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# Association Between Poverty Level and Prehypertension Risk Among U.S. Adults 

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# Abstract <br> Association Between Poverty Level and Prehypertension Risk Among U.S. Adults by <br> Cynthia Marie Adeyemi 

MPH, Walden University, 2010 BS, Central State University, Wilberforce, Ohio, 1986

Dissertation Submitted in Partial Fulfillment of the Requirements for the Degree of Doctor of Philosophy Public Health-Community Health Promotion and Education Track I

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#### Abstract

Prehypertension (PreHTN) is a significant risk factor for hypertension (HTN) that has been observed in both pediatric and adult populations. In the United States, adjusted mortality rate attributed to HTN in 2018 was highest among non-Hispanic Black (NHB) men followed by NHB females. The purpose of this study was to measure whether lowest, middle, and highest poverty group (LMHPG) and PreHTN varied by age, gender, race, and ethnicity (Hispanic compared to non-Hispanic [HcNH]) and whether the association varied by participants responding yes or no to having been told by a doctor or healthcare professional that they have PreHTN. This study, guided by a critical race theory (CRT) framework, used secondary data from the 2007-2008 National Health and Nutrition Examination Survey database ( $n=4,939$ ). Logistic regression and chi-square test of association were performed. There was a statistically significant association between LMHPG and PreHTN $X^{2}(d f=4,939)=8.684, p$-value $\left.=<.013\right)$. Being in the HPG compared to LPG favored increased odds of $44 \%(p<.010)$ for PreHTN. A withinrace subanalysis revealed that being NHB in the HPG was a significant predictor of $\operatorname{PreHTN}(O R 1.50,90 \%$ CI [1.049, 2.152], $p=<.062)$ compared to non-Hispanic White (NHW) and other race categories. The prospect for social change from this research comes from evidence that being in the HPG is a risk factor for PreHTN. This adds evidence of the need to go beyond traditional public health interventions (e.g., individual level) that tend to use race-neutral approaches and leverage tertiary-level prevention (e.g., public policy) such as reparations to American descendants of slavery and Baby Bonds to address the core roots of poverty and minimize its harmful effects.


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## Dedication

I dedicate this research to my ancestors who survived the trans-Atlantic slave trade, the breeding farms, the killing fields of the South, and oppressive conditions everywhere else in America. I thank my late and beloved mother, Lucy Mae-Dean CruseJones, who provided unconditional love and support. I think about you every day, Mama. My late maternal grandmother, Anna Mae Harris, your discipline, and love guide me. My late maternal great-grandmother, Maggie Maxwell, who made sure me and my sister Rhonda always had bread and butter with sugar and hot Lipton tea while listening to Aunt Bea's Bible Stories on the radio. My late father, Matthew Avery Blackmon, who used to let me, and Rhonda steer the car while we sat on his lap. As teens, my father always made sure we had safe and reliable cars to drive. I am so grateful for my big brother, Ronnie, whom I'll always regard as a second father. He is the first college graduate and law enforcement professional in the family; I'm so proud of you. My sister Rhonda, we have been together since Day 1, and I love you so. I look to you and Lorenzo Sr. as the epitome of a quality long-term marriage. Anna and Arlena, you are my "Golden Girls," and I love you. To my wonderful children, Adefemi, Folakemi, and Adeola, I am so grateful and proud of you. Adefemi, you are a wonderful father. Folakemi, we are so alike, and I admire your resilience and perseverance. Adeola, you are creative and a loving soul. Last, but certainly not least, I am grateful for my loving Man, Robin aka "Big Daddy"; you have been here for me since the beginning of this journey, cheering me on, lifting me up when I got my work back, and near tears.

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Last but certainly not least, I would like to acknowledge Walden University (WU) for its online learning model and scholar practitioner concept. When I started WU in 2008, online learning was looked down upon. Little did I know WU was light years ahead and knew digital learning focusing on critical thinking, collaborating, leveraging technology for meaningful cross-cultural interactions, and allowing students the freedom to explore understudied public health problems. This model is relevant and has real-world implications. The work was challenging, enjoyable, and allowed me the opportunity to contribute to the body of prehypertension disparities literature.

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Chapter 1: Introduction to the Study
Prehypertension (PreHTN) is a public health challenge and a significant hypertension (HTN) disease risk factor. PreHTN is defined as an elevated blood pressure (EBP) category identifying persons at high risk for HTN and designed to alert the individual and encourage lifestyle modifications to delay onset of HTN (Chobanian et al., 2003). The working definition of PreHTN as it pertains to this research is systolic blood pressure (SBP) 120-139 mmHg and diastolic blood pressure (DBP) $80-89 \mathrm{mmHg}$. The classification for PreHTN has undergone changes since the Sixth Joint National Committee (JNC6; Sheps et al., 1997), Seventh Joint National Committee (JNC7; Chobanian et al., 2003), and the American College of Cardiology/American Heart Association Task Force (ACC/AHATF) on Clinical Practice Guidelines (Whelton et al., 2017). There was no change for the classification in JNC8 (James et al., 2014). The 2017 clinical practice guidelines defined PreHTN as EBP (Whelton et al., 2017); however, for the current research, the term "PreHTN" is used. The changes in adult blood pressure (BP) categories are described in Table 1.

Table 1
Changes in Adult Blood Pressure ( mmHg ) Categories

| JNC 6 category |  | JNC7 |  | JNC8 | ACC/AHATF |  |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
|  | SBP/DBP |  | SBP/DBP | - |  | SBP/DBP |
| Optimal | $\begin{aligned} & <120 / 80 \\ & \mathrm{mmHg} \end{aligned}$ | Normal | $\begin{aligned} & <120 / 80 \\ & \mathrm{mmHg} \end{aligned}$ | - | Normal | $\begin{aligned} & <120 \text { and }< \\ & 80 \mathrm{mmHg} \end{aligned}$ |
| Normal | $\begin{aligned} & 120-129 / 80- \\ & 84 \mathrm{mmHg} \end{aligned}$ | PreHTN (lower limit) | $\begin{aligned} & 120- \\ & 129 / 80-84 \\ & \mathrm{mmHg} \end{aligned}$ | - | Elevated <br> (PreHTN) | $\begin{aligned} & 120-129 \\ & \text { and }<80 \\ & \mathrm{mmHg} \end{aligned}$ |
| Borderline | $\begin{aligned} & 130-139 / 85- \\ & 89 \mathrm{mmHg} \end{aligned}$ | PreHTN (upper limit) | $\begin{aligned} & 130- \\ & 139 / 85-89 \\ & \mathrm{mmHg} \end{aligned}$ | - | $\stackrel{-}{-}^{-}$ | - |
| Hypertension | > 140/90 | Hypertension | $>140 / 90$ | - | Hypertension |  |
| Stage 1 | $\begin{aligned} & 140-159 / 90- \\ & 99 \mathrm{mmHg} \end{aligned}$ | Stage 1 | $\begin{aligned} & 140- \\ & 159 / 90-99 \\ & \mathrm{mmHg} \end{aligned}$ | - | Stage 1 | $\begin{aligned} & 130-139 / \text { or } \\ & 80-89 \\ & \mathrm{mmHg} \end{aligned}$ |
| Stage 2 | $\begin{aligned} & 160-179 / 100- \\ & 109 \mathrm{mmHg} \end{aligned}$ | Stage 2 | $\begin{aligned} & \geq 160 />110 \\ & \mathrm{mmHg} \end{aligned}$ |  | Stage 2 | $\begin{aligned} & \geq 140 / \text { or } \geq \\ & 90 \mathrm{mmHg} \end{aligned}$ |
| Stage 3 | $\begin{aligned} & >180 / 110 \\ & \mathrm{mmHg} \end{aligned}$ | - | - |  | - | - |

The reclassification as defined in the 2017 ACC/AHATF was estimated to increase the overall crude prevalence of HTN by $14 \%$, with the highest prevalence observed among non-Hispanic Black (NHB) males (59\%) compared to their nonHispanic White (NHW) counterparts (47\%; Whelton et al., 2017). PreHTN estimates in the United States have been mixed.

Using the JNC7 PreHTN guidelines, Gupta et al. (2010) analyzed National Health and Nutrition Examination Survey (NHANES) data from 1999-2006 and found that PreHTN affected $36.3 \%$ of disease-free adults > 20 years of age. Gupta et al. (2010) described the term disease free as applying to persons not self-reporting cardiovascular diseases and not taking medication for BP.

Cardiovascular disease (CVD) is defined as abnormal functioning of the heart and circulatory system; it includes heart failure, arteriosclerosis, coronary artery disease, arrhythmia, heart attack, and stroke (Colangelo et al., 2015).

Estimates using data from 1999-2000 and 2011-2012 on U.S. disease-free adults 18 years of age and over reported PreHTN prevalence at $28.2 \%$. Among adults without normal BP, the prevalence of PreHTN decreased from 31.2\% in 1999-2000 to 28.2\% ( $p$ trend $=0.007$; Booth et al., 2017). Similar conclusions have been cited in other studies. For example, Zhang and Moran (2017) reported that PreHTN decreased among all age groups between 1999-2000 and 2013-2014, with the largest decrease observed among those $18-39$ years of age ( $32.2 \%$ vs. $23.4 \%$ ); within this age group, males had higher prevalence than females ( $33.6 \%$ vs. $12.8 \%$; 2013-2014). Zhang and Moran (2017) did not provide an aggregate PreHTN prevalence in their analysis to compare with other studies (Booth et al., 2017; Gupta et al., 2010). There are also mixed PreHTN prevalence estimates between high-income countries; refer to Table 2 (Joffres et al., 2013).

## Table 2

High-Income Countries

| Country | Time period | Participants | PreHTN measures | Prevalence |
| :---: | :---: | :---: | :---: | :---: |
| Canada | 2007-2009 | $\begin{aligned} & N=3,485 \\ & 20-79 \text { years } \end{aligned}$ | 120-139 or DBP <br> $80-89 \mathrm{mmHg}$ | 27.2\% |
| England | 2006 | $\begin{aligned} & N=6,873 \\ & 20-79 \text { years } \end{aligned}$ | $120-139 \text { or }$ <br> DBP <br> $80-89 \mathrm{mmHg}$ | 43.9\% |
| United States | 2007-2010 | $\begin{aligned} & N=10,003 \\ & 20-79 \text { years } \end{aligned}$ | 120-139 or DBP <br> $80-89 \mathrm{mmHg}$ | 36\% |

Among low- and medium-income countries, PreHTN prevalence estimates vary, as demonstrated in a meta-analysis of seven population based cross-sectional studies ( $n=$ $42,001)$ conducted in nine countries, as described in Table 3 (Irazola et al., 2016).

## Table 3

## Low- and Medium-Income Countries

| Country | Time period | Age in years | PreHTN measures mmHg | Prevalence |
| :---: | :---: | :---: | :---: | :---: |
| Pakistan | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 28.1\% |
| Uruguay | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 29.7\% |
| Chile | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 32.1\% |
| Argentina | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 32.8\% |
| China | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 33.6\% |
| Kenya | 2008-2013 | 35-74 | $\begin{aligned} & 120-139 \mathrm{SBP} / \\ & 80-89 \mathrm{DBP} \end{aligned}$ | 34.0\% |

The JNC7 PreHTN measures have been used for meta-analysis ( $n=591,664$ ) participants from 17 prospective studies, from Asia, Europe, and the United States; where PreHTN elevated the risk of coronary heart disease (CHD) (e.g., peripheral artery disease), relative risk $(R R)=1.4395 \% \mathrm{CI},[1.25,1.63], p<0.00001$ (Huang et al., 2015). The JNC7 PreHTN measures have been used among Framingham Heart Study participants $(n=5,181)$ for assessing the risk of cardiovascular disease associated with PreHTN (Qureshi et al., 2005).

Within this study, compared to normotensive (reference), PreHTN was significantly associated with myocardial infraction (MI) risk, $R R 3.5,95 \% \mathrm{CI}[1.6,7.5]$, at the 10 -year follow-up.

## Background

## Classification of Prehypertension

The classification for PreHTN is defined in Table 1 of the dissertation. Chobanian et al. (2003) gave attention to the PreHTN category to identify individuals at risk for HTN, encourage lifestyle change, and provide empirical evidence for pharmaceutical intervention (Kaplan, 1998). However, meta-analysis has confirmed an association of PreHTN and CVD risk (Guo et al., 2013a; Guo et al., 2013b; Huang et al., 2015). Lifestyle modification has demonstrated reduction in PreHTN risk and has been endorsed by other researchers (Dorough et al., 2014; Fuchs et al., 2016; Wu et al., 2019). For example, in a randomized study assessing the feasibility of electronically delivered PreHTN lifestyle interventions, at an 11-week assessment, persons assigned to the treatment group $(n=12)$ demonstrated a decrease in SBP $\mu=15.1 \mathrm{mmHg}$, SD 4.3 compared to the standard of care group $(n=11) \mu=4.6 \mathrm{mmHg}, S D 8.3, p<.01$ (Dorough et al., 2014).

In a meta-analysis (49 studies, $n=3,517$ ) on the antihypertensive effects of yoga, reported mean effect size was moderate in the intervention group ( $\mathrm{d}_{+} 0.47 ; 95 \% \mathrm{CI}, 0.62-$ $0.32 ; 5.0 \mathrm{mmHg})$ and $\mathrm{DBP}\left(\mathrm{d}_{+} 0.47 ; 95 \% \mathrm{CI}, 0.61-0.32 ; 3.9 \mathrm{mmHg}\right)$ compared to controls, $p<.001$ (Wu et al., 2019). Similarly, pharmaceutical interventions have shown promise (Fuchs et al., 2016).

The risk of developing CVD doubles with each 20 mmHg rise in SBP and 10 mmHg rise in DBP (Rapsomaniki et al., 2014). Whelton et al. (2020) reported that among a normotensive and elevated blood pressure (BP) population, a 10 mmHg increase in SBP was associated with atherosclerotic cardiovascular disease. Pharmaceutical interventions tend to reduce CVD risk (Fuchs et al. 2016). However, to the side effects of medications, such as dizziness and headaches, albeit nonsignificant, could make nonpharmaceutical interventions attractive.

An efficacy trial to assess the effects of a diuretic on the delay of incident HTN and targeted organ damage demonstrated at the 18-month follow-up that cumulative incident HTN was greater among the placebo group (19.5\%) compared to the treatment group ( $11.7 \% ; p<0.004$; Fuchs et al., 2016). The BP as defined by systolic and diastolic (SBP/DBP) 2017 ACC/AHATF reclassification was warranted because elevated BP is associated with CVD risk (Guo et al., 2013a; Rapsomaniki et al., 2014; Whelton et al., 2017). Some demographic groups might continue to be at risk for CVD morbidity and mortality attributed to HTN. This position is grounded in mortality trends from all-cause and CVD among HTN and nonhypertensive (nHTN) population using NHANES I ( $n=$ 10,852; 1971-1975) compared to NHANES III ( $n=12,420 ; 1988-1994$ ) data (Ford, 2011).

The mortality rate among HTN for NHANES I was $42 \%$ greater RR 1.42 than among nHTN ( $p<0.001$ ), and the mortality rate among HTN in NHANES III was $53 \%$ greater $R R 1.53$ than among nHTN ( $p<0.0001$; Ford, 2011) .

There is confusion with respect to PreHTN being defined as untreated SBP $<140$ and DBP $<90$ (Benjamin et al., 2018) compared to treated HTN (James et al., 2014). PreHTN overlaps with masked HTN (MHT; Shimbo et al., 2012). Depending upon the study, MHT is sometimes measured using PreHTN criteria (Asayama et al., 2014; Redmond et al., 2016; Shimbo et al., 2012). The term MHT refers to evaluated 24-hour ambulatory blood pressure monitoring (ABPM; Pickering et al., 2002) and is recorded at intervals using an automated device (Pogue et al., 2009). ABPM is based on 24-hour readings and is considered a better predictor of CVD, especially in the presence of organ damage (Eguchi et al., 2008). MHT places populations at elevated cardiovascular risk (Asayama et al., 2014; Peacock et al., 2014; Redmond et al., 2016).

A reason to be cautious about PreHTN is that empirical evidence reports normal clinic BP could mask HTN when compared to ABPM (Pogue et al., 2009; Wang et al., 2017). There appear to be diverse preferences for HTN measuring instruments among the HTN disparities experts (Anstey et al., 2018; Sharman et al., 2015).

For example, Stergiou et al. (2007) found a positive correlation between office blood pressure (OBP) and home blood pressure (HBP) for systolic/diastolic $S D$ ( $\mathrm{r}=$ $0.79 / 0.83)$; OBP and $\mathrm{ABPM}(0.76 / 0.77 S D$ and HBP and 24-h ABP (0.76/0.78; $S D$ for all correlations, $p<.001$ ) for predicting targeted organ damage (e.g., left ventricular mass index [LVMI]) among hypertensive patients. LVMI is a measure of heart damage (e.g., left ventricular hypertrophy [LVH]) and has been reported as an independent predictor of cardiovascular risk among both the general and HTN populations (Minamino-Muta et al., 2017).

LVH results from a thickened mass inside the heart's left pumping chamber and places the individual at heightened risk of heart attack, stroke, and/or death (Minamino-Muta et al., 2017, p. 1992).

In the Framingham Heart Study ( $n=3,220$ ) follow-up examining the association of LVMI to incident CV disease ( $n=208$ ), all-cause mortality ( $n=124$ ), and CV deaths ( $n=37$ ) found a positive association between LVMI and adverse health outcome (e.g., CVD and/or death; Levy et. al., 1990). Within the study, analysis was stratified by gender.

The 4-year age adjusted rate (AJR) of events for CVD for males with LVMI ( $n=$ 218) compared to males without LVMI $(n=1,185)$ was $12.1 \%$ versus $6.8 \%, \mathrm{p}<0.001$. For females with LVMI $(n=382)$ compared to those without LVMI $(n=1,435)$, the 4year AJR was $7.4 \%$ versus $4.2 \%, p<0.05$. CVD mortality among males with LVMI was higher compared to those without ( $4.8 \%$ vs. $1.0 \%, p<0.001$ ).

Among females with and without LVMI, there was a nonsignificant CVD mortality outcome, $1.0 \%$ compared to $0.4 \%$. All-cause mortality for males was significant for those with LVMI compared to those without LVMI, 9.1\% versus 4.0\%, $p$ $<0.01$; and females had a nonsignificant all-cause mortality with LVMI, $3.9 \%$ compared to without LVMI, 2.4\%.

Within the study, $(\mu)$ DBP was $81.67 \pm 9.66$ for males and $(\mu)$ DBP was $77.50 \pm$ 9.67 for females-measures associated with PreHTN. The key takeaway here is that $\mu$ DBP measure among males was within the PreHTN range (Saklayen \& Deshpande, 2016), providing evidence that this group is at risk for targeted organ damage and CVD mortality.

On the other hand, compared to clinic blood pressure (CBP), ABPM has been reported as a reliable tool for predicting CV events in patients without Type 2 diabetes (T2D), OR 1.20, 95\% CI [1.20,1.41], p.<0.01, and with T2D OR $1.44,95 \% \mathrm{CI}$ [1.44,1.80], $p .<0.01$ (Eguchi et al., 2008). Reliable tools for predicting CV events among low-risk groups such as patients without T2D demonstrate that specific BP measurement tools may help with early detection and timely interventions to reduce disease burden. Measuring and managing BP can help improve BP status and reduce overmedication (Doane et al., 2018).

Ulusoy et al. (2020) reported both ABPM and office BP monitoring identified statistically significantly more HTN among PreHTN patients ( $p<0.001$ ). White coat hypertension (WHC) is defined as unmedicated individuals with elevated clinical blood pressure but normal ABP (Cuspidi \& Mancia, 2016). It tends to occur when BP is taken by a physician compared to a technician (Lequeux et al., 2018; Pickering et al., 1988).

In a study estimating the prevalence of LVH ( $n=69 ; 6-20$ years of age) in obese ( $n=27$ ) compared to non-obese $(n=42)$ children, $32(46 \%)$ patients had WCH, 13 (19\%) had PreHTN, and 24 (35\%) had HTN; ABPM was used to analyze BP groups (Ramaswamy et al., 2016).

Within the study, $22(32 \%$; obese $=15$; nonobese $=7)$ children had LVH, and there were nonsignificant differences among WCH versus PreHTN versus HT ( $37.5 \%$ vs. $46 \%$ vs. $16.7 \%, p=$ nonsignificant). The ratio of LVH in the obese compared to the nonobese group was $55.5 \%$ to $16.6 \%(p<.001)$. Although there were no significant differences between BP groups, the study provides evidence that targeted organ damage appears among children with elevated BP. Based on the reported examples, BP measurement tools for the management, control, and detection of targeted organ damage might be a function of investigator preference (Gupta et al., 2010) and real-world setting (Doane et al., 2018).

Empirical evidence suggests that PreHTN overlaps with MHT (Shimbo et al., 2012). This was demonstrated in an observational study ( $n=813$ ) assessing diagnostic overlap and the interrelationships with left ventricular mass index, where 98 of the 117 individuals with MHT were PreHTN. Within the PreHTN population ( $n=287$ ), $26.5 \%$ of MHT was at the lower range of PreHTN (SBP $120-<130 /$ DBP $80-<85$ ) compared to the optimal BP group with MHT $(n=18), p<0.01$ (Shimbo et al., 2012). Study limitations included a large NHW population ( $>80 \%$ ), greater female representation (58.4\%), and lack of income data availability to determine an association between income by BP range.

Similar conclusions have been drawn regarding the prevalence of MHT and its association with subclinical cardiovascular disease among Jackson Heart Study participants $(n=909)$, where $82.3 \%$ of the MHT population $(n=187)$ were PreHTN (Redmond et al., 2016). The study authors combined normal clinic blood pressure and PreHTN as MHT.

Even after adjusting for the effect of age and gender, subclinical CVD was higher among the MHT group compared to the non-MHT group ( $p<0.001$ ) and clinic HTN ( $p$ $<0.038$ ) group. Study limitations included a population that was $100 \%$ NHB and lack of income data to align condition.

In an MHT pilot study $(n=73), 45.2 \%$ of the participants had MHT $(n=33)$, $p<.02$, compared to normotensive ( $n=40$; Larsen et al., 2014). Within the study, the MHT population had $\mu \mathrm{SBP}$ (124) and $\mu \mathrm{DBP}$ (77) in the lower PreHTN range. A gap in the study was the lack of financial data to see if an association of PreHTN exists by income, and the population was $100 \%$ NHB female. MHT overlap with PreHTN may undermine stroke risk as demonstrated by Satoh et al. (2016; $n=1,464$ ), where compared to sustained normal BP ( $n=776$;(ref) the HR for MHT ( $n=100$ ) was $2.05,95 \%$ CI [1.24, 3.41], $p<0.0001$. The MHT group had office BP in the lower range of PreHTN, which demonstrates that the lower range confers CVD risk. The absence of income data reduced the ability to observe an association and whether the association varied by age, gender, race, or ethnicity. PreHTN studies have not been modified to reflect the new HTN guidelines issued in November 2017. Therefore, Chapter 2 focuses on available peer-reviewed data based on JNC7 classification.

## Prehypertension as a Cardiovascular Risk Factor

Risk factors for PreHTN are the same as those for HTN, which include stress (Ford et al., 2016; Zambrana et al., 2016); racial (Glasser et al., 2011; Muntner et al., 2014) or ethnic group membership (Carson et al., 2011); metabolic syndrome (Gupta et al., 2010; Wang et al., 2017); body mass index (BMI; Pratima \& Chandra, 2015); gender
(Booth et al., 2017; Kshirsagar et al., 2006); dietary habits (Madanat et al., 2014); and family history of HTN (Najafipour et al., 2020; Xu et al., 2016). PreHTN is an indicator of cardiovascular risk linked to HTN (Chobanian et al. 2003) and targeted organ damage (Redmond et al., 2016). According to the literature, HTN control is defined as $\mathrm{SBP}<$ 140/DBP $<90$ (Chobanian et al., 2003, p. 25).

Other researchers have advocated for standardized HTN control definitions to monitor population trends (Crim et al., 2012). The problem with PreHTN measurement is that it may overestimate HTN control, as observed in prevalence, treatment, and control analysis citing recent control at $48.3 \%$ (Fryar et al., 2017). Within the analysis, highest control was among ages 40-59 (50.8\%), and younger individuals aged 18-39 demonstrated poorer control (32.5\%). HTN control reports could potentially minimize CVD risk and may lessen the vigilance required to prevent organ damage, cardiovascular events, and death. PreHTN is an important HTN risk factor. In a trend study between 1999-2000 and 2011-2012, the prevalence of incident HTN and CVD risk factors among PreHTN increased (Booth et al., 2017). EBP prevalence appears to cluster around lower income populations (Booth et al., 2017; Gupta et al., 2010; U.S. Census Bureau [USCB], 2021).

PreHTN classification has been used in longitudinal (Qureshi et al., 2005) and meta-analysis (Huang et al., 2015) studies assessing CVD endpoints (e.g., heart attack). There is some confusion with respect to PreHTN being defined as untreated SBP $<140$ and DBP $<90$ (Benjamin et al., 2018) compared to treated HTN (James et al., 2014) in terms of treated BP range.

This may be based on PreHTN overlapping with MHT (Shimbo et al., 2012). MHT places populations at elevated cardiovascular risk (Peacock et al., 2014). For example, Shimbo et al. (2012; $n=813, \geq 18$ years of age) concluded that MHT was found in 117 individuals, of which 98 were PreHTN. This study demonstrates that PreHTN overlaps with MHT, placing populations at risk.

A community-based study $(n=326)$ reported that at Year 2, 13.9\% $(n=11)$ of normotensive and $23.4 \%(n=30)$ of PreHTN population developed incident HTN (Mini et al., 2018). There is evidence that compared to normotension (reference), PreHTN is associated with any cardiovascular event (e.g., stroke) in women $H R 1.66,95 \%$ CI [1.44, 1.92], $p<0.001$ (Hsia et al. 2007) and total CVD in the general population relative risk $R R 1.44,95 \% \mathrm{CI}[1.35,1.53], p<0.001$ (Guo et al., 2013a). Within the latter study, total CVD RR was higher in the PreHTN upper range $R R 1.95,95 \%$ CI $[1.69,2.24], p<0.001$ compared to lower range PreHTN $R R 1.35,95 \%$ CI [1.10, 1.66], $p<0.004$. In a three-country comparison of HTN awareness, treatment, and control, Joffres et al. (2013) found that upper levels of PreHTN were positively associated with ischemic heart disease mortality. I believe HTN control reports could potentially minimize CVD risk and may project a distorted picture of national HTN control.

## Age, Gender, Race, and Ethnicity as Predictors of Prehypertension

The prevalence of PreHTN in the United States varies by age (Joffres et al., 2013; Koebnick et al, 2013), gender (Egan \& Stevens-Fabry, 2015), race (Glasser et al., 2011; Selassie et al., 2011), and ethnicity (Gupta et al., 2010; Zambrana et al., 2016).

Depending on the study, data measuring unmedicated HTN in a disease-free population $\geq$ 20 years of age $(n=10,380)$ estimated that $36.3 \%$ of the U.S. population met PreHTN status (Gupta et al., 2010); another study estimating PreHTN/HTN ( $n=30,958$ ) reported 28.2\% (Booth et al., 2017). The highest PreHTN prevalence was observed in the 60-69 age group (44.2\%) followed by 40-59 (42.3\%; Gupta et al., 2010). The age prevalence was different according to Booth et al. (2017), who reported a decrease in age-adjusted prevalence of PreHTN in those $\geq 60$ years. On the other hand, Kim and Lee (2015) concluded that specific CV risk in women $\geq 60$ years of age moderately increased the risk for PreHTN OR 1.04, 95\% CI [1.00, 1.07] compared to their normotensive male peers; no $p$ - value was given for this statistic. In a 2-year follow-up study, incident HTN in $>50$ years of age was $11.4 \%$ compared to $9 \%$ for $<50$ years of age (albeit not statistically significant; Mini et al., 2018).

The residual lifetime risk is defined as the unadjusted, cumulative absolute risk of developing an adverse health outcome during one's remaining life (Lloyd-Jones et al., 2006). The residual lifetime risk for developing HTN in a subset ( $n=6,313,20-85$ years of age) from the Lifetime Cardiovascular Pooling Project under the 2017 ACC/AHA threshold $(\geq 130 /<80)$ reported NHB men risk $86 \%, 95 \%$ CI [84.1, 88.1]; NHW men $83 \%, 95 \%$ CI [ 82.5, 85.0]; NHB women $85.7 \%, 95 \%$ CI [84.0, 87.5]; and NHW women $69.3 \%, 95 \%$ CI $[67.8 \%, 70.7 \%]$, respectively (Chen et al., 2019). These data show that reducing PreHTN must be a public health priority.

Age and the development of HTN have been observed in a Japanese cohort ( $n=$ 12,490), where older age conferred a moderate risk of HTN progression HR 1.16, $95 \% \mathrm{CI}$ [1.07, 1.27], $p<0.01$ (Ishikawa et. al, 2017). In Central China ( $n=8,565,18-98$ years of age), increasing age was associated with EBP ( $p<0.001$; Chen \& Yuan, 2018). Older age as a risk factor was also observed in an international study assessing HTN awareness, treatment, and control (Joffres et al., 2013).

Males are almost twice as likely to be PreHTN compared to females ( $44.8 \%$ vs. 27.3\%; Gupta et al., 2010). The gender difference was also supported by Booth et al. (2017), where compared to females (22.6\%), males presented with greater PreHTN (34.3\%). On the other hand, there has been evidence of females presenting with a greater risk of PreHTN in the presence of CV risk compared to males (e.g., age and waist size).

Kim and Lee (2015) demonstrated this where PreHTN risk among females was moderately associated with older age $(\geq 60)$ and larger waist circumference $O R 1.04$, $95 \%$ CI $[1.00,1.07]$, compared to their male peers who did not have the risk factor; no $p$ value was given.

Depending upon the study's racial and ethnic group sample, PreHTN prevalence varies. For example, Gupta et al. (2010) reported PreHTN for NHB at $38.9 \%$, for NHW at $36.9 \%$, for Mexican Americans at $32.2 \%$, and for other at $33.0 \%$. Variations between racial group prevalence were also observed by Glasser et al. (2011), where the PreHTN prevalence for NHB was $62.9 \%$ compared to $54.1 \%$ for NHW.

In a pediatric PreHTN and HTN study, compared to other racial and ethnic groups, NHB children had a greater prevalence of PreHTN compared to their NHW, Hispanic, and Asian peers (Lo et al., 2013).

Variations in BP have been reported as a CVD (e.g., stroke) and renal failure risk independent of $\mu$ BP levels (Parati et al., 2012). Among participants in the AngloScandinavian Cardiac Outcomes Trial Blood Pressure Lowering Arm (ASCOT-BPLA) ( $n$ $=2,006$ ), intraindividual (e.g., person level) $\mu$ SBP variability measured from Medical Office Visit 1-7 was a strong predictor of stoke HR 1.43 95\% CI [1.18, 1.74] per 20 $\mathrm{mmHg}, \mathrm{p}<.0001$ (Rothwell et al., 2010). BP reduction of $10 \%-20 \%$ during sleep (e.g., circadian BP) is known as BP dipping (Parati et al., 2012). Zeng et al. (2019) reported that the circadian rhythm disorder group had higher daytime SBP (d-SBP), daytime DBP (d-DBP), and daytime Pulse Pressure (d-PP) but lower nighttime SBP (n-SBP), nighttime DBP (n-DBP), and nighttime PP (n-PP) than the normal circadian rhythm group ( $p<0.0001$ ).

In The Health ABC Study, a biracial longitudinal study (> 70 years of age), annual clinic diastolic BP variability was associated with all-cause mortality HR 1.18 per $1 S D, 95 \%$ CI [1.01, 1.37], $(p<0.05)$; and pulse pressure HR 1.11 , per $1 S D, 95 \% \mathrm{CI}$ [1.02, 1.20], ( $p<0.05$; Wu et al., 2017).

Variations in BP status have been observed among young adults of diverse races and/or ethnicities. For example, young NHBs are less likely to demonstrate BP dipping compared to their NHW peers, 48.3 vs $27.2, p .<0.001$ (Muntner et al., 2014).

Non-BP dipping has been observed in Black Hispanics compared to White Hispanics ( $82.6 \%$ vs. $53.9 \%, p<0.02$; Rodriguez et al., 2016). Empirical evidence has indicated that NHBs transition quicker from PreHTN to HTN compared to their NHW peers, HR $1.35,95 \%$ CI [1.30,1.40], $p<0.001$ (Selassie et al., 2011).

Cardiovascular risk factors within ethnic populations have been found to accelerate the transition from PreHTN to HTN. For example, a study assessing CV and metabolic predictors of progression from PreHTN to HTN among Native Americans $(n=2,894)$ reported that higher baseline SBP per 10 mmHg independently increased the odds of incident HTN in participants without cardiovascular risk (e.g., diabetes mellitus) OR 1.60, $95 \%$ CI [1.30, 2.00] and persons with diabetes mellitus $O R 2.73,95 \%$ CI [1.77, 4.21 ] both $p<.0001$ (De Marco et al., 2009). This observation provides empirical evidence of the relationship between PreHTN and incident HTN among a small ethnic group with and without known CV risk.

PreHTN tends to occur earlier in life for children of color, especially NHBs. In a cross-sectional study of pediatric HTN prevalence, kids meeting PreHTN criteria ( $n=25,370$ ) tended to be NHB ( $n=2,413 ; 15.5 \%$ ) compared to NHW ( $n=8,438 ; 11.8 \%$; Lo et al., 2013). These children (PreHTN) had body mass index (BMI) within normal range. This observation was validated by Kit et al. (2015; $n=1,482$ ), where the prevalence of PreHTN $(n=157)$ was greater among NHB 13.5\% (10.5-17.0) children compared to non-Hispanic Asian 6.9\% (3.3-12.4), NHW 8.3\% (5.5-12.0), and Hispanic 9.1\% (4.3-16.4) peers. Within this study, children diagnosed with PreHTN were overweight $10.9 \%$ (6.6-16.6) and obese $16.7 \%$ (10.8-24.1).

Another study cited variations in youth (6-17) PreHTN prevalence ( $n=74,501$ ) such that it was higher among NHW (34.9\%), compared to NHB (32.7\%), Hispanic (29.6\%), Asian/Pacific Islander (28.6\%), and other/unknown (32.6\%; Koebnick et al., 2013). Within the total study ( $n=237,248$ ), Hispanic children represented $50.4 \%$ of the population, NHW represented $24.4 \%$, other/unknown represented $12.3 \%$, NHB represented $6.8 \%$, and Asian/Pacific Islander represented $6.1 \%$. It must be noted that higher percentages of PreHTN among non-White children might indicate external factors contributing to PreHTN disparities, and this population may not get the timely consideration needed to reduce HTN disease burden in later life.

