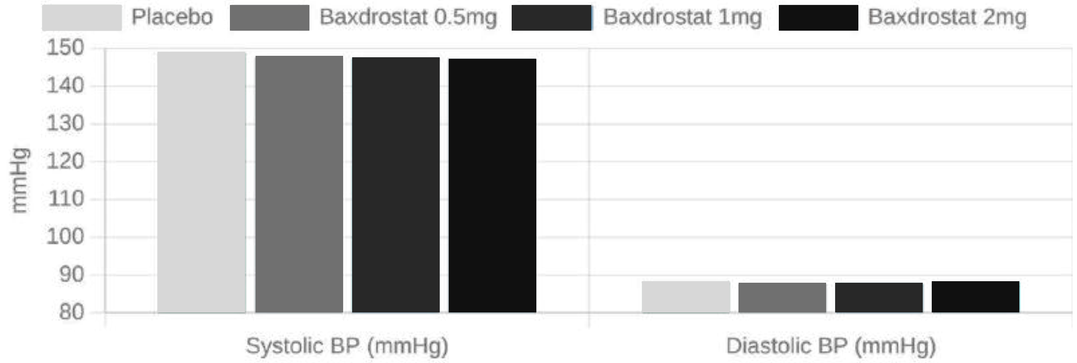
" BrigHTN, a multicenter, **randomized, double-blind, placebo-controlled, parallel-group, dose-ranging trial, had an adaptive design**. The trial enrolled men and women who were 18 years of age or older, were receiving stable doses of at least three antihypertensive medications (one of which was a diuretic), and had a mean blood pressure of at least 130/80 mm Hg while seated. Blood pressure was determined as the average of three measurements obtained with the use of an automated in-office blood-pressure monitor."

Apprisal

Question 5: Were the study groups similar at the start of the randomised controlled trial?

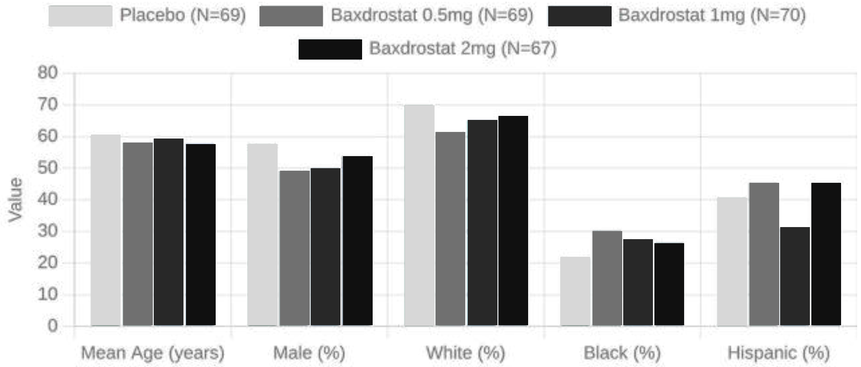
■ YES ☐ NO ☐ CAN'T TELL

Blood Pressure at Baseline

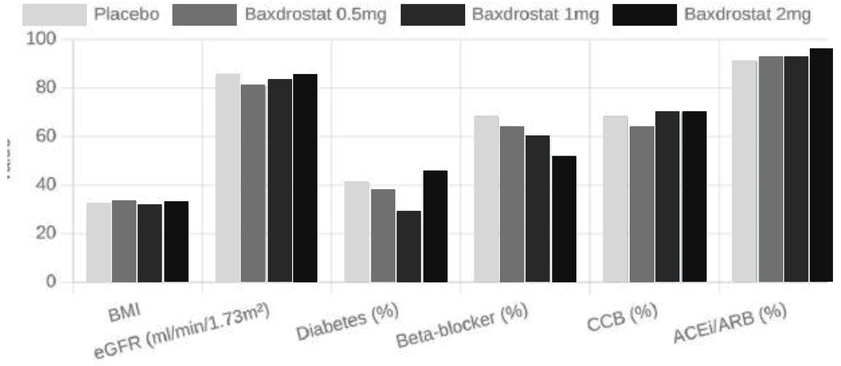


"A total of 779 patients underwent screening, and 360 were included in the placebo run-in period. A total of 275 patients were randomly assigned to receive once-daily baxdrostat at a dose of 0.5 mg (69 patients), 1 mg (70 patients), or 2 mg (67 patients), or placebo (69 patients). One patient who was randomly assigned to receive baxdrostat in the 1-mg group never received the drug, so the modified intention-to-treat population included 274 patients. **The trial groups were similar with respect to demographic and clinical characteristics at baseline**. The trial population was predominantly White (70%). Black patients constituted a higher percentage of patients in the trial (28%) than their proportionate representation in the U.S. population . The trial included a sizable percentage of patients who identified as Hispanic and a small percentage who identified as Asian"

Demographic Characteristics



Clinical Characteristics



Apprisal

Question 6: Apart from the experimental intervention, did each study group receive the same level of care?

