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Hypercortisolism in Difficult-to-Control HTN and Diabetes Mellitus Type 2 (DM type 2)

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Catalyst Trial Overview and the Role of Mifepristone, a Competitive Glucocorticoid Receptor Antagonist, in Improving Diabetes Mellitus Type 2 Control.

The majority of incidentally discovered adrenal tumors are benign adrenocortical adenomas, and the prevalence of adrenocortical adenomas is around 1–7% on cross-sectional abdominal imaging. These can be non-functioning adrenal tumors or they can be associated with autonomous cortisol secretion on a spectrum that ranges from rare clinically overt adrenal Cushing syndrome to the much more prevalent mild autonomous cortisol secretion (MACS) without signs of Cushing syndrome. MACS is diagnosed (based on an abnormal overnight dexamethasone suppression test) in 20–50% of patients with adrenal adenomas. MACS is associated with cardiovascular morbidity, obesity, fragility fractures, decreased quality of life, HTN, and DM type 2 and increased mortality. Management of MACS should be individualized based on patient characteristics and may include adrenalectomy, medications that counteract increased cortisol secretion, or conservative follow-up with treatment of associated comorbidities. Identifying patients with MACS who are most likely to benefit from adrenalectomy or medications is challenging, as adrenalectomy or medications that counteract increased cortisol secretion result in improvement of cardiovascular risk factors, DM, etc., as per recent studies in patients with MACS. The new studies showed promising results in the treatment of uncontrolled DM type 2 with corticosteroid receptor blockers.

It surprised us how common MACS was: it was diagnosed in nearly half the patients with a benign adrenal incidentaloma. Notably, 70% of patients with MACS were women, most of whom were postmenopausal (over 50 years old).

Compared to those without MACS, it was found that patients with MACS were more likely to have hypertension and to need three or more anti-hypertensive medications to control their blood pressure. Among patients with type 2 diabetes, those with MACS were twice as likely to be insulin-dependent, suggesting that other medications haven't effectively managed their blood sugar levels. Analysis of urine steroids in patients with MACS, compared to those with normal 1mg-DST results, showed increased excretion of cortisol and related metabolites. In contrast, the excretion of androgen metabolites was decreased in patients with MACS. Since adrenal androgen production is stimulated by adrenocorticotropic hormone (ACTH), this likely relates to cortisol's negative feedback on the pituitary, reducing ACTH secretion. Only a minority of patients with adrenal incidentalomas are referred to an endocrinologist and undergo an optimal work-up to exclude MACS. Just one in six patients underwent a 1mg-DST in a recently published population-based study. If left undiagnosed, patients with MACS are at risk of developing adverse cardiometabolic consequences and presenting with poorly controlled hypertension and type 2 diabetes, as well as obesity and Osteopenia/ Osteoporosis. So-called Big 4. If patients have those four conditions, it is very reasonable to screen for MACE using a 1 mg Dexamethasone suppression test.

Considering that around two out of three of these patients are women, MACS is potentially a key contributor to women's metabolic health, particularly after menopause. MACS progresses to Cushing syndrome in less than 1% of patients. These are, according to current understanding, two distinct pathophysiological processes[1,2].

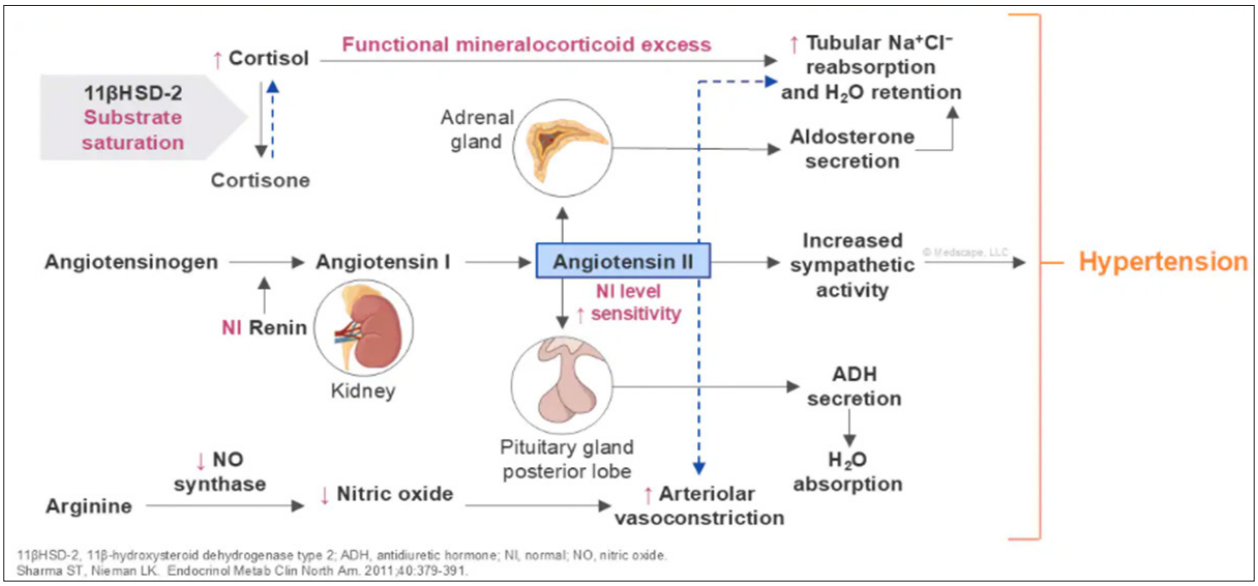
Certain Populations Have Higher Rates of Hypercortisolism

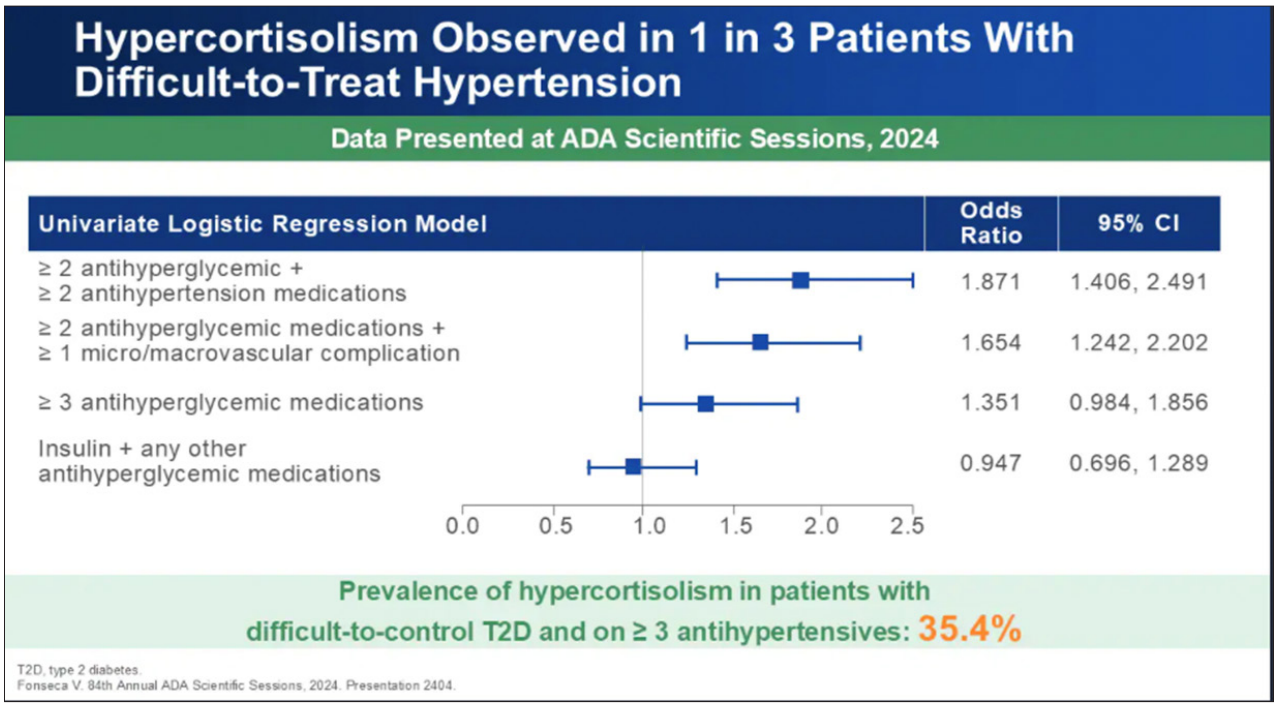
- While incidence of hypercortisolism in the general population is low, recent data suggest a higher prevalence in those with certain risk factors.
- Screening for hypercortisolism should occur in patients who have multiple risk factors
  - Increased pre-test probability of hypercortisolism
  - Better positive predictive value of the screen

- If pre-test probability for hypercortisolism is high, further evaluation is recommended even with normal results?

Pathogenesis of HTN in patients with MACS is discussed below[3].

Elevated Cortisol Pathophysiology

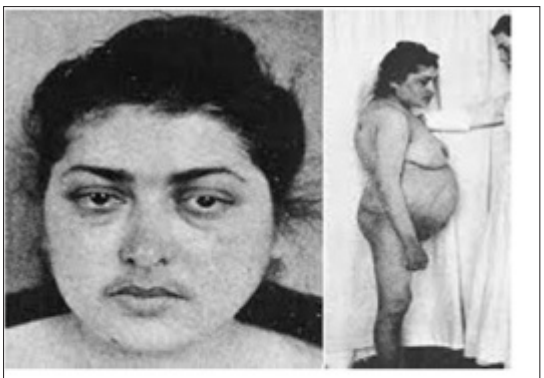




Hyperglycemia Secondary to Hypercortisolism: A Commonly Missed Diagnosis of Difficult- to-Treat T2DM

Some Patients with Hypercortisolism Present with Classic Phenotypic Features of Cushing Syndrome

- Easy bruising
- Facial plethora
- Proximal myopathy (or proximal muscle weakness)
- Striae (especially of reddish purple and >1 cm wide) Dorsocervical fat pad ("buffalo hump")
- Facial fullness
- Obesity
- Supraclavicular fullness
- Acne
- Hirsutism



Original publication: Bulletin of the Johns Hopkins Hospital, 1932. Reprint: *Obes Res*, 1994. Accessed November 21, 2022. doi:10.1002/j.1550-8528.1994.tb00097.x

