HHS Public Access

Author manuscript

Hypertension. Author manuscript; available in PMC 2025 August 25.

Published in final edited form as:

Hypertension. 2025 October ; 82(10): 1696–1704. doi:10.1161/HYPERTENSIONAHA.124.24517.

Lower Pre-Pregnancy Cardiovascular Health is Associated with Hypertensive Disorders of Pregnancy: The CARDIA Study

Leah V. Dodds[1], **Daniel J. Feaster**[1,2], **Catarina I. Kiefe**[3], **Erica P. Gunderson**[4], **Natalie A. Bello**[5], **Tatjana Rundek**[6], **Michael J. Paidas**[7], **Shathiyah Kulandavelu**[8], **Tali Elfassy**[9]

[1]Department of Public Health Sciences, University of Miami Miller School of Medicine, Miami, FL, USA

[2]Department of Biostatistics, University of Miami Miller School of Medicine, Miami FL, USA

[3]Department of Population and Quantitative Health Sciences, University of Massachusetts Chan Medical School, Worcester, MA, USA

[4]Bernard J Tyson Kaiser Permanente School of Medicine, Pasadena, CA, USA

[5]Smidt Heart Institute, Cedars-Sinai Medical Center, Los Angeles, CA, USA

[6]Department of Neurology, Evelyn F. McKnight Brain Institute, University of Miami Miller School of Medicine, Miami, FL, USA

[7]Department of Obstetrics, Gynecology, and Reproductive Sciences, University of Miami Miller School of Medicine, Miami, FL, USA

[8]Department of Pediatrics & Interdisciplinary Stem Cell Institute, University of Miami Miller School of Medicine, Miami, FL, USA

[9]Katz Family Division of Nephrology and Hypertension, University of Miami Miller School of Medicine, Miami, FL, USA

Abstract

Background: Hypertensive disorders of pregnancy (HDP), including gestational hypertension, preeclampsia, and eclampsia, contribute to increased maternal morbidity and mortality and long-term cardiovascular disease risk. It is unclear whether HDP arises from pregnancy-specific complications or pre-existing maternal cardiovascular traits unmasked during pregnancy. This paper will evaluate whether cardiovascular health prior to pregnancy, assessed by the American Heart Association's Life's Essential 8 (LE8) score, is associated with HDP risk.

Methods: CARDIA is a longitudinal cohort study of 5,115 Black and White men and women, aged 18-30 at baseline (1985-86) and followed for over 30 years ($n_{BlackWomen}$=1,480; $n_{WhiteWomen}$=1,307). LE8 score (range: 0-100) was calculated using health behaviors (diet, smoking, physical activity, sleep) and clinical metrics (BMI, blood pressure, cholesterol, glucose).

Corresponding author information: Leah Verna Dodds, PhD, Division of Prevention Science and Community Health, Department of Public Health Sciences, University of Miami Miller School of Medicine, Clinical Research Building, Room 822, Miami FL 33136, lvd10@med.miami.edu.

DISCLOSURES

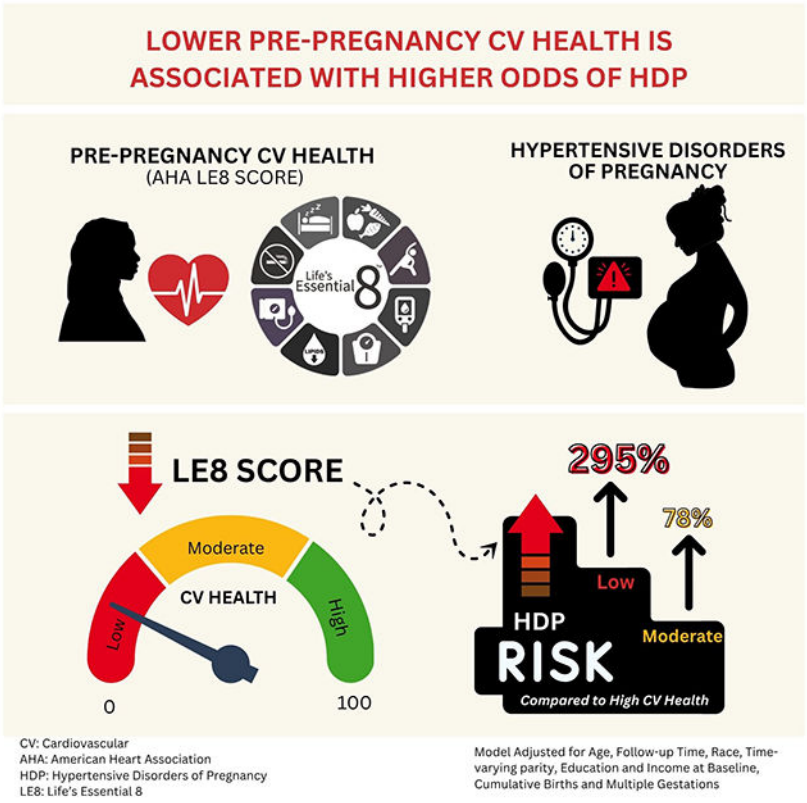
The authors have no relevant financial or non-financial interests to disclose.

Cardiovascular health was categorized as low (LE8 <50), moderate (LE8 50-79), or high (LE8 ≥80). HDP was self-reported as gestational hypertension, preeclampsia, or eclampsia in pregnancies lasting ≥23 weeks. Generalized mixed models assessed the association between LE8 and HDP among 2,036 pregnancies from 1,227 women, adjusting for age, time-varying parity, education, income, follow-up time, cumulative births and multiple gestation.

Results: Mean baseline age was 24.1 years, 48.7% were Black women, and 19.9% reported HDP. Women with HDP had lower baseline LE8 scores (77.0 vs. 79.5; p<0.01). Compared to high cardiovascular health, moderate (OR: 1.78; 95% CI: 1.13-2.81) and low cardiovascular health (OR: 3.95; 95% CI: 1.05-14.88) were associated with increased HDP risk.

Conclusions: Lower pre-pregnancy cardiovascular health is an independent risk factor for HDP. Improving cardiovascular health may reduce HDP risk.

Graphical Abstract



Keywords

Hypertensive Disorders of Pregnancy; Cardiovascular Health; Pre-conception; Pre-pregnancy; preeclampsia; Cardiovascular Disease; Life's Essential 8

INTRODUCTION

Hypertensive disorders of pregnancy (HDP) such as gestational hypertension (GH), preeclampsia, and eclampsia are major contributors to maternal morbidity and mortality.[1]

HDP have been on the rise, complicating approximately 2% to 8% of pregnancies worldwide,[2] and are considered a major contributor to maternal mortality in the US.[3,4] Beyond immediate obstetric complications, HDP confer long-term cardiovascular (CV) risks for affected women, including an increased predisposition to cardiovascular disease (CVD) later in life.[5–7] HDP have been associated with a variety of metabolic abnormalities that are known risk factors for CVD including higher waist circumference, waist/hip ratio, body mass index (BMI), serum insulin level, and lower glucose/insulin ratio.[8] Pre-existing hypertension and compromised CV health prior to pregnancy have also been identified as risk factors for the development of HDP.[9] For example, several studies have consistently shown women with unfavorable CV risk profiles such as those with hypertension, elevated serum lipids and high BMI to have increased risk of HDP.[10–12] HDP can therefore be considered a pivotal event, offering insight into a subset of women already predisposed to heightened cardiometabolic risk from early adulthood. Consequently, the American Heart Association (AHA)[13] and the American College of Obstetricians and Gynecologists[14] stress the importance of prioritizing women's CV health throughout their lifespan, particularly in adolescence.