## Prehypertension and Income

Few studies have assessed the relationship between PreHTN and income. For example, in a Taiwanese study, income $\geq \$ 1,660$ U.S. per month was not a predictor of PreHTN and HTN in adults who had EBP as children $(n=303)$ compared to those without EBP as children ( $n=486$; Su et al., 2014).

In another PreHTN prevalence study ( $n=331$ ), compared to normotensive ( $n=130$ ), less than half of those diagnosed with PreHTN $(n=148)$ had incomes above the federal poverty guidelines (Madanat et al., 2014). On the other hand, income may not be a good predictor of PreHTN disparities.

In the Reasons for Geographic and Racial Differences in Stroke (REGARDS) study $(\operatorname{PreHTN}=5,553)$, income as a risk factor for PreHTN was similar among NHW with $\$ 20,000$ household income OR $1.26,95 \%$ CI $[1.01,1.56]$ compared to NHW making $>\$ 20,000$ OR 1.24, 95 CI [1.04,1.46] and NHB making $\$ 20,000$ OR $0.95,95 \%$, CI [0.69,
1.31] compared to NHB making $>\$ 20,000$ OR $1.05,95 \%$ CI [0.79, 1.42], p. $<0.001$ (Glasser et al., 2011). In another study $(\mathrm{n}=2,558)$ assessing racial disparities in health outcomes among persons making > \$175,000 annually, reported HTN OR 2.9, 95\% CI [1.3, 6.2] for NHB compared to Hispanics $O R 1.6,95 \%$ CI [0.5, 4.9], Asians $O R$ 1.3, $95 \%$ C [0.5, 3.1], and 3.1\% for NHW (ref ; Wilson et al., 2017). It was unknown if PreHTN status was reflected as HTN in this study. According to the USCB (2021), 2020 median household income was highest among Asians $(\$ 94,903)$, compared to NHWs (\$74,912), Whites (\$71,231), Hispanics (any race; $\$ 55,321)$, and NHBs $(\$ 45,870)$.

The PreHTN disparities literature is mixed regarding its association to income (Glasser et al., 2011; Su et al., 2014; Wilson et al., 2017). These data are relevant to the association between income group and PreHTN after controlling for race and ethnicity. In the broader health disparities milieu, there appears to be an association between income group and adverse health (Egen et al., 2017).

Gender may also play a role in PreHTN outcome (Hsia et al., 2007), especially if PreHTN children are raised in households headed by females. In 2020, 23.4\% of femaleheaded households lived below the federal poverty guidelines compared to male-headed households (11.4\%; USCB, 2021a). Low-income status may confer a level of stress among care providers, who may adopt unhealthy behaviors to cope (Ibrahim et al., 2021; Lopez-Cepero et. al., 2018; McEwen, 2012), and these behaviors might be adopted by offspring. By race and ethnicity, some groups such as NHBs are relegated to permanent underclass status in the United States, as corroborated by health and other social measures such as employment, income, and spending patterns (Bureau of Labor Statistics
[BLS], 2018; Noël, 2018; USCB, 2014, 2020c, 2021); intergenerational wealth (Collins \& Wanamaker, 2017); and disproportionate incarceration compared to their NHW and other ethnic peers (Federal Bureau of Prisons [FBP], 2019). Being an underclass group might contribute to higher prevalence of PreHTN in the NHB community.

## Problem Statement

Estimated health care costs for HTN in 2016 were $\$ 79$ billion, $95 \%$ CI ( $\$ 72.6$ billion- $\$ 86.8$ billion; Dieleman et al., 2020). A supplemental report estimates that by 2035, the total cost of HTN as a risk factor for CVD will be $\$ 334$ billion (Khavjou et al., 2016).

An economic case can be made for reducing PreHTN prevalence, as the U.S. government's share of national health care spending is projected to grow 5.5 per year between 2017 and 2026, which may result in a rise in gross domestic product (GDP) spending from $17.9 \%$ in 2016 to $19.7 \%$ by 2026 (Centers for Medicare and Medicaid Services [CMS], 2018). A strong case can be made for decreasing PreHTN among normotensives and incident HTN among those with PreHTN. By preventing PreHTN, public expenditures could be leveraged toward rebuilding deteriorating neighborhoods, thus improving home values, which might translate into family wealth. Better home values may influence improved health outcomes (Mehdipanah et al., 2017), promote safer neighborhoods to walk, support the construction of biking networks (Liu \& Shi, 2017), attract full-service grocery stores versus low-end convenience stores (Shannon, 2020), and move marginalized communities from a sick-care environment to primary CVD prevention and health promotion.

## Purpose

Poverty income ratio (PIR) is a measure of income level accounting for family size (Mazidi et al., 2018) and is defined in this dissertation as lowest, middle, and highest poverty group (LMHPG). The purpose of this research was to measure whether LMHPG and PreHTN varied by age, gender, race, and ethnicity (Hispanic compared to nonHispanic [ HcNH ]); and if the association varies by participants responding yes or no to having been told by a doctor or other health professional if they have PreHTN.

A secondary analysis of existing data was used to assess for associations between LMHPG and PreHTN and whether the associations varied by age, gender, race, and ethnicity (HcNH). Secondary analysis of existing data determined that an association between variables, estimated population prevalence, estimated risk, and findings could potentially be used for public policy. Research questions are shown in Table 4.

## Table 4

## Research Questions and Hypotheses

| Research question | Null hypothesis | Alternative hypothesis |
| :---: | :---: | :---: |
| 1. Is there an association between lowest, middle, and highest poverty group (LMHPG) and prehypertension (PreHTN)? | There is no association between LMHPG and PreHTN. | There is an association between LMPHG and PreHTN. |
| 2. Is there an association between LMHPG and PreHTN controlling for age, gender, race (NHB, other race [OR], and NHW); ethnicity (Hispanic [Mexican and other Hispanic (OR)] compared to nonHispanic [HcNH])? | There is no association between LMHPG and PreHTN controlling for age, gender, race (NHB, OR, and NHW), and ethnicity ( HcNH ). | There is an association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity (HcNH). |


| Research question | Null hypothesis | Alternative hypothesis |
| :--- | :--- | :--- |
| 3. Is there a difference in the | There is no difference in the | There is a difference in |
| prevalence of PreHTN between | prevalence of PreHTN between | the prevalence of |
| LMHPG controlling for age, | LMHPG controlling for age, | PreHTN between |
| gender, race (NHB, OR, and | gender, race, and ethnicity | LMHPG controlling for |
| NHW); and ethnicity (HcNH)? | $(H c N H)$. | age, gender, race, and <br> ethnicity (HcNH). |

## Conceptual Framework

The critical race theory (CRT) framework was used because race and ethnicity were study covariates. The theory was developed out of legal scholarship resulting from limitations of Civil Rights legislation as framed by Derrick Bell, Jr. (1975); Richard Delgado (1989); and Allan Freeman (1978; Ford et al., 2010; Ford \& Airhihenbuwa, 2018) and provides an analysis of race and racism from a legal point of view that can be leveraged across disciplines.

Key concepts within the CRT framework include racism as an embedded social construct; interest convergence; racialized socialization; a Black and White binary of race; racism as it intersects with gender, class, national origin, sexual orientation, and how these combinations play out in various situations; and how counter narratives from impacted individuals contribute to the meaning of disempowerment (Bell, 1975; Delgado, 1989; Freeman et al., 2017). The CRT citations above reflect the most current research. The original architects of CRT (Bell, 1975; Delgado, 1989; Freeman, 1978) promoted the concept from a legal context. CRT has been used as an analysis tool in the public health field (Aymer, 2016; Ford et al., 2009; Freeman et al., 2017; Irizarry \& Raible, 2014) and in educational environments (Allen \& White-Smith, 2018).

The race and ethnicity covariates were expected to strengthen the relationship between the independent and dependent variable, as reported by other researchers (Baron \& Kenny, 1986). However, findings did not predict risk exposure by race (ref) and ethnicity (in table 17, p. 136) using aggregated data and estimated PreHTN prevalence by poverty group (in table 18, p. 138). A sub analysis within the race category did find a statistically significant association between the HPG and PreHTN (table 19, p. 139). When investigating health disparities, race and ethnicity can be a useful proxy to estimate disease burden and reveal social inequities (Paradies et al., 2015; Williams et al., 2016; Williams et al., 2019). This has been shown in morbidity and mortality data reports attributed to HTN (Benjamin et al., 2019; Murphy et al., 2021).

Evidence illustrates the effects of the U.S. racial caste system as demonstrated in health services (Sivashanker et al., 2020) and public health outcomes (Cobbinah \& Lewis, 2018). The racial caste system has been described as involving unequal and sustained distribution based on race of privileges, opportunities, as well as duties and obligations, resulting in Whites compared to African Americans receiving almost all privileges and prestige in society (Givel, 2021). The higher prevalence of EBP among non-White racial and ethnic groups compared to their NHW peers might be an expression of social hierarchy (Glasser et al., 2011; Gupta et al., 2010; Hardy et al., 2017; Lo et al., 2013). Other researchers have described a caste system as a hierarchal structure that designates population groups into social classes (Harawa \& Ford, 2009; Shaikh et al., 2018). Racism, also known as White supremacy, is best defined as a system of dominance, power, and privilege based on racial group designations resulting in a racial
caste system designed to exclude nondominant groups from power, esteem, and/or fair access to goods, services, and opportunities (Bailey et al., 2021; Harrell, 2000; Ibrahim et al., 2021). Within the system of racism, the classification of race was used by colonial health practitioners (Harawa \& Ford, 2009) to scientifically rank groups solely on phenotypic characteristics. The race classification was leveraged to justify inhumane treatment including medical experimentation, placing persons of West African descent, specifically the enslaved population, at the lowest strata (Harawa \& Ford, 2009). Official government agencies tasked with reporting define race as self-identification with a social group (General Publishing Office [GPO], 1997; USCB, 2020a).

Ethnicity has been described as involving shared histories, beliefs, practices, and rituals conferring a sense of pride, identity, and socialization processes (Helms \& Talleyrand, 1997; Markus, 2008). On the other hand, the USCB (2020a) reduces ethnicity to Hispanic compared to non-Hispanic. In any event, a subanalysis stratified by race and ethnicity did provide additional knowledge about the burden of PreHTN, a preventable condition, and the need for bold steps to mitigate the problem.

## Nature of Study

A quantitative approach, specifically secondary analysis of existing data obtained from the 2007-2008 NHANES dataset, was used. These data were appropriate because they contained the independent variable (LMHPG) and dependent variables (PreHTN) to include covariates (Centers for Disease Control and Prevention [CDC], n.d.a). The NHANES database is produced by the National Center for Health Statistics (NCHS) under the CDC and is a government-sanctioned data source (CDC, 2017a). The goal for

NHANES is to produce nutrition and health behavioral data representative of the noninstitutionalized U.S. population (Mirel et al., 2013). The selected variables were analyzed to estimate prevalence of PreHTN, predict the odds of PreHTN, and determine whether the outcome varied by age, gender, race, and ethnicity.

## Definitions

Ambulatory blood pressure measuring (ABPM) is based on 24-hour readings and is considered a better predictor of CVD, especially in the presence of organ damage (Eguchi et al., 2008).

Gross domestic product (GDP) is defined as the market value of the goods, services, and structures produced by the nation's economy in a period minus the value of the goods and services used up in production (U.S. Department of Commerce [USDC], n.d.).

Hypertension is clinically defined as $\mathrm{SBP}>140 \mathrm{mmHg}$ or $\mathrm{DBP}>90 \mathrm{mmHg}$, averaged over two readings, on two visits, after an initial screening (Crim et al., 2012).

Low-, middle-, and high-income countries are defined as follows: Lower middleincome economies have a gross national income (GNI) per capita between $\$ 1,006$ and $\$ 3,955$; upper middle-income economies have a GNI per capita between $\$ 3,956$ and $\$ 12,235$; and high-income economies have a GNI per capita of $\$ 12,236$ or more (World Bank, n.d.).

Masked hypertension (MHT) is defined as nonelevated clinic blood pressure and elevated ambulatory blood pressure (Pickering et al., 2002) and can be detected at intervals using an automated device (Pogue et al., 2009).

Normotensive is defined as person with $\mathrm{SBP} \leq 120$ and DBP $\leq 80$ (Chobanian et al., 2003).

Odds ratio (OR) is a measure of association between an exposure and outcome, where the odds of an outcome will occur given a specific exposure (e.g., LMHPG) compared to an absence of that exposure as demonstrated in studies predicting PreHTN risk (De Marco et al., 2009; Guo et al., 2013a; Madanat et al. 2014).

Poverty income ratio (PIR) is a measure of income level accounting for family size (McClurkin et al., 2015).

Prehypertension (PreHTN) is clinically defined as SBP at $120-139 \mathrm{mmHg}$ and/or DBP at $80-89 \mathrm{mmHg}$ and is an indicator of HTN risk (Chobanian et al., 2003).

The original plan was to use the percentage of PIR leveraging the 2007 U.S. Department of Health and Human Services (USDHHS) poverty guideline for a household of two $(\$ 13,690)$. This method would have produced more participants with a PIR > $350 \%(\$ 13,690 \times 350 \%=\$ 18,482)$ (USDHHS, n.d.a). Therefore, this study used the lowest, middle, and highest poverty income categories as described in Table 5.

## Table 5

Poverty Categories

| Value | Value description |
| :--- | :--- |
| $* \$ 0-\$ 19,000$ | Highest poverty group |
| $\$ 20,000-\$ 54,999$ | Middle poverty group |
| $\geq \$ 55,000$ | Lowest poverty group |
| * Cut points |  |

[^0]Relative risk $(R R)$ is the risk rate of something happening in one group compared to another. A RR of 1 means that there is no difference between the two groups in terms of an outcome rate (e.g., PreHTN). A RR greater than 1 or less than 1 means a specific factor (e.g., age, race, gender, etc.) increases or decreases a group's risk rate (National Cancer Institute [NCI], n.d).

Socioeconomic status is defined as characteristics of an individual or collective group as measured by income, education, and employment classification (D. R. Williams et al., 2016).

Specific poverty threshold is defined as the minimum requirements to meet basic needs such as food and shelter (USCB, 2018). Differential characteristics identified between and within groups often reveal inequities in access to resources (e.g., wealth) from childhood to adulthood (Percheski \& Gibson-Davis, 2020), as well as issues related to privilege, power, and control (Bhutta et al., 2020; Kochhar \& Cilluffo, 2017). This was relevant to my research questions because poverty group was the independent variable, and the dependent variable was PreHTN.

## Assumptions

The assumptions of the study were that socioeconomic status is a proxy for race and ethnicity (D. R. Williams et al., 2016), gender (Egan \& Stevens-Fabry, 2015), and age (Joffres et al., 2013) and may illustrate a pathway predicting populations at elevated risk for PreHTN. The basis for these assumptions is rooted in PreHTN and HTN prevalence data (Booth et al., 2017; Fryar et al., 2017) and CVD incidents (Guo et al., 2013a). Another assumption of this study was self-reported data accuracy. Public health
researchers have found inconsistencies of self-reported HTN and objective measurement of the condition (Dave et al., 2013; J. H. Williams et al., 2016). Depending upon the health condition and/or behavior, self-report and objective measures have been similar. This was observed by Glanz et al. (2009), who found congruency between self-reported use of sunscreen and confirmation of use via skin swabs.

On the other hand, Poltavski et al. (2018) found incongruency between self-report of indirect aggression and participants' global physiological response (e.g., heart rate). The point here is that self-report could underestimate results.

Generalizability was addressed in the 2007-2008 NHANES dataset, which contains 12,943 subjects who have answered questions by a screener, of which 10,149 were interviewed inside the home and 9,764 underwent physical examinations (Johnson et al., 2013). The 2007-2008 Data Documentation, Codebook, and Frequencies for Blood Pressure and Cholesterol interview data represent persons 16 years of age and older who responded to the PreHTN question ( $n=6,546 ;$ CDC, 2009b), and the numbers are prior to the research exclusion and inclusion criteria. The dissertation used data from the inhome interviews only; inclusion and exclusion criteria are described in Chapter 3 (see Figure 1).

## Scope and Delimitations

To date, few studies have demonstrated a relationship between LMHPG as a proxy for PIR and a health outcome (Bailey et al., 2017a; Mazidi et al., 2019). PIR as a covariant has been used to assess low serum vitamin D concentrations and EBP in children (Moore \& Liu, 2017). No study to date has considered the relationship between

LMHPG as an independent variable and PreHTN as the dependent variable when controlling for age, gender, race, and ethnicity.

## Limitations

Limitations to this study included the accuracy of the database analyzed. To minimize inaccurate data, NHANES used a quality assurance and quality control approach to check data after they had been collected by field staff for accuracy and completeness of response (CDC, 2009a, 2009b). To further assure data accuracy, a subset of household interviews was followed up to verify responses; additionally, some household interviews were audio recorded so that NCHS staff and outside contractors could review them (CDC, 2009a). Exclusion of missing data may result in lack of generalizable data. This was demonstrated in a large population study where participants with invalid exercise data may have distorted the outcome (Loprinzi et al., 2014). NHANES manages missing data by assigning symbols and numeric characters for recoding purposes to avoid biased estimates (CDC, 2009b). This dissertation used data that had both the independent and dependent variables, to include covariates.

Cross-sectional studies cannot determine causality because assessment is accomplished at one point in time as demonstrated in public health studies estimating PreHTN prevalence (Booth et al., 2017; Gupta et al., 2010). This is opposite to prospective research with repeated measures over prolonged periods of time providing strong evidence of health outcomes attributed to exposure (Mayne et al., 2019; Pletcher et al., 2008; Qureshi et. al., 2005).

## Significance

PreHTN is comparable to the yellow traffic signal in terms of warning an individual to take heed by implementing realistic strategies to prevent and delay incident HTN. This research is expected to contribute to the body of PreHTN health disparities literature by illustrating the burden of PreHTN by age (Lo et al., 2013), gender (Gupta et al., 2010), and race, and ethnicity (Muntner et al., 2018; Selassie et al., 2011; Zambrana et al., 2016). The ACC/AHA 2017 BP guidelines increased the estimated prevalence of HTN from $32 \%$ to $46 \%$ by increasing the SBP for Stage 1 HTN to 130 and DBP to 80 (Whelton et al., 2017). An analysis of 2011-2014 NHANES BP data among U.S. adults 20 years of age and over $(n=9,623)$ found that $13.7 \%(n=1,276)$ of the population reported BP at 130-139/80-89 and were not taking antihypertension medication (Muntner et al., 2018). This set of the population may be unaware of their CV and targeted organ damage risk resulting from EBP.

Through secondary analysis of existing data, I was able to test hypotheses and identify new associations between variables via inferential statistics to predict risk factors contributing to the outcome (e.g., PreHTN). The social change implication from the results could be used by public health practitioners to create and/or modify existing interventions based on the history and realities of population groups, political will, and the context in which interventions will be implemented (e.g., public policy level). In other words, a one-size-fits-all approach to minimize PreHTN burden and delay incident HTN may not be appropriate.

Rather, simultaneous interventions enacted at the legislative, community, organizational, group (e.g., social networks), and individual levels may reduce the burden of PreHTN and delay incident HTN. The dynamics of all levels working concurrently over a prolonged period (e.g., $>75$ years) may demonstrate positive BP outcomes and achieve the overreaching goals of Healthy People 2030 and beyond (USDHHS, 2020).

## Summary

PreHTN is a public health issue influenced by the complex interplay of personal, environmental, and social factors (Benjamin et al., 2019; Ford et al., 2016; Madanat et al., 2014; Xu et al., 2016). Moving from a sick care environment to one of primary prevention may improve quality-of-life measures, reduce HTN expenditures, and redirect taxpayer dollars to activities that may shift movement toward healthier communities. The theoretical framework used in the research provided a lens facilitating examination of nonmodifiable factors that may contribute to PreHTN disparities.

The definitions of key terms have been included, as well as the research questions and null and alternative hypotheses. Chapter 2 provides a critical evaluation of literature identifying gaps and limitations of existing knowledge. Chapter 3 covers methods for the study and why particular analyses were chosen. Chapter 4 provides results, and Chapter 5 presents interpretations of findings, limitations, recommendations, implications for future research, and conclusions.

## Chapter 2: Literature Review

## Introduction

PreHTN is a public health challenge and a significant HTN disease risk factor. PreHTN is a classification used to identify individuals at elevated risk of transitioning to HTN (Chobanian et al., 2003; Glasser et al., 2011; James et al., 2014)y, and it appears to impact non-White identified populations earlier in life (Flack et al., 2010; Hardy et al., 2017; Lo et al., 2013). Prevalence estimates in the United States have been mixed. For example, data from NHANES 1999 to 2006 indicated that PreHTN affected an estimated $36.3 \%$ of disease-free adults $\geq 20$ years of age (Gupta et al., 2010). A recent estimate using NHANES data from 1999-2000 and 2011-2012 of disease-free adults $\geq 20$ years of age found PreHTN prevalence at $28.2 \%$ (Booth et al., 2017). Among adults without normal BP, the prevalence of PreHTN decreased between 1999-2000 (31.2\%) and 20112012 (28.2\%; $p=0.007$; Booth et al., 2017, p. 277). In the Reasons for Geographic and Racial Differences in Stoke (REGARDS), a biracial prospective study subset $(n=9,799)$ reported a $56 \%$ PreHTN prevalence (Glasser et al., 2011). Glasser et al.'s (2011) study used income as a control variable.

In Chapter 2, I explain the literature review search method. Additionally, I evaluate the strengths and limitations of PreHTN prevalence studies. I address the metabolic risk attributed to PreHTN; the role of race and ethnicity, age, and gender; and the cost of PreHTN. I also describe the conceptual framework guiding the research. The chapter ends with a summary and conclusion.

## Literature Review Search Strategy

After formulating the research questions, I examined the extant literature and found few peer-reviewed articles focusing on the association between income and PreHTN. Public health research examining the relationship between income and health outcomes have examined PIR, ideal CV health associated with fatty liver (Mazidi et al., 2019); nutrient intake by PIR (Bailey et al., 2017a); PIR as a covariant for racial/ethnic prevalence of obesity (Gupta et al., 2021); association between insurance status and ideal CV health after controlling for PIR (McClurkin et al., 2015); correlates of measured PreHTN and HTN among Latina women after controlling for income (Madanat et al., 2014); and flavonoid consumption by participant's socioeconomic status (Vieux et al., 2020). To date, there has not been specific research assessing the association between poverty group and PreHTN.

The search strategy involved evaluating pertinent literature available via the Google search engine and Walden Library databases (in table 6). The key words and conceptual framework identified were cost of PreHTN/HTN; critical race theory; elevated blood pressure; high normal blood pressure; PIR and health outcomes; PreHTN/HTN prevalence/incidence; income and health outcomes; racial and ethnic prevalence/incidence of PreHTN/HTN; and risk factors for PreHTN/HTN. The Google search engine provided governmental websites (e.g., PubMed), Google Scholar, and technical reports from governmental and nongovernmental offices. Walden's library provided a wider selection of databases that produced empirical and historical information.

## Table 6

## Literature Review Research Approach

| Database | Type of documents | Usefulness |
| :---: | :---: | :---: |
| Academic Search Complete (ASC) | Primary, secondary, and 20 grey literature articles | Identified many secondary sources and grey literature of historical significance (e.g., lynching, etc.). Most of the nonprimary sources led me to primary sources. This database went beyond PreHTN/HTN and critical race theory; it was a onestop source. |
| Bureau of Justice Statistics | Inmate race and ethnicity | Provided current year and other demographic data to evaluate sentencing disparities. |
| Centers for Disease Control and Prevention | NHANES variables, codebooks, analysis instructions, morbidity/mortality data | Provided updated statistics on all morbidity and mortality health conditions linked to PreHTN/HTN. Provided tutorials for statistical analysis and other technical resources. |
| CINHAL and Medline Combined Search | Primary and review articles | Primary articles specific to PreHTN and HTN. This database was helpful for public health topics. The database appeared to have the same articles as ASC. |
| Federal Bureau of Prisons | Inmate statistics | The site provided demographic data that were useful for showing sentencing disparities. |
| Federal Reserve | Wealth data and working papers | Provided historical and current data on wealth by demographic groups. Defined reports on proportion of wealth in unique categories (e.g., stocks, retirement, etc.) and attribution to wealth disparities. |


| Database | Type of documents | Usefulness |
| :---: | :---: | :---: |
| Google Scholar | Primary, secondary, and technical reports | When using this source, specificity with search terms is required. Searches can be frustrating and time consuming. A few links to articles were nonworking. The database did provide current Heart Disease and Stroke Statistic Updates (HDSSU) and university library link tool. The site was useful in locating special wealth reports by geographic region (e.g., Boston). |
| Pew Research | Technical reports | Specific African, Asian, and Caribbean immigration data. Wealth inequality data. |
| ProQuest | Primary articles | Site contained most of the same resources as ASC. |
| PubMed | Primary articles | Site was not as useful for articles, except for articles that did not require purchase. Found more abstracts, had to go through Walden's library for updated and unusual reports. Site was useful for publicly available reference checks. |
| National Center for Health Statistics | Morbidity and mortality data | Extremely useful for validating the accuracy of articles and technical reports. |
| U.S. Bureau of Labor Statistics | Labor market, other income data, expenditure reports, and quarterly labor statistics | Labor market reports by quarter and year. Special reports by demographic characteristics (e.g., household income/expenditures). |
| U.S. Census Bureau | Detailed population and household data. | Great source for median household earnings by race/ethnicity, income and poverty reports, aggregate wealth data, and historical census data. |

I used the following inclusion criteria when selecting articles:

- published in English
- available in full text
- published 1996 to 2021; I used earlier articles if more current were not available
- focused on studies using NHANES population samples
- peer reviewed was preferred, but I used technical reports to supplement primary data
- PreHTN/HTN prevalence/incidence, risk factors, age, gender, race and/ethnicity
- studies showing the association between income and health outcome, income as a control variable, morbidity, and mortality data

The literature selection strategy involved reading the study abstract to determine whether the article met inclusion criteria; if so, I obtained the full text for further evaluation. If the text was not available, I made a request through Walden's document delivery service. From there, pertinent information was recorded in the literature review matrix specific to that topic. For example, studies citing CRT and adverse health would be recorded in the CRT literature review matrix. Articles were evaluated on strengths and limitations. This allowed for easier identification of gaps to form the bases to answer research questions.

## Literature Review

## Prehypertension Prevalence Studies

In baseline data from the Framingham Heart Study (FHS), $41 \%$ of the population was PreHTN (Qureshi et al., 2005). In the Bogalusa Heart Study ( $n=1,379$ ), a prospective study assessing the natural history of arteriosclerosis (e.g., artery plaque) found a $27 \%$ PreHTN prevalence (Toprak et al., 2009). A limitation was lack of income data to assess if PreHTN varied by age, gender, race, and ethnicity. In the Heart Study, a prospective investigation of Mexican Americans and NHW ( $n=2,767$; ages 25-65) found prevalence of PreHTN to be $31.3 \%$ and being Mexican with PreHTN increased the risk for Type 2 diabetes (Mullican et al., 2009).

Among high-income countries, PreHTN prevalence ranged from a high of 47.9\% in England to 27.2\% in Canada (Joffres et al., 2013). Among low- and medium-income countries, PreHTN prevalence was highest among Kenyans (34\%) and lowest among Peruvians (24\%; Irazola et al., 2016). In Malaysia, a southeast Asian country, compared to normal BP (28.9\%), the prevalence of PreHTN was reported to be $35.7 \%$ ( $p<0.001$; Rafan et al., 2018).

In the FHS cohort ( $n=5,209$; female $n=2,873$, and male $n=2,336$ ), at the 10 year follow-up ( $n=5,181$ ), compared to normotensive ( $n=1,012$ ), the risk of developing HTN over time was significantly higher for persons with PreHTN $(n=2,127) R R 2.0$, 95\% CI [1.9, 2.2] (Qureshi et al., 2005). Within the study, the PreHTN group had lower range SBP (128)/DBP (80) as noted by the JNC7 Report (Chobanian et al., 2003).

In the second-generation FHS cohort ( $n=1,635$ ), $\mu$ follow-up was 26 years, 481 (29.4\%) offspring developed HTN; $H R 1.93,95 \%$ CI [1.13, 3.29], $(p<0.02)$ for one parent with early onset of HTN ( $<45$ years of age); both parents were associated with a HR 3.56, $95 \%$ CI [2.06, 6.16], $(p<0.001$; Niiranen et al., 2017). A major strength of the FHS is its repeated measures; limitations are the use of NHW population, and that income was not used as a control variable.

In the Multi-Ethnic Study of Atherosclerosis (MESA; $n=3,146$ ), reported at Year 4-year follow-up, 910 normotensives developed HTN (Carson et al., 2011). Within the study, there were no data on PreHTN to assess progression from PreHTN to incident HTN; however, this population was middle aged, which has been found to be a contributor to incident HTN (Chen \& Yuan, 2018). Income was not used as a covariant, and a study strength was the use of specific ethnic populations (e.g., Chinese). In a West Indian prospective study $(n=708)$, reported that at 4-year follow-up, incidence rate for HTN among PreHTN persons was 70.4 per 1,000 person-years compared to 15.2 per 1,000 for normotensive ( $\mathrm{p}<0.001$; Ferguson et al., 2010). At baseline, PreHTN ( $n=$ 254) status increased the risk of progression to HTN compared to persons with normal BP $(n=454)$, which resulted in an incidence rate ratio $(\operatorname{IRR})$ of $4.62,95 \%$ CI $[2.9,7.43], p$ $<0.001$; middle age and weight status were found to be contributors to progression. Study limitations included a Jamaican population; therefore, findings could not be generalized to Americans. The strength was the use of participants 25-79 years of age.

In a prospective cohort study $(n=18,865)$ assessing incident HTN at Year 7-year reported $64 \%$ of the population progressed to HTN; compared to normotensive ( $n=3,069$ ), most progressors who met PreHTN status $(n=8,976)$ were NHB, male, diabetic, had chronic kidney disease, and were young ( $p<0.001$; Selassie et al., 2011). Study strengths included repeated measures and association controlled by age, gender, race, and cardiovascular risk factors (e.g., diabetes). A limitation was lack of income data.

In a Japanese cohort subset ( $n=2,227 ; n=707$ normotensive, $n=702$ PreHTN, and $n=818 \mathrm{HTN}$ ) reported that at Year 11, compared to the normotensive group ( $n=47$ ), the PreHTN ( $n=183$ ) group progressed to HTN HR 3.57, 95\% CI [2.56, 4.88], $p<0.01$, and the mean age was $>45$ (Ishikawa et al., 2017). Strengths included repeated measures, and a limitations were lack of income data and analysis took place in Asia. In a meta-analysis of ( $n=20$ studies, $n=250,741$ ), six studies reported progression from PreHTN to incident HTN; progression ranged from 28 months to 5 years, and the ages ranged from 35-60 (Guo et al., 2011). A strength was the pooled studies, and a limitation was lack of income data to determine if PreHTN varied by age, gender, race, and ethnicity.

An updated report from the American College of Cardiology/American Heart Association Task Force (ACC/AHATF) on clinical practice guidelines provided a clearer picture of who is at risk for heart disease and stroke based on new SBP and DBP ranges (Whelton et al., 2017). The new guidelines essentially recategorized the HTN threshold and have been described (in table 1, p. 2). The reclassification is estimated to increase the
prevalence of HTN by $14 \%$, with the highest prevalence observed in NHB males (59\%) compared to their NHW counterparts ( $47 \%$; Whelton et al., 2017).

The BP reclassification was warranted; however, some groups might continue to be at risk for CV mortality attributed to HTN based on age, gender, income, race, and ethnicity. This position is grounded in mortality trends from all-cause and cardiovascular disease (CVD) among HTN and non-hypertensive (nHTN) populations using NHANES I ( $n=10,852 ; 1971-1975)$ compared to NHANES III $(n=12,420)(1988-1994)$ data (Ford, 2011). According to NHANES I, there were 3,963 deaths $(\mathrm{HTN}=2,859$ and $\mathrm{nHTN}=$ $1,104)$ compared to 2,346 in NHANES III ( $\mathrm{HTN}=1,346$ and $\mathrm{nHTN}=1,000$ ). Compared to the nHTN group, over $2 / 3$ of deaths occurred among the HTN group (Ford, 2011).

Within the study, HTN mortality varied by gender and race. For example, across assessment periods, compared to HTN females, the mortality rate for HTN males was greater (NHANES I: 23.5 vs. 15.0 per 1,000 person-years, $p<0.001$; NHANES III: 15.7 vs. 13.2 per 1,000 person-years, $p<0.025$. Compared to NHB, NHW had lower mortality rates across assessment periods (NHANES I: 24.5 vs. 18.1 per 1,000 personyears, $p<0.004$; NHANES III: 19.1 vs. 13.7 per 1,000 person-years, $p<0.001$ ).

All cause and CVD mortality were not broken down by gender race, race age, and income to determine if variations exist. According to the author, other racial groups were not included because Black and White were the only categories available in NHANES I (Ford, 2011). The above mortality patterns between groups demonstrate that EBP can potentially reduce life expectancy.

For example, in 2018, the mortality rate attributed to HTN was 11.0 per 100,000 ( $n=35,835$; Murphy et al., 2021) compared to 2017 , when it was 10.8 per $100,000(n=$ 35,316; Kochanek et al., 2019).

In the United States, HTN-related mortality from 2000-2018 increased from 171,259 to 270,839; the authors reported an increase among White men; however, disparities among Blacks were 2 folds higher (Rethy et al., 2020). A limitation in this article was lack of age, gender, ethnicity, and income data. A major strength was the periods analyzed. According to the 2021 American Heart Association Heart Disease and Stroke Statistics Report (AHA), the overall age-adjusted mortality rate attributed to HTN in 2018 was 24.1 per 100,000 (Virani et al., 2021) compared to the previous year at 23.1 per 100,000 (Virani et al., 2020).

## Table 7

Age-Adjusted Death Rate Primarily Attributed to Hypertension, 2018

| Race/Ethnicity | Female | Male |
| :--- | :--- | ---: |
| Non-Hispanic Black (NHB) | 37.5 per 100,000 | 56.0 per 100,000 |
| Non-Hispanic American <br> Indian/Alaskan Native (NHAI/AN) | 22.5 per 100,000 | 32.3 per 100,000 |
| Non-Hispanic White (NHW) | 19.6 per 100,000 | 24.1 per 100,000 |
| Hispanic | 16.7 per 100,000 | 23.1 per 100,000 |
| Non-Hispanic Asian/Pacific Islander <br> (NHAI/AN) | 14.9 per 100,000 | 17.2 per 100,000 |

Based on the above data, preventing PreHTN could potentially reduce CV morbidity and mortality, especially among groups demonstrating the greatest burden.

