Updates in Primary Care

Max Rusek, MD, MPH

Outline

- AHA 2026 Dyslipidemia Guidelines
- Prostate cancer screening
- AHA 2025 Hypertension Guidelines

- **Quick hits**
  - Coffee and atrial fibrillation recurrence
  - Extended duration apixaban after DVT/PE
  - Shingles and dementia risk
  - MASH- Yet another reason for GLP-1s

No disclosures to report

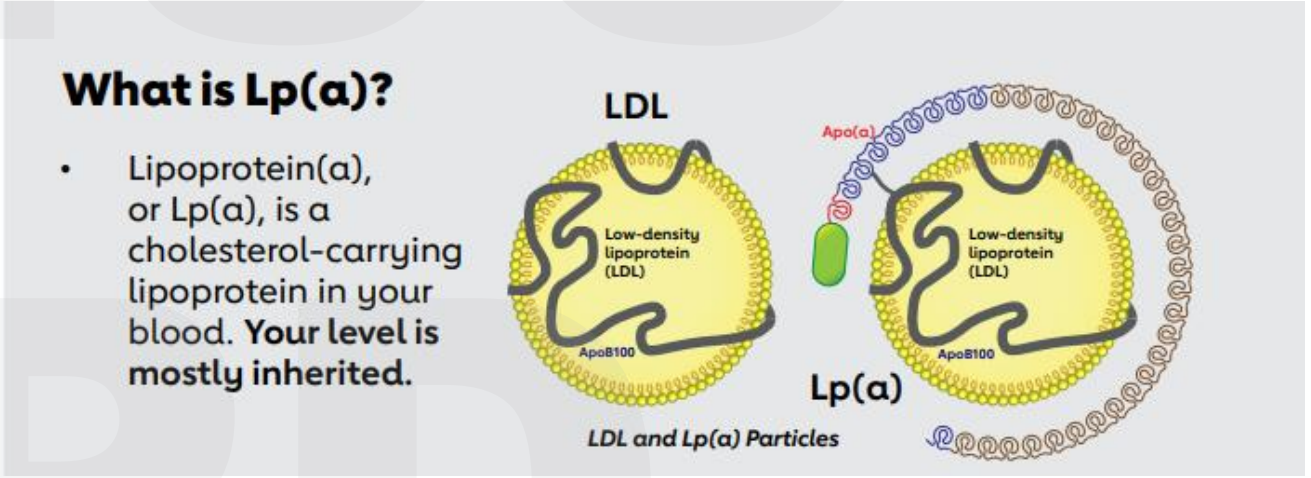
CLINICAL PRACTICE GUIDELINES

2026 ACC/AHA/AACVPR/ABC/ACPM/ADA/AGS/APhA/ASPC/NLA/PCNA Guideline on the Management of Dyslipidemia: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation, Association of Black Cardiologists, American College of Preventive Medicine, American Diabetes Association, American Geriatrics Society, American Pharmacists Association, American Society for Preventive Cardiology, National Lipid Association, and Preventive Cardiovascular Nurses Association

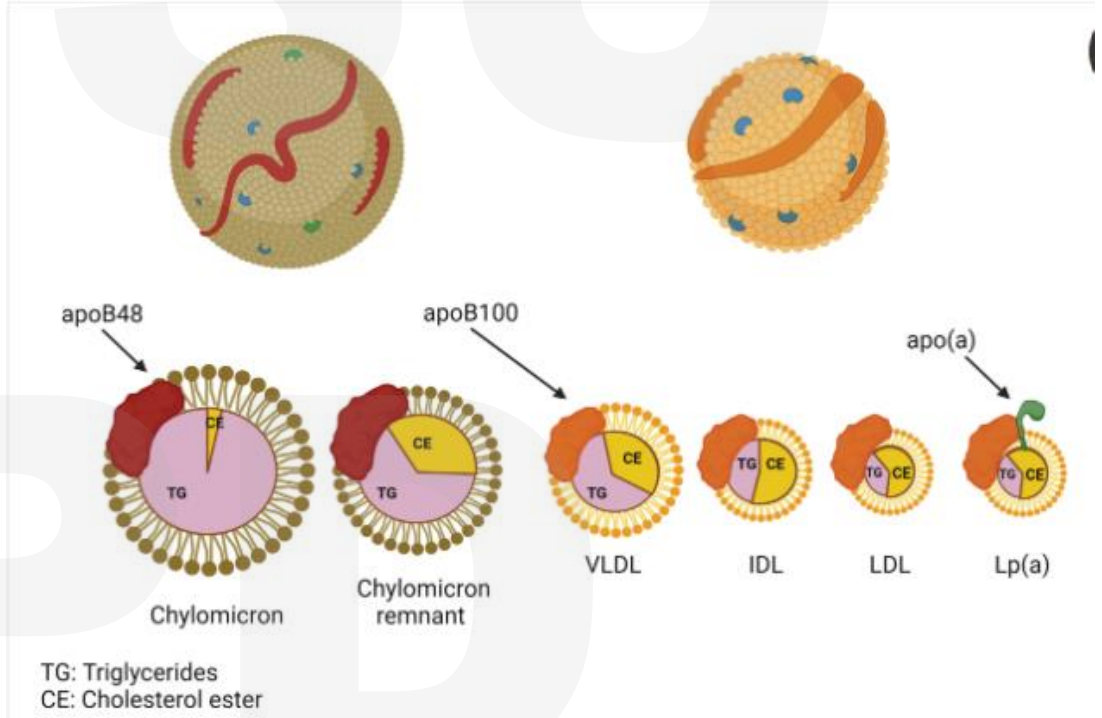
Definitions – Lipoprotein a or Lp(a)

- Lp(a) is an LDL-like particle - distinct from LDL
- Lp(a) levels predominantly genetic
- Elevated levels associated with increased ASCVD risk



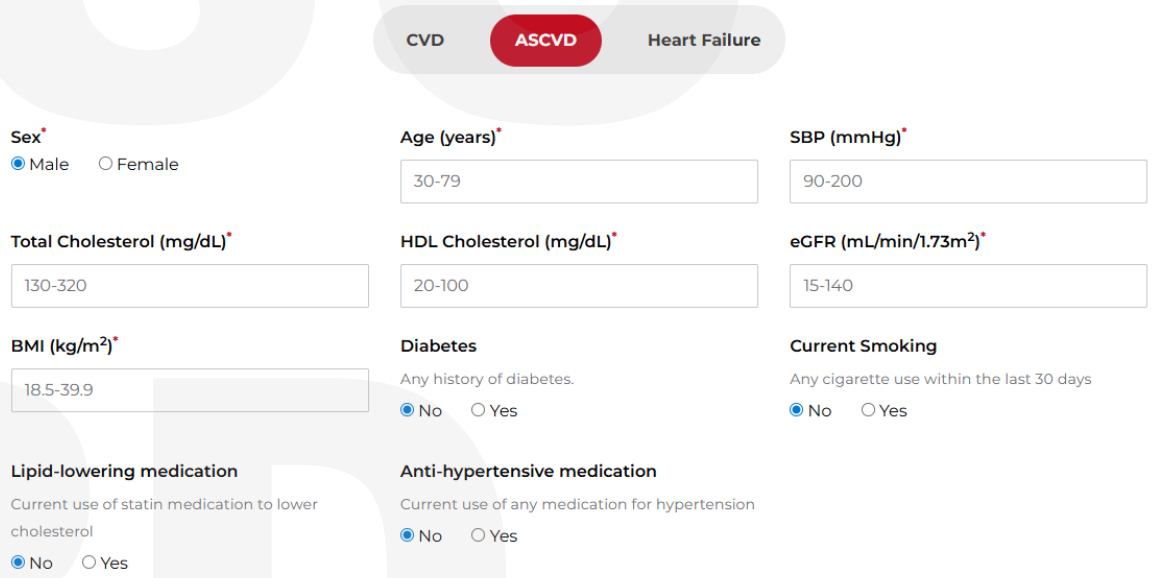
Definitions – Apolipoprotein B or ApoB

- Structural component of atherogenic particles
- ApoB more accurately predicts ASCVD risk than LDL-C in cases of discordance



Bottom Lines Upfront

- **PREVENT-ASCVD Calculator**
  - 10-year + 30-year risk
- **CPR + (R) model**
  - **Calculate** risk
    - PREVENT-ASCVD
  - **Personalize** risk
    - Risk modifying patient factors
      - CKD 3, HIV, inflammatory disorders
    - Lipoprotein A
    - Apo B
    - CAC score
  - **Reclassify** as needed
    - PREVENT ASCVD / CAC scoring /ApoB
  - **(R)e-assess**



Bottom Lines Upfront

- **Goals are back**
  - LDL + non-HDL (2a evidence)
  - 30-50% reduction in LDL (class 1)

- **ApoB Testing**
  - Consider to guide therapy + intensification particularly if
    - Elevated TG > 200, Diabetes, LDL <70 in secondary prevention

Bottom Lines Upfront

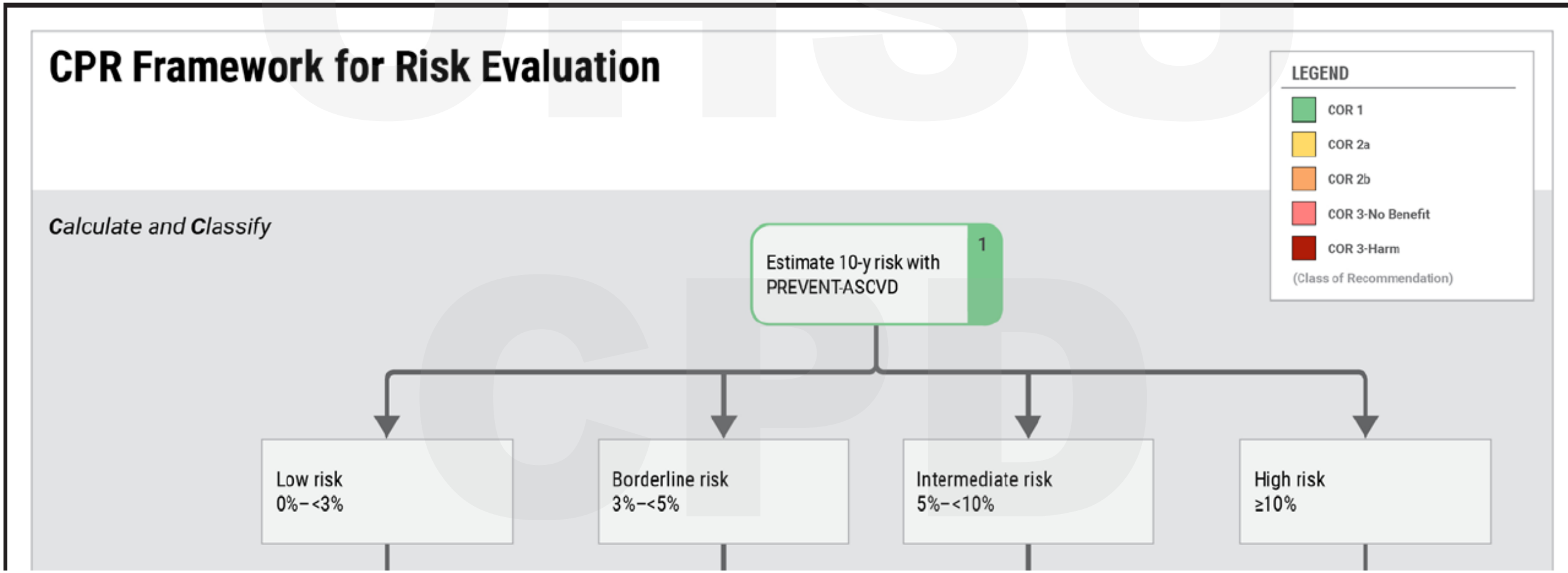
- **Lipoprotein(a) = Lp(a)**
  - One time universal screening recommended

- **CAC score guidance** and **Incidentally found coronary calcium**

- **Secondary prevention**
  - LDL <55 for ‘Very High Risk’
  - LDL <70 for ‘Not very high risk’
    - optional goal of LDL <55
  - Optional ApoB goals <55

Diving In

Primary Prevention



CVD | ASCVD | Heart Failure

Sex*
(•) Male ( ) Female

Age (years)*
30-79

SBP (mmHg)*
90-200

Total Cholesterol (mg/dL)*
130-320

HDL Cholesterol (mg/dL)*
20-100

eGFR (mL/min/1.73m^2)*
15-140

BMI (kg/m^2)*
18.5-39.9

Diabetes
Any history of diabetes.
(•) No ( ) Yes

Current Smoking
Any cigarette use within the last 30 days
(•) No ( ) Yes

Lipid-lowering medication
Current use of statin medication to lower cholesterol
(•) No ( ) Yes

Anti-hypertensive medication
Current use of any medication for hypertension
(•) No ( ) Yes

Primary Prevention Thresholds

- PREVENT ASCVD 10 Year
  - <3% = Low Risk
  - 3-5% = Borderline risk
  - 5-10% = Intermediate risk
  - >10% = High Risk
- PREVENT ASCVD 30 Year
  - >10 % = threshold to treat

Table 12. Crosswalk Between 10-Year Risk ASCVD Estimates From PCE and PREVENT-ASCVD Equations

	Approximate Equivalent Ranges of 10-Year ASCVD Risk Estimates*	
Risk Group	PCE	PREVENT-ASCVD
Low	<5%	<3%
Borderline	5% to <7.5%	3% to <5%
Intermediate	7.5% to <20%	5% to <10%
High	≥20%	≥10%

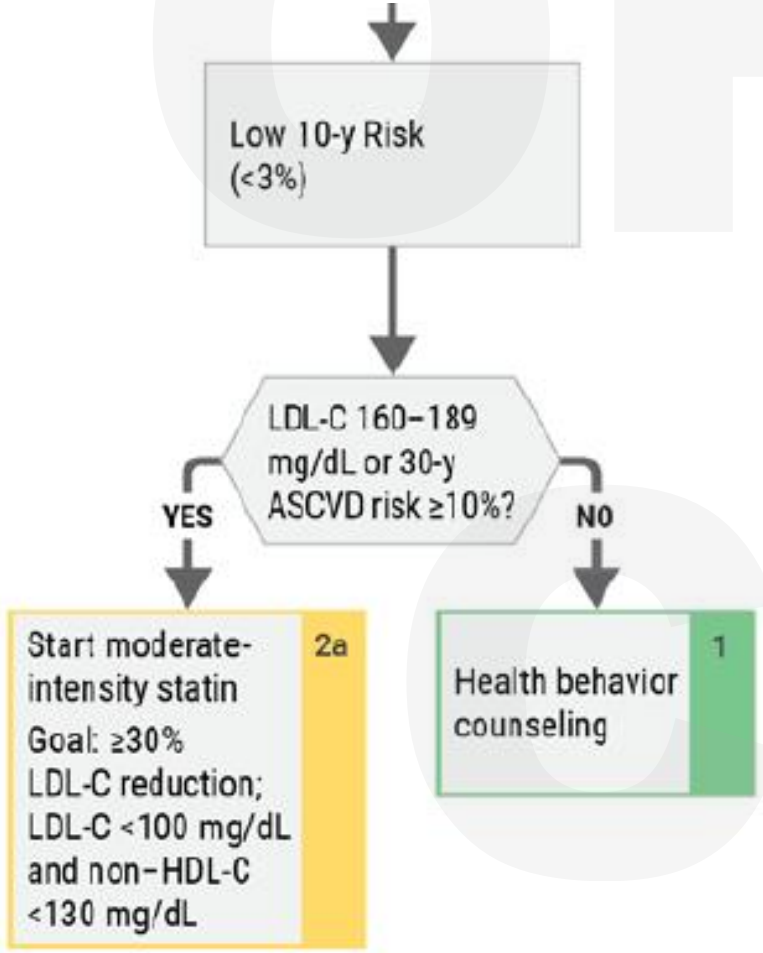
*The PREVENT-ASCVD equations generally provide 10-year risk estimates that are 40% to 50% lower than the PCE estimates because the PCE calculator often overestimated the risk for adults.

ASCVD denotes atherosclerotic cardiovascular disease; and PCE, pooled cohort equations. Adapted from Khan et al.[1,3]

Rationale for New 10-y Risk Thresholds in Lipid Lowering Therapy Using PREVENT-ASCVD

Rationale to start LLT in patients at borderline (3% to <5%), intermediate (5% to <10%), and high (≥10%) predicted 10-y risk
Estimates from contemporary PREVENT-ASCVD equations ~40%–50% lower than older PCE
Similar numbers of US adults recommended to consider statin therapy using PCE ≥5% or PREVENT-ASCVD ≥3% 10-y risk
Net benefit (benefit > potential harm) for statin therapy ≥3% 10-y event rate in primary prevention RCTs

Primary Prevention - 30-79 YO
Low Risk Group – **LDL 160 a new threshold**



IF PREVENT ASCVD 10 y < 3% AND LDL <160 = Lifestyle

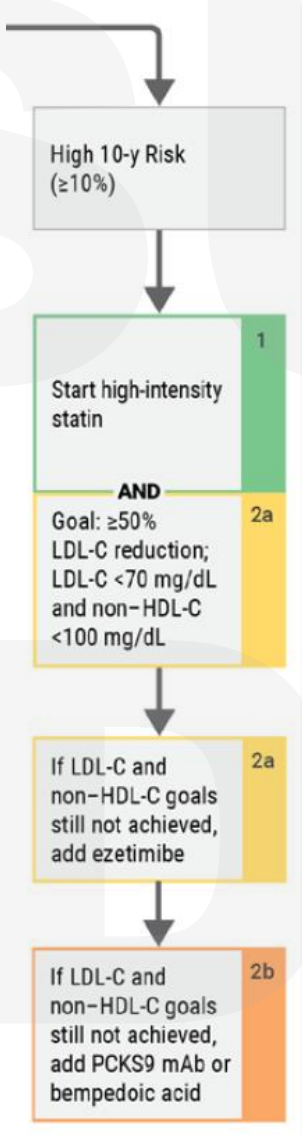
IF PREVENT ASCVD 10 y <3% AND **LDL>160** = consider mod statin

IF PREVENT ASCVD 30 year >10% = consider mod statin

LDL goal <100, non-HDL <130

Primary Prevention - 30-79 YO High Risk

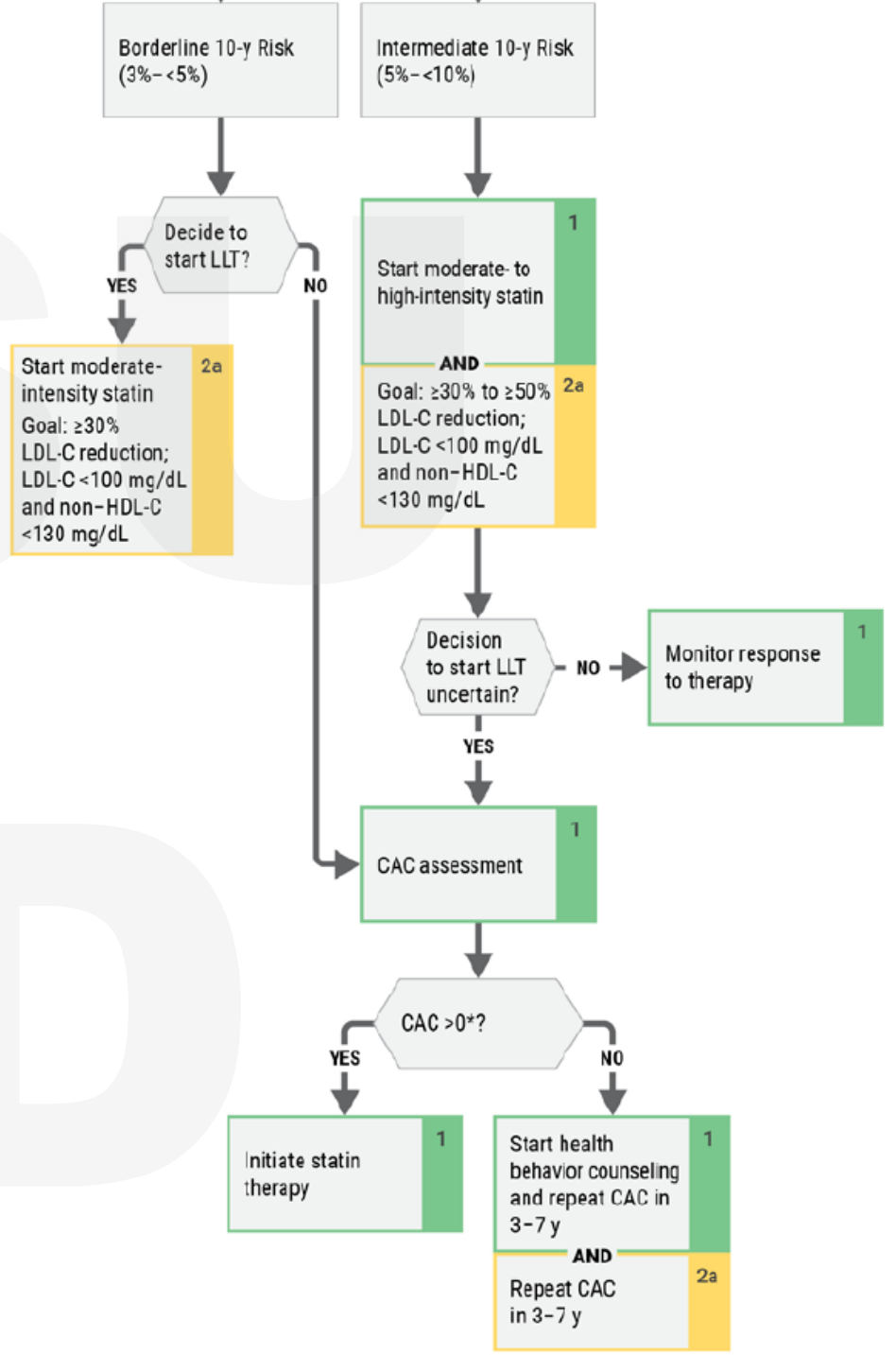
- PREVENT ASCVD 10 year >10%
- High risk = High intensity
- LDL goal <70, non-HDL <100



Primary Prevention 3-<10% Borderline – Intermediate

- Moderate intensity recommended
- Goal LDL <100, non-HDL <130
- Consider CAC to initiate
- IF CAC = 0
  - Defer LLT + Consider repeat 3-7 years

CAC to be avoided if:
Familial Hyperlipidemia, LDL>190
diabetes, cigarette smoking, strong family hx, premature ASCVD