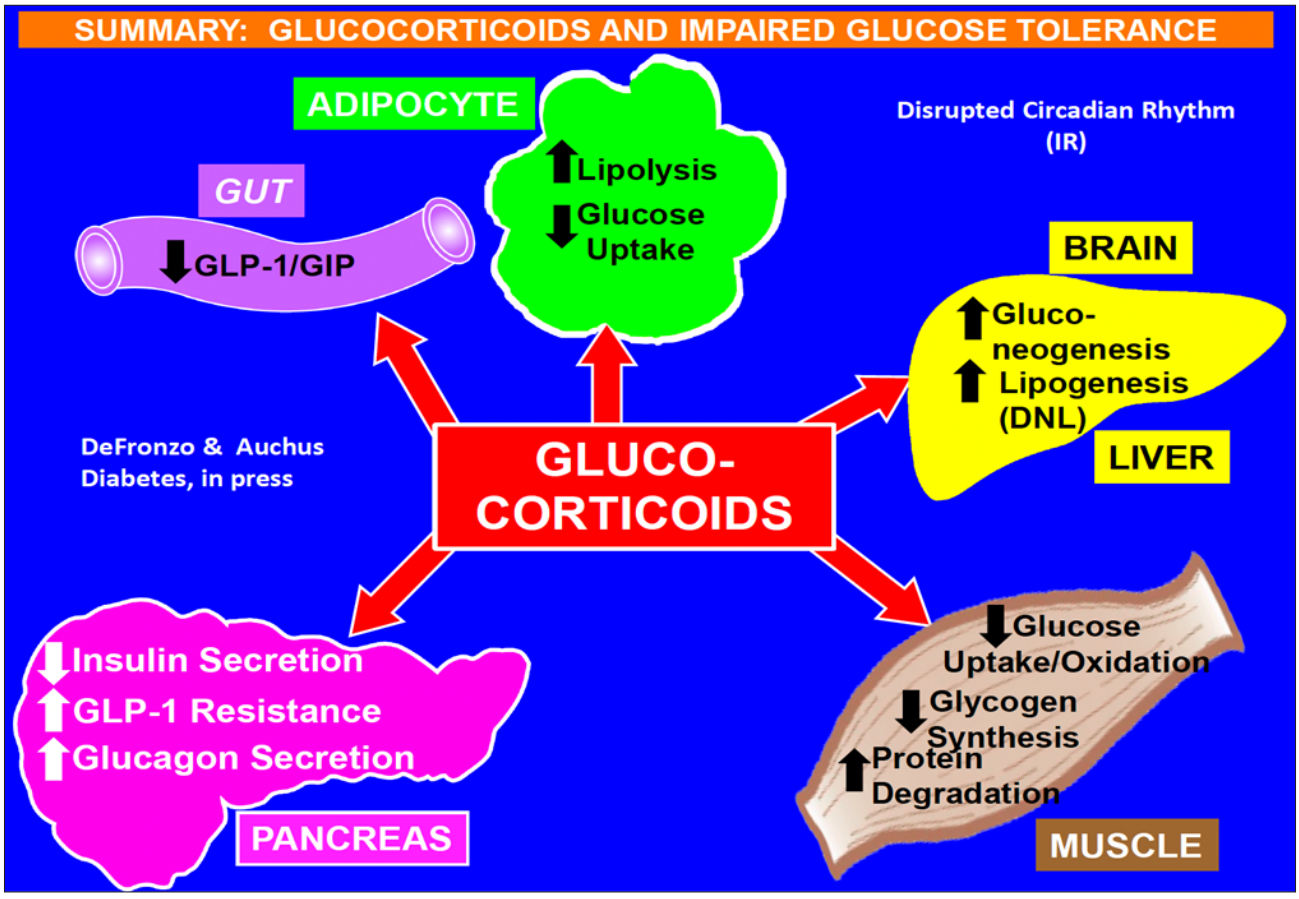
DTC = Difficult to Control

Most Individuals with Hypercortisolism Present without Classic Phenotypic Features: Big Four

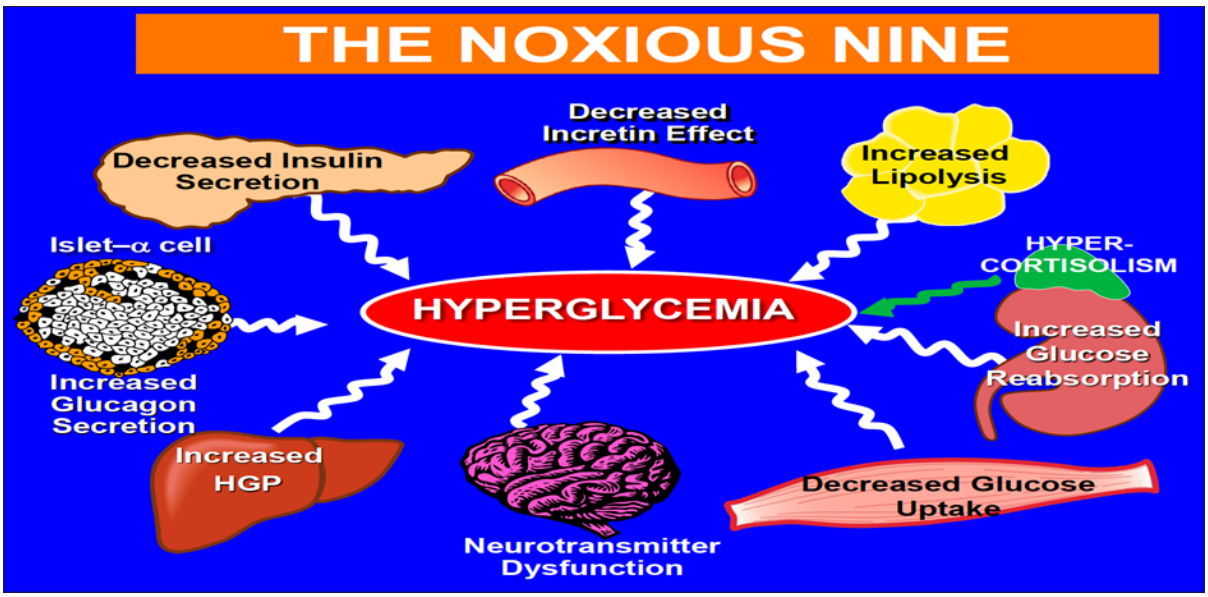
- Type 2 Diabetes (DTC)
- Hypertension (DTC)
- Obesity (visceral)
- Osteoporosis/
- Fractures

We are discussing MACS, which is a distinct process from Cushing syndrome (CS) and progresses to CS in only 0.27% of patients.

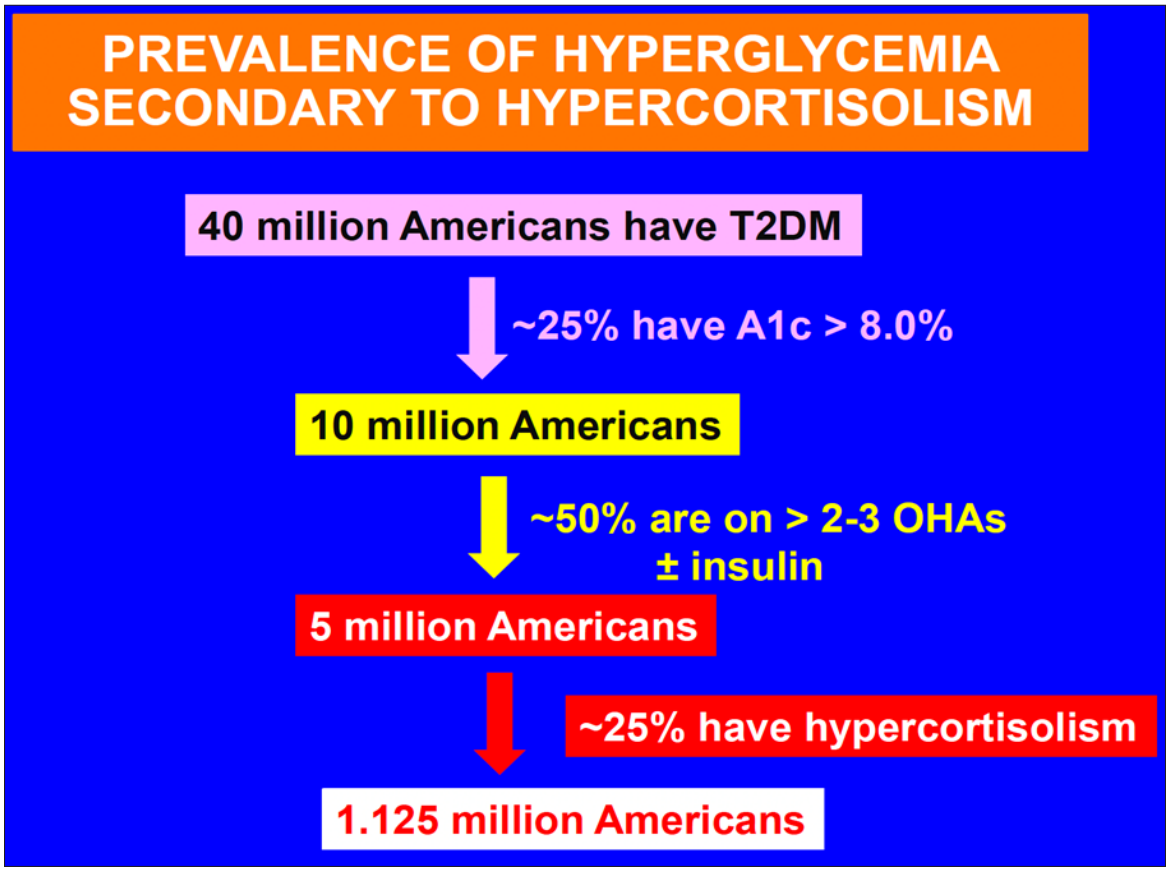
How Does Hypercortisolism cause Hyperglycemia?