PreHTN has been demonstrated to increase CV morbidity and mortality (Guo et al., 2013a; Hsia et al. 2007; Huang et al., 2014; Huang et al., 2020; Qureshi et al., 2005; Satoh et al., 2016). The problem with HTN measures as addressed in JNC7 (SBP $<140$ and DBP $<90$ ) is that it may overestimate HTN control, as observed in prevalence, treatment and control analysis citing control during 2015-2016 at (48.3\%; Fryar et al., 2017).

Overestimating control may undermine HTN risk through a casual pathway called Masked HTN and place populations at elevated CV risk (Asayama et al., 2014; Peacock et al., 2014). Empirical evidence report PreHTN share overlap with masked HTN (MHT; Shimbo et al., 2012). This was demonstrated in an observational study ( $n=813$ ) where 98 of the 117 individuals with MHT were PreHTN. Within the PreHTN population ( $n=287 ; 26.5 \%$ ) of MHT was at the lower range of PreHTN (SBP 120-129/DBP 80-84) compared to the optimal BP group with MHT $(n=18) p<0.01$ (Shimbo et al., 2012). Study limitations include large NHW population (>80\%), greater female representation $58.4 \%$, and income data was not available to determine an association between income by bp range.

In a MHT pilot study $(n=73), 45.2 \%$ of the participants had MHT $(n=33)$ ( $p<.02$ ) compared to normotensive ( $n=40$; Larsen et al., 2014). Within the study, the MHT population had lower range PreHTN $\mu$ clinic SBP (124) and DBP (77). A gap in the study was the lack of income data to determine if an association between PreHTN exist, and the population was $100 \%$ NHB female.

Masked HTN overlap with PreHTN may undermine stroke risk as demonstrated by Satoh et al. $(2016 ; n=1,464)$ where compared to sustained normal BP ( $n=776$; ref) the $H R$ for MHT ( $n=100$ ) was 2.05, $95 \%$ CI [1.24, 3.41], $p<0.0001$. The Masked HTN group had office (e.g., clinic) BP in the lower range of PreHTN, which demonstrates the lower range confers CV risk. The absence of income data reduced the ability to detect an association and whether the association varied by demographic characteristics. The PreHTN populations as identified in the JNC7 may have missed an opportunity to benefit from early intervention that might have reduced CVD morbidity and mortality attributed to HTN. Few PreHTN studies have been modified to reflect the new HTN guidelines issued in November 2017. Therefore, Chapter two focus is on available peer reviewed data based on the JNC7 classification.

## Risk Factors

In the United States (U.S), the distribution of PreHTN prevalence reflect variations by age, gender, race, and ethnicity. Prior to the new HTN guidelines (Whelton et al., 2017), PreHTN was measured at SBP 120-139 and DBP 80-89. PreHTN prevalence studies tend to rely on the average of three single readings at one visit (Mini et al., 2019; Zhang \& Moran, 2017) relative to the average of $>2$ readings at 2 or greater office visits, as recommended in the 2017 ACC/AHA Guidelines (Whelton et al., 2017, p. 27). A repeated measure during one point of time may not provide appropriate prevalence estimates. PreHTN estimates in the United States (U.S.) have been mixed.

For example, Gupta et al. (2010) used NHANES data from 1999 to 2006 and found PreHTN affected an estimated $36.3 \%(n=3,735)$ of disease-free adults 20 years of age and over. Within this study, PreHTN prevalence for men was (44.8\%) compared to women (27.3\%); NHB (38.9\%) compared to NHW (36.9\%), Mexican Americans (32.2\%), and other (33\%). The problem with this study was that it used the average of three BP measurements at one visit and did not measure income to evaluate if there was an association and its strength was the use of four cycles of NHANES data.

Using a similar population measurement approach, Booth et al. (2017) compared 1999-2000 PreHTN estimates to 2011-2012 and reported prevalence at $28.2 \%$ compared to $31.2 \%$. $p<0.007$ ). The strength in this study was multiple comparison years, use of NHANES data; the limitation was the lack of income to compare with BP group. Using 1999-2014 NHANES data, Zhang et al. (2017) found a decrease in PreHTN among 18 years of age and over. Young adults 18-39 years of age demonstrated the largest decrease from $32.2 \%$ in 1999-2000 to $23.7 \%$ 2013-2014. The problem with this study was that income was not measured to determine an association with PreHTN and its strength was the use of NHANES data. There may be a pattern of PreHTN disparities aligned with the group demonstrating the lowest median household income (USCB, 2021a) and wealth disparities (Bhutta et al., 2020; Kochhar \& Cilluffo, 2017). For example, median income for NHB households in 2020 was $\$ 45,870$ compared to NHW \$74,912; White \$71, 231, Hispanic \$55,321; and Asian \$94,903 (USCB, 2021a).

According to a technical report, post-recession median net worth of lower income White families was four times greater $(\$ 22,900)$ compared to NHB $(\$ 5,000)$ and three times the amount for Latinos (\$7,900; Kochhar \& Cilluffo, 2017). This information is relevant because RQ\#1, ask is there an association between LMHPG and PreHTN and RQ\#2 ask if the association varies by race and ethnicity. Earning a middle-class income may not assure security for all groups, rather, it may place certain groups at greater vulnerabilities (Assari, 2018; Assari et al., 2018; Glasser et al., 2011; Noël, 2018; Wilson et al., 2017).

Vulnerabilities include HTN attributed to hypervigilance (Hicken et al., 2014); stress eating (Ibrahim et al., 2021), risk of all-cause and coronary heart disease (CHD) attributed to low job control (Taouk et al., 2020) and higher probability of Type 2 Diabetes (Brody et al., 2016). Coronary Heart Disease (CHD) is a board definition of conditions such as arterial plaque buildup that affect normal heart functioning and increases risk for heart attack, stroke, and death (Gander et al., 2014).

An Asia, North American, and European meta-analysis (n=32 studies) assessing psychosocial work stressors and risk of all-cause and CHD mortality reported low job control increased the risk of all-cause mortality $H R 1.21,95 \% \mathrm{CI}[1.07,1.37, k=3]$ and CHD Mortality $H R 1.50,95 \%$ CI [1.42, 1.58, $k=5]$ (Taouk et al., 2020). According to the authors, moderate heterogeneity was observed $\mathrm{I}^{2}=59 \%, p=0.045$ for low job control and all-cause mortality. Stress is associated with EBP (Gawlik et al., 2019) and PreHTN in adolescence has been linked to predicted preclinical CVD in young adults (Koskinen et al., 2018).

A limitation of meta-analysis is aggregation of quantitative study findings which do not take into consideration variables such as income, PreHTN, and HTN which are measured at the individual study level to estimate associations (Allen, 2020). Fullerton and Anderson (2013) found that compared to NHW (ref), NHB (53\%), and Hispanics (77\%), $p<0.001$ demonstrated greater job insecurity. Within this study, controlling for occupation, race, ethnicity, insecurity levels, NHB, and Hispanics still demonstrated a significant difference in job insecurity compared to NHW.

Job insecurity is defined as a perceived threat of job loss and may explain an external factor contributing to racial and ethnic CVD disparities (Fullerton \& Anderson, 2013). This casual pathway might be indicative of how cognitive energy (e.g., perceived threat) is used to cope with external stressors. My research examined the association between income group and PreHTN. Prehypertension is a HTN risk factor and stress is linked to HTN (Dolezsar et al., 2014; Michaels et al., 2019).

High level cognitive coping might undermine physical health and may appear to cluster around socially marginalized groups. A study of Black college students ( $n=124$ ) found that Race Related Stress (RRS) was associated with higher DBP ( $F=3.31, d f=2$, $p \leq .05$; Lee et al., 2016). Race Related Stress (RRS) are the perceived demands on the minds, bodies, and wellbeing of non-White identified persons, primarily NHB, this specific stressor taxes existing resources (e.g., social capital) required to challenge unfair treatment within an environment where Racism is embedded, condoned, and implemented by dominant social groups to include their non-White allies who practice White Supremacist ideology (Bailey et al., 2021; Harrell, 2000).

A limitation of Lee et al. (2016) was lack of data to determine if there was an association between EBP at various levels of income.

In a subset of the U.S Longitudinal Study of Adolescent to Adult Health (Add Health) participants ( $n=5,366, \mathrm{NHB}=1,431$; NHW $=3,935$ ), high level coping in the face of disadvantage at age 16 undermined the health of high striving, disadvantage NHB participants at age 29 compared to their least disadvantaged race matched peers (Brody et al., 2016). Within the study, compared to least disadvantage groups $\beta=-0.104,95 \%, \mathrm{CI}$ $[-0.329,0.121], p=.37$, the most disadvantage group was more likely to present with T2D $\beta=0.248,95 \%, \mathrm{CI}[0.072,0.424], p<0.006$. This data demonstrates that the quality of resources may have within group benefits based on economic class. A study limitation was the absence of income data to assess cardiovascular risk by EBP status and gender. A major strength was the study's repeated measures.

In a longitudinal study of Finnish NHW men $(n=2,682)$, reported that CV risk was attributed to high level coping in the face of socioeconomic disadvantage (Mujahid et al., 2017). Within the study, compared to white collar workers (ref), acute myocardial infraction (AMI) was higher among blue collar $H R 3.14,95 \% \mathrm{CI}[1.65,5.98]$ and farm workers $H R 2.33,95 \%$, CI [1.04, 5.22]; additionally, compared to high income (ref), the low-income population showed a higher incident rate of AMI mean stress score (7.13 versus $13.11, p<0.001$ ). A limitation of this study was lack of EBP range to show participants with PreHTN across the 14.9 year assessment period.

## Metabolic Risk Attributed to Prehypertension

Metabolic issues contributing to PreHTN include body mass index (BMI) (Bersamin et al., 2009; Brody et al., 2016; Guo et al., 2011; Gupta et al., 2010; Hsia et al., 2007; Rafan et al., 2018) and sugary drink consumption (Chen et al., 2010;

DeChristopher et al., 2017; Madanat et al., 2014). Metabolic issues are defined as a group of traits such as impaired fasting blood glucose, above normal body mass index, and EBP which disrupts the natural body process and increases one's chance of developing nonoptimal BP and other cardiovascular problems (Mammeri et al., 2019).

In the Western New York Health Study 35-79 years of age $(n=564)$ (normotensive $=375$ and PreHTN $=189$ ) reported that impaired fasting glucose at baseline $O R 1.69,95 \%$ CI [1.06, 2.67], and weight gain $O R 1.28,95 \%$ CI [1.11, 1.58], $p=<0.05$, per 10 lbs . increase since age 25 , were predictors of PreHTN at follow-up (Donahue et al., 2014). A study strength was the use of a subsample of disease-free participants and the identification of PreHTN factors. Limitations include lack of income data, and the population was overwhelming NHW.

In an HTN awareness, treatment, and control study ( $n=2,780$, females $n=1,359$, and males $n=1,421), 33 \%$ of the total populations $(n=921)$ were PreHTN, of this number, $41 \%$ were overweight, $24 \%$ obese, and $5 \%$ were morbidly obese (Bersamin et al., 2009). A limitation in the study was lack of income data to determine if there were variations in PreHTN by age. gender, and the population was $100 \%$ Mexican American.

In a longitudinal study $(n=5,336, \mathrm{NHB}=1,431 ;$ NHW $=3,935)$ assessing predictors of type 2 diabetes (T2D) at age 29 found BMI was associated with T2D $\beta=$ $0.031,95 \%$ CI $[0.023,0.038], p<0.001$ (Brody et al., 2016). A limitation was not showing PreHTN burden, $100 \%$ NHB subanalysis, lack of income to assess a relationship and whether the association varied by gender. The relevance of Brody et al. (2016) as it relates to PreHTN confirms that BMI is a metabolic risk, and this association has been linked to PreHTN and places specific demographic groups at EBP risk.

A PreHTN prevalence and risk factor meta-analysis ( $n=26$ ); reported on 10 studies ( $n=49,552$ PreHTN and $n=37,919$ normotensive) that resulted in a pooled standardized mean differences (SMD) for BMI at 1.37, $95 \%$ CI [1.20,1.55], and heterogeneity was significant $X^{2}=135.74, I^{2}=86 \%$ (Guo et al., 2011). The $S M D$ is a strategy used in meta-analysis combining the means of many studies with different outcome measures (e.g., BMI, CVD, waist circumference [WC]) and summarized results (Allen, 2020). A limitation was between study variations (Inder et al., 2016) and data was combined which limited examinations at the individual levels. The strength was the use of large population groups from different studies.

In a PreHTN prevalence study $(n=10,380$, normal $n=5,095$, PreHTN $n=3,735$, HTN $=1,550$ ), BMI was significantly higher in subjects with PreHTN compared to normal BP ( $28.3 \%$ vs. $26.3 \%$ ), $p<0.001$ (Gupta et al., 2010). A limitation in this study was the lack of income data to assess if BMI varied by age, gender, race, and ethnicity.

In a subset $(n=60,785)$ of a cohort study $(n=161,806)$, compared to normotensive (ref; $n=16,002$ ), BMI was a significant metabolic risk among PreHTN ( $n=23,596$; and HTN $n=21,187$ ) group ( $p<0.0001$; Hsia et al., 2007). A study limitation was exclusively female population, over half had post high school education, and income greater than $\$ 30,000$ per year (Langer et al., 2003).

Data pooled from four longitunal studies ( $n=2,893,12-18$ years of age, $\mu$ follow23.4 years) reported that compared to normotensive, PreHTN $R R$ 1.4, 95\% CI [1.0, 1.9], HTN $R R 1.9,95 \%$ CI, [1.3, 2.9], overweight $R R 2.0,95 \%$ CI [1.4-2.9], and obesity $R R$ $3.7,95 \%$ CI [2.0-7.0], $p<0.05$ predicted high risk for carotid intima-media thickness (cIMT) in young adulthood (Koskinen et al., 2019). The study's strength were repeated measures, pooled data, populations were heterogenous and the use of clinic-based risk factors plus lipid profiles in predicting future CVD risk. A limitation was income not used as a covariant to assess if CV risk differed by LMHPG.

In a cross-sectional study assessing the relationship between behavior change and BP $(n=810)$, compared to $\mu$ baseline SBP/DBP $134.9+9.6 / 84.8+4.2 \mathrm{mmHg}$; at month 18 , reduction of 12 ounces of a sugary drink per day, reduced SBP by $\beta=1.76,95 \% \mathrm{CI}$, [1.17, 2.35], $p<0.001$ and DBP $\beta=1.08,95 \%$ CI [0.69, 1.47], $p<0.001$ (Chen et al., 2010). The strength of this study were repeated measures, randomized groups, and all participants were in the JNC7 PreHTN upper limit category ( $\mathrm{SBP} \geq 130 / \mathrm{DBP} \geq 85$ ). A limitation was the lack of data associating BP change by income group.

In a study assessing the relationship between total excess free fructose (tEFF) drink consumption (e.g., pop, apple juice, and fruit drinks) and coronary heart disease
(CHD) in persons 45-59 years of age ( $n=5 \%$ of 1,230 ), reported increase odds $2.82,95 \%$ CI, [1.16, 6.84], $p<0.023$ of CHD among persons consuming $\geq 5$ drinks per day compared to $\leq 3$ times per month (ref; DeChristopher et al., 2017). This study demonstrates liquid dietary intake has a positive relationship to CHD, especially through weight gain which is associated with EBP. A limitation was the small $(n=62)$ number of cases, middle age, and CHD was not categorized by income, gender, race, and ethnicity to determine variations.

The Framingham Heart Study ( $n=7,733$ ) reported lifetime risk of developing CHD for men at age 50 with no known risk to age 95 was $51.7 \%, 95 \%$ CI $[45.8,51.3]$ and $39.2 \%, 95 \%$ CI [37.0, 41.4] for women (no p-value was reported) (Lloyd-Jones et al., 1999). In other words, compared to persons with $\geq 2$ risk factors (e.g., PreHTN), no known risk conferred lower lifetime risk of developing CHD $68 \%$ vs $5.2 \%$ in men and $50.2 \%$ vs $8.2 \%$ female. The $\mu$ BP among participants was within the PreHTN range demonstrating the importance of reducing elevated BP. A major strength was its longitudinal design, limitations were homogeneous population (NHW) and lack of income data.

To demonstrate the association of weight gain from early to middle life and adverse health, compared to persons maintaining stable weight (e.g., $\leq 51 \mathrm{bs}$; ref), weight gain of $<10$ kilograms (e.g., 20lbs) was associated with HTN Incident Rate Ratio (IRR) $1.24(n=10,099) 95 \%$ CI [1.20,1.28], $p<.001$ among Nurses' Health Study participants ( $n=92,837$ ); and HTN $\operatorname{IRR} 1.21(n=2,370), 95 \%$ CI [1.21,1.31], $p<.001$ among Health Professionals follow-up study participants ( $n=25,303$; Zheng et al., 2017).

The incident rate ratio $(I R R)$ is an approach that finds the incident of disease by those exposed (e.g., weight gain) divided by the unexposed (e.g., stable weight/weight loss) (Ayerbe et al., 2018; Delfini, 2019). The two limitations of the study were an overwhelming NHW population and high income health services professionals. Major strengths were the large population of females and males, repeated measures over a generation, and multiple health outcome assessment (e.g., CVD, cancer, etc.).

Among Latino women ( $n=331$ ), compared to normotensive ( $n=130$ ), sugary drink consumption was found to be a risk factor for $\operatorname{PreHTN}(n=148)$ OR $1.34,95 \%$, CI [1.00, 1.80], $p<.047$ (Madanat et al., 2014). Excess sugar may have contributed to higher weight and weight is associated to HTN. Study limitations were $>50 \%$ of the participants lived below poverty, $100 \%$ were female, $100 \%$ self-identified as Latina (e.g., Mexican), and BP category was not broken down by income to determine variations by age. Eating and drinking calorically dense foods and beverages have been associated with obesity (Adam \& Epel, 2007; Ibrahim et al., 2021; Junne et al., 2017) which is a metabolic risk linked to EBP.

Eating comfort food may reduce anxiety by inhibiting the release of corticotropinreleasing factor (CRF), a key hormone involved in the body's response to stress (Adam \& Epel, 2007; Shen et al., 2020). The stress comfort food association has been assessed in animal models were compared to non-stressed rodents (NSR; $n=15$ ), stressed rodents ( $n$ $=15)$ were less likely to eat commercial chow $21.90 \pm 0.61 \mathrm{vs}$ comfort food $24.05 \pm 0.77, p$ $<0.035$ (Ortolani et al., 2011).

Among a population of American women $(n=457)$ perceived stress was associated with eating non nutritious food $(r=.154, p<.001)$, and an increased lack of control $(r=.321$, $p<.001$; Groesz et al., 2012). Perceived stress was significantly correlated with emotional eating ( $r=0.26$; Shen et al., 2020)

## Cardiovascular Risk Attributed to Prehypertension

The JNC7 officially classified PreHTN as a designation identifying populations at high risk for HTN, an important CV risk factor (Chobanian et al., 2003). Empirical evidence report an association between PreHTN and CV disease risk; however, the findings are mixed. For example, in a follow-up of Framingham Heart Study participants ( $n=5,181$; Normotensive $n=1,012$; PreHTN $n=2,127$ and HTN $n=2,042$ ) reported that compared to normotensive (ref), $\operatorname{PreHTN}(n=138$ events) was associated with incident heart attack $R R 3.5,95 \%$ CI [1.05, 7.5]; PreHTN ( $n=56$ events) was not associated with all stokes compared to normotensive $(n=4) R R 2.3,95 \%$ CI [0.8, 6.3], no $p$ value was reported (Qureshi et al., 2005). Major limitations of the Framingham Heart Study was that findings are not generalizable because the population was overwhelming White, middle class, non-White identified people were not included until the 1990's (Framingham Heart Study [FHS], n.d), stroke cases were small, and a strength was the repeated measure design.

In another longitudinal study ( $n=8986 ; n=1,662$ normotensive; $n=2,708$ PreHTN and $n=4,616$ HTN) compared to normotensive (ref); PreHTN was associated heart attack, stroke, or congestive heart failure $H R 1.79,95 \% \mathrm{CI}$ [1.43, 2.24], (no p value was reported; Liszka et al., 2005).

Within the study, PreHTN upper range (SBP 130-139/DBP 85-89) resulted in a twofold risk for heart attack, heart failure and stroke $O R 2.13,95 \% \mathrm{CI}[1.64,2.76]$ (no p values reported). Several major strengths in the study were the use of NHANES data and large number of PreHTN participants 20-50 years of age (70.91\%), suggesting that CV risk may start early in life (Hardy et al., 2017; Koebnick et al., 2013; Lo et al., 2013; Nwabuo et al., 2021; Pollock et al., 2018). A study limitation was lack of income data to assess if there were variations by age, gender, race, and ethnicity. Hazard ratio $(H R)$ is a statistical approach measuring the outcome of an event between the exposed and unexposed group from study entry to endpoint (e.g., CV event; Yoshitaka et al., 2017).

Between 1999-2000 and 2011-2012, an increase in prediabetes (9.6\% vs. 21.6\%), diabetes mellitus ( $6.0 \%$ vs. $8.5 \%$ ), overweight ( $33.5 \%$ vs. $37.3 \%$ ) and obesity ( $30.6 \%$ vs. $35.2 \%$ ) occurred among persons with PreHTN (Booth et al., 2017). These factors increase risk for incident HTN. A study limitation was the lack of income as a variable and the strength was the use of NHANES data from 1999-2012.

In a meta-analysis of 29 studies $(n=1,010,858)$ assessing the association of PreHTN ranges and the risk of CV disease (e.g., stoke); 21 studies concluded both the low and high range (SBP 120-129/DBP 80-85) $R R$ 1.24, 95 \% CI [1.10, 1.39], $p<.001$; $R R 1.56,95 \%$ CI [1.36, 1.7], $p<0.001$ influences total CV risk (Guo et al., 2013a). The limitation of the meta-analysis was absence of income data, and the strength was the inclusion of $>1$ million participants from Asia, Europe, and North America.

Another meta-analysis $(n=19$ articles) $(n=468,561)$ to determine the association between PreHTN and CV disease morbidity found that compared to normotensive (ref); PreHTN increased CV risk $R R 1.55,95 \%$ CI [1.41, 1.71], $p<0.00001$ (Huang et al., 2013). Within the study, the risk remained significant at the lowest range $R R 1.21,95 \%$ CI [1.32,1.62]; and differences between upper and lower range was significant ( $p<0.02$ ). Huang et al. (2015) later assessed the risk of CHD in Asian and Western populations, where compared to optimal BP, PreHTN increased CHD $R R 1.43$, $95 \%$ CI [1.26, 1.63$], p$ $<0.001$. Compared to Asian counties $R R 1.25,95 \%$ CI [1.12, 1.38], CHD risk was higher in Western countries $R R 1.70,95 \%$ CI [1.49, 1.94]. The Population Attributed Risk (PAR) showed 8.4\% of CHD in Asian was attributed to PreHTN compared to 24.1\% in Western Countries. Strengths include multiple prospective studies from Asian and Western counties, adjustments for traditional risk factors (e.g., age, BMI, gender, etc), that were not considered in the prior analysis (Huang et al., 2013, 2014). A limitation was the study did not include income as a covariant in the analysis. This data demonstrates that PreHTN is a public health issue, especially in Western Countries such as the United States.

Clark et al. (2017) reported among ( $n=3,770$ ) Jackson Heart Study participants, at follow-up ( $n=349$ ) developed CVD, an estimated $40 \%$ of incident CVD was attributed to HTN and PreHTN. Limitations $100 \%$ NHB from the Southeast U.S., not differentiating HTN vs PreHTN, and lack of income data. Strength was repeated measures.

Han et al. $(2019 ; 29$ studies, $n=491,666)$ reported compared to normal BP, PreHTN is associated with increased risk for total CVDs $R R 1.40,95 \%$ CI [1.34, 1.46], CHD $R R 1.40,95 \%$ CI [1.28, 1.52], heart attack $R R 1.86,95 \%$ CI [1.50, 2.32], stroke $R R$ $1.66,95 \% \mathrm{CI}[1.56,1.76], p<0.05$; and population attributable risk percentage (PAR\%) for PreHTN was $13.29 \%, 24.60 \%$ and $19.15 \%$ respectfully. Within the study, a subgroup analysis reported PreHTN in the upper range $R R 1.8195 \%$ CI [1.56, 2.10] compared to the PreHTN lower range $R R 1.42,95 \%$ CI [1.29, 1.55] increased risk of total CVD, $p<$ 0.017. Study limitations include lack of income data to observe if there was an association with PreHTN; and the strength was the findings are relevant and mirror earlier meta-analysis findings demonstrating the deleterious association of PreHTN on CV health. A takeaway from these studies is PreHTN increases CVD risk and primary prevention could be a potent intervention.

A meta-analysis ( $n=20$ studies; $n=73,556$, normotensive $n=44,170$, PreHTN $n=17,314$, and $\operatorname{HTN} n=12,072$ ), found that left ventricular (LV) mass index and relative wall thickness (RWT) were greater in PreHTN compared to normotensives (standard means difference: $0.32 \pm 0.07$ and $0.30 \pm 0.07$, respectively, $p<0.001$; Cuspidi et al., 2018). The study strength was the large population inclusive of Africa (1), Asia (9), Europe (4), North (4), and South America (1). A limitation was lack of person level data which is characteristic of meta-analysis which aggregates data.

In a three (3) country comparison of HTN awareness, treatment, and control, Joffres et al. (2013) found the upper levels of PreHTN was positively associated with ischemic heart disease mortality (e.g., stroke). However, this association was limited to

England and might be attributed to treatment polices that delay initiation of pharmaceutical intervention for PreHTN (National Institute for Health and Clinical Excellence [NIHCE], 2016). A gap in this study was the lack of data comparing countries by BP categories and household income. PreHTN is a risk factor for CV disease risk via incident HTN.

Cardiovascular risk was demonstrated in a Japanese cohort ( $n=93,303$, ages 1888), where the risk of incident HTN at follow-up in persons $<50$ was greater among those with high normal DBP (80-89) HR 17.5, $95 \%$ CI [15.7, 19.6] compared to high normal SBP (130-139) HR 10.5, 95\% CI [9.6, 11.5], $p<.001$ (Kanegae et al., 2017). A study limitation was the lack of data to detect an association of PreHTN by income and whether the associations varied by gender and age; additionally, Japan is a homogenous country therefore, racial, and ethnic comparisons could not be made.

A subset of Framingham Heart Study participants ( $n=6,539$, female $n=3,479$, male $n=3,060$ ) reported that every increase in baseline DBP of 10 mmHg in populations $<50$ years old was predictive of CHD $H R 1.34,95 \% \mathrm{CI}[1.15,1.74], p<.001$ at followup compared to SBP $H R 0.95$ [ $95 \%$ CI, $0.83,1.09$ ] (Franklin et al., 2001). A limitation was a $100 \%$ NHW cohort and the strength was the study's repeated measures.

In another Coronary Artery Risk Development Study (CARDIA) ( $n=5,111$; sub analysis $n=3,429$ ), a biracial cohort, ages 18-30, concluded at Year 10, that each 10 mmHg higher level of baseline SBP and DBP was associated with reduced kidney functioning as measured by glomerular filtration rate (GFR) eGFR of $-0.09,95 \% \mathrm{CI}$, $[-0.13,-0.06]$ and $-0.07,95 \%$ CI $[-0.12,-0.03] \mathrm{mL} / \mathrm{min} / 1.73 \mathrm{~m}$ per year, the authors did
not report a p - value but did cite statistical significance (Ku et al., 2018). A strength of the study was a biracial cohort of young adults with no comorbidities and repeated measures. A limitation was the outcome was not aligned by BP category, gender, income, age, race.

In the Strong Heart Study, a cohort of Native American tribes $(n=3,563$, w/diabetes $n=1,872$, w/o diabetes $n=1,619$ ) and no CHD at baseline, reported at year 12.2, among persons w/diabetes each 5 mmHg increase in SBP predicted CHD $H R$ 1.05, $95 \% \mathrm{CI}[1.02,1.07], p<0.001$, the same risk extended to persons w/o diabetes HR 1.04, $95 \%$ CI [1.00, 1.08], $p<0.049$ (Xu et al., 2012). Study strengths include Native American population from 4 states, all participants were free of CHD at baseline and SBP was within the PreHTN range (120-139). A strong conclusion can be drawn that SBP in the PreHTN range increases CHD risk in groups with and without metabolic disorders. Limitation was lack of income data to align with CHD risk.

In another CARDIA subanalysis ( $n=3,560$ ), compared to normotensive ( $n=$ 2,925), PreHTN ( $n=635$ ) with high SBP prior to age 35 and scores $>30 \mathrm{mmHg}$-years were more likely to have Coronary Calcium ( $38 \%$ vs. $15 \%$, $p<0.001$ ); scores greater than 10 mmHg -years ( $27 \%$ vs. $10 \%$; $p<0.001$ ), and a scores greater than $100(10 \%$ vs. $3 \% ; \mathrm{p}<0.001$ (Pletcher et al., 2008). Coronary Calcium is calcified plaque in the coronary arteries and a predictor of coronary heart disease (Pletcher et al., 2008).

Even though the above studies demonstrated an association between PreHTN and CV risk, PreHTN has not been associated with all-cause mortality. For example, in a meta-analysis ( $n=13$ studies, $n=870,678$ participants) an association between PreHTN
and all-cause mortality was not found (Guo et al., 2013b). However, within the study, PreHTN did present a CV mortality risk across levels (e.g., lower, and upper range) $R R$ $1.32,95 \%$ CI $[1.16,1.50], p<0.001$, making this classification worthy of close attention. This conclusion was shared by Huang et al. (2014) ( $n=20$ studies, 1,129,098 participants), where there was a risk of CV mortality attributed to PreHTN. Within the study, the upper range $R R 1.28,95 \% \mathrm{CI}[1.16,1.50]$ compared to the lower range RR $1.08,95 \% \mathrm{CI}[0.98,1.18], p<.0001$, made a greater impact. The limitation with the above meta-analysis was the lack of income data to compare an association.

In an analysis on the impact of PreHTN and Diabetes Mellitus (DM) on all-cause mortality and cardiovascular mortality ( $n=23,622$, non-White $n=13,235$, White $n=$ 10,387 ), in a population without CVD at baseline, compared to persons without PreHTN and $\mathrm{DM}(n=12,272)$, CV mortality $H R 1.70,95 \% \mathrm{CI}[0.88,3.27], p<0.001$ for persons with PreHTN/DM ( $\mathrm{n}=2,752$ ) (Huang et al., 2020). A limitation was lack of income data and dichotomized racial category. The strength was the use of NHANES dataset.

To further illustrate the deleterious effects of PreHTN relative to CV risk, Park et al. (2020) $(n=544)$ reported carotid artery characteristics (CAC) of PreHTN negatively changes organ structures, the $O R$ of IMTmax, LDmax, LDmin, peak-systolic FV (PFV), end-diastolic FV (EFV), PFV/LDmin, EFV/LDmax, compliance, and $\beta$ - stiffness were 4.20 ( $p<0.001$ ), $2.70(p<0.01), 3.52(p<0.001), 2.41(p<0.01), 3.06(p<0.001)$, $3.56(p<0.001), 3.29(p<0.001), 2.02(p<0.01)$, and $1.84(p<0.05)$ times higher than normotensive. Strength was the use of JNC7 BP criterion and participants (18-85 years of age). Limitations were lack of income data, and the study took place in South Korea.

Among youth ages 11-18 $(n=303)$, there was a graded increase in LVMI by normotensive (L), PreHTN (M) and HTN (H) (L=31.2 g/m2.7, M=34.2 g/m2.7, and $\mathrm{H}=34.9 \mathrm{~g} / \mathrm{m}$ ), $p<0.01$ (Urbina et al., 2019). This demonstrates abnormal cardiac structural changes have been identified in non HTN groups. Study strengths include multi-specialty clinic sites and recruiting PreHTN kids. Limitation includes lack of family income to assess structural change by BP level and primarily White population. The above studies provide an insight on how PreHTN might place younger populations at risk for CV disease.

## Race and Ethnicity

In the Multi-Ethnic Study of Atherosclerosis (MESA), ages 45-84, subset ( $n=3,146$, NHW $n=1,348$, NHB $n=630$, Chinese $n=424$, and Latino $n=734$ ), at Year 5, NHB had the highest HTN incident rate ratio $I R R 1.65,95 \%$ CI [1.39, 1.96] compared to NHW $\operatorname{IRR}$ (ref), Hispanic $\operatorname{IRR}$ of $1.29,95 \%$ CI [1.06, 1.57]; and Chinese $I R R 1.05$, $95 \% \mathrm{CI}[0.81,1.35]$, ( p values were not provided) in progressing from normotensive to incident HTN (Carson et al., 2011). Within the study, NHB represented 20\% of participants and carried the greatest HTN disease burden. Three study limitations were lack of specific Hispanic groups (e.g., Mexican, Puerto Rican, Dominican, etc.); the absence of income data to assess if HTN varied by gender, age, race, ethnicity, and the population was middle age. Study strengths include multiple sites and repeated measures.

PreHTN prevalence in the U.S. by racial ethnic group tend to vary by study. According to Glasser et al. (2011) compared to NHW ( $n=3780 ; 54.1 \%$ ), NHB ( $n$ $=1773 ; 62.9 \%$ ) have PreHTN burden. Within the NHB PreHTN group, $65.2 \%$ reported
annual household incomes $\$ 20,000$ compared to their NHW peers (58.6\%). However, in the model, income as a covariant predicted PreHTN among NHW OR $1.26,95 \%$, CI [1.01,1.56] compared to NHB OR $0.95,95 \% \mathrm{CI}[0.69,1.31], p<0.001$. A limitation of this study was that it only assessed a middle age population $\geq 45$ from the southeastern part of the U.S known as the Stroke Belt and Stroke Buckle because of the excess rates of deaths attributed to stokes above the national rate (Lanska \& Kuller, 1995).

In the CARDIA study ( $n=4,480$, NHB $n=2,473$, NHW $n=2,407$ ) at the 25 -year follow-up, 210 CV events occurred, of this number 140 were NHB ( 2.85 per 1,000 person-years) compared to NHW ( $n=70 ; 1.37$ per 1,000 person-years); at baseline $1 S D$ increase in SBP (11mmHg) in NHB favored an 32\% increase risk in CV events $H R$ 1.32, $95 \%$ CI $[1.09,1.61]$, compared to NHW $H R$ of 0.82 [0.57-1.18] 95\%, CI [0.88,1.53], $p<$ .05 ; whereas $1 S D(10 \mathrm{~mm})$ increase in DBP in NHW favored a $74 \%$ increase risk in CV event $H R 1.74,95 \%$ CI [1.21, 2.50], compared to NHB $H R 1.05,95 \%$ CI [0.82,1.18], $p .<01$ (Yano et al., 2017). A limitation was lack of income data by BP status, a strength was the repeated measures assessing adverse health outcomes.