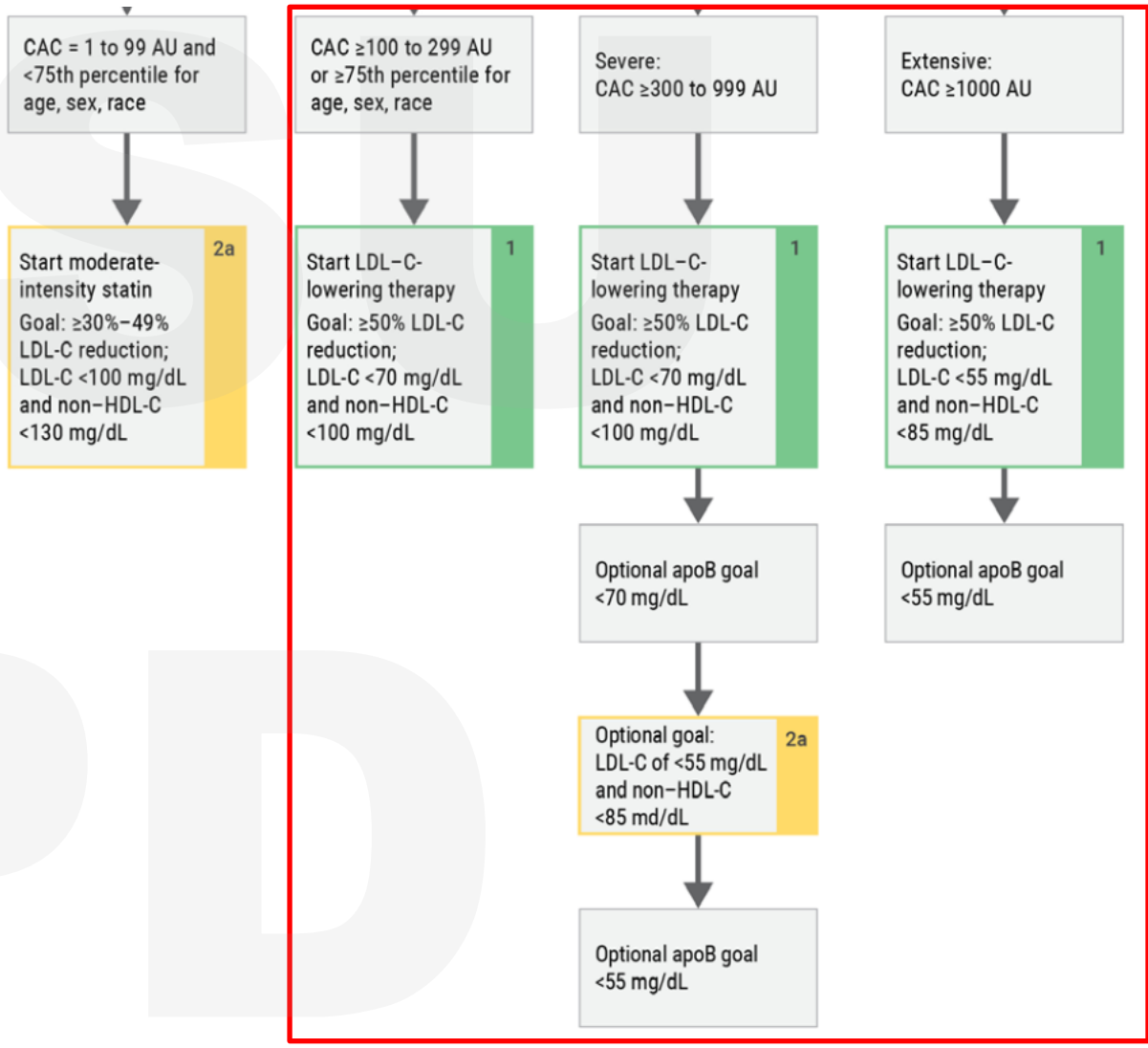
4.2.4. Severe Hypercholesterolemia (LDL-C ≥190 mg/dL [4.9 mmol/L])

4.2.4.1. Role of Risk Assessment in HeFH

Recommendations for Role of Risk Assessment in HeFH Referenced studies that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
2b	**B-NR**	1. In adults with HeFH, FH-specific risk scores may be useful in predicting short-term ASCVD risk.[1–5]
3: Harm	**C-EO**	2. In individuals with HeFH, standard risk assessment tools developed for the general population should not be used to calculate 10- or 30-y ASCVD risk.

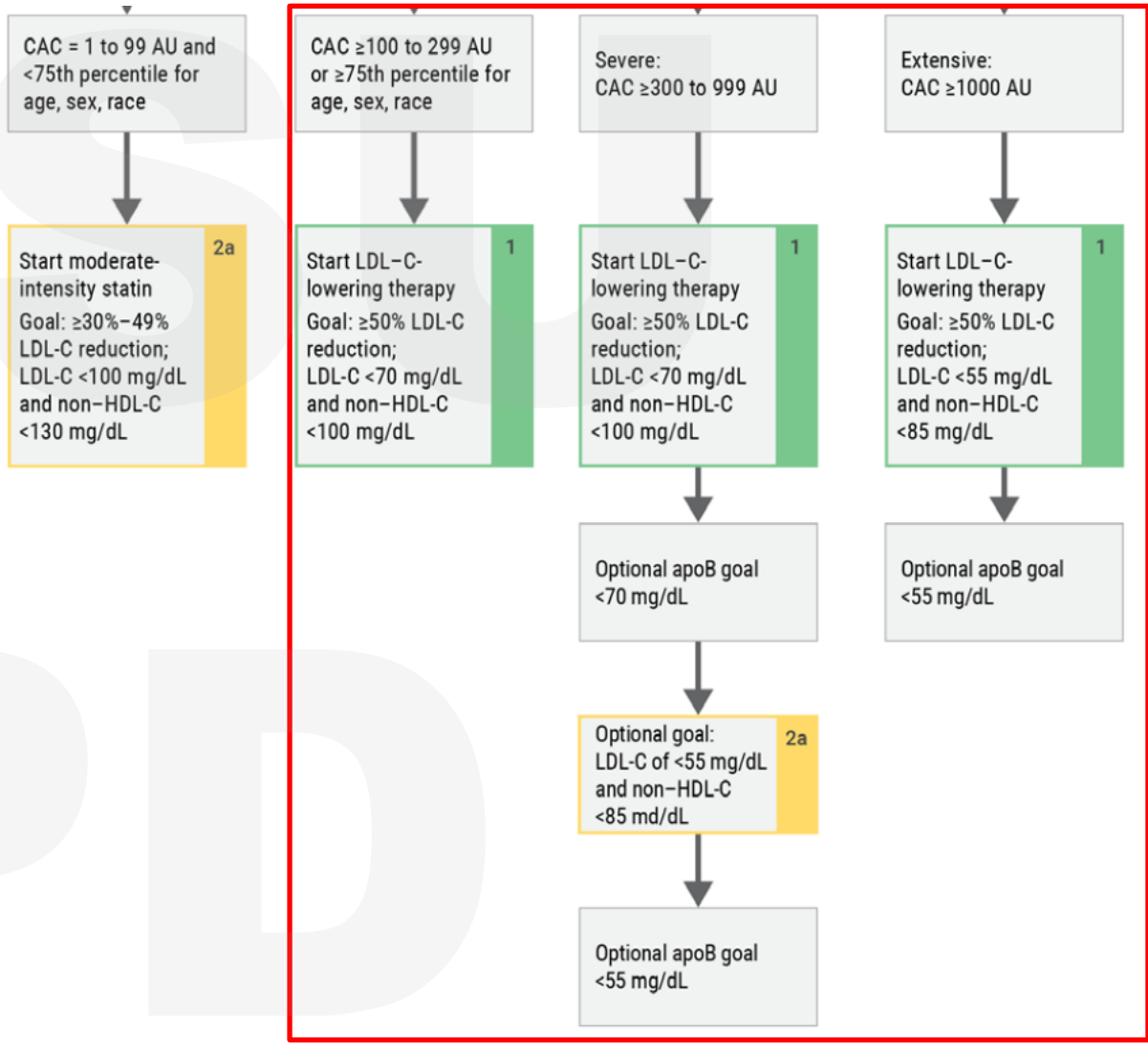
CAC

- Coronary Artery Calcium
- If > 0 – recommending statin
- CAC >100
  - Functionally secondary prevention goals



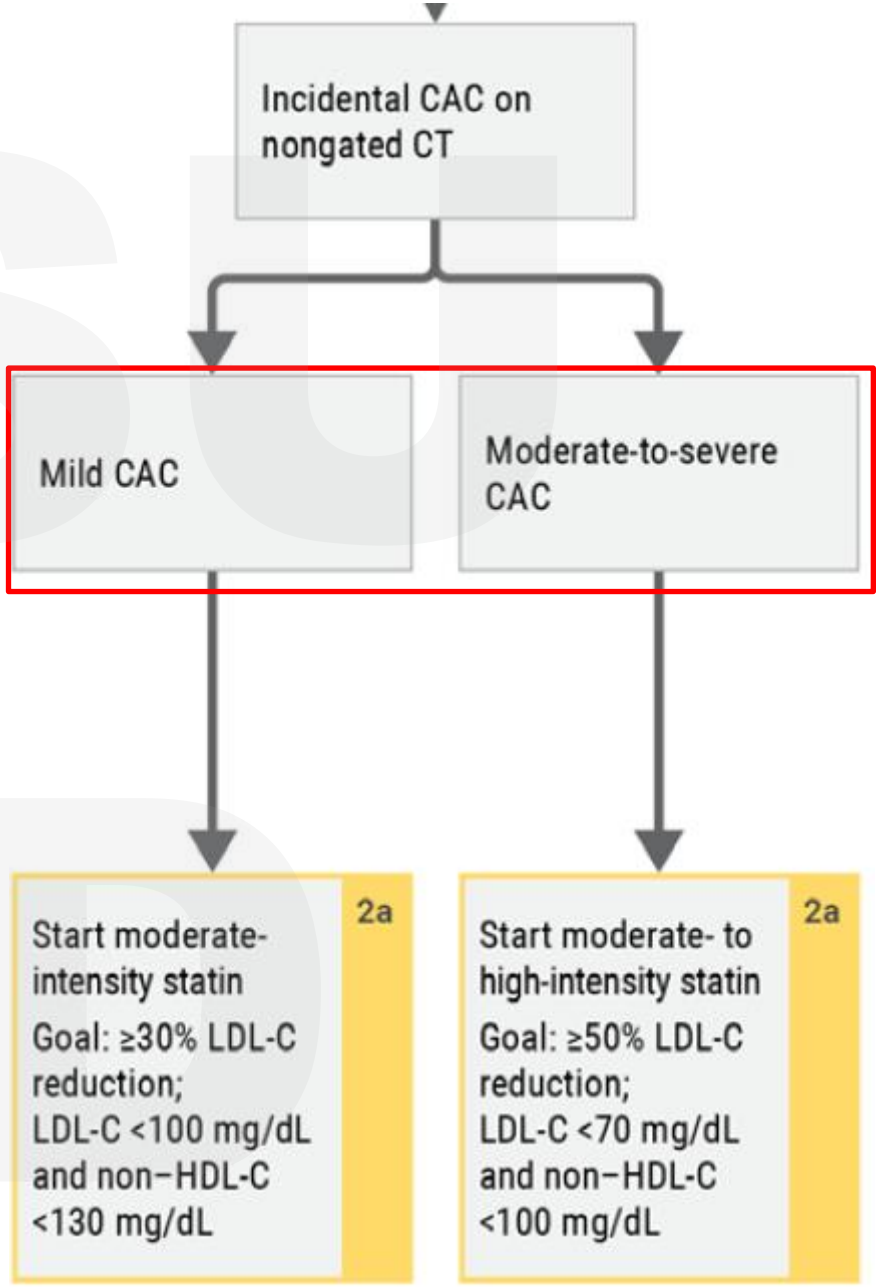
CAC

- CAC 100-299
  - LDL <70, non-HDL <100
- CAC 300-999
  - LDL <55-70, non-HDL<85-100
- CAC >1000
  - LDL < 55, non-HDL < 85



Incidental CAC

- Recommending statin therapy
- Carotid plaques also count
- Consider radiology over-read
  - Comment on mild vs. >mod
- Consider formal CAC

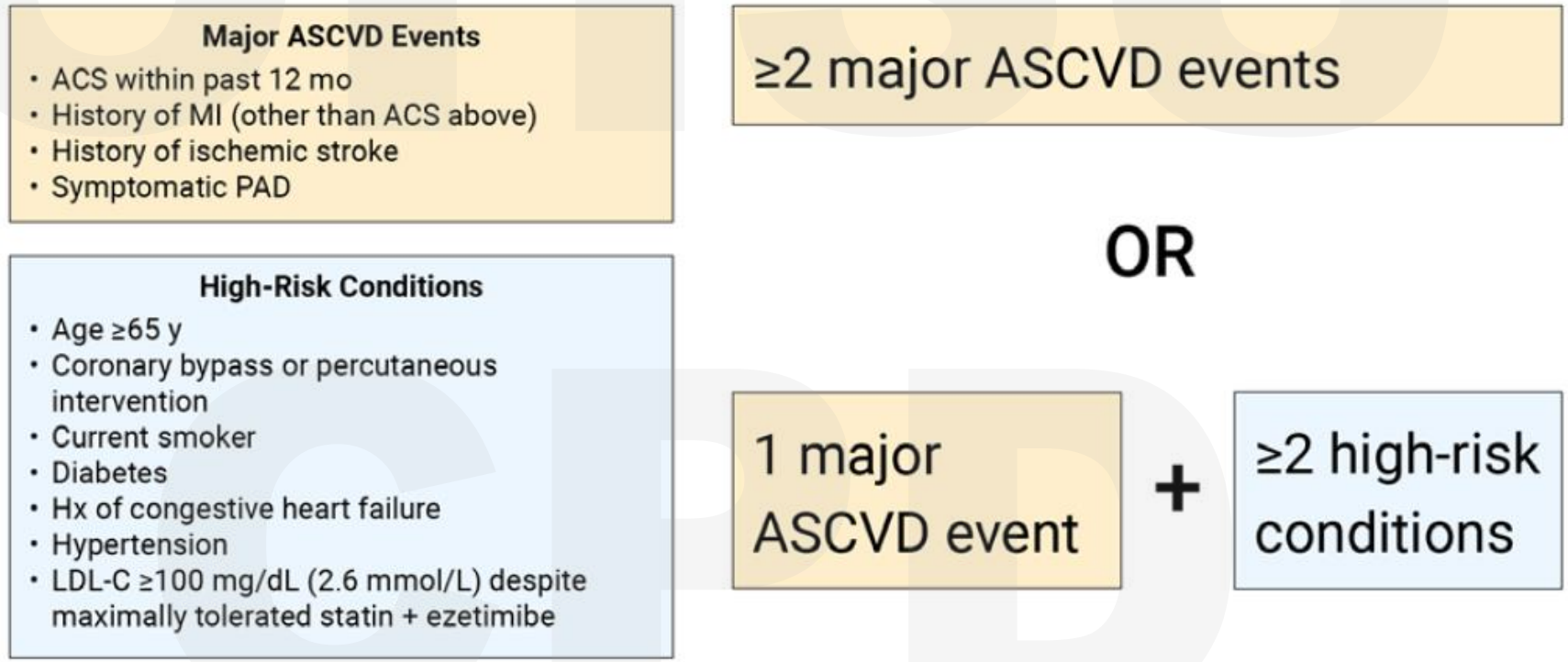


Secondary Prevention

Have Clinical ASCVD

Secondary ASCVD Prevention for Adults at Very High Risk

Clinical ASCVD: Criteria for Defining "At Very High Risk" in Adults



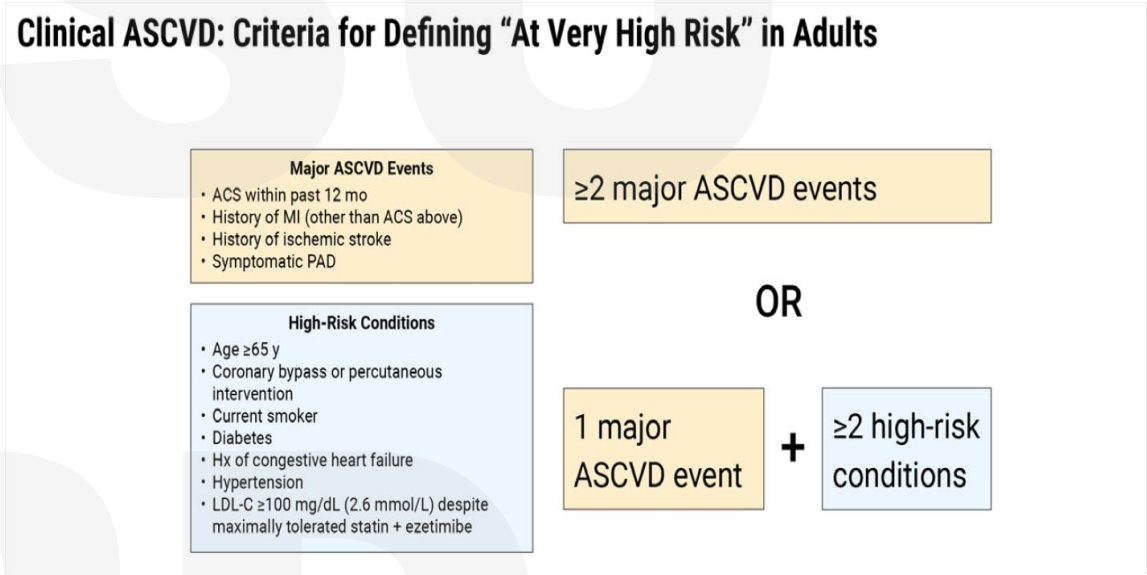
2ndary prevention

- ‘Not very high risk’
- LDL 55-70
  - Reasonable for either
    - <70 - class 1A
    - <55 – class 2a

Clinical ASCVD Not at Very High Risk*		
1	A	1. In adults with clinical ASCVD who are not at very high risk (Figure 10), high-intensity statin therapy should be initiated to achieve a ≥50% reduction in LDL-C and a goal of LDL-C <70 mg/dL (1.8 mmol/L) and non–HDL-C <100 mg/dL to reduce the risk of recurrent ASCVD events.[1–3]
2a	B-R	2. In adults with clinical ASCVD who are not at very high risk and on maximally tolerated statin therapy, it is reasonable to add ezetimibe, a PCSK9 mAb, or bempedoic acid (selection depending on degree of LDL-C lowering needed and patient preference) to achieve a goal of LDL-C <70 mg/dL (1.8 mmol/L) and non–HDL-C <100 mg/dL to reduce the risk of ASCVD events.[4–6]
2a	B-R	3. In adults with clinical ASCVD who are not at very high risk and on maximally tolerated statin therapy, it is reasonable to add ezetimibe, a PCSK9 mAb, or bempedoic acid (selection based on the degree of LDL-C lowering needed and patient preference) to achieve a goal LDL-C <55 mg/dL (1.4 mmol/L) and non–HDL-C <85 mg/dL (2.2 mmol/L) and to reduce the risk of ASCVD events.[4–6]

Summary 2ndary prevention

- LDL <70 for all
- LDL <55 for ‘Very High Risk’
  - **Acknowledge - Lots of ppl fitting into <55 group**
- Consider ApoB for further target

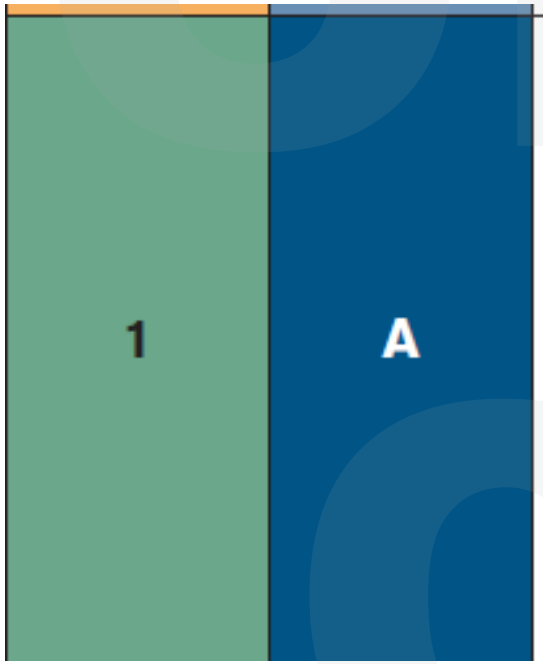


Older Adults - >75 YO

- 2.5 year life expectancy threshold for initiation of LLT

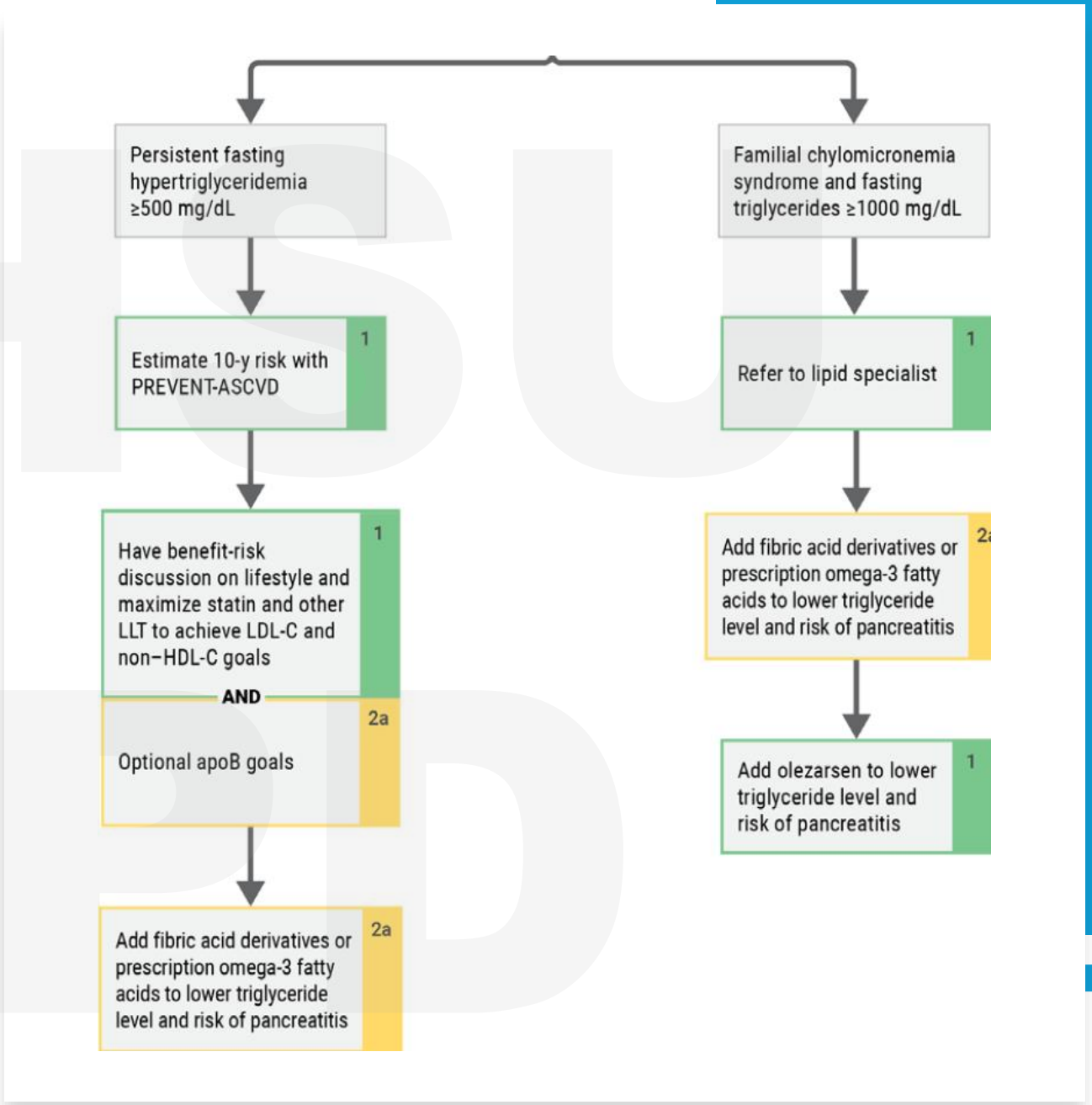
Recommendations for Older Adults (Continued)		
COR	LOE	Recommendations
2b	B-NR	2. In adults aged >75 y with an estimated life expectancy of at least 2.5 y, it may be reasonable to initiate moderate-intensity statin therapy after a clinician–patient discussion of potential benefits and risks to reduce ASCVD risk.[4–6,7]
2b	B-R	3. In patients with a life expectancy of <1 y, it may be reasonable to discontinue LDL-lowering therapy to avoid unnecessary medication use or adverse medication effects.[8,9]
2b	B-NR	4. In adults aged >75 y with an estimated life expectancy of at least 2.5 y, and for whom the decision regarding LLT is uncertain, it may be reasonable to measure CAC to reclassify those with minimal (1–10) or no CAC to avoid LLT.[10–13]