With the addition of the Cortisol role, the pathogenesis of type 2 DM looks like having 9 pathogenetic arms- Ominous Octet



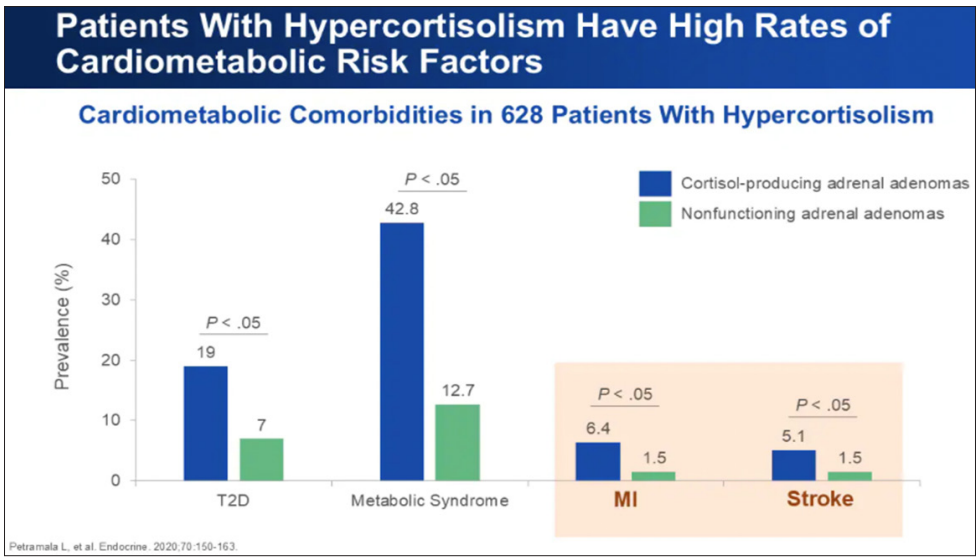
Further studies confirm the role of gut microbiota and decreased secretion of Amylin in the pathogenesis of DM type 2 so we can add 2 more pathogenetic mechanisms[4,5].



Hypercortisolism was not frequently screened in patients with difficult-to-control DM type 2 and HbA1c between 7.5% and 11.5% because they did not have classical physical signs suggestive of Cushing syndrome. Patients with hypercortisolism (MACS) have increased Cardiovascular, cardiometabolic, and adverse renal risk factors as discussed [6,7].

The patient was not overtly affected by Cushing's syndrome, but instead had MACS, which comprised the population of the CATALYST TRIAL. In the first part of the trial, the prevalence of hypercortisolism in difficult-to-control DM type 2 was discussed, and in the second part, whether the treatment of hypercortisolism with competitive glucocorticoid receptor antagonist Mifepristone can improve the control of DM type 2 [8,9].

Patients With Hypercortisolism Have High Rates of Cardiometabolic Risk Factors



Petramala L, et al. Endocrine. 2020;70:150-163.

What Happens If We Do Not Treat Hypercortisolism?

Elevated serum cortisol is associated with

- **Microalbuminuria** in T2D[1]
- All-cause **mortality in end-stage kidney disease**[2]
- **Endothelial dysfunction**[3]

Patients with **resistant hypertension** are at **increased risk for CV death, MI, and stroke**[4]

In patients with elevated cortisol, medical therapy did not improve blood pressure, whereas unilateral **adrenalectomy brought about significant reductions in CV abnormalities**[5]

CV, cardiovascular; MI, myocardial infarction.
1. Zhang X, M, et al. Int J Med Sci. 2020;17:2998-3004; Sagmeister MS, et al. Front Endocrinol (Lausanne). 2023;13:1075809; 3. Broadley AJ, et al. J Am Coll Cardiol. 2005;46:344-50; 4. Kumbhani DJ, et al. Eur

A Multidisciplinary Approach Is Important in Managing Patients With Hypercortisolism

Atypical Signs of Hypercortisolism[1,2]

T2D

- Difficult-to-control glucose
- Multiple antihyperglycemic medications

Hypertension

- Resistant hypertension
- Multiple antihypertensive medications

Include the nephrologist, as renal function can be negatively impacted[3,4]

Hypercortisolism may cause

- Podocyte damage
- Glomerulosclerosis
- Reduced eGFR

New Prevalence Data for Hypercortisolism: CATALYST Trial

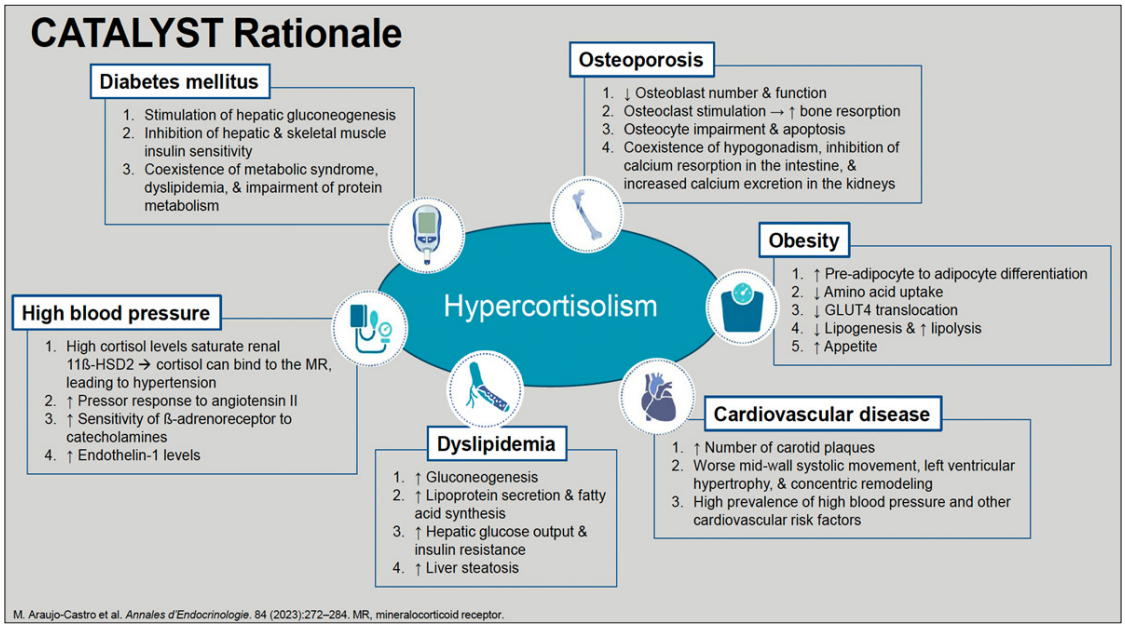
HyperCortisolism in PATients with Difficult-to-control Type 2 DiAbetes Despite Receiving Standard-of-care Therapies: PrevaLence and Treatment with KorlYm® (MifepriSTone)

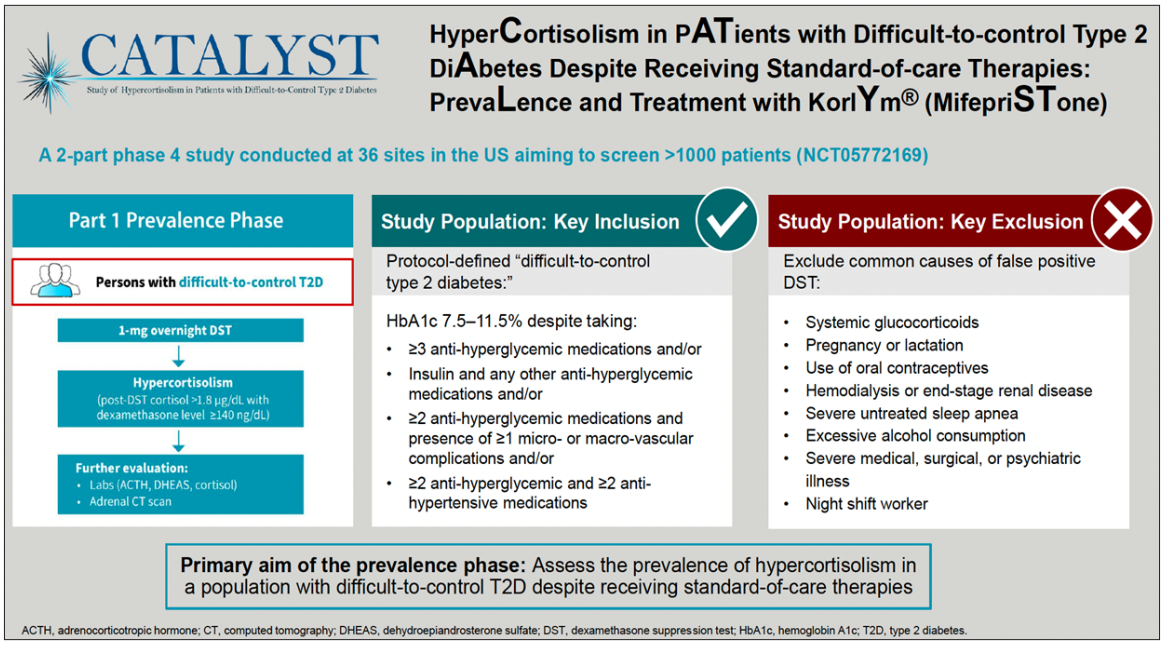
A 2-part, phase 4 study conducted in 36 sites in the United States to screen >1000 patients

Part 1 Aim: provide a robust estimate of the prevalence of hypercortisolism among patients with difficult-to-control T2D

- Endogenous hypercortisolism is a potential underlying driver of T2D

- **Hypercortisolism** is a recognized contributor to type 2 diabetes, metabolic syndrome, and cardiometabolic risk
- **Part 1:** What is the prevalence of hypercortisolism in individuals with difficult-to-control type 2 diabetes?
- **Part 2:** Can treatment with a cortisol-directed therapy improve glycemia in patients with type 2 diabetes?

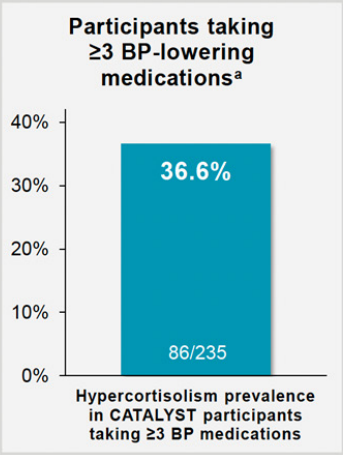




Screening Phase

Population of Interest: 36.6% of Those Taking ≥3 Antihypertensives Had Hypercortisolism

- **22.2%** of CATALYST participants were taking ≥3 blood pressure-lowering medications (235/1057)
  - Among those, the prevalence of hypercortisolism was **36.6%**
- The odds of having hypercortisolism were **2× as high** in those taking ≥3 blood pressure-lowering medications (OR 2.281, 95% CI 1.66, 3.127)

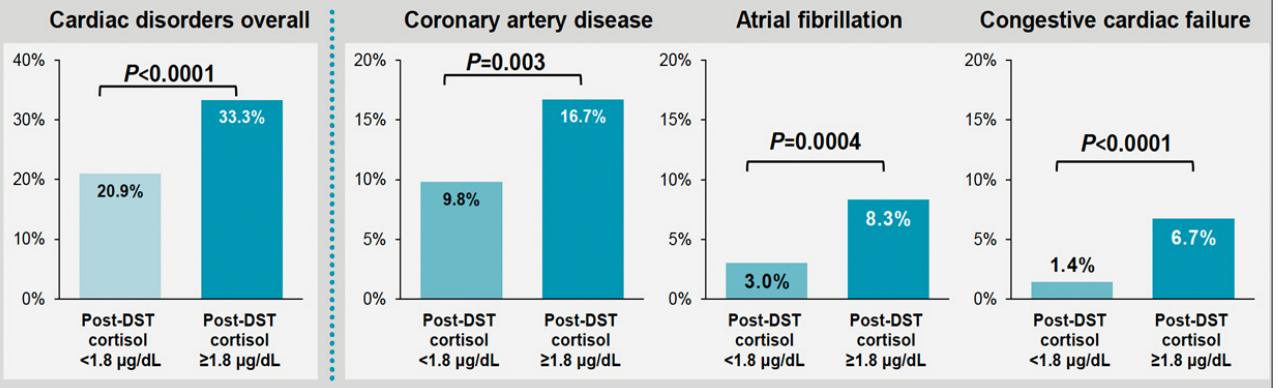


Buse, Kahn et al. *Diabetes Care* 2025:dc242841. [a]Odds ratio and 95% CI from a univariate logistic regression model for hypercortisolism vs no hypercortisolism performed separately for each variable. BP, blood pressure; CI, confidence interval; DST, dexamethasone suppression test; OR, odds ratio; T2D, type 2 diabetes.