In a biracial study ( $n=176$, NHB $n=76$, and NHW $n=100$ ) examining the relationship between discrimination and stress, $\mu$ SBP (123)/ DBP (73.92) was higher in NHB compared to NHW SBP (117.85)/DBP (68.87), $p<.05$ (Tomfohur et al., 2016). PreHTN prevalence in the NHB community might be a function of the gene (e.g., skin color, hair texture, etc.), social environment (e.g., linage) interaction as it relates to generational, and everyday experiences (Curry, 2020; Diez-Roux, 2011; Glasser et al., 2011; Selassie et al., 2011).

These experiences are expressed via accrued disadvantages from the legacy of Chattel Slavery and Jim/Jane Crow (Collins \& Wanamaker, 2017) and may better explain between group PreHTN disease disparities (Diez-Roux, 2011; Bailey et al., 2017b).

This data may shed light on RQ\#2 because the association between poverty group and PreHTN is controlled by race and ethnicity. Similar conclusions relating to race/ethnicity and its association with BP levels have been inferred in the literature (Boulter et al., 2015; Hardy et al., 2017; Hicken et al., 2014; Howard et al., 2013; Krieger \& Sidney, 1996).

For example, Lee et al. (2016; female $n=96$; male $n=28$ ) found a marginally significant effect on $\operatorname{DBP}(F=3.31, d f=2, p \leq .05)$ among black college students who scored high on Race Related Stress (RRS) at baseline and follow up 80.38 (9.44) compared to black students who scored low 75.83 (7.44) and moderate 78 (6.18). Although RRS was not directly implicated in elevated SBP, there was a main effect of depression on $\operatorname{SBP}(F=3.87, d f=2, p<.03$; Lee et al., 2016, p. 586). Within this study, $41.9 \%$ of the population were PreHTN compared to $22.9 \%$ normotensive. A study limitation was the absence of income as a covariant to better detect an association with PreHTN and if that association varied by age and gender.

Empirical evidence report race and or ethnic group membership might be a predictor of PreHTN. For example, in a cross-sectional study ( $n=10,380$ ) NHB (45.8\%) tend to have less normal BP compared to NHW (51.3\%), Mexican American (59.4\%), and other (55.2\%); which increased the prevalence percentage of PreHTN among NHB
(38.9\%) compared to NHW (36.9\%); Mexican Americans (32.2\%); and other (33\%; Gupta et al., 2010).

In a trend for PreHTN and HTN risk factor study, after adjusting for age and gender, NHB showed higher PreHTN prevalence compared to their NHW and other racial peers (Booth et al., 2017). A limitation was lack of income to determine an association. In an HTN prevalence study of Mexican Americans ( $n=2,780$ ), the prevalence of PreHTN was $33 \%$ ( $\mathrm{n}=921$; Bersamin et al., 2009). Within this study, income was not a covariant, however, Mexican Americans are the largest Hispanic/Latino group (63.4\%) in the United States (USCB, 2011) and the rate of poverty Hispanic (any race) is (17\%), higher than White (10.1), NHW (8.2\%) and Asian (8.1\%), and lower than (19.5\%) NHB (USCB, 2021a). Hispanic/Latinos as an ethnic group which include Mexican who overwhelming self-identify as White (Golash-Boza \& Darity, 2008; USCB, 2011). Additionally, White identity among Hispanic/Latinos could potentially distort BP disparities within the U.S Hispanic/Latino population.

Variations of targeted organ damage associated with EBP among Hispanic subgroups ( $n=1,437$, Dominican $n=175$; Other $n=260$; Mexican $n=801$; and Puerto Rican (PR; $n=201$ ), found that PreHTN was similar across groups; however, Dominicans had higher DBP 74.9 compared to PR 72.3; Other 71.6, and Mexican 70.7 ( $p$ $<.05$; Allison et al., 2008). Compared to other Hispanic subgroups, the prevalence of HTN was highest among Dominicans $52.9 \%$ compared to Mexicans 37.6 \%, PR 43.3\% , and Other $42.1 \%, p<0.05$. Study limitations include a middle age population ( $\mu 60.9$ ), absence of income data to determine variations by age, gender, and subgroup.

Strength includes different groups versus aggregating all persons of Spanish speaking lineage into one group.

Being Hispanic Black with low perceived racism was associated with non-BP dipping ( $n=21 ; 83 \%$ ) compared to White Hispanics $\left(n=19,54 \%, \chi^{2}=5.2, p<0.02\right.$; Rodriguez et al., 2016). Of the 180 participants, $41 \%$ self-identified as White, $34.4 \%$ Black, $1 / 4$ did not self-identify and $78 \%$ of the population were Dominican. A study limitation was $\mu 67$ years of age and family income could not be determined by dipping status and race identification. Within the Hispanic/Latino community, Colorism is practiced, and Dominicans tend to be darker with West African features (Chavez-Dueñas et al., 2014). Colorism is defined as within group discrimination based on skin color, hair texture, and other features that are non-Eurocentric (Quiros \& Dawson, 2013).

In a prospective PreHTN cohort study of adults $(n=18,865)$, NHB $(5,733)$ and NHW (13,132); NHB with PreHTN converted to HTN one year earlier following diagnosis compared to their NHW peers (626 days versus 991 days, $p<.001$; Selassie et al., 2011). Within the study, compared to normal BP ( $n=5,196 ; 27.5 \%$ ) and lower range PreHTN ( $n=5134 ; 27.2 \%$ ), the upper range of PreHTN (SBP 130-139/DBP 80-89; $n=$ 8,535; 45.24\%) significantly predicted incident HTN $H R$ of $2.02,95 \%$ CI [1.94, 2.11], $p<0.001$ regardless of racial group membership. This study lacked income data to ascertain an association of PreHTN by covariates.

According to Pletcher et al. $(2008 ; n=3560)$, of the 638 participants who developed PreHTN prior to 35 years of age, $61 \%$ were NHB compared to NHW ( $p<$ 0.001 ). There was a noticeable difference by annual income $<\$ 25,000$; compared to
normotensive population (14\%), 21\% of the PreHTN population reported incomes $<$ $\$ 25,000, p<0.01$. This suggests a portion of the PreHTN population have fewer tangible resources. The study did not report if the PreHTN group differed by income, age, gender, race, and ethnicity; however, over $60 \%$ of the PreHTN population was NHB, indicating a probable association by race.

In a subset of the Bogalusa Heart Study (BHS) participants ( $n=1,379,65 \%$ NHW and $35 \%$ NHB), compared to NHW, NHB had a higher prevalence of PreHTN ( $27 \%$ vs $29 \%$ ), $p<0.001$, and NHB women had a significantly higher prevalence compared to their NHW peers ( $25 \%$ vs $20 \%$ ), $p<0.05$ (Toprak et al., 2009). Within the study, income was not used to assess PreHTN by age, gender, race, or ethnicity; however, in another study using a similar BHS subset ( $n=1,266$, NHB $n=325$, and NHW $n=9$ 42), $63 \%$ of NHB had household incomes of $\leq \$ 15,000$ compared to $23 \%$ NHW (Deshmukh-Taskar et al., 2007).

In a cohort of Western Alaskan Natives $(n=4,562)$, the prevalence of PreHTN was $28 \%(n=1,300$; Jolly et al., 2015). A limitation was the lack of data comparing tribes by BP category, age, gender, and income. A strength of the study was the use of different Western Alaskan tribal groups.

A pilot study $(n=338)$ assessing CV disease risk among Parsi Zoroastrians, a homogenous subset of Asian Indians, found PreHTN prevalence at 46\% compared to 16\% for HTN, respectfully (Vazquez-Vidal et al., 2016). Study strengths include a subset of Asian Indians who marry within their tribe and a limitation is the lack of data to evaluate BP category by income.

Within the Asian American community, Asian Indians tend to have higher median household income ( $\$ 126,705$; USCB, 2020b) compared to the overall median Asian income ( $\$ 94,903$; USCB, 2021a). Thus, a case can be made that PreHTN varies across income levels and financial resources may not attenuate CV risk.

Among a population of normotensive Hispanic women, at the three-year followup, $27.3 \%$ were identified as PreHTN (Zambrana et al., 2016). A limitation in the study was the lack of financial information; however, baseline data from the Women's Health Initiative from which the subset was analyzed reported $<\$ 20,000$ per year family income among Hispanic and American Indians (Langer et al., 2003). This figure was opposite to NHW women who were 1.9 times more likely than American Indian and Hispanic, and 1.6 times more likely than NHB women to report family income above $\$ 50,000$.

PreHTN prevalence tend to be high among racial ethnic groups; however, in a study ( $n=23,622$ ) assessing whether pre-diabetes and or PreHTN increased the risk of all-cause and cardiovascular mortality reported higher risk of cardiovascular death for White $H R 1.47,95 \%$ CI [1.12, 1.94] compared to non-White population $H R 0.98,95 \% \mathrm{CI}$ [0.74, 1.32], ( $p<0.001$; Huang et al., 2020). This study demonstrates that specific metabolic risk places Whites at increased mortality. Study strengths include the use of NHANES dataset, the independent variables, and high-risk endpoints. The limitations were lack of income data and not specifying non-White groups (e.g., Chinese, Mexican American, NHB, etc.). Not disaggregating the non-White groups may have obscured between group outliers.

In the Enhanced Coal Workers, Health Surveillance Program (ECWHSP), an intervention providing free mobile medical services to Coal Miners, a blue-collar cohort ( $n=1402$ ), reported the prevalence of PreHTN at $42 \%$ ( $n=644$; Casey et al., 2017). Within the study, compared to females, males had higher prevalence ( $48 \%$ vs. $35 \%$ ), $p<.0244$. Ages $15-44$ had the highest prevalence $55 \%(n=259)$ compared to $>65(33 \%)$, $p<.05$. The challenge with this study was most participants were NHW (93\%), male (94\%), and there was no income data to assess an association with PreHTN. A conclusion can be made that the younger group presenting with PreHTN is at risk for targeted organ damage (Ku et al., 2018).

In a pediatric epidemiologic study, PreHTN was greater in non-White identified communities $(\mathrm{A} / \mathrm{PI}=2,952$, Hispanic $=4,372, \mathrm{NHB}=2,413)$ relative to $\mathrm{NWH}(8,438)$ and Other/Unknown (OU; 7,195) peers, all $p<.001$ (Lo et al., 2013). Limitations were missing data for OU group that might lead to biased estimates (Johnson et al., 2013), and lack of income data to align with PreHTN estimates. A study strength was the inclusion of different racial and ethnic community-based participants.

On the other hand, Koebnick et al. $(2013 ; n=74,501)$ reported higher PreHTN among NHW (34.9\%) compared to NHB (32.7\%), Other/Unknown (32.6\%), Hispanic (29.6\%) and A/PI (28.6\%). Within the study, Hispanic children represented $50.4 \%$ of the study population ( $n=237,248$ ). A strength was the use of repeated BP measures over a 36-month period. A limitation was lack of BP category associated with income. Within the latter study obesity was cited as a contributor for PreHTN, whereas Lo et al. (2013) reported being overweight (90th-94th percentile).

Hardy et al. (2017) reported estimated annual net transition probabilities in White American (WA) boys by age 8 (ANTP) from PreHTN to HTN was $1.2 \%, 95 \%$ CI [0.7\%$1.8 \%$ ], compared to African American (AA) boys $1.8 \%, 95 \%$ CI [1.0\%-2.65]. Within the study, ANTP increased into early adulthood among AA females $2.3 \%, 95 \%$ CI [1.9\%2.7\%] versus their race matched peers who experienced transitions to HTN later in their adult life. This information might account for the higher prevalence of HTN among NHB compared to their race and ethnically matched peers. Without timely interventions, this demographic group continues to be at risk for adverse CV outcomes. A limitation in the study was lack of income data and strength include using NHANES data.

Empirical evidence reports after adjusting for covariables, NHB transition quicker from PreHTN to HTN $H R 1.35,95 \%$ CI [1.30, 1.40], $p<0.001$ compared to their NHW peers (Selassie et al. 2011). Cardiovascular risk factors have been found to increase transition from PreHTN to HTN. For example, in a study assessing CV and metabolic predictors of progression from PreHTN to HTN among Native Americans ( $n=2,894$ ), at the 4-year follow-up, higher baseline SBP (per 10 mmHg ) independently increased the odds of incident HTN in persons without Diabetes Mellitus (DM) OR 1.60, 95\% CI [1.30, 2.00] and those with DM OR 2.73, 95\% CI [1.77, 4.21] both $p<.0001$ (De Marco et al., 2009). A major limitation is the lack of data to assess an association between BP and income. This study established that DM, a metabolic predictor, increases the risk of incident HTN, a cardiovascular risk.

Arterial stiffness, a marker for CV risk, usually occurs at older age, has been shown to occur earlier in life among NHB compared to NHW. For example, in a cross-
sectional analysis participants 20-70 years of age ( $n=855$, NHB $n=385$; NHW $n=470$ ), assessing mean Pulse Wave Velocity (PWV), a measure of arterial stiffness, was greater among NHB compared to NHW PWV $7.4 \pm 1.6$ vs $7.1 \pm 1.6, p<0.001$ (Morris et al., 2013). Income was not assessed, however, within the study, NHB tended to be younger and less likely to be college graduates. Clinical measures such as BP (Tomfohur et al., 2016; Yano et al., 2017); impaired vascular function (Morris et al., 2013); BMI (Lee et al., 2016) and Type 2 Diabetes (Bancks et al., 2017; CDC, 2017b) have limitations in terms of primary prevention, and early detection of PreHTN could potentially delay deterioration of cardiovascular health.

Racism is a chronic stress (Clark et al., 1999) and there is data citing perceived racial discrimination is associated with EBP (Dolezsar et al., 2014; Michaels et al., 2019) and other adverse health outcomes (Carter et al., 2019; Clay et al., 2018; Paradies et al., 2015) contributing to health disparities. The higher prevalence of PreHTN in nondominant groups compared to dominant groups fail to recognize the role stress may play relative in the context of historical trauma, accrued disadvantages, and loss by marginalized groups (Brave Heart et al., 2011; Curry, 2020; Hadden et al., 2016; McCutcheon, 2019) and may contribute to differential PreHTN outcomes in socially marginalized communities. This is relevant to my research because race and ethnicity are control variables. PreHTN is a risk factor for HTN and HTN has been linked to chronic stress (Ford et al., 2016; Sims et al., 2020).

Notwithstanding of education and higher income (Assari 2018); a group's racial classification is the most powerful determinant of its members wealth status (Bhutta et al., 2020), income (USCB, 2021a) BP (Booth et al., 2017; Glasser et al., 2011; Krieger \& Sidney, 1996; Virani et al., 2020) and other social measures (BLS, 2020a, 2020b; Collins \& Wanamaker, 2017; Houle \& Addo, 2019; FBP, 2019).

For example, In a study assessing adverse health outcomes (e.g., HTN) among individuals with annual incomes $>\$ 175,000(n=3,047$, NHW $n=2,391$, NHB $n=173$, Hispanic $n=189$, Asian $n=294$ ), NHB self-reported odds of HTN was $2.8,95 \%$ CI, [1.7, 4.7] compared to NHW(ref), Hispanic $O R 0.7,95 \%$ CI [0.4,1.2], and Asian $O R$ $1.00,95 \%$ CI [0.4,1.2], no p value (npv) cited (Wilson et al., 2017). A study limitation was not differentiating between the level of EBP and if BP might have been at the PreHTN level. A strength was stratifying health outcomes by race and ethnic identity as well as showing the small number of NHB participants making $>\$ 175,000$, indicating higher income may rest on social, political, and institutional factors outside group control and less likely to buffer against adverse BP.

A similar conclusion was found in a longitudinal study $(n=2,270)$, examining the interactions of race and socioeconomic status (SES) on health inequities, where higher SES (HSES) NHB $(n=705)$ were at increased risk for a CV event due to mean intimal medial thickness, a CV risk for stroke, 0.71 (0.12) compared to lower SES (LSES) NHB ( $\mathrm{n}=502$ ) $0.69(0.14)$, LSES NHW $(n=287) 0.67(0.13)$ and HSES NHW $0.67(0.13) p<$ 0.05 (Wendell et al., 2017). Higher SES NHB had the highest prevalence of HTN in this study (47\%) compared to their higher SES NHW peers (37.4\%).

A challenge with this study is not having data on the PreHTN population and income category assigned by outcome such as low compared to high poverty group to estimate if elevated BP outcome varied by age, gender, race, and ethnicity.

Higher income among NHB $(n=3,570)$ appear to have a less positive effect against chronic medical conditions compared to NHW ( $n=891$ ) $\beta-0.070 .10-0.04, p<$ $0.001 ; \beta-0.04-0.06-0.01, p<0.01$ (Assari, 2018). A study limitation was not aligning income by medical condition.

Pearson and Geronimus (2011; $n=8,566$; Black $n=537$, White Jewish $n=5,186$ and White $n=2,881$ ) reported that social identification of White advantage and its relationship to self-rated health has limitations among ethnic White Jews (WJ) in the U.S. Although WJ and NHW reported similar frequencies of excellent self-rated health relative to NHB; when adjusting for education and income; compared to NHW (ref), White Jews self-rated health $O R 0.68,95 \%$ CI $[0.59,0.79]$ was close to NHB $O R 0.71$, $95 \% \mathrm{CI}[0.55,0.91], p<.001$. A strength was the use of ethnic Whites, and the limitation was lack of BP measures.

Poor self-rated health (SRH) has been associated with mortality, this was demonstrated in study ( $n=8,251, \geq 16$ years of age and older) that anonymously linked census files to death and emigration records coving 30 years. Compared to excellent SRH in men (ref; $n=3,662$, deaths $=1,218$; ref) very poor self-rated health increased mortality HR $2.85,95 \% \mathrm{CI}[1.25,6.51], p<0.05$; and among women $(n=4,297$, deaths $=1,118)$ excellent SRH (ref) compared very poor $H R 1.30,95 \%$ CI [0.18, 9.35], p. $<0.05$ (Bopp et
al., 2012). Within the study there was a moderate increase in mortality for every 1 mmHg increase in BP for both genders $\mathrm{HR} 1.01,95 \% \mathrm{CI}[1.01,1.01], p<0.05$. Study strengths include 30-year follow-up, participants within the PreHTN range at baseline, almost half reported intermediate education, and marriage. Limitations include homogeneous population and study was implemented in Switzerland. The above studies citing education, income, and marriage status have limits in buffering against adverse health and mortality.

Arab Americans who are considered ethnic White (USCB, 2021b) reported more depressive symptoms 7.6 compared to NHB (6.6) and NHW (5.2), $p<.05$ (Ajrouch et al., 2018). Study strengths include a group of ethnic whites, NHB and NHW population from a midwestern state. Limitations include lack of bloop pressure and income data. Depression has been linked to EBP. For example, Still et al. (2020) identified a small correlation between DBP and depressive symptoms ( $r=.16, p<.03$ ). Race and ethnicity might be a reliable predictor of self-rated health, adverse BPs, CV risk and may contribute to PreHTN disparities in the U.S. This makes minimizing PreHTN and delaying incident HTN a national priority.

## Age

Age is a useful predictor of incident HTN and cardiovascular outcomes because SBP increases with age (Ostchega et al., 2020; Rapsomaniki et al., 2014) which might account for higher HTN prevalence in persons 60 years of age and over. A central belief attributing greater SBP with aging is arterial stiffness (Alghatrif et al., 2017).

This was demonstrated in the Baltimore Longitudinal Study on Aging (BLSA; $n=449$, male $n=201$; female $n=248$ ), 306 were normotensive at baseline, at Year 5, incident HTN $(n=105)$ was moderately associated with increased SBP manifested via Pulse Wave Velocity, a marker for central arterial stiffness, $H R 1.10,95 \%$ CI [1.00, 1.30], $p<0.03$ (Najjar et al., 2008).

Arterial stiffness attributed to frail aging was reported among a subset of Framingham Heart Study participants ( $n=2,171, \geq 60$ ), where the adjusted least square means between 67, 70 and 73 years of age were $9.9,95 \% \mathrm{CI}[9.8,10.1], 10.2,95 \% \mathrm{CI}$ [10.0, 10.3], and $10.7 \mathrm{~m} / \mathrm{s} 95 \%$, CI [10.4,10.9], $p<0.001$ (Orkaby et al., 2019). Adjusted least square means (ALSM) is used to measure the true relationship of variables among two or more groups, when discounting for covariates (e.g., age, gender, race, etc.). This method has been used in randomized clinical trials (Sabatine et al., 2017), and prospective studies (Hruby \& Jacques, 2019).

For example, in a double-blind study evaluating the effectiveness of a cholesterol lowering medication ( $n=27,564$ ), at week 48 , the ALSM percentage reduction in LDL cholesterol levels with medication ( $n=13,784$ ) was $59 \%, 95 \% \mathrm{CI}[58,60], p<0.001$, from 92 mg per deciliter to 30 mg per deciliter, compared to the placebo group (Sabatine et al., 2017). The strength of the study was the large multi-country population, and the limitations were a large NHW participants ( $>80 \%$ ), predominately male, and lack of income data. ALSM was used in the Framingham Offspring Cohort Study ( $n=2,061$ ), to assess the association of protein intake and inflammatory markers; where higher intake of dietary proteins, mainly plant based was associated with lower inflammatory markers
$0.77 \pm 0.17$ in quartile 1 compared to quartile $40.31 \pm 0.19 ; p<0.02$ (Hruby \& Jacques, 2019).

There is evidence that arterial stiffness might be intergenerationally transmitted. This was demonstrated in normotensive third generation Framingham Heart Study cohort ( $n=1,564$ ); where compared to offspring of parents without HTN $(n=468)$, those with at least one HTN parent $(n=715)$ had a mean arterial pressure (MAP) of $2.6(0.5)$, and 2 parents with HTN ( $n=381$ ) MAP was 3.6 ( 0.7 ), $p<0.001$ (Andersson et al., 2016).

Mean arterial pressure (MAP) is the average of SBP and DBP divided by 3 at a single clinic encounter (Andersson et al., 2016), and is a marker for cardiovascular risk (Maggio et al., 2018). A limitation of the study was that MAP was not broken down by age, gender, race, ethnicity, and income. A strength was the use of longitudinal third generation data of disease free young to middle-aged adults to evaluate the role of intergenerational CV risk transmission. A limitation was the homogenous population which cannot be generalized beyond NHW.

Age was moderately associated with PreHTN in a southeast Asian primary care population $a O R 1.06,95 \% \mathrm{CI}[1.02,1.11], p<0.007$ (Rafan et al., 2018). A strength was the inclusion of household income status, and the limitation was the study took place in Malaysia where estimated per capita income is $\$ 26,400$ (CIA, n.d.c).

PreHTN increases with age, in a prevalence study among disease free population ( $n=10,380$ ), within the PreHTN group ( $n=3,735$ ), the highest prevalence was observed among ages 60-69 (44.2\%) compared to 20-39 (31.2\%; Gupta et al., 2010).

Middle age appears to increase PreHTN prevalence, this was demonstrated in a subset of the Women's Health Initiative participants ( $n=60,785$ ), where compared to the normal group ( $n=16,002$ ), the PreHTN group $(n=23,596)$ was slightly older $62.6+6.8$ vs $60.7+6.8$ and when factors such as $\mathrm{BMI}, \mathrm{T} 2 \mathrm{D}$, and hypercholesterolemia were combined, the results were significant ( $p<0.0001$; Hsia et al., 2007).

Conversely, in a subset of San Antonio Heart Study participants ( $n=2,767,25-65$ years of age), younger age (25-49) compared to older age ( $\geq 50$ ) was associated with PreHTN $p<0.002$; and PreHTN was primarily attributed to high blood sugar (Mullican et al., 2009). A study strength was the inclusion of Mexican Americans and limitation was the lack of income data to assess PreHTN by age, gender, race, and ethnicity.

According to the clinical practice guideline for screening and management of high BP in children and adolescence, EBP for this group is defined as $\mathrm{SBP} \leq 120-129$ / DBP $\leq 80$ (Flynn et al., 2017). Investigations have demonstrated that EBP may appear earlier in life as evidenced in a pediatric observational study ( $n=199,513$, ages 3-17) reporting $12.7 \%$ PreHTN prevalence ( $n=25,370$; Lo et al., 2013). Study limitation was PreHTN measures were not repeated and lack of parental income to align with estimated prevalence. The strength was the use of large health systems records that include multiethnic pediatric populations.

Koebnick et al. (2013) reported that PreHTN prevalence among children 6-17 ( $n=237,238$ ) was $31.4 \%(n=74,501)$, an estimate close to $36.3 \%$ for disease free adults $\geq 20$ years of age (Gupta et al., 2010).

A limitation with Koebnick et al. (2013) was the overwhelming large Hispanic population and lack of income status associated with PreHTN. A strength of the study was the repeated measures over the assessment period. These study provided insight that PreHTN in the U.S. adolescent population is a problem and must be continuously evaluated because this population might be at risk for earlier CVD and targeted organ damage.

Continuous monitoring of BP was demonstrated in a trend study evaluating dyslipidemia (e.g., elevated fats in the blood) $(n=1,482)$ and EBP $(n=1,665)$ in adolescence 8 to 17 from 1999-2000 versus 2011-2012; where 9.4\% of the EBP group had borderline high BP, with a non-significant change in EBP from 1999-2000 and 2011$20127.6 \%, 95 \%$ CI [5.8, 9.8] vs $9.4 \%, 95 \%$ CI, [7.2, 11.9]; $p=.90$ (Kit et al., 2015). Within this study, over $1 / 3$ of the total EBP group was overweight $(n=267)$ or obese $(n=347)$ and although there were no significant changes between assessments, these kids might be at elevated CVD risk later in life. A major design strength was the use of NHANES data, the limitation was lack of income to evaluate an association controlling for age, gender, race, and ethnicity.

In a 1999-2008 NHANES analysis of adolescence 12-13 years of age (ref) and 14-19 years of age, $(n=3,383)$; found that compared to youth with normal BMI $(\geq 5-<85$ percentile) ( $n=1,921$ ), youth with $\mathrm{BMI}(\geq 85-<95$ percentile) $(n=299)$ presented with 2 CV risk factors (e.g., PreHTN and HTN), $p<.05$ (May et al., 2012).

Limitation include lack of income to determine if there was an association by age, gender, race, and ethnicity. Strength was the use of NHANES database that targeted population 12-19 years of age.

Moderate to vigorous physical activity (MVPA) (e.g., 60 minutes daily) has been promoted as a strategy to stabilize BP via weight control (Poitras et al., 2016). However, there appear to be mixed conclusions regarding the relationship between physically active adolescence, BMI, and EBP. This was shown in an analysis of preparticipation physical examinations (PPE; $n=2,678$ ) among student athletics 10-20 years of age, from 20092012, PreHTN prevalence in 2009 was $15 \%(n=112) ; 201010.6 \%(n=60) ; 201114.6 \%$ ( $n=96$ ); and 2012 12.8\% ( $n=90$; Kropa et al., 2016). One (1) in five (5) of the total population was overweight and $28 \%$ were obese, resulting in an association with EBP $p<0.0001$; but not PreHTN. Major strength was the large sample size, multiple assessment periods and geographic location (Northeast U.S). Limitations include race and ethnicity data was only posted for $2012,<15 \%$ of population was Hispanics, Asian, and other. PreHTN and EBP was not measured by race, ethnicity, and lack of parental income as a covariant.

EBP was also observed among student athletics $(n=7,705), 14-18$ years of age, 2009-2013, $\mu$ SBP was in the PreHTN range $122.7 \pm 13.1$, and $>40 \%$ of this group was overweight (20.3\%) or obese (23.5\%) compared to normotensive (56.2\%; Stiefel et al., 2016). Within the study, predictors of elevated BP were male gender 1.24, $95 \%$ CI [1.10, 1.38], number of sports ( 1 vs $>3$ ) 1.27 [1.09, 1.64], $p<0.05$ and NHB $1.2395 \% \mathrm{CI}$ [1.11-1.35], $p<0.01$.

The study strengths include $>1$ sport measured, geographic region (Southeast), and large sample size. Limitations identified $>69 \%$ male, smaller ethnic samples Hispanic (1.2\%), Asian (0.5\%), lack of PreHTN measures, and income data to determine if elevated BP varied by age, race, and ethnicity. It is worth noting that Kropa et al. (2016) reported 71\% of the 2012 population was NHB. Stiefel et al. (2016) reported $66.6 \%$ of the total student population was NHB. A conclusion could be made that a proportion of PreHTN and HTN cases within both studies were NHB, a group with greater BP throughout life and moderate to vigorous activities may not buffer CV risk.

In a subset from the Cardiovascular Risk in Young Finns, a longitudinal population-based study ( $n=1,635$ ), reported that childhood cardiovascular risk factors BMI, SBP, blood glucose, vegetable consumption, smoking and physical activity ( $p \leq .023$ ) was associated with systemic vascular resistance in adulthood (Kähönen et al., 2021). Study strengths include initial data collection in 1980 and follow-up in 2007. The limitations include the study conducted in Finland, homogeneous population and income was not used as a variable.

As with adults (Park et al., 2020), targeted organ damage has been shown in youth with elevated BP (Urbina et al., 2019). High normal BP at a younger age has been associated with later in life cardiovascular risk (Kähönen et al., 2021).

The PreHTN and HTN adolescent and pediatric studies (Kit et al., 2015; Koebnick et al., 2013; Kropa et al., 2016; Lo et al., 2013; Stiefel et al., 2016) did not explore the association between parental income and PreHTN and if it varied by age, gender, race, and ethnicity.

In a study that estimated annual net transition probabilities from ideal BP and PreHTN (8-80 years of age; $n=17,747$, AA $(n=4,973)$; WA $(n=8,886)$; MA $(n=3,888)$ reported as early as 8 years of age, males had a lower prevalence of ideal BP (86.6\% $88.8 \%$ ) compared to females ( $93.0 \%-96.3 \%$; Hardy et al.,2017). The limitation was the lack of income data to align BP status and strengths were the use of 4 cycles of NHANES data and specifying racial and ethnic groups.

Among Enhance Coal Workers Health Surveillance Program participants ( $n=$ $1,402)$, the prevalence of PreHTN was highest in ages $15-44$ at $55 \%(n=259)$ compared to $>65$ at $33 \%, p<.05$ (Casey et al., 2017). The challenge with this study was most participants were NHW (93\%), male (94\%) and there was no income data to assess an association by age, gender, race, and ethnicity. The relevance of the study was the prevalence of PreHTN (46\%) was higher compared to the general population (36.3\%) (Gupta et al., 2010); thus, demonstrating that specific workers may carry a higher PreHTN burden compared to the general population.

In a Japanese cohort study ( $\mathrm{n}=2,227$ ) 35-90 years of age, assessing progression from PreHTN to HTN and risk of CVD found at follow-up, 241 of the 702 PreHTN at baseline progressed to HTN (26.1\%), CVD risk was HR 2.95, 95\% CI [1.05, 8.33], $p<.041$ (Ishikawa et al., 2017). Older age (per 10-year increment) $H R 1.16,95 \% \mathrm{CI}$ [1.07,1.27], $p<0.01$, was reported as a contributor to HTN progression and CVD risk. Study limitations include a homogenous population ( $98.1 \%$ identify as Japanese; Central Intelligence Agency [CIA], n.d.a), over $1 / 3$ of population was male and income data was not available.

The strengths were using urban and rural communities and the cohort was part of a prospective study. Infant mortality and life expectancy are a good indicators of population health (World Health Organization [WHO], 2021). Compared to the U.S., estimated infant mortality rate in Japan is 2.0 per 1,000 live births vs 5.67 per 1,000 for the U.S. and life expectancy is 85.5 years of age compared to life expectancy in the U.S. 77.8. years of age (Arias et al., 2021; CIA, n.d.a; Ely \& Driscoll, 2020). These data suggest that age may not buffer healthier populations from PreHTN and incident HTN.

## Gender

Empirical evidence suggest men are at greater risk for PreHTN across ages compared to women as demonstrated in both cross-sectional and prospective studies (Booth et al., 2017; Gupta et al., 2010; Hardy et al., 2017; Joffres et al., 2013; Kit et al., 2015; Lo et al., 2013; Toprak et al., 2009; Yano et al., 2017).

In an analysis of BP trends between 1999-2000 and 2011-2012, PreHTN prevalence in 1999-2000 among males was at 57.3\% compared to females at $43.5 \%$ and 2011-2012 PreHTN prevalence among males was at $53.5 \%$ versus at $41.2 \%$ for females (Booth et al., 2017). Booth et al. (2017) did not cite statistical significance in BP category by gender. A major limitation was lack of income as a variable to evaluate association with EBP. A strength was the use of NHANES data.

Gender differences in PreHTN prevalence ( $n=3,735$ ) studies reported that compared to females, males carried the PreHTN burden (44.8\% vs. 27.3\%), $p<0.001$ (Gupta et al., 2010). A limitation include lack of income data to determine an association with PreHTN. The strength was the use of NHANES dataset.

In the Bogalusa Heart Study, a prospective biracial cohort ( $n=1,379$ ) assessing the association of PreHTN and CV risk, $(n=377)$ PreHTN prevalence was greater for males compared to females ( $35 \%$ vs. $22 \%$ ), $p<0.001$ (Toprak et al., 2009). Income data was not available to assess an association with PreHTN. The study strength was repeated measures and a young adult population 20-44.

In a cross-sectional analysis of HTN predictors among a sample of Mexican Americans ( $n=2,780$ ) reported PreHTN prevalence among males was at $40 \%$ compared to females at $26 \%$. The study strength was the use of NHANES data and a large sample of Mexican Americans, a limitation was the absence of income data (Bersamin et al., 2009). In a trend study after adjusting for race/ethnicity, females were less likely to be PreHTN or HTN compared to males (Booth et al., 2017). A strength of this analysis was the comparing 1999-2000 and 2011-2012. A limitation was lack of income data aligned with BP level.

In a prevalence study describing dyslipidemia (e.g., high cholesterol) and BP among U.S children and adolescents ( $8-17 ; n=1,665$, males $n=842$, females $n=823$ ), reported the prevalence of either high or borderline high $\mathrm{BP}(>120 / 80)$ among males was at $15.4 \%, 95 \% \mathrm{CI}[11.0,20.9]$ compared to females at $6.8 \%, 95 \% \mathrm{CI}[4.0,10.6], p<.01$ (Kit et al., 2015). A limitation of this analysis was the lack of parental income data to estimate an association.

The study's strength was the use of the NHANES database which has a large sample representative of the U.S population.

Similarly, in another pediatric observational study, $\operatorname{PreHTN}(n=25,370)$, was greater in males $(n=15,463)$ compared to females $(n=9,907), p<.001$ (Lo et al., 2013). A limitation of this analysis was the lack of parental income data to estimate an association and the strength was multiple sites and repeated measures to determine true hypertension. The higher prevalence aligns with Hardy et al. (2017) who reported that by age 8, PreHTN prevalence among boys was [10\%-11.0\%] compared to girls [2.7\%-5.9\%]. These data support PreHTN is a public health issue, especial for youth.