While HDP is now recognized as a major risk factor for subsequent CVD, it remains unknown whether HDP is merely a marker of preexisting CVD and/or represents an independent risk factor.[15] To better elucidate the magnitude of HDP risk conferred by CVD factors unmasked by the stressful nature of pregnancy and/or from pregnancy-related pathophysiology, our study aims to investigate the association between pre-pregnancy CV risk factors and the incidence of HDP. To operationalize pre-pregnancy CV risk, we will utilize AHA's Life's Essential 8 (LE8) framework. Briefly, LE8 is an updated set of eight health indicators used to monitor ideal health for preventing CVD.[16] LE8 metrics include measures of: diet, physical activity, nicotine exposure, BMI, blood lipids, blood glucose, blood pressure (BP), and recently included sleep health.[16] To do this work we leveraged data from The Coronary Artery Risk Development in Young Adults (CARDIA), a community-based prospective cohort study of Black and White young adults followed for thirty years across the reproductive years.

METHODS

Data Availability

Because of the sensitive nature of the data collected for this study, requests to access the dataset from qualified researchers trained in human subject confidentiality protocols may be sent to CARDIA Coordinating Center at CARDIAdataquestions@uabmc.edu and jwammack@uabmc.edu.

Study Population

In 1985-86, the CARDIA prospective cohort study enrolled 5,115 Black and White US men and women ($n_{BlackWomen}$=1,480; $n_{WhiteWomen}$ =1,307) designed to assess the determinants of CVD and its risk factors. Participants were aged 18-30 were recruited from four field centers in Birmingham, AL; Minneapolis, MN; Chicago, IL; and Oakland, CA and followed at years 2, 5, 7, 10 and then every 5 years through 2016. Participants were selected with

similar sample sizes in subgroups of sex, age, race (Black vs. white), and education (≤high-school vs. >high-school) across centers. One participant withdrew consent. Standardized protocols were used to gather demographic, socioeconomic, medical history and clinical data, including pregnancy information.[17]

Study Sample

Out of 2,787 women who participated in CARDIA, 1,316 self-reported at least one birth after the 1985 baseline visit. Among these women, 83 had a documented history of HDP at baseline, and 6 participants did not provide HDP data, and were consequently excluded from the analysis, Figure S1. As we did not have information on CV risk factors prior to the 1985 baseline visit, we only included pregnancies occurring after baseline, at exam year 2 (1987-1988) onward, so that we could establish temporality between our exposure and outcome. Only pregnancies resulting in live births or still births (n=2,036) were included in the study, defined as self-reported gestational age greater than 23 weeks not resulting in miscarriage, abortion, tubal pregnancy. The unit of analysis is pregnancies; therefore, a multiple gestation pregnancy (i.e. twins) was considered as one observation, 2.3% (n=46) our sample were multiple gestation pregnancies. If a woman reported two or more pregnancies in between exams, each pregnancy was recorded as a distinct observation. The analytic sample consisted of 2,036 pregnancies nested within 1,227 women who reported HDP data during the CARDIA study (1985-2015).

Exposure - Pre-pregnancy Cardiovascular Health

Pre-pregnancy CV health was conceptualized using the AHA's LE8,[16] Table S1. At all study visits (except where specified), participants underwent a comprehensive assessment of health behaviors (diet, physical activity, nicotine exposure, sleep health) and health factors (BMI, blood lipids, blood glucose, BP) used to estimate LE8.[16] Each measure is described in detail as follows.

Diet was measured as adherence to the Dietary Approaches to Stop Hypertension (DASH),[18] coded from responses to the interviewer-administered CARDIA dietary history questionnaire.[19,20] The CARDIA dietary history queried usual dietary practices and obtained a food frequency of the past month at exam years 0, 7 and 20.[19] Diet quality was based on 8 fundamental components of the DASH diet, including increased intake of fruits, vegetables, low-fat dairy products, whole grains, and nuts and legumes and reduced intake of sodium, sugar-sweetened beverages, and red and processed meats using the methods described by Chang et al[18], and categorized into quantiles according to Lloyd Jones et al.[16] Nicotine exposure was self-reported at each of the examinations using the CARDIA Tobacco Use questionnaire, and 3 cigarette-smoking groups were formed using previously defined criteria[21]: never, current, or former smoker; along with time since last smoked and household tobacco exposure. Participants also self-reported their physical activity (exercise units (EU)) using the validated CARDIA Physical Activity History questionnaire,[22,23] which includes 13 specific categories of recreational sports, exercise, leisure, and occupational physical activity in the past 12 months. A score of 300 EU is roughly equivalent to meeting national physical activity guidelines of ≥150 mins per week i.e. 2 EU for every 1 minute;[24] so a person who exercised 120 mins/week, will have 120*(300/150)= 240 EU.[25] Sleep

health (hours/night) was self-reported using the Sleep Questionnaire[26] as average hours of sleep per night, at exam years 15 and 20. Participants were asked "During the past month, how many hours of actual sleep did you get at night? (This may be different than the number of hours you spend in bed)". BMI (kg/m2) was calculated as a ratio of weight (kg)/height (m^2), measured by trained staff with the participant wearing light clothing and no shoes.[27] BP (mmHg) was measured on the right arm using a random zero sphygmomanometer (year 0 to 15; WA Baum Company, Copaigue, NY, USA) and a BP monitor (year 20 to 25; Omron Healthcare Inc., Lake Forest, Illinois), performed at 1-min intervals after the participant had been sitting quietly for 5 min in a still room.[28] The average of the second and third measurements was taken as the pressure of record.[29] Anti-hypertensive medication use was self-reported, and used in defining hypertension and for LE8 scoring (−20 points, Table S1). Blood lipids (mg/dL) were measured from fasting blood drawn from an antecubital vein in EDTA tubes. High-density lipoprotein cholesterol (HDL-C) was quantified using the dextran sulfate-magnesium chloride precipitation method[30]. Total cholesterol were determined using enzymatic procedures[31] on the ABA 200 bichromatic instrument (Abbott Laboratories, North Chicago, IL). Non-HDL cholesterol, the lipid used in this study, was calculated as total cholesterol minus HDL-C. The use of medication for blood lipids was self-reported and used for LE8 scoring (−20 points, Table S1). Blood glucose (mg/dL) was also measured from fasting blood in fluoridated tubes using the hexokinase ultraviolet method,[32] at all years except for years 2 and 5. For LE8 scoring, fasting blood glucose values were converted to calculated HbA1c using the formula: HbA1c = 2.6 + 0.03 × FBG (mg/dl)[33]. All blood samples collected at CARDIA clinics were sent to laboratories for testing.