CATALYST

Screening Phase

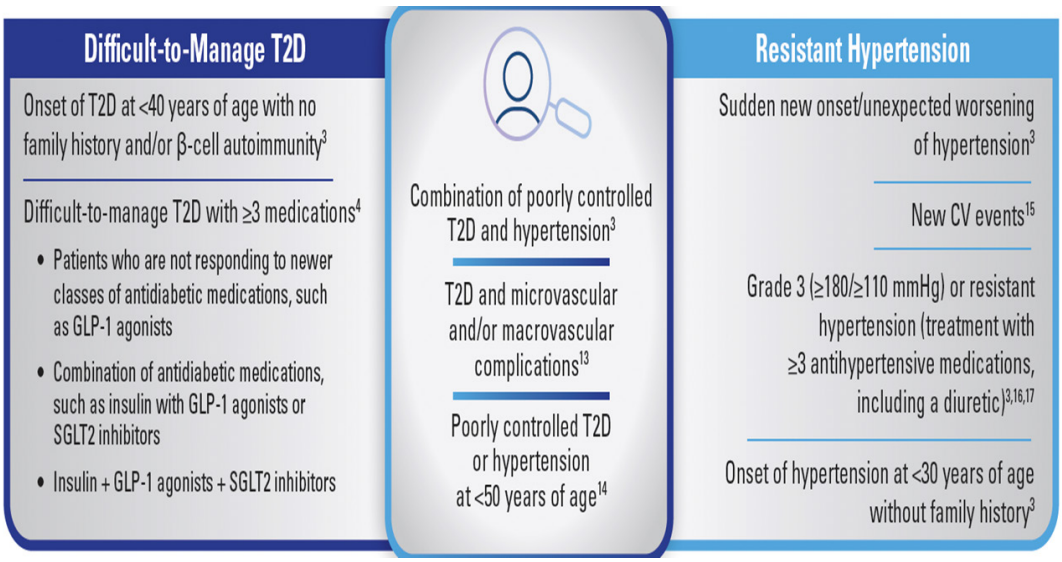
Comorbidity of Interest: Participants With Hypercortisolism Had More Cardiovascular Disease

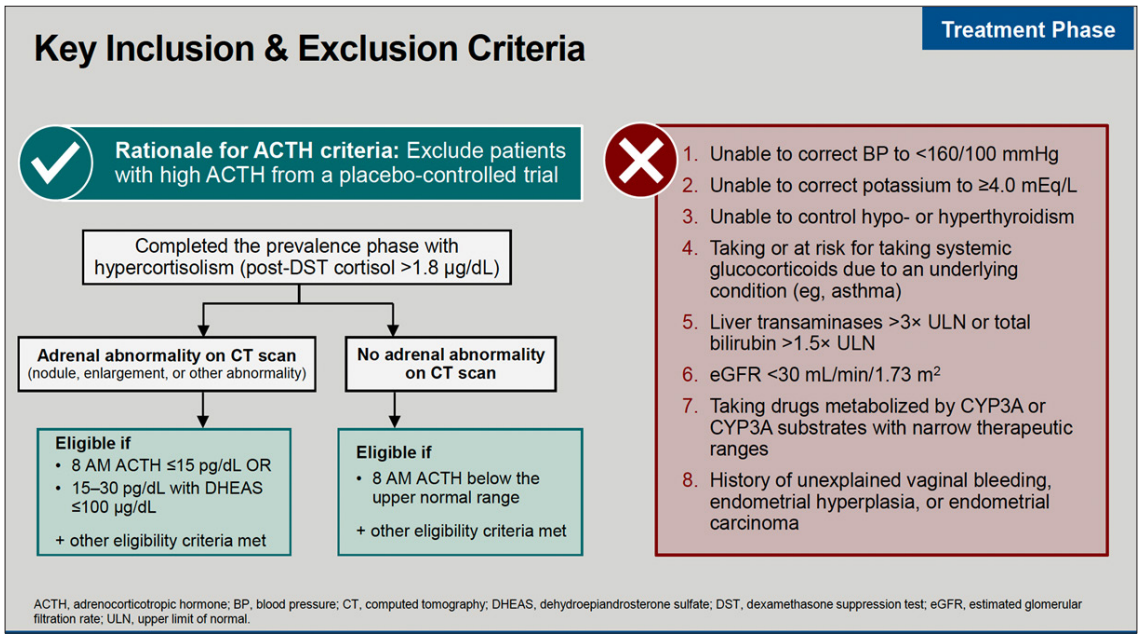


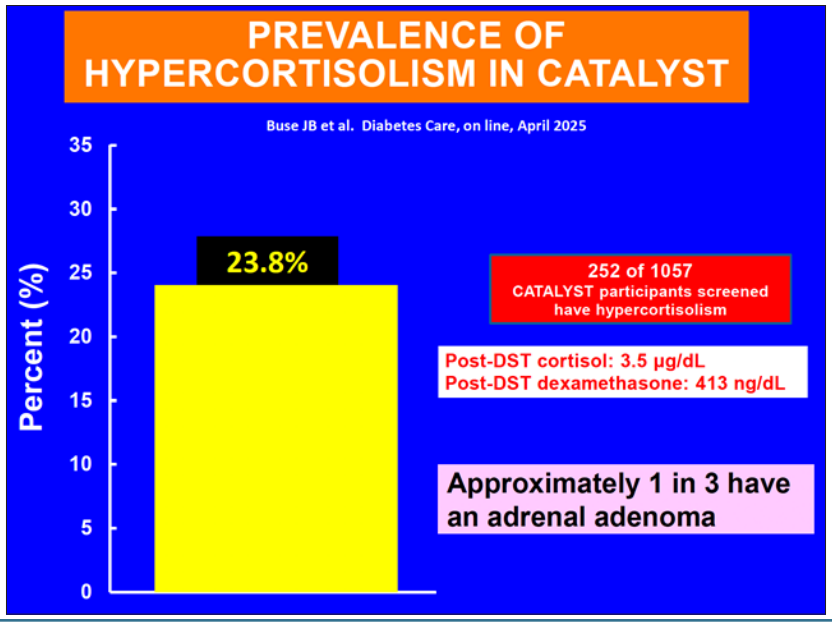
- Patients with T2D are at higher CVD risk, and this risk appears to increase further in those who also have hypercortisolism
- Consistent with the observation that CATALYST participants with hypercortisolism had a higher overall medication burden

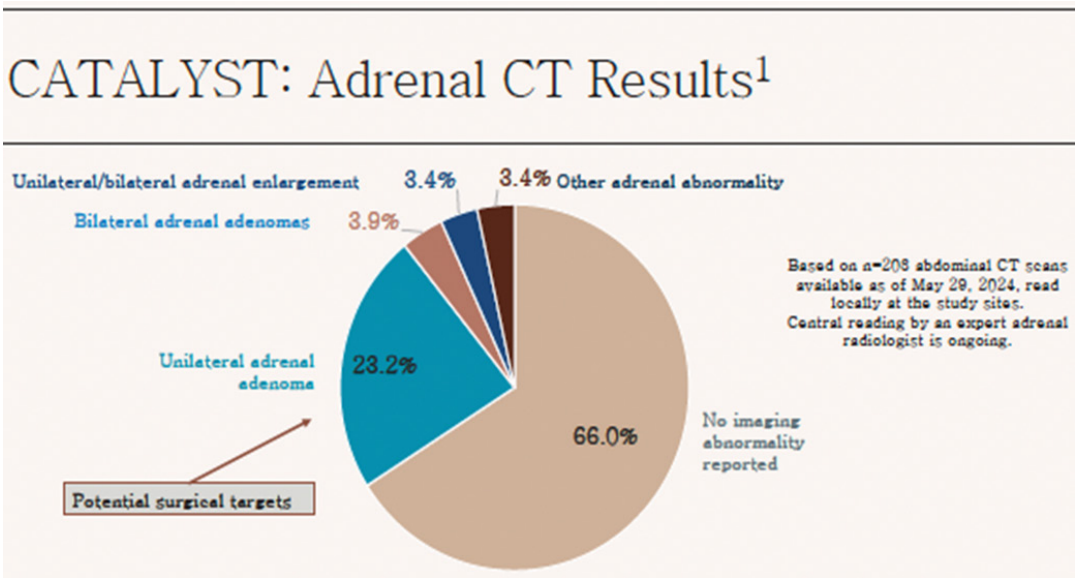
Buse, Kahn et al. *Diabetes Care* 2025:dc242841. *P*-values from a chi-squared test. CVD, cardiovascular disease; DST, dexamethasone suppression test; T2D, type 2 diabetes.