■ YES ☐ NO ☐ CAN'T TELL

Protocol:

Clearly defined study protocol with standardized procedures

Background medications:

Patients continued their existing antihypertensive regimens

Follow-up intervals:

Same schedule for all groups (days 3, 8, 15, 22, 43, 64, and 85)

Assessments

Identical monitoring and safety evaluations across groups

"**The use of background medication was similar among the trial groups**. All the patients received a diuretic, 91 to 96% received an angiotensin-converting–enzyme inhibitor or angiotensin-receptor blocker, and 64 to 70% received a calcium-channel blocker"

Apprisal

Question 7: Were the effects of intervention reported comprehensively?

☐ YES ☐ NO ■ CAN'T TELL

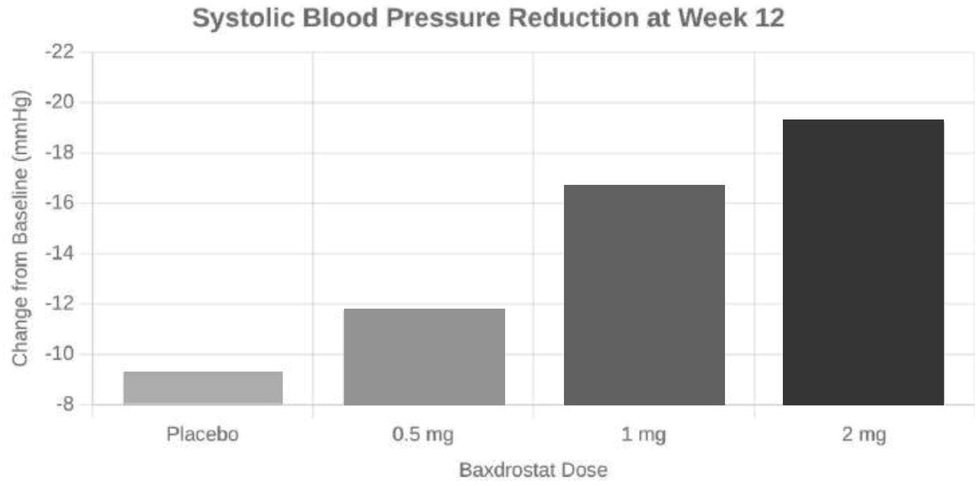


Table 2. Adverse Events That Occurred during the Treatment Period.

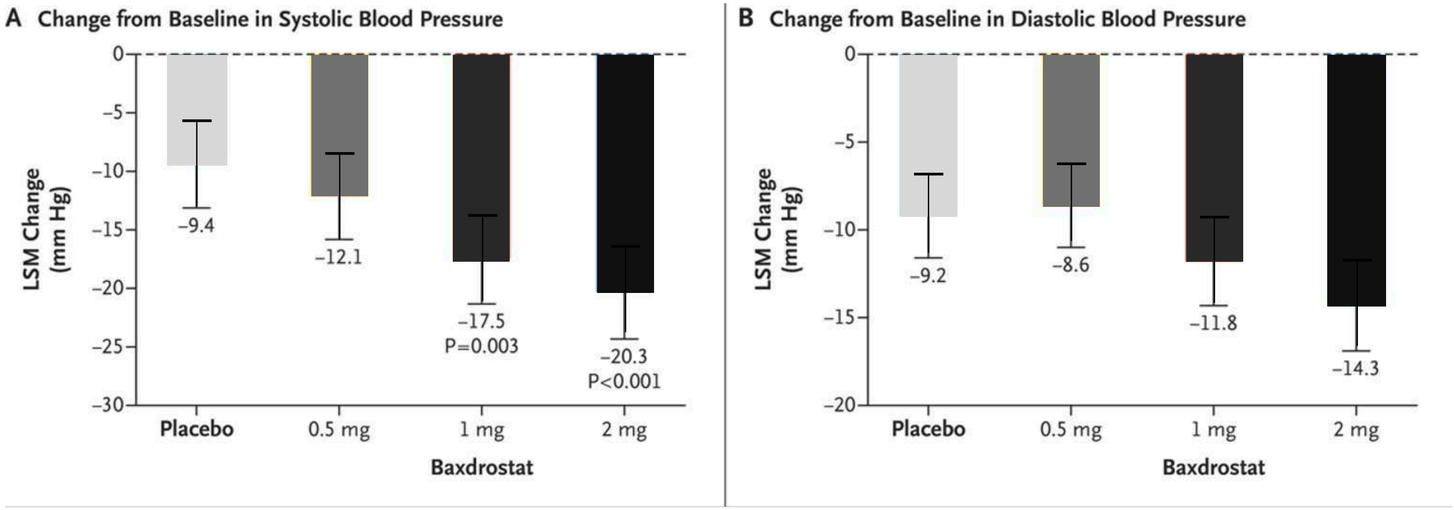
Event	Placebo (N=69)		Baxdrostat, 0.5 mg (N=69)		Baxdrostat, 1 mg (N=69)		Baxdrostat, 2 mg (N=67)	
	No. of Patients with Event (%)	No. of Events	No. of Patients with Event (%)	No. of Events	No. of Patients with Event (%)	No. of Events	No. of Patients with Event (%)	No. of Events
Any serious adverse event*	2 (3)	3	0	0	2 (3)	3	6 (9)	12
Any adverse event	28 (41)	50	24 (35)	38	36 (52)	77	32 (48)	67
Adverse event of special interest†	0	0	1 (1)	1	5 (7)	6	2 (3)	3
Hyponatremia	0	0	0	0	2 (3)	2	1 (2)	1
Hypotension	0	0	0	0	1 (1)	1	0	0
Potassium level ≥6.0 mmol/liter	0	0	0	0	2 (3)	2	1 (2)	1
Potassium level between 5.5 and 5.9 mmol/liter on at least two consecutive occasions‡	0	0	1 (1)	1	2 (3)	2	1 (2)	1