Triglycerides – Fasting <500 a threshold



5. In adults aged 40 to 75 y without a history of ASCVD or diabetes who have persistently elevated TG levels ≥150 to 499 mg/dL (≥1.7–5.6 mmol/L), it is recommended to estimate 10-y ASCVD risk by the PREVENT-ASCVD equations to guide the benefit-risk discussion regarding further optimization of diet and lifestyle management as well as the potential initiation of statin therapy to reduce ASCVD risk (Figure 11).[11]

Fasting triglycerides >500 & >1000



3.4. Measurement of Lp(a)

Recommendations for Measurement of Lp(a)
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	B-NR	1. In all adults, measurement of Lp(a) concentration is recommended at least once for ASCVD risk assessment.[1–4]
1	B-NR	2. In individuals with FH, premature ASCVD, or high Lp(a), cascade testing of first-degree family members for high Lp(a) concentration is recommended to identify those at increased ASCVD risk.[5–8]

4.2.10. Approach to Patients With Elevated Lp(a)

Recommendations for the Approach to Patients With Elevated Lp(a) Referenced studies that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	**B-NR**	1. In all individuals with elevated Lp(a) (≥125 nmol/L or ≥50 mg/dL), optimal early control of modifiable cardiovascular risk factors is recommended to reduce ASCVD risk.[1–4]
1	**B-R**	2. In individuals with clinical ASCVD and elevated Lp(a) who have not achieved LDL-C and non–HDL-C treatment goals on maximally tolerated statin therapy, the addition of a PCSK9 mAb with proven cardiovascular benefit is recommended to achieve treatment goals and reduce ASCVD risk.[4–7]

Table 4. ASCVD Risk Related to Lp(a) Concentrations*

Lp(a) concentration nmol/L (mg/dL)	ASCVD Relative Risk: Increase Compared With Population Median (20 nmol/L, 7 mg/dL)
430 nmol/L (180 mg/dL)	4-fold
350 nmol/L (150 mg/dL)	3-fold
250 nmol/L (100 mg/dL)	2 -fold
125 nmol/L (50 mg/dL)	1.4-fold
75–124 nmol/L (30-49 mg/dL)	1.2-fold
<75 nmol/L (<30 mg/dL)	Reference

Controversies

- Lp(a)
  - **Negatives**
    - Screening for something cannot specifically treat
    - Unclear outcome changing (primary prevention especially)
  - **Positives**
    - Risk modify earlier
    - Consensus across society guidelines ACC/AHA, ESC/EAS, EAS Consensus, and Canadian Cardiovascular Society
    - Future role for PCSK9 inhibitors?
    - Risk-based aspirin approach?

TABLE 13 Risk Enhancers

Risk Enhancers
History of premature ASCVD in a parent or sibling (onset age <55 y for men, <65 y for women)
Higher risk ancestry (eg, South Asian, Filipino)
High polygenic risk (if measured) (Section 4.2.3.5, **"Polygenic Risk Scores"**)
Chronic inflammatory diseases (eg, systemic lupus, rheumatoid arthritis, advanced psoriasis, inflammatory arthritis)
Lp(a) ≥125 nmol/L or ≥50 mg/dL
hsCRP ≥2 mg/L on >1 occasion (if measured)
TG persistently ≥175 mg/dL (2 mmol/L) (if nonfasting) and ≥150 mg/dL (1.7 mmol/L) (if fasting)
CKM syndrome
LDL-C persistently ≥160-189 mg/dL (4.1-4.9 mmol/L), non-HDL-C ≥190-219 mg/dL or apoB ≥120 mg/dL*
Reproductive risk markers (premature menopause, preeclampsia, gestational diabetes, gestational hypertension, preterm delivery; Section 4.2.3.4, **"Reproductive Risk Marker"**)

Note that all available information should be included in risk estimates derived from the PREVENT-ASCVD equations, including albuminuria, HbA1c, and zip code for assessment of neighborhood-level social determinants of health. Given the recent publication of the PREVENT-ASCVD equations, it remains to be demonstrated for most risk enhancers that risk is incremental to the PREVENT-ASCVD equations.

*Although LDL-C is not included in the PREVENT-ASCVD equations (total cholesterol and HDL-C are included), it is included here because persistent elevation of LDL-C may be a useful factor to include in risk-benefit discussions about LDL-C–lowering therapy, given that it is the target of that therapy. See Section 4.2.3.4, **"Reproductive Risk Marker,"** for more detail regarding reproductive risk factors.

Controversies

- CAC
  - Much expanded

Coronary Artery Calcium (CAC) - Redux: Part I. The "Power of Zero" Is a Myth

JAMES H. STEIN, MD

JAN 25, 2026

The clinical problem arises when a CAC score of zero is treated as a durable negative test rather than a conditional and time-limited observation. All probabilities are conditional and based on pre-test risk. In individuals with lifetime exposure to high LDL-C or other risk factors, CAC = 0 does not imply low lifetime risk or even low risk at 7 years. It simply indicates that calcification has not yet occurred. Using CAC to defer preventive therapy in this setting prioritizes short-term reassurance over long-term risk reduction.

This approach also creates **missed opportunities for prevention**. Observational studies consistently show that individuals with CAC = 0 who later experience ASCVD events have higher levels of modifiable risk factors, including LDL-C. Early statin therapy reduces cumulative atherosclerotic burden over time. Delaying treatment on the basis of a lagging imaging marker conflicts with the biological reality that atherosclerosis develops gradually and that earlier intervention yields greater absolute benefit.

CAC scanning also introduces nontrivial downsides that I have reviewed previously. The test adds cost, exposes patients to ionizing radiation, and frequently leads to incidental findings that trigger downstream imaging and testing without clear benefit. Anxiety and misinterpretation of results are common reasons for referral to cardiology clinics. **These costs and risks are justified only if the information meaningfully improves decision-making. In younger adults with elevated LDL-C, it does not.**

Therefore, the "power of zero" is best understood as an imperfect, temporary snapshot. In patients with substantial lifetime risk, it provides misguided reassurance. Sure, CAC = 0 occasionally can influence treatment decisions, particularly in highly risk-averse patients who are 65-75 years old, but these situations are far less common

Further…

- Contextual benefits
  - At a 3% 10-year risk, the absolute risk reduction is about -0.66%
    - NNT of ~150 over 10 years
- Supplements
  - SPORT trial cited
    - Rosuvastatin vs. supplements vs. placebo
    - Berberine specifically not included

4.1.5. Dietary Supplements

Recommendation for Dietary Supplements
Referenced studies that support the recommendation are summarized in the Evidence Table.

COR	LOE	Recommendation
3: No Benefit	**B-R**	1. In individuals with dyslipidemia, the use of dietary supplements is not recommended to lower LDL-C or TG based on limited and inconsistent data and/or limited benefits in lipid-lowering and reduction in ASCVD risk.[1–4]

DynaMed EBM Focus Editorial Team

Alan Ehrlich, MD, FAAFP

Ultimately, this guideline, like most, should be used to inform, nay, *guide* clinical care. The opportunity, and responsibility, for primary care providers is to use these recommendations as a starting point, while maintaining a clear focus on individualized decision-making, careful communication of absolute benefits and harms, and respect for what matters most to each patient.

Prostate Cancer Screening

Case

A 58 YOM presents for a preventative health visit. He inquires about PSA testing. He denies any family history of prostate cancer or other first degree family members with cancer, and he identifies as White European-American. He asks about expected mortality benefit from PSA screening… which you reply

A. PSA screening improves overall mortality
B. PSA screening worsens overall morality
C. PSA screening improves prostate-cancer related mortality
D. PSA screening worsens prostate-cancer related mortality

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 OCTOBER 30, 2025 VOL. 393 NO. 17

European Study of Prostate Cancer Screening — 23-Year Follow-up

Monique J. Roobol, Ph.D.,[1] Ivo I. de Vos, M.D.,[1] Marianne Månsson, Ph.D.,[2] Rebecka A. Godtman, M.D., Ph.D.,[2] Kirsi M. Talala, Ph.D.,[3] Elly den Hond, Ph.D.,[4] Vera Nelen, M.D., Ph.D.,[4] Arnauld Villers, M.D., Ph.D.,[5] Gregoire Poinas, M.D.,[6] Maciej Kwiatkowski, M.D., Ph.D.,[7-9] Stephen Wyler, Ph.D., M.D.,[7,8] Franz Recker, M.D., Ph.D.,[7] Donella Puliti, Ph.D.,[10] Giuseppe Gorini, M.D., Ph.D.,[10] Marco Zappa, Ph.D.,[10] Alvaro Paez, M.D.,[11] Marcos Lujan, M.D.,[12] Chris H. Bangma, M.D., Ph.D.,[1] Teuvo Tammela, M.D., Ph.D.,[13,14] Fritz H. Schröder, M.D., Ph.D.,[1] Sebastiaan Remmers, Ph.D.,[1] Jonas Hugosson, M.D., Ph.D.,[2] and Anssi Auvinen, M.D., Ph.D.,[15] for the ERSPC Investigators*

ERSPC = European Study of Prostate Cancer Screening

Started in 1993

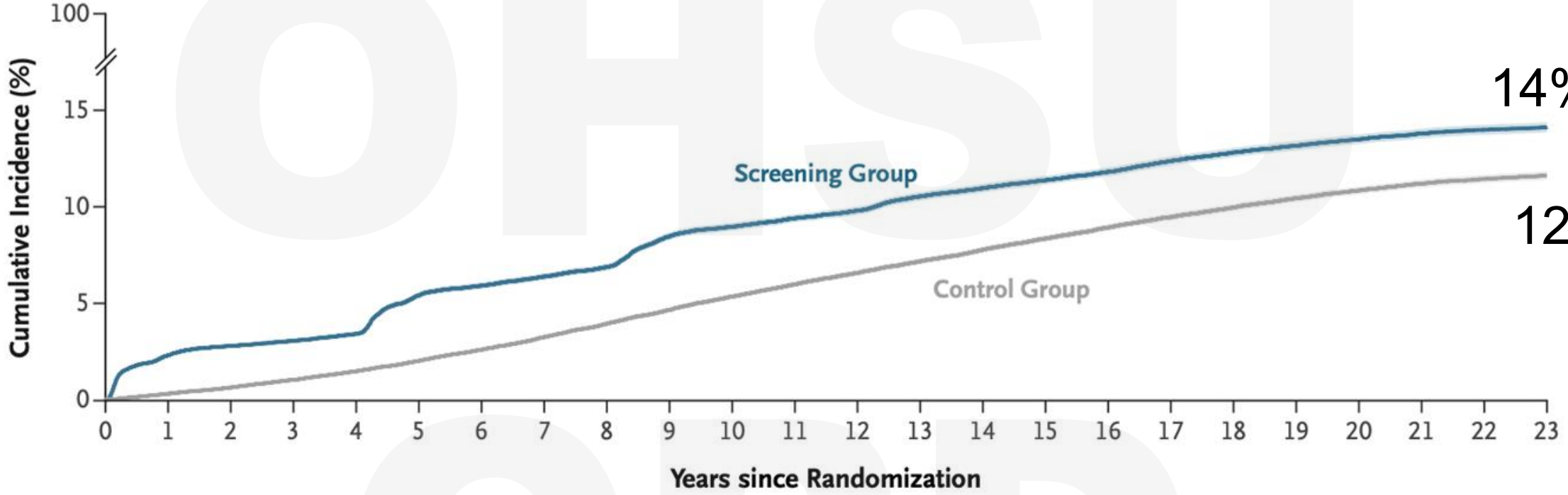
Eight European countries - Netherlands, Belgium, Sweden, Finland, Italy, Spain, Switzerland, France

Reported on **23 year outcomes** for **predefined core age group (55 - 69 years of age at the time of randomization).**

Intervention group - Mailed invitation for PSA Screening via national registry system

Control group - Routine care

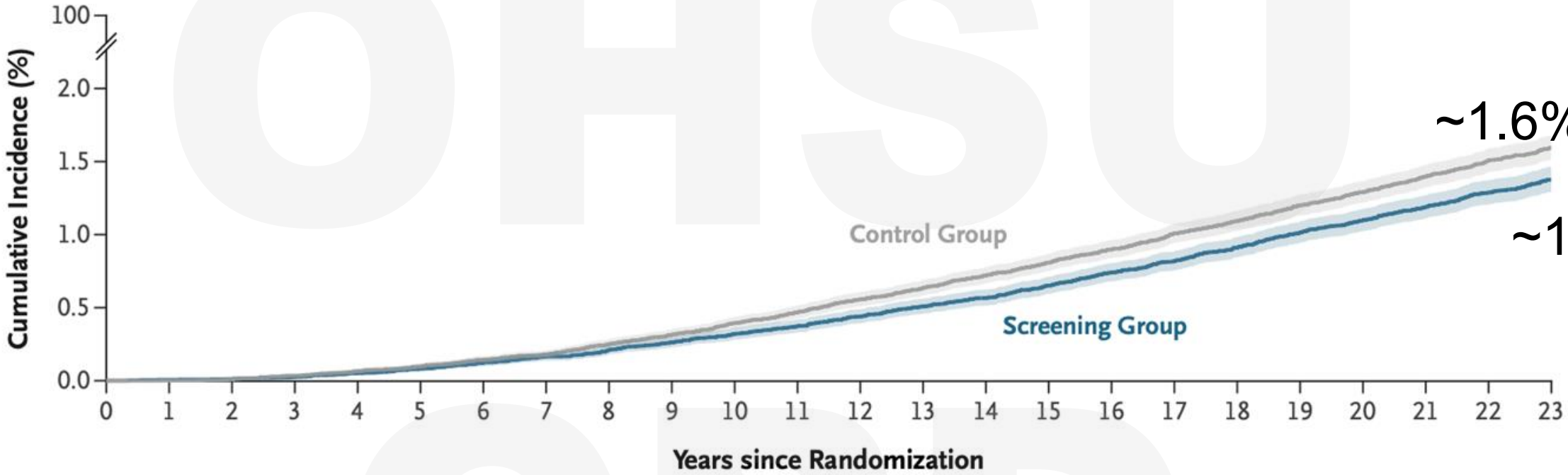
A Prostate Cancer Diagnosis



No. at Risk (no. of events)

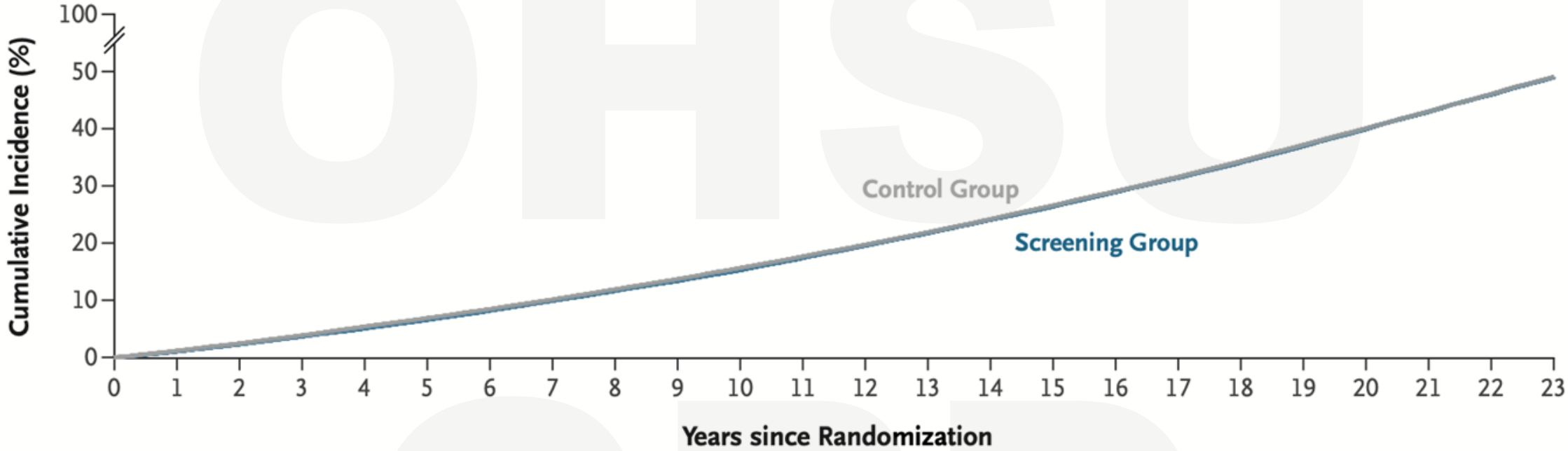
Screening group	72,888 (0)	64,312 (3929)	55,870 (6533)	46,834 (8292)	27,657 (9748)	11,843 (9995)
Control group	89,348 (0)	81,494 (1800)	71,036 (4777)	59,344 (7462)	32,961 (9529)	12,306 (9870)

B **Prostate Cancer–Specific Mortality**



No. at Risk (no. of events)						
Screening group	72,888 (0)	68,052 (60)	61,574 (233)	53,239 (475)	41,639 (796)	26,069 (969)
Control group	89,348 (0)	83,131 (87)	75,068 (352)	64,954 (722)	51,054 (1150)	31,634 (1385)

C **Other-Cause Mortality**



No. at Risk (no. of events)

Screening group	72,888 (1)	68,052 (4776)	61,574 (11,081)	53,239 (19,174)	41,639 (28,911)	26,069 (34,493)
Control group	89,348 (2)	83,131 (6130)	75,068 (13,928)	64,954 (23,672)	51,054 (35,588)	31,634 (42,444)

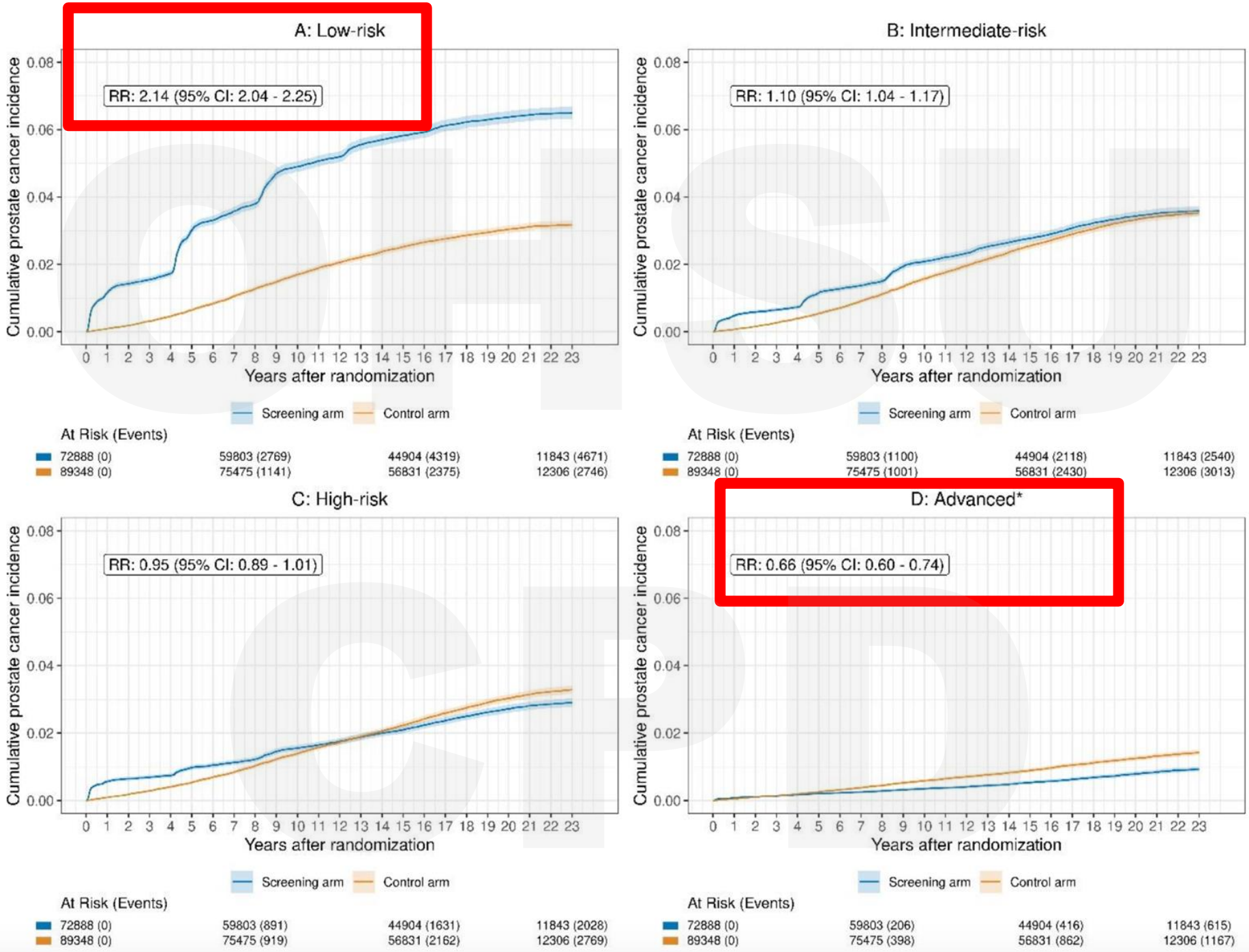
Table 2. Prostate Cancer Mortality According to Length of Follow-up.*

Length of Follow-up	Rate Ratio (95% CI)	Relative Risk Reduction	Absolute Risk Difference (95% CI)	No. Needed to Invite (95% CI)	No. Needed to Diagnose (95% CI)
		percent			
9 yr	0.83 (0.69–1.01)	17	0.05 (0.00–0.09)	1919 (903–14308)	73 (34–567)
11 yr	0.79 (0.67–0.93)	21	0.10 (0.03–0.15)	1041 (679–3486)	36 (22–118)
13 yr	0.80 (0.70–0.91)	20	0.12 (0.05–0.19)	803 (538–1927)	27 (18–62)
16 yr	0.82 (0.73–0.92)	18	0.16 (0.07–0.24)	628 (419–1481)	18 (12–45)
23 yr	0.87 (0.80–0.95)	13	0.22 (0.11–0.33)	456 (306–943)	12 (8–26)

* Confidence intervals (CIs) are presented without adjustment for multiplicity and should not be used to infer definitive statistical significance or for formal hypothesis testing.