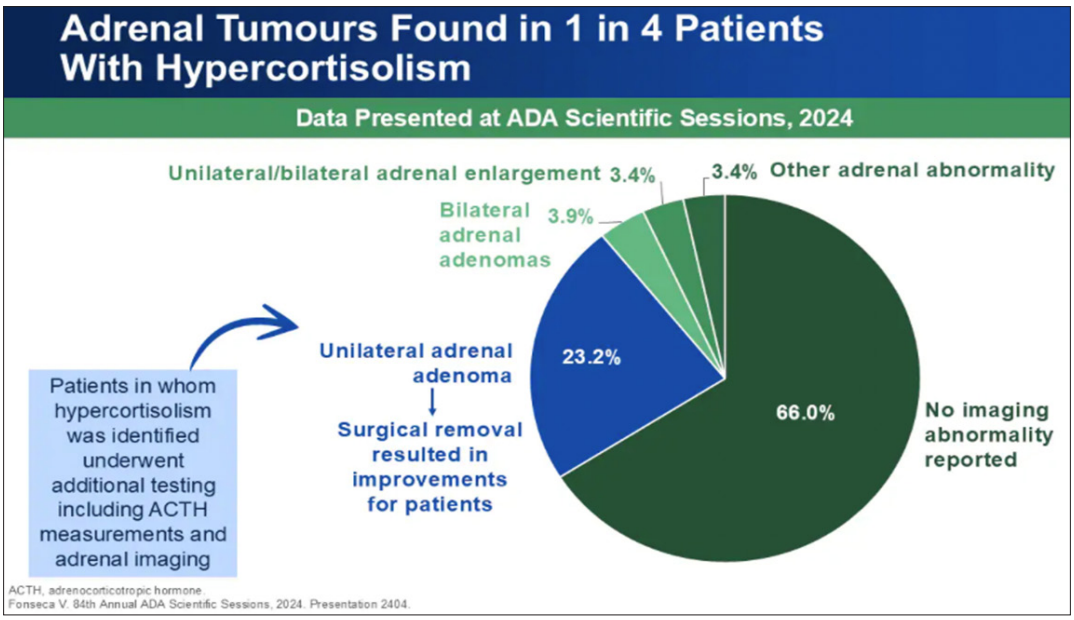
CATALYST

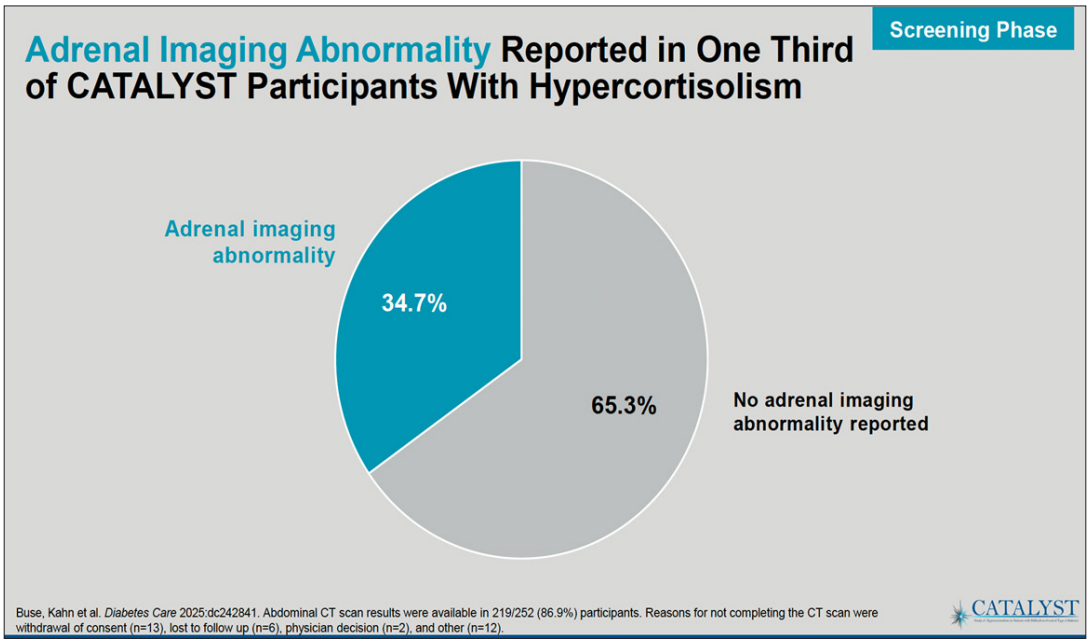

Difficult-to-Manage T2D
Onset of T2D at <40 years of age with no family history and/or β-cell autoimmunity[3]
Difficult-to-manage T2D with ≥3 medications[4]
• Patients who are not responding to newer classes of antidiabetic medications, such as GLP-1 agonists
• Combination of antidiabetic medications, such as insulin with GLP-1 agonists or SGLT2 inhibitors
• Insulin + GLP-1 agonists + SGLT2 inhibitors
Combination of poorly controlled T2D and hypertension[3]
T2D and microvascular and/or macrovascular complications[13]
Poorly controlled T2D or hypertension at <50 years of age[14]
Resistant Hypertension
Sudden new onset/unexpected worsening of hypertension[3]
New CV events[15]
Grade 3 (≥180/≥110 mmHg) or resistant hypertension (treatment with ≥3 antihypertensive medications, including a diuretic)[3,16,17]
Onset of hypertension at <30 years of age without family history[3]

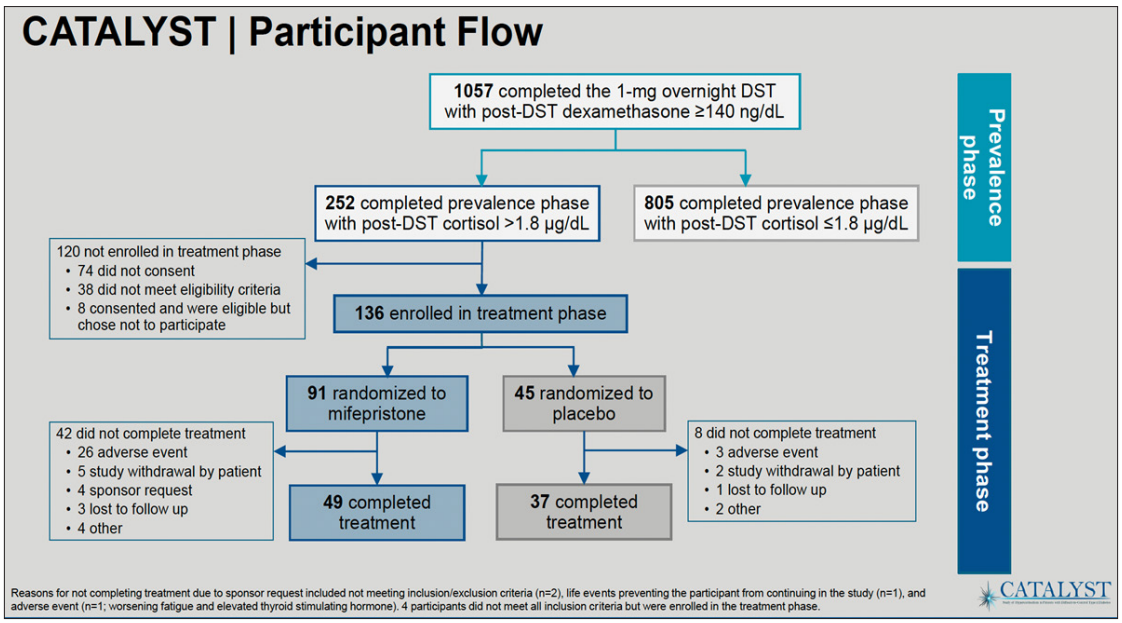

Key Inclusion & Exclusion Criteria
Treatment Phase
Rationale for ACTH criteria: Exclude patients with high ACTH from a placebo-controlled trial
Completed the prevalence phase with hypercortisolism (post-DST cortisol >1.8 µg/dL)
Adrenal abnormality on CT scan (nodule, enlargement, or other abnormality)
No adrenal abnormality on CT scan
Eligible if
• 8 AM ACTH ≤15 pg/dL OR
• 15–30 pg/dL with DHEAS ≤100 µg/dL
+ other eligibility criteria met
Eligible if
• 8 AM ACTH below the upper normal range
+ other eligibility criteria met
1. Unable to correct BP to <160/100 mmHg
2. Unable to correct potassium to ≥4.0 mEq/L
3. Unable to control hypo- or hyperthyroidism
4. Taking or at risk for taking systemic glucocorticoids due to an underlying condition (eg, asthma)
5. Liver transaminases >3× ULN or total bilirubin >1.5× ULN
6. eGFR <30 mL/min/1.73 m²
7. Taking drugs metabolized by CYP3A or CYP3A substrates with narrow therapeutic ranges
8. History of unexplained vaginal bleeding, endometrial hyperplasia, or endometrial carcinoma
ACTH, adrenocorticotropic hormone; BP, blood pressure; CT, computed tomography; DHEAS, dehydroepiandrosterone sulfate; DST, dexamethasone suppression test; eGFR, estimated glomerular filtration rate; ULN, upper limit of normal.


PREVALENCE OF HYPERCORTISOLISM IN CATALYST
Buse JB et al. Diabetes Care, on line, April 2025
Percent (%)
35
30
25
20
15
10
5
0
23.8%
252 of 1057
CATALYST participants screened have hypercortisolism
Post-DST cortisol: 3.5 µg/dL
Post-DST dexamethasone: 413 ng/dL
Approximately 1 in 3 have an adrenal adenoma


CATALYST: Adrenal CT Results[1]
Unilateral/bilateral adrenal enlargement
3.4%
3.4% Other adrenal abnormality
Bilateral adrenal adenomas
3.9%
Based on n=208 abdominal CT scans available as of May 29, 2024, read locally at the study sites.
Central reading by an expert adrenal radiologist is ongoing.
Unilateral adrenal adenoma
23.2%
66.0%
No imaging abnormality reported
Potential surgical targets


Adrenal Tumours Found in 1 in 4 Patients With Hypercortisolism
Data Presented at ADA Scientific Sessions, 2024
Unilateral/bilateral adrenal enlargement 3.4%
3.4% Other adrenal abnormality
Bilateral adrenal adenomas
3.9%
Unilateral adrenal adenoma
23.2%
66.0%
No imaging abnormality reported
Surgical removal resulted in improvements for patients
Patients in whom hypercortisolism was identified underwent additional testing including ACTH measurements and adrenal imaging
ACTH, adrenocorticotropic hormone.
Fonseca V. 84th Annual ADA Scientific Sessions, 2024. Presentation 2404.