Following AHA guidelines for LE8,[16] at each study visit, each of the 8 individual components were scored in a range of 0 to 100, Table S1. For components that were not assessed at every study visit (e.g. diet, sleep health, and blood glucose), we carried forward or backwards the most recent value and imputed missing data with these responses. For example, diet was only measured at Year 0, 7 and 20; therefore, diet at Year 0 was used to impute diet score at Year 2, while diet at Year 7 was used to impute the diet score at Year 5 and Year 10 (the most proximal visit). The same was done for individual missing scores. At each study visit, the overall LE8 score was defined as the average of all 8 individual component scores. CV health was defined based on overall LE8 score and categorized as low (LE8 score <50), moderate (LE8 score 50-79) or high (LE8 score ≥80)[16].

Outcome - Hypertensive Disorders of Pregnancy

HDP was assessed in the pregnancy questionnaire asked in all years of the CARDIA study: *"Since last exam, did you have any of these illnesses or complications during this pregnancy: a. toxemia, including all of the following: high BP, albumin in urine and swelling of the ankle? b. high BP without toxemia?"* An affirmative response to either question was considered HDP. However, in order to elucidate actual cases of GH i.e. hypertension after 20 weeks gestation,[2] women who answered yes to question *b,* who also had hypertension prior to pregnancy were not classified as HDP (n=4). This question was asked for each reported pregnancy since the last exam attended, so only pregnancies occurring after baseline were used to measure incident HDP.

Covariates

Age, race, years of education completed, household income, age at pregnancy, date of delivery, and cumulative pregnancies were obtained via questionnaire at each study year. Follow-up time from baseline to exam year when pregnancy was reported was also recorded. Time from baseline to date of delivery was recorded to determine time to index pregnancy. Time-varying parity was based on self-report from the CARDIA pregnancy questionnaire item *"how many times have you been pregnant*?" at exam 0 and "*how many times have you been since the last exam*?". Parity here is defined as any pregnancy regardless of the pregnancy outcome. Multiple gestation was based on self-report data from the CARDIA pregnancy questionnaire item "*how many babies were born during this birth*?". Gestation number greater than 1 was considered multiple gestation. Hypertension was defined using systolic BP (SBP) and diastolic BP (DBP) according to The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (JNC7)[34] (SBP ≥140mmHg and/or DBP ≥90mmHg) as this was the clinical standard at the time of the study (1985-2015), or self-reported use of anti-hypertensive medication.

Statistical Analysis

We described demographic, socioeconomic, and clinical characteristics overall and based on whether women ever reported HDP throughout the study period. Next, we estimated cumulative incidence of ever reporting HDP overall and according to CV health category at baseline.

To determine the association between CV health and incident HDP, we utilized multivariable adjusted generalized linear mixed models, using a binomial distribution with a logit link function, including random intercepts at the participant level to account for multiple pregnancies within the same individual and repeated measures across study visits. This is important to account for repeated measures (pregnancies) overtime. LE8 was modeled as continuous variables (in 5-unit decrements of overall LE8 score and 10-unit decrements for each LE8 score component) and as a categorical variable (high, moderate, or low CV health). We included both fixed (income and education at baseline) and time-varying covariates in all models. All time-varying covariates were time-updated to exam year prior to the reported pregnancy. Model 1 was adjusted for time-varying age, follow-up time, time from baseline to date of delivery, race, education and income at baseline. Model 2 included model 1 adjustments in addition to time-updated parity, cumulative pregnancies and multiple gestations. Using fully adjusted model 2, a sensitivity analysis was performed among women without hypertension at baseline and another by excluding imputed LE8 values for diet and sleep health in years when these components were unavailable. In such cases, we recalculated LE8 as the average of the remaining 6 or 7 available components. For example, diet and sleep data were both missing in Years 2, 5, 10, 25, and 30, so we computed an "LE6" score using the average of the six remaining components. We did not exclude individuals who developed hypertension throughout the study period, as this would also exclude women who developed preeclampsia superimposed on chronic hypertension. The association between individual LE8 components and HDP were also examined using the

same models and approach. Significance was determined at two-tailed $\alpha = 0.05$. Analyses were conducted using SAS Version 9.4.

RESULTS

Mean age at baseline was 24.1 years (SE: 3.6), 48.7% were Black women, 19.9% had a family income <$16,000, 33.8% had greater than a high school education, 24.5% were married, and 53.2% were nulliparous at baseline. Baseline CV health factors are also shown on Table 1. Across the study period, 244 women (19.9%) reported HDP in at least one pregnancy. Women who reported HDP compared to women who did not were more likely to be Black (p=0.015), unmarried (p=0.010), have less than two pregnancies at baseline (p=0.020), and have greater baseline BMI (p<0.001) and SBP (p<0.001).

At baseline, the average overall LE8 score was 79.0 (SD:11.0) with significantly lower scores (i.e. worse CV health) among women who developed HDP compared to those who did not (77.0 vs 79.5, p=0.002, Table 2). Average LE8 score declined over the study period across both groups; however, individuals with HDP consistently showed lower LE8 scores compared to those without HDP (p<0.001). Difference in LE8 scores between individuals with and without HDP nearly doubled over the study period, increasing from 2.5 points at baseline (year 0) to 5.7 points by year 20 (Figure 1). Average baseline scores for individual LE8 components were lower among women who developed HDP compared to those who did not for sleep health (76.8 vs. 81.1, p=0.022), BMI (83.5 vs. 89.1, p=0.004), and BP (93.8 vs. 96.9, p=0.004). At baseline, 0.7% of women had low CV health, 49.3% had moderate CV health and 50.0% had high CV health. A smaller proportion of women who developed HDP during the study (43.0%) had high CV health at baseline, compared to those who did not develop HDP (51.8%), p=0.029. The cumulative incidence of HDP differed by CV health at baseline and was 17.1% (104/612) among women with high CV health, 22.5% among women with moderate CV health, and 37.5% among women with low CV health (p=0.010), Figure S2.

From fully adjusted generalized linear mixed models (model 2), with pregnancies (i.e. not women) as the unit of analysis, each 5-unit decrement in LE8 score was associated with 18% greater odds of developing HDP (OR: 1.18; [95%CI: 1.07, 1.30]), Table 3. Individual LE8 components were also associated with HDP. In fully adjusted models, every 10-unit decrement in LE8 component score was associated with higher odds of HDP by 18% for BMI (OR:1.18; [95%CI: 1.11, 1.27]), 21% for blood glucose (OR:1.21; [95%CI: 1.06, 1.39]), 10% for blood lipids (OR:1.10; [95%CI: 1.02, 1.19]), and 31% for BP (OR:1.31; [95%CI: 1.17, 1.46]). LE8 scores for diet, physical activity, nicotine exposure and sleep health were not significantly associated with odds of HDP. Overall, compared with high CV health, the odds of developing HDP were 78% greater with moderate CV health (OR: 1.78; [95% CI: 1.13, 2.81]) and 295% greater with low CV health (OR: 3.95; [95% CI: 1.05, 14.88]). In a sensitivity analyses restricted to women without hypertension at baseline, effect sizes were similar in magnitude to what was reported for the full sample, Table S2. In another sensitivity analysis using non-imputed data for diet and sleep health, results were also similar to our main findings, Table S3.

