

Biomarker Based Community Screening for Cardiovascular Risk

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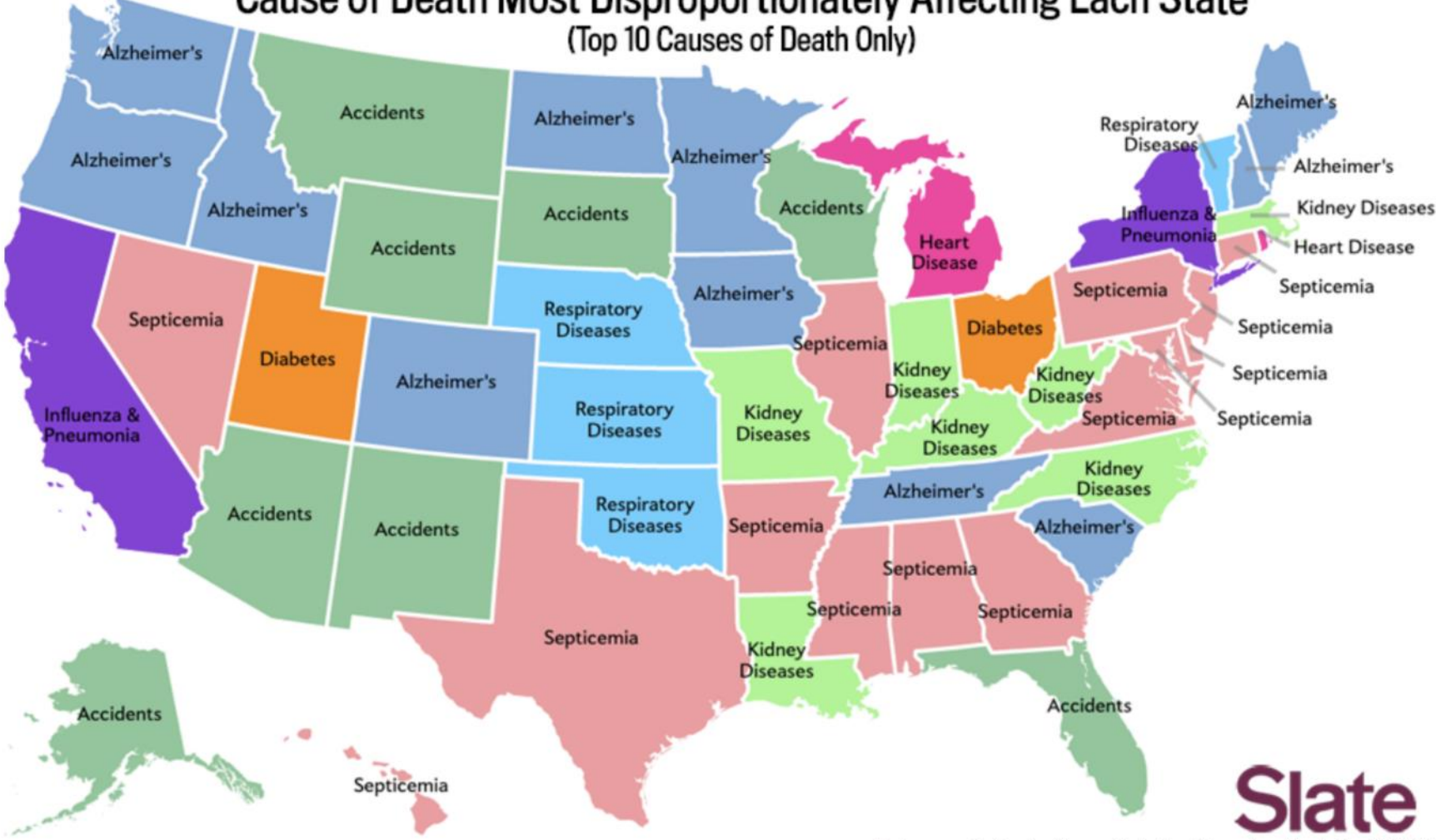
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Relevant Disclosures

- NIH/NHLBI: R01 HL153607; R01 HL163377; R01 HL146059; R01 HL127215; T32 HL120822
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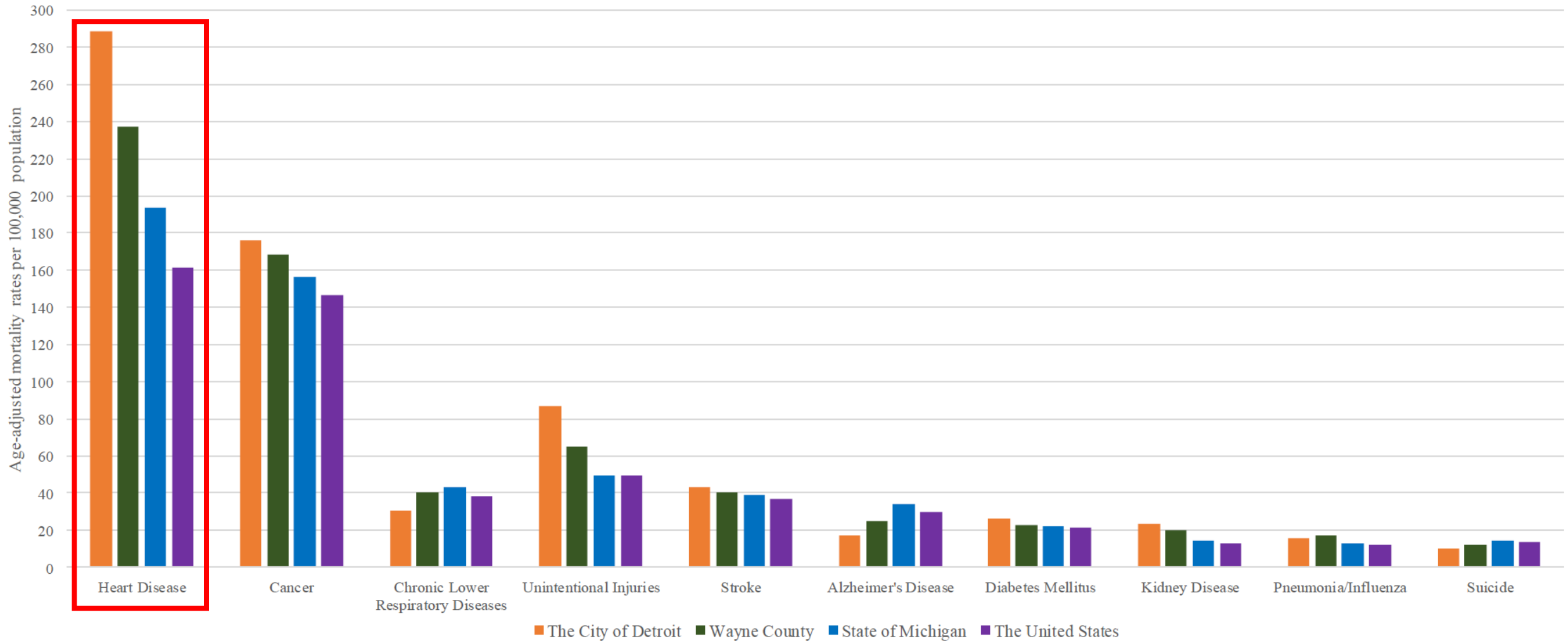
Cause of Death Most Disproportionately Affecting Each State (Top 10 Causes of Death Only)



Slate

Data source: Centers for Disease Control and Prevention. Map by Ben Blatt/Slate.

Age-adjusted Mortality Rates per 100,000 Population for the Ten Leading Causes of Death in the City of Detroit, Wayne County, State of Michigan, and the United States, 2019



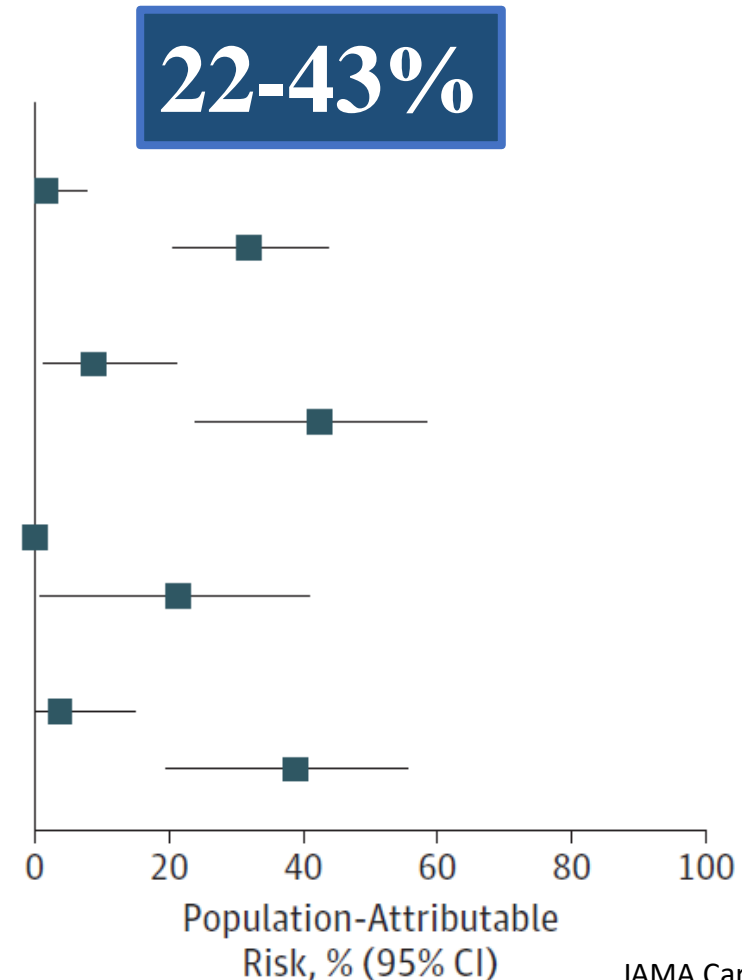
Source: 2019 Geocoded Michigan Death Certificate Registry. Division for Vital Records & Health Statistics, Michigan Department of Health & Human Services. National Center for Health Statistics.

Population-Attributable Risk for Cardiovascular Disease Associated With Hypertension in Black Adults

Jackson Heart Study (JHS)

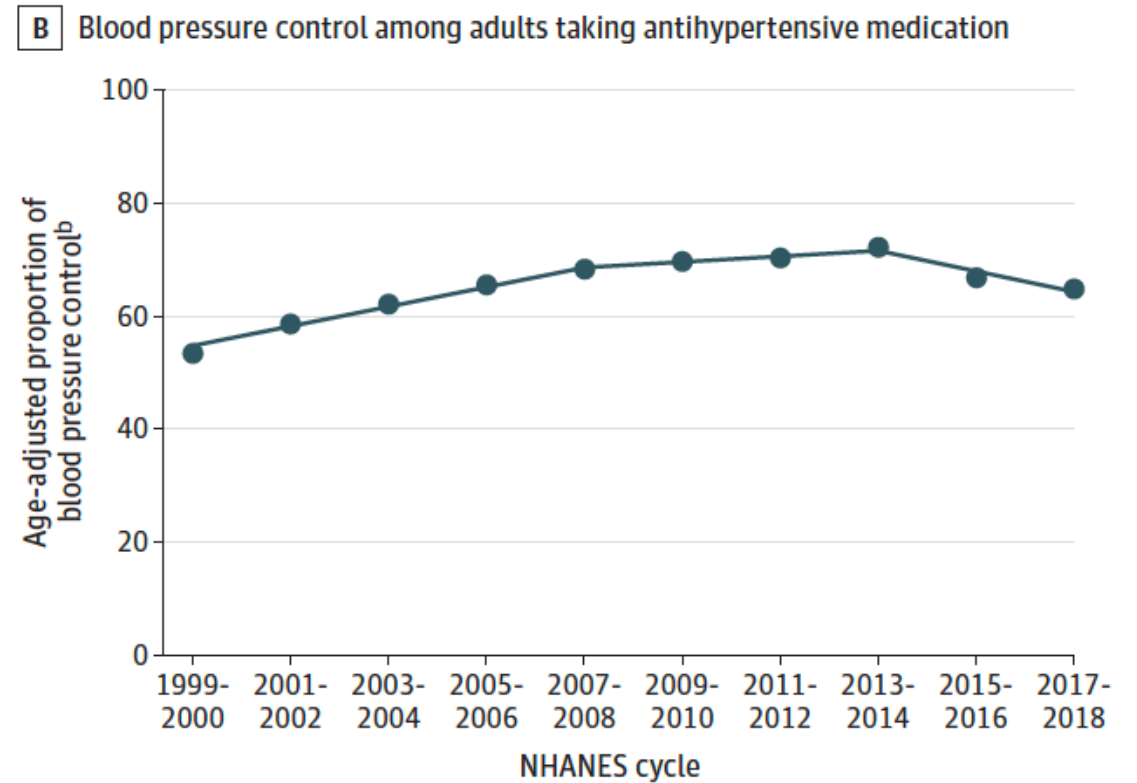
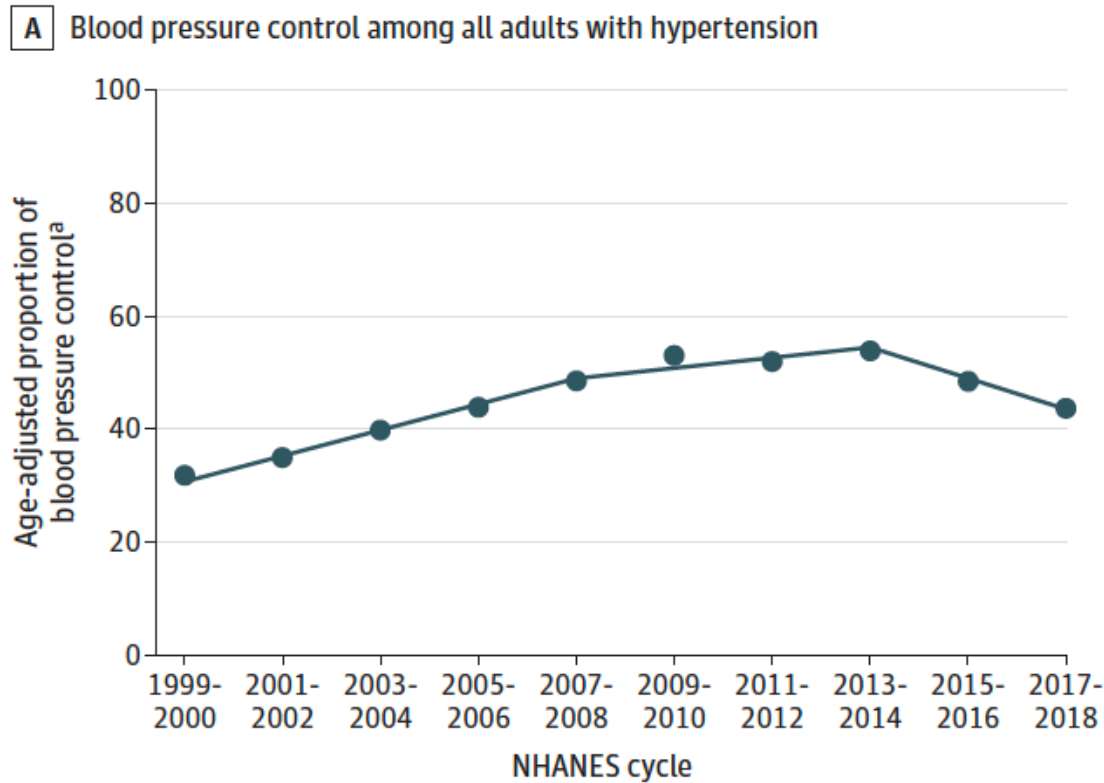
Reasons for Geographic and Racial Differences in Stroke (REGARDS) study

Disease	Population-Attributable Risk, % (95% CI)
Composite cardiovascular disease	
Elevated blood pressure	1.9 (0-7.5)
Hypertension	32.5 (20.5-43.6)
Coronary heart disease	
Elevated blood pressure	9.4 (1.2-20.9)
Hypertension	42.7 (24.0-58.4)
Heart failure	
Elevated blood pressure	0
Hypertension	21.6 (0.6-40.8)
Stroke	
Elevated blood pressure	4.0 (0-14.7)
Hypertension	38.9 (19.4-55.6)



Trends in Blood Pressure Control Among US Adults With Hypertension, 1999-2000 to 2017-2018

Paul Muntner, PhD; Shakia T. Hardy, PhD; Lawrence J. Fine, MD; Byron C. Jaeger, PhD; Gregory Wozniak, PhD; Emily B. Levitan, ScD; Lisandro D. Colantonio, MD, PhD



Three Public Health Interventions Could Save 94 Million Lives in 25 Years

Global Impact Assessment Analysis

Effect of Hypertension Treatment on Systolic Blood Pressure	Percent of Patients With Hypertension Treated, %*	Sodium Intake Reduction, %†	Number (Millions) of Deaths That Could Be Delayed (95% Uncertainty Interval)		
			Women	Men	Total
10 mm Hg	50	10	11.3 (10.1–12.5)	17.0 (15.1–18.8)	28.2 (25.2–31.3)
10 mm Hg	50	30	23.5 (20.7–26.2)	32.1 (27.8–36.0)	55.6 (48.5–62.2)
10 mm Hg	70	10	17.8 (16.0–19.5)	23.5 (21.3–25.7)	41.2 (37.3–45.2)
10 mm Hg	70	30	29.6 (26.4–32.8)	38.2 (33.5–42.4)	67.8 (59.9–75.2)
15 mm Hg	50	10	13.5 (12.1–14.9)	21.0 (19.0–23.1)	34.5 (31.1–38.0)
15 mm Hg	50	30	25.6 (22.6–28.4)	35.9 (31.4–40.1)	61.5 (54.0–68.5)
15 mm Hg	70	10	23.0 (20.8–25.2)	30.5 (27.8–33.3)	53.5 (48.6–58.5)
15 mm Hg	70	30	34.6 (31.1–38.2)	44.8 (39.9–49.5)	79.5 (71.0–87.7)

*Increasing hypertension coverage alone to 50% could delay 13.4 million (12.2–14.6) deaths if assuming a 10-mmHg decline and 19.8 million (18.1–21.7) deaths if assuming a 15-mmHg decline. With 70% coverage, the deaths delayed could be 26.7 million (24.3–29.2) with a 10-mmHg decline and 39.4 million (35.9–43.0) with a 15-mmHg decline.

†Reducing salt intake by 10% could delay 15.3 million (12.9–17.7) deaths, and reducing salt intake by 30% could delay 43.4 million (36.9–49.5) deaths globally.

The Surgeon General's Call to Action to Control Hypertension



Foreword from the Surgeon General, U.S. Department of Health and Human Services



As a physician, I've seen firsthand the devastating effects of hypertension. Left uncontrolled, it leads to heart attacks, stroke, kidney disease, and cognitive decline in later life, and it can impact mother and baby during and after pregnancy. In addition, as evidenced from the global COVID-19 outbreak earlier in the year, we've seen the broad impact of preventable health conditions on worse outcomes.

Hypertension is unfortunately common, but there are interventions and programs that have been successful in improving control. Our country has many hypertension control champions—doctors, practices, communities, and health systems that have excelled at achieving high rates of hypertension control among their patients. We need to learn from their many years of “blood, sweat, and tears” and apply their principles in new settings.

While hypertension is more prominent among older adults, it is not simply a condition of the elderly. All ages are impacted, and early identification and long-term control can preserve cardiovascular health now and into the future. We know that lifestyle changes, such as being physically active and adopting a healthy diet, can promote hypertension control, yet many communities have significant barriers that prevent people from making these changes. We also know that many people with hypertension require medications to achieve control. Access to high-quality health care, prescription of appropriate medications, and clinical and community support are needed to prevent and treat hypertension, publicize local resources, and establish a plan for care supportive of long-term control.

The Surgeon General's Call to Action to Control Hypertension summarizes recent data on hypertension control, identifies select goals and strategies, and provides recommendations for areas of focus when resources are limited. While the recent trends don't look good—we've hit a plateau in hypertension control—I believe that with focus and collaboration, we can improve our trajectory.

Join me in taking control of hypertension across our nation. Together, we've got this!

Jerome M. Adams, MD, MPH
Vice Admiral, U.S. Public Health Service
Surgeon General
U.S. Department of Health and Human Services

What Are Biomarkers and Why Are They Important? Transcript

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Biomarkers are characteristics of the body that you can measure. So your blood pressure is actually a biomarker.

Biomarkers are very important to medicine in general. We're all used to going to the doctor and getting all our test results, right, and even imaging — x-ray results or CAT scans — those are biomarkers that tell how the body's doing, and they're measurable.

Biomarkers are integral to drug development; they're really critical, because we need to measure the effects of investigational drugs on people during the clinical trials. And the way we do that is to look at their effect on biomarkers. And so it's really important that we have a wide range of biomarkers that can measure everything we want to know about the effect of the investigational drug in people.

Drug development today has many problems, and the major problem is the failure rate. So even drugs that have gone through the whole preclinical process and gone through all sorts of animal testing and all sorts of other types of assays, once they get into people, they have maybe a less than 1 in 10 chance of actually getting on the market. Nine out of 10 may fail during that development. And we have to do better than that if we're going to accelerate the treatment availability, if we're going to lower the cost of drug development and not have it continue to escalate, and if we're actually going to let a lot of innovators into this space of participating in drug development.

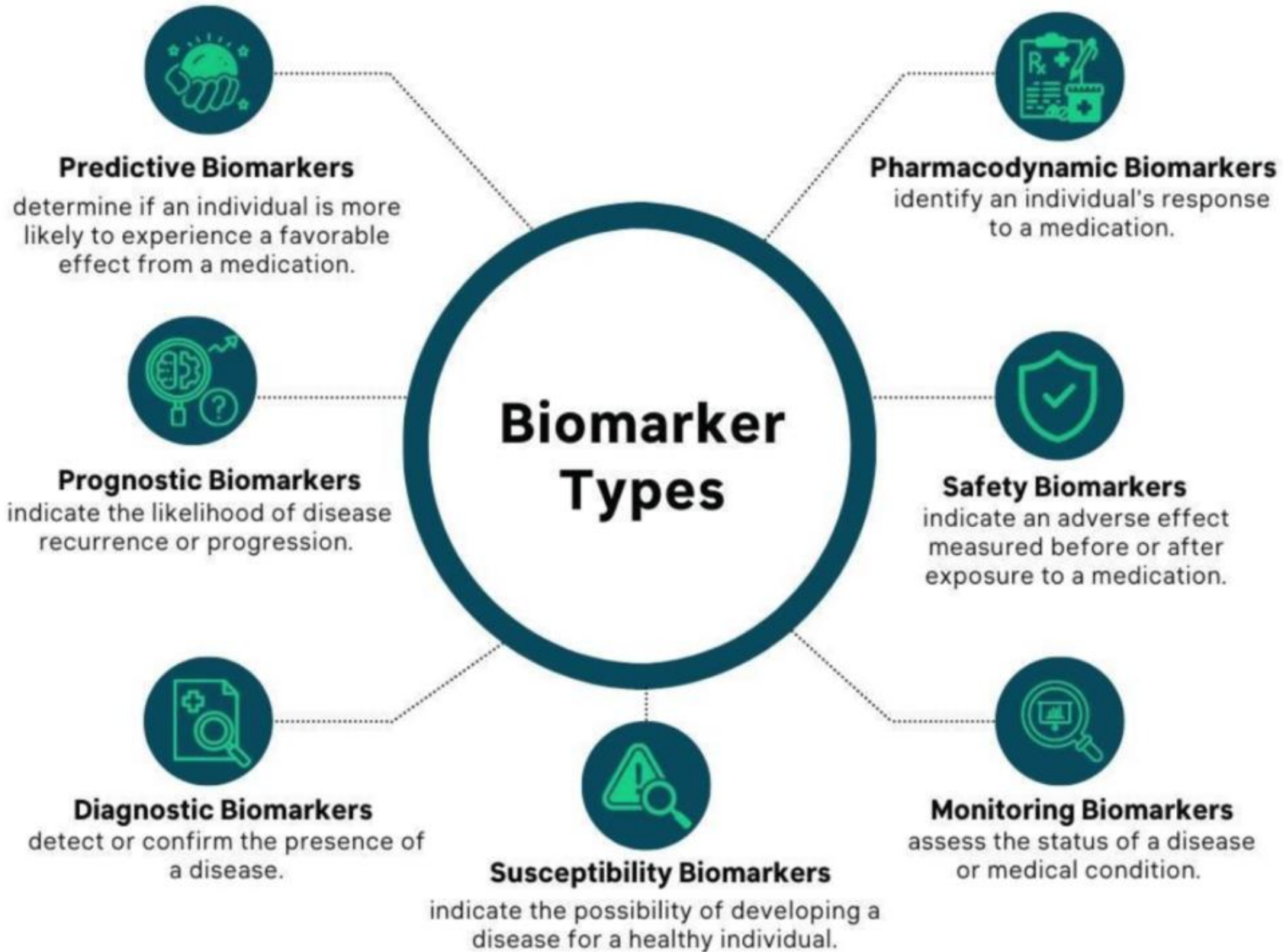
To really improve the success rate and improve the efficiency of drug development, we need a whole new generation of biomarkers that are more informative and that can tell developers earlier whether or not their drug may have toxicity or it really may not work at all, and to get that early read on what's going to be successful. And so those biomarkers are ones that have yet to be developed.

Content current as of:

03/03/2017



Regulated Product(s)

Drugs



ORIGINAL RESEARCH

Stage 1 Hypertension and the 10-Year and Lifetime Risk of Cardiovascular Disease: A Prospective Real-World Study

Xinyi Peng , MD;* Cheng Jin , MD;* Qirui Song, MD; Shouling Wu , MD, PhD; Jun Cai , MD, PhD

BACKGROUND: The 10-year and lifetime cardiovascular disease risk in the population with stage 1 hypertension and the effects of recovery from and progression of stage 1 hypertension remain undetermined.

METHODS AND RESULTS: This prospective cohort study included 96268 individuals with blood pressure measurements obtained in 2006 and again in 2010. The 10-year cardiovascular disease risk was estimated using the multivariable Cox proportional hazards model, and the lifetime risk was calculated using a modified survival analysis that accounted for the competing risk of death. Stage 1 hypertension was detected in 30.83% of the cohort. The 10-year cardiovascular disease risk was 2.80%, and the lifetime risk was 16.61%. Compared with the normal blood pressure group, the stage 1 hypertension group had a 35% higher 10-year risk (hazard ratio [HR], 1.35 [95% CI, 1.19–1.52]) and a 36% higher lifetime risk (HR, 1.36 [95% CI, 1.25–1.49]). By 2010, 12.57% of the participants with stage 1 hypertension had progressed to stage 2, with a significant 156% increase in 10-year risk (HR, 2.56 [95% CI, 2.11–3.11]) and an increased lifetime risk of 129% (HR, 2.29 [95% CI, 1.89–2.77]). There was no appreciable change in risk in those with stage 1 hypertension whose blood pressure returned to the normal-elevated range.

CONCLUSIONS: Stage 1 hypertension was associated with a significant increase in 10-year and lifetime cardiovascular disease risk. Progression to stage 2 hypertension was associated with a marked increase in lifetime risk. The current guidelines require revision to promote early detection and appropriate management of blood pressure.

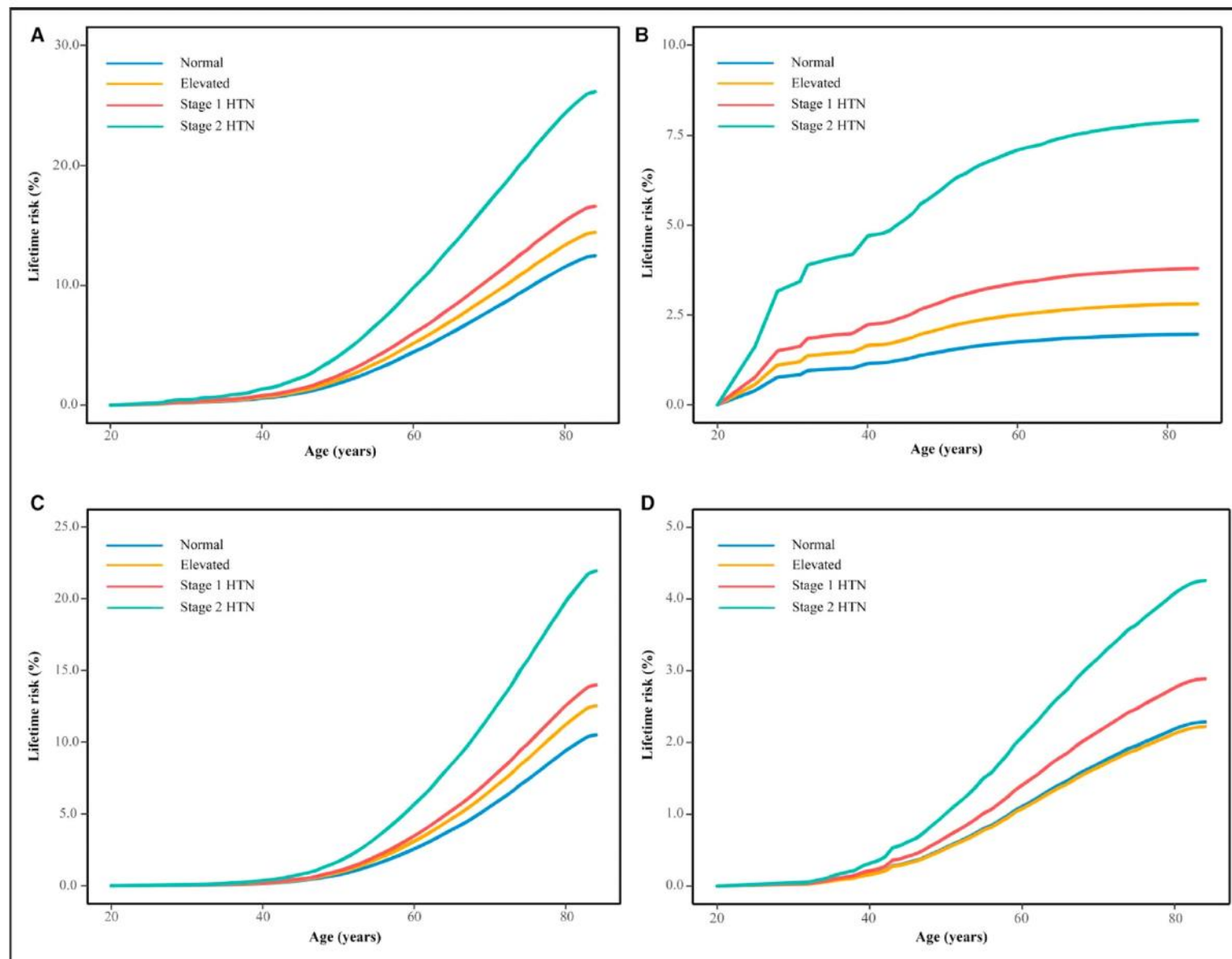


Figure 2. Lifetime risk of cardiovascular diseases, according to BP levels in 2006.