Screening Phase
Adrenal Imaging Abnormality Reported in One Third of CATALYST Participants With Hypercortisolism
Adrenal imaging abnormality
34.7%
65.3%
No adrenal imaging abnormality reported
Buse, Kahn et al. Diabetes Care 2025:dc242841. Abdominal CT scan results were available in 219/252 (86.9%) participants. Reasons for not completing the CT scan were withdrawal of consent (n=13), lost to follow up (n=6), physician decision (n=2), and other (n=12).
CATALYST

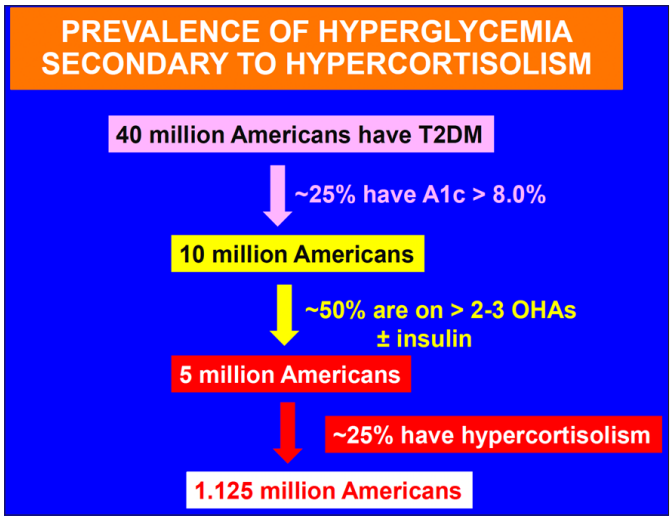


Enriched Population for Screening

According to the 2008 Endocrine Society Clinical Practice Guideline, screening should include (but not be limited to) the following

- Patients with unusual features for their age, such as osteoporosis/fragility fracture, T2D or hypertension in young individuals
- Patients with multiple and unexplained/progressive features, like worsening T2D outside of the normal progression or unexplained recent weight gain
- All patients with adrenal mass.

An observational study using a prospective hypercortisolism registry identified a prevalence of up to 50% using these screening criteria.

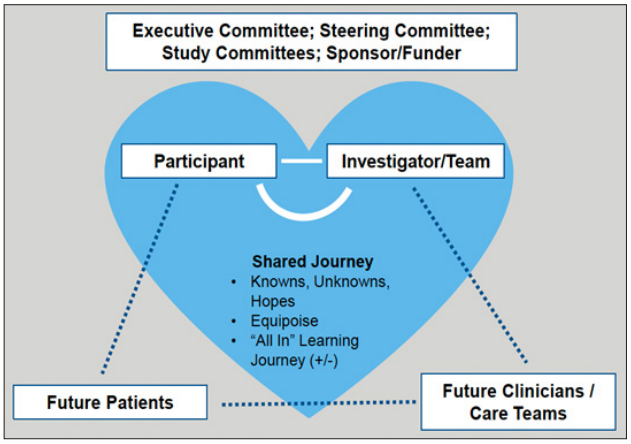


Summary: Design of the CATALYST Trial & Prevalence Results

CATALYST was the largest, prospective study to date assessing the prevalence and the impact of pharmacological treatment of hypercortisolism in patients with difficult-to-control T2D

Prevalence Phase Results

- **Hypercortisolism prevalence of 23.8%** in the overall study population (n=1057)
- **Hypercortisolism prevalence of 36.6%** among participants **who took ≥3 BP-lowering medications** in addition to their glucose-lowering medications
- **In one third of participants** with hypercortisolism, a standard abdominal **CT** scan revealed an adrenal abnormality



Key Endpoints for Part 2: Evaluate the Safety and Efficacy of Mifepristone Treatment in These Participants

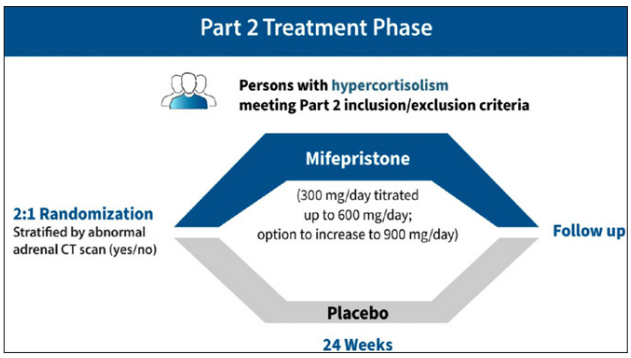
Enrolled 136 Participants

Primary Endpoint

- Change in HbA1c from baseline to week 24 for mifepristone compared to placebo

Key Secondary Endpoints

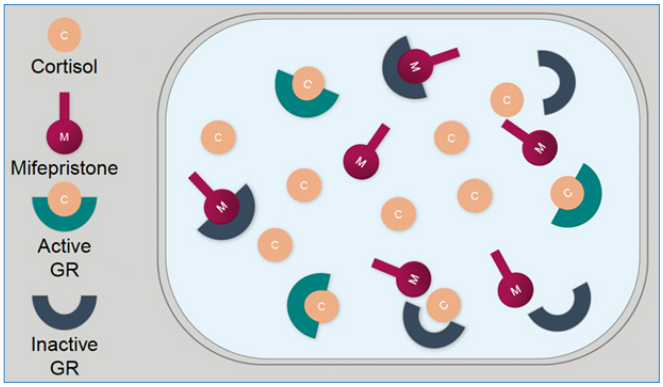
- Changes in glucose-lowering medications
- Changes in hypercortisolism-related comorbidities (eg, glycemic metrics, weight, waist circumference, BMI, BP, lipids)
- Differences between participants with and without adrenal imaging abnormalities



Statistical Methods

- 90% power to detect a difference in change from baseline to week 24 in HbA1c between groups of at least 1.5% with 114 participants, based on a t-test with a=0.05
- Primary endpoint analysis applied a restricted maximum likelihood-based mixed-effect model for repeated measurements in the intent-to-treat population

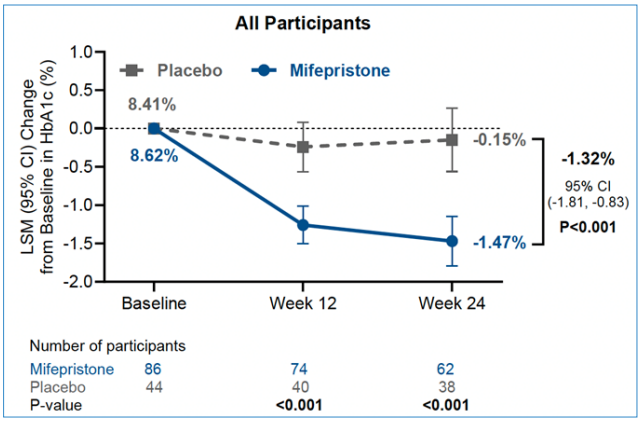
What Is Mifepristone and How Does it Work?



Mifepristone Is a Competitive Glucocorticoid Receptor Antagonist

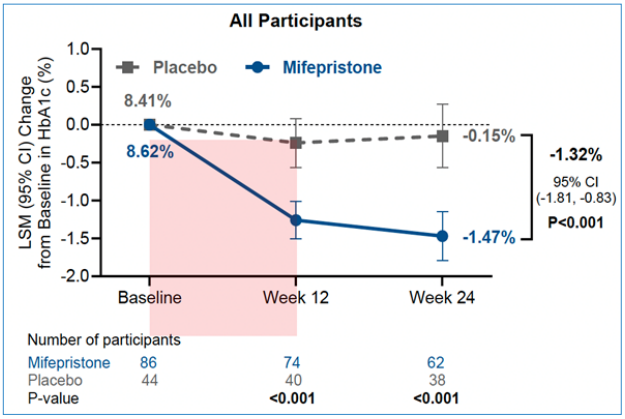
- Binds to the glucocorticoid receptor, decreasing cortisol-mediated signaling1-3 and reducing the clinical effects of hypercortisolism
- FDA approved for the treatment of hyperglycemia secondary to hypercortisolism in adult patients with endogenous Cushing syndrome who have T2D or glucose intolerance

Primary Endpoint: Statistically Significant Reduction in HbA1c



Of the 91 participants randomized to mifepristone, 65 (71%) received 600 mg and of those, 28 (31%) received 900 mg mifepristone during the study.

Primary Endpoint: Statistically Significant Reduction in HbA1c Despite Medication Reductions / Discontinuation



Of the 91 participants randomized to mifepristone, 65 (71%) received 600 mg and of those, 28 (31%) received 900 mg mifepristone during the study

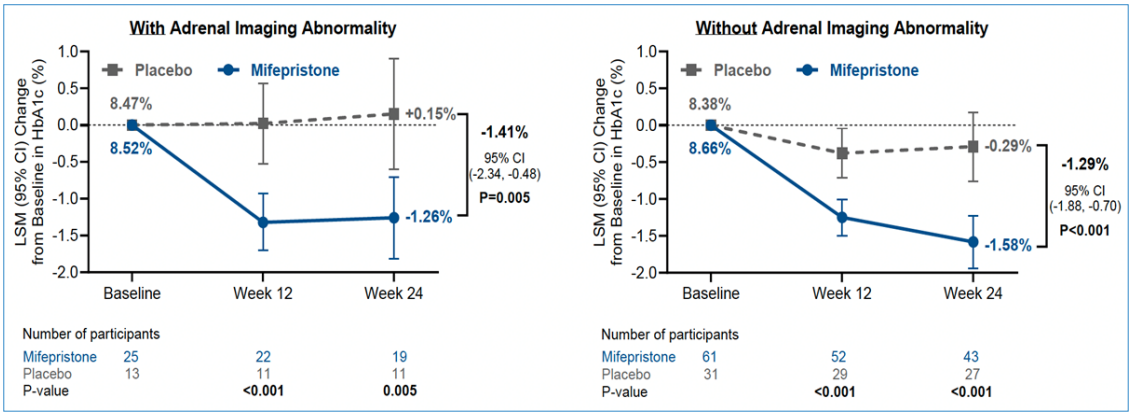
Discontinuations/Dose-reductions in Glucose-lowering Medications by Week 12

n/N[a] (%)	Mifepristone	Placebo
Long-acting insulin	32/65 (49.2%)	3/23 (13.0%)
Fast-acting insulin	10/33 (30.3%)	2/19 (10.5%)
Sulfonylureas	4/18 (22.2%)	2/19 (10.5%)
GLP-1 RAs	4/33 (12.1%)	0/19
Tirzepatide	2/19 (10.5%)	0/3
Metformin	1/67 (1.5%)	0/33
SGLT2 inhibitors	0/56	1/27 (3.7%)
Total	**53**	**8**