ERSPC - Details

- 16% of PSA tests yielded elevated results
- **24%** of the subsequent biopsies **confirmed prostate cancer**
- Concern **overdiagnosis** and subsequent **overtreatment**
  - Excess incidence of 27 cases per 1000 men
  - **Doubling in detection of low-risk cancers**

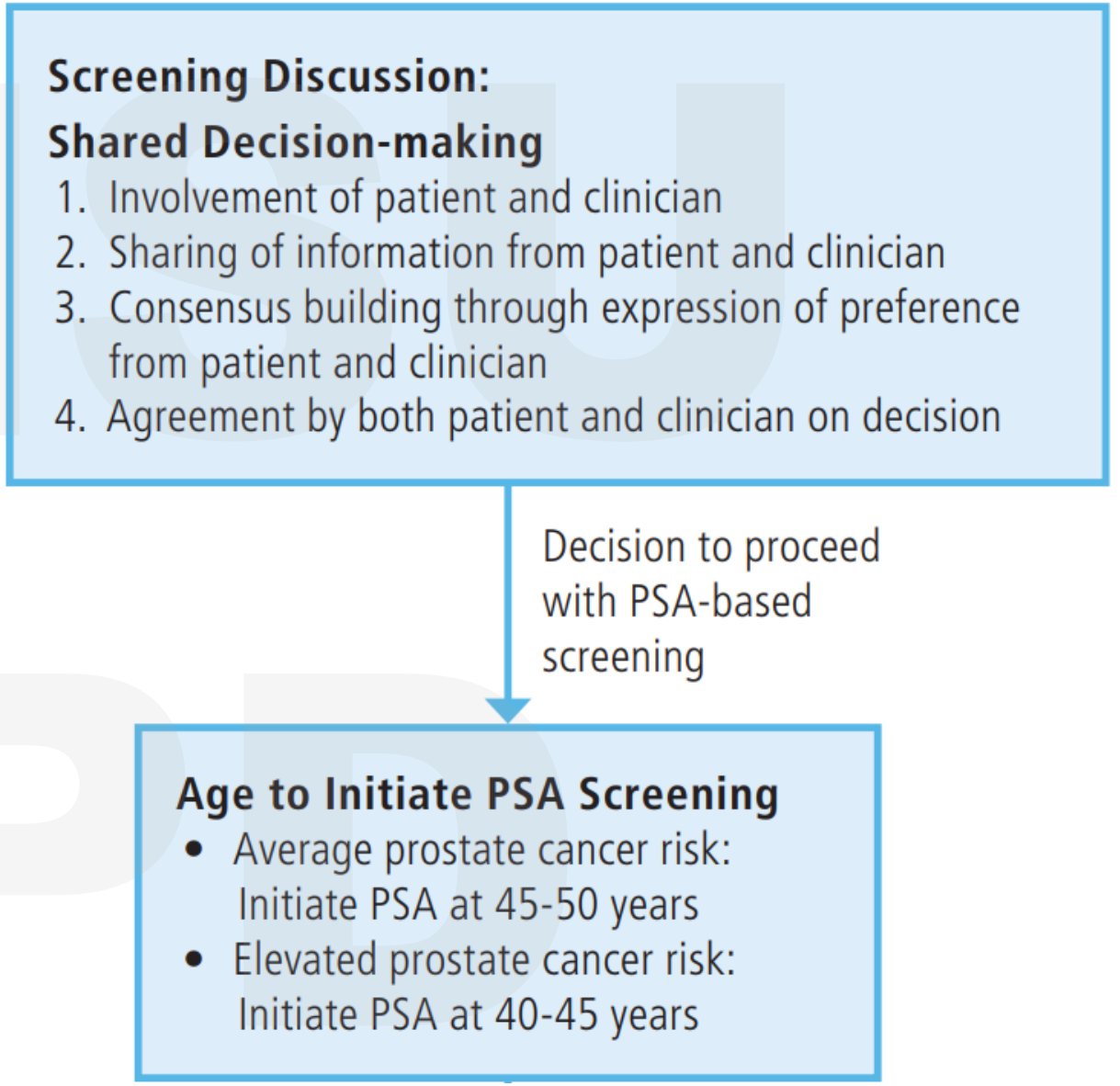

A: Low-risk
RR: 2.14 (95% CI: 2.04 - 2.25)
B: Intermediate-risk
RR: 1.10 (95% CI: 1.04 - 1.17)
C: High-risk
RR: 0.95 (95% CI: 0.89 - 1.01)
D: Advanced*
RR: 0.66 (95% CI: 0.60 - 0.74)
Cumulative prostate cancer incidence
0.00
0.02
0.04
0.06
0.08
0 1 2 3 4 5 6 7 8 9 10 11 12 13 14 15 16 17 18 19 20 21 22 23
Years after randomization
Screening arm
Control arm
At Risk (Events)
72888 (0) 59803 (2769) 44904 (4319) 11843 (4671)
89348 (0) 75475 (1141) 56831 (2375) 12306 (2746)
72888 (0) 59803 (1100) 44904 (2118) 11843 (2540)
89348 (0) 75475 (1001) 56831 (2430) 12306 (3013)
72888 (0) 59803 (891) 44904 (1631) 11843 (2028)
89348 (0) 75475 (919) 56831 (2162) 12306 (2769)
72888 (0) 59803 (206) 44904 (416) 11843 (615)
89348 (0) 75475 (398) 56831 (862) 12306 (1167)

What to do!?

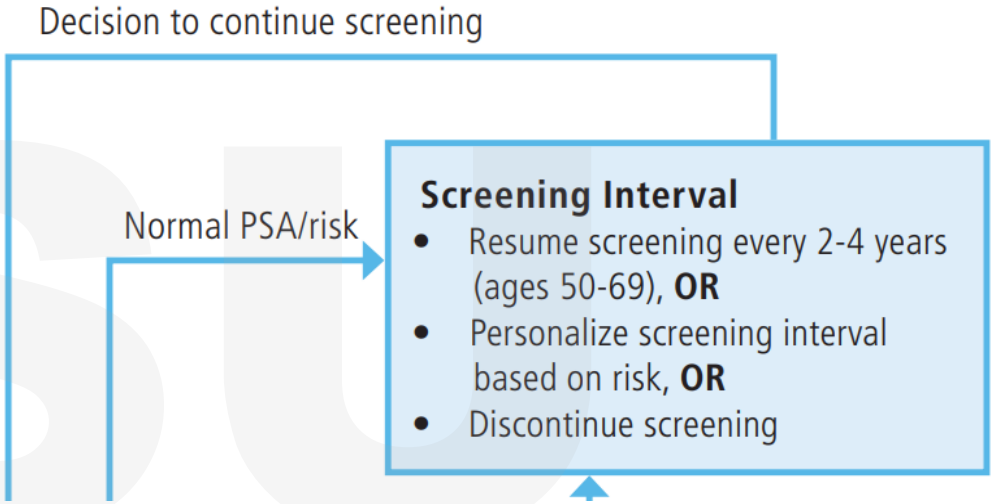
AUA 2026

Shared decision-making

- Higher risk patients
  - Black ancestry
  - Family history
    - Prostate (dx <65 especially)
    - Breast, ovarian, pancreatic
    - BRCA carriers
  - Agent Orange exposure



AUA 2026



Consider lengthening/stopping for:

- 60+ with PSA <1ng/mL - very low risk of mets/death
- 75+ with PSA <3 – very low risk of mets/death
- Life expectancy <10y – very low risk of prostate cancer being the cause of death

AUA 2026 Updates

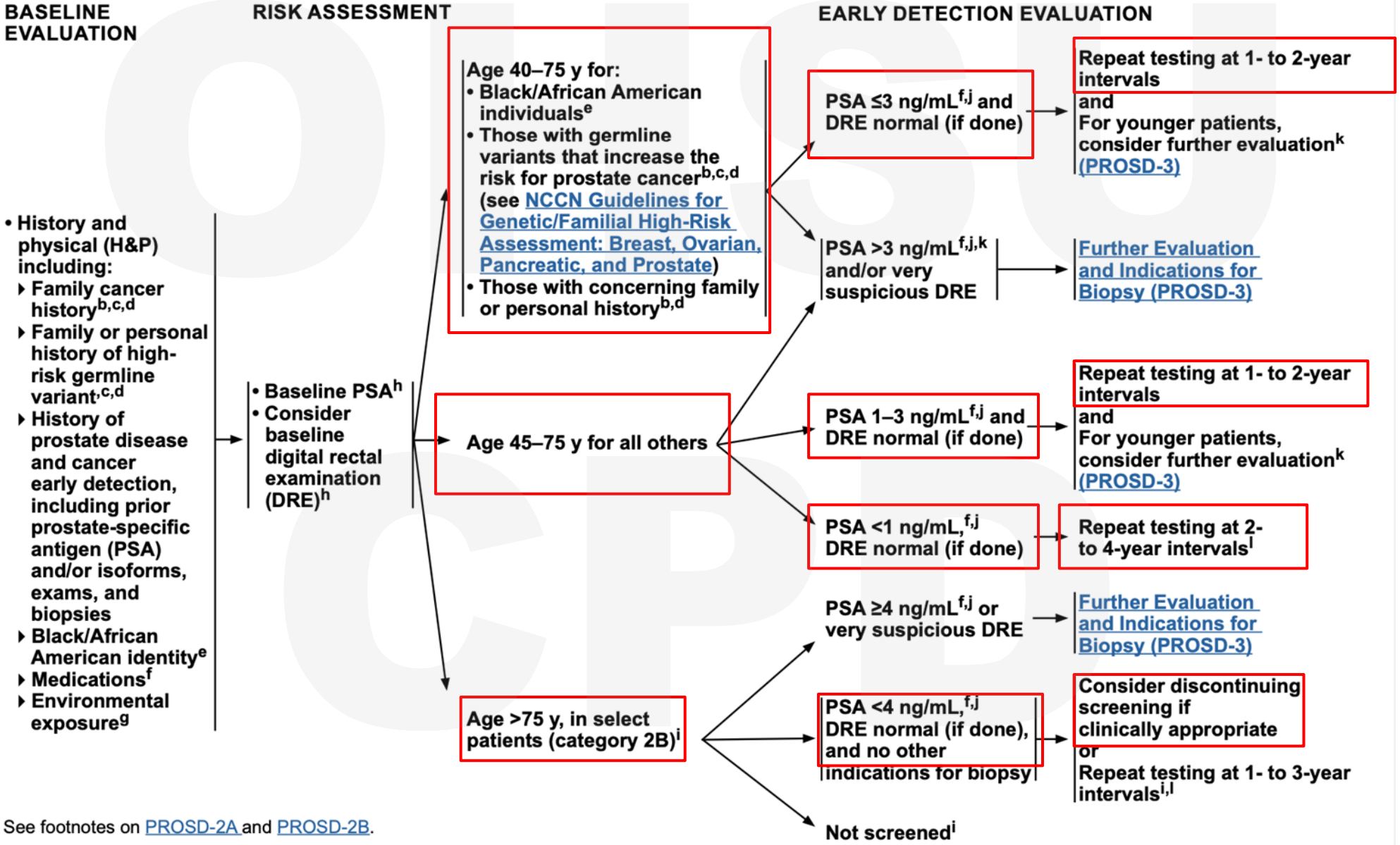
- Role of Prostate MRI
  - Evidence Level from Grade B -> Grade A

13. Clinicians may use MRI prior to initial biopsy to increase the detection of GG2+ prostate cancer. (*Conditional Recommendation*; *Evidence Level: Grade A*)

National Comprehensive Cancer Network®

NCCN Guidelines Version 1.2026
Prostate Cancer Early Detection

NCCN Guidelines Index
Table of Contents
Discussion



See footnotes on PROSD-2A and PROSD-2B.

Take home points

PSA is complicated!

Summary of evidence - **likely benefit for reduction of prostate cancer mortality without benefit in overall survival**

Combined AUA 2026 / NCCN 2026 highlights

- **Higher risk** – Shared decision making ~40-45
- **Average risk** - Shared decision making ~45-50
- **Stop screening -** ~70-75 for most people with prostates
- **Contextual screening intervals**
  - Higher risk q1-2 years
  - Average risk
    - IF PSA >1 -> consider 1-2 years (NCCN) vs. 1-4 years (AUA)
    - IF PSA < 1 -> consider 2-4 years (NCCN + AUA)

AUA 2026 + NCCN 2026

Consider lengthening/stopping for:

- 60+ with PSA <1ng/mL - very low risk of mets/death
- 75+ with PSA <3 – very low risk of mets/death
- Life expectancy <10y – very low risk of prostate cancer being the cause of death

Hypertension

2025 AHA/ACC/AANP/AAPA/ABC/ACCP/ACPM/AGS/AMA/ASPC/NMA/PCNA/SGIM Guideline for the Prevention, Detection, Evaluation and Management of High Blood Pressure in Adults: A Report of the American College of Cardiology/American Heart Association Joint Committee on Clinical Practice Guidelines

Developed in Collaboration With and Endorsed by American Academy of Physician Associates; American Association of Nurse Practitioners; American College of Clinical Pharmacy; American College of Preventive Medicine; American Geriatrics Society; American Medical Association; American Society of Preventive Cardiology; Association of Black Cardiologists; National Medical Association; Preventive Cardiovascular Nurses Association; and the Society of General Internal Medicine.

Table 4. Categories of Blood Pressure in Adults*

	SBP		DBP
BP Category			
Normal	<120 mm Hg	and	<80 mm Hg
Elevated	120 to 129 mm Hg	and	<80 mm Hg
Hypertension			
Stage 1	130 to 139 mm Hg	or	80 to 89 mm Hg
Stage 2	≥140 mm Hg	or	≥90 mm Hg

BP indicates blood pressure (based on an average of ≥2 careful readings obtained on ≥2 occasions, as detailed in Section 3 ("Evaluation and Diagnosis"); DBP, diastolic blood pressure; and SBP, systolic blood pressure.

Ambulatory BP Monitoring - ABPM
Home BP Monitoring - HBPM

3.1.4. ABPM and HBPM

Recommendations for ABPM and HBPM
Referenced studies that support the recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	A	1. In adults with suspected hypertension, out-of-office BP measurements by either ABPM or HBPM are recommended to confirm the diagnosis of hypertension.[1,2]
1	A	2. In adults who are taking antihypertensive medication, HBPM is recommended for monitoring the titration of BP-lowering medication, along with cointerventions such as patient education, telehealth counseling, and clinical interventions.[2–6]

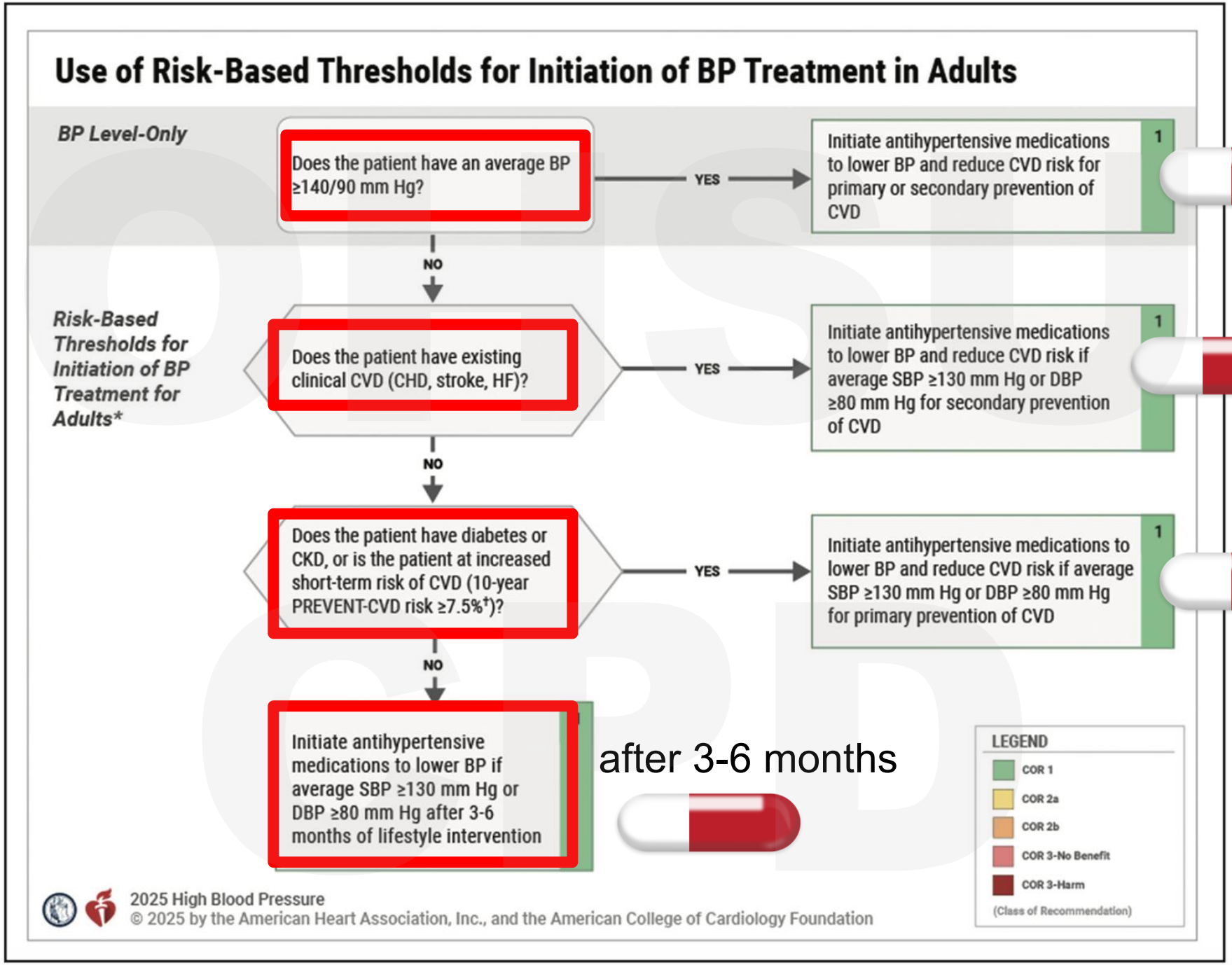


Figure 6. Use of Risk-Based Thresholds for Initiation of BP Treatment in Adults.

CVD | ASCVD | Heart Failure

Sex*
(•) Male ( ) Female

Current Smoking
Any cigarette use within the last 30 days
(•) No ( ) Yes

Lipid-lowering medication
Current use of statin medication to lower cholesterol
(•) No ( ) Yes

Age (years)*
30-79

HDL Cholesterol (mg/dL)*
20-100

BMI (kg/m^2)*
18.5-39.9

Total Cholesterol (mg/dL)*
130-320

SBP (mmHg)*
90-200

eGFR (mL/min/1.73m^2)*
15-140

Diabetes
Any history of diabetes.
(•) No ( ) Yes

Anti-hypertensive medication
Current use of any medication for hypertension
(•) No ( ) Yes

The following three predictors are optional for further personalization of risk assessment. When they are clinically indicated or available,
If avaliable or indicated, select "Yes" and enter the value.

UACR (mg/g)
UACR is clinically indicated for individuals with chronic kidney disease, diabetes, or hypertension
(•) No ( ) Yes

HbA1C
HbA1c is clinically indicated for individuals with diabetes, prediabetes, overweight, or obesity, or those with history of gestational diabetes
(•) No ( ) Yes

Zip Code
valid 5-digit zip code is needed to estimate social deprivation index [SDI]
(•) No ( ) Yes

5.2.7. BP Goal for Patients With Hypertension

Recommendations for BP Goal for Patients With Hypertension
Referenced studies that support recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	A	1. In adults with confirmed hypertension who are at increased risk* for CVD, an SBP goal of at least <130 mm Hg, with encouragement to achieve SBP <120 mm Hg, is recommended to reduce the risk of cardiovascular events and total mortality.[1–4]

Recommendations for Lifestyle and Psychosocial Approaches (Continued)

COR	LOE	Recommendations
2a	A	4. In adults with or without hypertension, potassium-based salt substitutes† can be useful to prevent or treat elevated BP and hypertension, particularly for patients in whom salt intake is related mostly to food preparation or flavoring at home, except in the presence of CKD or use of drugs that reduce potassium excretion where monitoring of serum potassium levels is indicated.‡[20–24]

The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 SEPTEMBER 16, 2021 VOL. 385 NO. 12

Effect of Salt Substitution on Cardiovascular Events and Death

B. Neal, Y. Wu, X. Feng, R. Zhang, Y. Zhang, J. Shi,* J. Zhang, M. Tian, L. Huang, Z. Li, Y. Yu, Y. Zhao, B. Zhou, J. Sun, Y. Liu, X. Yin, Z. Hao, J. Yu, K.-C. Li, X. Zhang, P. Duan, F. Wang, B. Ma, W. Shi, G.L. Di Tanna, S. Stepien, S. Shan, S.-A. Pearson, N. Li, L.L. Yan, D. Labarthe, and P. Elliott

5.2.4. Choice of Initial Monotherapy Versus Initial Combination Drug Therapy

Recommendations for Choice of Initial Monotherapy Versus Initial Combination Drug Therapy
Referenced studies that support the recommendations are summarized in the Evidence Table.