A, Cardiovascular disease; **B**, cerebral hemorrhage; **C**, cerebral infarction; and **D**) myocardial infarction. Participants were stratified by BP levels on the basis of the following criteria: (1) a normal BP group (SBP <120mmHg and DBP <80mmHg); (2) an elevated BP group (SBP 120–129mmHg and DBP <80mmHg); (3) a stage 1 hypertension group (SBP 130–139mmHg or DBP 80–89mmHg); and (4) a stage 2 hypertension group (SBP ≥140mmHg or DBP ≥90mmHg or currently taking antihypertensive agents). BP indicates blood pressure; DBP, diastolic blood pressure; HTN, hypertension; and SBP, systolic blood pressure.

2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk

A Report of the American College of Cardiology/American Heart Association Task Force on Practice Guidelines

*Endorsed by the American Association of Cardiovascular and Pulmonary Rehabilitation,
American Society for Preventive Cardiology, American Society of Hypertension,
Association of Black Cardiologists, National Lipid Association, Preventive Cardiovascular
Nurses Association, and WomenHeart: The National Coalition for Women With Heart Disease*

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SUBCOMMITTEE ON PREVENTION GUIDELINES

Sidney C. Smith, Jr, MD, FACC, FAHA, Chair; Gordon F. Tomaselli, MD, FACC, FAHA, Co-Chair

Recommendations	NHLBI Grade	NHLBI Evidence Statements	ACC/AHA COR	ACC/AHA LOE
Assessment of 10-Year Risk of a First Hard ASCVD Event				
1. The race- and sex-specific Pooled Cohort Equations* to predict 10-year risk of a first hard ASCVD event should be used in non-Hispanic African Americans and non-Hispanic whites, 40–79 years of age.	B (Moderate)	N/A	I	B ⁴⁻⁸
2. Use of the sex-specific Pooled Cohort Equations for non-Hispanic whites may be considered for estimation of risk in patients from populations other than African Americans and non-Hispanic whites.	E (Expert Opinion)	N/A	IIb	C
CQ1: Use of Newer Risk Markers After Quantitative Risk Assessment				
1. If, after quantitative risk assessment, a risk-based treatment decision is uncertain, assessment of ≥1 of the following—family history, hs-CRP, CAC score, or ABI—may be considered to inform treatment decision making.	E (Expert Opinion)	Appendix 4	IIb†	B ⁹⁻¹⁷
2. Routine measurement of CIMT is not recommended in clinical practice for risk assessment for a first ASCVD event.	N (No recommendation for or against)	Appendix 4	III: No Benefit†	B ^{12,16,18}
3. The contribution of ApoB, CKD, albuminuria, and cardiorespiratory fitness to risk assessment for a first ASCVD event is uncertain at present.	N (No recommendation for or against)	Appendix 4	—	—
CQ2: Long-Term Risk Assessment				
1. It is reasonable to assess traditional ASCVD risk factors‡ every 4–6 years in adults 20–79 years of age who are free from ASCVD and to estimate 10-year ASCVD risk every 4–6 years in adults 40–79 years of age who are free from ASCVD.	B (Moderate)	Appendix 5 CQ2/ES7	IIa	B ^{19,20}
2. Assessment of 30-year or lifetime ASCVD risk on the basis of traditional risk factors‡ may be considered in adults 20–59 years of age who are free from ASCVD and are not at high short-term risk.	C (Weak)	Appendix 5 CQ2/ES2, CQ2/ES3, CQ2/ES4, CQ2/ES5, CQ2/ES6	IIb	C ²⁰⁻²²

App should be used for primary prevention patients (those without ASCVD) only.

Current Age ⓘ *

Age must be between 20-79

Sex *

Male

Female

Race *

White

African American

Other

Systolic Blood Pressure (mm Hg) *

Value must be between 90-200

Diastolic Blood Pressure (mm Hg) *

Value must be between 60-130

Total Cholesterol (mg/dL) *

Value must be between 130 - 320

HDL Cholesterol (mg/dL) *

Value must be between 20 - 100

LDL Cholesterol (mg/dL) ⓘ ○

Value must be between 30-300

History of Diabetes? *

Yes

No

Smoker? ⓘ *

Current ⓘ

Former ⓘ

Never ⓘ

On Hypertension Treatment? *

Yes

No

On a Statin? ⓘ ○

Yes

No

On Aspirin Therapy? ⓘ ○

Yes

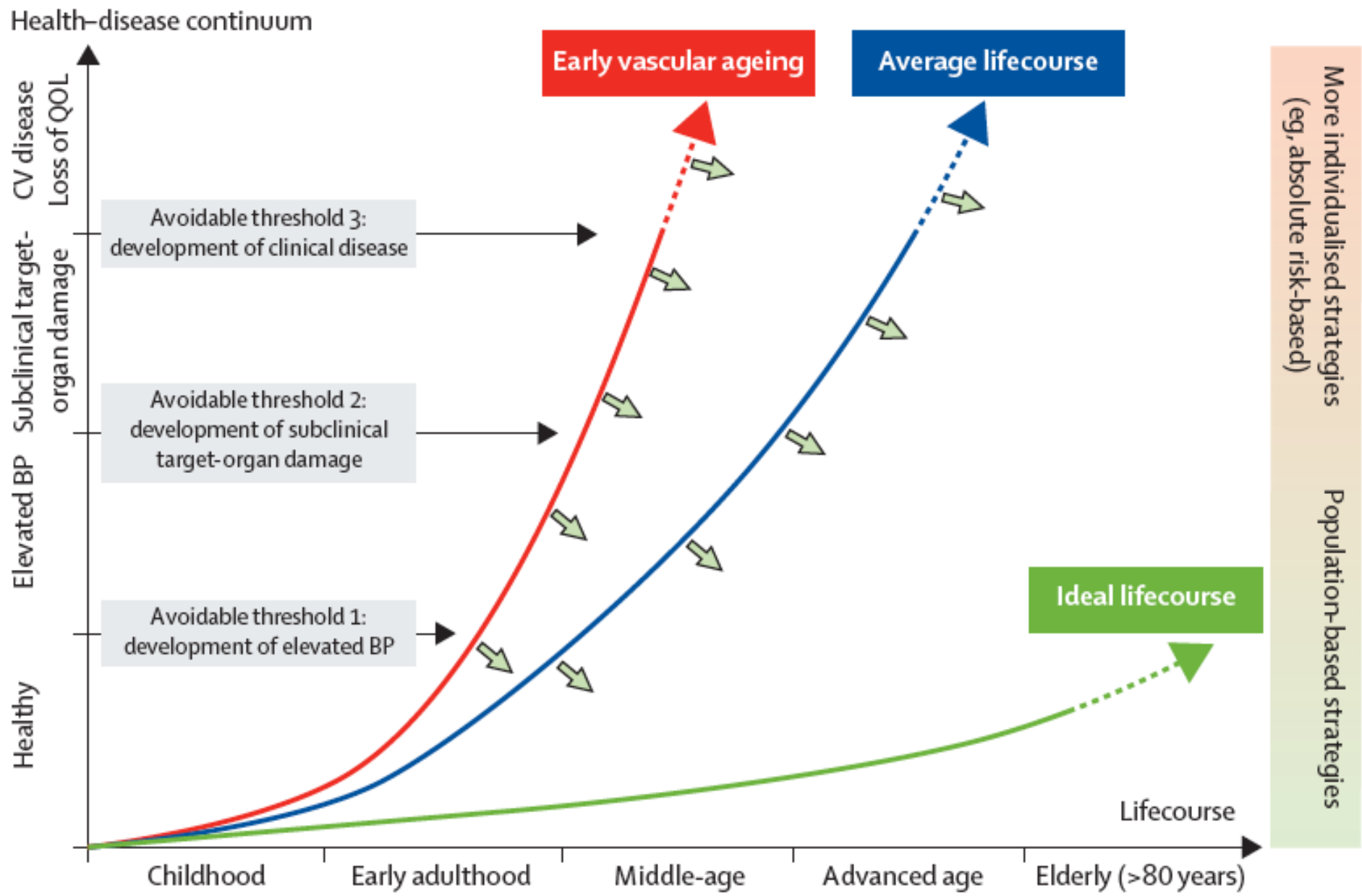
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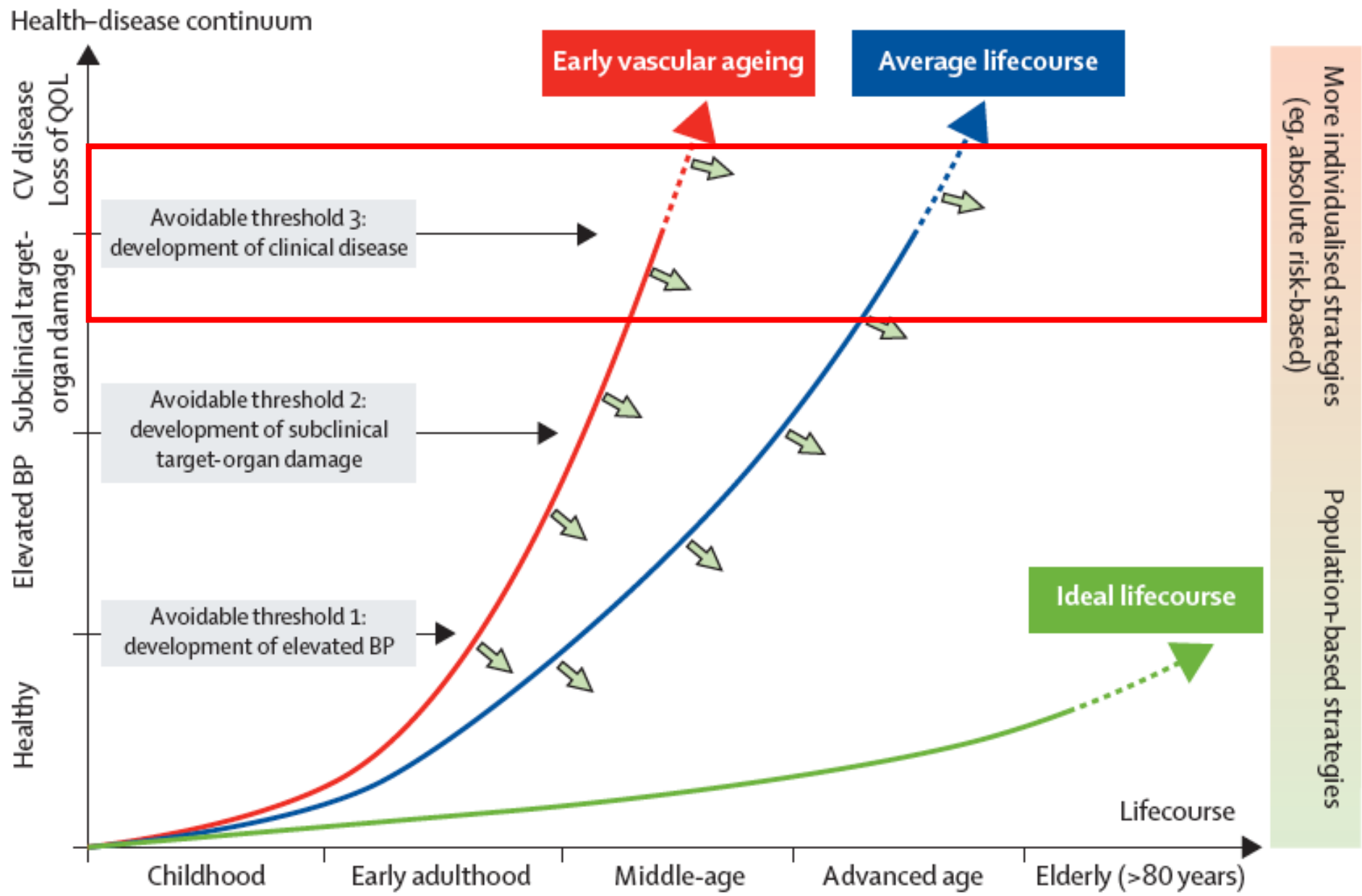
Do you want to refine current risk estimation using data from a previous visit? ⓘ ○

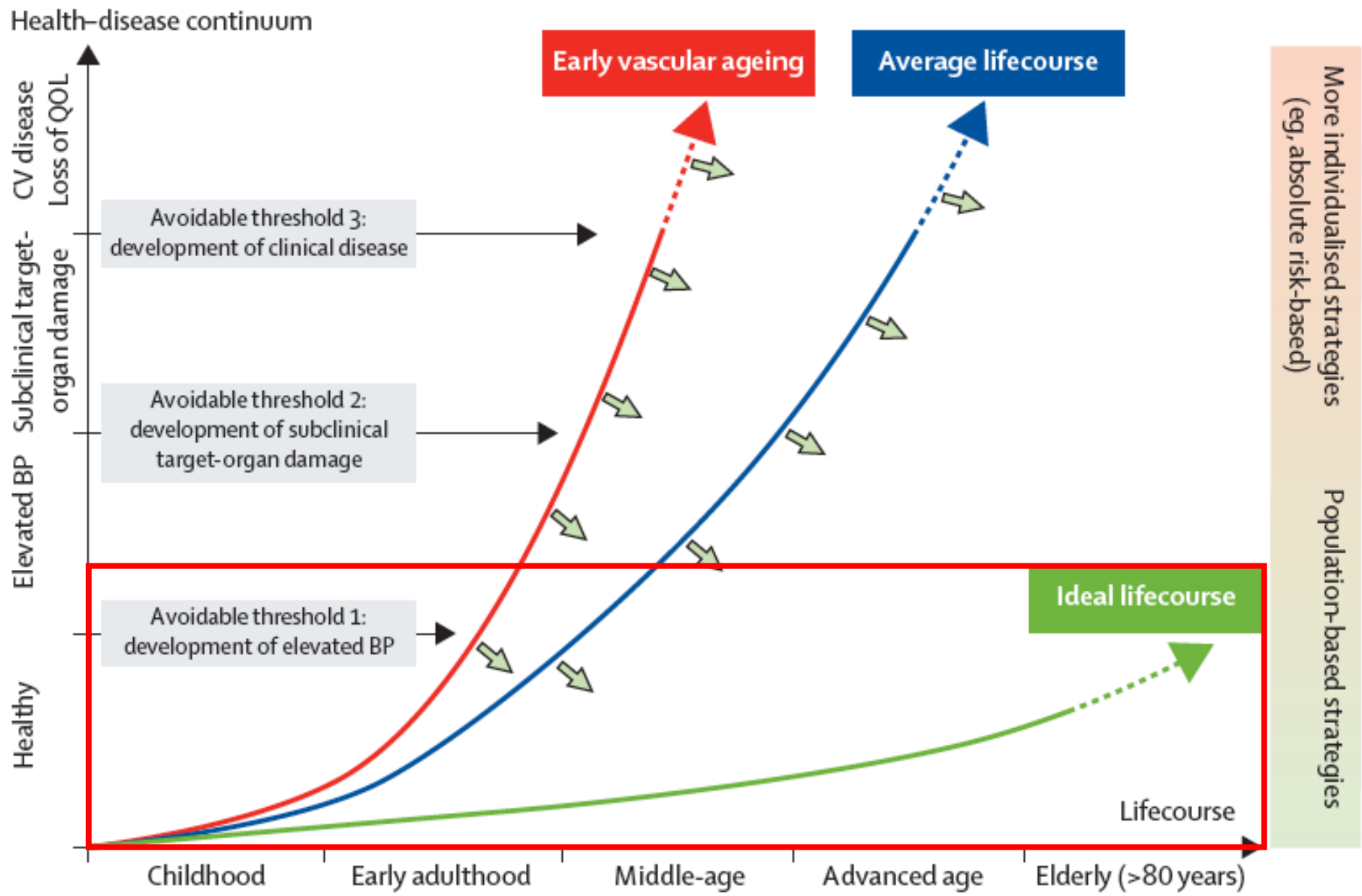
Yes

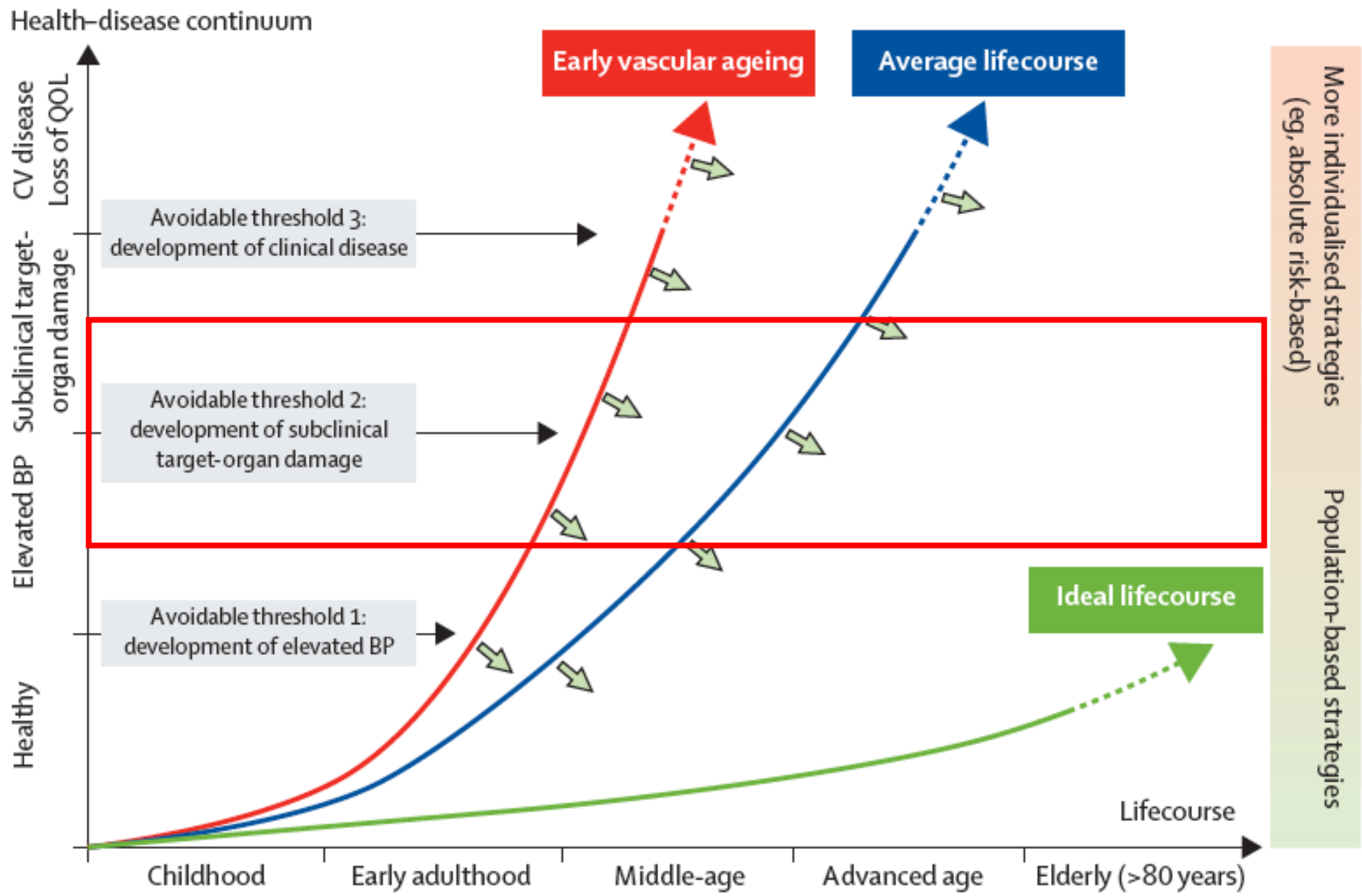
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Asymptomatic Hypertension in the Emergency Department: A Matter of Critical Public Health Importance

Phillip D. Levy, MD, MPH, and David Cline, MD

Abstract

Asymptomatic hypertension (HTN) is commonly encountered in the emergency department (ED), but in most circumstances little is done about it. While many factors may contribute to this, the failure to recognize asymptomatic HTN as a public health problem is particularly important. Given the established long-term consequences of elevated blood pressure (BP), a reconsideration of methods that could enhance surveillance and intervention in the ED is needed. In this article, we discuss the relevant epidemiology of asymptomatic HTN and present a novel approach using a modified version of the Haddon's matrix to systematically address the challenges that contribute to ineffective screening and suboptimal outcomes.

ACADEMIC EMERGENCY MEDICINE 2009; 16:1251–1257 © 2009 by the Society for Academic Emergency Medicine

Keywords: asymptomatic hypertension, public health, effective screening, Haddon's matrix

Subclinical Hypertensive Heart Disease in Black Patients With Elevated Blood Pressure in an Inner-City Emergency Department

Phillip Levy, MD, MPH, Hong Ye, MS, Scott Compton, PhD, Robert Zalenski, MD, Timothy Byrnes, MD, John M. Flack, MD, MPH, Robert Welch, MD, MS

Left-Ventricular Dysfunction	Left-Ventricular Hypertrophy		Total, No. (%)*
	Yes	No	
None	12	0	12 (8.2)
Diastolic dysfunction alone	60	49	109 (74.7)
Systolic dysfunction alone	2	1	3 (2.1)
Systolic and diastolic dysfunction	15	7	22 (15.1)
Total, No. (%)*	89 (61.0)	57 (39.0)	146 (100)

*Percentage of patients with subclinical hypertensive heart disease (n=146).

Effect of Lower Blood Pressure Goals on Left Ventricular Structure and Function in Patients With Subclinical Hypertensive Heart Disease

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BACKGROUND

Subclinical hypertensive heart disease (SHHD) is a precursor to heart failure. Blood pressure (BP) reduction is an important component of secondary disease prevention in patients with SHHD. Treating patients with SHHD utilizing a more intensive BP target (120/80 mm Hg), may lead to improved cardiac function but there has been limited study of this, particularly in African Americans (AAs).

METHODS

We conducted a single center, randomized controlled trial where subjects with uncontrolled, asymptomatic hypertension, and SHHD not managed by a primary care physician were randomized to standard (<140/90 mm Hg) or intensive (<120/80 mm Hg) BP therapy groups with quarterly follow-up for 12 months. The primary outcome was the differences of BP reduction between these 2 groups and the secondary outcome was the improvement in echocardiographic measures at 12 months.

RESULTS

Patients (95% AAs, 65% male, mean age 49.4) were randomized to the standard ($n = 65$) or the intensive ($n = 58$) BP therapy groups. Despite significant reductions in systolic BP (sBP) from baseline (-10.9 vs.

-19.1 mm Hg, respectively) ($P < 0.05$), no significant differences were noted between intention-to-treat groups ($P = 0.33$) or the proportion with resolution of SHHD ($P = 0.31$). However, on *post hoc* analysis, achievement of a sBP <130 mm Hg was associated with significant reduction in indexed left ventricular mass (-6.91 gm/m^{2.7}; $P = 0.008$) which remained significant on mixed effect modeling ($P = 0.031$).

CONCLUSIONS

In *post hoc* analysis, sBP <130 mm Hg in predominantly AA patients with SHHD was associated with improved cardiac function and reverse remodeling and may help to explain preventative effects of lower BP goals.

CLINICAL TRIALS REGISTRATION

Trial Number NCT00689819.

Keywords: African American; blood pressure; heart failure; hypertension; left ventricular hypertrophy; subclinical hypertensive heart disease; urban

doi:10.1093/ajh/hpaa108

Does Vitamin D Provide Added Benefit to Antihypertensive Therapy in Reducing Left Ventricular Hypertrophy Determined by Cardiac Magnetic Resonance?

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BACKGROUND

Left ventricular hypertrophy (LVH) and vitamin D deficiency have been linked to hypertension (HTN) and cardiovascular disease, particularly in African Americans (AAs). Our objective was to determine if the addition of vitamin D to antihypertensive therapy would lead to greater regression of LV mass index (LVMI) as determined by cardiac magnetic resonance (CMR) after 1 year in vitamin D deficient AA patients with uncontrolled HTN and LVH.

METHODS

This study was a randomized, double-blind, placebo-controlled, single-center study. AA patients with HTN (systolic blood pressure [BP] >160 mm Hg), increased LVMI, and vitamin D deficiency (<20 ng/ml) were randomized. All patients received antihypertensive therapy combined with biweekly 50,000 IU vitamin D3 (vitamin D group, $n = 55$) or placebo (placebo group, $n = 58$).

RESULTS

At 1 year, there were no statistical differences between the vitamin D and placebo groups in LVMI (-14.1 ± 14.6 vs. -16.9 ± 13.1 g/m²; $P = 0.34$) or systolic BP (-25.6 ± 32.1 vs. -25.7 ± 25.6 mm Hg; $P = 0.99$) reduction, respectively. Serum vitamin D levels increased significantly in the vitamin D group compared with placebo (12.7 ± 2.0 vs. 1.8 ± 8.2 ng/ml; $P < 0.001$).

CONCLUSIONS

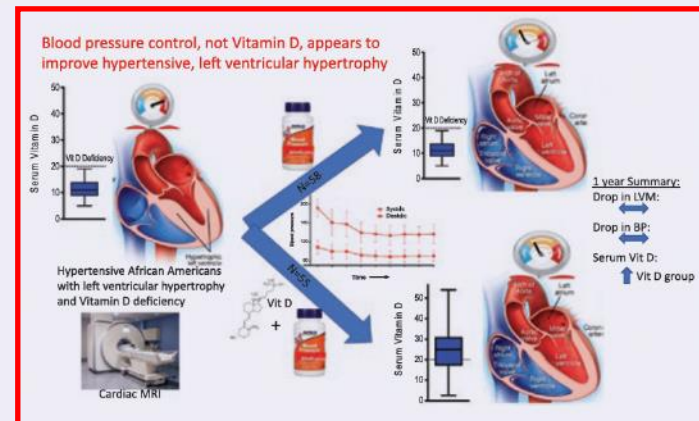
In this high-risk cohort of AAs we did not find an association between vitamin D supplementation and differential regression of LVMI or reduction in systolic BP. However, our study suffered from a small sample

size with low statistical power precluding a definitive conclusion on the therapeutic benefit of vitamin D in such patients.

CLINICAL TRIALS REGISTRATION

Trial Number NCT01360476. Full trial protocol is available from corresponding author.

GRAPHICAL ABSTRACT



Keywords: African Americans; blood pressure; cardiac magnetic resonance imaging; hypertension; left ventricular hypertrophy; subclinical hypertensive heart disease; vitamin D deficiency.