DISCUSSION

In a large community-based study of Black and White parous women, one in five reported HDP in at least one pregnancy over a 30-year period. Low pre-pregnancy CV health is a significant and independent risk factor for HDP. Women with low CV health before pregnancy had approximately four-fold increased odds of developing HDP, while those with moderate CV health had 78% greater odds. Enhancing pre-pregnancy CV health is crucial for HDP prevention, with our results demonstrating that even modest improvements, such as a 5-point increase in the AHA's LE8 score, can markedly reduce HDP risk.

Our findings align with previous research, linking lower CV health to adverse pregnancy outcomes like HDP.[35–39] In a multinational study, Perak et al. characterized CV health using 5 metrics —BMI, BP, lipids, glucose, and smoking — classified as ideal, intermediate, or poor using pregnancy-specific guidelines.[40] A modest increase in CV health score at 24 to 32 weeks' gestation was associated with a 33% lower risk for preeclampsia.[40] Similar to our findings, this study showed a graded relationship: compared to women with all ideal metrics, preeclampsia risk was 213% greater with at least one intermediate metric, 434% greater with one poor metric, and 830% with two or more poor metrics.[40]

We also found that lower scores on individual components of AHA's LE8, specifically BMI, blood lipids, glucose and BP, were associated with higher odds of HDP. This is consistent with research demonstrating that higher pre-pregnancy BMI and BP are associated with increased HDP risk,[8,11,41,42] with obesity alone accounting for 30% of HDP cases in the US.[37] Studies also show that women who develop pre-eclampsia have higher pre-pregnancy HbA1c, glucose, cholesterol, and triglycerides than those who do not.[35] Our findings support this, as individuals with HDP had lower baseline LE8 scores, and steeper declines over time. Even more concerning is the rising prevalence of pre-pregnancy obesity, chronic hypertension, and diabetes in the US,[43] highlighting the need for early interventions. Strategies such as telemonitoring combined with remote BP management have shown promise.[44] Surprisingly, we did not find significant associations between pre-pregnancy health behaviors (diet, physical activity, nicotine exposure, and sleep) and HDP, contrary to prior studies.[45–49] This may be due to their indirect, long-term effect on metabolic health,[50] self-reporting biases, or the persistence of underlying risks despite healthy behaviors. Reduced risk for HDP from these health behaviors may require early and sustained adoption, as early as adolescence,[13,14] which was beyond the scope of our study.

HDP and CVD share common risk factors, likely due to overlapping pathophysiologic mechanisms.[38] For example, changes in maternal BP from preconception to early pregnancy, even within normal ranges, may contribute to adverse pregnancy outcomes like HDP.[42,51] Early vascular dysfunction, such as reduced arterial compliance and retinal microvascular constriction, is also associated with increased HDP risk.[13,52,53] Placental malperfusion, resulting from improper remodeling of uterine spiral arteries, initiates vascular injury well before HDP symptoms emerge.[54,55] This process is influenced by maternal pre-pregnancy CV health, potentially through disrupted angiogenesis, oxidative stress and inflammation.[35,38,55] HDP may thus unmask pre-existing CVD risk, under the physiological demands of pregnancy. These findings underscore that poor pre-pregnancy CV health

precedes and contributes to HDP risk.[13] Importantly, pre-pregnancy CV health is shaped by social determinants, including income, education and broader social factors.[56,57] Addressing these determinants is essential for promoting pre-pregnancy CV health, particularly in vulnerable populations.

This study has limitations that must be considered. First, HDP was self-reported. Although maternal recall of HDP shows high specificity[58–60] , studies - particularly in CARDIA - report low sensitivity and positive predictive value[61,62]. We also lacked data on HDP severity (e.g., early vs. late onset), limiting precision of our findings and the strength of the conclusions we can draw. Future studies would benefit from clinically verified records. Additionally, due to self-report inaccuracy and lack of statistical power[61], we did not characterize HDP into its various subtypes (preeclampsia, eclampsia, and gestational hypertension). To ensure temporality, we excluded women with prior HDP at baseline; however, including women with previous normotensive births may have slightly biased the sample toward non-hypertensive pregnancies. Additionally, health behaviors were imprecisely measured, and sleep and diet data were missing for some visits. We addressed this by extrapolating LE8 scores from the most proximal available year and performed a sensitivity analysis which showed similar results to our main findings. Similarly, we used calculated HbA1c levels vs. actual measured HbA1c; however, derived HbA1c levels have been shown to correlate highly with directly measured values.[63] Additionally, due to small sample sizes in the low CV health group, we were unable to examine protective factors that may buffer the association between low CV health and HDP. Lastly, this is an observational study and as such we were unable establish causality or elucidate underlying mechanisms. Nonetheless, this study has several notable strengths. The data source is recent with a large proportion of Black women - an underrepresented population in research, and utilized longitudinal data across four national locations. Additionally, the application of LE8 as a CV health construct offers a comprehensive approach relevant for the peripartum period.

Recognizing the impact of underlying social factors on pre-pregnancy CV health, our study highlights the need for comprehensive approaches to reduce HDP risk and improve maternal and offspring health outcomes. Further research and intervention strategies aimed at optimizing preconception CV health are warranted to mitigate the burden of adverse pregnancy outcomes like HDP and reduce the risk of future CVD.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

SOURCES OF FUNDING

The Coronary Artery Risk Development in Young Adults Study (CARDIA) is conducted and supported by the National Heart, Lung, and Blood Institute (NHLBI) in collaboration with the University of Alabama at Birmingham (75N92023D00002 & 75N92023D00005), Northwestern University (75N92023D00004), University of Minnesota (75N92023D00006), and Kaiser Foundation Research Institute (75N92023D00003). This manuscript has been reviewed by CARDIA for scientific content. Derived pregnancy variables data were supported by R01 DK106201 (Gunderson, PI), R01 DK090047 (Gunderson, PI) from the National Institute of Diabetes, Digestive and Kidney Diseases.

Leah Dodds is currently supported by NIH/NHLBI (F31 HL165894-01A1). Tali Elfassy was supported by NIH/NIMHD (K01MD014158).