In an international study comparing $(\mathrm{n}=20,870)$ the distribution of BP by level (e.g., normal, PreHTN, and HTN), age, gender, and country (Canada $n=3,584$, England $n=7,382$, United States [U.S] $n=10,003$ ), found that across all countries, PreHTN prevalence (43.9\% in England, 27.2\% in Canada, and 36\% in U.S) was greater among males compared to females (Joffres et al., 2013). The problem with this study was the inability to combine the prevalence data by gender, age, race, ethnicity, and the exclusion of income to evaluate differences.

In the Reasons for Geographic and Racial Differences in Stroke (REGARDS) Study, reported similar PreHTN prevalence $(n=5,553)$ among females $(n=2,777)$ and males ( $n=2,776$ ); however, male gender was a predictor of PreHTN, only for NHB (Glasser et al., 2011). A limitation of the study includes only NHB and NHW populations. Strengths was the use large cohort from regions with higher-than-normal stroke prevalence and repeated measures (Lanska \& Kuller, 1995) .

In a PreHTN and HTN pediatric prevalence study ( $n=199,513$ ), PreHTN prevalence was at $12.7 \%(n=25,370)$; compared to females, males reported a higher PreHTN burden ( $39 \%$ vs $61 \%$ ), $p<.001$ (Lo et al., 2013). Within the study, parental income data was not available to determine an association by age, gender, race, and ethnicity. Study strengths include diverse population and the use of clinical records to confirm and compare results over time. Koebnick et al. (2013) reported PreHTN prevalence for males at $37.1 \%(95 \%$, CI: $36.8,37.3)$ compared to females at $26.0 \%$ $(25.8,26.4), p=$ value was not reported.

In a cross-sectional study of youth athletics ( $n=7,705$, female $31 \%$, male $69 \%$ ) compared to females, the risk of EBP were greater among males $R R 1.24,95 \%$ CI [1.10, 1.38], $p<.005$ (Stiefel et al., 2016). A limitation within the study was lack of parental income data to assess PreHTN association by age, gender, race, and ethnicity. Similarly, in a child/adolescent population, Kit et al. (2015) reported higher prevalence of borderline/high BP among males at $15.5 \%, 95 \% \mathrm{CI}[11.0,20.9]$ compared to females at $6.8 \%, 95 \%$ CI $[4.0,10.6], p<.01$ (Kit et al., 2015). A strong argument can be made that males regardless of age carry PreHTN burden.

## Cost of Prehypertension

Chen et al. (2017) assumed the cost of PreHTN to be the same as HTN. This is supported by studies demonstrating the advantage of pharmacologic treatment to delay incident HTN (Fuchs et al. 2016). Gross Domestic Product (GDP) is the total value of all the goods and services produced in a country during a period (e.g., year; International Monetary Fund [IMF], 2018).

Total health care spending in 2016 is estimated at $\$ 3.1$ trillion (17.9\% of Gross Domestic Product [GDP] \$9,655 per person), HTN accounted for $\$ 79$ billion, $95 \%$ CI [\$72.6, \$86.8] (Dieleman et al., 2020). Currently, HTN prevalence is estimated at 47.3\%, $95 \%$ CI [45.9, 49.2] (Virani et al., 2021) and greater HTN prevalence might result in higher economic commitment. A supplemental report estimates by 2035, the total cost of HTN as a risk factor for CVD to be $\$ 334$ billion (Khavjo et al., 2016). An economic case can be made for reducing PreHTN prevalence as the United States government's share of national health care spending is projected to grow 5.5 per year between 2017-2026, which may result in a rise in the Gross Domestic Product (GDP) spending from $17.9 \%$ in 2016 to $19.7 \%$ by 2026 (CMS, 2018).

## Conceptual Theory/Theoretical Foundation

The critical race theory (CRT) framework to follow because race and ethnicity are covariates for the research. The framework is useful to follow because race and ethnicity are moderating variables that can strengthen the association between the independent and dependent variable (Glasser et al., 2011; Gupta et al., 2010; Lo et al., 2013; Toprak et al., 2009).

Critical race theory (CRT) was developed out of legal scholarship and provides an analysis of race and racism from a legal point of view and can be leveraged across disciplines (Ford et al., 2010; Ford \& Airhihenbuwa, 2018). Critical race theory (CRT) was initially advanced through the work of Derrick Bell Jr. (1975), Richard Delgado (1989) and Allan Freeman (1978).

These legal scholars recognized the limitations of Civil Rights gains, particularly legal policies that on the surface appear to help non dominate groups, but only suppresses choices (Ford et al., 2010; Ford \& Airhihenbuwa, 2018).

The framework consists of Racism as an imbedded social construct; Interest Convergence; Racialized Socialization; Black and White Binary of race; Racism as it intersects with gender, class, national origin, sexual orientation, and how these combinations play out in various situations; the use of storytelling, and counter narratives challenge generally accepted belief systems of the dominant group and their allies (Bell, 1975; Delgado, 1989; A. D Freeman, 1978; Freeman et al., 2017).

The original architects of CRT (Bell, 1975; Delgado, 1989; Freeman, 1978)
promoted the concept from a legal context. This concept has been used as an analysis tool in public health and education (Allen \& White-Smith, 2018; Aymer, 2016; Ford et al., 2009; Freeman et al., 2017; Irizarry \& Raible, 2014). When used as a control variable, race and ethnicity has the potential to strengthen the relationship between the independent (LMHPG) and dependent variable (PreHTN; Baron \& Kenny, 1986). When investigating health disparities, race and ethnicity can be a useful proxy to estimate burden, predict risk and measure associations between the IDV and the DV. This has been shown in morbidity and mortality data reports attributed to HTN (Benjamin et al., 2019; Murphy et al. 2021).

Evidence illustrates the effects of the U.S racial caste system as demonstrated in health services and public health outcomes (see Appendix C; Cobbinah \& Lewis, 2018; Sivashanker et al., 2020).

The higher prevalence of EBP among racial and ethnic groups compared to their NHW peers (Glasser et al., 2011; Gupta et al., 2010; Hardy et al., 2017; Lo et al., 2013) might be an expression of social hierarchy.

Racism is a unique stressor (Clark et al., 1999; Lucas et al, 2016), specifically perceived racism (Dolezsar et al., 2014), and frequent exposure to racism (Hicken et al., 2014; Volpe et al. 2020) has been linked to HTN. Racism as a stressor is pertinent to RQ\#2 (in table 4) because controlling for race and ethnicity might demonstrate PreHTN burden linked to specific group identity. Race is an offspring of Racism, in that it is a classification creating caste groups (Givel, 2021; Harawa \& Ford, 2009; see Appendix C).

Including ethnicity as a control variable in RQ \#2 (in table 4) might strengthen the link between the independent and dependent variable (see Appendix C). Hispanics who reported higher perceived ethnic discrimination were less likely to demonstrate evening BP dipping, a cardiovascular risk (Rodriguez et al., 2016) and may contribute to lower BP control compared to other racial and ethnic groups (Virani et al., 2021).

Non-Hispanic Blacks have the highest prevalence of PreHTN (Glasser et al. 2011; Gupta et al., 2010; Toprak et al., 2009), lowest household income and highest poverty rate compared to other racial and ethnic groups in the U.S. (USCB, 2021a). This is relevant to the research topic and RQ\#3 (in table 4).

Income is an indicator of social standing and human capital (Galobardes et al., 2006; Noël, 2018; see Appendix C). Marginalized groups are clustered among lower income (USCB, 2021a) and high poverty group membership is associated with increased odds of PreHTN and CV risk.

For example, the 2001-2002 National Epidemiologic Survey on Alcohol and Related Conditions (NESARC), subset ( $n=32,752$, NHB $n=8,245$, NHW $n=24,507$ ), reported that of the total past year heart attacks ( $n=375$ ), the prevalence attributable to structural racism among NHB was at $1.3 \%$ compared to NHW at $1.1 \%$ (Lukachko et al., 2014). EBP is a risk factor for heart attack (Guo et al., 2013b). Within the study, the odds of a heart attack was associated with public policy enacted by elected officials (e.g., 1994 Crime Bill) NHB OR 1.30, $95 \%$ CI [1.09, 1.69] compared to NHW OR $0.8095 \%$ CI [0.70, 0.91], $p<0.0001$; Civilian Labor Force $O R$ of $1.22,95 \%$ CI [1.04, 1.44] verses OR $0.94,95 \%$ CI [0.82, 1.07]; and Employment OR 1.74, 95\% CI [1.48, 2.04], $p<0.001$ compared to $O R 0.90,95 \%$ CI [0.78, 1.04]. A limitation in the study was the absence of gender, age, and ethnic group (e.g., Dominican, Mexican, etc.), income data, and incident heart attack was self-reported. One thing is clear, being a bottom racial caste may place NHB at increased risk for heart attack while buffering the effects among their NHW peers. This data is relevant because PreHTN is a risk factor for heart attack (Guo et al., 2013b) and is relevant to the Research Questions 2 and 3 (in table 4).

In a pooled meta-analysis ( $n=29$ studies, $n=1,010,858$ individuals), assessing the association between PreHTN and CV outcomes, PreHTN increased the risk of heart attack $R R 1.79,95 \% \mathrm{CI}[1.45,2.22], p<0.001$ (Guo et al., 2013b).

Han et al. $(2019 ; 8$ studies, $n=86,513)$ reported that $<65$ years of age and having PreHTN increased the heart attack risk $R R 1.86,95 \%$ CI [1.50-2.32], $p<0.05$. A limitation with meta-analysis is the absence of psychosocial factors such as racial discrimination to determine group variations. A strength in the meta-analysis are the pooled studies inclusive of different counties. None the less, the analysis demonstrates the deleterious association between PreHTN on CV risk.

In a random effects meta-analysis ( $n=44 ; n=32,651$ individuals), perceived racial discrimination was associated with higher $\mathrm{BP} \mathrm{Z}_{\text {Hypertension }} .048$, $95 \% \mathrm{CI}[.013$, $.087], p<.05$ (Dolezsar et al., 2014). According to findings, factors strengthening racial differences in BP include older age, male gender, higher percent of NHB (62\%) participation and lower education attainment. A limitation was the lack of PreHTN and income data to estimate PreHTN prevalence. The strength was the use of pooled data from diverse populations throughout the US.

Critical race theory (CRT) as a framework was used to explore the effects of macro, meso, and micro racism in the context of poor health care engagement among a sample of HIV positive NHB and Hispanic Black (HB) individuals ( $n=37$ ); where participants described how contemporary and historical abuses by the dominate group created mistrust and how institutional level actors (e.g., physicians, nurses, etc.) exacerbated distrust by treating clients as if they were invisible (Freeman et al., 2017).

This analysis demonstrates counter narratives from a non-dominate group in the context that their mistrust is righteous because they deal with it from health care practitioners who take the oath of do no harm.

These harms create intentional barriers to optimal health. For example, compared to NHW (ref), NHB level of medical mistrust was $a O R 1.73,95 \%$ CI [1.15, 2.61], $p<.01$ and Hispanic $a O R 1.49,95 \% \mathrm{CI},[1.02,2.17], p .<05$ (Barzargan et al., 2021). Overtime, medical mistrust predicted lower chronic disease treatment adherence at follow-up ( $\beta$ $.08, S E 04, p<.03$; Dale et al., 2016).

In a subset of Jackson Heart Study (NHB $n=1,845$ ), of the HTN free participants at baseline, at follow-up $52 \%(n=954)$ developed HTN; compared to low stress from everyday discrimination, high stress from lifetime exposure to discrimination was associated with a higher risk of incident HTN $H R 1.19,95 \% \mathrm{CI}[1.01,1.40], p<0.05$; however, significance declined after behavior risks factor adjustments (Forde et al., 2020). This study illustrates that stress attributed to unfair treatment is associated with incident HTN and PreHTN is a significant risk factor for HTN, an avoidable CV risk.

Ford et al. (2009), used CRT ( $n=373,210$ female and 163 male) from a race consciousness perspective (e.g., awareness of health provider bias) and found perceived racism increased the odds of routine HIV testing in a neighborhood clinic $O R 1.64,95 \%$ CI [1.07, 2.52], $p<0.05$. From this perspective, a strong conclusion can be drawn that self-care is an anti-racism strategy at the micro level that aligns with the challenging narrative component of CRT.

Irizarry and Raible (2014) used the CRT framework to combine a Latino/a CRT (LatCrit) analytical tool; this approach was designed to question Whiteness as ordinary by Latino students $(\mathrm{n}=7)$ to include their experiences engaging in the school to prison pipeline.

Researchers identified a critical consciousness among participants relative to their sociopolitical position in society as providing a buffer mitigating the stress of Racism.

Allen and White-Smith (2018) combined CRT and cultural capital conceptual frameworks to understand working class Black mother's involvement in their son's schools and how the mothers drew upon their cultural capital to challenge the ordinariness of White middle class parental school involvement as not the norm. Aymer (2016) used a CRT framework demonstrating the ordinariness of Racism to describe the psychosocial stressors of state sanctioned violence on the lives of Black men. Ordinariness has been described by critical race theorist as typical White life in the U.S and is not perceived by some as abnormal (Ford et al., 2010).

Prolonged exposure to stress may impair the regulatory functions of the Hypothalamic-Pituitary-Adrenal (HPA) axis which is responsible for fight or flight behavior via excess glucocorticoids (e.g., cortisol) and has been linked to BP regulation (Dallman et al., 2003; McEwen, 2000; Nai-Li et al., 2019). There is a relationship between stress and EBP that could be inherited. For example, compared to controls, offspring's of prenatally stressed rats demonstrated elevated systolic pressure when exposed to restraint stress $F=5, p<0.05$ and recovery $F=4.4, p<0.05$ (Igosheva et al., 2007). In humans, prenatal stress among marginalized populations has been reported as a contributor to offspring's later in life cardiovascular risk (Reynolds et al., 2013).

Bierer et al. (2020) reported that among European Jewish Holocaust Survivors (EJHS), FKBP5 Methylation was higher in offspring's $(n=28)$ of mothers exposed to ethnoviolence $\leq 11$ years of age compared to mothers exposed $\geq 12$ years of age ( $n=87$ ) ( $p<0.028$ ) and controls ( $n=34 ; p<0.0005$ ). The FKBP5 Methylation process serves to regulate excess cortisol in the blood by activating the negative feedback loop (Binder, 2009).

In a small study assessing the association of preconception parental trauma among EJHS ( $n=32$ ) and its effect on offspring's ( $n=22$ ); Yehuda et al. (2016) found that compared to ethnically matched controls $(n=9)$, the offspring's of EJHS demonstrated lower mean FKBP5 Methylation in offspring's $53.13 \pm 1.00$, compared to controls 57.58 $\pm 1.63, \mathrm{~F}_{1,25}=5.03, p<0.034$. Post-Traumatic Stress Syndrome (PTSD) is an anxiety disorder and has been associated with FKBP5 Methylation (Yehunda et al., 2014). Human and rodent studies have found offspring's of one or both stressed parents demonstrated anxiety like behaviors and heighten sensitivity to stress (Chan et al., 2018). Post-Traumatic Stress Disorder (PTSD) has been linked to incident HTN (Sumner et al., 2016) and elevated SBP among a sample of healthy adults (Edmondson et al., 2018).

Ethnoviolence is defined as violence and intimidation directed at members of ethnic groups that have been stigmatized by dominate group members (Helms et al., 2012). This is relevant because PreHTN is a risk factor for HTN. Race and ethnicity may explain EBP burden in NHB, and other ethnic groups compared to NHW due to the body's inability to reduce cortisol sensitivity.

The CRT framework has public health implications because of its ability to use race and ethnicity as a covariant that predicted an association between poverty group and PreHTN (in table 20, p. 145).

## Summary and Conclusions

The literature review for this study provided a lens into the body of work that has been accomplished to include findings and limitations. These findings can serve as a baseline for the study's conclusions with the aim of this research to be replicated. The paucity of data assessing the association of poverty group and health outcomes have been used by other researchers (Bailey et al., 2017a; 2012; Gupta et al., 2021); Flavonoid intake linked to SES (Vieux et al., 2020). Bailey et al. (2017a) used a mixed methods approach to assess the uptake of micronutrients (MN) by PIR, comparing $<130 \%$ PIR group ( $\mathrm{n}=1,635$ ), with $\mathrm{PIR}>350 \%(\mathrm{n}=1,344), p<.001$. Micronutrients (MN) are defined as small amounts of substances needed for healthy growth and development and include vitamins A, B, C, D and E, zinc, folate, and magnesium (Allen et al., 2009). Vieux et al. (2020) found an association between Flavonoid intake by income level and method of use (e.g., tea versus vegetables). Flavonoids are plant-based compounds having antioxidant characteristics that can kick start key enzyme action and are essential for optimal health (Panche et al., 2016).

A meta-analysis evaluating MN on BP in a population of T2D patients $(n=11$ studies, 723 individuals, middle aged population) found increased vitamin D ( $n=7$ studies, $n=542$ patients, ages $50-66$ ) resulted in a 4.56 mmHg reduction in Weighted Mean Difference $95 \%$, CI [ $-7.65,-1.47] ; p<0.004$ on SBP and 2.44 mmHg WMD on

DBP 95\%CI [-3.49, -1.39], $p<0.001$ (De Paula et al., 2017). A study limitation was lack of disaggregation by age, gender, ethnicity, income, and race that might have shown patterns. A strength was the pooling of multiple randomized clinical trials (RCT).

In a cross-sectional assessment of CV risk with increasing Dietary Inflammatory Index $\left(\mathrm{DII}^{\circledR} ; n=17,689\right)$ compared to persons in $1^{\text {st }}$ quartile (better Income to Poverty[ITP]), persons in the $4^{\text {th }}$ quartile (worst ITP) had a $65 \%$ higher odds of DII ${ }^{\circledR}$, $95 \%$ CI $[44 \%, 89 \%]$ and differences in SPB was shown, quartile 1 SBP 118.2 $\pm 0.4$ compared to quartile $4 \mu 123.6 \pm, p<0.001$ (Mazidi et al. 2018). Within the study, $46.7 \%$ of persons in quartiles 1 and 4 were NHW compared to $16.8 \%$ vs $29.3 \% \mathrm{NHB}$, $16.5 \%$ vs $13.3 \%$ Mexican American: and $20.0 \%$ vs $10.9 \%$ Other Hispanic and Others (all $p<0.001$ ). Study strengths include the use of NHANES datasets and significant findings after adjusting for covariates (e.g., ITP). Limitation included the cross sectional method which cannot attribute cause and effect.

In a study assessing the racial/ethnic changes in obesity using age, gender, race, ethnicity, and family income-to-poverty ratio as an independent variables from 2003$2018(n=29,712)$ reported that all groups demonstrated an increase in grade 3 obesity (BMI $\geq 40$; Gupta et al., 2021). Within the study by race/ethnicity, low-income Mexican Americans at 53.3\% [48.5,58] had the highest prevalence in 2015-2018 compared to NHB at $46.8 \%,[42,51.7]$ and at NHW $40 \%[35.9,44.3](p<0.001)$. This information is relevant because obesity is an independent risk factor for PreHTN and HTN (El-Ashker et al., 2021; Hu et al., 2017; Rivera-Mancía et al., 2018). Gupta et al. (2021) limitation
was not reporting the proportion of Mexican Americans who self-identified as White or Black. A strength was the use of multiple cycles of NHANES data.

The CRT conceptual framework was used as a guide to analyze an association between LMHPG and PreHTN and whether the association varied by age, gender, race, and ethnicity. A within Race subanalysis (NHB, NHW and OR) was conducted and showed a statistically significant association and is detailed in Chapter 5 (p. 145) with specific recommendations made in Chapter 5 (p. 166). In conclusion, this dissertation is unique because there is no research to my knowledge using the independent variable LMHPG and the dependent variable PreHTN.

## Chapter 3: Research Method

Chapter 3 addresses the research design and the rationale for its selection. Additionally, I describe the population, sampling, recruitment, instruments, data collection, data analysis, threats to validity, and ethical procedures. The study assessed an association between LMHPG and PreHTN and whether the association varied by gender, age, race, and ethnicity. Understanding the association may facilitate better implementation of social policies for primary prevention of PreHTN and incident HTN (e.g., secondary prevention) targeting persons $\geq 20$ to 79 years of age. The most recent data on PreHTN prevalence in the U.S. population for this dissertation were collected by the 2007-2008 NHANES (CDC, 2009a).

## Research Design and Rationale

This study used the 2007-2008 NHANES dataset to examine association between LMHPG and PreHTN and whether the insights explained the association. Procedures to evaluate income and health outcomes were used. For example, PIR, defined as a measure of income level accounting for family size (McClurkin et al., 2015), was used to assess insurance status and ideal CV health, as a covariant assessing CV risk profile with increased DII ${ }^{\circledR}$ scores (Mazidi et al., 2018). Vieux et al. (2020) assessed the association between flavonoid intake linked to SES (e.g., income to poverty ratio). This dissertation used LMHPG in lieu of PIR. PIR is discussed because the initial proposal was intended to use the measure; however, a change was made and is explained in Chapter 4 (see p. 125).

PIR has also been described as the ratio (e.g., proportion) of family income to the HSDHHS poverty threshold, accounting for family size and inflation (McClurkin et al.,

2015; USDHHS, n.d.). The poverty threshold is defined as the minimum requirements to meet basic needs (e.g., food, shelter, etc.; USCB, 2018). Studies have used PIR as an ordinal variable to estimate the prevalence of nutritional intake (Bailey et al., 2017a), insurance status by ideal cardiovascular health (McClurkin et al., 2015), and $\mu$ flavonoid intake (Vieux et al., 2020).

Moore and Liu (2017) categorized PIR as low, middle, and high as a covariant to assess the association between pediatric BP and serum vitamin D concentrations. Gupta et al. (2021) categorized PIR as lowest, medium, and highest in their assessment of racial/ethnic disparities in obesity across time periods. This study used LMHPG to estimate prevalence and predict the odds of PreHTN controlling for age, gender, race, and ethnicity. The method is appropriate for public health research because categorical data are finite; additionally, it simplifies data analysis and might be easier to interpret to the general population. This was relevant to the research topic because income group may not be associated with optimal BP outcomes. For example, researchers have reported the odds of chronic diseases by income when controlling for race and ethnicity. Research outcomes have demonstrated limitations among some high-income groups in protecting against chronic medical conditions to include EBP (Assari, 2018; Wilson et al., 2017).

Power (P) has been described as the ability of a specific statistical test to detect a relationship. Factors improving power include estimated minimum sample size for detecting an identified size of effect, prior public health studies, and a priori calculation to estimate a strength of association (Academia.edu [AE], 2017). The alpha error is defined as the chance of rejecting the null hypothesis when in fact it is true.

The effect size estimates the magnitude of influence that the independent variable has on the outcome variable.

The dependent variable for this study was PreHTN, defined as an EBP category SBP 120-139/DBP 80-90 identifying persons at elevated risk for HTN and designed to alert and encourage lifestyle modifications to delay onset of HTN (Whelton et al., 2017).

The independent variable, LMHPG, came from the 2007-2008 NHANES dataset's annual household income (in table 5) and is described in Chapter 4 (see p. 125). The research covariates used were age, gender, race, and ethnicity as established by NHANES. It is worth noting that race and ethnicity have similarities in terms of both being socially constructed (see Appendix C).

During the 2007-2008 NHANES survey period, all Hispanics were oversampled (Johnson et al., 2013). The 2007-2008 NHANES dataset includes ( $n=10,149$; CDC, 2009a); Hispanics represent $33 \%$ of the total sample. Of the 10,149 participants, $(n=$ 4,939) 20-79 years of age met inclusion criteria, of which Hispanics represented $<30 \%$ of the population. The research questions, the null, and alternative hypotheses guiding the study are described in Table 4. My research analysis was possible because the 2007-2008 NHANES data was already collected, available for research, time saving, and widely accepted in public health research (Bailey et al., 2017a; Booth, 2017; Gupta et al., 2010; Vieux et al., 2020). Research questions were derived leveraging a data-driven approach. A data-driven approach is defined as evaluating specific databases to determine if the variables of interest are available to answer a research question (CDC, n.d.a).

Conversely, qualitative research would not have been appropriate because the research questions did not address how or why a process occurs (Fassinger \& Morrow, 2013). The interpretive nature of qualitative research is informative and is best used on small samples that would not have met the current research criteria. Finally, a mixed method approach was beyond the scope and resources of a student researcher (Guetterman et al., 2015).

The choice to use the 2007-2008 NHANES dataset was based on it being complex and on the use of a multistage probability sampling design to select participants representative of noninstitutionalized U.S. citizens; the dataset provided the needed participants (CDC, n.d.b). NHANES used a cross-sectional design, whereby all measurements were taken at one point of time with separate groups (e.g., age, gender, race, and ethnicity). Cross-sectional designs have been used in public health prevalence (Gupta et al., 2021; Gupta et al., 2010; Moore \& Liu, 2017; Vieux et al., 2020) and risk prediction research (Madanat et al., 2014; Rafan et al., 2018). A major benefit in using NHANES data is that the NCHS (2017) is the nation's leading health statistics agency as designated through legislative authority. According to the FY 2017 President's Budget request, $\$ 160,397,000$ was allocated for Health Statistics to collect data, analyze data, and purchase equipment (NCHS, 2016).

NHANES is a major program of the NCHS and is responsible for assessing the health and nutritional status of adults and children in the United States (CDC, 2017a). The strengths of using NHANES were that it has been in operation > 50 years, the 20072008 dataset was appropriate to answer my research questions, and the sample design
was reported as allowing the production of aggregate-level national estimates (Curtin et al., 2013). NHANES data collection measures have been validated, and it has a physical examination component to objectively measure biological samples (CDC, 2017a).

Because my research was an analysis of secondary data from a cross-sectional design, a potential limitation was the inability to show a cause-and-effect relationship as described by public health prospective research (Zhang et al., 2017). G*Power 3.1 software was used to determine a minimum number of study participants required to answer the research questions when using the following parameters: alpha $<.10$; power $=$ 0.90 ; effect size $=0.25($ see Appendix A).

A key question in the NHANES 2007-2008 BP questionnaire asked whether a person had been told by a health care provider whether they were prehypertensive (CDC, 2006a). The NHANES 2007-2008 dataset was appropriate, given its ability to align research questions with specific NHANES variables (in table 8).

## Table 8

Research Questions'Alignment, Variables, and Measurement Levels

| RQ | Variable type | Variable name | Variable source | Value | Level of measurement |
| :---: | :---: | :---: | :---: | :---: | :---: |
| $\begin{aligned} & 1,2, \text { and } 3 \\ & \text { refer to Table } 4 \end{aligned}$ | Independent | INDFMPIR | Annual household income | $\begin{aligned} & 1,2,3,4, \\ & 5 \end{aligned}$ | Categorical |
|  | Dependent | BP0Q52 | Blood Pressure Questionnaire told have prehypertension | Yes, No | Categorical |
|  | Covariant | RIAGEYR | Screener module | 20-79 | Continuous |
|  | Covariant | RIAGENDR | Screener module | Male, Female | Categorical |
|  | Covariant | RIDRETH1 | Screener module | Mexican, Other Mexican, NHW, NHB, OR | Categorical |

## Methodology

## Population and Setting

The target population for NHANES consists of civilians of all ages in the noninstitutionalized U.S. population (CDC, 2017a; Johnson et al., 2013). The population analyzed was from the NHANES 2007-2008 dataset because it was the only dataset that asked about PreHTN in the population compared to the other datasets within NHANES (CDC, n.d.a). The NHANES database was the most appropriate source for PreHTN information because CDC is a program under the NCHS and is tasked with providing relevant, accurate and timely data (CDC, 2019). The 2007-2008 NHANES dataset contained 12,943 screened subjects, of which 10,149 were interviewed and 9,764 underwent physical examination (Johnson et al., 2013).

## Sampling Procedures

The 2007-2008 NHANES dataset was appropriate because it used a complex, multistage probability design (Johnson et al., 2013). The study inclusion criteria for analysis indicated NHANES participants 20 to 79 years of age who had PIR, PreHTN, age, gender, race, and ethnicity data. Exclusion criteria for this study applied to participants $0-19$ years of age, participants 80 years of age, and participants with missing independent, dependent, and covariant data.

## Power Analysis

Because there were no previous data on the association between LMHPG and PreHTN, conditions set for analysis were $<.10$ alpha, 90 power, and medium effect to estimate minimum sample size for detecting an identified effect using logistic regression
(AU, 2017). G*Power Version 3.1.92 software was used to calculate sample size. Analysis for the research was from the in-home interviews only; physical examination and laboratory data were not used. The variables used are found on the NHANES variable search page (CDC, n.d.a). The justification for using the in-home interview data was based on the PreHTN question on the BP questionnaire dated September 8, 2006, and was used to collect data for the 2007-2008 NHANES dataset (CDC, 2006a).

Power is the ability of a statistical test to detect a relationship. Preanalysis power assessments are necessary to estimate a minimum sample size for detecting an identified size of effect. The effect size quantifies an association, the larger the effect size, the stronger the association. Having more participants can help minimize occurrence of Type II $\beta$ error, failure to reject the null hypothesis when the alternative is correct. For example, Bond et al. (2016) may have committed a Type II $\beta$ error when they concluded that the PreHTN group $(n=6)$ had elevated SBP after weightlifting (from $139 \pm 6$ to 205 $\pm 11 \mathrm{mmHg},+48 \%$ ) compared to the normotensive control group ( $n=6$; from $132 \pm 3$ to $145 \pm 3 \mathrm{mmHg},+10 \%)$; intergroup difference $p<0.001$. This may have been an erroneous conclusion due to the small sample size, and the study did not report a postanalysis power assessment.

For the current research, the effect size was medium/moderate. At the time of the research proposal, the effect size was unknown and was based on the body of literature where covariables (e.g., age, race, etc.) impacted the outcome (Booth et al., 2017; Glasser et al., 2011; Lo et al., 2013; McClurkin et al., 2015). A review of literature did not show prior studies assessing the association between LMHPG and PreHTN; therefore, a less
conservative alpha was favorable with the understanding that a higher error rate may occur. Another rationale undergirding the selection of $<.10$ alpha for this research was to minimize the occurrence of a Type I error, and the number of participants answering yes to having PreHTN was small. G*Power Version 3.1.92, free power analysis software available online, was used to estimate a minimum sample size using the set alpha. The power analysis framework was used to determine the minimum number of participants recommended (in table 9).

Table 9
Power Analysis Framework

Research question (RQ)

Null hypothesis $\left(\mathrm{H}_{0}\right)$

Alternative hypothesis $\left(\mathrm{H}_{\mathrm{a}}\right)$

Dependent variable
Covariates
Alpha

Power

Effect size
Calculated minimum sample
size

Software

1. RQ1: Is there an association between lowest, middle, highest poverty group (LMHPG) and prehypertension (PreHTN)?
$\mathrm{H}_{0}$ : There is no association between LMHPG and PreHTN.
$\mathrm{H}_{\mathrm{a}}$ There is an association between LMHPG and PreHTN.
2. RQ2: Is there an association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity (Hispanic compared to non-Hispanic [HcNH])?
$\mathrm{H}_{0}$ : There is no association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity ( HcNH ).
$H_{a}$ : There is an association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity ( HcNH ).
3. RQ3: Is there a difference in the prevalence of PreHTN between LMHPG controlling for age, gender, race, and ethnicity ( HcNH )?
$H_{0}$ : There is no difference in the prevalence of PreHTN between LMHPG controlling for age, gender, race, and ethnicity (HcNH).
$H_{a}$ : There is a difference in prevalence of PreHTN between LMHPG controlling for age, gender, race, and ethnicity (HcNH).

Prehypertension Yes/No
Age, gender, race, and ethnicity (HcNH)
$<0.10$
0.90
0.25 (moderate)

$$
378
$$

378

The first step was to compute an a priori analysis for a continuous predictor variable using logistic regression test based on a two-probability function because the DV is dichotomous, and the covariates moderately influenced the outcome. The effect size chosen for this research was based on the review of PreHTN prevalence estimates in the U.S., specifically those studies that used variables affecting the association between the independent and dependent variable and NHANES data (Booth et al., 2017; Gupta et al., 2010). My study used the estimated minimum sample size. The $\mathrm{G}^{*}$ Power a priori power analysis estimated 378 individuals needed to achieve .90 power to correctly reject the null hypothesis (see Appendix A).

Since this is an early study and the effect size was estimated to be medium/moderate, findings will require validation and reassessment using study designs that can estimate risk and inform discussions of possible causality (in table 9, for research hypothesis). These discussions could influence Health Equity policy. Depending on the context (e.g., environment), Health Equity is defined as everyone having a fair opportunity for optimal health and no one being deprived of achieving health (WHO, 2019a). Health Equity has also been defined as contextual and shared approach that removes obstacles impeding wellbeing and assures social justice in health (Braveman et al., 2011; Braveman, 2014). Health Equity policies are methods institutions implement using the Social Determinant of Health Framework to dismantle health inequities and ensure fair opportunities specifically for marginalized communities (Hall et al., 2016; WHO, 2019b). This is accomplished via Health Equity in all policies such as but not
limited to the use of Presidential Executive Orders to adequately address and reduce public health problems.

Social Determinants of Health are defined as conditions in which people are born, live, work, age; and are shaped by the distribution of money, power, and resources (WHO, 2019b) this also includes lineage and caste membership. Health Inequities are avoidable injustices in health (e.g., morbidity burdens), economic (e.g., wealth), and political (e.g., group interest) conditions between groups of people within and between countries (WHO, 2019b).

## Procedures for Recruitment, Participation, and Data Collection

A secondary analysis of existing data from the 2007-2008 NHANES dataset was used. No permission was needed to access the deidentified data because it is publicly available. The data files were downloaded from the NHANES website. The variables examined are categorical except for age. Age was transformed into groups (e.g., 20-39). Evaluation of data completeness was accomplished by comparing files downloaded against the data documentation code book and frequency count manuals for $\mathrm{BP} /$ cholesterol and demographic variables, and sample weights (CDC, 2009a; CDC, 2009b). The NHANES sampling methods are described elsewhere (Curtin et al., 2013). The criteria for data entry include participants 20-79 years of age who have data for the independent (ID) and dependent variable (DV) to include covariates collected at in-home interviews. The criteria for data exclusion are 0-19 and 80 years of age, missing ID and DV to include covariates.