<https://doi.org/10.1093/ajh/hpac096>

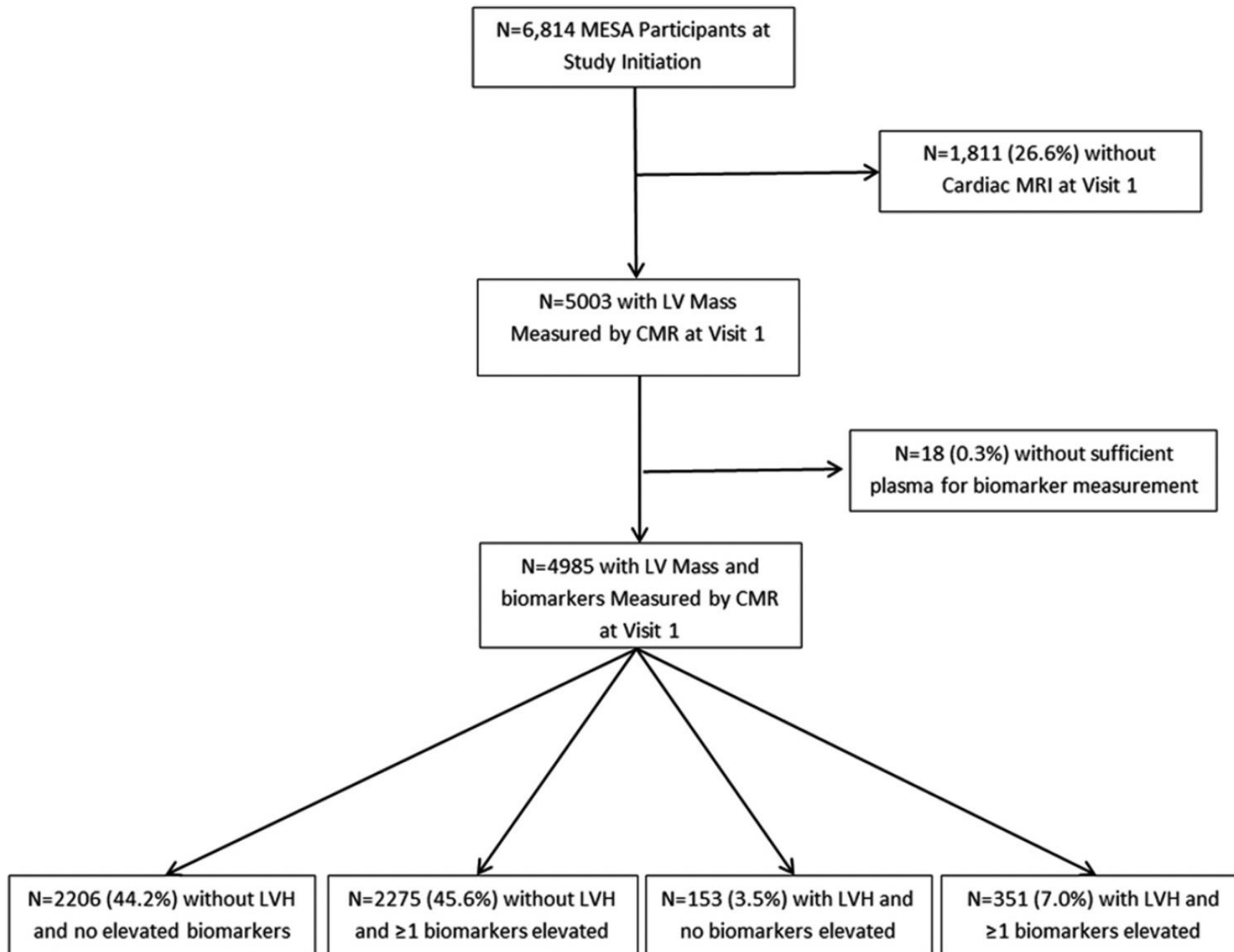
“Malignant” Left Ventricular Hypertrophy Identifies Subjects at High Risk for Progression to Asymptomatic Left Ventricular Dysfunction, Heart Failure, and Death: MESA (Multi-Ethnic Study of Atherosclerosis)

Matthew N. Peters, MD; Stephen L. Seliger, MD, MS; Robert H. Christenson, PhD; Susie N. Hong-Zohlman, MD; Lori B. Daniels, MD, MAS; Joao A.C. Lima, MD; James A. de Lemos, MD; Ian J. Neeland, MD; Christopher R. deFilippi, MD

Background—As heart failure (HF)-associated morbidity and mortality continue to escalate, enhanced focus on prevention is increasingly important. “Malignant” left ventricular (LV) hypertrophy (LVH): LVH combined with an elevated cardiac biomarker reflecting either injury (high-sensitivity cardiac troponin T), or strain (amino-terminal pro-B-type natriuretic peptide) has predicted accelerated progression to HF. We sought to determine whether malignant LVH identified community-dwelling adults initially free of cardiovascular disease at high risk of asymptomatic decline in LV ejection fraction or a clinical cardiovascular event.

Methods and Results—A total of 4985 of 6814 individuals without prevalent cardiovascular disease underwent baseline cardiac magnetic resonance for LVH in combination with measurement of plasma high-sensitivity cardiac troponin T and amino-terminal pro-B-type natriuretic peptide as part of MESA (Multi-Ethnic Study of Atherosclerosis) and were subsequently divided into 4 groups: (1) No LVH, no elevated biomarkers (n=2206; 44.3%); (2) No LVH, ≥ 1 elevated biomarkers (n=2275; 45.7%); (3) LVH, no elevated biomarkers (n=153; 3.0%); and (4) LVH, ≥ 1 elevated biomarkers (malignant LVH; n=351; 7.0%). Cardiac magnetic resonance was repeated 10 years later (n=2831) for assessment of LV ejection fraction $< 50\%$. Median follow-up was 12.2 years. Malignant LVH was associated with 7.0-, 3.5-, and 2.6-fold adjusted increases in incidence of HF, cardiovascular death, and asymptomatic LV dysfunction, respectively, versus group 1. New-onset HF was predominately HF with reduced ejection fraction (9.5-fold increase).

Conclusions—Malignant LVH is predictive of progression to asymptomatic LV dysfunction, HF (particularly HF with reduced ejection fraction), and cardiovascular death. Consequently, malignant LVH represents a high-risk phenotype among individuals without known cardiovascular disease, which should be targeted for increased surveillance and more-aggressive therapies. (*J Am Heart Assoc.* 2018;7:e006619. DOI: 10.1161/JAHA.117.006619.)



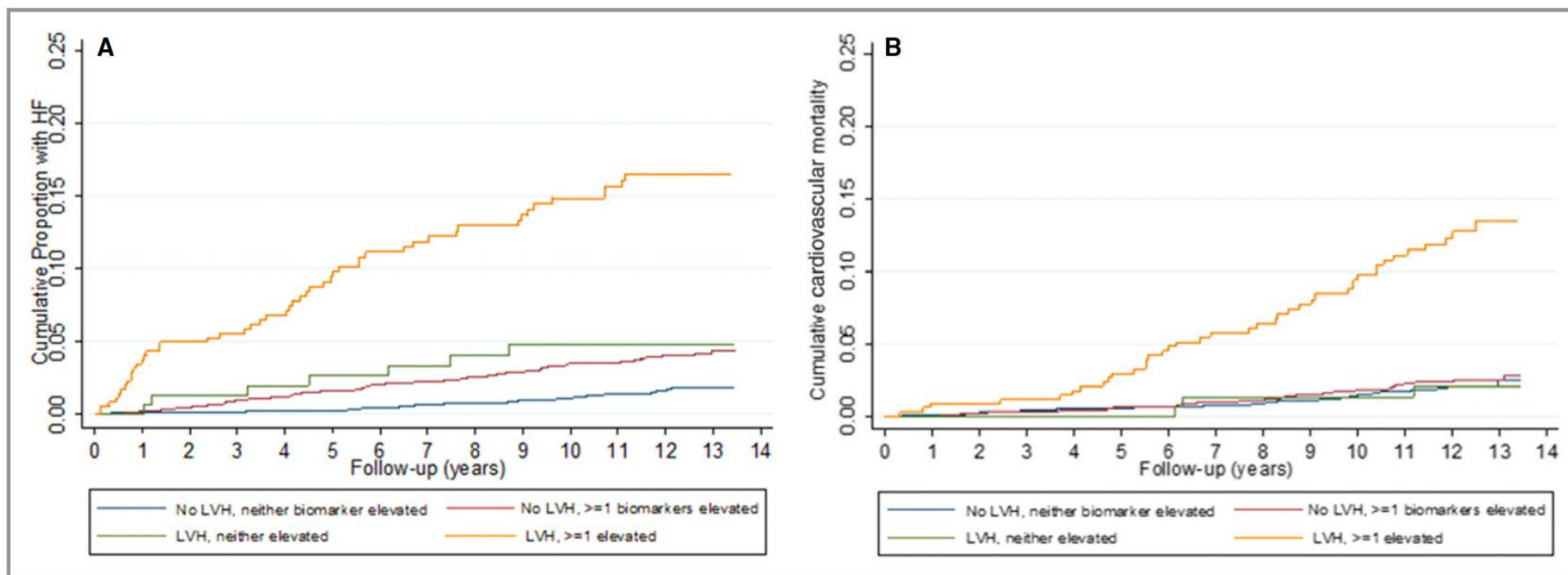


Figure 2. A, Cumulative incidence of HF, by LVH-biomarker group. Kaplan–Meier curve depicting cumulative risk of HF among: (1) No LVH, no elevated biomarker; (2) No LVH, ≥ 1 elevated biomarker; (3) LVH, no elevated biomarker; and (4) LVH, ≥ 1 elevated biomarker groups over median follow-up period of 12 years. HF indicates heart failure; LVH, left ventricular hypertrophy. B, Cumulative cardiovascular death, by LVH-biomarker group. Kaplan–Meier curve depicting cumulative risk of cardiovascular mortality among: (1) No LVH, no elevated biomarker; (2) No LVH, ≥ 1 elevated biomarker; (3) LVH, no elevated biomarker; and (4) LVH, ≥ 1 elevated biomarker groups over median follow-up period of 12 years. HF indicates heart failure; LVH, left ventricular hypertrophy.

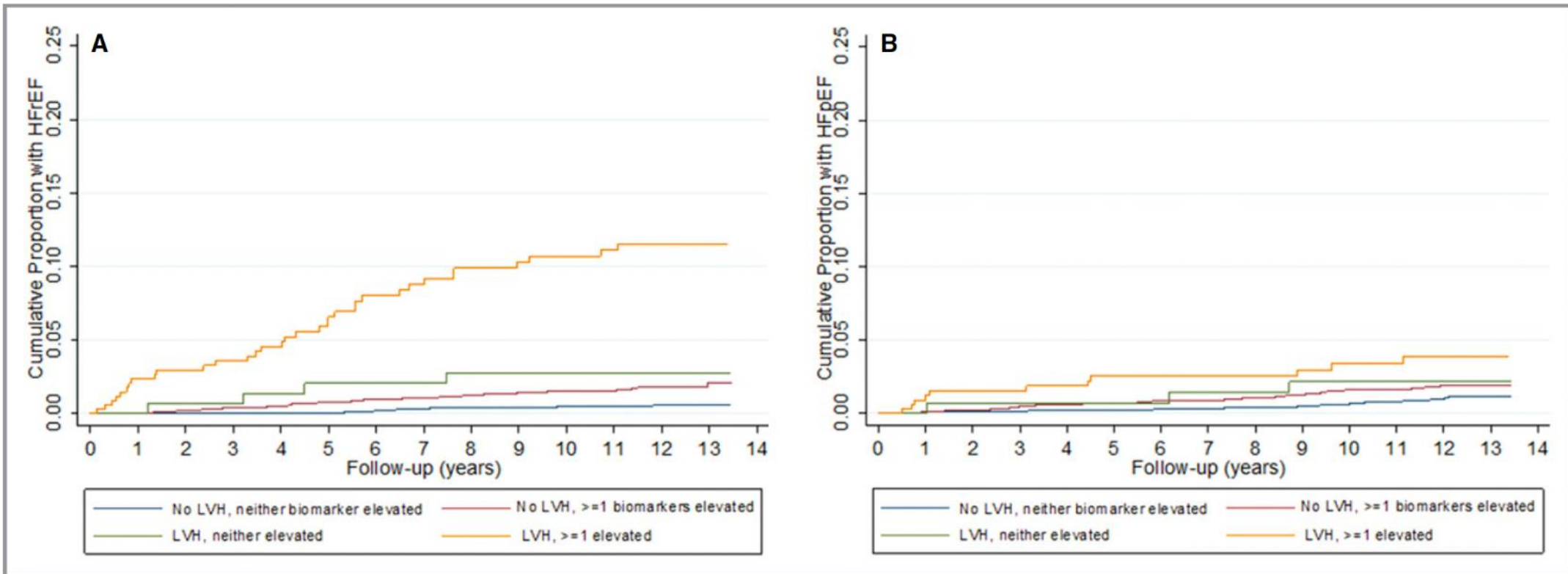


Figure 3. A, Cumulative risk of HFrEF, by LVH-biomarker group. Kaplan–Meier curve depicting cumulative risk of HFrEF among: (1) No LVH, no elevated biomarker; (2) No LVH, ≥ 1 elevated biomarker; (3) LVH, no elevated biomarker; and (4) LVH, ≥ 1 elevated biomarker groups over median follow-up period of 12 years. B, Cumulative risk of HFpEF, by LVH-biomarker group. Kaplan–Meier curve depicting cumulative risk of HFpEF among: (1) No LVH, no elevated biomarker; (2) No LVH, ≥ 1 elevated biomarker; (3) LVH, no elevated biomarker; and (4) LVH, ≥ 1 elevated biomarker groups over median follow-up period of 12 years. HFpEF indicates heart failure with preserved ejection fraction; HFrEF, heart failure with reduced ejection fraction; LVH, left ventricular hypertrophy.

Intensive Blood Pressure Lowering in Patients With Malignant Left Ventricular Hypertrophy



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ABSTRACT

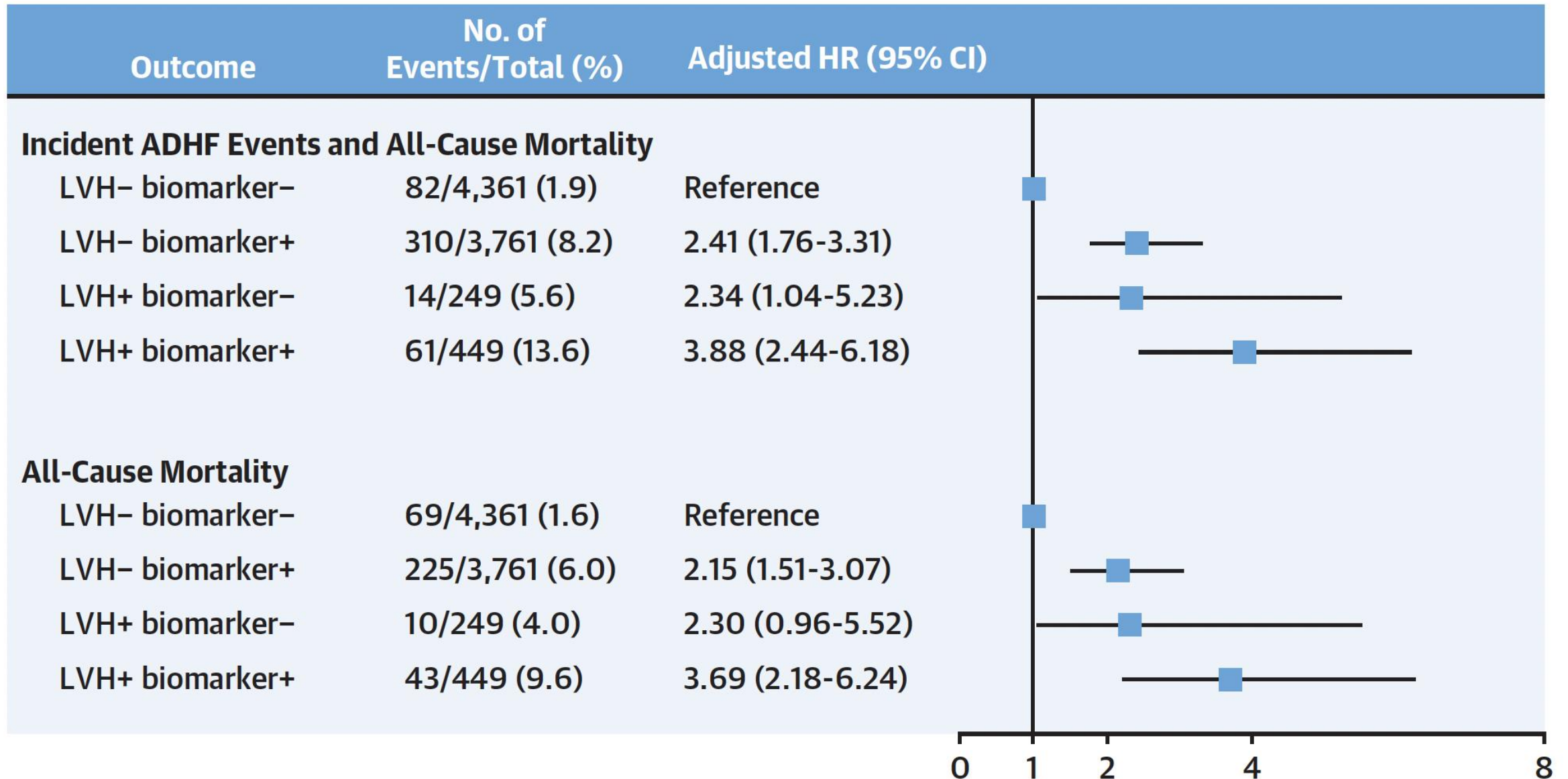
BACKGROUND Left ventricular hypertrophy (LVH) combined with elevations in cardiac biomarkers reflecting myocardial injury and neurohormonal stress (malignant LVH) is associated with a high risk for heart failure and death.

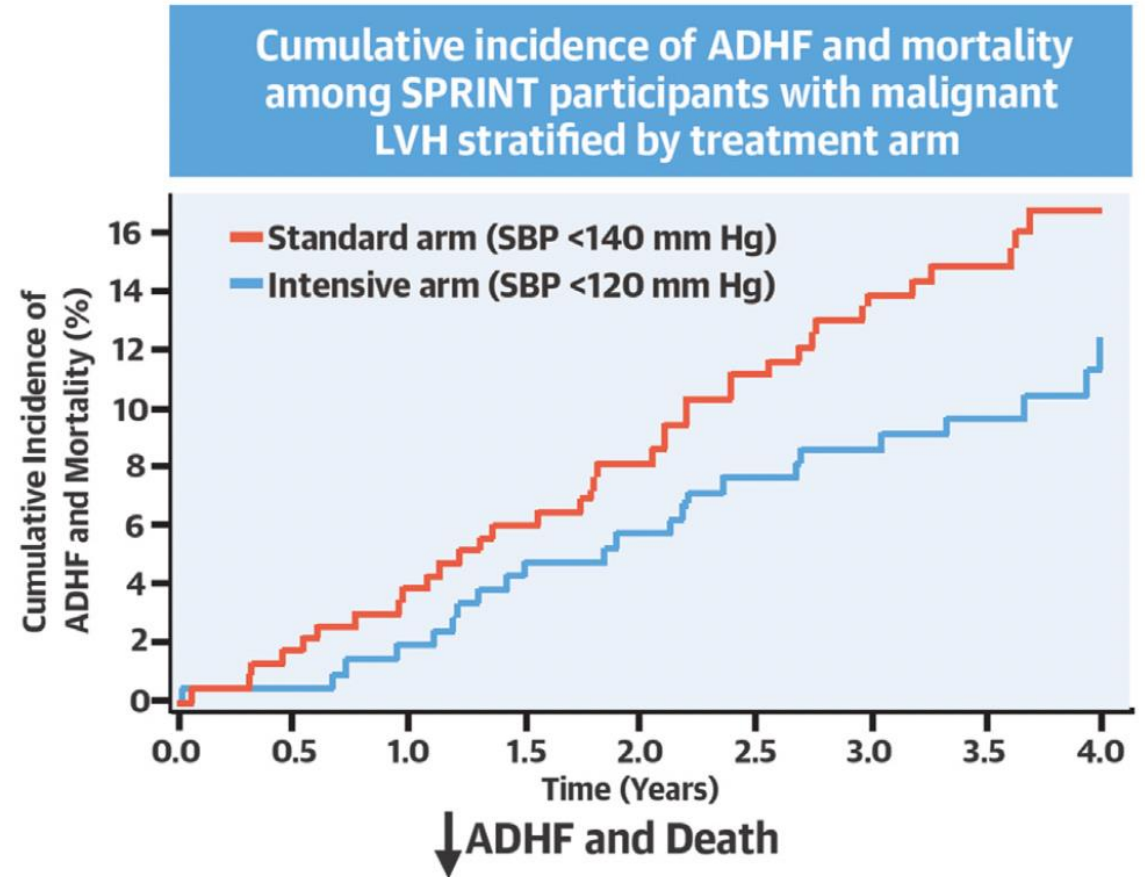
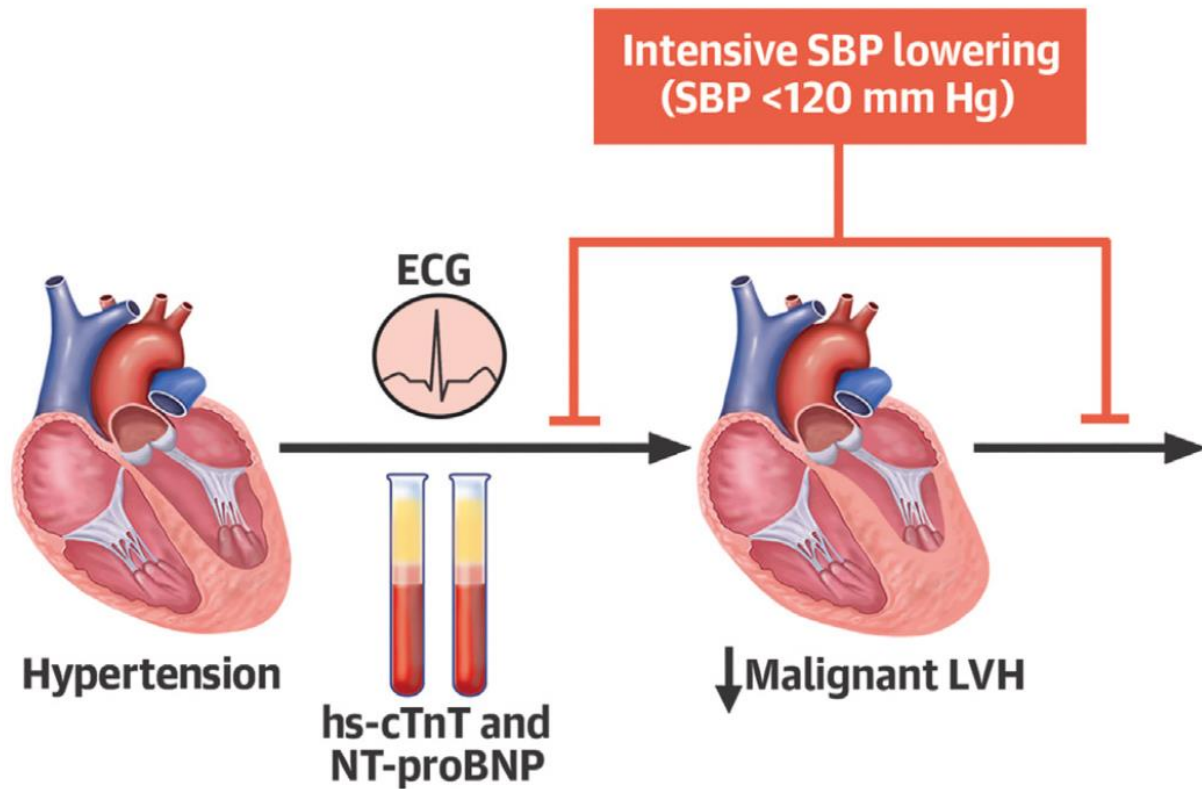
OBJECTIVES The aim of this study was to determine the impact of intensive systolic blood pressure (SBP) control on the prevention of malignant LVH and its consequences.

METHODS A total of 8,820 participants in SPRINT (Systolic Blood Pressure Intervention Trial) were classified into groups based on the presence or absence of LVH assessed by 12-lead ECG, and elevations in biomarker levels (high-sensitivity cardiac troponin T ≥ 14 ng/L or N-terminal pro-B-type natriuretic peptide ≥ 125 pg/mL) at baseline. The effects of intensive vs standard SBP lowering on rates of acute decompensated heart failure (ADHF) events and death and on the incidence and regression of malignant LVH were determined.

RESULTS Randomization to intensive SBP lowering led to similar relative reductions in ADHF events and death across the combined LVH/biomarker groups (P for interaction = 0.68). The absolute risk reduction over 4 years in ADHF events and death was 4.4% (95% CI: -5.2% to 13.9%) among participants with baseline malignant LVH ($n = 449$) and 1.2% (95% CI: 0.0%-2.5%) for those without LVH and nonelevated biomarkers ($n = 4,361$). Intensive SBP lowering also reduced the incidence of malignant LVH over 2 years (2.5% vs 1.1%; OR: 0.44; 95% CI: 0.30-0.63).

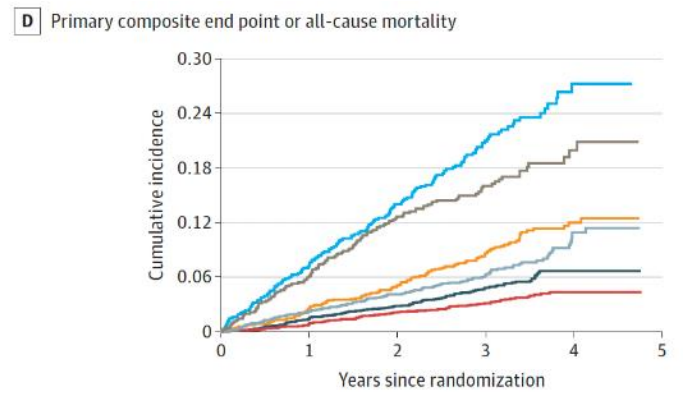
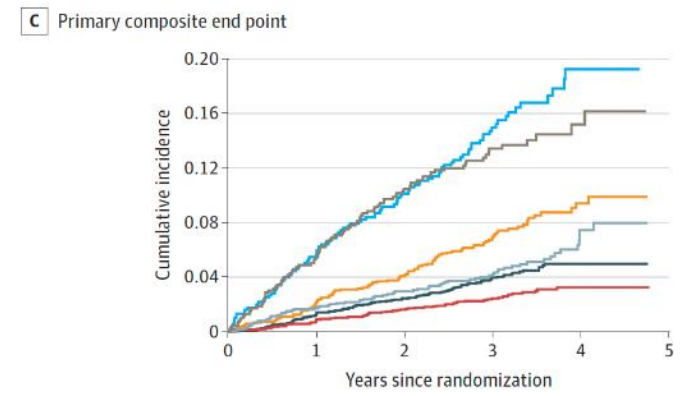
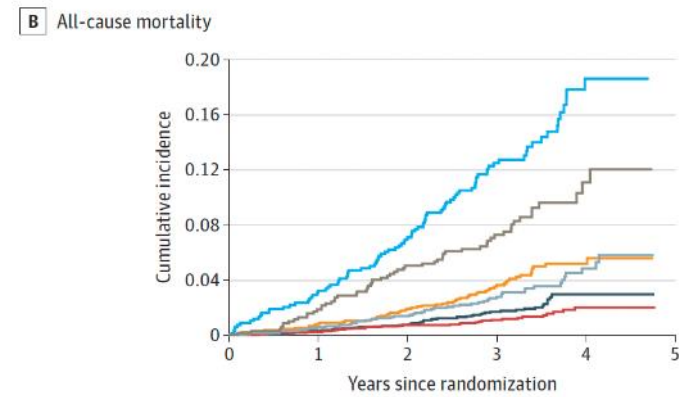
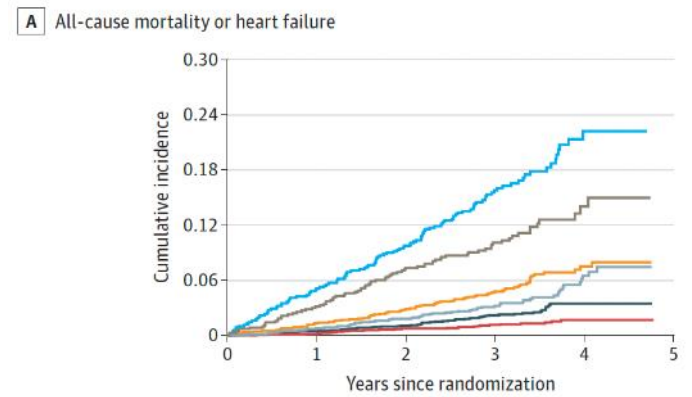
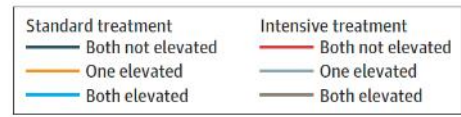
CONCLUSIONS Intensive SBP lowering prevented malignant LVH and may provide substantial absolute risk reduction in the composite of ADHF events and death among SPRINT participants with baseline malignant LVH. (J Am Coll Cardiol 2022;80:1516-1525) © 2022 by the American College of Cardiology Foundation.





Associations of High-Sensitivity Troponin and Natriuretic Peptide Levels With Outcomes After Intensive Blood Pressure Lowering Findings From the SPRINT Randomized Clinical Trial

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Incorporation of Biomarkers Into Risk Assessment for Allocation of Antihypertensive Medication According to the 2017 ACC/AHA High Blood Pressure Guideline

A Pooled Cohort Analysis

BACKGROUND: Risk for atherosclerotic cardiovascular disease was a novel consideration for antihypertensive medication initiation in the 2017 American College of Cardiology/American Heart Association Blood Pressure (BP) guideline. Whether biomarkers of chronic myocardial injury (high-sensitivity cardiac troponin T ≥ 6 ng/L) and stress (N-terminal pro-B-type natriuretic peptide [NT-proBNP] ≥ 100 pg/mL) can inform cardiovascular (CV) risk stratification and treatment decisions among adults with elevated BP and hypertension is unclear.