NONSTANDARD ABBREVIATIONS AND ACRONYMS

HDP	Hypertensive Disorders of Pregnancy
GH	Gestational Hypertension
CV	Cardiovascular
LE8	Life's Essential 8
AHA	American Heart Association
CARDIA	Coronary Artery Risk Development in Young Adults
DASH	Dietary Approaches to Stop Hypertension
FBG	Fasting Blood Glucose
EU	Exercise Units
JNC	Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure

REFERENCES

1. Bryant AS, Worjoloh A, Caughey AB, Washington AE. Racial/ethnic disparities in obstetric outcomes and care: prevalence and determinants. American journal of obstetrics and gynecology. 2010;202(4):335–343. [PubMed: 20060513]
2. American College of Obstetricians and Gynecologists. Gestational Hypertension and Preeclampsia: ACOG Practice Bulletin, Number 222. Obstetrics & Gynecology. 2020;135(6)
3. Joseph KS, Boutin A, Lisonkova S, et al. Maternal Mortality in the United States: Recent Trends, Current Status, and Future Considerations. Obstetrics & Gynecology. 2021;137(f5)
4. Ghulmiyyah L, Sibai B. Maternal mortality from preeclampsia/eclampsia. Elsevier; 2012:56–59.
5. Benschop L, Duvekot JJ, Roeters van Lennep JE. Future risk of cardiovascular disease risk factors and events in women after a hypertensive disorder of pregnancy. Heart. 2019;105(16):1273–1278. doi:10.1136/heartjnl-2018-313453 [PubMed: 31175138]
6. Brown MC, Best KE, Pearce MS, Waugh J, Robson SC, Bell R. Cardiovascular disease risk in women with pre-eclampsia: systematic review and meta-analysis. European Journal of Epidemiology. 2013/01/01 2013;28(1):1–19. doi:10.1007/s10654-013-9762-6 [PubMed: 23397514]
7. Wu P, Park K, Gulati M. The Fourth Trimester: Pregnancy as a Predictor of Cardiovascular Disease. Eur Cardiol. 2021;16:e31–e31. doi:10.15420/ecr.2021.18 [PubMed: 34603511]
8. Samuels-Kalow ME, Funai EF, Buhimschi C, et al. Prepregnancy body mass index, hypertensive disorders of pregnancy, and long-term maternal mortality. American journal of obstetrics and gynecology. 2007/11/01/ 2007;197(5):490.e1–490.e6. doi:10.1016/j.ajog.2007.04.043
9. Brown MA, Magee LA, Kenny LC, et al. Hypertensive Disorders of Pregnancy. Hypertension. 2018/07/01 2018;72(1):24–43. doi:10.1161/HYPERTENSIONAHA.117.10803 [PubMed: 29899139]
10. Lackland DT. Racial differences in hypertension: implications for high blood pressure management. Am J Med Sci. 2014;348(2):135–138. doi:10.1097/MAJ.0000000000000308 [PubMed: 24983758]

11. Lewandowska M, Więckowska B, Sajdak S. Pre-Pregnancy Obesity, Excessive Gestational Weight Gain, and the Risk of Pregnancy-Induced Hypertension and Gestational Diabetes Mellitus. J Clin Med. 2020;9(6)doi:10.3390/jcm9061980
12. Magnussen EB, Vatten LJ, Lund-Nilsen TI, Salvesen KÅ, Smith GD, Romundstad PR. Prepregnancy cardiovascular risk factors as predictors of pre-eclampsia: population based cohort study. BMJ. 2007;335(7627):978. doi:10.1136/bmj.39366.416817.BE [PubMed: 17975256]
13. Khan SS, Brewer LC, Canobbio MM, et al. Optimizing Prepregnancy Cardiovascular Health to Improve Outcomes in Pregnant and Postpartum Individuals and Offspring: A Scientific Statement From the American Heart Association. Circulation. 2023;147(7):e76–e91. doi:doi:10.1161/CIR.0000000000001124 [PubMed: 36780391]
14. Brown HL, Warner JJ, Gianos E, et al. Promoting risk identification and reduction of cardiovascular disease in women through collaboration with obstetricians and gynecologists: a presidential advisory from the American Heart Association and the American College of Obstetricians and Gynecologists. Circulation. 2018;137(24):e843–e852. [PubMed: 29748185]
15. Ying W, Catov JM, Ouyang P. Hypertensive Disorders of Pregnancy and Future Maternal Cardiovascular Risk. Journal of the American Heart Association. 2018;7(17):e009382. doi:doi:10.1161/JAHA.118.009382 [PubMed: 30371154]
16. Lloyd-Jones DM, Allen NB, Anderson CAM, et al. Life's Essential 8: Updating and Enhancing the American Heart Association's Construct of Cardiovascular Health: A Presidential Advisory From the American Heart Association. Circulation. 2022;0(0):10.1161/CIR.0000000000001078. doi:10.1161/CIR.0000000000001078
17. Friedman GD, Cutter GR, Donahue RP, et al. CARDIA: study design, recruitment, and some characteristics of the examined subjects. Journal of clinical epidemiology. 1988;41(11):1105–16. [PubMed: 3204420]
18. Chang A, Van Horn L, Jacobs DR Jr., et al. Lifestyle-Related Factors, Obesity, and Incident Microalbuminuria: The CARDIA (Coronary Artery Risk Development in Young Adults) Study. American Journal of Kidney Diseases. 2013;62(2):267–275. doi:10.1053/j.ajkd.2013.02.363 [PubMed: 23601954]
19. Linda Van Horn R, Marty Slattery R, Joan Hilner M, et al. The CARDIA dietary history: development, implementation, and evaluation. Journal of the American Dietetic Association. 1991;91(9):1104–1112. [PubMed: 1918764]
20. Liu K, Slattery M, Jacobs D Jr, et al. A study of the reliability and comparative validity of the cardia dietary history. Ethn Dis. 1994:15–27. [PubMed: 7742729]
21. Moreira HT, Armstrong AC, Nwabuo CC, et al. Association of smoking and right ventricular function in middle age: CARDIA study. Open Heart. 2020;7(1):e001270. doi:10.1136/openhrt-2020-001270 [PubMed: 32201592]
22. Bild DE, Jacobs DR Jr, Sidney S, Haskell WL, Anderssen N, Oberman A. Physical activity in young black and white women the CARDIA study. Annals of epidemiology. 1993;3(6):636–644. [PubMed: 7921312]
23. Jacobs DR, Hahn LP, Haskell WL, Pirie P, Sidney S. Validity and reliability of short physical activity history: CARDIA and the Minnesota Heart Health Program. Journal of Cardiopulmonary Rehabilitation and Prevention. 1989;9(11):448–459.
24. Gafni T, Gabriel KP, Shuval K, Yaffe K, Sidney S, Weinstein G. Physical activity trajectories, autonomic balance and cognitive function: The Coronary Artery Risk Development in Young Adults (CARDIA) study. Preventive Medicine. 2022/11/01/ 2022;164:107291. doi:10.1016/j.ypmed.2022.107291 [PubMed: 36209818]
25. Gabriel KP, Sidney S, Jacobs DR Jr, et al. Convergent validity of a brief self-reported physical activity questionnaire. Medicine and science in sports and exercise. 2014;46(8):1570. [PubMed: 24496119]
26. Lauderdale DS, Knutson KL, Yan LL, Liu K, Rathouz PJ. Sleep duration: how well do self-reports reflect objective measures? The CARDIA Sleep Study. Epidemiology (Cambridge, Mass). 2008;19(6):838. [PubMed: 18854708]