## Instrumentation and Materials

The NHANES survey is designed to assess the health and nutritional status of non-institutionalized children and adults in the U.S. (CDC, 2017a). It is a snapshot of the nation's health, and its outcomes are used to guide health policies, create new guidelines, and promote research. Instrumentation and materials are described elsewhere (Curtin et al., 2013). These attributes make NHANES a desirable database for answering my research questions. The criteria being used from the 2007-2008 NHANES dataset are demographic, PIR and self-report PreHTN data from participants 20 to 79 years of age collected at the in-home interview; inclusion and exclusion criteria have been described elsewhere. Mobile examination and laboratory center data will not be used. The age in year variable name is RIAGEYR, for males and females $0-150$ years of age (CDC, 2009a) and was collected using the screener instrument (SI). The gender variable name is RIGENDR for male and females 0-150 years of age and it too was collected using the SI. The Race/ethnicity variable name is RIDRETH1, and it reports race ethnicity information using SI. The Prehypertension variable name is BPQ052 Told you have Prehypertension and was collected on the BP Questionnaire (CDC, 2006a). The variable name for Income to Poverty Ratio is INDFMPIR and was collected on the income survey (CDC, 2006c; in table 10).

## Table 10

Dependent and Independent Variable Used in This Study-2007-2008 NHANES Data

| Research questions | Variable type | Variable name | Variable source/label | Value code | Level of measurement |
| :---: | :---: | :---: | :---: | :---: | :---: |
| Refer to Table 4 for RQs 1-3 | Independent | INDFMPIR | Ratio of family income to poverty | $\begin{aligned} & 1,2,3 \\ & 4, \& 5 \end{aligned}$ | Categorical |
|  | Dependent | BPQ052 | Blood Pressure <br> Questionnaire/ <br> Ever been told you have prehypertension | $\begin{gathered} 1,2,7 \\ \& 9 \end{gathered}$ | Categorical |
|  | Covariant | RIAGEYR | Screener module/age | $\begin{gathered} 0-79 \\ 80 \end{gathered}$ | Continuous |
|  | Covariant | RIAGEYR | Screener module/ gender | 1,2 | Categorical |
|  | Covariant | RIDRETH1 | Screener module race/ethnicity | $\begin{aligned} & 1,2,3 \\ & 4 \& 5 \end{aligned}$ | Categorical |

## Independent Variable

The independent variable is Lowest, Middle, and Highest Poverty Group is used as a proxy for Poverty Income Ratio (PIR) which is a measure of income level accounting for family size (Mazidi et al., 2019; McClurkin et al., 2015). Poverty Income Ratio data is generated from the 2007-2008 NHANES income questionnaire (CDC, 2006c). Specific poverty threshold is defined as the minimum requirements to meet basic needs (e.g., food, shelter, etc.; USCB, 2018).

The PIR is described as the ratio of family income to the United States Department Health and Human Services poverty threshold accounting for family size and inflation (McClurkin et al., 2015; USDHHS, n.d.a).

The method to be used to calculate PIR has been described as a good income measurement because it can be used over a period (e.g., 2 months; Johnson et al., 2013). My intention was to examine percent of PIR values of $0-130 \% ;>130 \%-350 \%$, and $\geq 350 \%$ to assess an association with PreHTN. These values have been used in other cross-sectional studies using NHANES datasets (Bailey et al., 2017a; Egan et al., 2020; Vieux et al., 2020). The approach was selected because it was least complicated to combine values exceeding $350 \%$ for analysis. Subjects with values $0-130 \%$ will be coded as zero; $>130 \%-350 \%$ coded as 1 and $\geq 350 \%$ will be coded as 2 . Chapter 4 will explain the challenge with this method and justification for using an alternative (see p.125).

## Rationale for Inclusion of Potential Covariates and/or Confounders

Confounding variables (CV) may influence the relationship between the independent and dependent variable (Ananth \& Schisterman, 2017). Researchers examining differences in PreHTN (Gupta et al., 2010; Selassie et al., 2011); Cardiovascular Health (Egan et al., 2020); Health Insurance Status and ideal CV Health (McClurkin et al., 2015); and adverse health among high income earners (Wilson et al., 2017) have used gender, income, race, ethnicity, and education to partially explain the relationship between the independent (IDV) and dependent (DV) variables. Confounding variables for this study are age, gender, race, and ethnicity (Hispanic compared to Non-Hispanic); and were adjusted using logistic regression modeling.

According to the United States Census Bureau (2011), 53\% of Hispanics selfidentified as White. The overrepresentation of White identity among Hispanics could potentially obscure within group PreHTN prevalence estimates and predicted risk. Empirical evidence report Hispanics self-identify as White to confer economic and social advantages that would be excluded if identified as non-White (Darity et al., 2005; Golash-Boza and Darity, 2008; Lopez et al., 2018; Quiros \& Dawson, 2013). The 20072008 NHANES dataset uses the Hispanic variable to differentiate from non-Hispanic. During data analysis this variable was adjusted by comparing cases who are Hispanic against non-Hispanic by age and gender (in table 17). Non-Hispanic is the reference group in the BLR model.

## Table 11

Covariates

|  | Definition |
| :--- | :--- |
| Age | Age is collected on the screening instrument as continuous, then <br> it is transformed into categories for analysis. Age in years, at the <br> time of the screening interview is reported for survey <br> participants between the ages of 1 and 79 years of age. There are <br> no missing values. |
| Gender is a categorical variable and is reported as (1) male or <br> (2) female; there are no missing values. The lack of other (e.g., <br>  <br> Scheim, 2013) and a false conclusion. |  |
| Including an Other category is not feasible because the <br> NHANES screener module assessing gender identification list <br> female, male, don't know or refused to answer (CDC, 2006b). <br> The other category is absent in the United States Census Bureau <br> questionnaire (USCB , n.d), a Federal Statistical Agency (United <br> States of America Government [USA.Gov], 2019). |  |
| A rationale for not including a Transgender category might be <br> attributed to the estimated prevalence of persons in the United <br> States identifying sa Transgender being close to 1 million <br> (Meerwijk \& Sevelius, 2017). |  |
| Race is a categorical variable identified as Hispanic-Mexican |  |
| Race/Ethnicity |  |
| American, Hispanic-Other, NHB, NHW and other (CDC, |  |
| 2009a). |  |

## Dependent Variable

The dependent variable for the research is Prehypertension. Prehypertension is reported in the 2007-2008 NHANES BP questionnaire and identified as BPQ052. The categorical variable has a three (3) values; 1) yes, 2) no, and 3) Don't know (CDC, 2006a). The Don't Know category indicates the person was not asked the question or given the test (CDC, 2018a).

There were no missing or refusal values on the PreHTN item. To answer research questions, responses are coded to reflect (1) no and (2) yes. According to the NHANES codebook (CDC, 2009a) subjects who answered "don't know" were excluded from the dataset. However, after downloading the data, the "don't know" numbers were in the data set, which were excluded from analysis (see Figure 1).

To determine PreHTN, the 2007-2008 NHANES collected BP awareness, treatment, and control data during the personal interview on the blood pressure questionnaire BPQ (CDC, 2006a). The BPQ excludes household members $<16$ years of age (CDC, 2006a). The set research inclusion are 20-79 years of age and $0-19$ years of age, and 80 years of age were excluded.

## Data Analysis Plan

Data files for the 2007-2008 NHANES are released in the Statistical Analysis Software (SAS) transport file format; this allowed extraction of data on Windows, UNIX, or Macintosh (CDC, n.d.d). Data was analyzed using Statistical Package for Social Sciences (SPSS), version 25.0 (International Business Solutions [IBM], 2018), which can analyze SAS transport files. Statistical Package for Social Sciences have been used in

PreHTN prevalence and risk prediction research to analyze data (Rafan et al., 2018). The SPSS program managed and organized the data to include coding, recoding, transformation, weighting variables, and data analysis. This software was optimal for analyzing complex variables from the 2007-2008 NHANES dataset. The 2007-2008 NHANES dataset incorporated weighing to account for non-responses, oversampling, and post stratification (CDC, n.d.e).

Prior to analysis, analytic guidance was followed as recommended by NHANES (Johnson et al., 2013). The first step was to determine if there were any missing data, this was accomplished after downloading the SAS file from CDC prior to analysis. According to the BP and cholesterol, documentation, codebook and frequency guide, there are $(n=6,546)$ responses to the PreHTN question (CDC, 2009b). The PreHTN group response frequency met inclusion criteria in terms of covariables and income data. Data was cleaned, recoded, and grouped for analysis via SPSS version 25.0; results are reported in Chapter 4 using text and visual interpretations.

A decision tree of the 2007-2008 NHANES data ( $n=10,149$ ) was constructed to provide a visual interpretation of included and excluded participants (see Figure 1). Of the 6,546 participants responding to the PreHTN questionnaire, there were $(n=336)$ yes, $(n=6,142)$ no, $(n=68)$ don't know and persons 16-19 years of age and 80 years of age were excluded ( $n=1,105$; see Figure 3). The research excluded persons $0-19$ years of age and 80 years of age; study entry were participants 20-79 years of age who had data for the independent and dependent variable.

Data was downloaded from the CDC website as a SAS file. Frequency counts was performed on data following transfer to ensure counts matched reported frequency for each variable as reported in the 2007-2008 Data Documentation, Codebook and Frequency Analytic notes (CDC, n.d.c). To achieve data quality, missing value counts was conducted to ensure agreement with the transferring files.

NHANES assigns missing values with a (.) for numeric characters, blank for character variable and missing values will be ignored. A table labeled Research Questions (RQ) and Variables Names (VN) was constructed to align RQ with VN (in table 12).

Table 12
Research Questions and Variable Names

| Research questions | Variable names |
| :--- | :--- |
| Is there an association between LMHPG and <br> prehypertension (PreHTN)? | Ratio of family income to poverty |
| Null hypothesis: There is no association between PIR |  |
| and PreHTN. | Told you have prehypertension. |
| Alternate hypothesis: There is an association between <br> LMHPG and PreHTN. |  |
| Is there an association between PIR and PreHTN <br> controlling for age, gender, race, and ethnicity <br> (Hispanic compared to non-Hispanic [HcNH])? | Ratio of family income to poverty |
| Null hypothesis: There is no association between | Told you have prehypertension |
| LMHPG and PreHTN controlling for age, gender, race, |  |
| and ethnicity (HcNH). | Gender |
| Alternate hypothesis: There is an association between |  |$\quad$ Race/Ethnicity (Mexican American, other.

## Procedures Used to Account for Multiple Statistical Tests

My research used descriptive statistics (DS) to describe the population by age, gender, race, and ethnicity. Descriptive statistic displays frequency tables, number, and proportion of the population exposed to PreHTN as demonstrated by other public health researchers (Caunca et al., 2020; Glasser et al., 2011; Gupta et al., 2010; Rafan et al., 2018). Prehypertension is the dependent variable and covariables are age, gender, race, and ethnicity (HcNH). Reporting was summarized by the frequency distribution and proportion of PreHTN self-report, yes or no.

Additionally, inferential statistics was used to draw conclusions about the 20072008 NHANES participants 20 to 79 years of age. Inferential statistics are frequently used in population based studies to inform empirical based decision making. Logistic regression (LR) is defined as the likelihood of an outcome (e.g., PreHTN) given the exposure risk (e.g., income) and if exposure varies by covariates (e.g., age; CDC, n.d.f). The goal of LR is to minimize standard errors. Logistic regression was an appropriate test to assess the association between LMHPG and PreHTN to include covariates. For example, Mazidi et al. (2018) used logistic regression analysis to examine the association between quartiles and increased inflammatory markers of DII ${ }^{\circledR}$. Jackson et al. (2017) used inferential statistics to assess the odds of antihypertension medication use by race and ethnicity; Rivera-Mancía et al. (2018) used LR to predict the type of body fat closely associated with PreHTN. Logistic regression (LR) helps the analyst predict the effect of exposure on the outcome after adjusting for confounders.

The test (e.g., logistic regression) is generally used when the independent variable is either categorical (e.g., having $>1$ categories) or continuous and the outcome variable is categorical (Aggarwal \& Ranganathan, 2017). When the outcome variable is dichotomous (e.g., yes, or no), the most appropriate test would be a bivariant analysis because of its categorical feature and the ability to assess relationship by covariant. Public health studies assessing prevalence of PreHTN with dichotomous dependent variable have been used (Rafan et al., 2018; Yang et al., 2017) to include design validity testing (Nikki et al., 2018). Binomial logistic regression (BLR) is a subset of logistic regression that predicts the probability that an observation falls into one or two categories of a dichotomous outcome variable if the selections vary by covariates (Rafan et al., 2018), and the term will be used going forward in this dissertation.

The research questions met the assumptions of BLR (in table 15). Logistic regression falls under a family of test called Generalized Linear Models (GzLM); which are models predicting a quantitative dependent variable by a set of independent variables that can be categorical or quantitative. Generalized linear model's have been used to predict PreHTN among samples demonstrating blood oxygenation changes (Wrobel et al., 2018). As a result, a logit transformation must be used to estimate the outcome (CDC, n.d.f). The logit function is designed to link the risk factors (e.g., LMHPG) to the outcome (e.g., PreHTN). If $p$ is the probability that a sample is PreHTN, then the logit is defined as ( $\mathrm{p} / 1-\mathrm{p}$ ) where PreHTN is the dependent variable and LMHPG is the independent variable.

To answer research question \#1: Is there an association between Lowest, Middle, and Highest Poverty Group (LMHPG) and Prehypertension (PreHTN)? The original plan was to use a Bivariant Pearson Correlation (BPC) to determine whether there is a significant relationship between LMHPG and PreHTN. The Binomial Logistic Regression (BLR) analysis was used in lieu of BPC because LMHPG and PreHTN are categorical variables and did not meet BPC assumptions. The BPC is an appropriate test for measuring continuous ID and DV variables. The chi-square $\left(x^{2}\right)$ test determined it was a good fit for BLR model $(p<.011)$. The chi square $\left(x^{2}\right)$ test have been used in PreHTN research to compare data between categorical variables (Owiredu et al., 2019). The BLR model was used to answer research question \#2 Is there an association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity ( HcNH )? The covariates age, gender, race, and ethnicity were entered, and age moderately increased the odds of PreHTN in the Highest Poverty Group.

To answer research question \#3 Is there a difference in the prevalence of PreHTN between LMHPG controlling for age, gender, race, and ethnicity ( HcNH ). The chi square $\left(x^{2}\right)$ test determined there was a statistically significant association between the IDV and DV ( $p<.013$ ). Researchers have used chi square $\left(x^{2}\right)$ to estimate prevalence and predict determinants of PreHTN and HTN by covariates (Fan et al., 2019; Khanam et al., 2015).

## How Results Are Interpreted

Descriptive statistics summary show frequencies, numbers, and estimated percentage of LMHPG and PreHTN (in Table 19). Categorical variables were
summarized by frequency distribution, number, and percentage of PreHTN. Inferential statistics was used to interpret BLR. Binomial logistic regression (LR) models predicted LMHPG is associated with PreHTN to include controlling for covariates. The level of significance for this research is $<.10$ and the rationale for its use is described elsewhere (see p. 103). The test was two tailed; any probability value of significance less than or equal to the level of significance was considered statistically significant. For this study Odds Ratio $(O R)$ was used to measure the influence of the IV on the DV. The assumptions for logistic regression are linearity (e.g., there is a relationship between the IV and DV); independence of error (e.g., same person not counted twice); multicollinearity (e.g., no duplicate predictor); and strongly influential outliers as demonstrated in the study.

## Threats to Validity

Bias can occur throughout the research process from failure to randomize samples to failure to report Type I errors. Secondary analysis of existing data can introduce methodological issues such as sampling, data collection, measurement, missing data, and non-response (Barrington \& James, 2017; Boo \& Froelicher, 2013; Yang et al., 2017). The SPSS software was used to adjust for survey weights to appropriately analyze the 2007-2008 NHANES dataset.

Researchers have used statistical software to adjust for complex sampling to estimate adult physical activity prevalence (W. M Williams, et al., 2018) and predict 10year cardiovascular disease risk (Borhanuddin et al., 2018).

The participant inclusion criteria for this study are household members 20-79 years of age with data for the independent and dependent variable. Exclusion criteria are participants 20-79 years of age without the IDV, DV, and covariates; and $0-19$, and 80 years of age. The 2007-2008 NHANES data collection methodology is described elsewhere (Curtin et al., 2013). The reliability of 2007-2008 NHANES data is guided by NCHS being an authorized federal statistical agency and professionally recognized authority in the field.

Threats to validity were addressed by controlling for age, gender, race, and ethnicity as covariates to estimate PreHTN prevalence and predict risk using logistic regression modeling. The biases that may have occurred have been from excluding participants 16-19 years of age and 80 years of age who have data for the IDV and DV and participants aged 20-79 missing the IDV/DV or had the IDV but not the DV and vice versa (see Figure 1).

## Ethical Concerns

This study used deidentified secondary data released by the CDC (n.d.b) and is available for public use. Sanitized secondary data analysis using NHANES data does not pose a disclosure or confidentiality risk for subjects. This research received Walden University's Institutional Review Board approval \#3-18-20-0131651 as required by Walden University.

## Summary

A detailed strategy to determine if there is an association between Lowest, Middle and Highest Poverty Group and PreHTN using 2007-2008 NHANES data was described
in Chapter 3. A rationale for the use of secondary data analysis; power analysis; selected statistical threshold (e.g., <10); procedures to account for multiple statistical test; rationale for inclusion of potential covariates; how results will be interpreted; threats to validity and ethical concerns were addressed. Chapter 4 presents and interprets statistical results, and Chapter 5 provides conclusions and recommendations for further research on this topic.

## Chapter 4: Results

## Introduction

The purpose for this quantitative, nonexperimental, cross-sectional study was to examine the association between (LMHPG) and (PreHTN. A subset of the 2007-2008 NHANES dataset was used to enable research. Household income was used in lieu of PIR, which did not have values required for the analysis (see pp. 125-126).

Chapter 4 addresses data collection and challenges encountered, as well as results and statistical assumptions, concluding with a summary. Results are presented and interpreted as aligned with each research question. In Chapter 4, I report on whether assumptions for the use of specific tests aligned with the analysis plan as described in Chapter 3, and I provide a summary transitioning to Chapter 5.

## Table 13

Research Questions and Variable Type and Recode Name

| Questions | Variable type-Name | Recode name and values |
| :---: | :---: | :---: |
| RQ 1, 2, \& 3; refer to Table 4 | Independent—INDFMPIR—Ratio of family income to poverty | PIR _Recode 3 groups-Lowest poverty group (Ref). ->\$55,000 |
|  |  | 2 - Middle poverty group-\$20,000-\$54,999 |
|  |  | $\begin{aligned} & 3 \text { - Highest poverty group- } \$ 0- \\ & \$ 19,999 \end{aligned}$ |
|  | Dependent-BPQ052-Told you have prehypertension | Prehypertension 0 - No 1 - Yes |
|  | Covariant RIDAGEYR | Age _Selection <br> 0 - Not of age ( $0-19 \& 80$ ) |
|  |  | 1 - Yes of age (20-79) |
|  |  | Filter_\$ 0 - Not selected |
|  |  | 1 - Selected |
|  |  | Age groups |
|  |  | 20-39 |
|  |  | 40-59 |
|  |  | 60-79 |
|  | Covariant RIAGENDR | No recode needed 1 - Male 2 - Female |
|  | Covariant RIDRETH1 | Race_Recode 1 - Non-Hispanic Black |
|  |  | 2 - Other race |
|  |  | 3 - Non-Hispanic White |
|  | Covariant RIDRETH1 | Ethnicity_Recode |
|  |  | 1- Hispanic (Mexican \& Other Hispanic) <br> 2- Non-Hispanic (Race) |

## Data Collection and Challenges Encountered

Upon receiving Institutional Review Board (IRB) approval (Walden IRB approval \#03-18-20-0131651), secondary data were obtained from the 2007-2008 NHANES dataset. NHANES did not require permission to download the dataset because it is publicly accessible (CDC, 2018b).

## Figure 1

Study Decision Tree


Note. Data were received for 10,149 participants; of these, 4,653 were excluded ( $45.8 \%$ ) based on age ineligibility. Of the 5,496 who were age eligible, $557(10.1 \%)$ were excluded based on missing essential data needed for analysis.

This study used secondary data from the 2007-2008 NHANES dataset, which contained 12,943 screened subjects, of which 10,149 had been interviewed and 9,764 underwent physical examination (Johnson et al., 2013).

The NHANES datasets do not go beyond 80 years of age; therefore, beyond 80 was not included. Compared to the recommended sample from the $\mathrm{G}^{*}$ power analysis $(n=378)$, this study used 4,939 participants who met inclusion criteria. The number of participants self-reporting PreHTN ( $n=286$ ) was below the minimum sample ( $n=378$ ); however, a decision was made to use all eligible participants to answer the research questions. The Demographic and Blood Pressure files from the 2007-2008 NHANES dataset were downloaded and transferred from SAS format to SPSS.

Quality assurance was implemented by checking the data for completeness by comparing files downloaded against the data documentation codebook and frequency count manuals for the variables of interest. Data were cleaned and recoded to facilitate analysis. As described in Table 13, the independent variable ratio of family income to poverty (INDFMPIR) was transformed from a scaled measure (ratio) to a nominal measure (categorical) to evaluate by LMHPG for comparison. By doing so, it was understood that a loss in power would occur; however, this was not the case because all eligible participants were included ( $n=4,939$ ), resulting in a larger sample size. The post hoc analysis using a medium effect size demonstrated achieved power of .99 (see Appendix B).

The rationale behind recoding INDFMPIR using INDHHIN2 (annual household income) data was that there were no assigned values in the former. For example, INDFMIR listed values (e.g., $1.00=$ "range of value") but no numeric income range. The INDHHIN2 had numeric values (e.g., $0-\$ 19,999$ ); therefore, for the subset of data used in this analysis, INDHHIN2 was used in lieu of INDFMPIR. The original plan was to use
the percentage of PIR using the 2007 Health and Human Services Poverty guideline for a household of two $(\$ 13,690)$. This method would have had more samples with a PIR $\geq$ $350 \%(\$ 13,690 \times 350 \%=\$ 18,482 ;$ USDHHS, n.d.a). I felt it best to categorize income groups from INDHHIN2 to PIR_recode (in table 14) using LMHPG, with the lowest serving as the reference group.

The cut point for each category was determined (in table 14). Of the 10,149 participants in the 2007-2008 NHANES dataset, 5,496 met age inclusion criteria; of this number, 557 participants were excluded due to missing independent variable, and the final number of eligible participants was 4,939 (see Figure 1). Another rationale for using poverty group classification is that income is not static (BLS, 2020) and public health interventions aimed at improving population health have not continuously evaluated for different socioeconomic impact of groups (McGill et al., 2015; Vieux et al., 2020).

## Table 14

Poverty Income Group Category

| Name | Label | Group definition |
| :---: | :---: | :---: |
| Lowest poverty group | LPG | $* \geq \$ 55,000$ |
| Middle poverty group | MPG | $\geq \$ 20,000-\$ 54,999$ |
| Highest poverty group | HPG | $0-\$ 19,999$ |

*Cut point determined by poverty group values.

## Figure 2

## Told You Have Prehypertension



Note. $n=6,546$. Original prehypertension question from the 2007-2008 Blood Pressure Questionnaire (BPQ52).
*Not included in final exclusion.
The dependent variable PreHTN was recoded from four possible responses (see Figure 1) to two, 0-no PreHTN and 1—yes PreHTN (see Figure 2). Subjects with missing and "don't know" responses were excluded.

## Figure 3

Prehypertension Recode


Age (RIAGEYR), a continuous measure, was changed to categorical for analysis and recoded three times. The first step was to dichotomize, selecting cases falling within the age range 0 -not of age ( $0-19$ and 80 ) and 1 -yes of age (20-79; see Figure 3). Step 2 required recoding a new variable named filter, which included 0 - not selected and 1 selected.

## Figure 4

Age Recode


Specifying an age range resulted in 5,496 subjects for analysis, representing $54.1 \%$ of the total population; however, 577 were excluded due to missing independent variable information (see Figure 1), resulting in 4,939, representing $48.6 \%$ of the total population $(n=10,149)$ from the 2007-2008 NHANES data.

According to a frequency count, of the 10,149 subjects, $41.5 \%(n=4,214)$ of the data set was $0-19$ years of age and $4.3 \%$ was 80 years of age $(n=439) .{ }^{1}$ It was understood that a loss in power would occur because of transformation; however, the

[^1]subsample was selected because the body of PreHTN prevalence literature used $\geq 20-79$ years of age (Booth et al., 2017; Gupta et al., 2010) and 8-80 years of age for analysis (Hardy et al., 2017).

The third step was to transform and recode age groups by $0-19,20-39,40-59$, $60-79$, and 80. The 2007-2008 NHANES includes populations $<1$; that is why 0 is an included age grouping (CDC, 2006b). At the time of the screening interview, the interviewer documents ages $1-79$; all responses 80 years of age and older are coded as 80 to protect disclosure, and participants $<1$ are coded as 1 year of age (CDC, 2009a). If the date of birth is missing or the respondent refuses to provide data, it is imputed as 7; corrections are made to imputed information for respondents who are $<1$ year of age. When study analysis includes $0-11$ months of age, the investigator can code as 0 . The purpose for age grouping was to get the proportion of the total population not included in the analysis (see Figure 1). The gender variable RIAGENDR did not require recoding because the NHANES dataset list two options: 1—male (reference) and 2—female. There were no missing gender data (see Figure 5).

## Figure 5

## Gender



The NHANES 2007-2008 dataset combined race and ethnicity. To analyze this variable, race/ethnicity (RIDRETH1) was dichotomized, making race and ethnicity separate variables. For example, NHB, Other/Race and NHW was recoded to Race_Recode (see Figure 6). Ethnicity (RIDRETH1) was recoded to Ethnicity_Recode 1—Hispanic and 2-Non-Hispanic. Mexican American and Other Hispanics were in this classification. The non-Hispanic population are listed in the Ethnicity_Recode variable (see Figure 7) to better understand comparison.

Figure 6
Race Recode


Figure 7
Ethnicity


A chi-square $\left(x^{2}\right)$ test of association was conducted between LMHPG and PreHTN. All expected cell frequencies were greater than five. There was a statistically significant association between the independent and dependent variable $x^{2}(d f,=4,939)$ $=8.684$, $p$-value $=<.013$; in Table 18). Statistical Assumptions are explained in Table 15 (p.133).

## Results and Statistical Assumptions

The 2007-2008 NHANES dataset consisted of 10,149 participants (Johnson et al., 2013), of this number, 20-79 years ( $n=5,496$ ) representing $54.2 \%$ of total participants from the dataset were available for analysis. analyzed. The mean age was 48.44, minimum 20, maximum was 79 and standard deviation 16.563. A frequency count was conducted and found IDV and DV missing data ( $n=577$; see Figure 1, p. 124). Missing information is a challenge when using secondary data, it is unknown if screeners skipped questions or participants chose not to respond, these missing data can bias results and how this was handled is discussed in Chapter 5 (pp. 163-164).

Table 15
Assumptions for Binominal Logistic Regression

## Explanation

## 1. Must have one dependent One dependent variable (PreHTN; yes/no). variable that is dichotomous.

## Met

2. Have one or more independent variables that are measured on either a continuous or nominal scale.

Met
3. Independence of observations

Met
4. There should be $\boldsymbol{>} \mathbf{1 5}$ cases per IV

## Met

5. There needs to be a linear relationship between the continuous independent variables and the logit transformation of the dependent variable.

Partially met
6. Data must not show multicollinearity.

Not met
7. There should be no significant outliers, high leverage points or highly influential points.

The continuous variable PIR was recoded to a lowest, middle, highest poverty group (LMHPG). LMHPG is a nominal variable and was used of for chi-square ( $x^{2}$ ) analysis. Nominal measurement is categorical.

Independence of observations, the IDV is ordinal but is being treated as nominal for chi-square ( $x^{2}$ ) analysis. Nominal measurement is categorical and ordinal measurement has the same characteristic as nominal but is ranked and/or ordered. This study treated ordinal as nominal.

Of the 5,496 participants available for analysis, there were 4,939 participants distributed among the IV data (LMHPG; see Figure 1).

The continuous variable (PIR) was transformed into a nominal variable labeled LMHPG.

Partially met means there was a linear relationship between the highest poverty group (2) and PreHTN compared to baseline lowest poverty group (ref). There was no significant relationship between middle poverty group (1) and PreHTN (in table 16).

There was only one IDV (LMHPG) measured at the nominal level. Not met means the IDV was categorical with three levels.

The IDV poverty group was transformed into a categorical variable, and the DV is dichotomous. A binominal logistic regression (BLR) was run on the data, and the casewise list did not detect any outliers. BLR is a subset of logistic regression predicting the probability that an observation falls into one or two categories of a dichotomous

## Explanation

outcome variable if the selections vary by covariates (Rafan et al., 2018).

Research Question 1: Is there an association between Lowest, Middle, and Highest Poverty Group and Prehypertension (PreHTN)?
$H_{0}$ : There is no association between LMHPG and PreHTN. The null hypothesis was Rejected. A chi-square ( $x^{2}$ ) test for association was run demonstrating a statistically significant association between LMHPG and PreHTN, $\chi^{2}(d f, N=4,939), p=<.013$ (in Table 18) and the BLR model was a good fit ( $p=<.011$ ).
$H_{a}$ : There is an association between LMHPG and PreHTN. The alternative hypothesis was Accepted because the nominal predictor variable, LMHPG, in the logistic regression analysis was found to contribute to the model. The unstandardized Beta ( $\beta$ ) weight for the Constant; $\beta=[-3.058], S E=[.119]$, Wald $=[660.923], p<.000$. The unstandardized $\beta$ weight for the Middle Poverty Group was not a statistically significant predictor to the baseline, Lowest Poverty Group $\beta=[.014], S E=[.160]$, Wald $=[.008], p$ .928 (in table 16).

The unstandardized $\beta$ weight for the Highest Poverty Group was a statistically significant predictor to the baseline, Lowest Poverty Group: $\beta=[.367], S E=[.148]$, Wald $=[.013], p<.013$. The estimated odds ratio favored an increase of $44 \%[\operatorname{Exp}(\beta)=$ [1.444], $90 \%$ CI $(1.132,1.841)$ for PreHTN for the Highest Poverty Group compared to the baseline, Lowest Poverty Group (in table 16).

## Table 16

Association Between LMHPG and Prehypertension

|  | $B$ | $S E$ | Wald | $d f$ | Sig | $\operatorname{Exp}(\beta)$ |
| :--- | :--- | :--- | :--- | :--- | :--- | :--- |
| *Lowest poverty group |  |  | 9.235 | 2 | .010 |  |
| Middle poverty group (1) | .014 | .160 | .008 | 1 | .928 | 1.015 |
| Highest poverty | $(2)$ | .367 | .148 | .013 | 1 | .013 |
| Constant | -3.058 | .119 | 660.923 | 1 | .000 | .0474 |

*Variables entered on Step 1: Poverty groups.

Table 17

Association Between LMHPG and Prehypertension

|  |  | $90 \%$ CI for <br> lower | $\operatorname{Exp}(\beta)$ <br> upper |
| :--- | :--- | :---: | :---: |
| Step 1 $^{\mathrm{a}}$ | * Lowest poverty group |  |  |
|  | Middle poverty group (1) | .780 | 1.320 |
|  | Highest poverty group (2) | 1.132 | 1.841 |
|  | Constant |  |  |
|  |  |  |  |

*Variables entered on Step 1a: Poverty groups.
Research Question 2: Is there an association between LMHPG and PreHTN when age, gender, race (NHB, Other Race, and NHW), and ethnicity (HcNH) are removed from analysis?
$H_{0}$ : There is no association between LMHPG and PreHTN controlling for age gender, race, and ethnicity $(\mathrm{HcNH})$. The null hypothesis is Rejected.

A logistic regression analysis to investigate the association between LMHPG and PreHTN was conducted. The predictor variable LMHPG was tested a priori using the G*Power software to verify there were no violation of assumptions of the linearity of the logit.

The predictor variable LMHPG, in the logistic regression analysis was found to contribute to the model $(p=.011)$. The unstandardized $\beta$ weight for the Constant; $\beta=[-$ 4.588], $S E[.356]$, Wald $=[166.313], p<.010$ (in table 17, p. 136). The unstandardized $\beta$ weight for the predictor variable: $\beta$ [.177], SE [ .079], Wald= [9.235], $p<.026$ shows for every one unit increase in poverty group, the estimated odds ratio for PreHTN increases by $19 \%[\operatorname{Exp}(\beta)=1.194], 90 \% \mathrm{CI}(1.048,1.360), p<.026$ (in table 17, p. 136).
$H_{a}$ : There is an association between LMHPG and PreHTN when adjusting for age, gender, race, and ethnicity (HcNH)? Partially accepted, an association continued after accounting for age. This means for every one (1) unit increase in age, the odds of PreHTN increased by $3 \%, \beta[0.29]$, $\mathrm{SE}[004]$, Wald $=[55.628], p<.000,90 \% \mathrm{CI}(1.023$, 1.036), $p<.000$ (in table 17, p. 136).

The association did not continue after accounting for gender ( $p=.631$ ), race (ref), and ethnicity ( $p=.746$ ). Race ( NH ) was selected as the baseline data because its frequency count was greater ( $n=3,853$ ) compared to Hispanic ( $n=1,643$ ). Therefore, Hispanic is coded as (1) and non-Hispanic (Race) is coded (2).

## Table 18

Model Controlling for Age, Gender, and Ethnicity

|  | $B$ | SE | Wald | $d f$ | Sig | $\operatorname{Exp}(\mathrm{B})$ |
| :---: | :---: | :---: | :---: | :---: | :---: | :---: |
| Step1 ${ }^{\text {a }}$ LMHPG | . 177 | . 079 | 9.235 | 1 | . 026 | 1.194 |
| Gender | -. 059 | . 123 | . 231 | 1 | . 631 | . 943 |
| Age_Recode | . 029 | . 004 | 55.628 | 1 | . 000 | 1.030 |
| Ethnicity_Recode (1) | -047 | . 145 | . 105 | 1 | . 746 | . 954 |
| Constant | -4.588 | . 356 | 166.313 | 1 | . 010 | . 047 |
| *Variables entered on Step 1 ${ }^{\text {a }}$ : LMHPG, Gender, Age_Recode, \& Ethnicity_Recode. |  |  |  |  |  |  |
| Model controlling for age, gender, and ethnicity |  |  |  | 90\% CI for lower |  | $\operatorname{Exp}(\mathrm{B})$ upper |
| Step 1 ${ }^{\text {a }}$ LM |  |  |  | 1.048 |  | 1.360 |
|  |  |  |  | . 770 |  | 1.154 |
| Age_Recode |  |  |  | 1.023 |  | 1.036 |
| Ethnicity_Recode (1) |  |  |  | . 751 |  | 1.212 |
| Constant |  |  |  |  |  |  |

*Variables entered on Step 1á: LMHPG, Gender, Age_Recode, \& Ethnicity_Recode.