METHODS: Participant-level data from 3 cohort studies (Atherosclerosis Risk in Communities Study, Dallas Heart Study, and Multiethnic Study of Atherosclerosis) were pooled, excluding individuals with prevalent CV disease and those taking antihypertensive medication at baseline. Participants were analyzed according to BP treatment group from the 2017 American College of Cardiology/American Heart Association BP guideline and those with high BP (120 to 159/ <100 mmHg) were further stratified by biomarker status. Cumulative incidence rates for CV event (atherosclerotic cardiovascular disease or heart failure), and the corresponding 10-year number needed to treat to prevent 1 event with intensive BP lowering (to target systolic BP <120 mmHg), were estimated for BP and biomarker-based subgroups.

RESULTS: The study included 12 987 participants (mean age, 55 years; 55% women; 21.5% with elevated high-sensitivity cardiac troponin T; 17.7% with elevated NT-proBNP) with 825 incident CV events over 10-year follow-up. Participants with elevated BP or hypertension not recommended for antihypertensive medication with versus without either elevated high-sensitivity cardiac troponin T or NT-proBNP had a 10-year CV incidence rate of 11.0% and 4.6%, with a 10-year number needed to treat to prevent 1 event for intensive BP lowering of 36 and 85, respectively. Among participants with stage 1 or stage 2 hypertension recommended for antihypertensive medication with BP $<160/100$ mmHg, those with versus without an elevated biomarker had a 10-year CV incidence rate of 15.1% and 7.9%, with a 10-year number needed to treat to prevent 1 event of 26 and 49, respectively.

CONCLUSIONS: Elevations in high-sensitivity cardiac troponin T or NT-proBNP identify individuals with elevated BP or hypertension not currently recommended for antihypertensive medication who are at high risk for CV events. The presence of nonelevated biomarkers, even in the setting of stage 1 or stage 2 hypertension, was associated with lower risk. Incorporation of biomarkers into risk assessment algorithms may lead to more appropriate matching of intensive BP control with patient risk.

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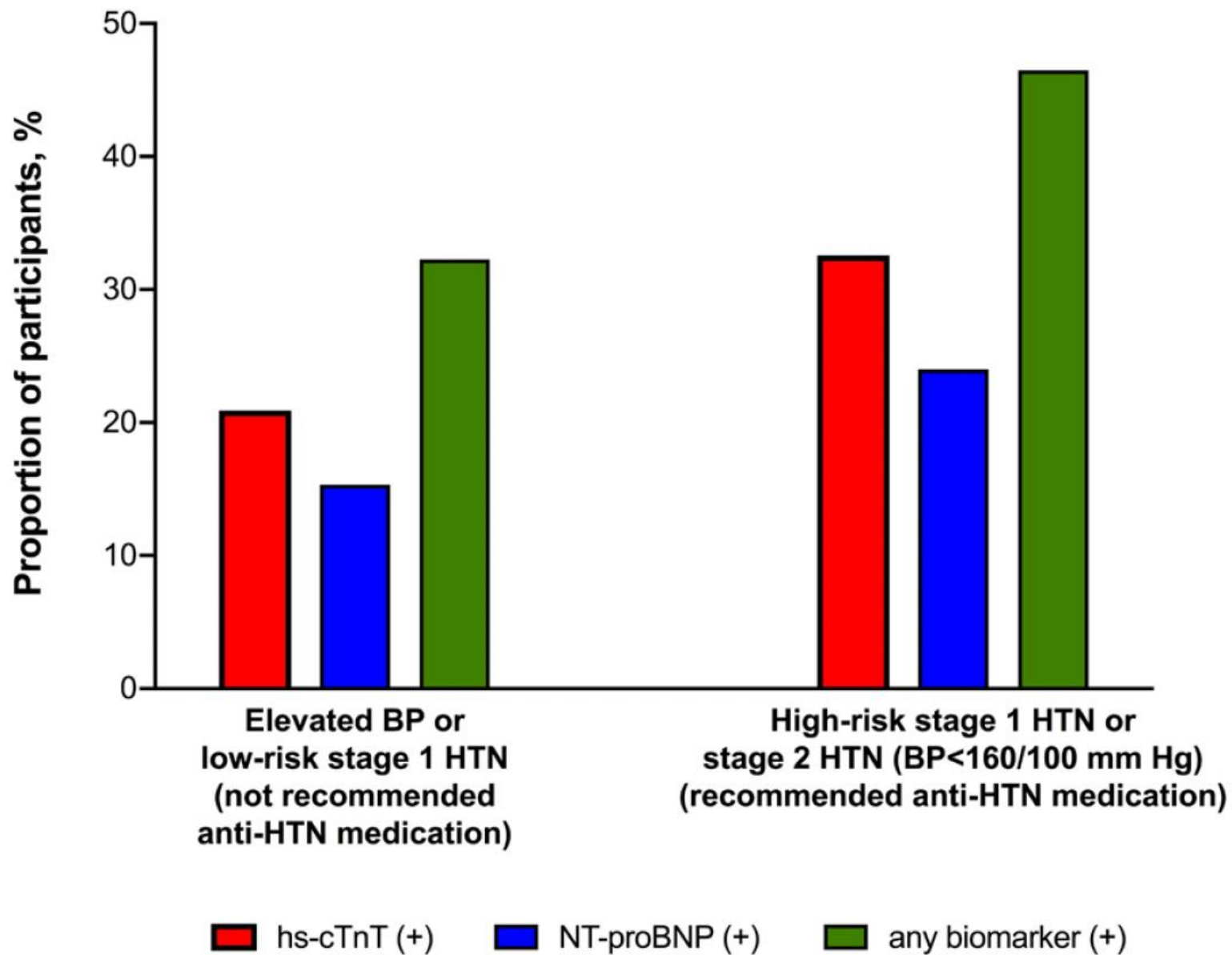
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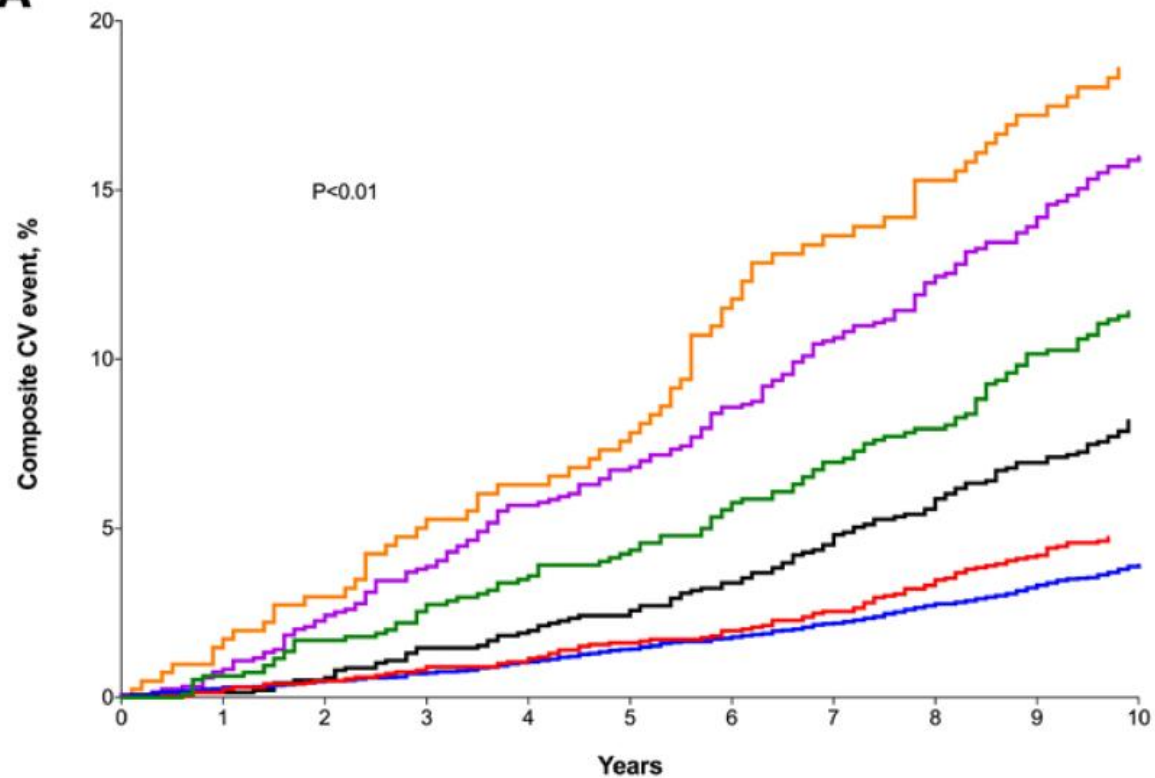
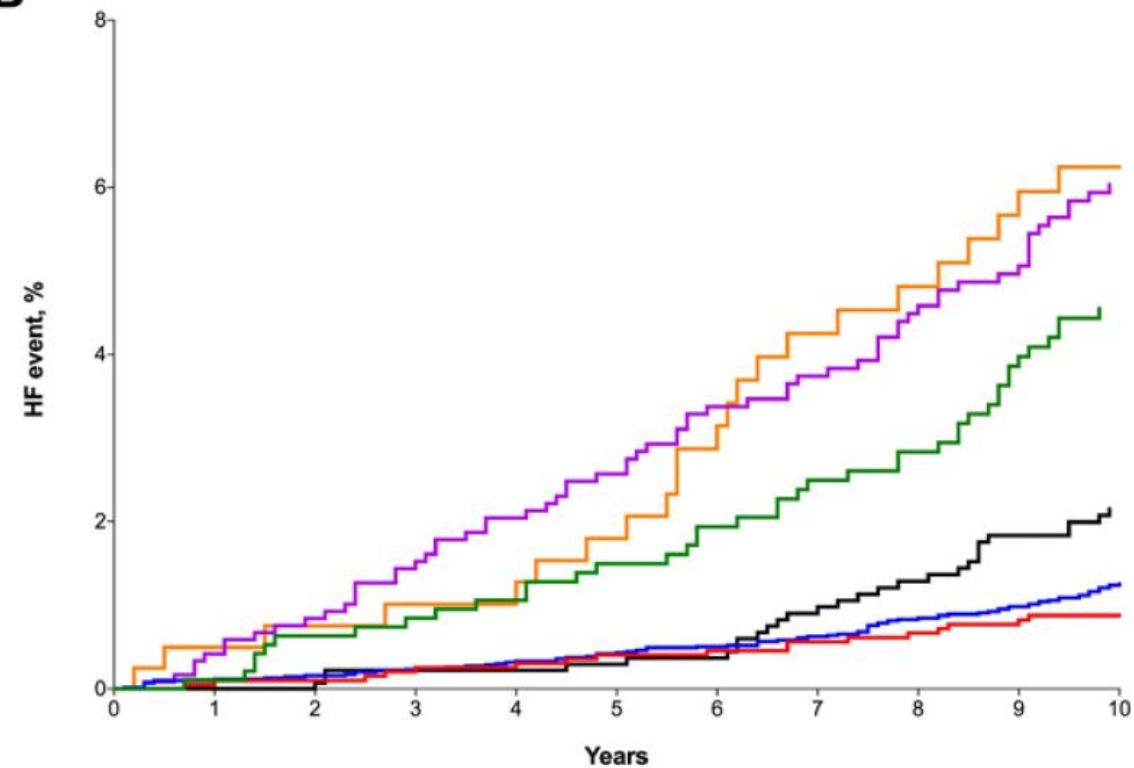
Key Words: biomarkers ■ heart failure
■ hypertension ■ myocardial infarction
■ risk

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A**B**

- Normal BP
- Elevated BP or low-risk stage 1 HTN, not recommended anti-HTN medication, both biomarker (-)
- Elevated BP or low-risk stage 1 HTN, not recommended anti-HTN medication, any biomarker (+)
- High-risk stage 1 HTN or stage 2 HTN (BP $< 160/100$ mmHg), recommended anti-HTN medication, both biomarker (-)
- High-risk stage 1 HTN or stage 2 HTN (BP $< 160/100$ mmHg), recommended anti-HTN medication, any biomarker (+)
- Stage 2 HTN (BP $\geq 160/100$ mmHg)

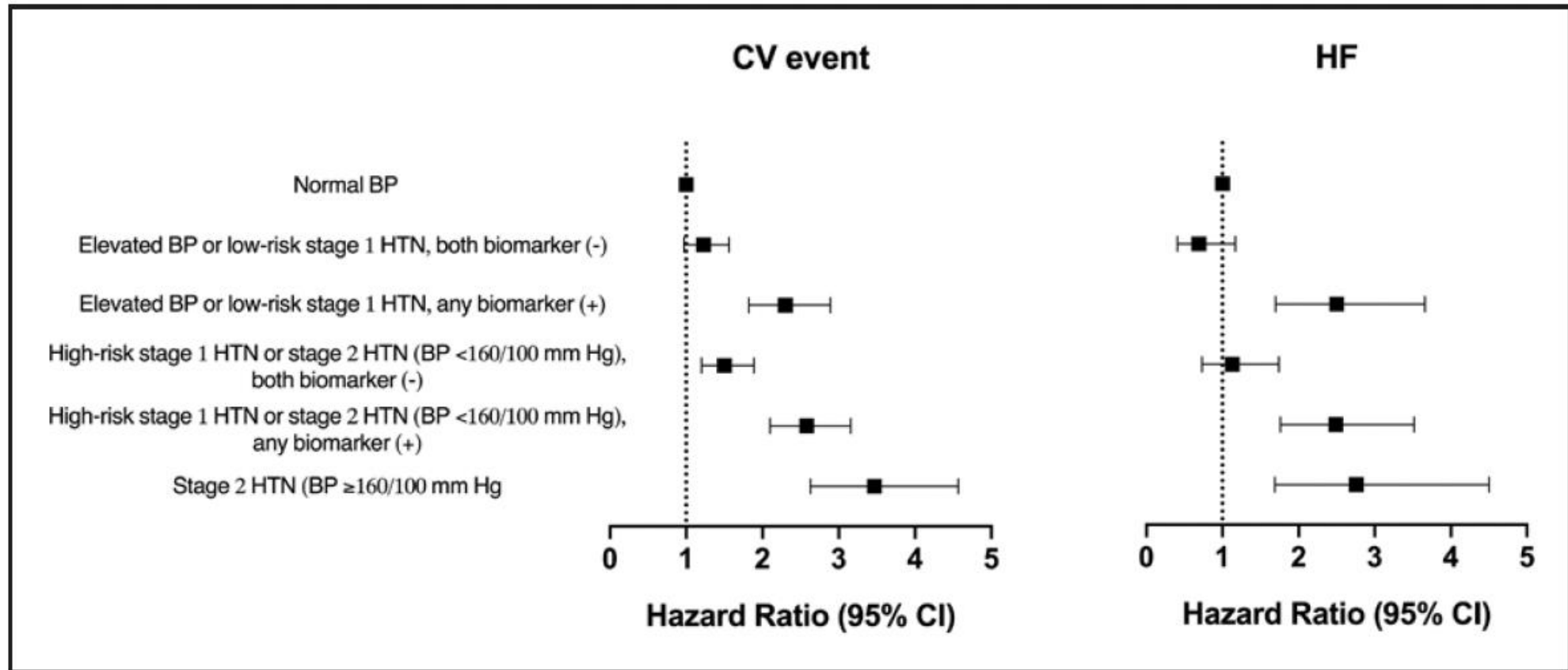


Figure 5. Multivariable adjusted association between the 2017 ACC/AHA BP guideline recommended treatment groups stratified by biomarker status and incident composite CV event (ASCVD or HF) and HF.

Multivariable adjusted Cox models were constructed to evaluate the association between the BP/biomarker-based study groups and risk of outcome (composite CV event [nonfatal MI, nonfatal stroke, HF, or CV death] or HF [referent group=normal BP]) with adjustment for the following potential confounders: demographics (age, sex, race), CV risk factors (BMI, diabetes mellitus status, smoking status), laboratory values (total cholesterol, HDL cholesterol, estimated glomerular filtration rate), medications (statin use), and study cohort. Elevated BP, 120 to 129/<80 mmHg; stage 1 HTN, 130 to 139/80 to 89 mmHg; stage 2 HTN, $\geq 140/90$ mmHg; high-risk stage 1 HTN was defined by the presence of any of the following: PCE-estimated 10-year ASCVD risk $\geq 10\%$, diabetes mellitus, estimated GFR <60 mL/min per 1.73 m², or age ≥ 65 years with systolic BP ≥ 130 mmHg; in the absence of all of these risk factors, individuals with stage 1 HTN were classified as low-risk; any biomarker (+), hs-cTnT ≥ 6 ng/L and/or NT-proBNP ≥ 100 pg/mL. ASCVD indicates atherosclerotic cardiovascular disease; BMI, body mass index; BP, blood pressure; CV, cardiovascular; GFR, glomerular filtration rate; HDL, high density lipoprotein; HF, heart failure; hs-cTnT, high-sensitivity cardiac troponin T; HTN, hypertension; MI, myocardial infarction; NT-proBNP, N-terminal pro-B-type natriuretic peptide; and PCE, Pooled Cohort Equation.

Combining Biomarkers and Imaging for Short-Term Assessment of Cardiovascular Disease Risk in Apparently Healthy Adults

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BACKGROUND: Current strategies for cardiovascular disease (CVD) risk assessment focus on 10-year or longer timeframes. Shorter-term CVD risk is also clinically relevant, particularly for high-risk occupations, but is under-investigated.

METHODS AND RESULTS: We pooled data from participants in the ARIC (Atherosclerosis Risk in Communities study), MESA (Multi-Ethnic Study of Atherosclerosis), and DHS (Dallas Heart Study), free from CVD at baseline (N=16 581). Measurements included N-terminal pro-B-type natriuretic peptide (>100 pg/mL prospectively defined as abnormal); high-sensitivity cardiac troponin T (abnormal >5 ng/L); high-sensitivity C-reactive protein (abnormal >3 mg/L); left ventricular hypertrophy by ECG (abnormal if present); carotid intima-media thickness, and plaque (abnormal >75th percentile for age and sex or presence of plaque); and coronary artery calcium (abnormal >10 Agatston U). Each abnormal test result except left ventricular hypertrophy by ECG was independently associated with increased 3-year risk of global CVD (myocardial infarction, stroke, coronary revascularization, incident heart failure, or atrial fibrillation), even after adjustment for traditional CVD risk factors and the other test results. When a simple integer score counting the number of abnormal tests was used, 3-year multivariable-adjusted global CVD risk was increased among participants with integer scores of 1, 2, 3, and 4, by \approx 2-, 3-, 4.5- and 8-fold, respectively, when compared with those with a score of 0. Qualitatively similar results were obtained for atherosclerotic CVD (fatal or non-fatal myocardial infarction or stroke).

CONCLUSIONS: A strategy incorporating multiple biomarkers and atherosclerosis imaging improved assessment of 3-year global and atherosclerotic CVD risk compared with a standard approach using traditional risk factors.

Table 2. Hazard Ratios (95% CIs) for the Associations of Biomarkers With Risk of Global CVD Events in the Combined Cohorts

	Unadjusted	Adjusted for Base Model*	Adjusted for Base Model and eGFR	Adjusted for Base Model, eGFR, and the Other Biomarkers
Total, n	16 581	16 551	16 506	16 506
Number of events	553	553	551	551
Biomarkers as categorical variables				
ECG-LVH	1.11 (0.87–1.43)	1.10 (0.85–1.41)
hs-CRP ≥3 mg/L	1.4 (1.18–1.65)	1.30 (1.08–1.56)	1.28 (1.07–1.54)	1.23 (1.03–1.48)
NT-proBNP ≥100 pg/mL	2.06 (1.73–2.44)	2.20 (1.82–2.67)	2.16 (1.78–2.62)	2.00 (1.65–2.43)
hs-cTnT ≥5 ng/L	2.16 (1.80–2.60)	1.47 (1.21–1.79)	1.45 (1.19–1.77)	1.32 (1.08–1.62)
Plaque and/or IMT >75th percentile (ARIC only)	3.31 (2.69–4.08)	1.96 (1.57–2.45)	1.95 (1.56–2.44)	1.8 (1.43–2.26)
CAC >10 (DHS and MESA only)	5.07 (4.24–6.05)	2.38 (1.96–2.90)	2.38 (1.96–2.89)	2.2 (1.81–2.68)
Plaque and/or IMT >75th percentile and/or CAC >10 (combined cohorts)	3.28 (2.69–3.99)	2.16 (1.76–2.67)	2.15 (1.75–2.65)	1.99 (1.61–2.46)
Biomarkers as log-transformed continuous variables				
hs-CRP (log)	1.26 (1.16–1.36)	1.18 (1.07–1.29)	1.17 (1.06–1.28)	1.13 (1.03–1.24)
NT-proBNP (log)	1.87 (1.72–2.03)	1.65 (1.52–1.80)	1.70 (1.55–1.87)	1.59 (1.44–1.75)
Hs-cTnT (log)	1.90 (1.77–2.04)	1.36 (1.24–1.49)	1.35 (1.23–1.49)	1.19 (1.08–1.31)

ARIC indicates Atherosclerosis Risk in Communities study; CAC, coronary artery calcium score; ECG-LVH, left ventricular hypertrophy by ECG; HDL, high-density lipoprotein; hs-CRP, high sensitivity C-reactive protein; hs-cTnT, high-sensitivity cardiac troponin T; IMT, intima-media thickness; LDL, low-density lipoprotein; and NT-proBNP, N-terminal prohormone of B-type natriuretic peptide.

*Base model adjustment included pooled cohort equation variables (age, sex, black race, total cholesterol, high-density lipoprotein [HDL] cholesterol, systolic blood pressure, antihypertensive medication use, current smoking, and diabetes mellitus status) and body mass index.

Table 3. Hazard Ratios (95% CIs) for the Associations of Biomarkers With Risk of Atherosclerotic CVD Events in the Combined Cohorts

	Unadjusted	Adjusted for Base Model*	Adjusted for Base Model and eGFR	Adjusted for Base Model, eGFR, and the Other Biomarkers
Total, n	16 581	16 565	16 519	16 519
Number of events	260	260	259	259
Biomarkers as categorical variables				
ECG-LVH	1.15 (0.89–1.50)	0.98 (0.68–1.42)
hs-CRP ≥3 mg/L	1.31 (1.02–1.67)	1.12 (0.92–1.56)	1.19 (0.91–1.55)	1.14 (0.88–1.49)
NT-proBNP ≥100 pg/mL	1.85 (1.43–2.38)	1.76 (1.33–2.33)	1.73 (1.3–2.31)	1.66 (1.25–2.21)
hs-cTnT ≥5 ng/L	1.8 (1.38–2.34)	1.07 (0.81–1.42)	1.06 (0.8–1.41)	0.98 (0.74–1.31)
Plaque and/or IMT >75th percentile (ARIC only)	3.43 (2.49–4.72)	2.03 (1.45–2.85)	2.01 (1.43–2.83)	1.91 (1.36–2.67)
CAC >10 (DHS and MESA only)	4.14 (3.25–5.28)	1.66 (1.27–2.16)	1.66 (1.27–2.16)	1.56 (1.20–2.03)
Plaque and/or IMT >75th percentile and/or CAC >10 (combined cohorts)	3.12 (2.35–4.13)	1.80 (1.34–2.41)	1.78 (1.33–2.4)	1.69 (1.26–2.28)
Biomarkers as log-transformed continuous variables				
hs-CRP (log)	1.20 (1.07–1.35)	1.11 (0.97–1.27)	1.10 (0.96–1.27)	1.08 (0.94–1.24)
NT-proBNP (log)	1.63 (1.45–1.84)	1.37 (1.21–1.55)	1.39 (1.21–1.59)	1.33 (1.16–1.52)
Hs-cTnT (log)	1.81 (1.62–2.01)	1.21 (1.05–1.38)	1.20 (1.04–1.38)	1.10 (0.95–1.27)

ARIC indicates Atherosclerosis Risk in Communities study; CAC, coronary artery calcium score; ECG-LVH, left ventricular hypertrophy by ECG; HDL, high-density lipoprotein; hs-CRP, high sensitivity C-reactive protein; hs-cTnT, high-sensitivity cardiac troponin T; IMT, intima-media thickness; LDL, low density lipoprotein; and NT-proBNP, N-terminal prohormone of B-type natriuretic peptide.

*Base model adjustment included pooled cohort equations variables (age, sex, black race, total cholesterol, high-density lipoprotein cholesterol, systolic blood pressure, antihypertensive medication use, current smoking, and diabetes mellitus status) and body mass index.

Table 4. Changes in c-Statistic With All Four Biomarkers Added to the Base Model*, Both as Categorical and as Continuous Variables, in the Combined Cohorts

Global CVD			Atherosclerotic CVD		
c-Statistic Base Model (95% CI)	c-Statistic Base Model+Biomarkers (95% CI)	P Value for c-Statistic w/ vs w/o Biomarkers	c-Statistic Base Model (95% CI)	c-Statistic Base Model+Biomarkers (95% CI)	P Value for c-Statistic w/ vs w/o Biomarkers
Biomarkers as categorical variables [†]					
0.780 (0.762, 0.798)	0.801 (0.784, 0.818)	<0.0001	0.799 (0.774, 0.823)	0.812 (0.789, 0.835)	0.006
Biomarkers as log-transformed continuous variables					
0.780 (0.762, 0.798)	0.805 (0.788, 0.822)	<0.0001	0.799 (0.774, 0.823)	0.811 (0.788, 0.834)	0.006

CAC indicates coronary artery calcium; CVD, cardiovascular disease; hs-CRP, high-sensitivity C-reactive protein; IMT, intima-media thickness; NT-proBNP, N-terminal pro B-type natriuretic peptide.

*Base model includes traditional CVD risk factors, including pooled cohort equation variables (age, sex, black race, total cholesterol, high-density lipoprotein [HDL] cholesterol, systolic blood pressure, antihypertensive medication use, current smoking, and diabetes mellitus status), and bone mass index.

[†]Thresholds were hs-CRP ≥ 3 mg/L; NT-proBNP ≥ 100 pg/mL; hs-cTnT ≥ 5 ng/L; plaque and/or IMT >75th percentile and/or CAC >10.

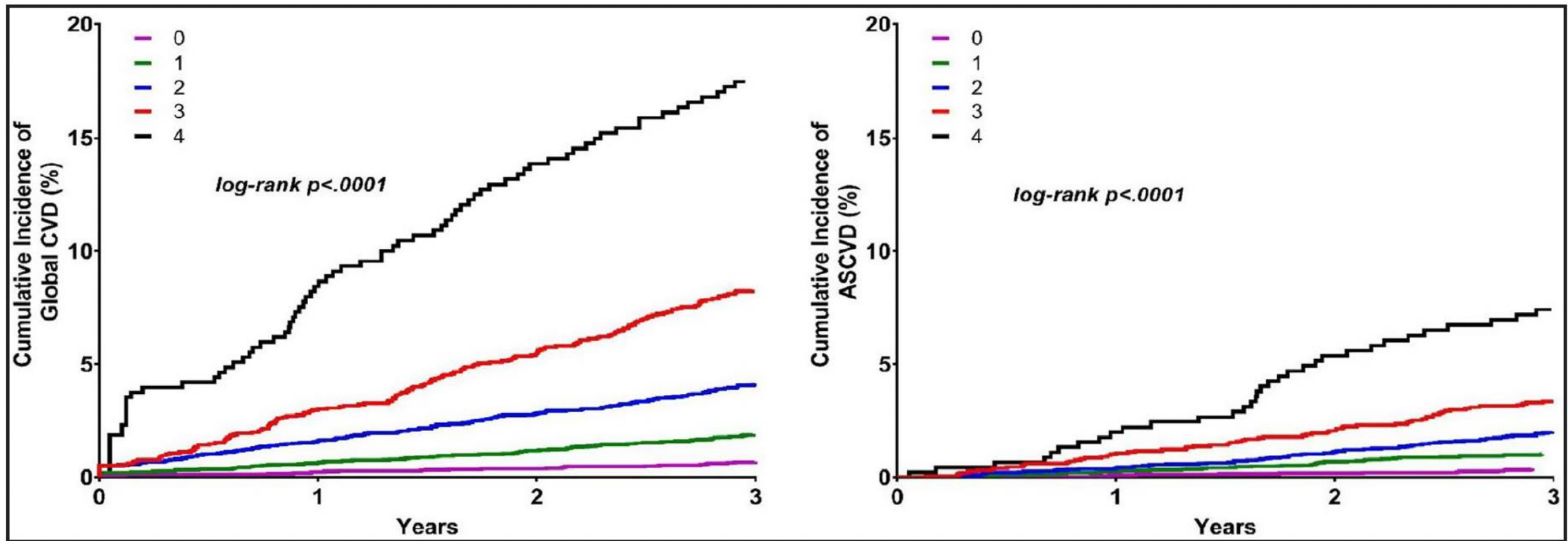


Figure 1. Cumulative incidence rates of global cardiovascular disease (CVD) and atherosclerotic cardiovascular disease composite outcomes stratified by the number of abnormal test results.