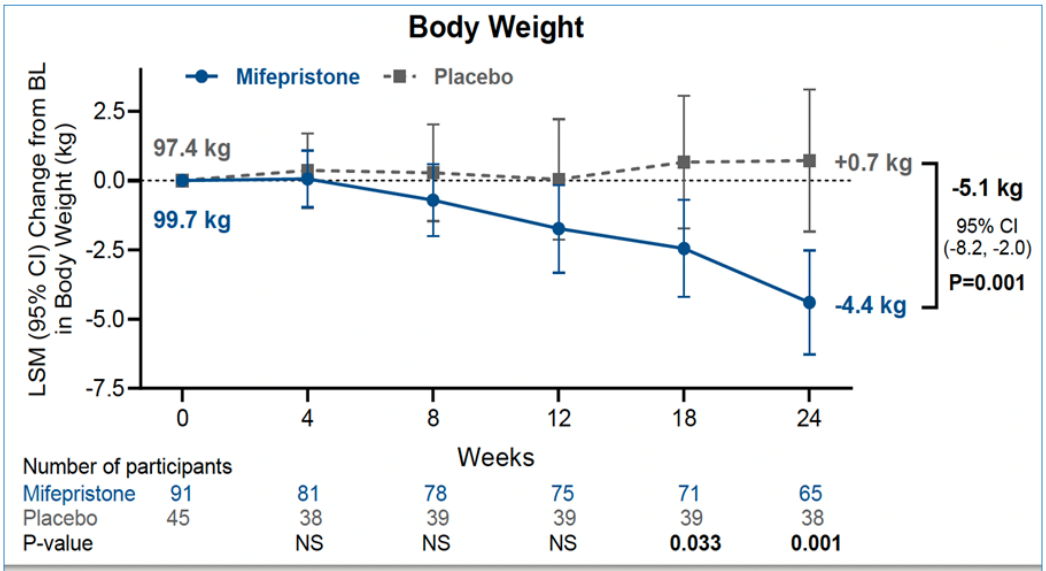
HbA1c : Subgroup Analyses Support the Primary End Point

HbA1c (%)	Mifepristone	Placebo	Difference from Placebo	P-value
Participants who completed 24 weeks of treatment				
Baseline, mean (SD) [n]	8.82 (1.33) [47]	8.42 (1.02) [36]	—	—
Change from baseline to week 24, LSM (95% CI) [n]	**-1.69** (-2.09, -1.30) [47]	-0.13 (-0.58, 0.32) [36]	**-1.56** (-2.12, -1.00)	**<0.001**
Participants who received 3 tablets (900 mg mifepristone)				
Baseline, mean (SD) [n]	9.10 (1.35) [24]	8.62 (1.09) [25]	—	—
Change from baseline to week 24, LSM (95% CI) [n]	**-2.01** (-2.63, -1.39) [23]	-0.16 (-0.76, 0.45) [25]	**-1.85** (-2.67, -1.04)	**<0.001**

Similar HbA1c Reductions in Participants with and without Adrenal Imaging Abnormalities

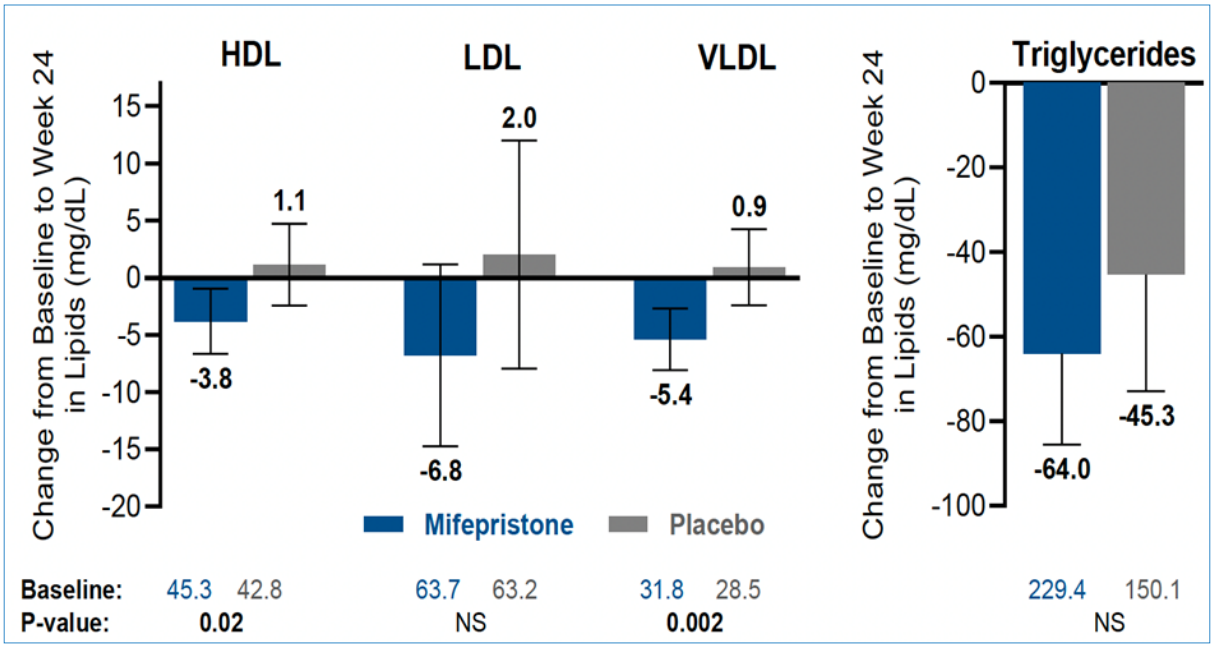


Body Weight Loss with Mifepristone

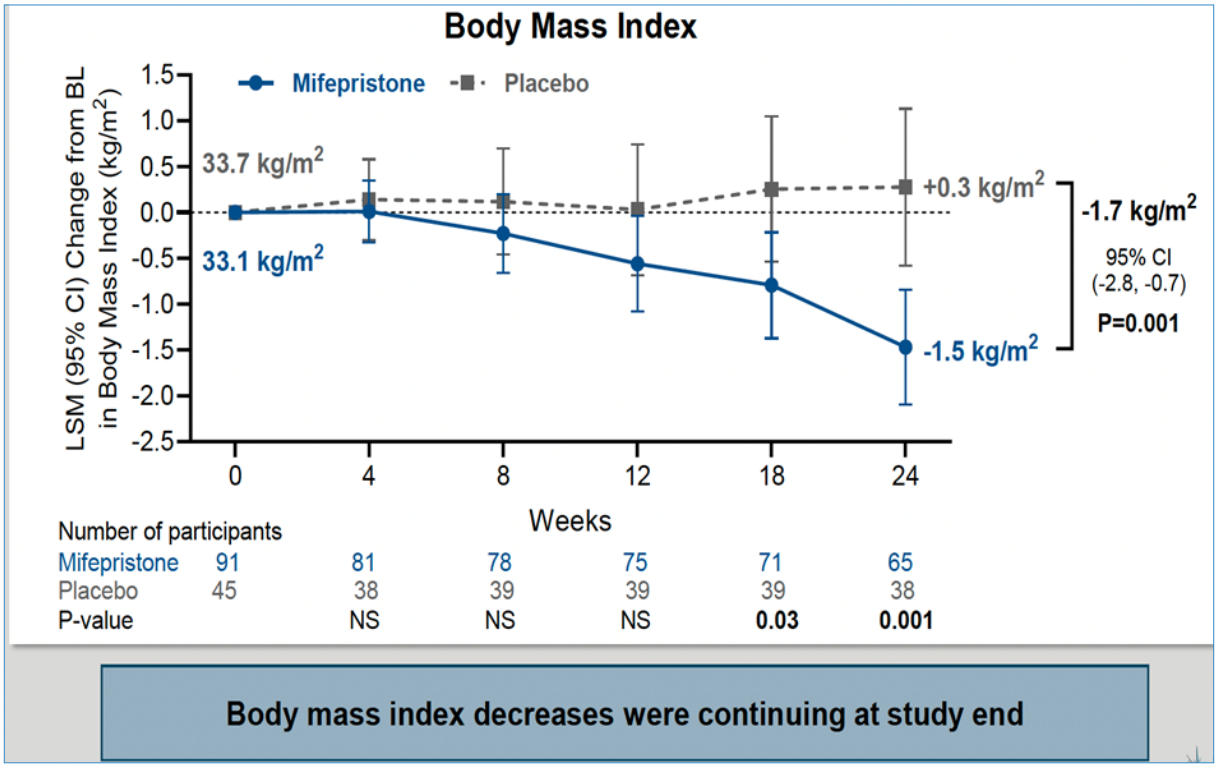


By week 24, participants taking mifepristone had a 3.6% reduction in body weight compared to an increase of 0.3% in those taking placebo

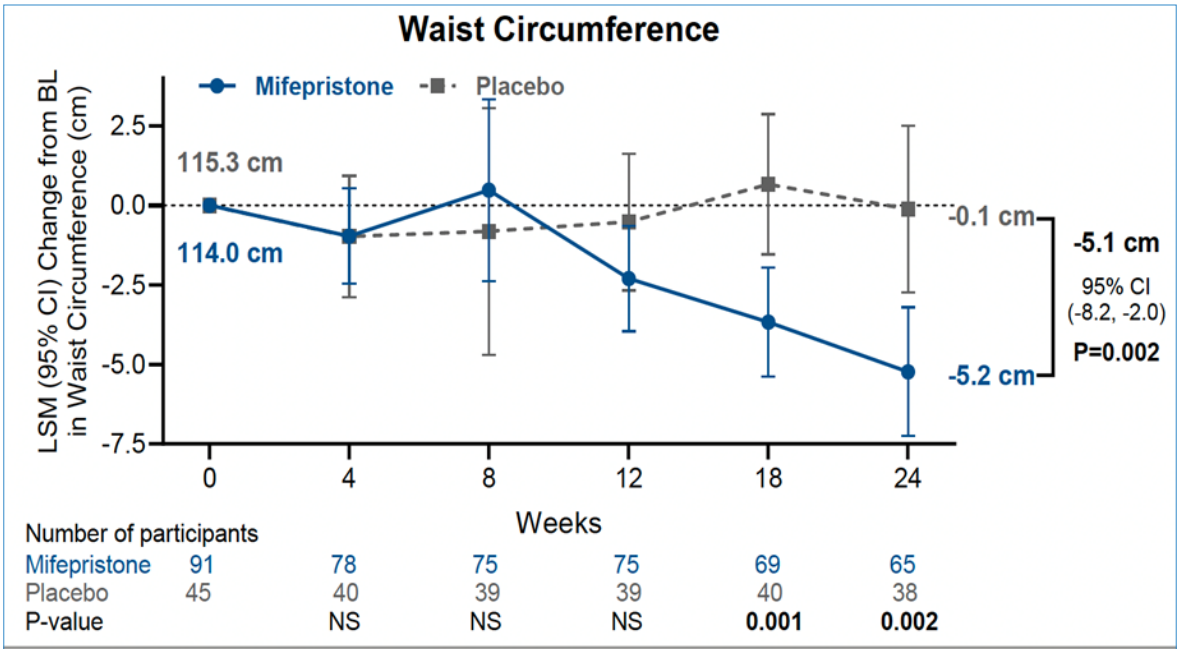
Effects of Mifepristone on Lipids



Body Mass Index Decreased with Mifepristone



Waist Circumference Decreased with Mifepristone



Waist circumference decreased along with body weight, suggesting loss of primarily visceral fat mass