Apprisal

Question 8: Was the precision of the estimate of the intervention or treatment effect reported?

■ YES ☐ NO ☐ CAN'T TELL

95% confidence intervals (CIs) reported for all key endpoints



Apprisal

Question 9: Do the benefits of the experimental intervention outweigh the harms and costs?

☐ YES ☐ NO ■ CAN'T TELL

Benefits

-Significant reduction in systolic BP (-11.0 mmHg vs placebo at 2 mg dose)
-Dose-dependent effect showing clear pharmacological activity
-Potential alternative for patients who cannot tolerate mineralocorticoid receptor antagonists

Harms

-No serious adverse events attributed to baxdrostat
-No instances of adrenocortical insufficiency
-Hyperkalemia (K+ ≥6.0 mmol/L) in only 2 patients, resolved after temporary discontinuation

Assessment

While the trial provides detailed data on blood pressure effects, the lack of clinical endpoints makes it impossible to determine if the intervention comprehensively improves patient outcomes beyond surrogate markers.

Apprisal

Question 10: Can the results be applied to your local population/in your context?

☐ YES ☐ NO ■ CAN'T TELL

Racial/Ethnic Representation:

Seldom Asian individuals included in the trial population

Geographic Scope

Trial conducted entirely in the United States

"The trial population was predominantly **White (70%)**. **Black** patients constituted a higher percentage of patients in the trial **(28%)** than their proportionate representation in the U.S. population. The trial included a sizable percentage of patients who identified as **Hispanic (43%)** and a small percentage who identified as **Asian (2%)**"

Apprisal

Question 11: Would the experimental intervention provide greater value than existing interventions?

☐ YES ☐ NO ■ CAN'T TELL

Trial Limitations:

No Direct Comparison:

Not compared with spironolactone or other antihypertensives

Short Duration:

12-week Phase 2 trial, not designed for long-term assessment

Patient Selection:

Excluded eGFR < 45 ml/min/1.73 m^2

Future Needs:

Phase 3 trials required for long-term safety and efficacy

Apprisal

CASP Appraisal Summary: Baxdrostat Phase 2 Trial

V		I		P	
Clear Question	●	Comprehensive	◐	Benefits > Harms	◐
Randomized	●	Precise	●	Applicable Locally	◐
Loss to Follow-up	●			Clinical Result Value	◐
Blinding	●				
Baseline Characteristics	●				
Beyond Intervention	●				

● YES
◐ CAN'T TELL
○ NO

Comparison of MRAs and Baxdrostat

NAME	Spironolactone	Eplerenone	Finerenone	Baxdrostat
Drug Class	Steroidal MRA	Steroidal MRA	Non-Steroidal MRA	Aldosterone Synthase Inhibitor
Mechanism of Action	Blocks aldosterone by binding to mineralocorticoid receptor less selective	Selectively blocks aldosterone by binding to mineralocorticoid receptor	Highly selective non-steroidal MRA; unique MR binding	Inhibits aldosterone synthase (CYP11B2)
Pharmacokinetics	Metabolized in liver to active metabolites; renal excretion	Peak: 1.5h; Bioavailability: ~70%	Linear PK; half-life 2-3h; rapid absorption; satisfactory oral bioavailability	Limited published data

Comparison of MRAs and Baxdrostat

NAME	Spironolactone	Eplerenone	Finerenone	Baxdrostat
Efficacy	Greater antihypertensive effect in primary hyperaldosteronism	The antihypertensive efficacy is approximately 50–75% that of spironolactone	Reduced CKD progression and CV events in T2D; cardiac anti-fibrotic effects	8.1-11.0 mmHg SBP reduction in resistant hypertension
Potency	Effective in decreasing BP, especially in abdominal obesity	50-75% as potent as spironolactone as an antimineralocorticoid	High potency and selectivity	Highly selective and potent aldosterone synthase inhibitor
Side Effects	Gynecomastia, menstrual irregularities, hyperkalemia	Hyperkalemia, fewer endocrine-related effects	Hyperkalemia (less than steroidal MRAs)	Limited long-term safety data

Comparison of MRAs and Baxdrostat

NAME	Spironolactone	Eplerenone	Finerenone	Baxdrostat
Taiwan NHI Price	1.50-2.00 NTD/tab	12.40 NTD/tab(25mg) 24.90 NTD/tab(50mg)	80 NTD/tab(self paid)	