We pooled data from participants in 3 large population-based cohorts, free from CVD at baseline (N=16 581), with measurements including N-terminal pro-B-type natriuretic peptide; high-sensitivity cardiac troponin T; high-sensitivity C-reactive protein; and a composite imaging measure of subclinical atherosclerosis (coronary artery calcium or carotid intima-media thickness or plaque). Using an integer score to count the number of abnormal tests in each study participant, higher integer scores were associated with higher incidence of global CVD events (myocardial infarction, stroke, coronary revascularization, incident heart failure, or atrial fibrillation) and atherosclerotic CVD events (fatal or non-fatal myocardial infarction or stroke) during 3 years of follow-up. ASCVD indicates atherosclerotic cardiovascular disease; and CVD, cardiovascular disease.

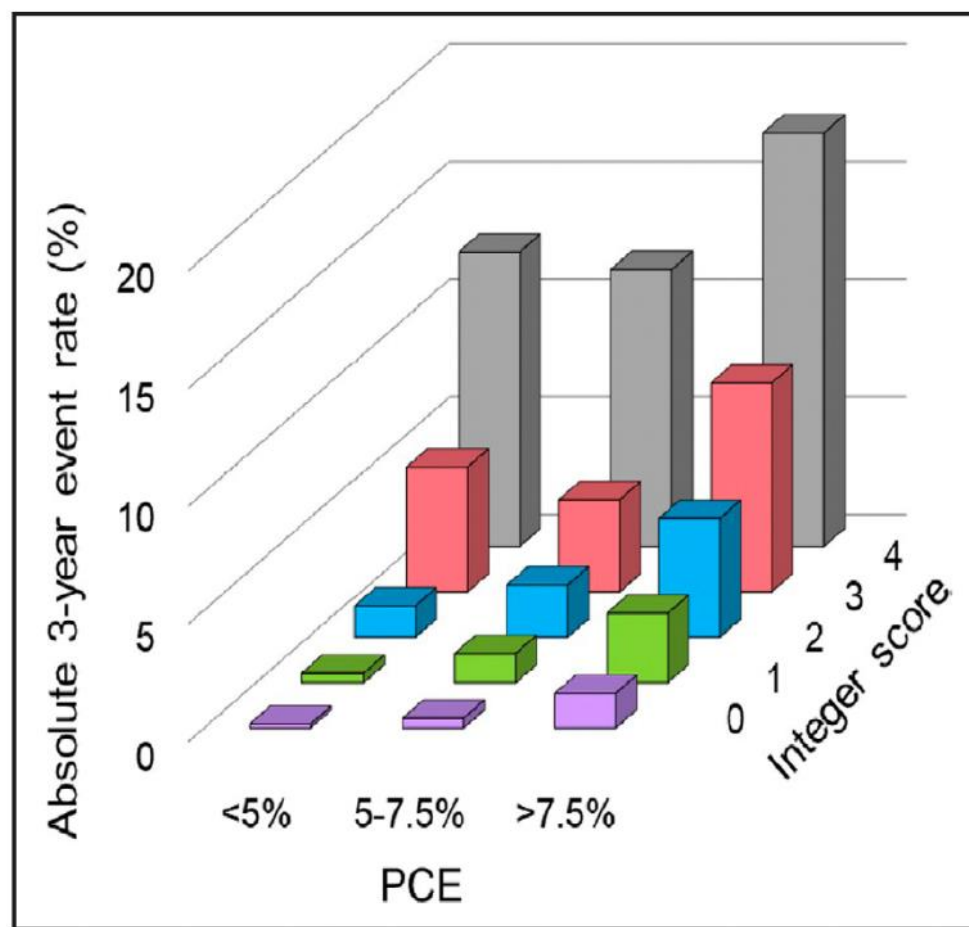


Figure 3. Absolute 3-year global cardiovascular disease event rates in the combined cohorts stratified by pooled cohort equation-estimated risk and the number of abnormal test results.

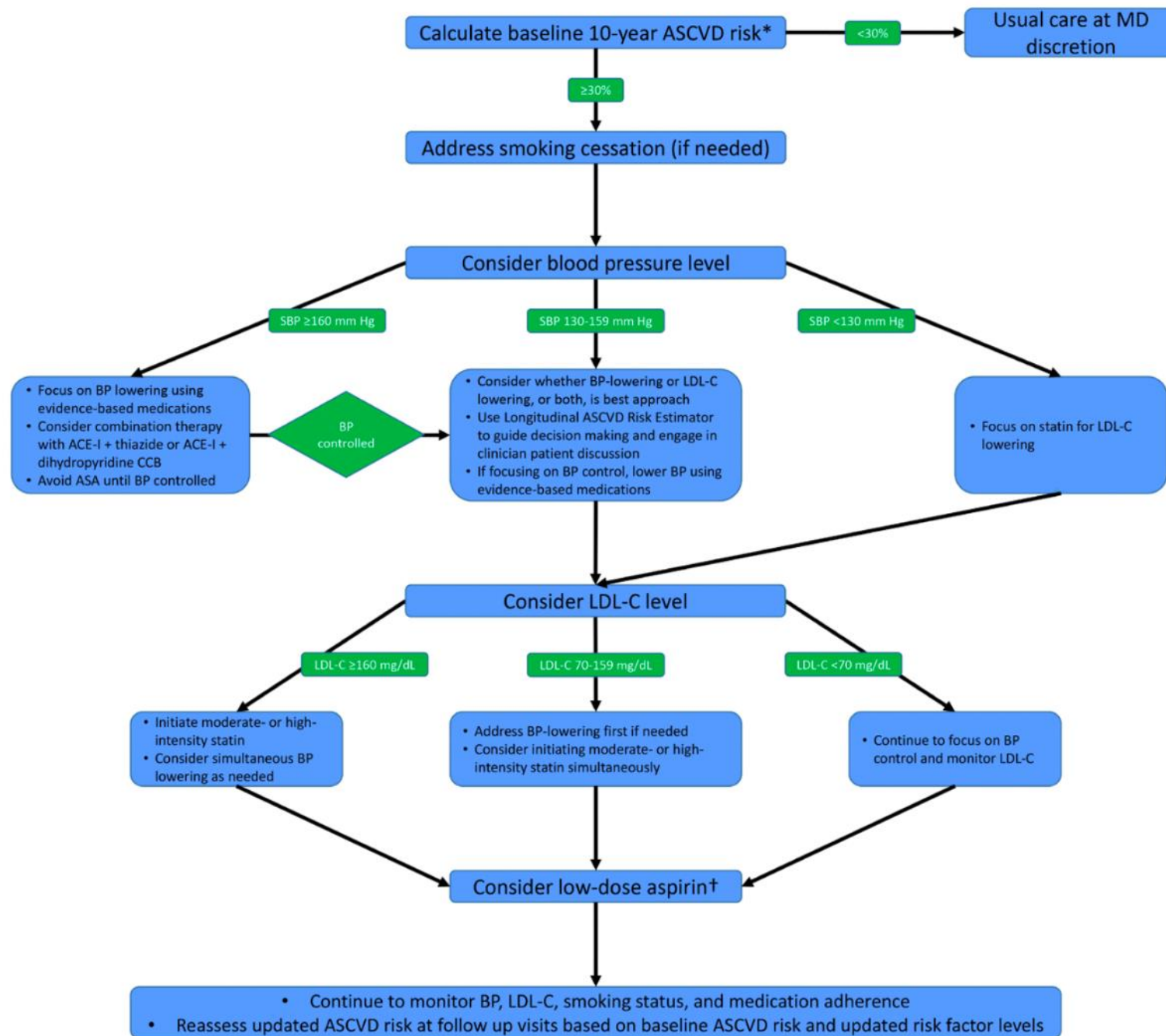
Higher integer scores (representing the number of abnormal test results) were associated with higher 3-year global cardiovascular disease risk across pooled cohort equation categories, and, conversely, a low integer score (0 or 1) was associated with lower global cardiovascular disease risk even among individuals with borderline or elevated 10-year risk by pooled cohort equations. PCE indicates pooled cohort equations.

Estimating Longitudinal Risks and Benefits From Cardiovascular Preventive Therapies Among Medicare Patients

The Million Hearts Longitudinal ASCVD Risk Assessment Tool: A Special Report From the American Heart Association and American College of Cardiology

ABSTRACT: The Million Hearts Initiative has a goal of preventing 1 million heart attacks and strokes—the leading causes of mortality—through several public health and healthcare strategies by 2017. The American Heart Association and American College of Cardiology support the program. The Cardiovascular Risk Reduction Model was developed by Million Hearts and the Center for Medicare & Medicaid Services as a strategy to assess a value-based payment approach toward reduction in 10-year predicted risk of atherosclerotic cardiovascular disease (ASCVD) by implementing cardiovascular preventive strategies to manage the “ABCS” (aspirin therapy in appropriate patients, blood pressure control, cholesterol management, and smoking cessation). The purpose of this special report is to describe the development and intended use of the Million Hearts Longitudinal ASCVD Risk Assessment Tool. The Million Hearts Tool reinforces and builds on the “2013 ACC/AHA Guideline on the Assessment of Cardiovascular Risk” by allowing clinicians to estimate baseline and updated 10-year ASCVD risk estimates for primary prevention patients adhering to the appropriate ABCS over time, alone or in combination. The tool provides updated risk estimates based on evidence from high-quality systematic reviews and meta-analyses of the ABCS therapies. This novel approach to personalized estimation of benefits from risk-reducing therapies in primary prevention may help target therapies to those in whom they will provide the greatest benefit, and serves as the basis for a Center for Medicare & Medicaid Services program designed to evaluate the Million Hearts Cardiovascular Risk Reduction Model.

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2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure



A Report of the American College of Cardiology/American Heart Association
Joint Committee on Clinical Practice Guidelines

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Clyde W. Yancy, MD, MSc, MACC, FAHA, FHFSA[†]

[†]ACC/AHA Representative. [‡]ACC/AHA Joint Committee on Clinical Practice Guidelines Liaison. [§]ACC/AHA Task Force on Performance Measures Representative. ^{||}HFSA Representative.

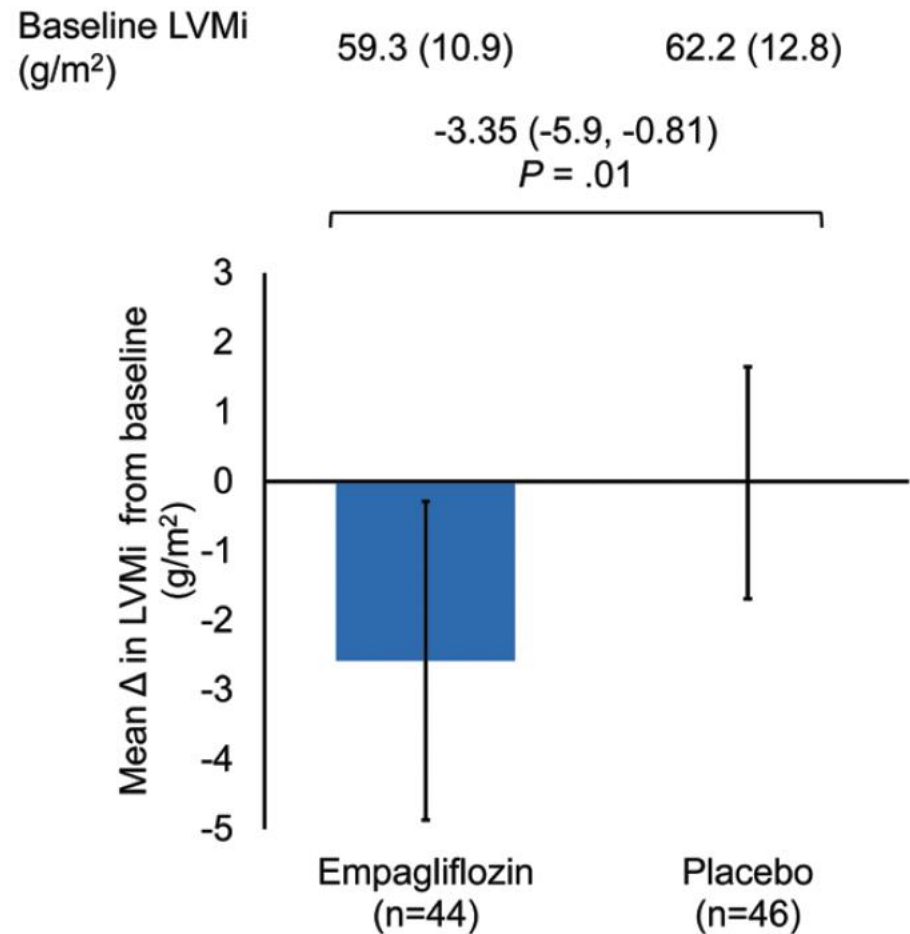
Guideline Directed Medical Therapy Across Heart Failure Stages

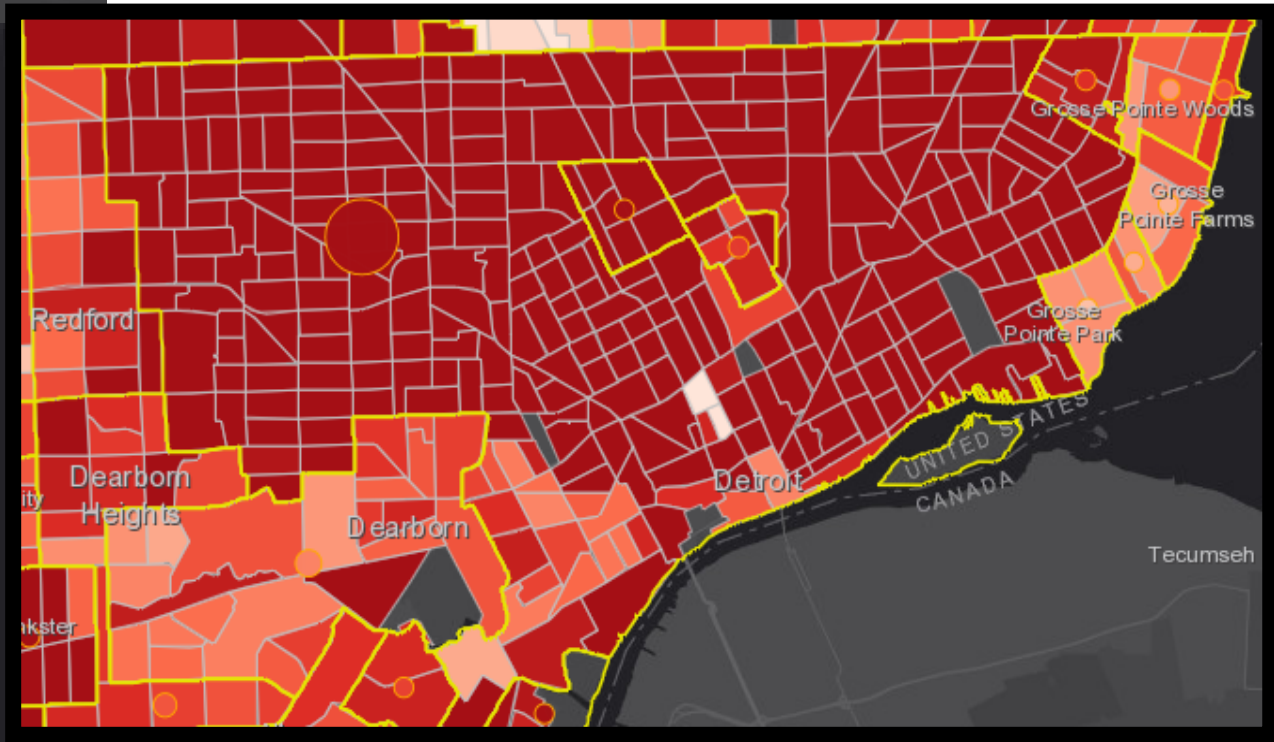
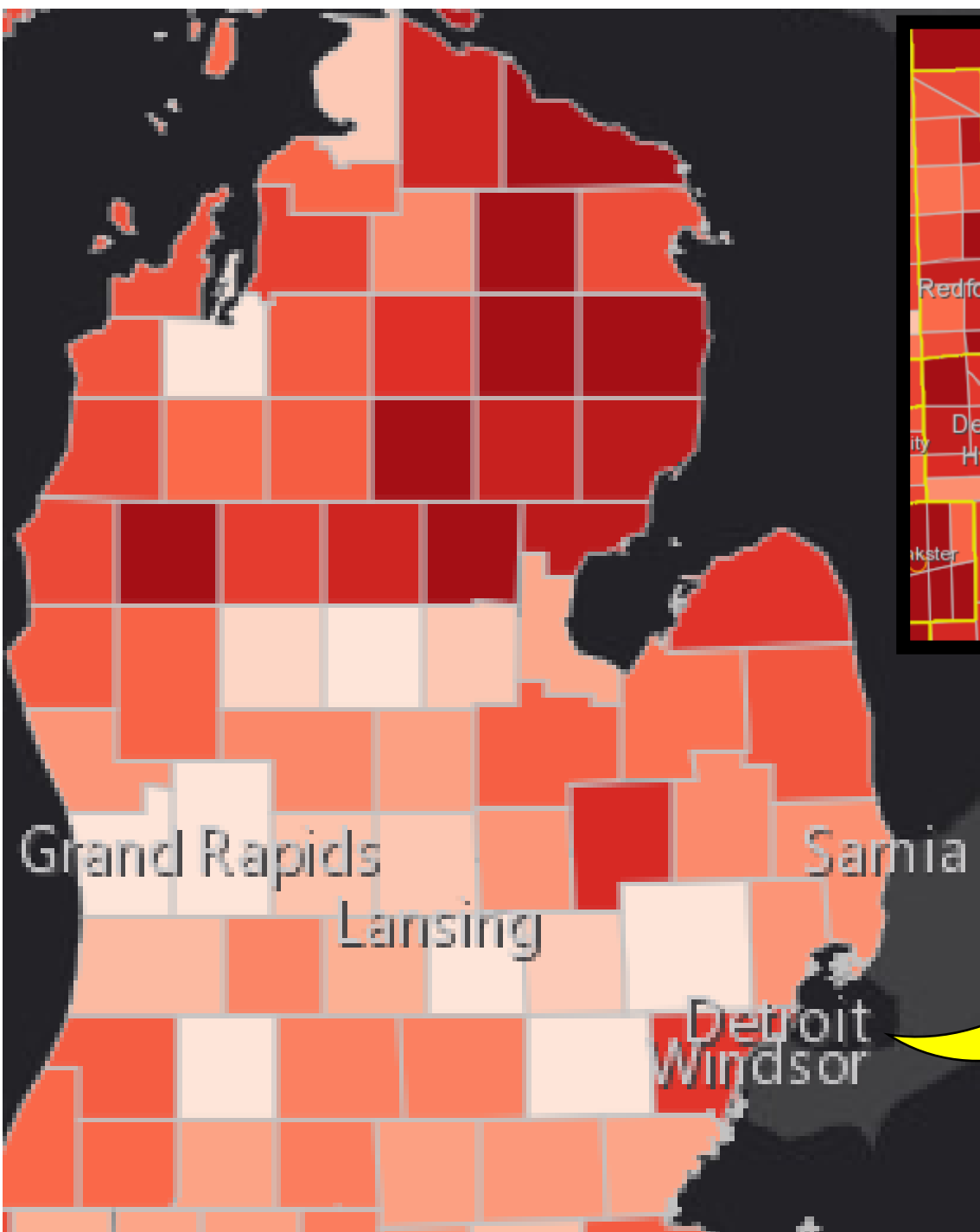
Use this tool to reference guideline directed medical therapy (GDMT) across the four ACC/AHA stages of Heart Failure (HF) as outlined in the 2022 AHA/ACC/HFSA Guideline for the Management of Heart Failure. See the guideline for specific patient population criteria.

	Stage C & D					
	Stage A	Stage B	Stage C: Symptomatic Heart Failure & Stage D: Advanced Heart Failure			
	At-Risk for Heart Failure	Pre-Heart Failure	HFREF LVEF ≤40% HFmrEF LVEF 41-49% HFrEF LVEF ≥50%			
GDMT of major medication classes	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i in pts with DM (1)</div>	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i in pts with DM (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">ACEi (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">ARB if ACEi intolerant (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Beta blocker (1)</div>	<table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 33%; border-right: 1px dashed black; padding: 5px;"> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">ARNi in NYHA II-III; ACEi or ARB in NYHA II-IV (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Beta blocker (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">MRA (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Hydral-nitrates for NYHA III-IV, in African American pts (1)</div> </td> <td style="width: 33%; border-right: 1px dashed black; padding: 5px;"> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ACEi, ARB, ARNI (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">MRA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Beta blocker (2b)</div> </td> <td style="width: 33%; padding: 5px;"> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ARNi (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">MRA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ARB (2b)</div> </td> </tr> </table>	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">ARNi in NYHA II-III; ACEi or ARB in NYHA II-IV (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Beta blocker (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">MRA (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Hydral-nitrates for NYHA III-IV, in African American pts (1)</div>	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ACEi, ARB, ARNI (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">MRA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Beta blocker (2b)</div>	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Diuretics, as needed (1)</div> <div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">SGLT2i (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ARNi (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">MRA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">ARB (2b)</div>
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Additional Medical Therapies once GDMT optimized	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Optimal control of BP (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Optimal management of CVD (1)</div>	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Optimal control of BP (1)</div> <div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">Optimal management of CVD (1)</div>	<table style="width: 100%; border-collapse: collapse;"> <tr> <td style="width: 33%; border-right: 1px dashed black; padding: 5px;"> <div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">Ivabradine (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Vericiguat (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Digoxin (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">PUFA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Potassium binders (2b)</div> </td> <td style="width: 33%; border-right: 1px dashed black; padding: 5px;"></td> <td style="width: 33%; padding: 5px;"></td> </tr> </table>	<div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">Ivabradine (2a)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Vericiguat (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Digoxin (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">PUFA (2b)</div> <div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">Potassium binders (2b)</div>		
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	<div style="background-color: #4CAF50; color: white; padding: 5px; margin-bottom: 5px;">1 (strong)</div>	<div style="background-color: #FFEB3B; color: white; padding: 5px; margin-bottom: 5px;">2a (Moderate)</div>	<div style="background-color: #FF9800; color: white; padding: 5px; margin-bottom: 5px;">2b (Weak)</div>			

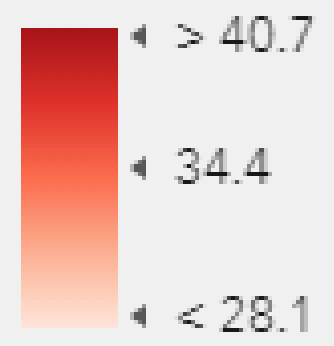
Effect of Empagliflozin on Left Ventricular Mass in Patients With Type 2 Diabetes Mellitus and Coronary Artery Disease

The EMPA-HEART CardioLink-6 Randomized Clinical Trial

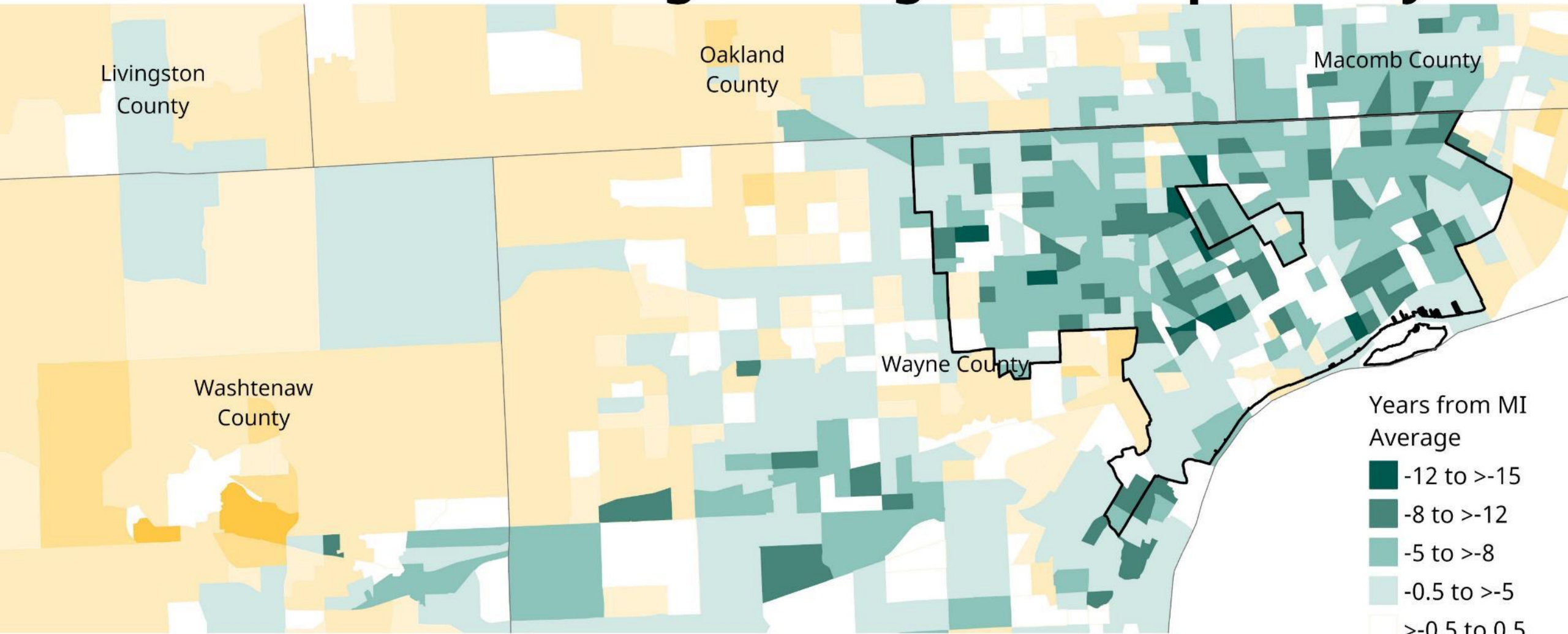




High blood pressure crude prevalence (%)





















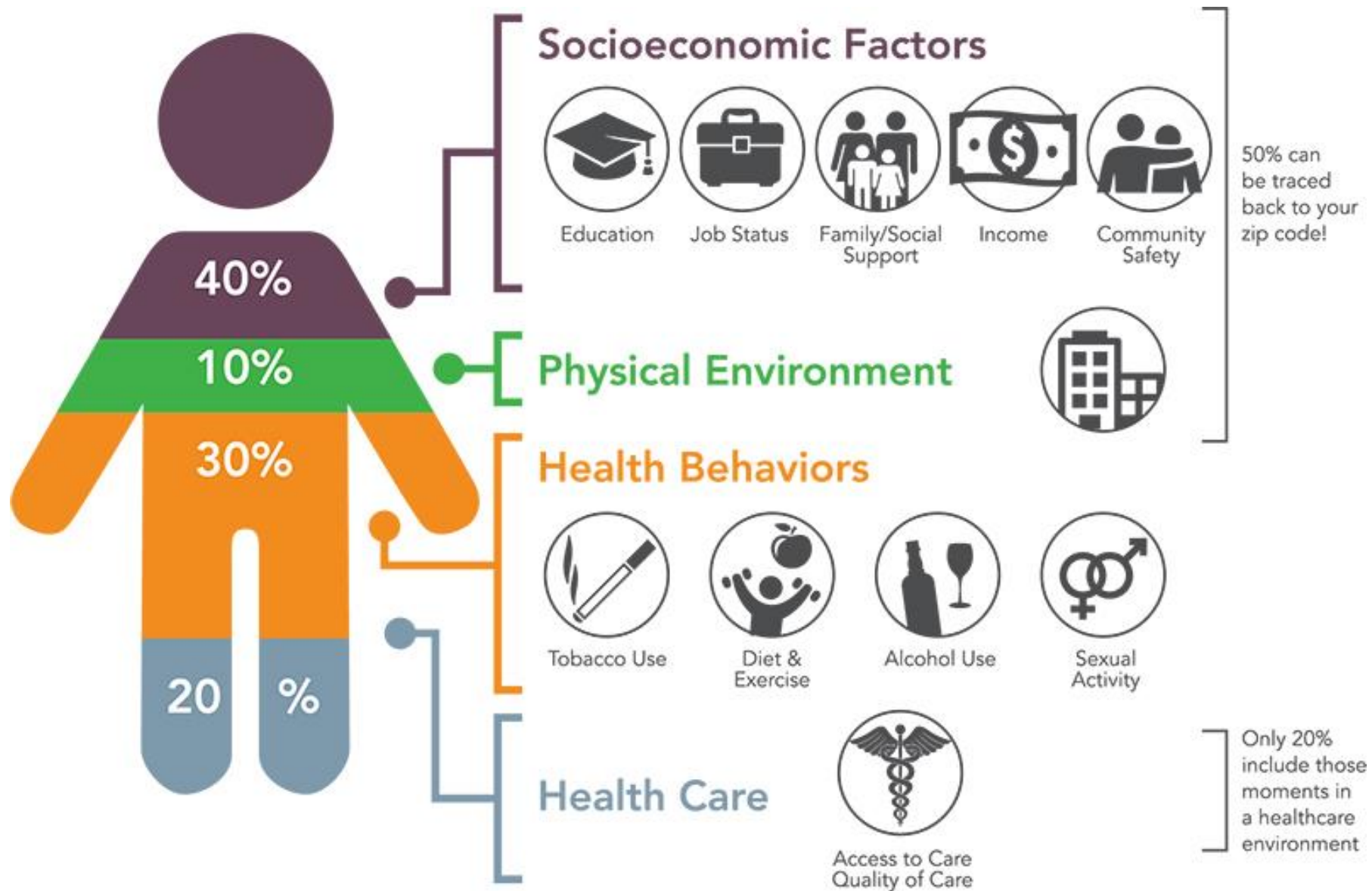
Years from Average Michigan Life Expectancy



Average Michigan life expectancy is 77.7 years (at birth).