Question 3: Is there a difference in the prevalence of PreHTN between Lowest, Middle, and Highest Poverty Group when age, gender, race, and ethnicity ( HcNH ) are removed from analysis?
$H_{0}$ : There is no difference in the prevalence of PreHTN between Lowest, Middle, and Highest Poverty Groups when age, gender, race, and ethnicity (HcNH) are removed from analysis. This hypothesis was rejected.
$H_{a}$ : There is a difference in the prevalence of PreHTN between Lowest, Middle, and Highest Poverty Groups when age, gender, race, and ethnicity ( HcNH ) are removed
from analysis, The alternative hypothesis was accepted. A chi-square ( $\mathrm{x}^{2}$ ) test of association was conducted between LMHPG and PreHTN adjusted for age, gender, race, and ethnicity $(\mathrm{HcNH})$ and there was a statistically significant difference between the IDV and DV $x^{2}(d f, N=4,939)=8.684, p=<.013$ (in table 19).

Table 19
Prevalence of Prehypertension by Poverty Group

| Poverty group | PreHTN |  |  |
| :--- | :---: | :---: | :---: |
|  | No | Yes | Total |
| Lowest | 1,306 | 69 | 1,375 |
|  | $(95 \%)$ | $(5 \%)$ |  |
| Middle | 1,558 | 82 | 1,640 |
|  | $(95 \%)$ | $(5 \%)$ |  |
| Highest | 1,789 | 135 | 1,924 |
|  | $(93 \%)$ | $(7 \%)$ |  |
| Total | 4,653 | 286 | $* 4,939$ |
|  | $(94.2 \%)$ | $(5.8 \%)$ |  |

*Represents valid $n=4,939$ out of $n=5,496, X^{2}(d f, N=4,939)=8.684, p=<.013$. Adjusted for age, gender, race, and ethnicity ( HcNH ).

## Chapter 5 explains findings, study research limitations, describes

recommendations for study replication, discusses positive social change and considers realistic steps based on study's findings to reduce PreHTN in the United States.

Chapter 5: Discussion, Conclusions, and Recommendations

## Introduction

The association between poverty group and PreHTN found in this study builds upon the body of PreHTN research. Guo et al. (2013a) reported that PreHTN in the lower range (120-129/80-84) $R R 1.24,95 \% \mathrm{CI}[1.10-1.39], p<0.001$ and upper range (130-139/85-89) $R R 1.56,95 \% \mathrm{CI}[1.36-1.78], p<0.001$ were associated with increased risk of developing and dying of total CVD disease. Upon further analysis, Guo et al. (2013b) later reported that both ranges of PreHTN pose a risk of CVD mortality $R R 1.32,95 \%$ CI [1.16, 1.50], $p<0.001$. However, when stratified by range, the upper range $R R 1.26,95 \%$ CI [1.13, 1.41], $p<0.001$, compared to the lower range $R R 1.10,95 \%$ CI $[0.92,1.30]$, $p=0.287$, demonstrated greater risk of CVD mortality.

Faustini and Davoli (2020) noted that population attributable risk (PAR) "helps determine which exposures are most important to a specific community and is calculated as the incidence of a disease in the total population minus the incidence of the group of those unexposed to the specific risk factor" (p. 3). According to Han et al. (2019), PAR associated with PreHTN for CHD was $13.26 \%$, MI $26.6 \%$, and Stroke $19.15 \%$. This finding is important because effective control of PreHTN could potentially prevent $>10 \%$ of CV cases. In a subset of the Jackson Heart Study $(n=3,770)$ without prevalent CVD Clark et al. (2018) reported PAR associated with HTN and PreHTN in African Americans was $42 \%$. The PAR data reinforce the importance of PreHTN primary prevention, especially in that my findings demonstrated a statistically significant association between poverty group membership and PreHTN.

A major limitation with Han et al. (2019) meta-analysis was the inability to account for environmental exposures such as poverty that place specific groups at elevated risk.

My findings demonstrated that being in the highest poverty group is associated with increased risk of PreHTN by $44 \%$ ( $<.10$ ) compared to baseline lowest poverty group; there was a significant difference between being in the highest poverty group compared to lowest poverty group ( $p<.013$ ). If poverty were decreased, PreHTN as a risk factor might prevent CVD cases.

PreHTN has been reported in pediatric epidemiologic studies with estimated prevalence ranging from $12.7 \%$ (Lo et al., 2013) to $31.4 \%$ (Koebnick et al., 2013). There is evidence that at 8 years of age, $<89 \%$ of males compared to $>96 \%$ of females have ideal BP (Hardy et al., 2017). PreHTN among this same group is higher among males (12\%) compared to females (6\%). By 19 years of age, the PreHTN burden is observed in males and African Americans (Hardy et al., 2017). The relevance of this study is that it provides evidence of the importance of maintaining normal BP, specifically among youth, and this is a public health concern.

The cost to treat PreHTN is the same as the cost to treat HTN (Chen et al., 2017). According to Moise et al. (2016), treating high-risk groups such as those with PreHTN can reduce CVD events, improve quality of life, and save money. Muntner et al. (2018) estimated HTN prevalence at $45.6 \%$, which is 103.3 million people $\geq 20$ years of age. Ostchega et al. (2020) used 2017-2018 data to estimate HTN prevalence in $>18$ years of age at $45.4 \%$, with $22.4 \%$ in adults $18-39$ years of age.

HTN prevalence increases with age and delaying incident HTN could reduce needless suffering and minimize costly treatment.

By preventing PreHTN and delaying incident HTN, public expenditures could be used to rebuild deteriorating neighborhoods to improve home values that may translate into family wealth. Better home values may influence improved health outcomes (Mehdipanah et al., 2017), promote safer neighborhoods to walk, lead to the construction of quality bicycle networks (Lui \& Shi, 2017), attract full-service grocery stores versus low-end convenience stores (Shannon, 2020), and move Americans from a sick care environment to primary CVD prevention and health promotion. The savings could also be invested to close the lineage/racial wealth gap (Bhutta et al., 2020; Federal Reserve [FR], 2021, 2020b; Hamilton \& Linden, 2018).

The paucity of literature examining the association between LMHPG and PreHTN is of concern because public health interventions aimed at improving population health have not continuously evaluated for the different socioeconomic impact of groups. The purpose for this quantitative, nonexperimental, cross-sectional research was to examine the association between LMHPG and PreHTN when controlling for age, gender, race, and ethnicity (HcNH); and if that association varies by participants, responding yes or no to having been told by a doctor or other health professional that they have PreHTN.

Narratives and tables were used to illustrate estimated PreHTN prevalence and test the null hypothesis, and logistic regression was used to assess the association between LMHPG and PreHTN from 4,939 out of 5,496 non-institutionalized U.S. citizens ages 20-79 years of age (see Figure 1, p. 124).

The review of literature for this research evaluated findings that used 95\% CI estimates and conservative alphas (e.g., $<0.05$ ); however, my key findings are based on $90 \%$ CI estimate and a less conservative alpha. $<10$, and this decision has been discussed previously.

## Key Findings

- The predictor variable LMHPG was found to contribute to the logistic regression model and was statistically significant. The estimated odds ratio favored an increase of $44 \%[\operatorname{Exp}(\beta)=[1.444], 90 \% \mathrm{CI}(1.132,1.841) p=$ $<.010$ for PreHTN in the HPG compared to the baseline, LPG.
- The estimated odds ratio favored a statistically significant increase of $19 \%$ $[\operatorname{Exp}(\beta)=1.194], 90 \% \mathrm{CI}(1.048,1.360), p=<.026$ in PreHTN for every one unit increase in poverty group.
- Even after adjustments for age in the BLR, there remained a $3 \%[\operatorname{Exp} \beta)=$ [1.030], $90 \% \mathrm{CI}(1,030,1.036), p=.000$ increase in poverty group membership for age. This might be attributed to reduction in household income due to retirement.
- The review of literature demonstrated statistically significant associations between Race and PreHTN; my analysis adjusted for the confounder, and a statistically significant association was not observed. However, a within-Race category (NHB, NHW and OR) subanalysis assessed for association between poverty group and PreHTN. Using the $90 \%$ CI estimate and $<.10$ alpha
showed that being NHB and in the HPG was a statistically significant predictor of PreHTN, OR $1.50,90 \%$ CI [1.049, 2.152], $p=.062$, compared to baseline HPG NHW, OR 1.12, $90 \%$ CI [.934, 1.356], $p=.298$, which was not a statistically significant predictor (in table 20).
- The overall prevalence of PreHTN by LMHPG was $5.8 \% x^{2}(d f, 4,939)=$ 8.684, $p=<.013$.

Table 20
Subanalysis Within Race Category Model


## Interpretations of Findings

In this section, I describe whether findings confirmed, disconfirmed, and extended the body of PreHTN health disparities literature using the CRT framework. The CRT framework was used because race and ethnicity were covariates for the research and had been reported as strengthening the relationship between the independent and dependent variables in public health studies and educational research (Allen \& White-Smith, 2018; Aymer, 2016; Freeman et al., 2017; Mayne et al., 2019). Research Questions 2 and 3 adjusted for race and ethnicity because it is known in empirical literature as a risk factor. However, a within-race subanalysis observed that being in the HPG and NHB was a significant predictor of PreHTN (in table 19, p. 139). Stratification was a good method for examining the NHANES 2007-2008 dataset because aggregated race obscured outliers and did not provide a clear picture of specific risk groups. This is where evaluating public health interventions by socioeconomic groups is paramount.

PreHTN is precursor to incident HTN and has been associated with CVD events (Han et al., 2019; Huang et al., 2015) and renal dysfunction (Ku et al., 2019). PreHTN prevalence has also been identified in pediatric studies (Koebnick et al., 2013; Lo et al., 2013) and the annual net transition probabilities from ideal BP to PreHTN over the life course (Hardy et al., 2017) indicating an early life presence.

To answer the first research question (Is there an association between LMHPG and PreHTN?), a chi-square ( $x^{2}$ ) test of association was conducted, and a significant association was confirmed, $\chi^{2}(d f, N=4,939), p=.031$, and was found to contribute to the BLR model.

To answer the second research question (Is there an association between LMHPG and PreHTN controlling for age, gender, race, and ethnicity?), the BLR model observed that for every one unit increase in poverty group, the estimated $O R$ for PreHTN increased by $19 \%,[\operatorname{Exp}(\beta)=1.194], 90 \% \mathrm{CI},(1.048,1360), p=<.026$. The 2007-2008 NHANES dataset aggregated race, which obscured within-group association between LMHPG and PreHTN.

A within-group subanalysis was conducted using $90 \% \mathrm{CI}$ and $<.10$ alpha and found that being in the HPG and NHB increased the $O R$ for PreHTN by $50 \%,[\operatorname{Exp}(\beta)=$ $1.503], 90 \% \mathrm{CI},(1.49,2.152), p=<.062$ compared to HPG and NHW OR $1.125,90 \%$ CI, (.934, 1.356), $p=.298, O R$ and Other Race $O R .971,90 \% \mathrm{CI},(.658,1.434), p=.902$ (in table 20). The use of BLR for the subanalysis confirmed that marginal groups are at increased risk of PreHTN and would have been overlooked when aggregated data are used.

The third research question identified a statistically significant difference in prevalence of PreHTN by LMHPG controlling for age, gender, race, and ethnicity ( $p=.013$ ). My findings build upon the PreHTN disparities literature that adjusted for confounders (Glasser et al., 2011; Gupta et al., 2021; Madanat et al., 2014; Su et al., 2014) or as an independent variable for health outcomes (Wilson et al., 2017).

To date, there has been no specific research examining the association of LMHPG and self-report of PreHTN. My findings demonstrated a statistically significant association between stratified income groups and PreHTN; specifically, being in the HPG
$\leq \$ 19,999$ was associated with self-report of PreHTN. A strong case can be made that HPG membership is risk factor for PreHTN.

The only known research using income as an independent variable with high BP as one of many outcomes was that of Wilson et al. (2017), who used household income $>\$ 175,000$ to estimate racial disparities in health outcomes. The association between income group and EBP is not limited to the HPG as reported by Wilson et al. (2017). High income group membership may not buffer against adverse health outcomes for specific demographic groups (Assari, 2018; Assari et al., 2018; Vazquez-Vidal et al., 2016) to include buffering against unfair treatment during childhood (Assari et al., 2021), thus indicating racial and/or ethnic group as a CRT attribute may account for differences. The subanalysis conducted for this dissertation observed that being NHB and in the HPG increased PreHTN risk.

The current research found that after adjusting for age using BLR, the covariant remained a statistically significant predictor of PreHTN OR $1.03,90 \%$ CI [1.023, 1.036], $p<.10$. Similar conclusions were found by Madanat et al. (2014) that after adjustment, age remained a predictor for PreHTN among Latina Women 18-72 years of age, $O R$ $1.03,95 \%$ CI [1.00, 1.05], $p<0.05$.

The CARDIA study reported that PreHTN $<35$ years of age independently predicted a graded association with coronary calcium (cc) later in life (cc prevalence $15 \%, 24 \%, 38 \%$ for $0,1,30$, and $>30 \mathrm{mmHg}$ years exposure, $p<0.001$ ), and the risk remained after 35 years of age (Pletcher et al., 2008). Rafan et al. (2018) reported that age as a predictor was associated with an $6 \%$ increased risk in PreHTN $a O R 1.06,95 \% \mathrm{CI}$
[1.02, 1.11], $p<0.007$. Conversely, other researchers have reported greater predictive odds of PreHTN controlling for age (Glasser et al., 2011; Kim \& Lee, 2015).

Among a Korean population, compared to 20-39 years of age, 40-59 years of age were predictors of PreHTN $a O R 1.79,95 \%$ CI [1.55,2.05], $p<0.05$ (Kim \& Lee, 2015). An increase odds of PreHTN by age (per 10 years) was reported by Glasser et al. (2011) among a biracial cohort, NHW OR 1.43, $95 \% \mathrm{CI}$, [1.35, 1.53], NHB OR 1.30, $95 \% \mathrm{CI}$, [1.18, 1.44], $p<0.001$. Within the study, household income of $\$ 20,000$ was a significant predictor of PreHTN among NHW OR 1.26, $95 \%$ CI, [1.01, 1.56], $p<0.001$ but not for NHB OR $0.95,95 \% \mathrm{CI},[0.69,1.31]$. The higher predicted odds ratio might be attributed to the study having 56\% participation from the Stroke Belt/Buckle Region of the United States. Within the study, PreHTN prevalence was higher (65.2\%) among NHB with household incomes $<\$ 20,000$ compared to NHW (58.6\%). Other REGARDS analysis have been reported $>70 \%$ HTN prevalence and $24 \%-32 \%$ household incomes (HHI) $<\$ 20,000$ among NHB compared to $>55 \%$ HTN prevalence and $11 \%-18 \%<\$ 20,000 \mathrm{HHI}$ among NHW (Limdi et al., 2016). A conclusion can be drawn that NHB are less likely to have higher household incomes and more likely to have HTN compared to their NHW peers.

Among middle-aged females, Kim and Lee (2015) reported a moderate association between large waist size and PreHTN, $a O R 1.04,95 \%$ CI, [1.00,1.07], no pvalue given. Koskinen et al. (2019) reported that having PreHTN in early childhood predicted preclinical atherosclerosis in adulthood. Within the study parental income was not used as a control variable.

In a CARDIA analysis, compared to participants without elevated systolic PreHTN $<35$ years of age, those with PreHTN with a graded association across 0, 1-30, and $>30 \mathrm{mmHg}$ of exposure years, independently predicted a greater relationship with Coronary Calcium, a cardiovascular risk (Coronary Calcium prevalence $15 \%, 24 \%$, and $38 \%, p<0.001$; Pletcher et al, 2008). In other words, the higher the BP, the greater the CVD risk. Income was not a predictor; however, the author did report participants with incomes $<\$ 25,000$ were just as likely as those with incomes $>\$ 100,000$ to be PreHTN.

In an analysis of Framingham Heart Study participants, at Year 4, having PreHTN at baseline (34-64 years of age) predicted incident HTN OR 11.6, 95\% CI [9.6, 14.0], $p<0.001$ (Vasan et al., 2001). Kanegae et al. (2017) reported at Year 8, incident HTN in $<50$ years of age was significantly higher in the isolated diastolic high-normal (IDHN; SBP $<130 \mathrm{mmHg}$ and DBP $85-89 \mathrm{mmHg}$ ) group compared to isolated systolic highnormal blood (ISHN; SBP 130-139 mmHg and DBP $<85 \mathrm{mmHg}, p<0.001$.

BP variability that progress to incident HTN places populations at risk for CVD morbidity (Clark et al., 2017; Guo et al., 2013a; Han et al., 2019; Huang et al., 2013; Qureshi et al., 2005; Yano et al., 2017) and CVD mortality (Huang et al., 2020; Ford et al., 2011). Income as a predictor of PreHTN by age were not found in the above literature, this lends evidenced to the paucity of research examining the association between LMHPG and PreHTN.

Gender was not a predictor in my the LMHPG and PreHTN BLR model. A similar conclusion was reported by Owiredu et al. (2019) when adjusting for gender was not a statistically significant predictor of PreHTN $a O R 1.48,95 \% \mathrm{CI}(0.84,2.61) p<.17$.

Authors limitations include absence of income data, and the study took place in Ghana, where per capita annual income is $<\$ 5,000$ U.S. dollars (CIA, n.d.b). Kim and Lee (2015) reported middle-aged women $\geq 60$ with large waist circumference were a predictor of PreHTN compared to their age matched peers $a O R 1.04,95 \%$ CI [1.00, 1.07], no pvalue was given. Among student athletics, male gender was a predictor of EBP compared to females $O R 1.28(1.15,1.41), p<.005$ (Stiefel et al., 2016). A major limitation identified during the review of literature was most studies measured PreHTN prevalence (Gupta et al., 2010; Koebnick et al., 2013; Lo et al., 2013); CV risk associated with PreHTN (Clark et al., 2017; Glasser et al., 2011; Guo et al., 2013a; Han et al., 2019; Huang et al., 2020; Koskinen et al., 2018); early life predictors of later life CV risk (Hardy et al., 2018); CV events associated with PreHTN (Huang et al., 2020; Qureshi et al., 2005; Yano et al., 2017); PreHTN to HTN progression (Carson et al., 2011; Ishikawa et al., 2017; Selassie et al. 2011); metabolic risk associated with EBP (Casey et al., 2017; Mullican et al., 2009; Stiefel et al; 2016); CV risk factors among females (Hsia et al., 2007) and trend studies (Booth et al., 2017; Hardy et al., 2019). Few if any used income as a predictor and PreHTN as an outcome.

My study is guided by critical race theory (CRT) framework which posits that Racism is a system intricately woven into the fabric of the United States and used by Euro-Americans (EA) and Honorary EA to maintain advantage over non-EA's, specifically NHB, as articulated by legal scholars from the Civil Rights era (Bell, 1975; Delgado, 1989; Freeman, 1978). Leveraging the CRT framework was useful for RQ\# 2 because a subanalysis found that being in the HPG and NHB was a statistically
significant risk for PreHTN (in table 20). Bonilla-Silva (2004) describes honorary Whites/EA as light skin Latinos, Americans of Chinese/Filipino/Korean/Japanese descent, Asian Indians, most Multiracial (e.g., black father/white mother and vice versa) and Middle Eastern Americans (e.g., Iranians, Israelis, Syrians, etc.).

Race is the offspring of Racism, and it is used at all social levels (e.g., macro, meso, and micro) to assign values and structure opportunities across generations. This means that Racism is a system which assigns values and structures opportunities based on racial group classification and too some extent lineage. Theses classifications are required to maintain a bottom caste across generations. Within this research analysis, Race as an aggregate did not contribute to the LMHPG and PreHTN BLR model. Therefore, a within Race subanalysis was conducted. Being NHB in the HPG was associated with 50\% increase odds of PreHTN OR 1.50, $90 \%$ CI [1.049, 2.152], $p<.062$ compared to NHW OR $1.125,90 \%$ CI $[.934,1.356], p>.298$; and Other Race $O R .971,90 \%$ CI [.658, 1.434], $p>902$. The differences between the Race aggregate compared to within Race subanalysis demonstrated that outliers can be obscured and health promotion opportunities to mitigate PreHTN in specific groups could be missed.

This study's subanalysis finding was opposite to Glasser et al. (2011) where NHW with household income at $\$ 20,000$ had a $26 \%$ increased odds of PreHTN compared to NHB, OR $0.95,95 \% \mathrm{CI}[0.69,1.31], p<0.001$. Pletcher et al. (2008) reported the presence of PreHTN $<35$ years of age where more likely to be Black race/ethnicity $a O R$ $2.00,95 \% \mathrm{CI}[1.6,2.4], p<.001$, and male gender aOR 4.2, $95 \% \mathrm{CI}[3.4,5.1], p<.000$. Exposure to systolic PreHTN $<35$ years of age showed a graded association with
coronary calcium risk prevalence $15 \%, 24 \%$, and $38 \%$ for $0,1,30$ and $>30 \mathrm{mmHg}$ years of exposure, respectively p. $<0.001$. This association remained $>35$ years of age. Within the study, NHB represented $46 \%$ of the PreHTN group, lower income $<\$ 25,000$ with no college education group and were twice as likely as participants with post graduate education and income $>\$ 100,000$ to be PreHTN prior to 35 years of age. The takeaway is a stratified subanalysis showed PreHTN racial group differences, and exposure prior to 35 years of age was a strong predictor of later in life coronary calcium risk.

My subanalysis by Race found that being NHB in the HPG, strengthen the connection between the IDV and DV. When investigating health disparities, stratified Race should be leveraged to estimate burden, predict risk, and measure associations. Evidence report racial group classification, specifically lineage, determines NHB's social trajectory (Collins \& Wannamaker, 2017).

Research Question \#2, subanalysis found that being NHB in the HPG predicted PreHTN demonstrating specific group risk. On the other hand, researchers have found regardless of income, NHB with annual incomes of $>\$ 175,000$ had greater odds of selfreporting HTN $O R 2.8,95 \% \mathrm{CI}[1.17,4.7]$, no p-value given, compared to Asian, Hispanic and NHW (ref; Wilson et al., 2017). In the context of this dissertation findings and body of health disparities literature, racial group membership continuously shape BP patterns in the U.S (Colangelo et al., 2020; Hardy et al., 2017; Krieger \& Sidney, 1996).

Using CRT as an analytical tool in public health research has been used by Aymer (2016) to describe the psychosocial stressors of state sanctioned violence on the lives of Black men.

Research reports prolonged exposure to stress may impair the regulatory functions of the Hypothalamic-Pituitary-Adrenal (HPA) Axis which is responsible for fight or flight behavior via excess glucocorticoids (e.g., cortisol) and has been linked to BP regulation (Goosby et al., 2018; McEwen, 2000). There is a relationship between stress (e.g., Exposure to unfair treatment) and EBP that could be inherited. This is relevant to the Research Question \#2 because Race and Ethnicity are control variables.

The relationship between stress and elevated BP has been shown among offspring's of prenatally stressed rats that demonstrated greater cardiovascular responses (e.g., SBP) to restraint stress $F=5, \mathrm{p}<0.05$ and recovery $F=4.4, \mathrm{p}<0.05$ compared to controls (Igosheva et al., 2007). Intergenerational stress transmission is an emerging discipline (Franklin et al., 2010). In humans, prenatal stress among marginalized groups has been reported as a contributor to offspring's later in life cardiovascular risk (Reynolds et al., 2013).

Public health researchers have reported an association between anxiety and HTN (Bacon et al, 2014); anxiety, depression, stress and HTN (Haggart et al., 2018; Mushtaq \& Najam, 2014). A limitation with Bacon et al. (2014) is the study took place in Canada, and the strength include a population free of HTN at baseline and within 1 year almost half of the participants progressed to HTN. A strength of Haggart et al. (2018) was the study took place in the U.S. with a young adult population (18-39 years of age) and the use of electronic health records to validate outcomes. Limitation includes lack of income as a predictor of EBP.

Mushtaq and Najam (2014) study took place in Pakistan which does not lend generalizability in the U.S. and the strength was the use of a covariant that showed a decrease in the odds of HTN with the security of having a monthly income.

Critical race theory (CRT) as a framework was used by Freeman et al. (2017) to understand poor health care engagement among a sample of HIV positive NHB and Hispanic Black (HB) individuals ( $n=37$ ); where participants described how historical and contemporary abuses by the dominate group create mistrust and how institutional level actors (e.g., physicians) exacerbated mistrust by treating clients as invisible. The participants motivation to seek health services served as a counter narrative challenging structural barriers created via Racism. Mistrust in healthcare providers may contribute to higher chronic condition burden among racial ethnic populations (Bazargan et al., 2021).

Ethnicity (HcNH) did not contribute to the model and a subanalysis was not performed because there were only two (2) options, Hispanic and non-Hispanic. Based on review of literature, I believed Hispanic ethnicity would have contributed to the model. For example, in a subset of Women's Health Initiative (WHI), a history of depression at baseline among Hispanic women was associated with PreHTN OR 1.27 (1.01, 1.61) (Zambrana et al., 2016). A major limitation among ethnic WHI participants was not adjusting for the $40 \%$ of Hispanics and American Indians with family income $<\$ 20,000$. It was also unknown what specific Hispanic (e.g., Mexican, Cuban, etc.) or American Indian ethnic groups (e.g., Chippewa, Seminole, etc.) were assessed and whether they self-identified as White.

There is evidence that Hispanics who self-identify as White have a $52 \%$ increased odds of reporting excellent and very good physical health compared to their peers who do not identify as White (Lopez et al., 2018). Hispanics who socially identify as Minority /White $(\mathrm{M} / \mathrm{W})$ are more likely not to report health care discrimination $a O R=0.61,95 \%$ CI $[0.39$, $0.95], p<0.05$ compared to their minority peers (ref; MacIntosh et al., 2013). Within the analysis $56 \%$ of NHW households reported income $>\$ 35,000$ compared to $43 \%$ (MW) and $31.4 \%$ Minority/Minority. The White Advantage among Hispanics might contribute to within group BP differences (Elfassy et al., 2020).

Among Latino women ( $n=331$ ), compared to normotensive ( $n=130$ ), sugary drink consumption was reported as a risk factor for PreHTN $(n=148)$ OR $1.34,95 \%$, CI [1.00, 1.80], $p<.047$ (Madanat et al., 2014). On the other hand, high income ( $>\$ 175,000$ ) Hispanics were $30 \%$ less likely to have HTN OR . $07,95 \%$ CI [0.4, 1.2], compared to NHB 2.8, $95 \%$ CI [1.7, 4.8] no-p value given (Wilson et al., 2017). Hispanics are not a monolith, among a group of HTN free Hispanics at baseline, at follow-up, compared to other Hispanics, Cubans had a higher $I R R$ for HTN 45 per 1,000 PY, $p<0.05$ (Elfassy et al., 2020). Sorlie et al. (2014) reported the highest prevalence of HTN was among Dominican men (34.3\%) and lowest South American Women (17.2\%), and $<50 \%$ of total participant income ranged between $\$ 10,000-\$ 40,000$. The income level cited by Sorlie et al. (2014) may provide insight into the higher PreHTN prevalence among the Hispanic MPG (36\%) compared to LPG (33.3\%) and HPG (30.7\%) found in the dissertation subanalysis (not shown). The United States Census Bureau allow Hispanics to self-identify as any race, which may account for $53 \%$ self-reporting as White (USCB,

2011; USCB, 2020a). This is relevant because Research Questions 2 and 3 adjusted for race and ethnicity. The proportion of Hispanics living with PreHTN might be underestimated and this group could be at elevated CV risk.

An association between stress and EBP in ethnic populations have been reported in the literature (Ford et al., 2016; Hicken et al., 2014; Rammah et al., 2019; Tomfohur et al., 2016; Zambrana et al., 2016).

Psychosocial stress (e.g., anxiety and depression) and HTN was reported among a sample of Mexican and Hispanic origin population in Texas, compared to controls, case prevalence odds ratio $(P O R$ ) was $1.36,95 \%$ CI $[1.06,1.75], p<0.05$ (Rammah et al., 2019). PreHTN is a significant risk factor for HTN, and stress has been linked is linked to HTN.

Mixed conclusions regarding EBP and discrimination by ethnic groups have been reported. For example, in a population of African American (AA; $n=130$ ) and Latina Women (LW; $n=48$ ); racial discrimination was positively correlated to stress ( $r=.229$, $p<.02$ ) and negatively correlated to ethnicity ( $r=-.277, p<.001$; Shin et al., 2017). Within this sample, $43.8 \%$ were PreHTN, $>50 \%$ of participants reported incomes above federal poverty, indicating income has limited buffering against elevated BP correlated to racial discrimination.

Ethnicity in the U.S. has been described as Hispanic or non-Hispanic (USCB, 2020a). Others have described ethnicity as shared histories, beliefs, practices, lineage, and rituals conferring a sense of pride and identity (Helms \& Talleyrand, 1997; Markus, 2008).

The problem with ethnicity is that ethnic group affiliation does not necessarily expose groups to unfair treatment because of skin color and lineage; in fact, USCB extends honorary White identity to specific African, Asian subgroups, and to any Hispanic group (USCB, 2020a).

A central challenge confirming LMHPG and PreHTN among Hispanic/Latinos, might be attributed to Hispanic as an ethnic group overwhelming self-identify as White (Golash-Boza \& Darity, 2008; USCB, 2011) and White identity may offer levels of health protection (Assari, 2018; MacIntosh et al., 2013; Malat et al., 2018). However, the level of protection has limitations for specific Hispanic groups (e.g., Cubans, Puerto Ricans).

The overall PreHTN prevalence based on self-report in the study was $5.8 \%$. Prevalence differed by poverty group (in table 18, p. 138). The lower prevalence found might be attributed to having three (3) poverty groups and using one (1) NHANES data cycle. PreHTN prevalence by income group did not align with Glasser et al. (2011) who used a larger population and four years of data. PreHTN prevalence without income as a control variable in various studies have been reported. For example, Booth et al. (2017) reported at 28\%; 42\% (Casey et al., 2017); 36.3\% (Gupta et al., 2010); 31.3\% (Mullican et al., 2009); 27\% (Toprak et al., 2009) and 41\% (Qureshi et al., 2005).

The hypothesis that PreHTN prevalence would differ by age was supported by the literature (Glasser et al., 2011; Gupta et al., 2010; Hardy et al., 2017; Qureshi et al., 2005), except for Glasser et al. (2011), these studies did not adjust for income or use it as an IDV.

The findings from this research identified a trend, as populations aged, so did the prevalence of PreHTN increase between poverty group (not shown), albeit nonsignificant. Less than ideal BP was to be expected since HTN increases with age (Ostchega et al., 2020), with some demographic groups transitioning from PreHTN to HTN later in their adult life (Hardy et al., 2019).

Within the Coal Miners Study, higher PreHTN prevalence was observed in 15-44 years of age (57\%), and lowest in $>65$ years of age ( $33 \%$; Casey et al., 2017). In the Framingham Heart Study (ages 20-80) the PreHTN prevalence was not categorized by age, however, within the study, $\mu$ age was $42.9 \pm 8.2$, and slightly older than normotensive ( $p<0.05$; Qureshi et al., 2005).

Glasser et al. (2011) reported PreHTN prevalence was highest in 55-64 years of age group (43\%) and lowest in $\geq 75$ years of age group (12\%). The low prevalence in the $\geq 75$ years of age group may be attributed to the group's larger national HTN burden (74.5\%; Ostchega et al., 2020). Older age has been reported as a risk factor for incident HTN in prospective studies (Vasan et al., 2001, 2002). Among young adults (20-44) Toprak et al. (2009) reported PreHTN prevalence at $27 \%$, the problem with this data is it was not broken down by age, however, $\mu$ age was $36+5$. Within the study, PreHTN was not controlled by income, however, another study using BHS subset ( $n=1,266$, NHB $n=$ 325 , and NHW $n=942$ ), reported $63 \%$ of NHB had household incomes $<\$ 15,000$ compared to NHW 23\% (Deshmukh-Taskar et al., 2007).

The criterion for this study was 20-79 years of age with both the independent and dependent variable (see Figure 1, p. 124).

Even though study inclusion included populations 20-79 years of age, it must be noted that Lo et al. (2013) reported the prevalence of PreHTN was $>25 \%$ among $15-17$ years of age. Annual net transition probabilities between ideal BP and PreHTN has been reported at 8 years of age where certain kids transitioned from ideal BP to PreHTN earlier than others (Hardy et al., 2017). Less than normal BP in youth is predictive of adult PreHTN and HTN (Theodore et al., 2015). Among young athletics, a population perceived to be healthy, investigators reported EBP (Kropa et al., 2016; Stiefel et al., 2016). This early warning can serve as a catalyst to implement population wide strategies to minimize incident HTN and later in life adverse CV events. Minimizing EBP among our youth must be a public health priority.

A subanalysis of PreHTN prevalence in this study by gender did not show a significant difference in males (5.6\%) compared to females (5.8\%; not shown). The overall PreHTN prevalence by gender was not consistent with extant PreHTN disparities literature, in terms of greater prevalence among males compared to females. Gupta et al. (2010) reported prevalence in males at $44.8 \%$ compared to females at $27.3 \%$. Trend data (1999-2012) reported females were less likely to be PreHTN/HTN compared to males (Booth et al., 2017). In 2013-2014, compared to young adult females (18-3 9 years of age), higher PreHTN prevalence was reported in males $33.6 \%$ vs $12.8 \%$ (Zhang \& Moran, 2017). Among Coal Miners, the prevalence of PreHTN was higher in males compared to females $48 \%$ vs $35 \%$ (Casey et al., 2017), given that $94 \%$ of the study population was male, a higher prevalence was expected.

Higher PreHTN prevalence among males compared to females was observed in a Framingham Heart Study follow-up, 30\% vs 20\% (Vasan et al., 2001). In the Bogalusa Heart Study, Toprak et al. (2009) PreHTN prevalence was higher for males compared to females $35 \%$ vs $22 \%$.

Male gender was a predictor of EBP among a population of student athletics (Stiefel et al., 2016). On the other hand, Glasser et al. (2011) reported the proportion of PreHTN were similar among males and females.

The overall prevalence of PreHTN for my research was $5.8 \%$. A within group subanalysis by Race and poverty group was conducted PreHTN prevalence in the HPG was highest among NHW (29.4\%) compared to NHB (5.4\%) and Other Race (3.9\%), $x^{2}$ (4) $=218.169, p=.000$ (not shown). This means, within Race showed a significantly significant difference in prevalence in the HPG and PreHTN. In a trend study, NHB were more likely to be PreHTN compared to NHW, the differences were not significant (Booth, 2017). Gupta et al. (2010) reported slight difference in PreHTN prevalence in NHB 38.9\% compared to NHW 36.9\%.

On the other hand, other studies have reported differences. For example, the REGARDS study reported PreHTN prevalence $62.9 \%$ among NHB compared to $54 \%$ NHW (Glasser et al., 2011). Toprak et al. (2009), reported PreHTN prevalence at 29\% NHB compared to $27 \%$ NHW. Except for Glasser et al. (2011) none of the cited studies used income as a control or independent variable.