Treatment Phase

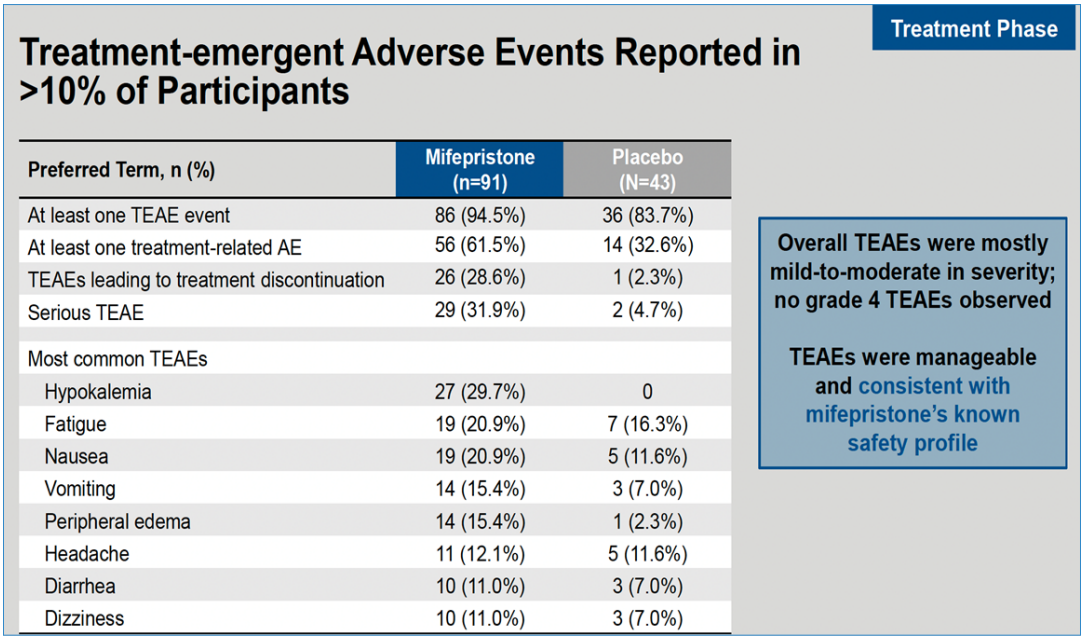
Treatment-emergent Adverse Events Reported in >10% of Participants

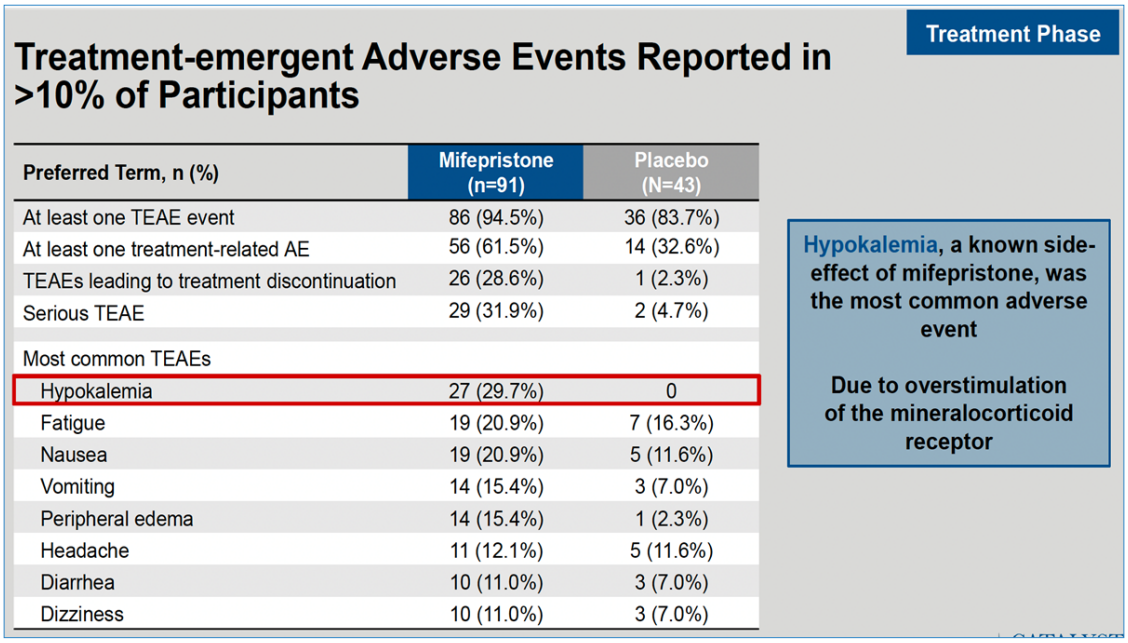
Preferred Term, n (%)	Mifepristone (n=91)	Placebo (N=43)
At least one TEAE event	86 (94.5%)	36 (83.7%)
At least one treatment-related AE	56 (61.5%)	14 (32.6%)
TEAEs leading to treatment discontinuation	26 (28.6%)	1 (2.3%)
Serious TEAE	29 (31.9%)	2 (4.7%)
Most common TEAEs		
Hypokalemia	27 (29.7%)	0
Fatigue	19 (20.9%)	7 (16.3%)
Nausea	19 (20.9%)	5 (11.6%)
Vomiting	14 (15.4%)	3 (7.0%)
Peripheral edema	14 (15.4%)	1 (2.3%)
Headache	11 (12.1%)	5 (11.6%)
Diarrhea	10 (11.0%)	3 (7.0%)
Dizziness	10 (11.0%)	3 (7.0%)

TEAE, treatment-emergent adverse event.

Many of the most common TEAEs were consistent with glucocorticoid withdrawal, which can occur with any treatment for hypercortisolism, surgical or pharmacological

CATALYST





Treatment of hypokalemia with mineralocorticoid receptor antagonists-Spironolactone or Eplerenone

Summary and Conclusions

- In a cohort with difficult-to-control type 2 diabetes and hypercortisolism, mifepristone resulted in clinically & statistically significant improvements in HbA1c and other comorbidities
- Comparable reductions in HbA1c were observed in participants with and without adrenal imaging abnormalities
- Adverse events were consistent with mifepristone's known safety profile
- The most common adverse events were consistent with glucocorticoid withdrawal syndrome
- Hypokalemia may be addressed by proactive initiation of a potassium-sparing diuretic, eg, spironolactone, in clinical practice (as opposed to a double-blind trial).

Key Takeaway

In individuals with inadequately controlled type 2 diabetes and hypercortisolism, cortisol-directed medical therapy with mifepristone significantly reduced HbA1c

Summary - Treatment phase

- No new safety signals for mifepristone were identified
- One death was reported during the study in the placebo arm (attributed to cardiovascular disease)
- Serious treatment-emergent adverse events were reported more frequently in the mifepristone arm (32% versus 5%)
- Serious treatment-emergent adverse events occurring in more than 3% of participants were hypokalemia (5%; mifepristone) and euglycemic ketoacidosis (3%; mifepristone [all on SGLT2i])
- Many of the most common adverse events were consistent with glucocorticoid withdrawal, which can occur with any treatment for hypercortisolism

Summary

- When there is difficult-to-treat hypertension or diabetes despite adherence to optimal medication, consider the possibility of hypercortisolism
- It is more prevalent than most doctors have appreciated
- Overnight dexamethasone suppression test is the best way to diagnose
- Multidisciplinary teams are key
- Involve the endocrinologist, nephrologist, primary care physician, preventative cardiologist

References

1. Prete, A., Bancos, I. Mild autonomous cortisol secretion: pathophysiology, comorbidities and management approaches. *Nat Rev Endocrinol 20*, 460–473 (2024). https://doi.org/10.1038/s41574-024-00984-y.
2. Antonio Prinzi MD 1 2, Ausilia Maria Lombardo MD 1, Salvatore Finocchiaro MD et al. Expanding the Clinical Profile of Mild Autonomous Cortisol Secretion: New Diagnostic Markers and Emerging Complications.Endocrine Practice, 2025. https://www.sciencedirect.com/journal/endocrine-practice. https://doi.org/10.1016/j.eprac.2025.09.009
3. Sharma, ST, Nieman LK. Cushing's syndrome: all variants, detection and treatment. *Endocrinol Clin North Am*. 2011 Jun; 40(2): 379-391. DOI:10.1016/j.ecl.2011.01.0064.
4. S. Erzen, G. Tonin,D. Erzen et al. Amylin, Another Important Neuroendocrine Hormone for the Treatment of Diabesity. *Int. J. Mol. Sci. 2024, 25*(3), 1517; https://doi.org/10.3390/ijms25031517
5. Baars D, Fondevilla M, Meijnikman A et al. The central role of gut microbiota in the pathophysiology and management of Type 2 diabetes. Cell and host microbe, Vol. 32, Issue 8, P1280-1300, August 14, 2024. htpps;//doi.org/10.1016/j.chom.2024.07.017.
6. Petramala L, Olmati F, Concistrè A, Russo R et al. Cardiovascular and Metabolic Risk Factors in Patients with Subclinical Cushing Syndrome Endocrine. 2020 Oct;70(1):150-163. DOI: 10.1007/s12020-020-02297-2. Epub 2020 Apr 16.
7. Xiaodan Zhang1, Xiaoyi Deng1, Jianlong Zhou et al. The Association of Serum Cortisol Level with Microalbuminuria in Patients with Type 2 Diabetes and Prediabetes. *International Journal of Medical Sciences 2020; 17*(18): 2998-3004. DOI: 10.7150/ijms.48742
8. Buse JB, Kahn SE, Aroda VR, Auchus RJ et al. CATALYST Investigators. Prevalence of Hypercortisolism in Difficult-to-Control Type 2 Diabetes. Diabetes Care. 2025 Apr 18:dc242841. DOI : 10.2337/dc24-2841.
9. DeFrozo R, Fonseca V, Aroda V et al. Catalyst investigators.Inadequately Controlled Type 2 Diabetes and Hypercortisolism: Improved Glycemia With Mifepristone Treatment. Diabetes Care 06/2025. https://doi.org/10.2337/dc25-1055