COR	LOE	Recommendations
1	B-R	1. In adults with stage 2 hypertension (SBP ≥ 140 mm Hg and DBP ≥90 mm Hg), initiation of antihypertensive drug therapy with 2 first-line agents of different classes, ideally in a single-pill combination (SPC), is recommended to improve BP control and adherence.[1–6]

3.2.3. Secondary Forms of Hypertension

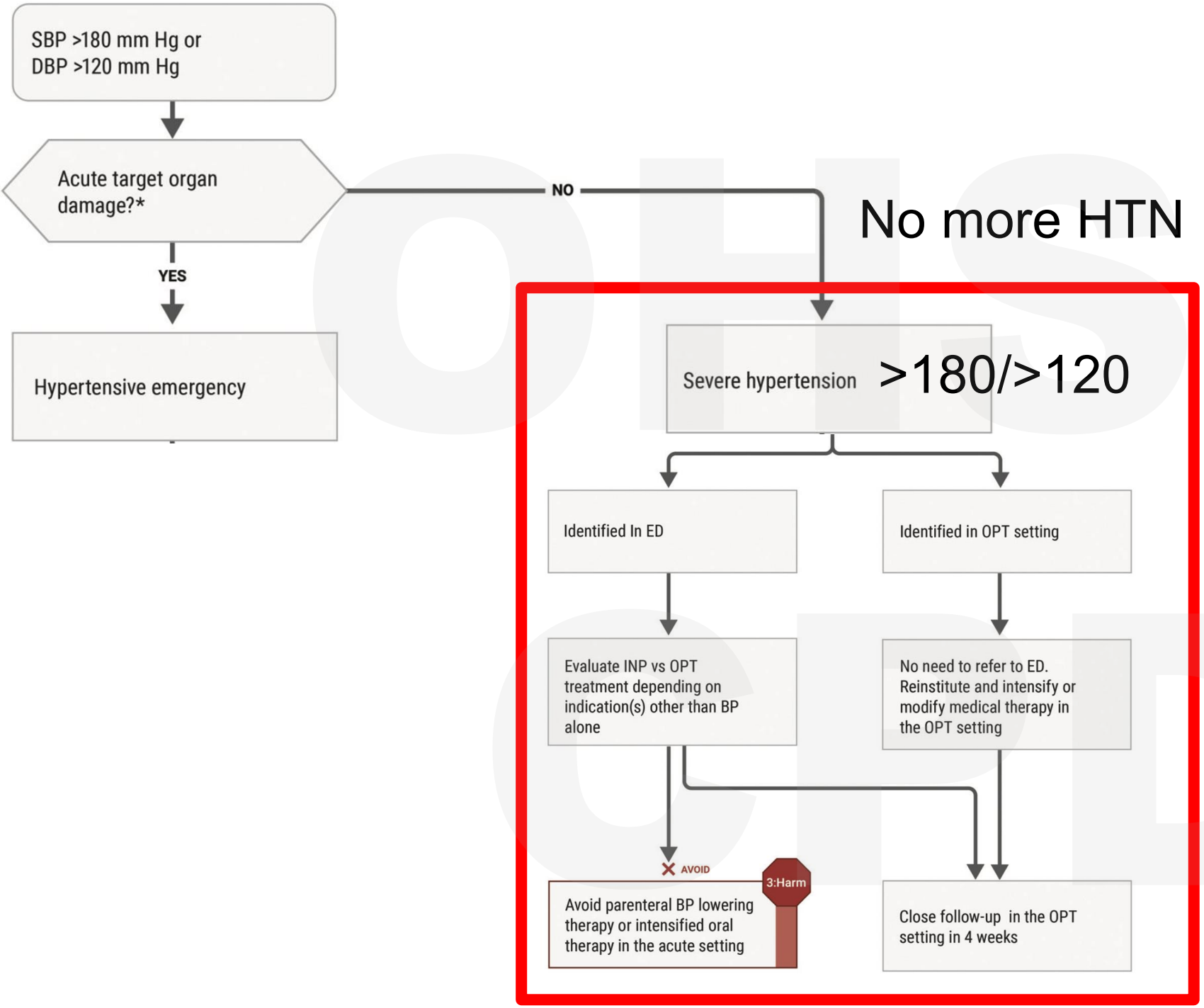
Recommendations for Secondary Forms of Hypertension References that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	C-EO	1. In adults with hypertension, screening for specific forms of secondary hypertension is recommended when clinical suspicion is present (Table 10, Figure 5) to increase rates of detection, diagnosis, and specific targeted therapy.
1	B-NR	2. In adults with resistant hypertension, screening for primary aldosteronism is recommended regardless of whether hypokalemia is present to increase rates of detection, diagnosis, and specific targeted therapy.[1,2]

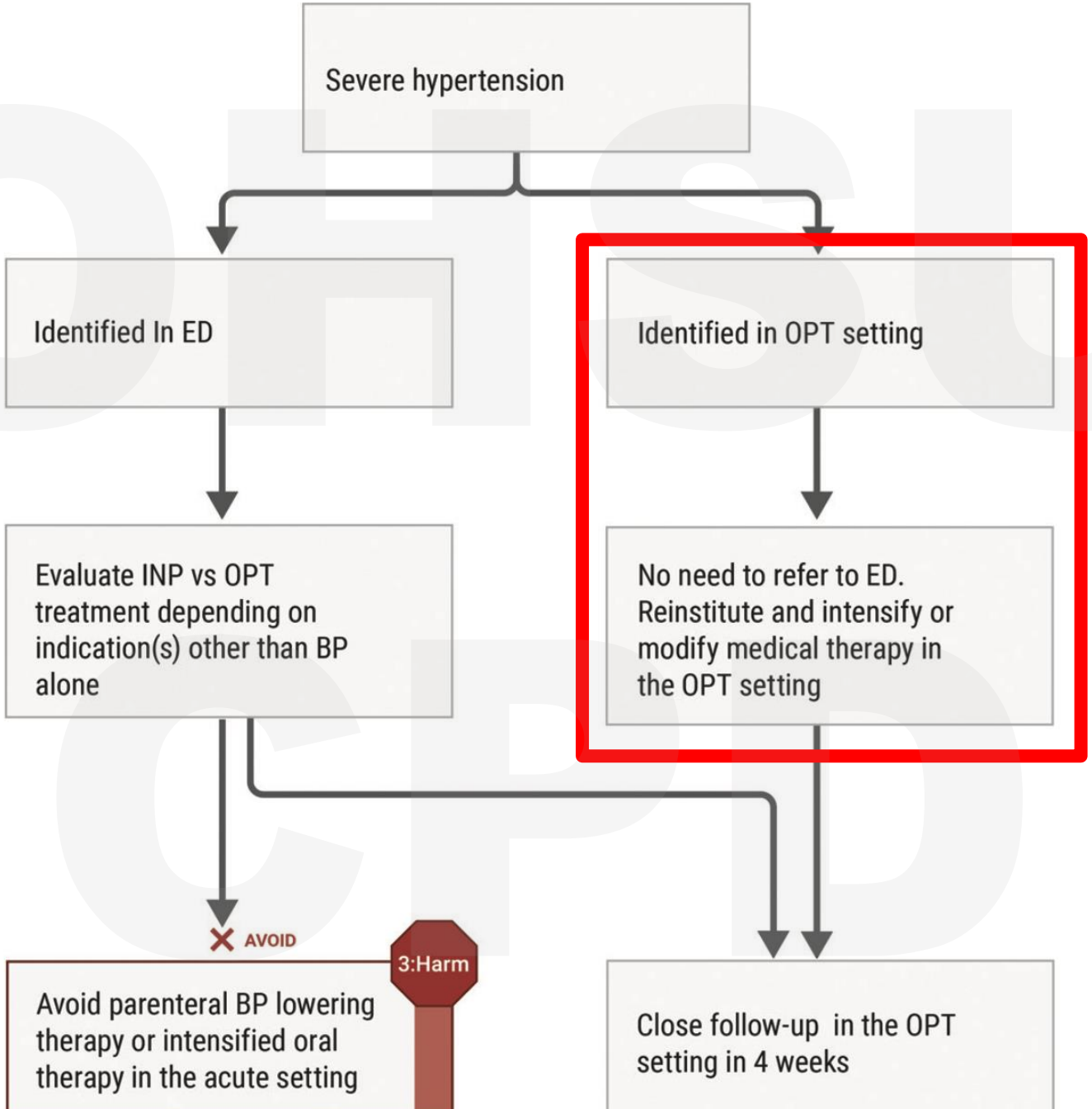
3.2.3.1. Primary Aldosteronism

Recommendations for Primary Aldosteronism		
COR	**LOE**	**Recommendations**
1	**C-EO**	1. In adults with hypertension, screening for primary aldosteronism is recommended in the presence of any of the following conditions to increase rates of detection, diagnosis, and specific targeted therapy: resistant hypertension (regardless of whether hypokalemia is present), hypokalemia (spontaneous or diuretic induced), OSA, incidentally discovered adrenal mass, family history of early-onset hypertension, or stroke at a young age (<40 years).
2b	**C-EO**	2. In adults with stage 2 hypertension, screening for primary aldosteronism may be considered to increase rates of detection, diagnosis, and specific targeted therapy.

Renal Denervation		
2b	**B-R**	4. In carefully selected patients with systolic and diastolic hypertension (office SBP 140-180 mm Hg and DBP ≥90 mm Hg) and eGFR ≥40 mL/min/1.73 m^2 who have resistant hypertension despite optimal treatment, or intolerable side effects to additional antihypertensive drug therapy, renal denervation (RDN) may be reasonable as an adjunct treatment to BP medications and lifestyle modification to reduce BP.[12–14]

3: Harm	**B-NR**	4. For adults with severe hypertension (>180/120 mm Hg) who are hospitalized for noncardiac conditions without evidence of acute target organ damage, intermittent use of additional IV or oral antihypertensive medications are not recommended to acutely reduce BP.[8,10,11]


SBP >180 mm Hg or
DBP >120 mm Hg
Acute target organ damage?*
NO
YES
Hypertensive emergency
No more HTN Urgency!!!
Severe hypertension >180/>120
Identified In ED
Identified in OPT setting
Evaluate INP vs OPT treatment depending on indication(s) other than BP alone
No need to refer to ED. Reinstitute and intensify or modify medical therapy in the OPT setting
AVOID
3:Harm
Avoid parenteral BP lowering therapy or intensified oral therapy in the acute setting
Close follow-up in the OPT setting in 4 weeks


Severe hypertension
Identified In ED
Identified in OPT setting
Evaluate INP vs OPT treatment depending on indication(s) other than BP alone
No need to refer to ED. Reinstitute and intensify or modify medical therapy in the OPT setting
AVOID
3:Harm
Avoid parenteral BP lowering therapy or intensified oral therapy in the acute setting
Close follow-up in the OPT setting in 4 weeks

AHA 2025 HTN - Take Home Points

Overarching blood pressure treatment goal is **<130/<80 mm Hg for all adults**

Continued emphasis on **risk-based approach** (prior CVD, **PREVENT score**)

Home BP readings (HBPM) to confirm and **manage** given greater correlation to CVD outcomes

Preference for **single-pill combination (SPC) for stage 2 HTN (>140/>90)**

Hypertensive Urgency no more!

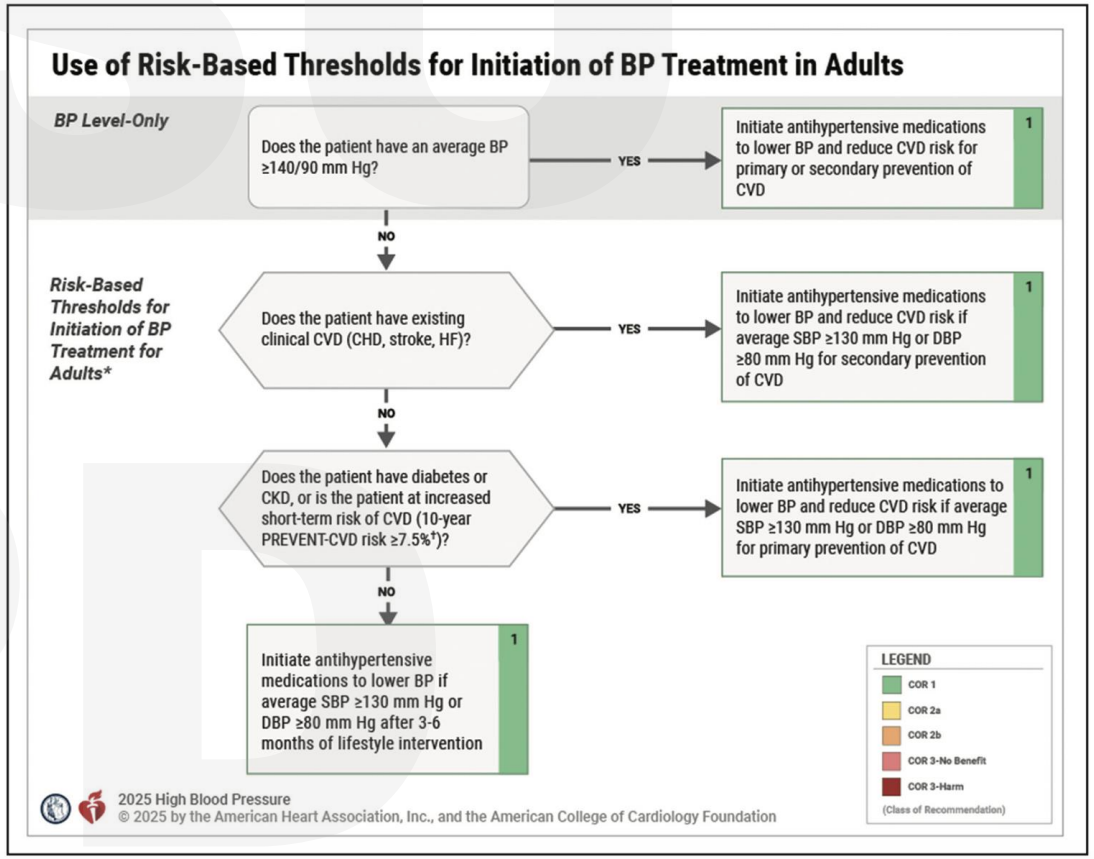


Figure 6. Use of Risk-Based Thresholds for Initiation of BP Treatment in Adults.

Quick Hits

Afib and Coffee
Extended Apixaban after DVT/PE
Shingles and Dementia
GLP 1 and MASH

JAMA | Original Investigation

Caffeinated Coffee Consumption or Abstinence to Reduce Atrial Fibrillation
The DECAF Randomized Clinical Trial

Christopher X. Wong, MBBS, MPH, PhD; Christopher C. Cheung, MD, MPH; Gabrielle Montenegro, BA; Hannah H. Oo, BS; Isabella J. Peña, BA; Janet J. Tang, MPH, PhD; Samuel J. Tu, MBBS; Grace Wall, BA; Thomas A. Dewland, MD; Joshua D. Moss, MD; Edward P. Gerstenfeld, MD; Zian H. Tseng, MD, MAS; Henry H. Hsia, MD; Randall J. Lee, MD, PhD; Jeffrey E. Olgin, MD; Vasanth Vedantham, MD; Melvin M. Scheinman, MD; Catherine Lee, PhD; Prashanthan Sanders, MBBS, PhD; Gregory M. Marcus, MD, MAS

DECAF - Context

- Caffeinated coffee has historically been **considered proarrhythmic**

- **Observational data is conflicting**

*"An **accurate understanding of any effect of caffeinated coffee** on **atrial fibrillation** would be of **great interest to patients and physicians alike**"*

DECAF Trial Overview

Randomized Control Trial

Patient with **persistent** atrial fibrillation or Fib/Flutter who had **successful cardioversion**

Allocated to
1) **Coffee consumption** group
2) **Coffee abstinence** group

Primary Outcome - **Recurrence** of atrial fibrillation OR atrial flutter **>30 seconds**

DECAF Trial Overview

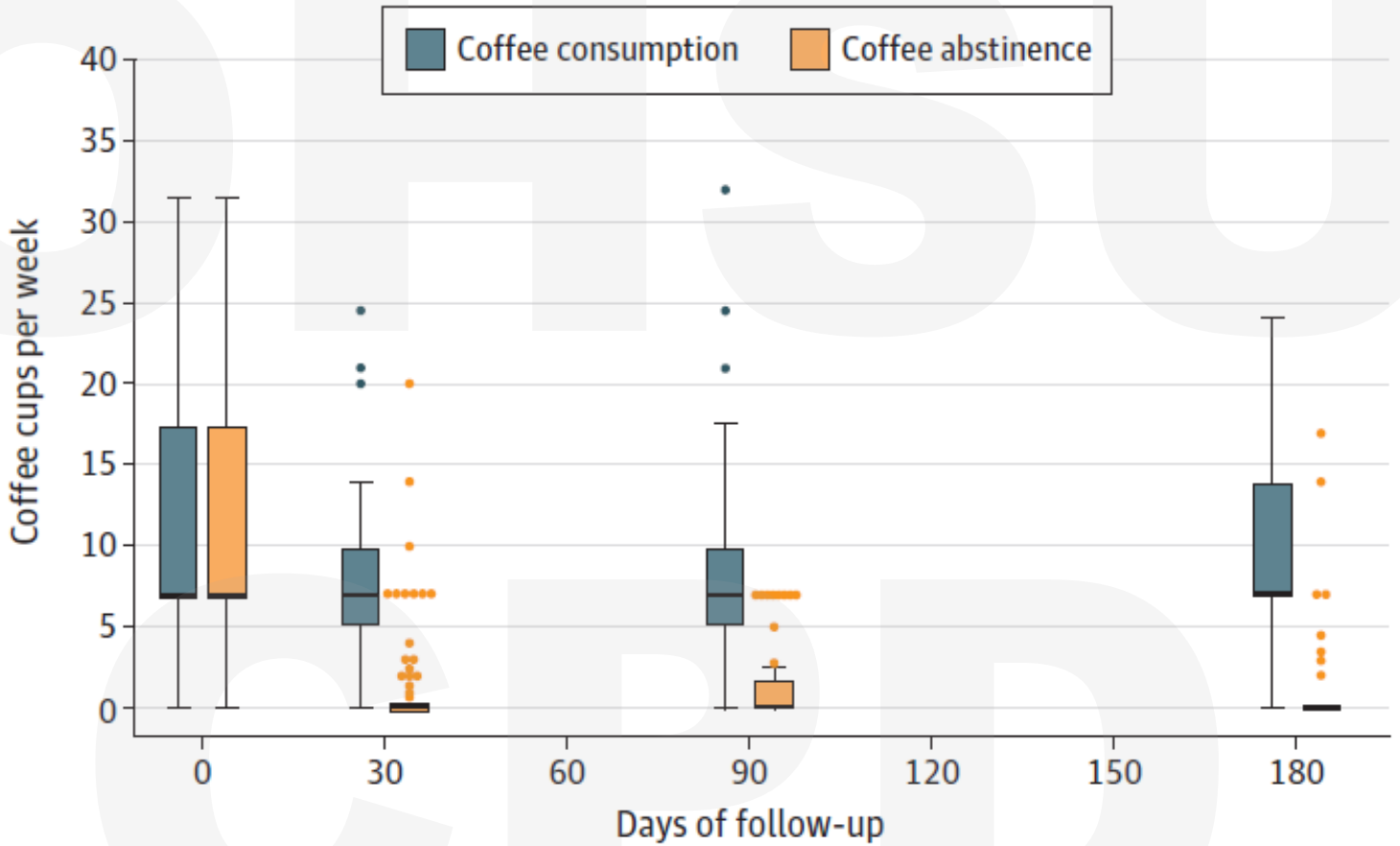
Coffee consumption group

- Encouraged to drink at least 1 cup of caffeinated coffee or espresso shot
- Continue other caffeine-containing products per their usual lifestyle
- Not intentionally increase or decrease

Coffee abstinence group

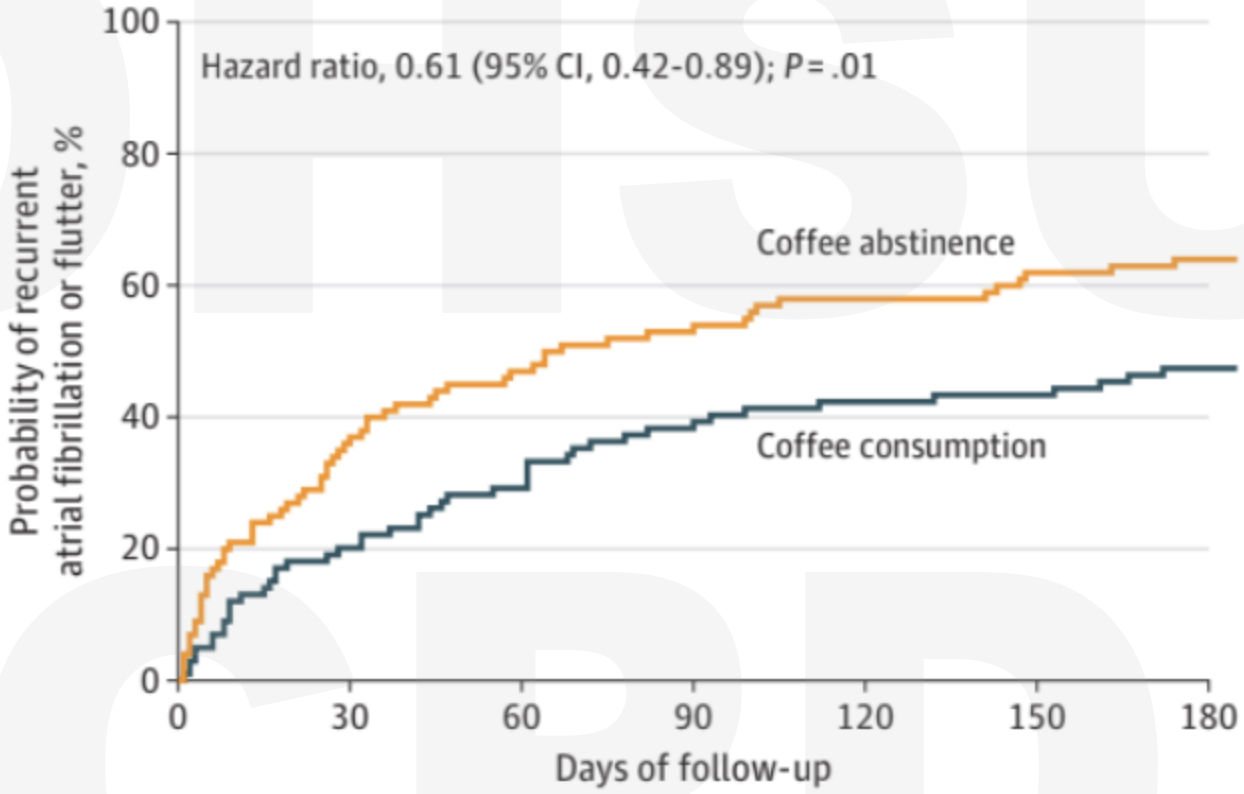
- encouraged to completely abstain from coffee, decaffeinated coffee, and other caffeine products