Data from National Center for Health Statistics. U.S. Small-Area Life Expectancy Estimates Project (USALEEP): Michigan [2010-2015]. Methodology by Escobedo et al., 2018.

Study Population	Methods	Results					
 <p>NHANES</p> <p>21,664 US adults (10,658 men & 11,006 women)</p> <ul style="list-style-type: none"> - ≥18 years - Non-pregnant 	<p>Cross-sectional analysis</p> <p>Survey-weighted Poisson regression</p> <p>Outcomes</p> <p>Hypertension: BP ≥130/80 or current medication use</p> <p>Stage 2 hypertension: BP ≥140/90</p> <p>Controlled BP</p>	<p>Race/Ethnicity</p> <p>NH Black</p> <p>NH Asian</p> <p>Hispanic</p> <p>No Routine Place for Healthcare</p> <p>Uninsured</p> <p>Unmarried</p> <p>Unemployed</p> <p>Poor</p> <p>Some College</p> <p>≤ High School</p> <p>Foreign-born</p>	          	<p>Hypertension</p>   <p>↑</p> <p>↑</p> <p>↑</p> <p>-</p> <p>↓</p> <p>↓</p> <p>-</p> <p>-</p> <p>↑</p> <p>↑</p> <p>↑</p> <p>↑</p> <p>-</p> <p>-</p>	<p>Stage 2 Hypertension</p>   <p>↑</p> <p>↑</p> <p>↑</p> <p>-</p> <p>-</p> <p>-</p> <p>-</p> <p>↑</p> <p>↑</p> <p>↑</p> <p>↑</p> <p>-</p> <p>-</p>	<p>Controlled BP</p>   <p>↓</p> <p>-</p> <p>-</p> <p>↓</p> <p>↓</p> <p>↑</p> <p>↑</p> <p>-</p> <p>-</p> <p>-</p> <p>-</p>	
<p>Conclusion: SDoH were independently associated with hypertension, Stage 2 hypertension, and controlled BP.</p>							



Life expectancy by county, race, and ethnicity in the USA, 2000–19: a systematic analysis of health disparities



GBD US Health Disparities Collaborators*

Summary

Background There are large and persistent disparities in life expectancy among racial–ethnic groups in the USA, but the extent to which these patterns vary geographically on a local scale is not well understood. This analysis estimated life expectancy for five racial–ethnic groups, in 3110 US counties over 20 years, to describe spatial–temporal variations in life expectancy and disparities between racial–ethnic groups.

Methods We applied novel small-area estimation models to death registration data from the US National Vital Statistics System and population data from the US National Center for Health Statistics to estimate annual sex-specific and age-specific mortality rates stratified by county and racial–ethnic group (non-Latino and non-Hispanic White [White], non-Latino and non-Hispanic Black [Black], non-Latino and non-Hispanic American Indian or Alaska Native [AIAN], non-Latino and non-Hispanic Asian or Pacific Islander [API], and Latino or Hispanic [Latino]) from 2000 to 2019. We adjusted these mortality rates to correct for misreporting of race and ethnicity on death certificates and then constructed abridged life tables to estimate life expectancy at birth.

Findings Between 2000 and 2019, trends in life expectancy differed among racial–ethnic groups and among counties. Nationally, there was an increase in life expectancy for people who were Black (change 3·9 years [95% uncertainty interval 3·8 to 4·0]; life expectancy in 2019 75·3 years [75·2 to 75·4]), API (2·9 years [2·7 to 3·0]; 85·7 years [85·3 to 86·0]), Latino (2·7 years [2·6 to 2·8]; 82·2 years [82·0 to 82·5]), and White (1·7 years [1·6 to 1·7]; 78·9 years [78·9 to 79·0]), but remained the same for the AIAN population (0·0 years [–0·3 to 0·4]; 73·1 years [71·5 to 74·8]). At the national level, the negative difference in life expectancy for the Black population compared with the White population decreased during this period, whereas the negative difference for the AIAN population compared with the White population increased; in both cases, these patterns were widespread among counties. The positive difference in life expectancy for the API and Latino populations compared with the White population increased at the national level from 2000 to 2019; however, this difference declined in a sizeable minority of counties (615 [42·0%] of 1465 counties) for the Latino population and in most counties (401 [60·2%] of 666 counties) for the API population. For all racial–ethnic groups, improvements in life expectancy were more widespread across counties and larger from 2000 to 2010 than from 2010 to 2019.

Interpretation Disparities in life expectancy among racial–ethnic groups are widespread and enduring. Local-level data are crucial to address the root causes of poor health and early death among disadvantaged groups in the USA, eliminate health disparities, and increase longevity for all.

Funding National Institute on Minority Health and Health Disparities; National Heart, Lung, and Blood Institute; National Cancer Institute; National Institute on Aging; National Institute of Arthritis and Musculoskeletal and Skin Diseases; Office of Disease Prevention; and Office of Behavioral and Social Science Research, US National Institutes of Health.

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*Collaborators are listed at the end of the Article

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Detroit is Among the Most Disadvantaged

	Detroit	National Average
Children in poverty (%)	52.2	20.4
Income inequity score	-39.6	-1.1
Racial segregation score	40.3	10.9
Unemployment (%)	18.6	6.8
3rd Grade reading proficiency (%)	19.2	46.2
Violent Crime (per 100,000)	1900.4	436.1
Air pollution (PM2.5)	9.7	8.5
Housing w/ Lead Risk (%)	44.2	17.6
Limited access to healthy food (%)	48.3	63.9
Smoking (% adults)	28.9	16.7
Physical inactivity (%)	37.6	23.9
Obesity (%)	43.6	30.4

The Population Health Outcomes and Information Exchange (PHOENIX) Program - A Transformative Approach to Reduce the Burden of Chronic Disease

Steven J. Korzeniewski^{1*}, Carla Bezold², Jason T. Carbone¹, Shooshan Danagouljian¹, Bethany Foster¹, Dawn Misra¹, Maher M. El-Masri¹, Dongxiao Zhu¹, Robert Welch¹, Lauren Meloche¹, Alex B. Hill¹, Phillip Levy¹

¹Wayne State University, ²Detroit Health Department

ABSTRACT

This concept article introduces a transformative vision to reduce the population burden of chronic disease by focusing on data integration, analytics, implementation and community engagement. Known as PHOENIX (The Population Health Outcomes and Information Exchange), the approach leverages a state level health information exchange and multiple other resources to facilitate the integration of clinical and social determinants of health data with a goal of achieving true population health monitoring and management. After reviewing historical context, we describe how multilevel and multimodal data can be used to facilitate core public health services, before discussing the controversies and challenges that lie ahead.

Keywords: Health information exchange; data integration; epidemiology; electronic health record; translational science; social determinants of health.

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DOI: 10.5210/ojphi.v12i1.10456

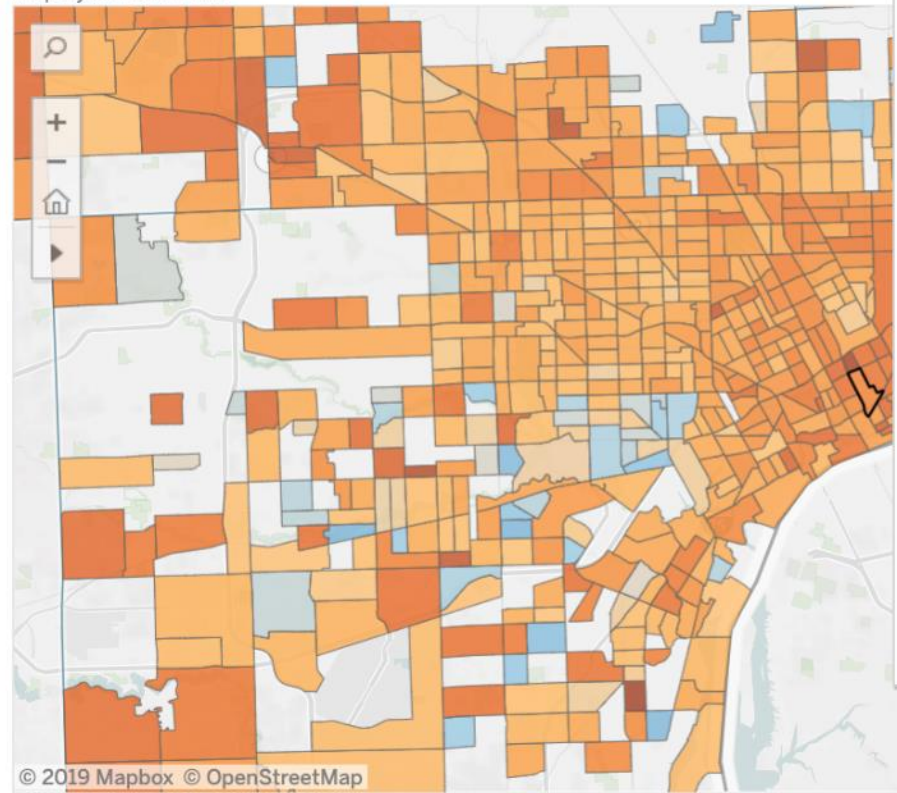
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Hypertension Dashboard Metro-Detroit (Way

Select a Vital Category to Explore:

Map by Census Tract

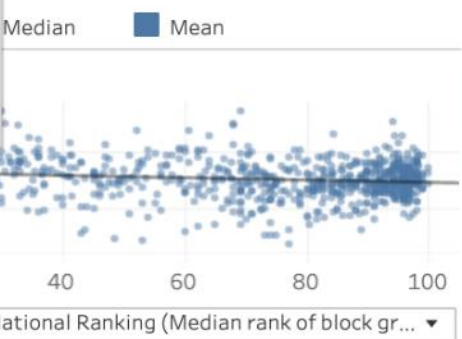
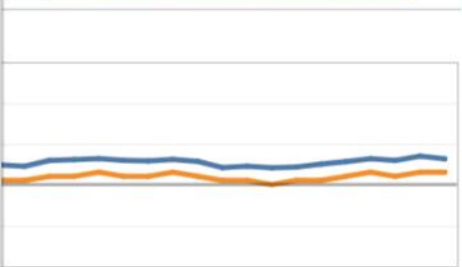
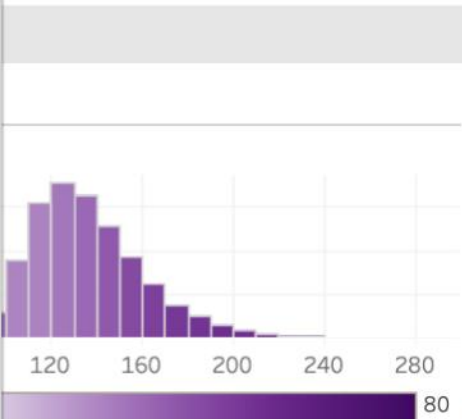


Set map legend midpoint as: (Midpoint: 130)

Tract ID: 26163518900
 Number of Patient Records: 4,493
 Mean SBP: 137.22
 Mean DBP: 83.01
 Mean HR: 88.64
 Median SBP: 133.00
 Median DBP: 81.00
 Median HR: 88.00

Census Tract Information

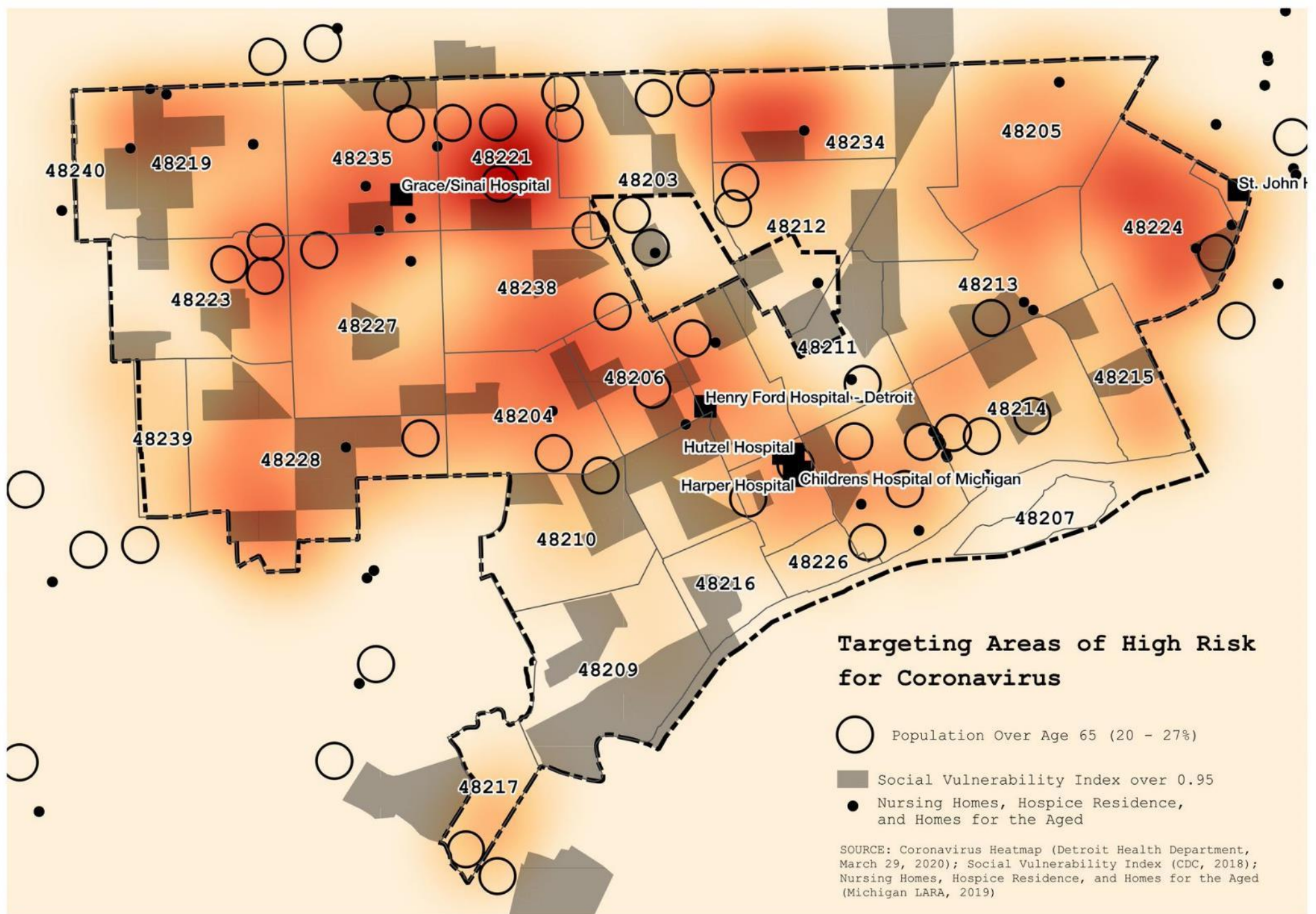
Population: 2,122
 Housing Units: 953
 Occupied Housing Units (Households): 843
 Median Age: 29.30
 Population Age 65+: 15.08%
 Median Household Income: \$13,764
 Families Below Poverty Level: 60.9%
 Unemployment Rate: 35.30%
 Uninsured Population: 12.40%
 Life Expectancy at Birth: 72.10 years
 Non-White Population: 96.09%
 Renter-Occupied Housing Units: 99.29%
 Unoccupied Housing Units: 11.54%
 ADI National Ranking (median of block groups within tract): 100.00
 ADI State Ranking (median of block groups within tract): 10.000



Filter Data:

- Select County:
- Select City:
- Date of Arrival: (All)
- Gender:
- Age at Time of Visit: to
- Race/Ethnicity:
 - (All)
 - Black/African American
 - Other Race or More than On..
 - Spanish/Hispanic
 - Unknown
 - White

Race/Ethnicity	Number of Records	Mean SBP	Mean DBP	Mean HR	Mean Age	Mean Temp	% of Total
Black/African American	422,654	135.20	81.98	89.00	42	36.76	76.61%
White	82,775	135.72	80.33	90.02	46	36.72	15.00%
Other Race or More than On..	31,395	136.51	81.22	87.79	48	36.73	5.69%
Unknown	8,950	134.57	80.85	90.05	42	36.77	1.62%
Spanish/Hispanic	5,916	134.17	81.47	89.49	42	36.72	1.07%
Grand Total	551,690	135.33	81.67	89.11	43	36.75	100.00%





COVID-19 Testing for Anyone Draws a Crowd in Hard-Hit Detroit

By Reuters

April 28, 2020



DETROIT — Detroit residents waited for hours on Tuesday to get free COVID-19 tests at a new facility that for the first time offered testing to people who did not already have symptoms of the disease and a doctor's authorization for the test.

"I don't want to take a chance," said Cheryl Albright, a 58-year-old Detroit woman with hypertension and chronic obstructive pulmonary disease (COPD), conditions she knows put her at higher risk. Albright said she has family members who have tested positive for COVID-19, the respiratory illness caused by the new coronavirus.

She lined up at 10 a.m. ET (1400 GMT) for a test, and was still in the line with about 100 others more than two hours later.

"This is important to me. I think this is something everyone should do," she said.

Tuesday's tests were the start of a free program for Detroit residents, said Dr. Phillip Levy, professor of emergency medicine at Wayne State University. Even those without symptoms can get tested with a nasal swab for the virus as well as have their blood drawn to test for antibodies.



Michigan Coronavirus Racial Disparities Task Force

Recommendations for Collaborative Policy, Programming and Systemic Change





HEALTH

Community-informed strategies improved Detroit's COVID response

By NAIMA © January 27, 2022



Wayne Health Mobile Unit

Patient Visits

89,518

Unique Patients

60,968

Covid Tests

53,502

Negative Results

49,083

Positive Results

4,419

Covid Vaccines

15,235

First Dose

7,023

Second Dose

5,421

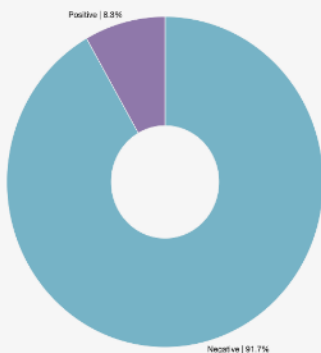
Third Dose/Booster

2,684

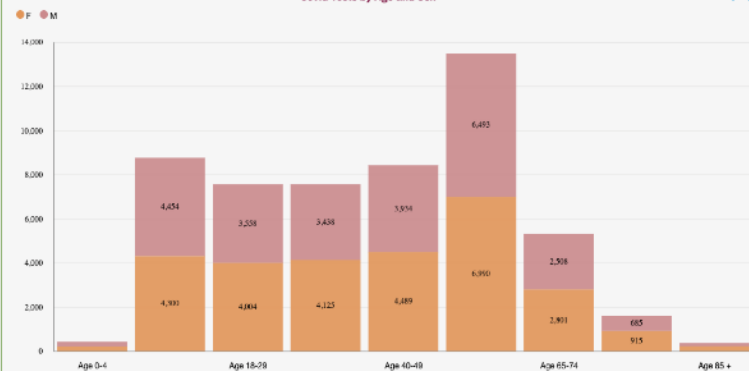
Patients Seen by Month



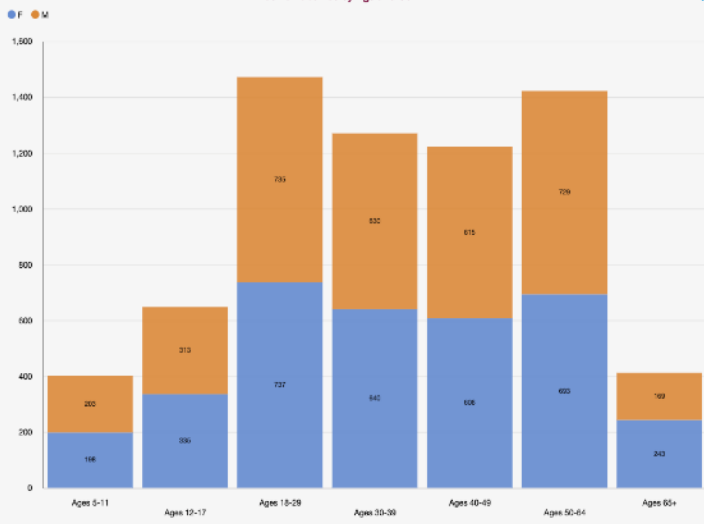
Covid Test Results %



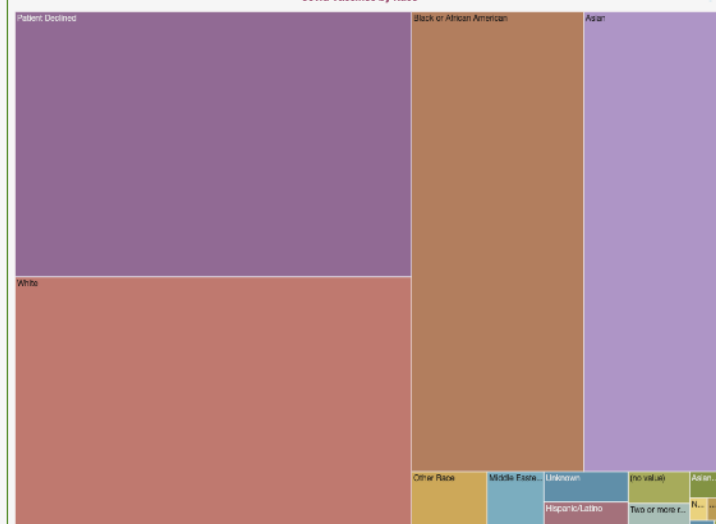
Covid Tests by Age and Sex



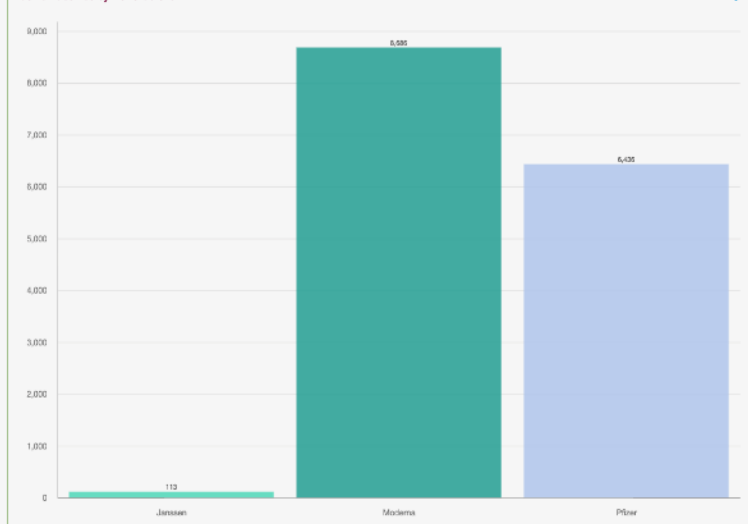
Covid Vaccines by Age and Sex



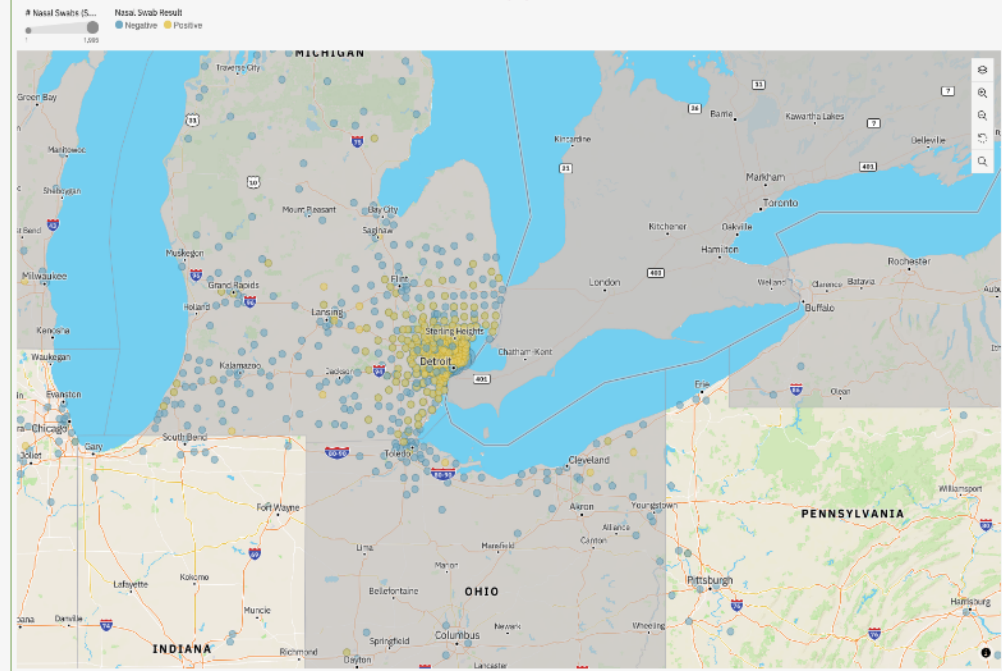
Covid Vaccines by Race



Covid Vaccines by Manufacturer



Covid Tests by Zip Code





WAYNE
HEALTH
MOBILE
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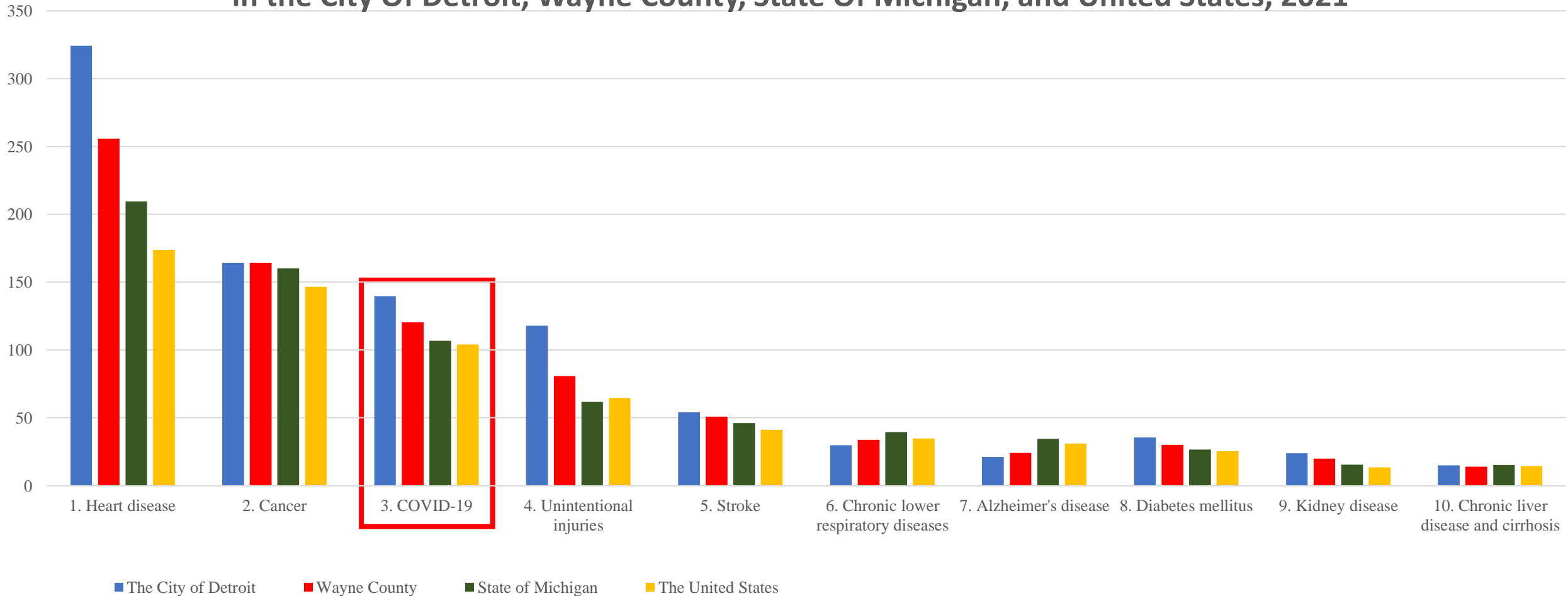
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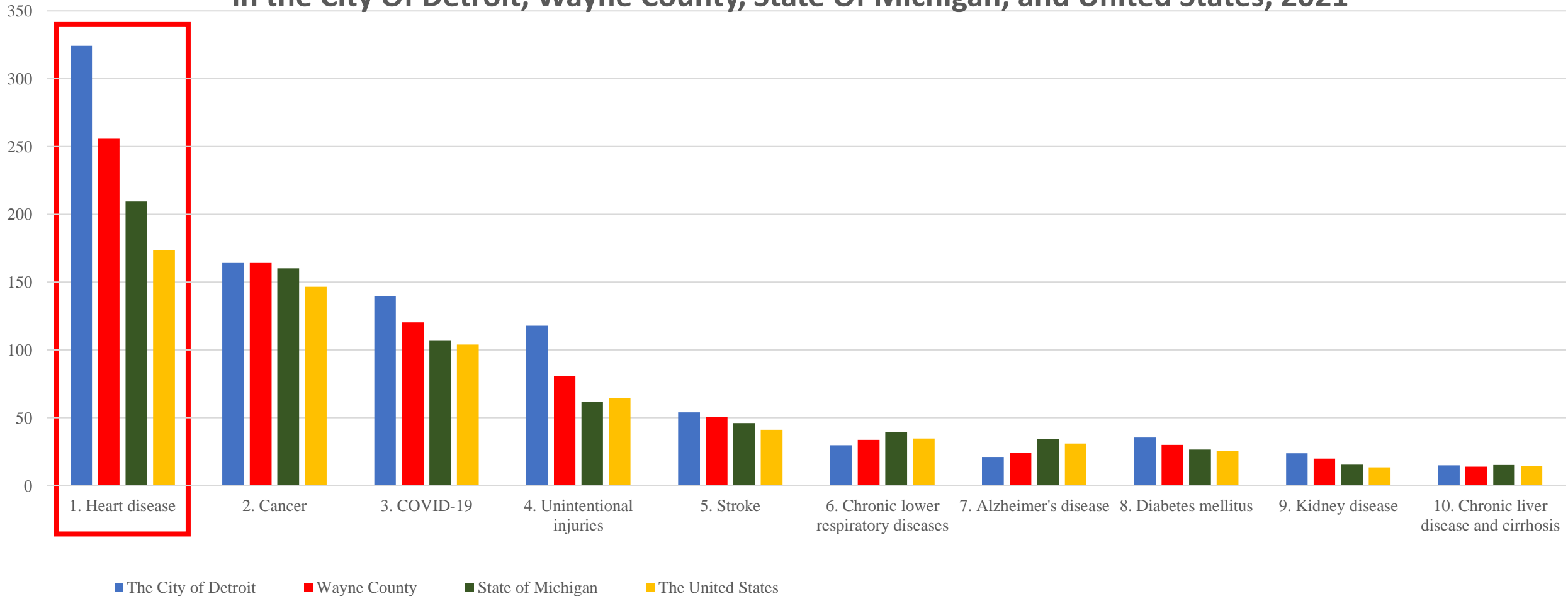


Age-adjusted Mortality Rates/100,000 Population for the Ten Leading Causes Of Death in the City Of Detroit, Wayne County, State Of Michigan, and United States, 2021



Source: 2021 Geocoded Michigan Death Certificate Registry. Division for Vital Records & Health Statistics, Michigan Department of Health & Human Services. National Center for Health Statistics. NCHS Data Brief No. 456, 2022.