27. Burke GL, Jacobs DR Jr, Sprafka JM, Savage PJ, Sidney S, Wagenknecht LE. Obesity and overweight in young adults: the CARDIA study. Preventive Medicine. 1990;19(4):476–488. [PubMed: 2204915]
28. Jacobs DR Jr, Yatsuya H, Hearst MO, et al. Rate of decline of forced vital capacity predicts future arterial hypertension: the Coronary Artery Risk Development in Young Adults Study. Hypertension. 2012;59(2):219–225. [PubMed: 22203738]
29. Dyer AR, Liu K, Walsh M, Kiefe C, DR J Jr, Bild DE. Ten-year incidence of elevated blood pressure and its predictors: the CARDIA study. Journal of human hypertension. 1999;13(1):13–21. [PubMed: 9928747]
30. Warnick G, Benderson J, Albers J. Dextran sulfate-Mg2+ precipitation procedure for quantitation of high-density-lipoprotein cholesterol. Clinical chemistry. 1982;28(6):1379–1388. [PubMed: 7074948]
31. Warnick GR. Enzymatic methods for quantification of lipoprotein lipids. Methods in enzymology. Elsevier; 1986:101–123.
32. Manolio TA, Savage PJ, Burke GL, et al. Association of fasting insulin with blood pressure and lipids in young adults. The CARDIA study. Arteriosclerosis: An Official Journal of the American Heart Association, Inc. 1990;10(3):430–436. doi:doi:10.1161/01.ATV.10.3.430 [PubMed: 2188641]
33. Temsch W, Luger A, Riedl M. HbA1c Values Calculated from Blood Glucose Levels Using Truncated Fourier Series and Implementation in Standard SQL Database Language. Methods Inf Med. 2018/01/18 2008;47(04):346–355. doi:10.3414/ME0468 [PubMed: 18690368]
34. Chobanian AV, Bakris GL, Black HR, et al. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension. Dec 2003;42(6):1206–52. doi:10.1161/01.HYP.0000107251.49515.c2
35. Retnakaran R, Shah BR. The adverse cardiovascular risk factor profile of women with pre-eclampsia develops over time in the years before pregnancy. BJOG: An International Journal of Obstetrics & Gynaecology. 2022;129(9):1512–1520. [PubMed: 34954865]
36. Jin M, Liu X, Liu X, et al. Association of pre-/early pregnancy high blood pressure and pregnancy outcomes: a systemic review and meta-analysis. The Journal of Maternal-Fetal & Neonatal Medicine. 2024/01/02 2024;37(1):2296366. doi:10.1080/14767058.2023.2296366 [PubMed: 38151254]
37. Wang MC, Freaney PM, Perak AM, et al. Trends in prepregnancy obesity and association with adverse pregnancy outcomes in the United States, 2013 to 2018. Journal of the American Heart Association. 2021;10(17):e020717. [PubMed: 34431359]
38. Khan SS, Cameron NA, Lindley KJ. Pregnancy as an early cardiovascular moment: peripartum cardiovascular health. Circulation research. 2023;132(12):1584–1606. [PubMed: 37289905]
39. Wang S, Mitsunami M, Ortiz-Panozo E, et al. Prepregnancy Healthy Lifestyle and Adverse Pregnancy Outcomes. Obstetrics & Gynecology. 2023;142(6)
40. Perak AM, Lancki N, Kuang A, et al. Associations of gestational cardiovascular health with pregnancy outcomes: the hyperglycemia and adverse pregnancy outcome study. American journal of obstetrics and gynecology. 2021;224(2):210. e1–210. e17.
41. Yang Z, Phung H, Freebairn L, Sexton R, Raulli A, Kelly P. Contribution of maternal overweight and obesity to the occurrence of adverse pregnancy outcomes. Australian and New Zealand Journal of Obstetrics and Gynaecology. 2019;59(3):367–374. doi:10.1111/ajo.12866 [PubMed: 30024043]
42. Nobles CJ, Mendola P, Mumford SL, et al. Preconception Blood Pressure and Its Change Into Early Pregnancy. Hypertension. 2020/09/01 2020;76(3):922–929. doi:10.1161/HYPERTENSIONAHA.120.14875 [PubMed: 32755413]
43. Ayyash MK, McLaren R Jr, Shaman M, Al-Kouatly HB. Trends in Preeclampsia Risk Factors in the US From 2010 to 2021. JAMA. 2024;332(2):167–169. doi:10.1001/jama.2024.8931 [PubMed: 38857021]
44. Clark III D, Woods J, Zhang Y, Chandra S, Summers RL, Jones DW. Home blood pressure telemonitoring with remote hypertension management in a rural and low-income population. Hypertension. 2021;78(6):1927–1929. [PubMed: 34757773]