Figure 2. Changes in Coffee Intake by Randomization Group



No. at risk							
Coffee consumption	100	79	70	61	57	56	52
Coffee abstinence	100	64	53	47	42	38	36

Figure 3. Time to Recurrence of Atrial Fibrillation or Flutter



No. at risk							
Coffee consumption	100	79	70	61	57	56	52
Coffee abstinence	100	64	53	47	42	38	36

DECAF - Discussion

Possible mechanisms

- Caffeine has adenosine-antagonism properties -> Adenosine can induce AF through ‘sympathoexcitatory effects”
- Coffee with anti-inflammatory properties
- Increased physical activity (extrapolation from CRAVE trial)

Take Home Points

Typical consumption - averaging 1 cup a day of caffeinated coffee was associated with **less recurrence of detected AF / Flutter at 6 months**

The NEW ENGLAND JOURNAL of MEDICINE

ORIGINAL ARTICLE

Apixaban for Extended Treatment of Provoked Venous Thromboembolism

Gregory Piazza, M.D.,[1,2] Behnood Bikdeli, M.D.,[1-3] Arvind K. Pandey, M.D.,[2]
Darsiya Krishnathasan, M.S.,[1] Candrika D. Khairani, M.D.,[1] Antoine Bejjani, M.D.,[1,4]
Ruth H. Morrison, R.N., B.S.N.,[1] Heather Hogan, R.N., B.S.N.,[1]
Sina Rashedi, M.D., M.P.H.,[1] Mariana Pfeferman, M.D.,[1]
Junyang Lou, M.D., Ph.D.,[1,2] John Fanikos, R.P.H.,[1,5] Nicole Porio, B.A.,[1]
Lisa Rosenbaum, M.D.,[6] Piotr Sobieszczyk, M.D.,[2] Zhou Lan, Ph.D.,[1,7]
Marie Gerhard-Herman, M.D.,[2] Umberto Campia, M.D.,[1,2] and
Samuel Z. Goldhaber, M.D.,[1,2] for the HI-PRO Trial Investigators*

Study design

1. VTE
2. Transient provoking factor
3. Chronic persistent risk factor
4. 3 months of anticoagulation

Apixaban 2.5 mg BID

300 patients

Placebo

300 patients

Chronic Persistent Risk Factors

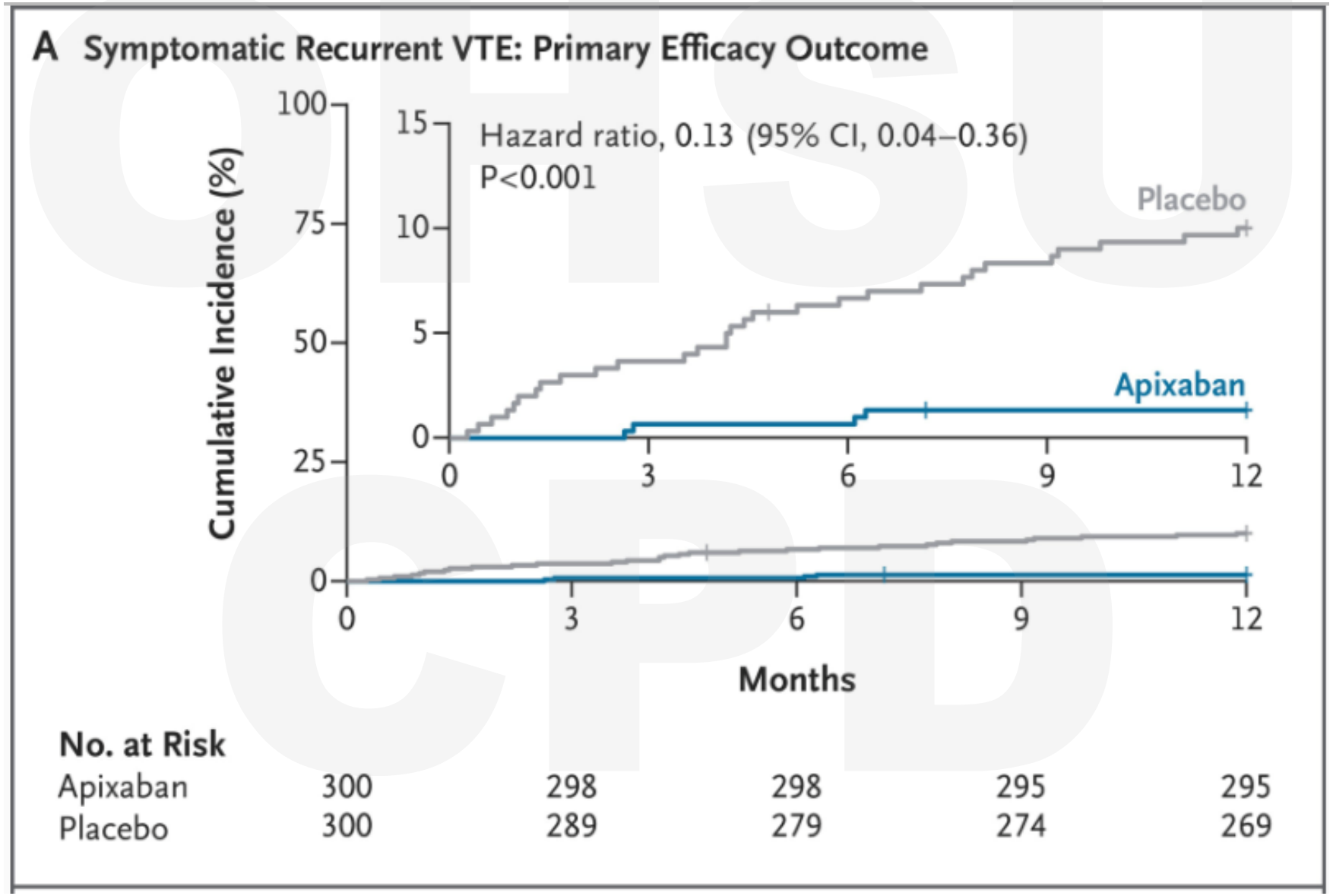
- Persistent immobility (defined as paralysis, other inability to ambulate freely, bed-bound, wheelchair-bound)
- Obesity (defined as BMI ≥ 30 kg/m^2)
- Heart failure (systolic, diastolic, or combined)
- Chronic lung disease (COPD, asthma, interstitial lung disease)
- Chronic kidney disease (eGFR <60 $mL/min/1.72m^2$)
- Chronic inflammatory/autoimmune disorder (inflammatory arthritis, vasculitis, inflammatory bowel disease, chronic infection)
- Atherosclerotic cardiovascular disease (coronary, cerebrovascular, or peripheral artery disease) (up to 35% in each study group may have atherosclerotic cardiovascular disease as a qualifying persistent risk factor)

Chronic Persistent Risk Factors in HI-PRO

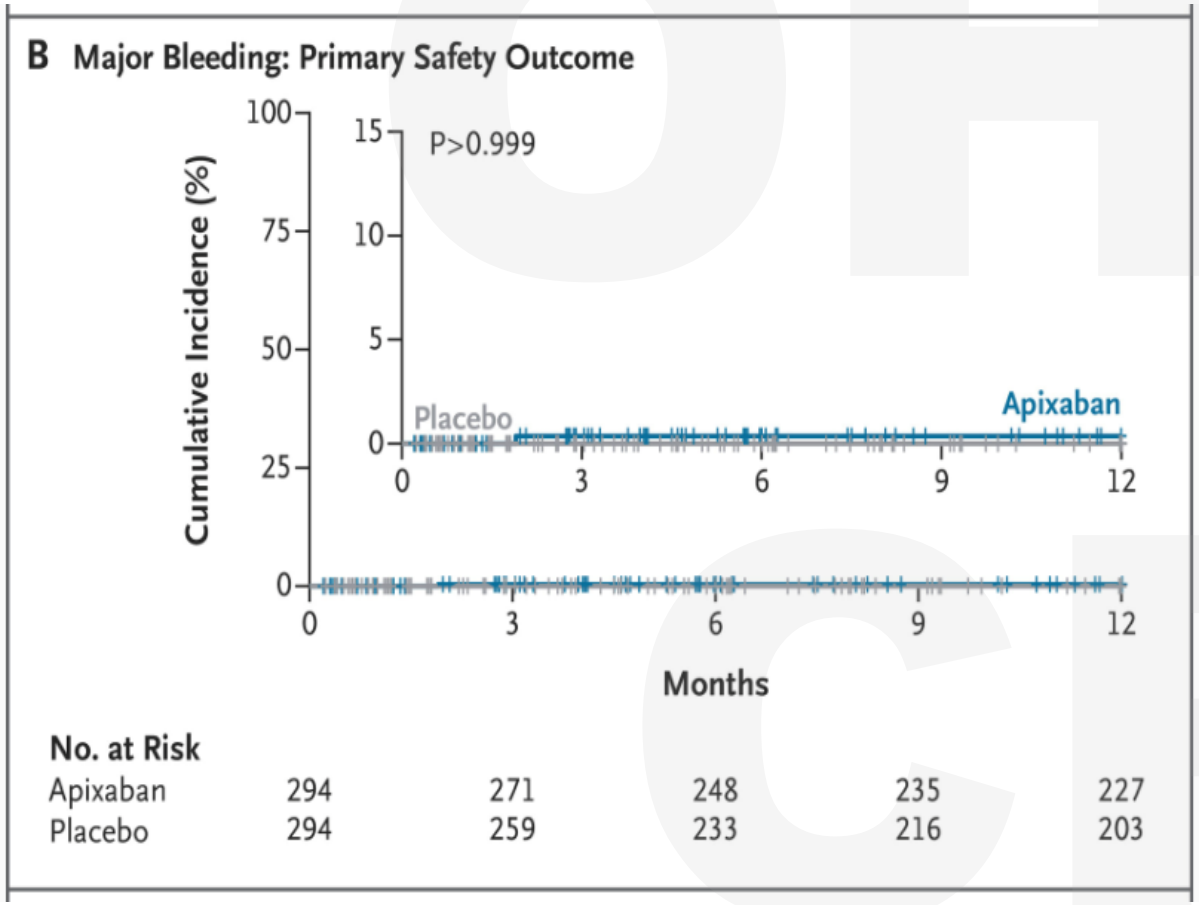
Enduring risk factors for VTE — no. (%)†				
Persistent immobility	39 (6.5)	15 (5.0)	24 (8.0)	12.2
Obesity: BMI ≥30	289 (48.2)	141 (47.0)	148 (49.3)	4.7
Heart failure	15 (2.5)	10 (3.3)	5 (1.7)	10.7
Chronic lung disease	134 (22.3)	78 (26.0)	56 (18.7)	17.7
Chronic inflammatory or autoimmune disorder	313 (52.2)	152 (50.7)	161 (53.7)	6.0
Atherosclerotic cardiovascular disease	176 (29.3)	86 (28.7)	90 (30.0)	2.9
Chronic kidney disease	64 (10.7)	32 (10.7)	32 (10.7)	0
Chronic liver disease	23 (3.8)	10 (3.3)	13 (4.3)	5.2

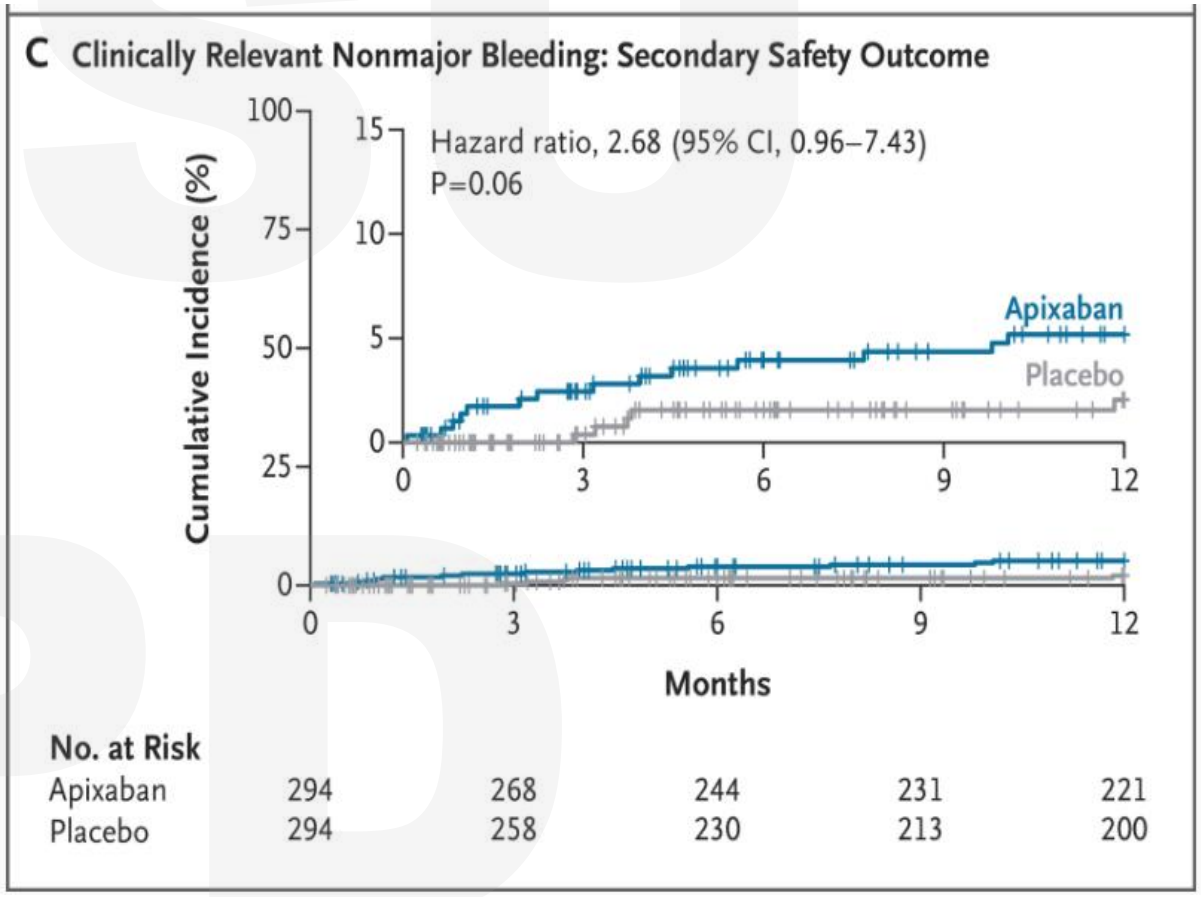
Provoking factors for VTE — no. (%)†				
Acute medical illness‡	110 (18.3)	56 (18.7)	54 (18.0)	1.7
Surgery	201 (33.5)	102 (34.0)	99 (33.0)	2.1
Trauma	115 (19.2)	62 (20.7)	53 (17.7)	7.6
Pregnancy	11 (1.8)	7 (2.3)	4 (1.3)	7.5
Infection	99 (16.5)	45 (15.0)	54 (18.0)	8.1
Hormonal contraceptive or replacement therapy	69 (11.5)	42 (14.0)	27 (9.0)	15.7
Hospitalization ≤3 mo before the VTE event	56 (9.3)	25 (8.3)	31 (10.3)	6.9
Immobility	188 (31.3)	81 (27.0)	107 (35.7)	18.8
Blood transfusion§	2 (0.3)	1 (0.3)	1 (0.3)	0
Coronavirus disease 2019§	49 (8.2)	23 (7.7)	26 (8.7)	3.7
Long-haul travel§	100 (16.7)	46 (15.3)	54 (18.0)	7.2
Other factor¶	53 (8.8)	26 (8.7)	27 (9.0)	1.2

Outcomes

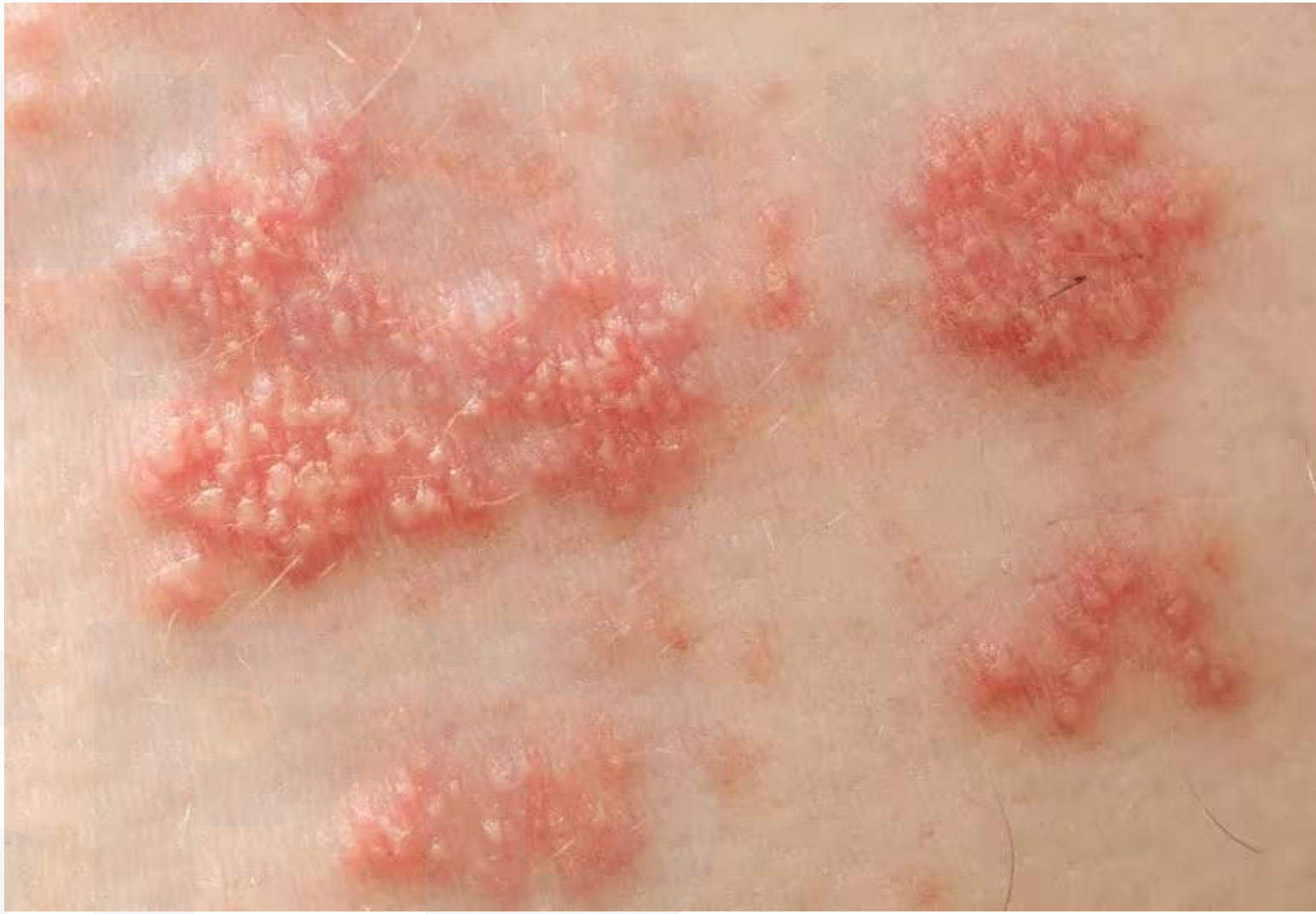

A Symptomatic Recurrent VTE: Primary Efficacy Outcome
Cumulative Incidence (%)
100
75
50
25
0
15
10
5
0
Hazard ratio, 0.13 (95% CI, 0.04–0.36)
P<0.001
Placebo
Apixaban
0
3
6
9
12
Months
No. at Risk
Apixaban 300 298 298 295 295
Placebo 300 289 279 274 269

Adverse Bleeding





Shingles and Dementia Associations



https://health.clevelandclinic.org/when-your-shingles-pain-lingers-5-options-may-help