Age-adjusted Mortality Rates/100,000 Population for the Ten Leading Causes Of Death in the City Of Detroit, Wayne County, State Of Michigan, and United States, 2021



Source: 2021 Geocoded Michigan Death Certificate Registry. Division for Vital Records & Health Statistics, Michigan Department of Health & Human Services. National Center for Health Statistics. NCHS Data Brief No. 456, 2022.

Funding: Funding was supplied by donors and non-profit organizations including United Way for Southeastern Michigan, the Community Foundation of Southeast Michigan/Detroit Medical Center Foundation, the Ralph C. Wilson Foundation, Community Organized Relief Effort (CORE), DTE Energy Foundation, Blue Cross Blue Shield of Michigan, and the Cielo Foundation. Michigan Department of Health and Human Services (MDHHS) also collaborated and contributed funding to support further growth and extension of services. A CDC funded program (1817) with the MDHHS Heart Disease and Stroke Prevention Unit allowed for cardiometabolic risk factor screening. In addition, funding for the PHOENIX program was provided by the Michigan Health Endowment Fund and Delta Dental Michigan.

RESEARCH ARTICLE

From pandemic response to portable population health: A formative evaluation of the Detroit mobile health unit program

Phillip Levy¹, Erin McGlynn^{1*}, Alex B. Hill¹, Liying Zhang², Steven J. Korzeniewski², Bethany Foster¹, Jasmine Criswell³, Caitlin O'Brien³, Katee Dawood³, Lauren Baird³, Charles J. Shanley⁴

1 Department of Emergency Medicine, Wayne State University School of Medicine, Detroit, Michigan, United States of America, **2** Department of Family Medicine and Public Health Sciences, Wayne State University School of Medicine, Detroit, Michigan, United States of America, **3** Wayne Health, Wayne State University, Detroit, Michigan, United States of America, **4** Department of Surgery, Wayne State University School of Medicine, Detroit, Michigan, United States of America

* ekmcglynn@wayne.edu



Portable Population Health

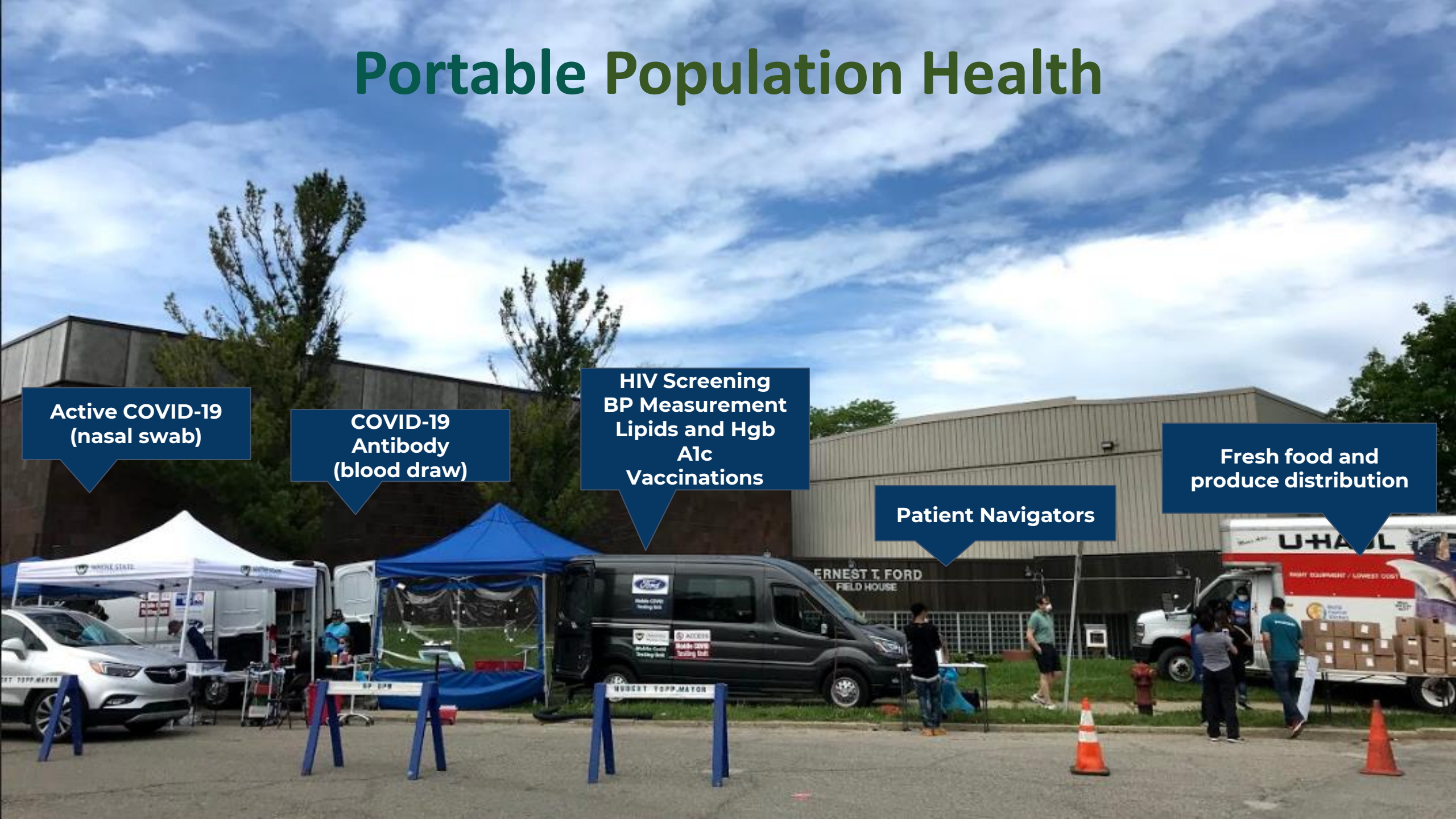
Active COVID-19
(nasal swab)

COVID-19
Antibody
(blood draw)

HIV Screening
BP Measurement
Lipids and Hgb
A1c
Vaccinations

Patient Navigators

Fresh food and
produce distribution



RESEARCH LETTER

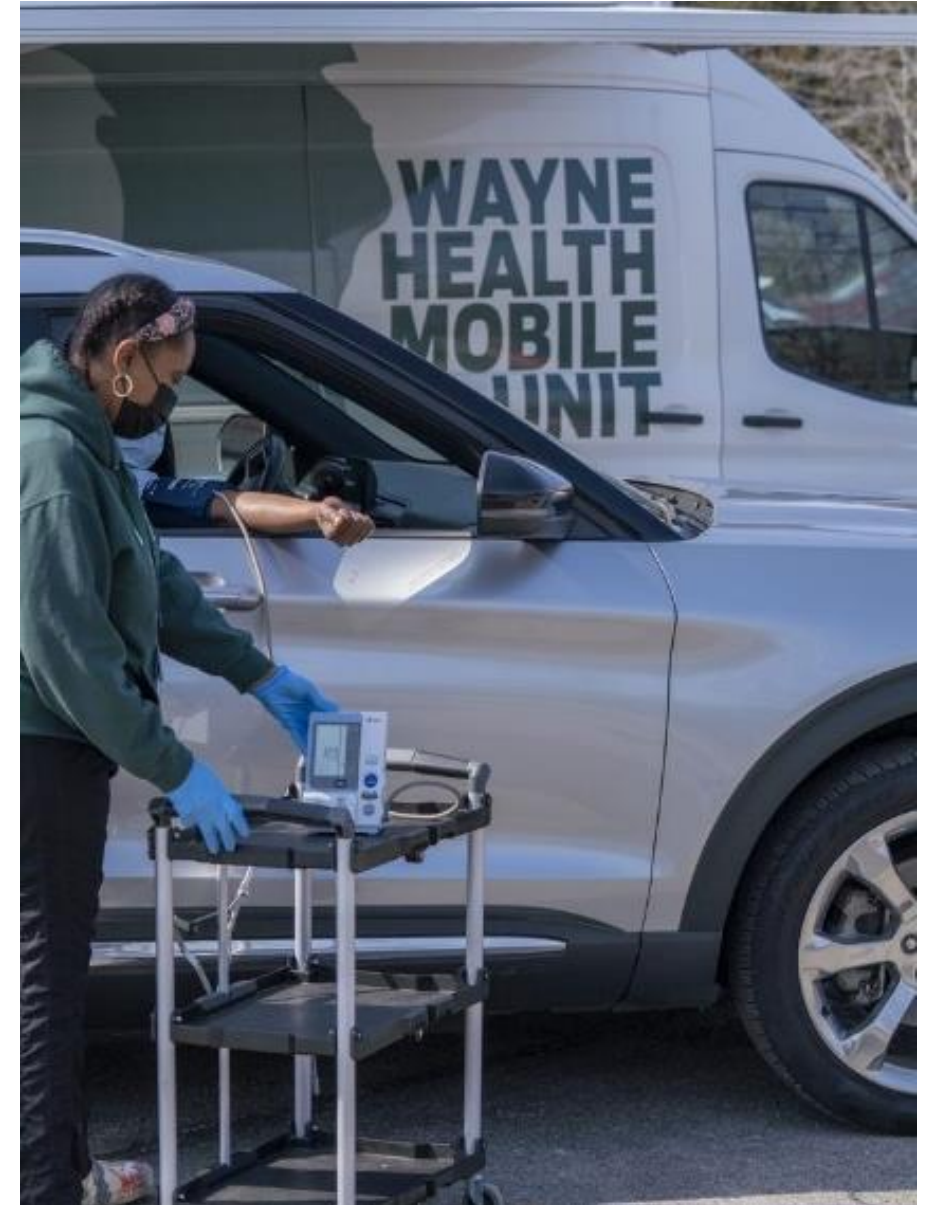
Utilizing Mobile Health Units for Mass Hypertension Screening in Socially Vulnerable Communities Across Detroit

Robert D. Brook¹, Katee Dawood, Bethany Foster, Randi M. Foust, Catherine Gaughan, Paul Kurian, Brian Reed, Andrea L. Jones, Barbara Vernon², Phillip D. Levy³

Nearly half of all adults in the United States have hypertension, defined as a blood pressure (BP) $\geq 130/80$ mmHg. However, both the prevalence (56%) and control rates (18%) are worse in Black patients.¹ Numerous social determinants of health in socially vulnerable populations further exacerbate these disparities while reducing hypertension awareness and access to health care.² Few places exemplify this crisis like the city of Detroit (78% Black race) where hypertension rates are the highest in Michigan (<https://www.cdc.gov/places>) and all census tracts are in health professional shortage areas (<https://data.hrsa.gov/tools/shortage-area/>). As such, the public health importance of large-scale screening efforts to identify the enormous number of individuals with hypertension cannot be overstated.³ We here describe the first-year results using our novel Wayne Health Mobile Unit program developed in

Given the large population serviced (while also ensuring resiliency of the program during cold weather and COVID restrictions), we developed a high-throughput method to offer screening for high BP (defined as $\geq 120/80$ mmHg) beginning in November 2020. Those driving to a site ($\approx 90\%$) rested inside their parked car for ≥ 5 minutes. BP was then measured using an Omron 907XL monitor following a guideline-consistent protocol—up to an average of triplicate upper arm readings (1-minute intervals) using a correct cuff size with the arm supported at heart level (door armrest) and feet resting on the car floor. A minority ($< 10\%$) of walk-up patients had seated BP measured in MHU canopy rooms. As privacy was limited, BP measurements were attended and cuffs were placed over long-sleeves when relevant.

All patients are provided follow-up care in the Wayne Health system per individual needs/wishes. Health information, including prior hypertension status, is collected but not currently available for the entire cohort. Individuals with a



Categories	Number (%)	BP* (mm Hg)
All patients	3,039	126.9 ± 23.1 / 76.8 ± 14.7
Normal BP Systolic BP <120 and diastolic BP <80 mm Hg	1136 (37%)	105.5 ± 9.28 / 65.0 ± 8.34
High BP Categories**		
Elevated BP Systolic BP 120-129 and diastolic BP <80 mm Hg	306 (10%)	124.2 ± 2.8 / 70.1 ± 6.44
Hypertension categories*** Systolic BP ≥130 and/or diastolic BP ≥80 mm Hg	1597 (53%)	142.7 ± 19.39 / 86.4 ± 12.43
Stage I Systolic BP 130-139 and/or diastolic BP 80-89 mm Hg	629 (21%)	127.7 ± 8.73 / 80.3 ± 6.84
Stage II Systolic BP ≥140 and/or diastolic BP ≥90 mm Hg	968 (32%)	152.4 ± 18.15 / 90.4 ± 13.6

The PHOENIX Program at Wayne State University

the Population Health OutcomEs aNd Information eXchange program

Mission

*CONNECTING COMMUNITIES WITH DATA FOR POSITIVE
SOCIAL CHANGES IN PURSUIT OF LIFESPAN EQUALITY*

Our team informs public health action through targeted analytics, storytelling and commentary about the constellation of factors that drive local health disparities



We help with data to help others

Our team ingests, transforms, merges, analyzes and shares multi-level health data



Access management

Plan and implement who can access what



Ingestion and storage

Move data into the cloud



Standardize and transform

Adopt standards to foster data sharing and team science



Integration and analytics

Describe patterns, assess trends and answer questions



Sharing and dissemination

Visualize and report health information to stakeholders

Source	Data Ingested	Clinical		Social		Built Env.		Natural Env.		Policy Env.		Index		Region		State		County		ZIP		Tract		Block	
Brookings Institute	Metro Recovery Index									X	X			X											
US Centers for Disease Control & Prevention	500 Cities & PLACES		X														X	X	X						
	Atlas of Heart Disease and Stroke	X															X								
	COVID-19 Surveillance	X													X	X									
	Drug Overdose Mortality	X													X										
	Drug Overdose Surveillance and Epidemiology (DOSE): Non-Fatal Overdose	X													X										
	Hospital Capacity	X		X												X									
	Life Expectancy	X																			X				
	Modified Retail Food Environment Index		X	X																		X			
	Monkeypox Surveillance	X													X										
	National Environmental Public Health Tracking Network			X	X												X				X				
	National Health Interview Survey	X	X											X											
	Social Vulnerability Index	X	X	X						X						X					X				
	State Unintentional Drug Overdose Reporting System (SUDOR): Fatal Overdose	X	X													X									
	Tick Surveillance				X												X								
	US Chronic Disease Indicators	X													X										
	US Chronic Disease Indicators: Cardiovascular Disease	X													X										
	US Chronic Disease Indicators: Diabetes	X													X										
US Chronic Disease Indicators: Reproductive Health	X													X											
WONDER: Natality	X														X										
City of Detroit Open Data Portal	COVID-19 Surveillance	X																X							
	Parcels			X												X	X	X							
Columbia University	Drinking Water Contaminants			X												X									
Electronic Health Records	Detroit Medical Center - Emergency Department Surveillance	X														X	X	X							
	Henry Ford Health- Emergency Department Surveillance	X														X	X	X							
Diversity Data Kids	Child Opportunity Index								X									X	X						
Environmental Protection Agency	Environmental Justice Screening		X	X	X															X					
	Facility Registry Service			X																		X			
	Walkability Index								X													X			
Federal Housing Finance Agency	High Opportunity Areas		X	X					X											X					
	Underserved Areas		X	X					X											X					
Global Burden of Disease	Police Violence US Subnational Collaborators		X										X												
Google	Mobility Reports			X					X		X		X		X				X						
Gun Violence Archive	Gun Violence		X	X												X				X					
Harvard Database	Presidential Election Returns							X								X									
Health Resources & Services Administration	Health Professional Shortage Areas	X		X												X				X					
	Medically Underserved Areas	X		X												X				X					

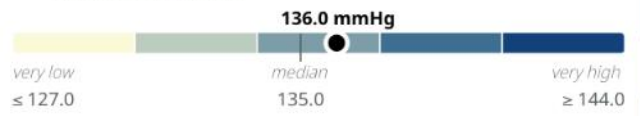
METRICS

browse: [vitals](#) **medical** [social](#) [natural](#) [built](#)

selected geography:
Tract 521900, Wayne County MI
3.1K residents (estimated total population)

Systolic Blood Pressure

Emergency department median
DEC 2018 - MAR 2023



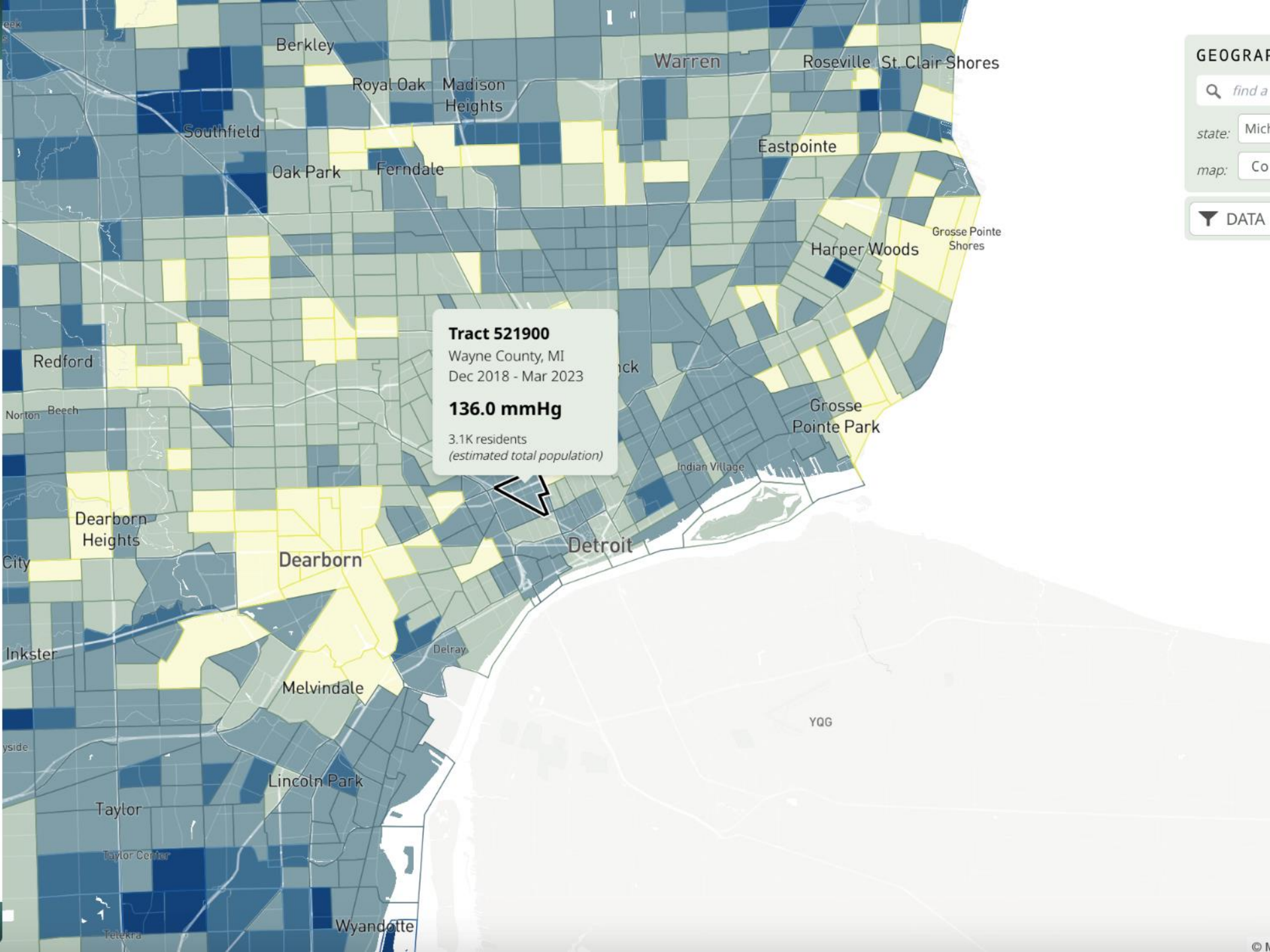
advanced display: Related Metrics

time period: All Data Last 12 Months

click on a related metric to view it on the map



[view trends and determinants summary >>](#)



Tract 521900
Wayne County, MI
Dec 2018 - Mar 2023

136.0 mmHg
3.1K residents
(estimated total population)

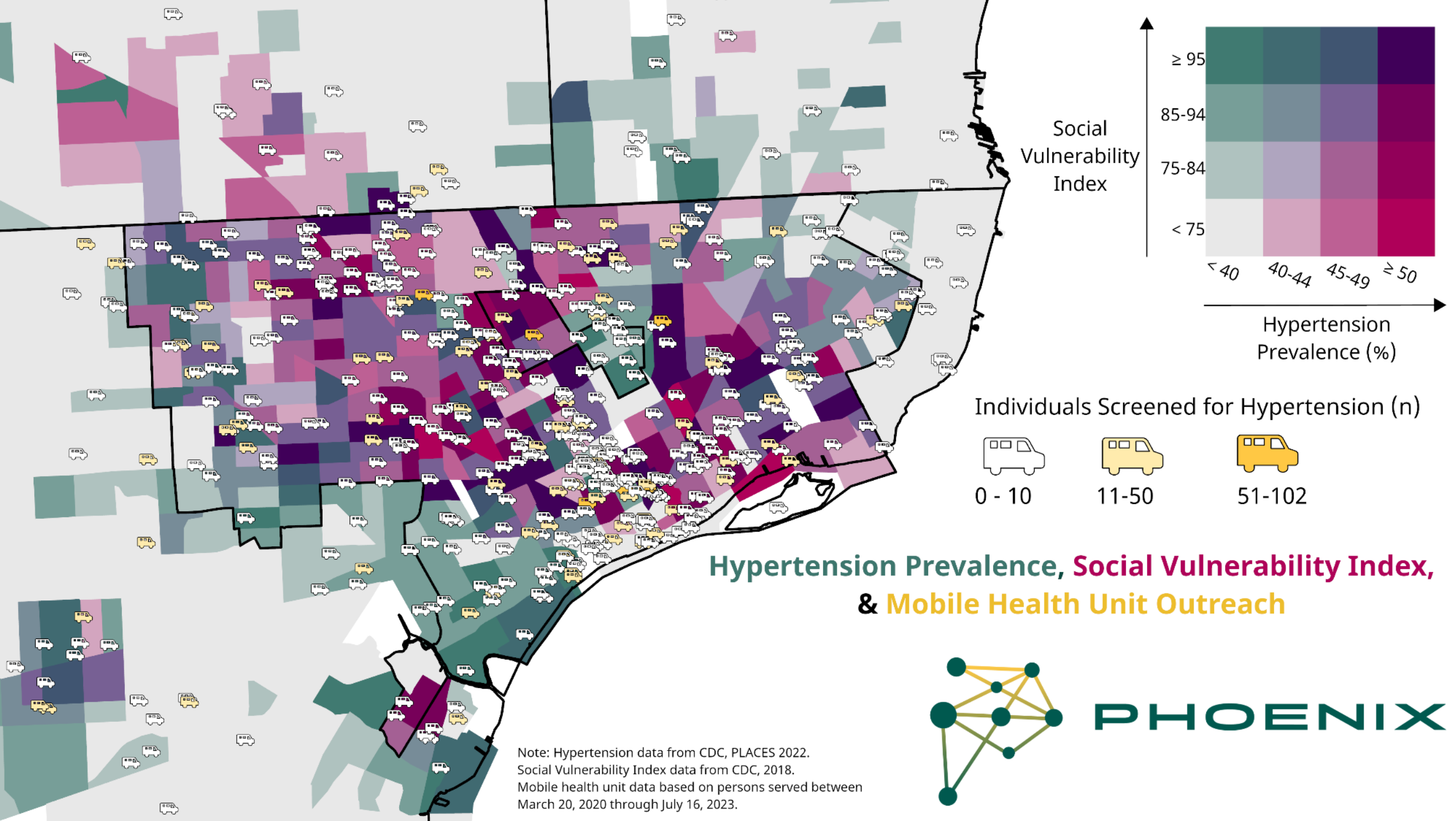
GEOGRAPHY

find a

state: Michigan

map: County

DATA





Wayne Health Mobile Unit

BP Screenings

7,371

Screening Labs

14,940

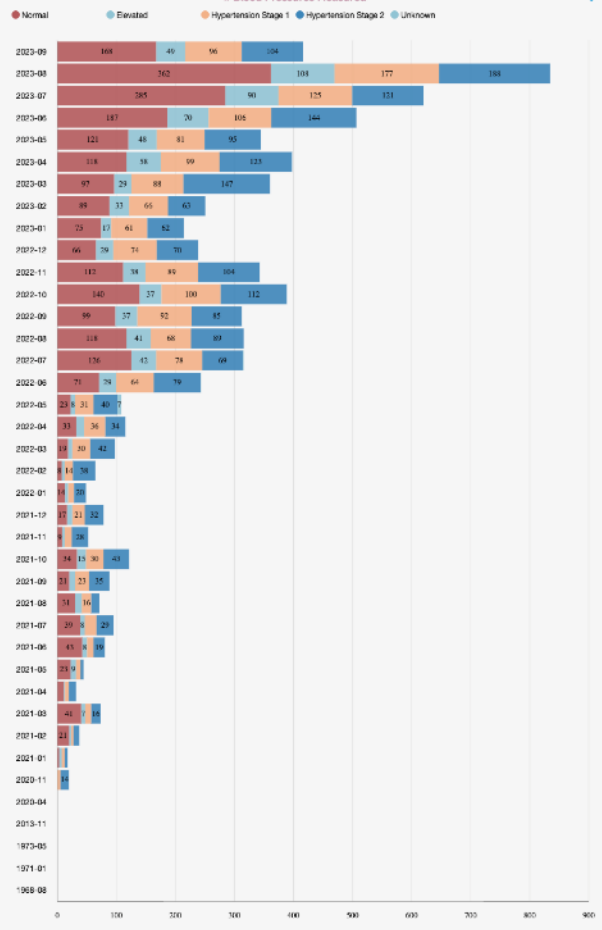
Referred to PCP

391

Referred to Specialist

146

Blood Pressures Measured



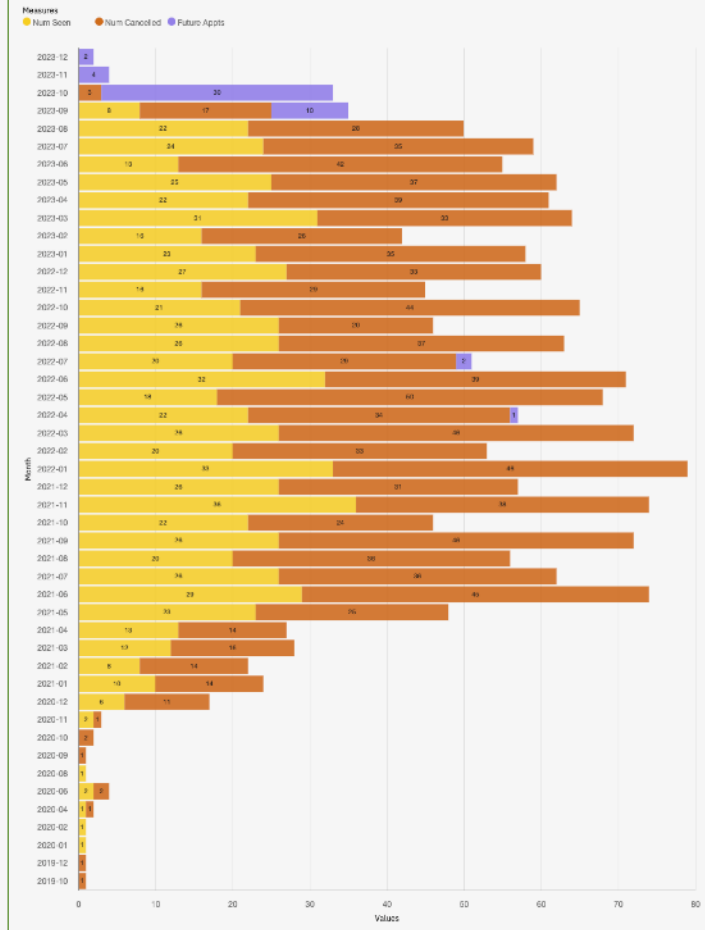
Screening Labs Ordered

Num_Labs_Ordered	2023-09	2023-08	2023-07	2023-06	2023-05	2023-04	2023-03	2023-02	2023-01	2022-12	2022-11	2022-10	2021-10
ALBUMIN/CREATININE, MASS RATIO, URIN...	0	0	6	2	1	0	0	0	0	0	0	0	0
BMP, SERUM OR PLASMA	0	0	0	0	0	0	0	0	0	0	0	0	0
C-REACTIVE PROTEIN, QUANTITATIVE	0	0	0	0	0	0	0	0	0	0	1	0	0
CBC W/ ALTO DIFF	0	0	0	0	0	0	1	0	0	0	0	0	0
CBC W/ DIFF	0	0	0	0	0	0	0	1	0	0	0	0	0
CD4 T-CELLS, BLOOD	1	0	0	0	0	0	0	0	0	0	0	0	0
CMP, SERUM OR PLASMA	260	479	340	234	209	213	156	116	101	69	104	207	207
GLUCOSE, SERUM OR PLASMA	0	0	0	0	0	0	0	0	0	0	0	0	0
HBA1C (HEMOGLOBIN A1C), BLOOD	298	479	333	231	201	211	134	115	101	69	103	204	204
HEMOGLOBIN (Hb), BLOOD	0	0	0	0	0	0	0	0	0	0	0	0	0
HEPATITIS C 10G AB, QUAL, SERUM	1	0	0	0	0	0	0	0	0	0	0	0	0
HIV 1 + 2, MEANINGFUL USE SET	1	0	0	0	0	0	0	0	0	0	0	0	0
HIV-1 RNA, QUANTITATIVE, PCR, SERUM...	1	0	0	0	0	0	0	0	0	0	0	0	0
LEAD, BLOOD	0	0	0	0	0	0	0	0	0	0	0	0	0
LEAD, QUANT, VENOUS BLOOD	1	7	4	2	3	0	1	1	0	0	0	0	0
LYPD PANEL, SERUM	259	476	339	232	201	211	159	116	101	69	103	206	206
PHO BNP (PHO B-TYPE NATRIURETIC PEPT...	7	20	14	6	7	8	7	6	2	0	0	0	0
TSH, SERUM OR PLASMA	0	0	0	0	0	0	0	1	0	0	0	0	0
TNI ISTD I AS	0	0	0	1	0	0	0	0	0	0	0	0	0
VITAMIN B12, SERUM	0	0	0	0	0	0	1	0	0	0	0	0	0
VITAMIN D, 25-HYDROXY, TOTAL, SERUM	0	0	0	0	0	0	1	0	0	0	0	0	0
Summary	789	1,457	1,036	708	618	643	476	356	305	208	310	617	617