The hypothesis maintaining there is a difference in PreHTN prevalence between Non-Hispanic compared to Hispanic was non-significant ( $p=\mathrm{ns}$ ). Support for this position was based on the body of PreHTN prevalence literature (Bersamin et al., 2009; Booth et al., 2017; Gupta et al., 2010). A sub analysis was done by Hispanic Ethnicity and poverty group; PreHTN prevalence was highest in the MPG (36\%) compared to LPG (33.3\%) and HPG (30.7\%) $p=\mathrm{ns}$ (not shown).

What was not expected in this study was the higher PreHTN prevalence in the MPG compared to HPG. The differences by poverty group prevalence might be attributed to the HPG cutoff $<\$ 19,999$. It is also important to keep in mind the estimated median household income for Hispanics in 2020 was $\$ 55,321$, less than Asian \$94,903, NHW \$74,912 but higher than NHB \$45,870 (USCB, 2021a).

Bersamin et al. (2009) used 1999-2004 NHANES data and reported 33\% PreHTN prevalence among Mexican Americans. In the HTN Awareness, Treatment and Control assessment by race/ethnicity (NHANES 2013-2016); Hispanics were more likely to be aware of HTN $54.5 \%$ compared to NH Asian (NHA) at 52.9\%, least likely than NHB at $68.8 \%$, and NHW at $66.5 \%$; least likely to be treated at $42.1 \%$ compared to NHA at $43.7 \%$, NHB at $57.1 \%$, NHW at $55.4 \%$ and least likely to have control at $19.3 \%$ compared to NHB at $22 \%$, NHW at $26.8 \%$, but higher than NHA at $14.9 \%$ (Virani et al., 2020).

To summarize the interpretation of findings in the present study, an association between LMHPG and PreHTN was reported; association controlling for age was supported but not for gender, race, and ethnicity. PreHTN prevalence by LMHPG was
estimated by the same covariates. These findings build upon the body of PreHTN literature, specifically by adding new information that being in the HPG is predictive of self-report of PreHTN compared to MPG and LPG, with the exception for Hispanics. The non-significant findings by gender, aggregated race and ethnicity require replication.

The subanalysis stratified by racial group lend support for public policy on closing the lineage/racial wealth gap by addressing the hidden rules of race in tax laws that boost resources among the wealthiest population who are overwhelmingly NHW (Collins et al., 2020; Hamilton \& Linden, 2018). Income can boost wealth, yet we must be cognizant that not every dollar earned is equal between demographic groups (BLS, 2020a, 2020b).

## Limitations of Study

The first limitation was the paucity of public health literature examining the association of poverty group category and PreHTN when controlled for age, gender, race, and ethnicity. The analysis for this study was guided by the critical race theory (CRT) framework, using race and ethnicity as separate control variables to estimate the strength between the IDV and the DV. Stratifying Race is important because outlier categories can mask significant findings when subanalysis are not explored. I would have expected a significant difference between racial categories relative to PreHTN based on stressors related to Racism (Bird et al., 2021; M. T Williams et al., 2018).

The study findings could be attributed to the use of self-report compared to validated examination data. Ethnicity in the 2007-2008 NHANES dataset was limited to

Hispanic, within the category, Mexican American and Other Hispanic were the options. According to the Census Brief of the Hispanic Population (USCB, 2011), Hispanics can be of any Race. In 2010, (94\%) or 47.4 million of the Hispanics reported one racial group of this number, $>53 \%$ or 26.7 million self-identified as White (USCB, 2011).

Despite challenges, noteworthy results were identified and could be attributed to the quantitative approach, specifically controlling for age, gender, race, and ethnicity.

Second, cross sectional study designs cannot determine causality because assessment is accomplished at one point in time. Third, one (1) cycle of valid and reliable NHANES 2007-2008 data was used and the study relied on self-report of PreHTN rather than validating with BP data from the examination file. Participant recall bias might have also played a role in PreHTN self-report, or it could be possible that populations were PreHTN and or HTN at the time of the in-home interview and were not aware.

Third, there were survey respondents with missing values for the independent variable ( $n=577$ ). The covariant Race_Recode had a substantial amount of missing data $(\mathrm{n}=1,403)$ that were excluded from analysis. A conclusion could be made that the race groups were undercounted, and a greater number of participants could have been PreHTN. This opened the door for bias or imprecision. This was observed in a longitunal study where children who received public economic support, had a decrease odds of HTN as adults, this was only recognized among females because many male participants were lost to follow-up (Barrington \& James, 2017). Analysis of existing data can introduce methodological issues such as sampling, data collection, measurement, missing data, and non-response (Boo \& Froelicher, 2013; Yang et al., 2017).

## Strengths

There were several notable strengths, this was the first study to my knowledge examining the association between LMHPG and PreHTN after controlling for age, gender, race, and ethnicity. Second, the study used modeling for prediction, estimated prevalence and identified trend data from a large nationally representative sample. The 2007-2008 NHANES dataset consisted of 10,149, of these, 5,496 were $20-79$ years of age. Of these, 4,939 had data for the both the IDV and DV required to answer the research questions.

Third, the review of literature drew upon $>5$ prospective studies, The Baltimore Longitudinal Study of Aging (Najjar et al., 2008); The Bogalusa Heart Study (Pollock et al., 2018); Cardiovascular Risk in Young Finns (Kähönen et al., 2021); Coronary Artery Risk Development in Young Adults (CARDIA; Chae et al., 2020; Colangelo et al., 2020; Krieger \& Sidney, 1996; Kue et al., 2018; Pletcher et al., 2008; Yano et al., 2017); Framingham Heart Study (Andersson et al., 2016; Levy et al., 1990; Lloyd-Jones et al., 1999; 2006; Orkaby et al., 2019;Vasan et al., 2001, 2002); Jackson Heart Study (Clark et al., 2017; Forde et al., 2016; Redmond et al., 2016); Jichi Medical School Cohort Study (JMC; Ishikawa et al., 2017); Multi-Ethnic Study of Atherosclerosis (MESA; Allison et al., 2008; Carson et al., 2011; Mayne et al., 2019): Reasons for Geographic and Racial Differences in Stoke Study (REGARDS; Glasser et al., 2011; Limbi et al., 2016); San Antonio Heart Study (Mulligan et al., 2008); U.S. Longitunal Study of Adolescent to Adult Health (Add Health; Brody et al., 2016); and The Western Alaska Tribal Collaborative for Health Study (WATCH; Jolly et al., 2015).

## Recommendations

First, replication of this study using examination data to validate self-report of PreHTN may strengthen outcomes. The use of income demonstrates resources to meet immediate needs, specifically what a household must use for expenses and vary based on market conditions (e.g., recession), acute diseases (e.g., Covid-19), intentional violence (e.g., terrorism), and natural disasters (e.g., Tornado's; Muhammad-Asante et al., 2017; Taylor \& Meschede, 2018). Income can build wealth when there are fewer barriers encountered. The use of wealth as an independent variable would have been a better measure because it is the value of assets (e.g., stocks, house, cash, etc.) owned minus the debt (e.g., student loans, credit cards, etc.) owed (USCB, 2020d), its generational, shows distribution of economic and political power and is quantifiable.

Wealth provides a buffer during emergencies; pays college tuition; facilitates access to higher amenity neighborhoods; exert political influence via campaign contributions; pay for quality legal help if confronted by the judicial system; provides vital capital to start a new business, and is intergenerational (Benton \& Keister, 2017; Bhutta et al., 2020; Collins \& Wannamaker, 2017; Darity \& Mullen, 2020). Being in the HPG may indicate lack of wealth and chronic social disadvantage.

PreHTN is a risk factor for HTN, a cardiovascular risk. Higher income (USCB, 2020c) and wealth (FR, 2021) tend to be salient among dominant groups. This research found an association between HPG and PreHTN. Social gradient defined as members of higher SES are more likely to have better health outcomes compared to members of lower SES (Nguyen et al., 2014) and is relevant to this research.

To provide context, total unadjusted wealth in the U.S. during the $1^{\text {st }}$ quarter of 2021 was 129.6 trillion (t) of this number NHW held 109.4 ( t , NHB 5.1 ( t ), 2.97\% Hispanic and 12.08\% Other (Asian, PI, Native Americans, etc.; FR, 2021.) Future research should use the wealth variable in the context of PreHTN controlling for age, gender, residential segregation, race (NHB, NHW), and ethnicity (Asian Indian, Black immigrant, Hispanic Black compared to Hispanic Other/White).

In the context of residential segregation controlled for income and wealth, Mayne et al. (2019) found an association between racial group membership and cardiovascular health. Within the study, adjusting for neighborhood and individual SES; living in a more segregated neighborhood predicted cardiometabolic risk (e.g., HTN) for NHB (high vs low segregation: $\mu$ difference 0.17 SD units, $95 \%$ CI 0.02 to 0.32 ); (medium vs low segregation: $\mu$ difference $0.18 S D$ units, $95 \%$ CI 0.03 to 0.33 ). Non-Hispanic Whites living in more segregated neighborhoods did not show adverse cardiometabolic risk (high vs low segregation $\mu$ difference 0.00 units, $95 \%$ CI -0.02 to 0.01 ) (medium vs low; $\mu$ difference -0.01 units, $95 \% \mathrm{CI}(-0.01$ to 0.00$)$; and cardiometabolic risk was also minimal among Hispanics (high vs low: $\mu$ difference 0.11 units, $95 \%$ CI ( -0.03 to 0.25 ; medium vs low: $\mu$ difference 0.03 units, $95 \% \mathrm{CI}(-0.13$ to 0.20$)$. A compelling argument could be made that cardiometabolic risk is observed in bottom caste populations which tend to be higher poverty groups (USCB, 2021a).

In an HTN prevalence study among a subgroup of Hispanics, Dominicans had the highest prevalence $52.9 \% p<0.05$ compared to Mexican 32.6\%, Other Hispanic 42.1\% and Puerto Rican 43.3\% (Allison et al., 2008).

Elfassy et al. (2020) used the 2017 ACC/AHAA guidelines (Whelton et al., 2017) and reported incident HTN was highest among Cuban males compared to other Hispanic subgroups.

The risk of HTN among NHB and too an extent Hispanics might be explained through the concept called "Weathering" which occurs from living in a race-conscious society, exposure to repeated experiences of social exclusion, economic adversity, and political marginalization resulting in cumulative exposure to stress and deterioration of health (Geronimus et al., 2006; Simons et al., 2020). It appears that income and wealth provide little buffering against adverse health in NHB (Assari, 2018; Boen, 2016; Wilson et. al., 2017). This is relevant because Research Question 2 controlled for race and ethnicity.

With the use of wealth as an IDV, I propose an intervention at the public policy level to minimize the economic and social burdens resulting from accrued disadvantages for marginalized groups and limited tax cuts for wealthier populations. According to $20211^{\text {st }}$ quarter wealth data, the top 1 percentile has $41.2(\mathrm{t})$, top $90-99^{\text {th }}$ percentile 48.8 $(\mathrm{t}), 50-90^{\text {th }}$ percentile $36.52(\mathrm{t})$ and bottom 2.92 ( t ; FR, 2021). By racial group classification, Whites hold $>120(\mathrm{t})$ in wealth. Wealth is calcified and without public policy, groups on the margin face economic genocide and less optimal BP.

Wealth in the U.S is rooted in Chattel Slavery and the massive profits from the sale of humans, labor exploitation and commodities (e.g., cotton) produced by the enslaved population that created the American economy and solidified generational wealth (Hammond, J, 1866; USCB, 1791, 1832, 1854a, 1854b).

This information is relevant because a subanalysis by race found a statistically significant association between in HPG NHB and PreHTN compared to their race matched peers.

After the U.S government reneged on the promise of 40 acres of land to newly emancipated Blacks as demonstrated by General William T. Sherman's, Special Field Order \#15 (Grimsley, 2014; National Archives, 2016a), the failure of Senate Bill 1176 to Provide Pensions for Freedmen and so Forth Financial Support (National Archives, 2017); not withstanding grievous barriers, Southern Blacks were able to purchase 15 million acres of land, unfortunately through land grabs, and White terrorism, an estimated 1 million acres are currently owned by Southern descendants of persons enslaved in the U.S. defined here as ADOS (Darity, 2008; Hinson, 2018). This historical information is relevant because a subanalysis by Race found that NHB in the HPG had almost twice the risk of PreHTN compared to their race matched peers.

After Emancipation Proclamation in 1863 (National Archives, 2019), ratification of the $13^{\text {th }}$ Amendment (National Archives, 2016); and Plessy v. Ferguson (1896) (Library of Congress.Gov [LCG], n.d) that legalized racial discrimination also known as Jim Crow; ADOS, encountered immense obstacles to wealth building and maintaining. The Homestead Act of 1862, PL 37-64, extended wealth opportunities to landless Whites, newly arrived European immigrants and other non-Europeans and excluded most Blacks because the majority were enslaved (United States Senate [USS], n.d).

The New Deal, a series of programs enacted by President Franklin D. Roosevelt in 1933 was a tool that mutually reinforced polices providing economic repair for White citizens at the expense of ADOS, thus solidifying the lineage/racial wealth gap (Davies \&

Derthick, 1997; FR, 2021; Faber, 2020). This information is relevant because a sub analysis found that NHB in the highest poverty group increased the odds of PreHTN selfreport. PreHTN is a risk factor for HTN, and lifetime discrimination has been linked to incident HTN (Forde et al., 2020). Compared to the lowest poverty group, my research found an association between the highest poverty group and PreHTN.

Not having adequate resources to meet needs could potentially create stress and stress has been associated with EBP (Gawlik et al., 2019; Haggart et., 2018). An independent analysis of 2016 Federal Reserve data found that wealth is not homogenous, after controlling for depreciable assets (e.g., car, jewelry, furniture, etc.), investigators found that Black families $(n=380,000)$ at the $99^{\text {th }}$ percentile were worth $\$ 1,574,000$ compared to $>13$ million White families worth $>\$ 12$ million (Moore \& Bruenig, 2017). This is relevant to the research questions because higher poverty group membership was associated with PreHTN, and a stratified subanalysis found that NHB in the HPG had almost twice the odds of PreHTN compared NHW and OR peers.

While most studies report wealth data for Blacks, Hispanic/Latino, and Whites; Asians are left out. Asians are not homogenous and wealth differences between groups are striking. This is important because the research questions controlled for ethnicity and the 2007-2008 NHANES dataset did not extend ethnicity data beyond Hispanic.

Economic resources by Asian ethnicity is different, for example, In The Color of Wealth in Los Angeles, a report by the Federal Reserve Bank of Los Angeles and partners, reported median net worth for Japanese at $\$ 592,000$, Asian Indians at $\$ 460,000$, Chinese at $\$ 408,200$, Filipino at $\$ 243,000$, Vietnamese at $\$ 61,500$, compared to Korean at
$\$ 23,400$ (De La Cruz-Viesca et al., 2016). Within the report, median net worth for Whites was $\$ 355,000$ and $\$ 72,000$ African Blacks compared to U.S. Blacks (ADOS) \$4,000.

Hispanic ethnic groups differ as well; the Federal Reserve Bank of Boston and partners reported that median net worth for U.S Blacks was $\$ 8.00$ compared to Caribbean Blacks $\$ 12,000$ (Muñoz et al., 2015). Within the report, Whites median worth was $\$ 247,000$. The median net worth for Puerto Ricans was $\$ 3,020$ and Other Hispanics $\$ 2,700$ compared to Dominicans $\$ 0$. Among Hispanics in the Color of Wealth in Miami, median net worth is highest among Cubans $\$ 22,000$, Other Hispanics $\$ 10,500$, and Puerto Ricans $\$ 3,940$ versus South Americans $\$ 1,200$ (Aja et al., 2019). Within the report the median wealth among Whites was $\$ 107,000$. Caribbean Blacks had greater net worth $\$ 12,000$ compared to U.S. Blacks $\$ 3,700$. These data suggest that racial and ethnic groups are not homogenous, and differences must be considered in PreHTN and health promotion research.

Research assessing the association between LMHPG and PreHTN must integrate lineage/racial wealth gap data. By doing so, findings can be used as an additional approach to challenge the federal tax code, specifically the 2017 Tax Cuts and Jobs Act (TCJA), PL 115-97, (Congress.Gov [CG], 2018). This is based on empirically derived evidence that racial group and economic resources are associated with adverse health outcomes (Assari et al., 2017a; Assari et al., 2017b; Assari, 2018; Boen, 2016; Wilson et al., 2017). The 2017 Tax Cuts and Jobs Act (CG, 2018; Gale et al., 2018) made it easier for wealthier individuals to receive larger reductions.

For example, the top quantile is estimated to receive $2.9 \%$ tax reduction compared to the lowest quantile 0.4 (Gale et al. 2018, p. 598). The impact of this policy decision is relevant to the research questions because higher poverty group is associated with increased odds of PreHTN, a preventable HTN risk factor.

The U.S. government's ability to pay for social change interventions has been demonstrated. During the Recession of $2008>\$ 2$ trillion was generated to bail out banks and aid the ailing economy (Hamilton \& Darity, 2010) and because of the Coronavirus Preparedness and Response Appropriations Act, PL 116-123, trillions of dollars have been infused into the economy to address Covid-19 (Congress.Gov [CG], 2020). Taxes are revenue sources and a redistribution of wealth used by the U.S government to support all human activities (Congressional Budget Office [CBO], 2020). Eliminating generous tax breaks for the wealthy is socially just and has the potential to generate critically needed revenue.

The revenue from equitable taxation can be invested in restitution for grievous injustice to ADOS, childhood savings accounts (e.g., Baby bonds), updating infrastructures (e.g., sidewalks, bridges, bike trails, etc.), ensure quality affordable housing, livable wages, and a lifetime of preventive health services. This approach is relevant to my research questions because higher poverty group is associated with PreHTN.

First, the research found an association between the HPG and PreHTN. A sub analysis between Race found an increased risk of PreHTN among HPG NHB. Being a member of a specific racial group has been linked to stress and stress is associated to

HTN (Gawlik et al., 2019; Michaels et al., 2019; Whitaker et al., 2018; M.T. Williams et al., 2018). Revenue must be directed to a comprehensive program to pay Reparations to ADOS because this group inherited the accrued injustices from Chattel Slavery, legal segregation, and chronic racial discrimination and stigmatization (Collins \& Wanamaker, 2017; Darity, 2008; Darity \& Mullen, 2020).

The prevalence of PreHTN (Booth et al., 2017; Glasser et al., 2011) and HTN (Ostchega et al., 2020) is high in the NHB community. Current research using epidemic modeling reported that a restitution program paid to Black American Descendants of Persons Enslaved in the U.S. could have decreased the impact of Covid- 19 in Louisiana, thus situating Reparations as a viable public health intervention (Richardson et al., 2021). Empirical data report inheritances, family gifts (e.g., college tuition, capital to start a business, etc.) and intrafamily transfers (e.g., land deeds) account for more of the lineage/racial wealth gap than any other demographic and socioeconomic indicators including education, income, and household structure (Benton \& Keister, 2017; Gittleman \& Wolff, 2004; Niimi \& Horioka, 2018).

Reparations paid by the U.S. government for grievous harms are not new. The Civil Liberties Act of 1987, P.L. 100-383 (Congress.gov [CG], 1988) paid restitution to Japanese civilians interned during World War II. Native Americans have received good faith payment for injustices (United States Department of Interior [USDOI], n.d; United States Department of Justice [USDOJ], 2016) and although the Jewish Holocaust happened in Europe, the U.S has paid restitution to survivors and heirs (United States Department of States [USDOS], 2019).

The Obama Administration specifically allocated $\$ 12$ million from Health and Human Services for 130,000 Jewish Holocaust survivors in the U.S. (Wassermanshultz.gov [WSHG], 2015). Under the National Defense Authorization Act for Fiscal Year 2017, PL-114-328 (CG, 2016; Hofschneider, 2020), the same administration earmarked reparation to Guam for grievous harms committed during World War II.

For the general population, establishing child savings accounts, also known as Baby Bonds, could narrow the lineage/wealth gap of young Black adults from \$2,900 to $\$ 57,850$ and young Whites from $\$ 46,000$ to $\$ 79,143$ (Zewde 2020). Additional investments must be targeted toward updating aging infrastructure to walk, ride bikes, road repair, expand broadband internet access, and upgrade sanitation systems in rural communities. The latter is of great concern because third world conditions should not be experienced by U.S citizens, as demonstrated by the Hookworm problem in Lowndes County, Alabama (McKenna et al., 2017). Within the study, $>1 / 3$ of households tested positive for Hookworms.

Second, within Hispanic ethnicity, using only Mexican and Other Hispanic minimizes subgroup heterogeneity. This is important because the research questions controlled for ethnicity. Although Mexicans represent $63 \%$ of Hispanics in the U.S. and Other Hispanics (Central and South Americans) 21.5\%; using two categories to measure Hispanic health fosters a catchall category. This catchall category misses Puerto Ricans (9.6\%), Cubans (3.5\%), and Dominicans (2.8\%; USCB, 2011).

For example, in assessing risk factors for subclinical cardiovascular disease in a subgroup of Hispanics, Dominicans DBP was higher compared to Puerto Ricans, Other Hispanics, and Mexicans $p<0.05$ (Allison et al., 2008). Daviglus et al., (2012) reported among Hispanic males, Dominicans presented with greater proportion of HTN 32.6\% compared to Cuban 28.9\%, Puerto Rican 27.4\%, Central American 25\%, Mexican 21.4\%, and South American 19.9\%. Dominicans have been reported with zero wealth compared to Puerto Ricans \$3,020 and Other Hispanics \$2,700 (Muñoz et., 2015).

Compared to Central Americans ( $\mu 1.45$ ), Cubans ( $\mu$ 1.39), Dominicans ( $\mu 1.86$ ), and Mexicans ( $\mu 1.38$ ), Puerto Ricans had higher $\mu$ stress 2.19 ( $p<.0001$ )(Rodriquez et al., 2021); and depression, a stress condition, is associated with PreHTN (Ford et al., 2016: Zambrana et al., 2016). Recent HTN Awareness, Treatment and Control using 2017 ACC/AHA guidelines reported greater incident HTN among Cubans compared to other Hispanic subgroups (Elfassy et al., 2020). Hispanics overwhelming self-identify as White ( $>50 \%$ ); Dominicans and Puerto Ricans are more likely to classify as Black (USCB, 2011).

Black Hispanics with low perceived Racism was associated with non-BP dipping compared to White Hispanics (Rodriguez et. al., 2016). White identity among Hispanics may confer different levels of BP and other health benefits (López et al., 2018). Black Hispanics might be a group at elevated risk and understudied in PreHTN and HTN disparities research.

Third, poverty varies within Asian subgroups, it would be expected that if there were an association between poverty and PreHTN, addressing this group would be necessary because EBP might be underexplored. For example, compared to low acculturated Asians, high acculturation was associated with $34 \%$ increased risk of HTN (Divney et al., 2019). Within this study, $>45 \%$ of Asian reported annual household income $>\$ 70,000$. Acculturation is a stressor (Wong et al., 2017) and stress has been linked to HTN.

There is scant data on the prevalence of PreHTN among Asian subgroups in the U.S. (Jung et al., 2019; Vazquez-Vidal et al., 2016) to include pediatric populations (Lo et al., 2013). In the MESA study, at follow-up, Chinese participants had the lowest rate of incident HTN IRR 52.2 per 1,000 persons years (py) compared to NHB 85.9, Hispanic 65.7 and NHW 56.8 (Carson et al., 2011). Hypertension prevalence among Asians are mixed. For example, The National Center for Health Statistics [NCHS] (2019), reported that in 2018, age adjusted HTN prevalence among Asian Adults $\geq 18$ at $21.9 \%$ (NCHS, 2019). On the other hand, recent data reported HTN prevalence among Non-Hispanic Asian men at $46.4 \%$ and $36.4 \%$ among females (Virani et al., 2020).

Race and ethnicity are control variables for the research questions because both has the potential to increase association between poverty group and PreHTN. Asians represent $5.6 \%$ (17.3 million) of the U.S. population, and between 2000-2010 (10.2 million to 14.7 million) their presence has grown by $43 \%$ (USCB, 2012). Eighty five percent ( $85 \%$ or 14.7 million) of Asians report 1 race, among those reporting Asian and another race ( 2.6 million) $60 \%$ self-identify as White.

The five (5) largest Asian populations in the U.S. are Chinese (4,010,114), Asian Indian $(3,183,063)$, Vietnamese $(1,737,433)$, Korean $(1,706,822)$, and Japanese $(1,304,286)$ (USCB, 2012). As demonstrated by the Los Angeles Color of Wealth Report, within group wealth differences between this group was observed (De La Cruz-Viesca et al., 2016).

Asians were not included in the 2007-2008 NHANES dataset, which clearly demonstrates this group is under analyzed and within group variations in EBP exist. For example, Jung et al. (2019) reported among foreign born Asians ( $n=600$; 201-Chinese, 198-Korean, and 198 Vietnamese); $34 \%(n=204)$ were PreHTN and $29.2 \%$ HTN ( $n=$ 175). Although between group differences were not statistically significant, HTN among Vietnamese was $31.9 \%$ compared to $21.8 \%$ Chinese, $29.9 \%$ Korean and PreHTN was not broken down by ethnic group. Vazquez-Vidal et al. (2016) reported 46\% PreHTN prevalence among Parsi Zoroastrians and $16 \%$ HTN. Based on these studies a case can be made that homogenizing Asians may introduce unintentional bias by enabling masking of problems within smaller subgroups which may deprive opportunities to examine within group PreHTN disparities for timely intervention.

Poverty varies within Black subgroups; it would be expected that when an association between poverty and PreHTN is reported, this area would require close examination. Black race is not a monolith, this is undergirded in the fact that of the 41 million Blacks $\geq 37$ million are ADOS and an estimated 4.3 million are Black immigrants (USCB, 2020c). All Black immigrants are self-selected, meaning they have voluntarily immigrated and do not share the unique ethnic identification, experiences, and accrued
disadvantages of ADOS (USCB, 1791, 1832, 1854a, 1854b, 1961). Prior to the 1952 Immigration Act, PL 82-414 (Homeland Security Digital Library [HSDL], n.d); quotas were used for specific Europeans (e.g., Eastern and Southern) and non-European immigrants (e.g., Asian Indians, etc.). Before the 1964 Civil Rights Acts (CRA) PL 88352, very few Black Immigrants lived in the United States (Gibson \& Jung, 2006; GovInfo.gov [GIG], n.d.a; Reid, 1938; USCB, 1961). Caribbean's from English speaking countries were the main Black immigrants, and the portion of Black immigrants was to the right of the decimal point.

The CRA of 1964 opened the door for the 1965 Hart Cellar Act, PL 89-236 (GIG, n.d.b) that effectively eliminated the quota system for specific immigrants (Eastern and Southern Europeans); thus, paving the way for favorable migration policies; only later did this legislation and other policies benefit immigrant Blacks (Anderson, 2015; Howard \& Borgella, 2020; McCleary-Gaddy \& Miller, 2018). African immigration increased $137 \%$ between 2000-2013 (Anderson, 2015) and $>47 \%$ of Nigerians immigrated post 2010 (USCB, 2020e). Black immigrants are not representative of their origin country peers in terms of average years of education.

For example, years of education in Nigeria is 9 (CIA, n.d.d) in contrast $20.9 \%$ of Nigerians in the U.S. have some college or an associate degree (USCB, 2020e). Trinidad/Tobago has one of the highest literate populations in the world at $99 \%$ (CIA, n.d.e) and $31.2 \%$ of Trinidad/Tobago population in the U.S have some college or an associate degree (USCB, 2020g); to put into context, ADOS mean years of education is 12 years (USCB, 2020c).

Less than $15 \%$ of ADOS have undergraduate degrees (13.9\%) compared to Black immigrants from Ghana ( $25 \%$; USCB, 2020f), Nigerian (36.6\%) and Trinidadian and Tobagonian (18\%). Median household income in 2019 for ADOS was \$43,862 (USCB, 2020c) compared to $\$ 69,021$ Ghanaian (USCB,2020g), $\$ 57,451$ Haitian (USCB, 2020h), $\$ 62,044$ Jamaican (USCB, 2020i), and $\$ 65,672$ Nigerians (USCB, 2020e). An argument can be made that these groups have benefited from the CRA 1964.

Racism is a stressor and has been associated with EBP in NHB compared to NHW (Gawlik et al., 2019; M. T. Williams et al., 2018). Evidence found that being a foreign-born Black provides a $39 \%$ protection against HTN compared to U.S. Born Blacks (Brown et al., 2017). The recommendations presented have the potential to extend quality of life, save money, and contribute to a collective strategy that may contribute to optimal BP in the United States.

## Implications for Positive Social Change

The present study revealed an association between LMHPG and PreHTN among a representative sample of U.S. noninstitutionalized individuals 20-79 years of age. Social Change from this research comes from assessing if poverty group level when controlled for age, gender, race, and ethnicity is a good predictor of PreHTN, a significant HTN and CVD risk. Given the association between PreHTN, incident HTN, BMI, Stroke, and EBP's adverse impact on quality of life and life span; this confirmation adds evidence of the need to go beyond traditional public health interventions to address the root cause of poverty. The implications of my findings are that higher poverty group level controlled for age, gender, race, and ethnicity is a good predictor of PreHTN, a preventable CV risk.

Social change is synonymous to wound debridement in terms of going to the root cause, cleaning it out, medicate to allow substantial healing, and continuous evaluation to ensure infection will never return; if it does, its impact will be neglectable due to the problem being made a significant public health priority.

## Conclusion

The lack of research data on the association between income and PreHTN warrants further investigation. A detailed analysis of race and ethnic heterogeneity must be incorporated in PreHTN public health research to obtain a clearer picture on within group differences. The CRT framework was useful for the covariant Race in the sub analysis only but not ethnicity. This study confirmed an association between LMHPG and PreHTN when controlling for age, gender, race, and ethnicity $(\mathrm{HcNH})$ and estimated prevalence of PreHTN. Study replication is required to build upon the strengths and limitation, but also to generate data to inform novel PreHTN and HTN interventions that specifically addresses the root causes of poverty. Additionally, research using the association between wealth and PreHTN is warranted to inform public policy. My aim as a Public Health Scholar Practitioner is to use these finding as part of a collective, taking radical and unpopular actions advocating for restorative justice with a goal of optimal BP and the associated enhanced quality of life attributes such as low cardiovascular risk, perceived safety, livable wage, quality affordable housing and generational wealth.

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## Appendix A: G* Power Sample Size Analysis

| B1 G*Power 3.1.9.2 |  |  | $\square \times$ |
| :---: | :---: | :---: | :---: |
| File Edit View Tests Calculator | r Help |  |  |
| Central and noncentral distributions Protocol of power analyses |  |  |  |
|  |  | $=1.64485$  |  |
| Test family Statistical test |  |  |  |
| $z$ tests Logistic regression |  |  |  |
| Type of power analysis |  |  |  |
| A priori: Compute required sample size - given $\alpha$, power, and effect size |  |  |  |
| Input Parameters Output Parameters |  |  |  |
| Tail(s) | Two $\checkmark$ | Critical z | 1.6448536 |
| Determine $=>\quad$ Odds ratio | 3.4390244 | Total sample size | 378 |
| $\operatorname{Pr}(\mathrm{Y}=1 \mid \mathrm{X}=1) \mathrm{HO}$ | 0.06 | Actual power | 0.9002338 |
| $\alpha$ err prob | 0.10 |  |  |
| Power (1- $\beta$ err prob) | 0.90 |  |  |
| $\mathrm{R}^{\mathbf{2}}$ other X | . 25 |  |  |
| X distribution <br> X parm $\pi$ | Binomial $\checkmark$ |  |  |
|  | 0.3 |  |  |
| Options |  | $X-Y$ plot for a range of values | Calculate |

## Appendix B: G* Post Hoc Analysis



Appendix C: Racial Caste, Ethnicity, and Income as an Indicator of Social Standing
Race determines a group's social trajectory. For example, datasets comparing the income of NHB and NHW father son dyads from 1880 to 2000 found that compared to NHW, NHB large intergenerational mobility gaps was attributed to lack of human capital perpetuated by structural racism (e.g., Mutually reinforcing systems) (Collins \& Wannamaker, 2017). The problem with this study was that it did not cite 20th century policies such as New Deal which excluded NHB from skilled blue-collar jobs, homeownership, collective bargaining, social security benefits, and other anti-poverty resources (Hirsch, 2000; Katznelson, 2021). The analysis did not cite the creation of harsh criminal policies (e.g., Convict leasing) (Haley, 2013); ethnic cleansing through lynching (Stuart, 2020) and banishment (Lancaster, 2010). Additionally, mass incarceration in the $21^{\text {st }}$ century has negatively impacted NHB and Hispanic communities (Bureau of Justice Statistics [BJS], 2019; Graff, 2015).

Ethnicity in the United States have been described as Hispanic or Non-Hispanic (USCB, 2020a). Others have described ethnicity as shared histories, beliefs, practices, lineage, and rituals conferring a sense of pride and identity (Helms \& Talleyrand, 1997; Markus, 2008). In the United States, race is measured by phenotypic characteristics and is visible (e.g., skin color, hair texture, etc.) to include caste membership (Harawa \& Ford, 2009) and ethnicity is based on self-identification, shared histories, language, and lineage (e.g., Dominican, Jewish, Mexican, Yoruba etc.) (Helms \& Talleyrand, 1997; Markus, 2008).

The problem with ethnicity is that ethnic group affiliation does not necessarily expose groups to unfair treatment because of skin color; in fact, USCB extends honorary white identity to specific African and Asian subgroups and to any Hispanic group (USCB, 2020a).

Income is an indicator of social standing and human capital (Galobardes et al., 2006; Noël, 2018) has limitations for American Descendants of Slavery (ADOS) also known as NHB. To provide context, even with a college degree, and working hard, access to social capital that provide economic and social security is limited for ADOS (Hamilton et al., 2015; Muhammad-Asante et al., 2017 [see Appendix D]; Noël, 2018). The social construction of race is based on the subjective evaluation in the context of skin color, hair, and nose, by individuals, groups and structures that hold power (Lopez, 1994). This power is leveraged through structural racism defined as "the totality of ways in which societies foster racial discrimination through mutually reinforcing systems of housing, education, employment, law, earnings, benefits, credit, media, and health care (Bailey et al., 2017b). Bottom caste groups are clustered among lower income (USCB, 2021a) and high poverty group membership is associated with increased odds of PreHTN and CV risk.

Appendix D: Middle Class Earnings by Race/Ethnicity
Earning Middle-Class Income Does Not Guarantee Black Families Middle-Class Economic Security


Source: Middle Income Range. Pew Research Center "The Anerican Mddele Class is Losng Ground" Income Quinties and Associted Household Weoth. The New School Ouke Center for Sociol Equity


*Note: Zero Wealth, How the Racial Wealth Divide is Hollowing out America's
Middle Class, p. 7


[^0]:    * Cut points based on numeric values.

[^1]:    ${ }^{1}$ NHANES does not go beyond 80 years of age.