Article

A natural experiment on the effect of herpes zoster vaccination on dementia

https://doi.org/10.1038/s41586-025-08800-x

Received: 4 November 2023

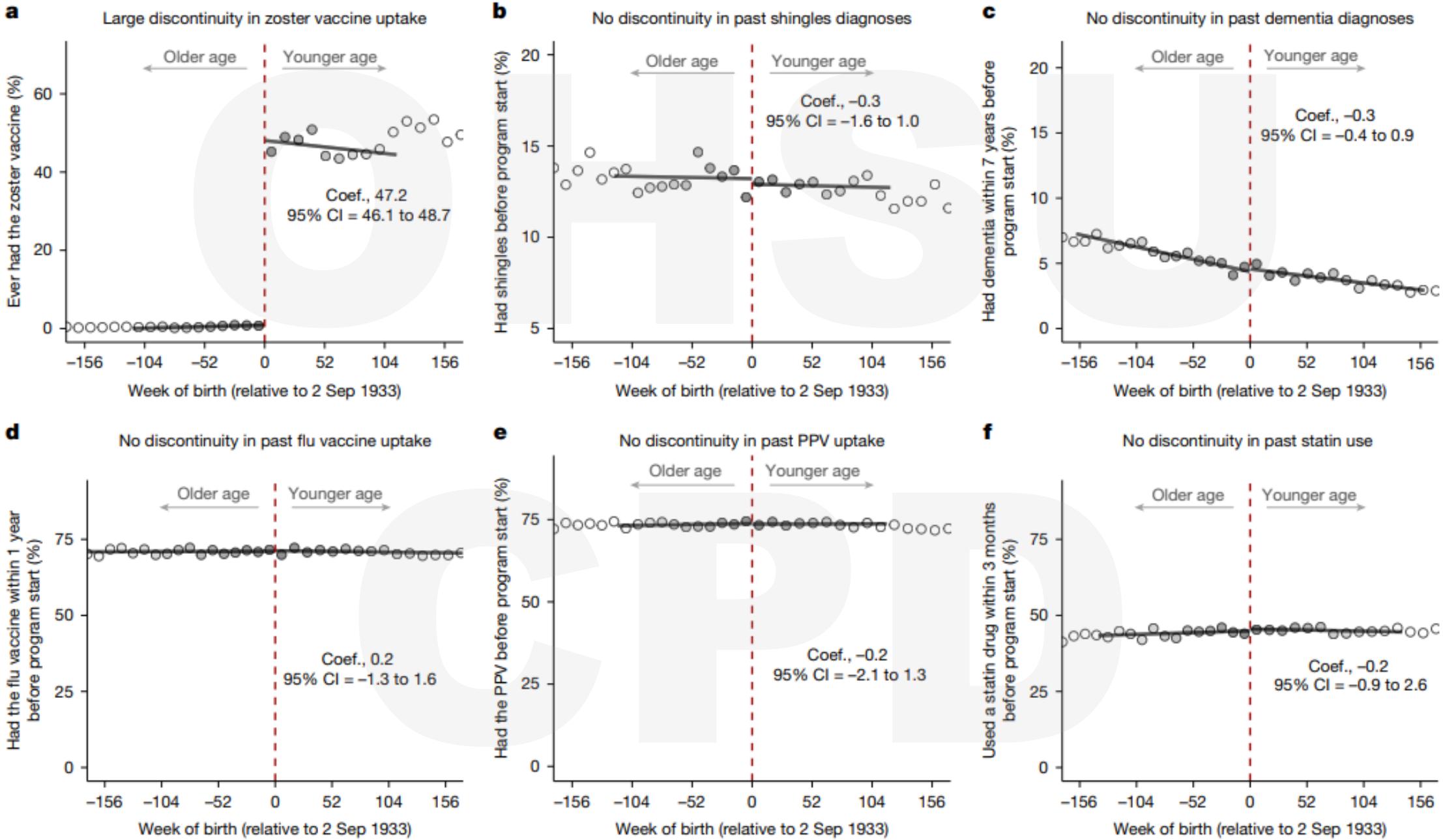
Accepted: 18 February 2025

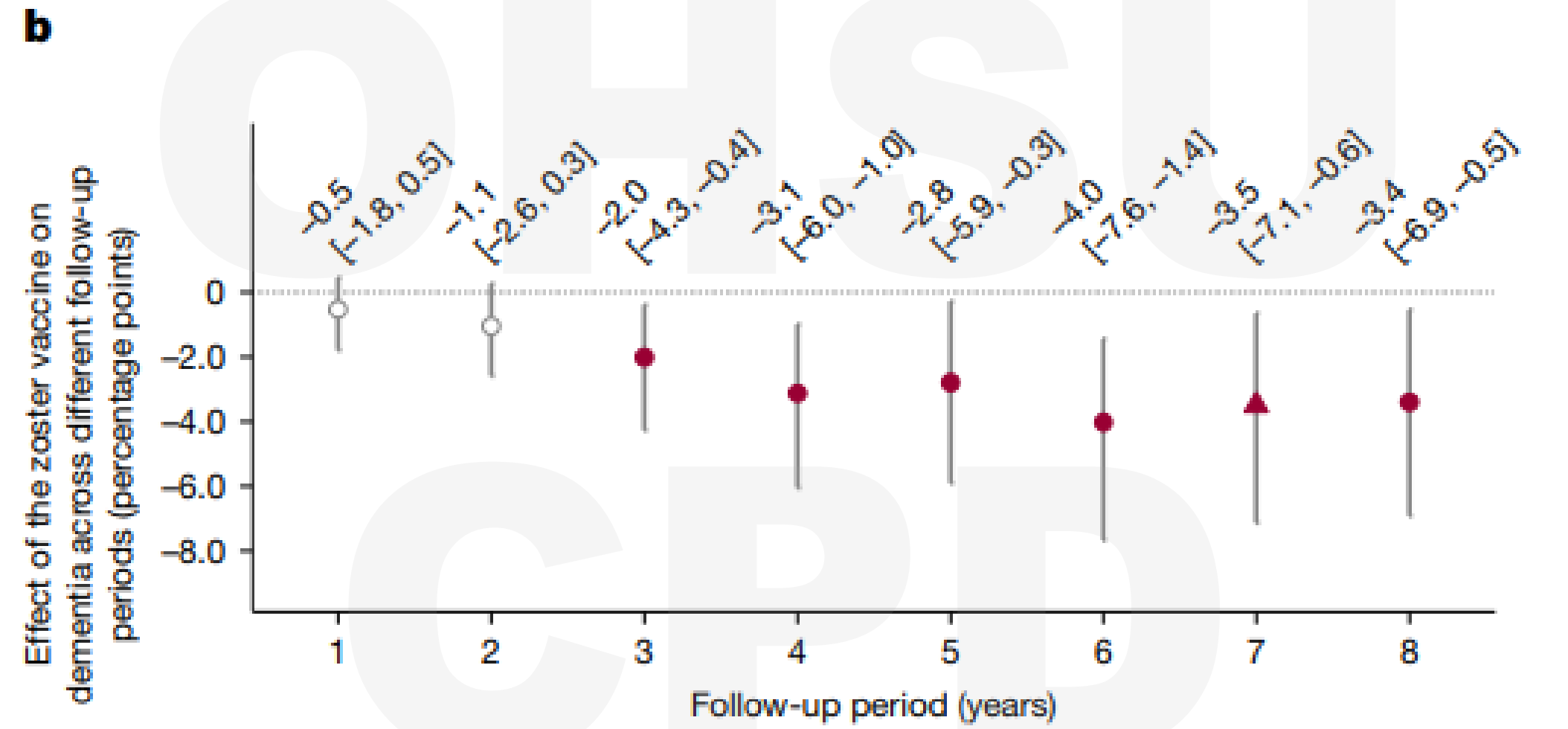
Markus Eyting[1,2,3,9], Min Xie[1,4,9], Felix Michalik[1,4], Simon Heß[5], Seunghun Chung[1] & Pascal Geldsetzer[1,6,7,8]

Context

- **Welsh herpes zoster vaccination program**
  - September 1, 2013

- Eligibility was **determined strictly by date of birth:**
  - **On or after September 2, 1933** were eligible for at least 1 year of free vaccination
  - **Before September 2, 1933** were permanently ineligible


a
Large discontinuity in zoster vaccine uptake
Older age
Younger age
Ever had the zoster vaccine (%)
Coef., 47.2
95% CI = 46.1 to 48.7
Week of birth (relative to 2 Sep 1933)
b
No discontinuity in past shingles diagnoses
Had shingles before program start (%)
Coef., −0.3
95% CI = −1.6 to 1.0
c
No discontinuity in past dementia diagnoses
Had dementia within 7 years before program start (%)
Coef., −0.3
95% CI = −0.4 to 0.9
d
No discontinuity in past flu vaccine uptake
Had the flu vaccine within 1 year before program start (%)
Coef., 0.2
95% CI = −1.3 to 1.6
e
No discontinuity in past PPV uptake
Had the PPV before program start (%)
Coef., −0.2
95% CI = −2.1 to 1.3
f
No discontinuity in past statin use
Used a statin drug within 3 months before program start (%)
Coef., −0.2
95% CI = −0.9 to 2.6


b
Effect of the zoster vaccine on dementia across different follow-up periods (percentage points)
0
−2.0
−4.0
−6.0
−8.0
−0.5 [−1.8, 0.5]
−1.1 [−2.6, 0.3]
−2.0 [−4.3, −0.4]
−3.1 [−6.0, −1.0]
−2.8 [−5.9, −0.3]
−4.0 [−7.6, −1.4]
−3.5 [−7.1, −0.6]
−3.4 [−6.9, −0.5]
1
2
3
4
5
6
7
8
Follow-up period (years)

nature medicine

Article

https://doi.org/10.1038/s41591-025-03972-5

Varicella-zoster virus reactivation and the risk of dementia

Vitaly Polisky[1], Maria Littmann[1], Aleksei Triastcyn[2], Max Horn[2], Andreas Georgiou[2], Robyn Widenmaier[3], Bruno Anspach[4], Halima Tahrat[4], Sanjay Kumar[5], Carolyn Buser-Doepner[5], Pascal Geldsetzer[6,7], Cornelia M. Van Duijn[8] & Patrick Schwab[2]

Study Design

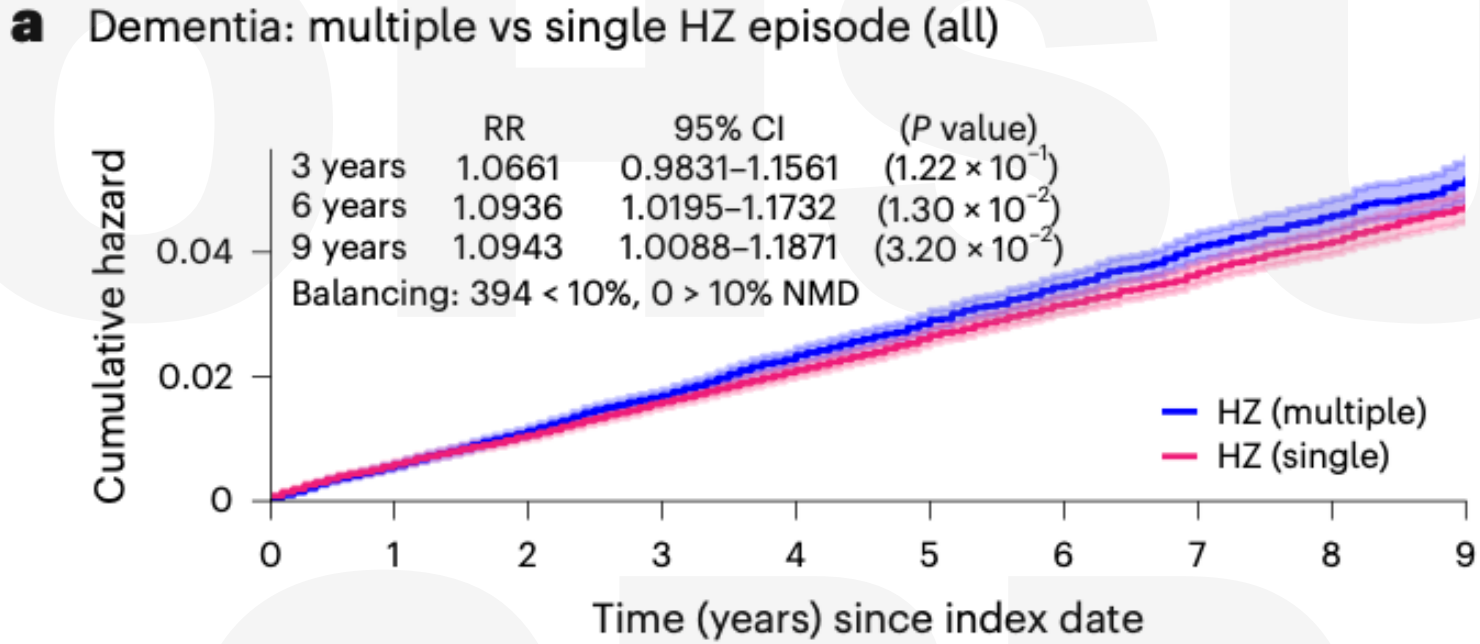
- Retrospective, observational, matched-cohort study with pairwise comparisons
- Optum EHR database
- Between October 2007 and September 2023
- Inclusion: Documented HZV or vaccination compared to cohorts without documented event

Five Assumptions

1. Increase in VZV reactivation = Increased dementia risk
2. Lower burden reactivation = decreased dementia risk
3. Strength of modulation of VZV reaction proportional to dementia risk reduction
4. Impact of VZV reactivation on dementia risk dementia risk proportional to dementia risk reduction
5. Those at higher risk reactivation benefit more from vaccination in terms of dementia risk

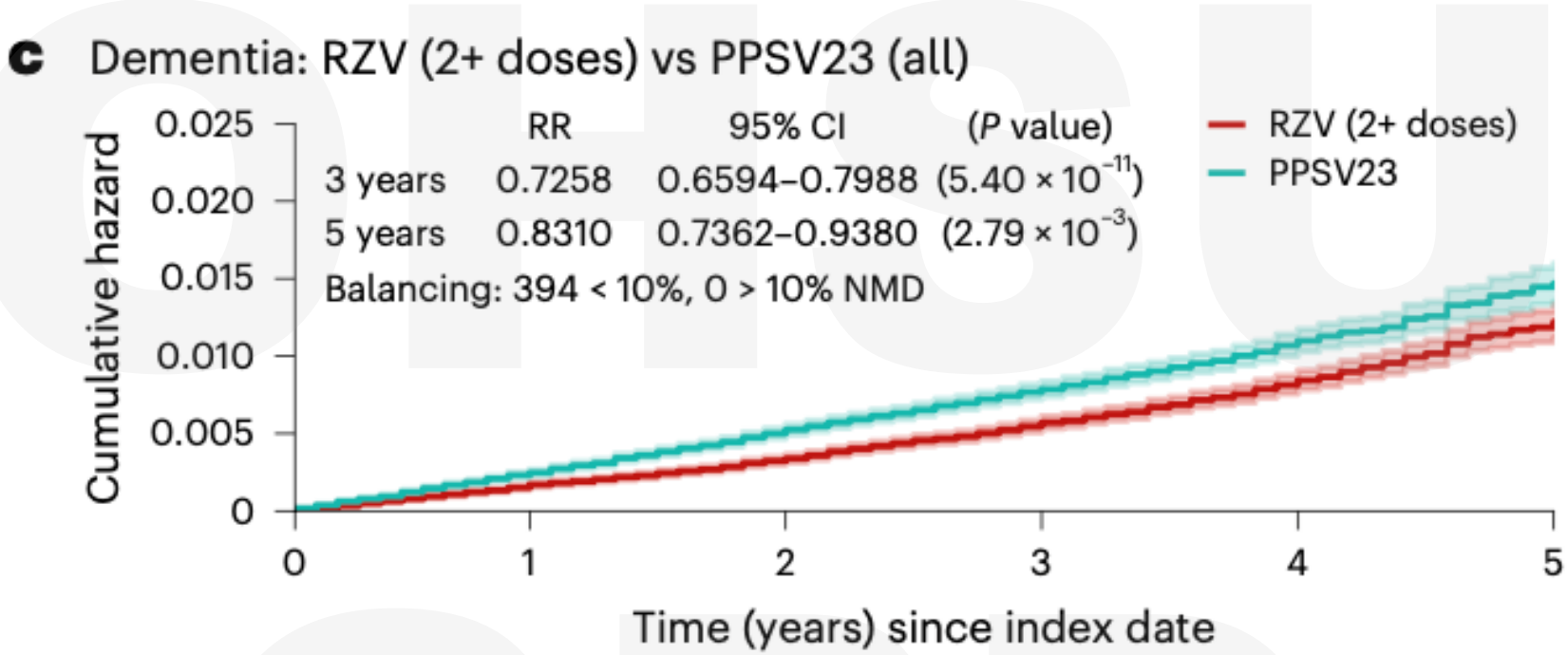
Assumption 1

Increase in HZ burden = Increased dementia risk



HZ (multiple)				
At risk	97,342	48,080	18,510	3,840
Censored[a]	0	48,068	77,062	91,553
Events[b]	0	1,194	1,770	1,949
HZ (single)				
At risk	97,342	57,546	29,727	10,589
Censored[a]	0	38,579	65,717	84,548
Events[b]	0	1,217	1,898	2,205

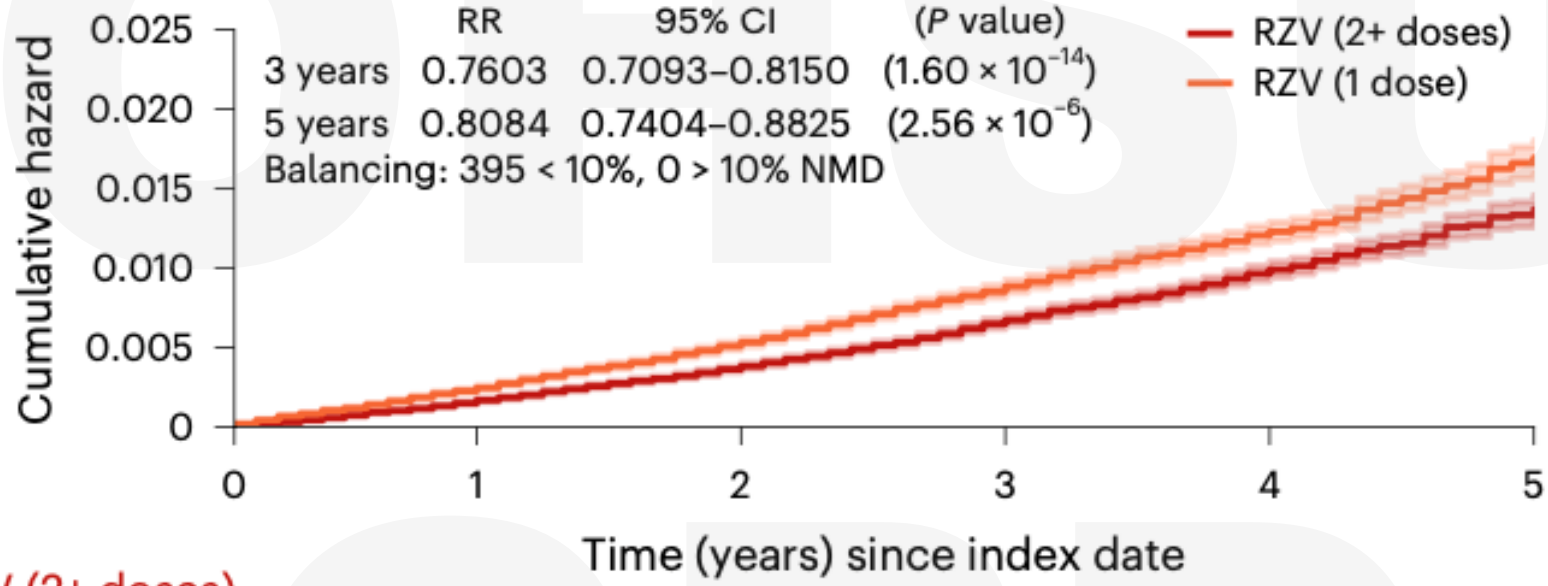
Assumption 2



RZV (2+ doses)						
At risk	234,309	184,310	120,043	68,247	32,534	9,031
Censored[a]	0	49,640	113,655	165,229	200,806	224,230
Events[b]	0	359	611	833	969	1,048
PPSV23						
At risk	234,309	172,126	108,595	59,736	26,552	7,121
Censored[a]	0	61,679	124,822	173,457	206,507	225,877
Events[b]	0	504	892	1,116	1,250	1,311

Assumption 3

b Dementia: RZV (2+ doses) vs RZV (1 dose) (all)



RZV (2+ doses)

At risk	416,765	328,757	212,314	117,202	55,024	16,756
Censored[a]	0	87,370	203,234	297,861	359,761	397,897
Events[b]	0	638	1,217	1,702	1,980	2,112

RZV (1 dose)

At risk	416,765	284,177	173,951	95,329	43,219	12,318
Censored[a]	0	131,719	241,292	319,427	371,289	402,067
Events[b]	0	869	1,522	2,009	2,257	2,380

Summary

- Dose-response, temporal relationship
- Possible mechanism
  - VZV reactivation -> linked to vasculopathy, amyloid deposition resembling Alzheimer pathology.

- Limitations include
  - Observational
    - Claims data
  - Healthy vaccine bias
    - Comparators PPSV23

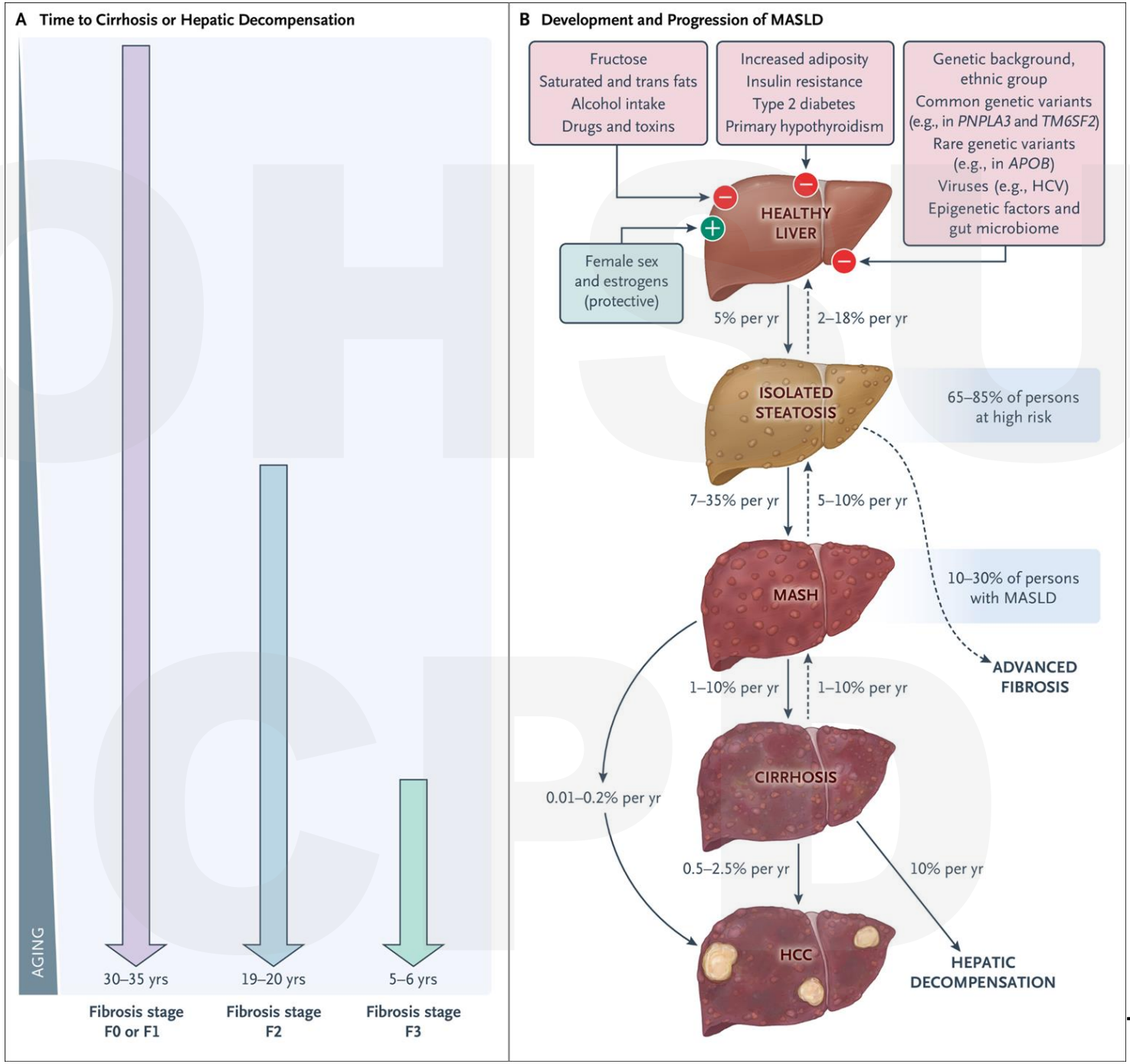
The NEW ENGLAND JOURNAL of MEDICINE

ESTABLISHED IN 1812 JUNE 5, 2025 VOL. 392 NO. 21

Phase 3 Trial of Semaglutide in Metabolic Dysfunction–Associated Steatohepatitis

Arun J. Sanyal, M.D.,[1] Philip N. Newsome, M.B., Ch.B., Ph.D.,[2,3] Iris Kliers, M.D.,[4] Laura Harms Østergaard, M.Sc.,[4] Michelle T. Long, M.D.,[4] Mette Skalshøi Kjær, M.D., Ph.D.,[4] Anna M.G. Cali, M.D.,[4] Elisabetta Bugianesi, M.D., Ph.D.,[5] Mary E. Rinella, M.D.,[6] Michael Roden, M.D.,[7-9] and Vlad Ratziu, M.D., Ph.D.,[10] for the ESSENCE Study Group*