Lab Results

	2021-08			2021-07			2021-06			2021-05			Summary		
	num	% Abnormal	Num Results	Num Abnormal	% Abnormal	Num Results	Num Abnormal	% Abnormal	Num Results	Num Abnormal	% Abnormal	Num Results	Num Abnormal	% Abnormal	
CHOLESTEROL, TOTAL	90	35%	152	51	34%	19	0	0%	69	3	4%	4,606	1,536	33%	
CREATININE	21	8%	151	16	11%	20	1	5%	69	6	9%	4,576	358	8%	
EGFR	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	(no value)	2,999	1,281	43%	
EGFR AFRICAN AMERICAN	134	53%	146	68	47%	20	10	50%	69	35	51%	1,559	829	53%	
EGFR NON-AFR. AMERICAN	76	30%	146	37	25%	20	5	25%	69	21	30%	1,559	484	31%	
HEMOGLOBIN A1C	29	11%	152	10	7%	20	1	5%	70	7	10%	4,520	582	13%	
Summary	350	28%	747	182	24%	99	17	17%	346	72	21%	19,819	5,070	26%	

Referred to PCP, Appointment Statuses



METRICS

browse: [vitals](#) **medical** [social](#) [natural](#) [built](#)

selected geography:
Tract 521900, Wayne County MI
3.1K residents (estimated total population)

Systolic Blood Pressure

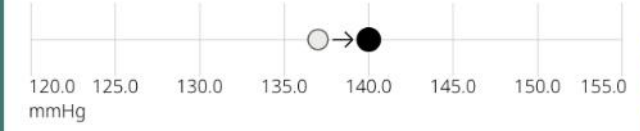
Emergency department median
APR 2022 - MAR 2023



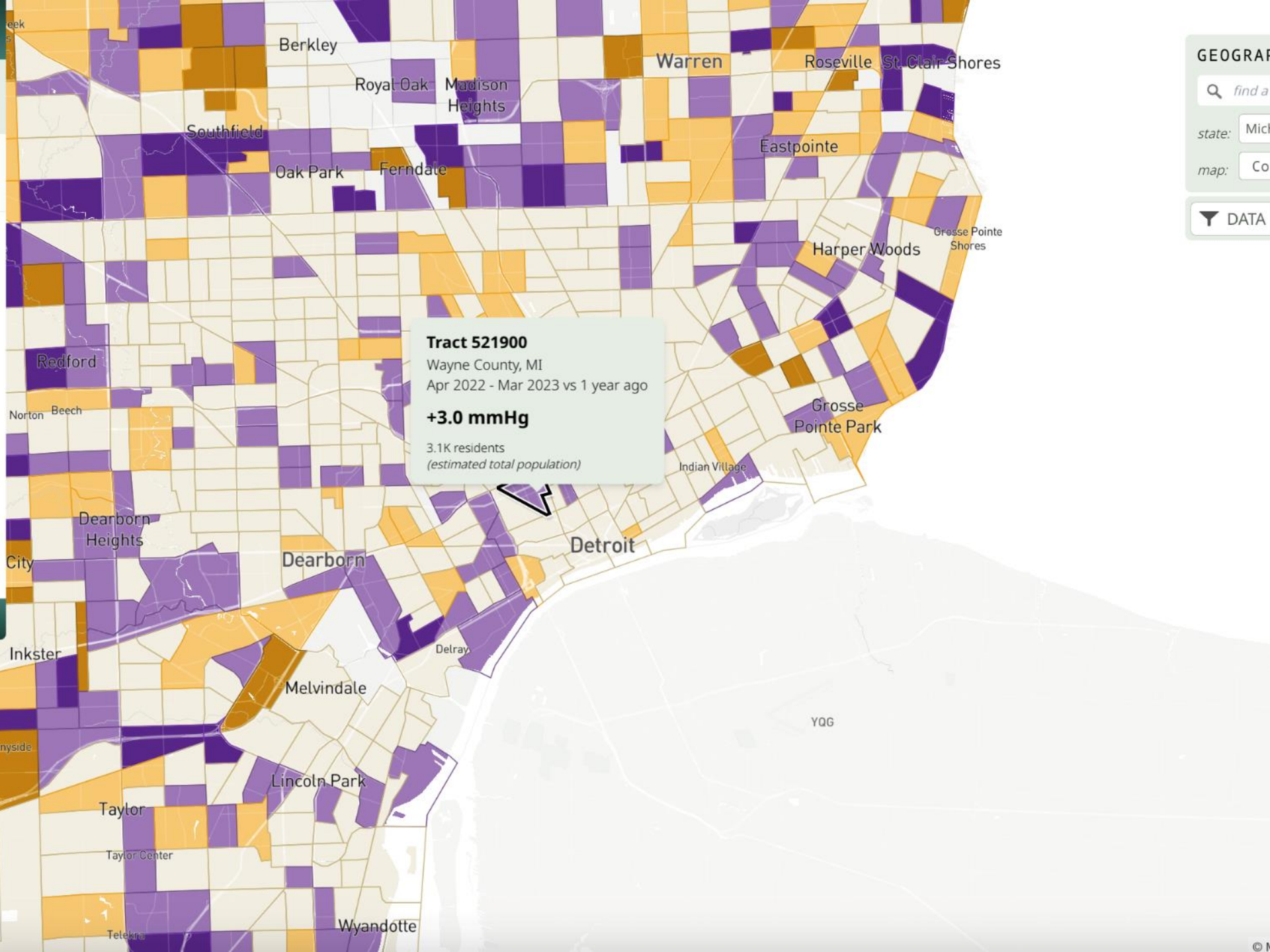
advanced display: Time Period Comparison

time period: All Data Last 12 Months

compare time period against: **1 year ago** 2 years ago



[view trends and determinants summary >>](#)



GEOGRAPHY

find a

state: Mich


map: Co

DATA

CARDIOVASCULAR PERSPECTIVE

Cardiometabolic Risk Factor Control During Times of Crises and Beyond

The world is currently suffering through one the greatest crises of the last century. The coronavirus disease 2019 (COVID-19) pandemic is taking an enormous toll on public health and stretching medical resources in an unprecedented fashion. Our priorities are rightly focusing on meeting this existential threat. Nonetheless, we wish to call to attention that during major catastrophes the health consequences of chronic diseases, in particular cardiometabolic risk factors (CMRFs), continue unabated. In fact, new and serious problems arise part-and-parcel with the catastrophe and conspire to hamper our already imperfect ability to control CMRFs.^{1,2} Our objective is to raise awareness that we need to anticipate (and not just be reactive to) the possible coming of a second crisis we term disastrous CMRFs. This refers to the worsening of CMRFs and their control rates during and following a major disaster.^{1,2} Health care providers, in particular cardiologists, need to recognize the potential for this serious problem as it could promote a burgeoning of cardiovascular morbidity and mortality if not addressed. The COVID-19 pandemic should also serve as a wake-up call to the antiquated flaws in our healthcare model that collude to undermine the successful management of CMRFs in general.³ This current crisis can be a catalyst for optimizing practices and creating critical new capacities that will be beneficial moving forward and serve as a bulwark against future crises.

Robert D. Brook , MD
Phillip Levy, MD, MPH
Sanjay Rajagopalan, MD

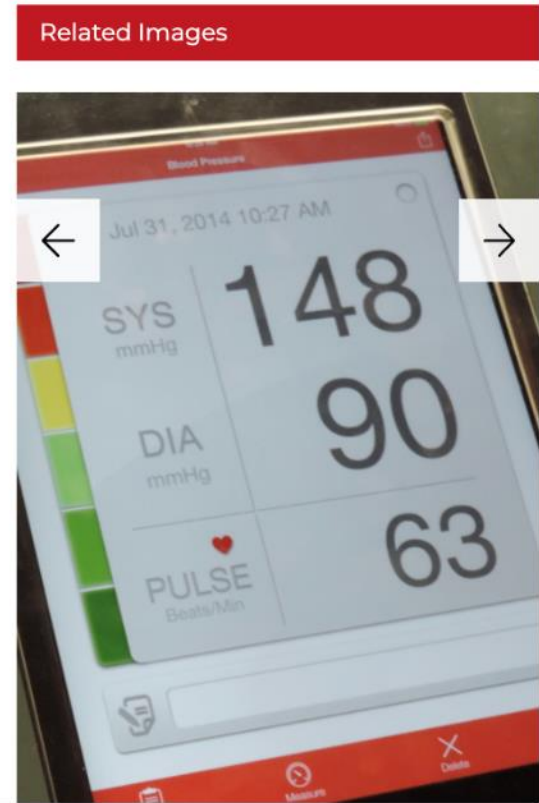


Newsroom / Search News Releases / \$20M awarded for scientific research to ensure health equity in preventing hypertension

Categories: [Program News](#) | Published: July 29, 2021

\$20M awarded for scientific research to ensure health equity in preventing hypertension

Teams from Beth Israel Deaconess Medical Center, Johns Hopkins University School of Nursing, NYU Grossman School of Medicine, University of Alabama at Birmingham and Wayne State University receive American Heart Association research grants to study high blood pressure prevention in underrepresented populations



Wayne State wins \$18 million from National Institutes of Health to intercept chronic disease in Black communities

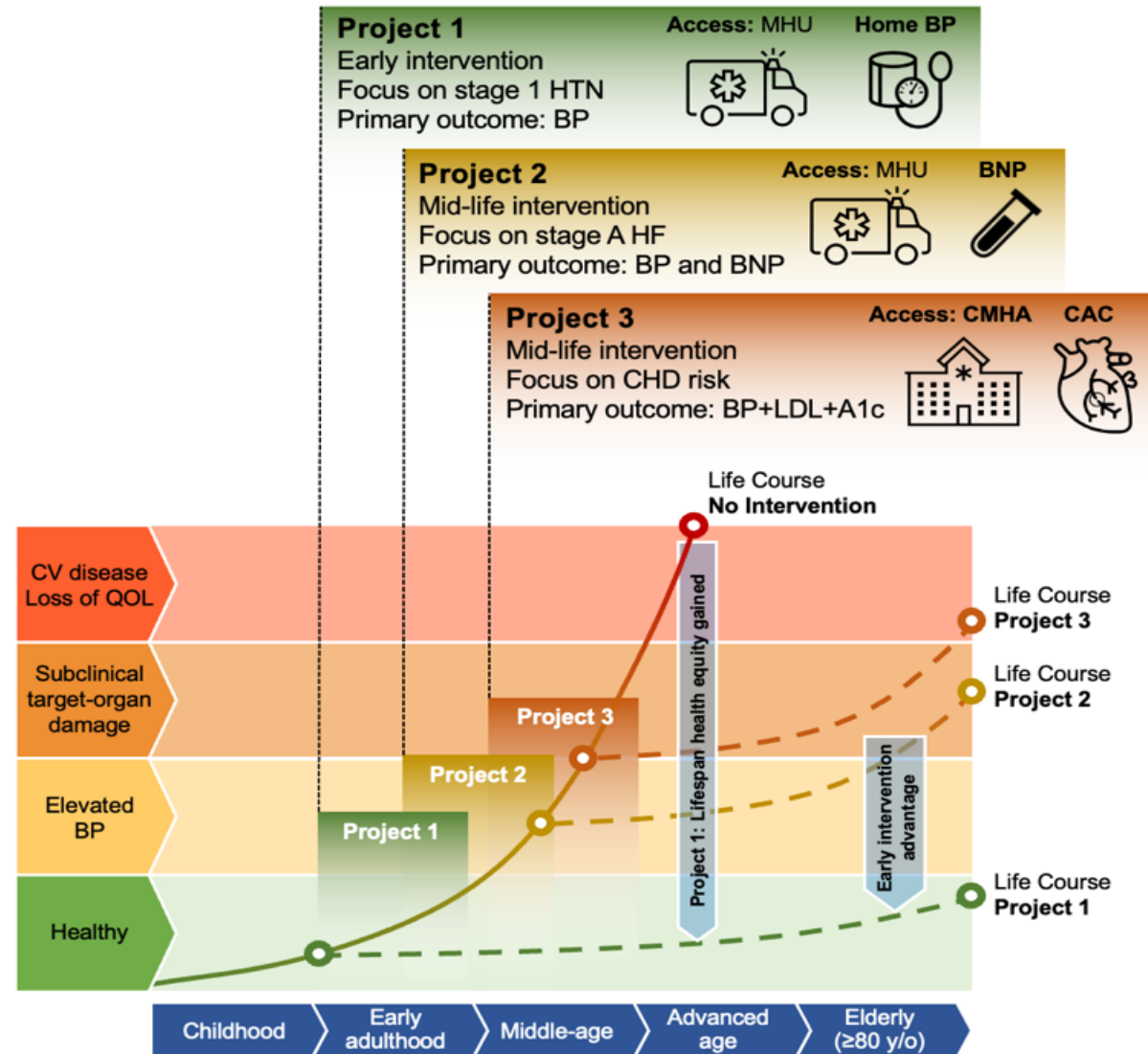
Center: [ACHIEVE GREATER](#)

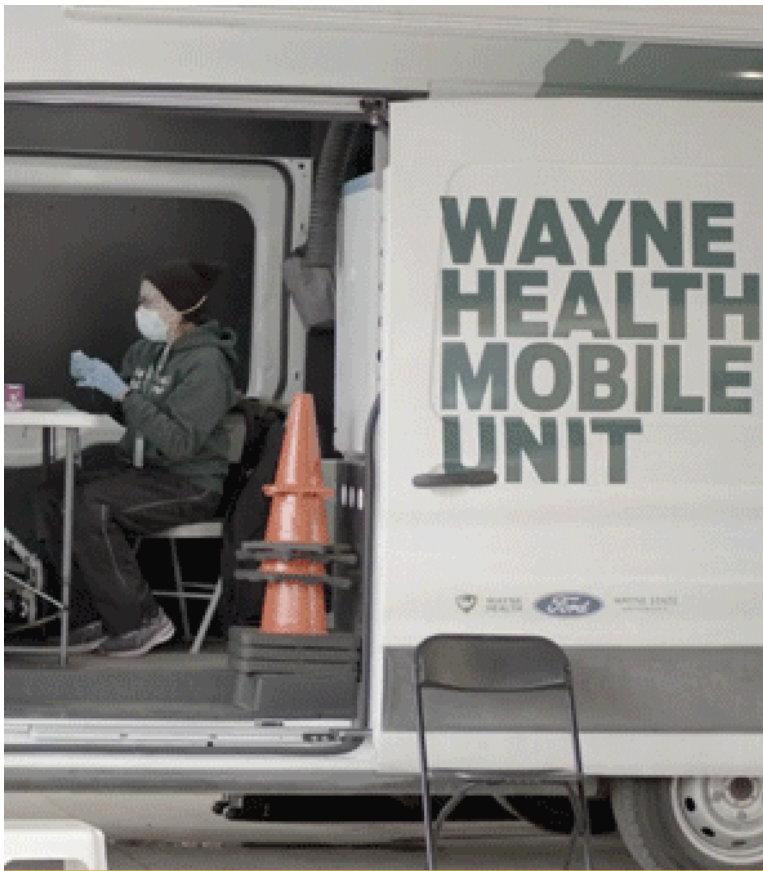
The National Institute on Minority Health and Health Disparities has awarded Wayne State University \$18.15 million over five years to establish a Center for Multiple Chronic Diseases Associated with Health Disparities: Prevention, Treatment, and Management that will use community-based interventions deployed from three research institutions to fight hypertension, heart failure and coronary heart disease in the Black population.

The Addressing Cardiometabolic Health Inequities by Early PreVENTion in the GREAT LakEs Region, or ACHIEVE GREATER, Center is a proactive versus reactive approach to reducing overwhelming cardiometabolic health disparities and downstream Black-White lifespan inequality in Detroit and Cleveland, two uniquely comparable cities.

ACHIEVE GREATER

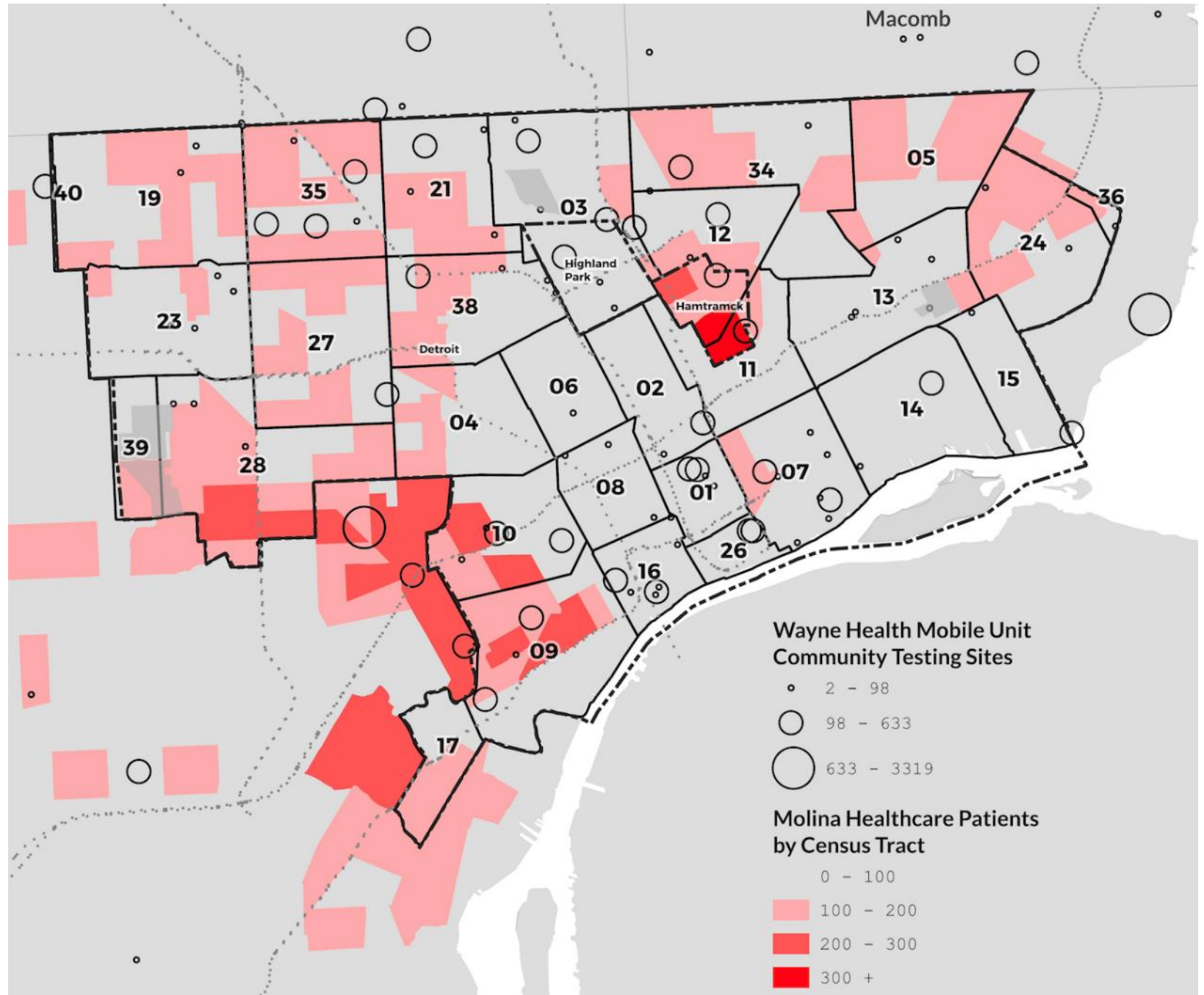
Addressing Cardiometabolic Health Inequities by Early PreVention in the GREAT LakEs Region

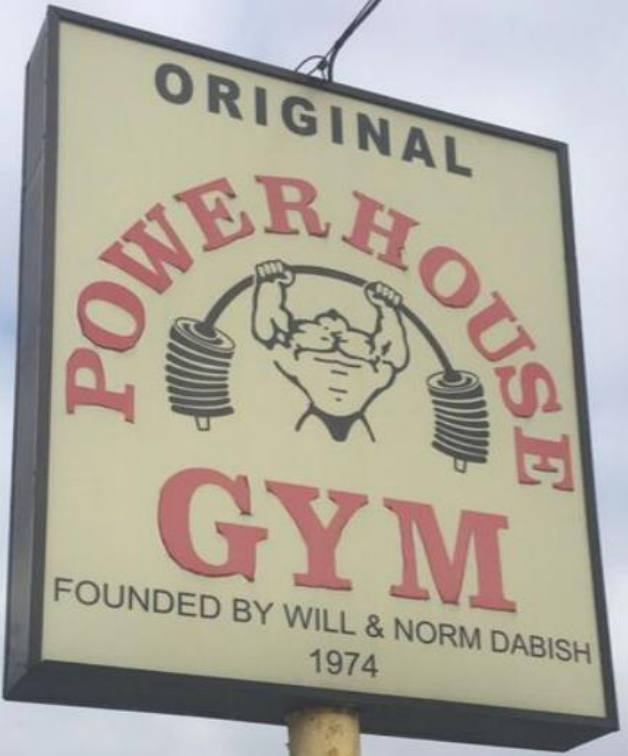


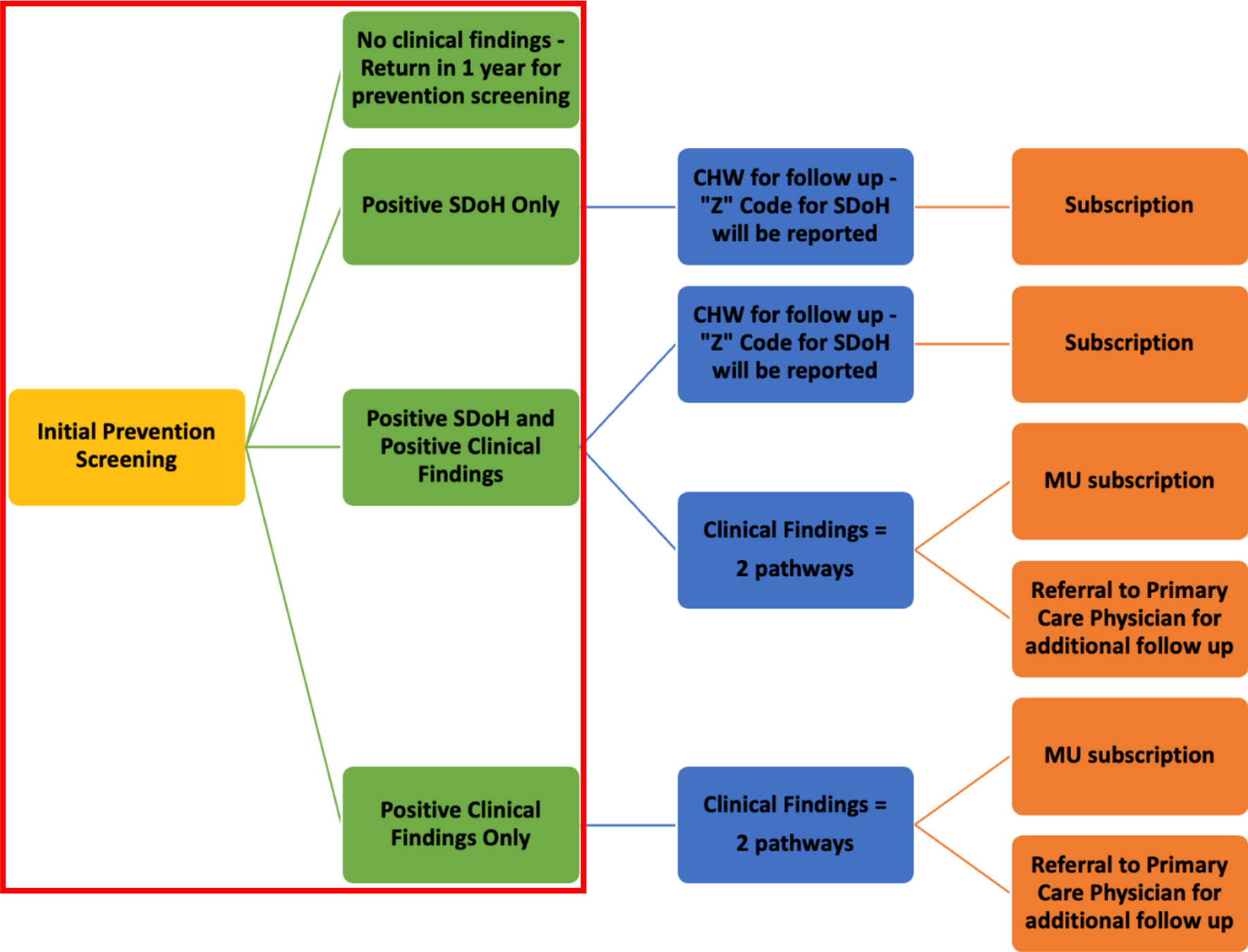


MolinaCares partners with the Wayne Health Mobile Unit

Providing health screenings for the Detroit community.









Play video to add notes



WAYNE
HEALTH

313.448.9850

waynehealthcares.org

00:30



A photograph of a modern building at dusk. The building features a large glass facade that is illuminated from within, and a prominent section with horizontal wooden slats. The sky is a deep blue with scattered clouds. In the foreground, there is a paved area and some landscaping with small trees and bushes. A yellow email address is overlaid on the image.

plevy@med.wayne.edu