45. Jarman M, Mathe N, Ramazani F, et al. Dietary patterns prior to pregnancy and associations with pregnancy complications. Nutrients. 2018;10(7):914. [PubMed: 30018227]
46. Tanaka K, Nishigori H, Watanabe Z, et al. Secondhand smoke exposure is associated with the risk of hypertensive disorders of pregnancy: the Japan Environment and Children's Study. Hypertension Research. 2023/04/01 2023;46(4):834–844. doi:10.1038/s41440-022-01144-3 [PubMed: 36732667]
47. Arvizu M, Stuart JJ, Rich-Edwards JW, Gaskins AJ, Rosner B, Chavarro JE. Prepregnancy adherence to dietary recommendations for the prevention of cardiovascular disease in relation to risk of hypertensive disorders of pregnancy. The American Journal of Clinical Nutrition. 2020;112(6):1429–1437. [PubMed: 32778894]
48. Arvizu M, Minguez-Alarcon L, Stuart JJ, et al. Physical activity before pregnancy and the risk of hypertensive disorders of pregnancy. American Journal of Obstetrics & Gynecology MFM. 2022;4(2):100556. [PubMed: 34933145]
49. Lu Q, Zhang X, Wang Y, et al. Sleep disturbances during pregnancy and adverse maternal and fetal outcomes: a systematic review and meta-analysis. Sleep medicine reviews. 2021;58:101436. [PubMed: 33571887]
50. Landers-Ramos RQ, Dondero K, Imery I, Reveille N, Zabriskie HA, Dobrosielski DA. Influence of cardiorespiratory fitness and body composition on resting and post-exercise indices of vascular health in young adults. Sports Medicine and Health Science. 2024;6(1):54–62. [PubMed: 38463670]
51. Cameron NA, Molsberry R, Pierce JB, et al. Pre-pregnancy hypertension among women in rural and urban areas of the United States. Journal of the American College of Cardiology. 2020;76(22):2611–2619. [PubMed: 33183896]
52. Valensise H, Novelli GP, Vasapollo B, et al. Maternal Diastolic Dysfunction and Left Ventricular Geometry in Gestational Hypertension. Hypertension. 2001/05/01 2001;37(5):1209–1215. doi:10.1161/01.HYP.37.5.1209 [PubMed: 11358930]
53. Vasapollo B, Novelli GP, Gagliardi G, Farsetti D, Valensise H. Pregnancy complications in chronic hypertensive patients are linked to pre-pregnancy maternal cardiac function and structure. American Journal of Obstetrics and Gynecology. 2020;223(3):425. e1–425. e13.
54. Helfrich BB, Chilukuri N, He H, et al. Maternal vascular malperfusion of the placental bed associated with hypertensive disorders in the Boston Birth Cohort. Placenta. 2017;52:106–113. [PubMed: 28454692]
55. McBride CA, Bernstein IM, Sybenga AB, McLean KC, Orfeo T, Bravo MC. Placental maternal vascular malperfusion is associated with prepregnancy and early pregnancy maternal cardiovascular and thrombotic profiles. Reproductive Medicine. 2022;3(1):50–61. [PubMed: 36923963]
56. Wang E, Glazer KB, Howell EA, Janevic TM. Social determinants of pregnancy-related mortality and morbidity in the United States: a systematic review. Obstetrics and gynecology. 2020;135(4):896. [PubMed: 32168209]
57. Braveman P, Gottlieb L. The Social Determinants of Health: It's Time to Consider the Causes of the Causes. Public Health Reports. 2014/01/01 2014;129(1_suppl2):19–31. doi:10.1177/00333549141291S206
58. Stuart JJ, Bairey Merz CN, Berga SL, et al. Maternal Recall of Hypertensive Disorders in Pregnancy: A Systematic Review. Journal of Women's Health. 2013/01/01 2012;22(1):37–47. doi:10.1089/jwh.2012.3740
59. Tomeo CA, Rich-Edwards JW, Michels KB, et al. Reproducibility and Validity of Maternal Recall of Pregnancy-Related Events. Epidemiology. 1999;10(6)
60. Roberts CL, Bell JC, Ford JB, Hadfield RM, Algert CS, Morris JM. The accuracy of reporting of the hypertensive disorders of pregnancy in population health data. Hypertens Pregnancy. 2008;27(3):285–297. doi:10.1080/10641950701826695 [PubMed: 18696357]
61. Gunderson EP, Chiang V, Lewis CE, et al. Long-term blood pressure changes measured from before to after pregnancy relative to nonparous women. Obstetrics and gynecology. Dec 2008;112(6):1294–1302. doi:10.1097/AOG.0b013e31818da09b [PubMed: 19037039]

62. Carter EB, Stuart JJ, Farland LV, et al. Pregnancy Complications as Markers for Subsequent Maternal Cardiovascular Disease: Validation of a Maternal Recall Questionnaire. Journal of Women's Health. 2015/09/01 2015;24(9):702–712. doi:10.1089/jwh.2014.4953

63. Misquith A, Rangareddy H, Chikkanarayanappa V, Sultana A, Ashakiran S. Mathematical model for assessing glycemic control in type 2 diabetes mellitus. Bioinformation. 2024;20(2):116. [PubMed: 38497074]

PERSPECTIVES

This study reinforces the importance of pre-pregnancy CV health in the development of HDP. Our findings suggest that suboptimal CV health prior to conception—particularly with respect to BP, BMI, glucose, and lipids—is associated with increased risk of HDP. This supports the growing recognition that the reproductive period can serve as a critical window for identifying women at elevated risk for future CVD. These results highlight the need for proactive strategies aimed at optimizing CV health before pregnancy. Incorporating CV risk assessment and health promotion into preconception and routine primary care may help prevent adverse pregnancy outcomes such as HDP, while also reducing long-term CV risk in women.

NOVELTY AND RELEVANCE

What is new?

Lower pre-pregnancy LE8 scores (poor CV health) measured repeatedly over 30 years was associated with onset of HDP.

What is Relevant?

These findings highlight the value of assessing and optimizing CV health well before conception to mitigate the risk of HDP.

Clinical/Pathophysiological Implications

Addressing modifiable CV risk factors before pregnancy offers a key opportunity to prevent HDP and reduce long-term CVD risk. These findings support integrating CV health screening into preconception care to improve maternal and CVD outcomes.

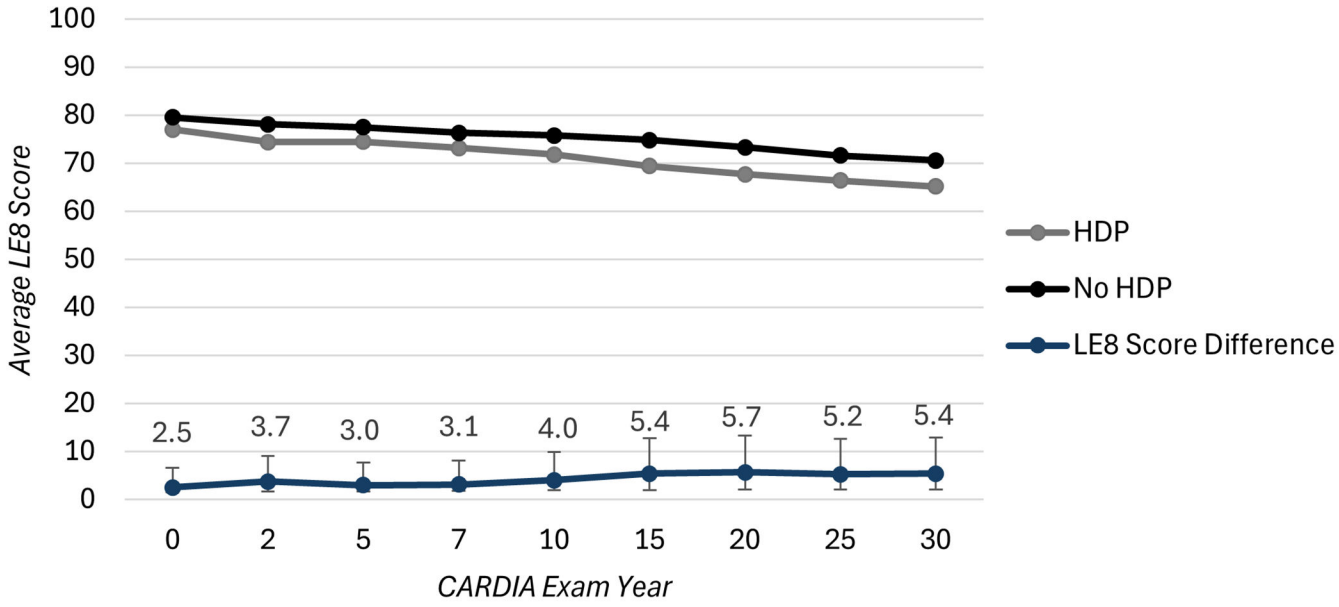


Figure 1.
Differences in LE8 scores were significicantly different at all exam years, p<0.001

Table 1.