ESSENCE Trial



Targher et al 2025

Study design

Documented steatohepatitis and liver fibrosis stage 2 or 3

NAS core >= 4 (pathology eval)

Randomly assigned 2:1 to subcutaneous semaglutide versus placebo

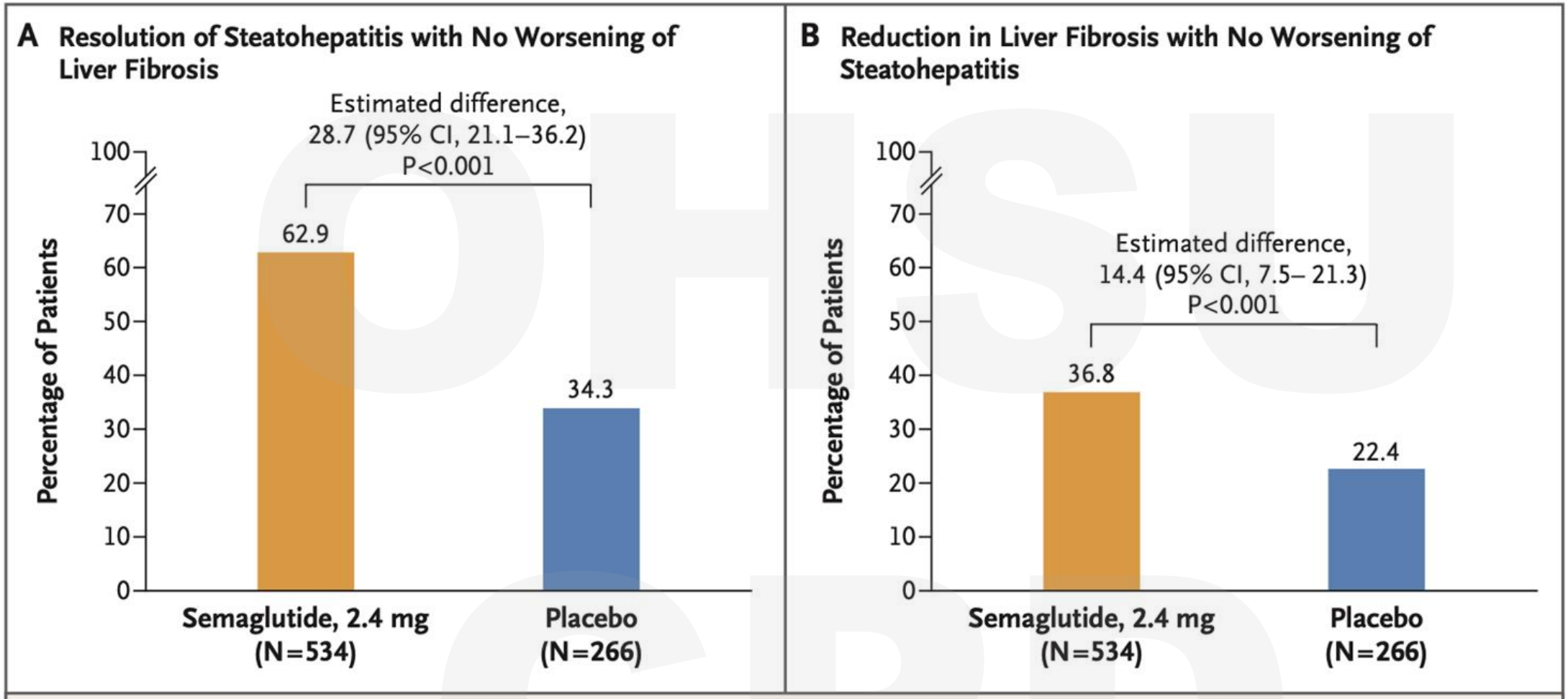
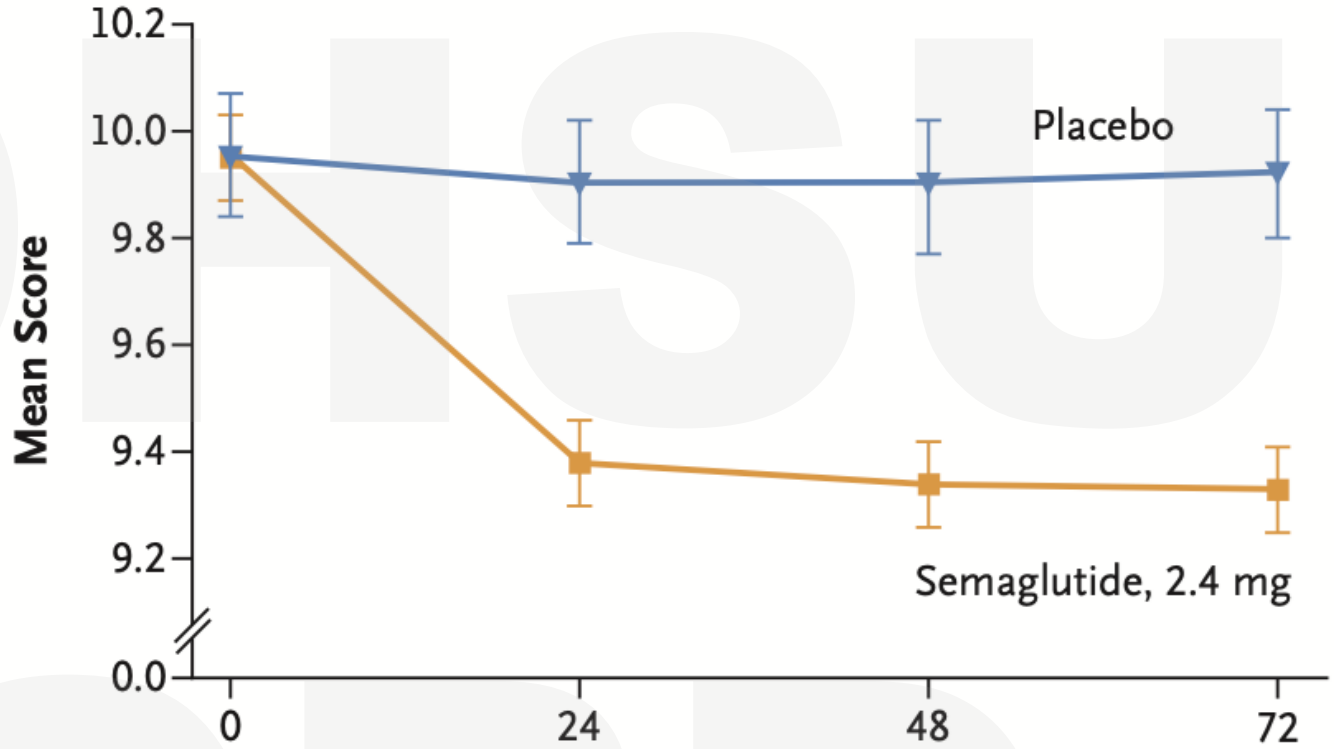


Figure 1. Primary End Points.

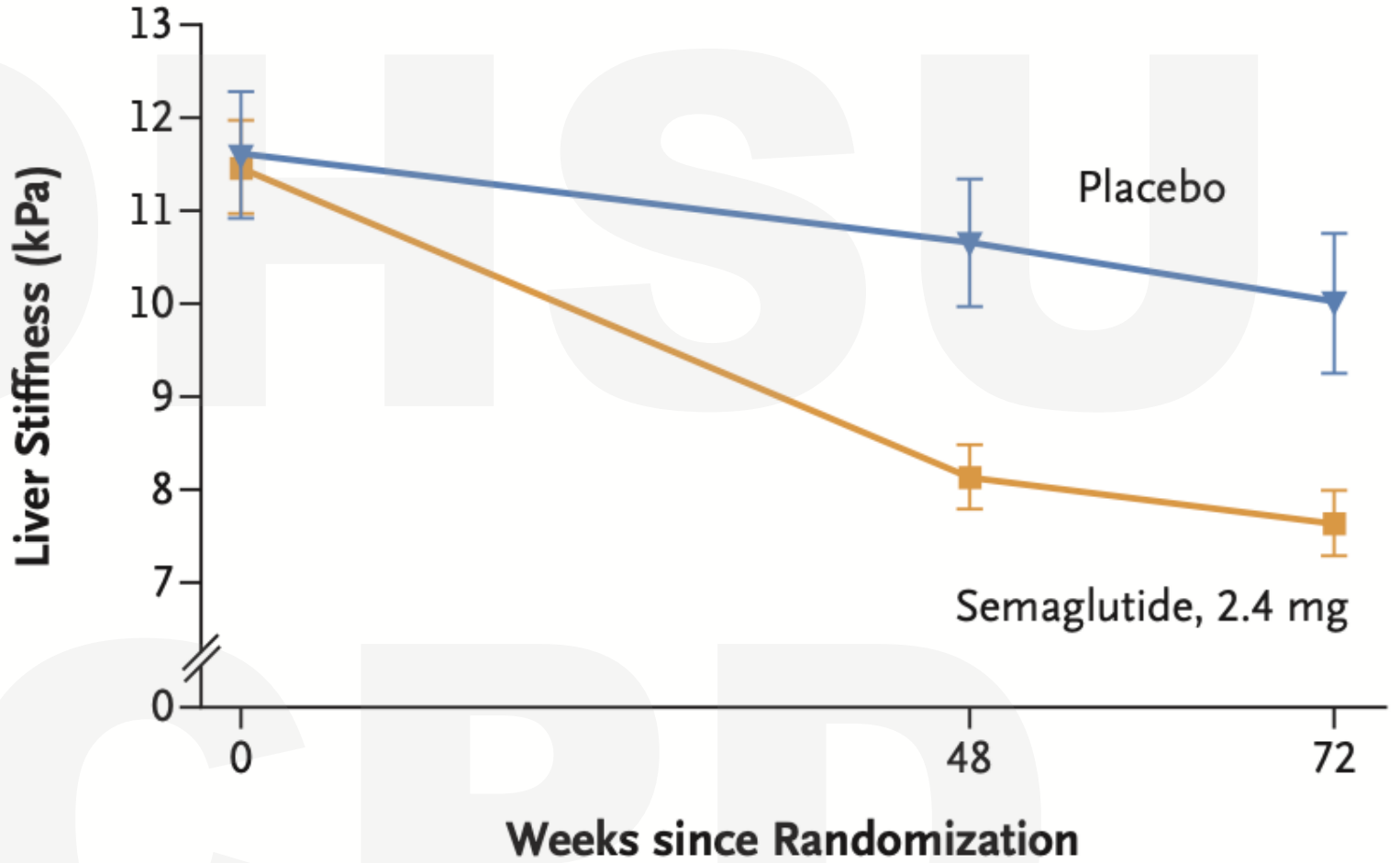
The figure shows the percentage of patients with fibrosis stage 2 or 3 who had resolution of steatohepatitis with no worsening of liver fibrosis (Panel A) and reduction in liver fibrosis with no worsening of steatohepatitis (Panel B) after 72 weeks, with the estimated difference expressed in percentage points.

A **Enhanced Liver Fibrosis Score**



No. of Patients				
Placebo	266	252	246	237
Semaglutide, 2.4 mg	534	511	504	492

B **Liver Stiffness Measured by Vibration-Controlled Transient Elastography**



No. of Patients

Placebo	216	204	193
Semaglutide, 2.4 mg	417	399	381

Table 3. Safety Analysis Population.*

Event	Semaglutide, 2.4 mg	Placebo
	number/total number (percent)	
Any adverse event	690/800 (86.2)	315/395 (79.7)
Fatal adverse event	3/800 (0.4)	6/395 (1.5)
Serious adverse event	107/800 (13.4)	53/395 (13.4)
Infection or infestation	22/800 (2.8)	10/395 (2.5)
Gastrointestinal disorder	20/800 (2.5)	6/395 (1.5)
Musculoskeletal or connective-tissue disorder	14/800 (1.8)	6/395 (1.5)
Injury, poisoning, or procedural complication	11/800 (1.4)	5/395 (1.3)
Nervous system disorder	11/800 (1.4)	5/395 (1.3)
Neoplasm†	11/800 (1.4)	8/395 (2.0)
Adverse event leading to trial discontinuation	21/800 (2.6)	13/395 (3.3)
Adverse event affecting ≥10% of patients in either group		
Nausea	290/800 (36.2)	52/395 (13.2)
Diarrhea	215/800 (26.9)	48/395 (12.2)
Constipation	178/800 (22.2)	33/395 (8.4)
Vomiting	149/800 (18.6)	22/395 (5.6)
Coronavirus disease 2019	134/800 (16.8)	74/395 (18.7)
Decreased appetite	112/800 (14.0)	11/395 (2.8)
Adverse event within safety focus area‡		
Gallbladder-related disorder	20/800 (2.5)	6/395 (1.5)
Acute pancreatitis	3/800 (0.4)	2/395 (0.5)
Malignant neoplasm	13/800 (1.6)	9/395 (2.3)
Hypoglycemia		
Patients with type 2 diabetes§	33/446 (7.4)	12/222 (5.4)
Patients without type 2 diabetes	1/354 (0.3)	1/173 (0.6)

Practice Changing Points

- AHA Dyslipidemia Guidelines
  - Lp(a). ApoB, LDL goals, CAC, incidental calcium
- Prostate Cancer Screening
  - Reduces prostate-specific mortality. Contextual screening intervals
- AHA Hypertension guidelines
  - <130/80, Single-pill combinations, hypertensive urgency no more

Practice Changing Points

- Coffee averaging ~1 cup a day not associated with worsening Afib recurrence at 180 days
- Consider extended duration apixaban for patients with persistent provoking risk factors and provoked VTE
- More arguments for encouraging Shingrix uptake
- Consider GLP-1s for management MASH/MASLD

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Notes

Lipid guidelines

- on-HDL cholesterol includes all cholesterol present in lipoprotein particles that is considered atherogenic, including LDL, lipoprotein(a), intermediate-density lipoprotein (IDL), and very low-density lipoprotein (VLDL)
- Fasting recommended if TG > 400 and trying to assess LDL-C
- Consider fasting if TG > 200 to better quantify values

- **Aspirin** – Aspirin appears to have a net clinical benefit (which outweighs the ris old who also have CAC ≥100, regardless of their ASCVD risk category. Shared de provider(s) are warranted prior to initiating therapy [46]. (See "Aspirin in the p
- The MESA study suggested that individuals with CAC ≥100 had a net benefit wit individuals with a score of 0 had potential for more harm than benefit (table 1) old with elevated CAC has not been well studied.

- Per UpToDate
  - The MESA study (Multi-Ethnic Study ofAtherosclerosis)
  - individuals with CAC ≥100, <70 YO, had a netbenefit with aspirin
  - Conversely, CAC≥100 and CAC≥400 identified subgroups in whicl overall (for CAC≥100, NNT_5=140 versus NNH_5=518
    - ASCVD event vs. Major bleed

1	B-NR	3. In adults and children who have undergone a standard lipid profile, use of either the Martin/Hopkins equation or the Sampson/National Institutes of Health (NIH) equation is preferred over calculation by the Friedewald equation to estimate LDL-C.[6,7,11–13]
1	B-NR	4. In adults and children who have undergone a standard lipid profile, use of either the Martin/Hopkins equation or Sampson/NIH equation is preferred over direct LDL-C measurement (other than by beta-quantification) to estimate LDL-C.[7,11,14,15]
1	B-NR	5. In adults and children who have undergone a standard lipid profile, reporting of non–HDL-C is recommended for ASCVD risk assessment and to guide initiation and monitoring of LLT.[16–19]

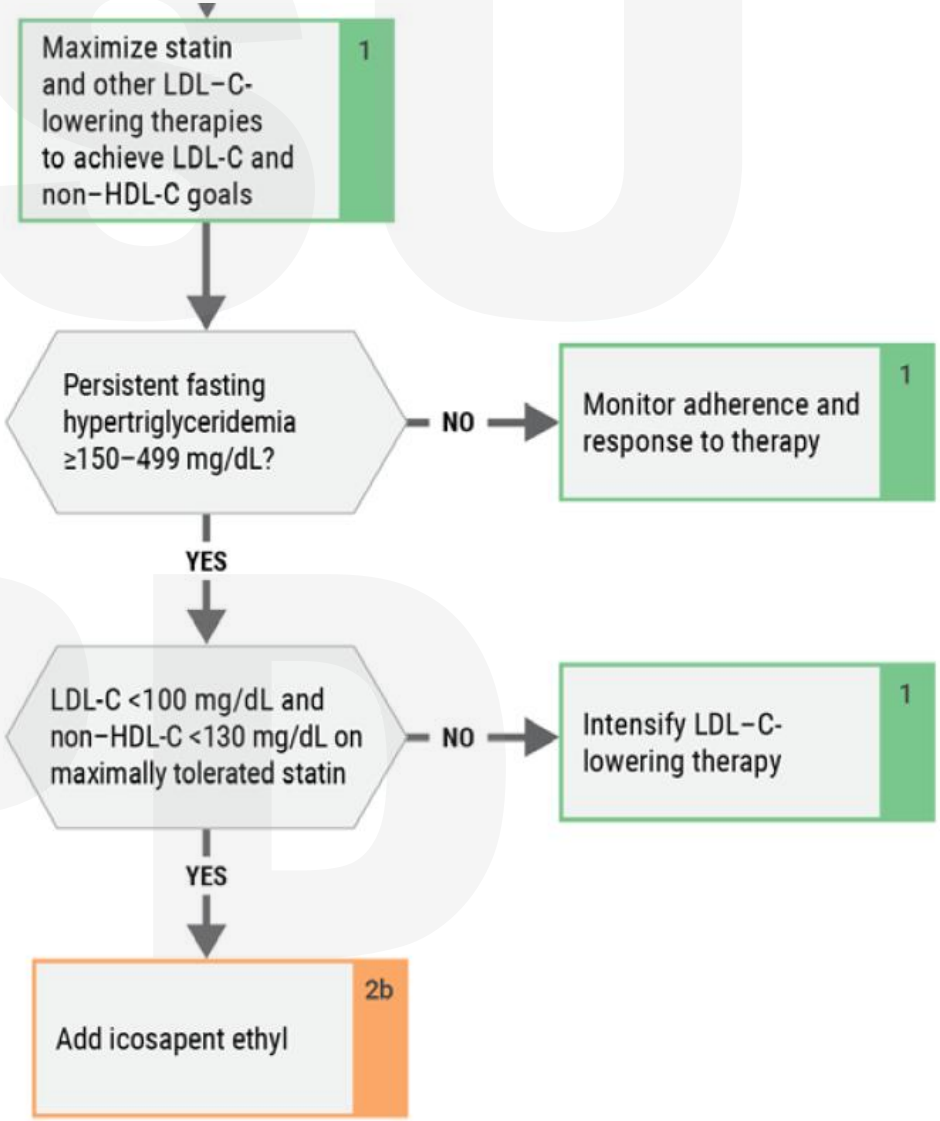
Lipid Guidelines

4.2.4.2. Genetic Testing for FH

Recommendations for Genetic Testing for FH Referenced studies that support recommendations are summarized in the Evidence Table.		
COR	**LOE**	**Recommendations**
1	**B-NR**	1. In adults with possible, probable, or definite FH, panel-based genetic testing for pathogenic/likely pathogenic rare variants for FH is beneficial to identify individuals at highest risk of cardiovascular events and to facilitate cascade screening.[1–4]
2a	**B-NR**	2. In adults with severe hypercholesterolemia with an LDL-C ≥190 mg/dL (4.9 mmol/L) without an identified secondary cause, panel-based genetic testing for pathogenic/likely pathogenic rare variants for FH can be useful to identify those with FH who are at higher risk of ASCVD events.[1,2]
2b	**B-NR**	3. In adults with an elevated LDL-C of 160 to 189 mg/dL (4.1–4.9 mmol/L) without an identified secondary cause, panel-based genetic testing for pathogenic/likely pathogenic rare variants for FH may be considered to identify those with FH who are at higher risk of events.[1,2]

Triglycerides in Secondary Prevention

- IF Fasting TG >150-500 despite maximally tolerated statin
  - Consider icosapent ethyl (Vascepa)



ERSPC - Limitations / Acknowledgements

- **Heterogeneity** between countries
  - Screening **invitations** (2-8 times)
  - Screening **intervals** (2-7 years)
  - **PSA thresholds for biopsy** (3 ng/mL vs. 4 ng/mL w/ supplementary tests)

- **?Reflecting modern practice**
  - Trial initiated in 1993
  - **Core analysis of group 55-69 YO ->** updated guidelines suggesting shared-decision making 40-50 YO depending on risk

- **Pragmatic / Population design**

- **Quantification of harms not described**

DECAF

Variable	Coffee Consumption (N=100)	Coffee Abstinence (N=100)
Baseline coffee consumption		
None – no. (%)	3 (3)	6 (6)
1-3 cups per month – no. (%)	4 (4)	1 (1)
2-4 cups per week – no. (%)	15 (15)	11 (11)
5-7 cups per week – no. (%)	2 (2)	5 (5)
1 cup per day – no. (%)	38 (38)	41 (41)
2-3 cups per day – no. (%)	29 (29)	25 (25)
4-5 cups per day – no. (%)	2 (2)	5 (5)
≥ 6 cups per day – no. (%)	3 (3)	3 (3)
Don't know – no (%)	4 (4)	3 (3)
Type of coffee consumption		
Drip coffee – no. (%)	49 (49)	45 (45)
Espresso drinks – no. (%)	37 (37)	36 (36)
Iced coffee – no. (%)	4 (4)	4 (4)
Decaffeinated coffee – no. (%)	7 (7)	11 (11)
Other – no. (%)	14 (14)	7 (7)
Don't know – no (%)	0 (0)	0 (0)
Patient-reported frequency of coffee triggering AF		
Never – no. (%)	60 (60)	65 (65)
Rarely – no. (%)	2 (2)	1 (1)
Sometimes – no. (%)	4 (4)	4 (4)
Often – no. (%)	1 (1)	0 (0)
Always – no. (%)	1 (1)	0 (0)
Don't know – no. (%)	32 (32)	30 (30)
Patient-reported symptoms from coffee abstinence		

DECAF

- Moreover, a continued separation of survival curves over time implies that this difference may be more attributable to a benefit of coffee consumption rather than harm from abrupt coffee cessation and withdrawal.