Baseline Characteristics among parous women who had at least one pregnancy (n=1227 women) and no history of HDP at baseline (1985-86), stratified by the cumulative incidence of HDP during the CARDIA study 1987-2015 *

	Parous Women without HDP at baseline			
	All n=1227	**HDP n=244, 19.9%**	**No HDP n=983, 80.1%**	**p-value***
Characteristic	**n (%)**	**n (%)**	**n(%)**	
Mean Age (SD): 17-31	24.1 (3.6)	23.8 (3.6)	24.1 (3.7)	0.223
Black Women	598 (48.7)	136 (55.7)	462 (47.0)	0.015
†Family Income				
<$16,000	244 (19.9)	57 (23.4)	187 (19.0)	
<$25,000	148 (12.1)	35 (14.3)	113 (11.5)	
<$35,000	199 (16.2)	35 (14.3)	164 (16.7)	
<$50,000	202 (16.5)	32 (13.1)	170 (17.3)	0.316
<$75,000	177 (14.4)	39 (16.0)	138 (14.0)	
≥$75,000	129 (10.5)	23 (9.4)	106 (10.8)	
Missing	128 (10.4)	23 (9.4)	105 (10.7)	
Greater than HS education	415 (33.8)	68 (27.9)	347 (35.3)	0.072
Married	301 (24.5)	46 (18.8)	255 (25.9)	0.010
Parity				
0	653 (53.2)	127 (52.1)	526 (53.5)	
1	298 (24.3)	74 (30.3)	224 (22.8)	0.020
2+	276 (22.5)	43 (17.6)	233 (23.7)	
LE8 FACTORS	**Mean (SE)**	**Mean (SE)**	**Mean (SE)**	
BMI (kg/m^2)	23.4 (4.7)	24.4 (5.5)	23.2 (4.5)	<0.001
Systolic BP (mmHg) (SD)	105.4 (8.9)	107.2 (9.5)	105.0 (8.8)	<0.001
Glucose (mg/100ml)	79.3 (8.0)	79.7 (6.9)	79.2 (8.2)	0.349
Non-HDL Cholesterol (mg/dL)	120.3 (31.8)	121.2 (32.6)	120.1 (31.7)	0.616
Current Smoker (n %)	314 (25.6)	67 (27.0)	247 (25.2)	0.381
Mean Physical Activity (EU)	355.2 (259.4)	336.9 (232.7)	359.7 (265.6)	0.183
† Average Sleep hours	6.8 (6.6)	6.5 (1.3)	6.9 (7.4)	0.100
Diet (Dash Diet score quartile)	1.68 (1.2)	1.60 (1.1)	1.70 (1.2)	0.190

* Differences between HDP and No HDP groups were assessed using chi-squared tests for categorical variables and t-tests for continuous variables.

† Income based on Y5 (1990); Sleep hours based on Y15 (2005-2006) i.e. not at baseline

Abbreviations: HDP: hypertensive disorder of pregnancy HS: High school; BMI: Body mass index; BP: Blood Pressure; EU: Exercise Units

Table 2:

Baseline average LE8 score, LE8 components score, and CV health status stratified by HDP: CARDIA, 1985-2015 (n=1227 women)

	Parous women without HDP at baseline			
	All (n=1227)	**HDP (n=244)**	**No HDP (n=983)**	
Characteristic	**Mean (SD)**	**Mean (SD)**	**Mean (SD)**	**p-value**
LE8 TOTAL SCORE	79.0 (11.0)	77.0 (10.9)	79.5(11.0)	0.002
LE8 Factor Scores:				
Diet	43.2 (31.8)	41.0 (30.2)	43.8 (32.2)	0.219
Physical Activity	80.6 (26.3)	80.4 (25.4)	80.6 (26.6)	0.911
Nicotine Exposure	61.4 (40.8)	59.3 (41.4)	61.9 (40.7)	0.377
Sleep Health	80.2 (24.9)	76.8 (26.2)	81.1 (24.5)	0.022
Body mass index	88.0 (24.0)	83.5 (27.4)	89.1 (22.9)	0.004
Blood glucose	99.1 (6.9)	99.1 (7.1)	99.1 (6.9)	0.944
Blood lipids	83.7 (24.4)	82.2 (24.8)	84.1 (24.3)	0.288
Blood pressure	96.3 (12.4)	93.8 (15.9)	96.9 (11.3)	0.004
†**CV HEALTH**	**n (%)**	**n (%)**	**n (%)**	
Low	8 (0.7)	3 (1.2)	5 (0.5)	
Moderate	605 (49.3)	136 (55.7)	469 (47.7)	0.029
High	614 (50.0)	105 (43.0)	509 (51.8)	

Abbreviations - LE8: American Heart Association Life's Essential 8; CV: Cardiovascular; HDP: Hypertensive Disorders of Pregnancy; SD: Standard Deviation.

†CV health defined based on overall LE8 score (low: LE8 score <50, moderate: LE8 score 50-79), high: LE8 score ≥80).

Table 3.

Longitudinal association between LE8 score (5-unit decrements), CV health, and LE8 components with HDP during a pregnancy, CARDIA study 1985-2015 (n=2036 births)

	Model 1 OR (95% CI)	Model 2 OR (95% CI)
LE8 OVERALL SCORE	1.16 (1.06, 1.26) *	1.18 (1.07, 1.30) *
Per 5-unit decrement		
LE8 COMPONENT SCORE		
Per 10-unit decrement		
Diet	0.98 (0.92, 1.05)	0.99 (0.92, 1.06)
Physical Activity	0.97 (0.91, 1.03)	1.00 (0.94, 1.07)
Nicotine Exposure	1.03 (0.98, 1.09)	1.03 (0.97, 1.08)
Sleep	1.08 (0.98, 1.20)	1.09 (1.00, 1.20)
Body Mass Index	1.17 (1.10, 1.25) *	1.18 (1.11, 1.27) *
Blood Glucose	1.13 (0.99, 1.29)	1.21 (1.06, 1.39) *
Blood Lipids	1.09 (1.01, 1.18) *	1.10 (1.02, 1.19) *
Blood Pressure	1.21 (1.09, 1.34) *	1.31 (1.17, 1.46) *
†**CV HEALTH**		
High	Ref	Ref
Moderate	1.74 (1.13, 2.69) *	1.78 (1.13, 2.81) *
Low	3.37 (0.98, 11.61)	3.95 (1.05, 14.88) *

* $p<0.05$

† CV health defined based on overall LE8 score (low: LE8 score <50, moderate: LE8 score 50-79), high: LE8 score ≥80)

Odds ratios were estimated using generalized linear mixed models (GLMMs) with random intercepts for individual subjects.

Model 1: adjusted for age, follow-up time, and time from baseline to delivery date, race, education at baseline, and income at baseline

Model 2: Model 1 + time-varying parity, cumulative pregnancies and multiple gestation.