

Increased Physical Activity Reduces the Odds of Elevated Systolic Blood Pressure Independent of Body Mass or Ethnicity in Rural Adolescents

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Abstract

Objectives: Reduced Physical Activity (PA) has been implicated in the increased prevalence of adolescent obesity and Systolic Blood Pressure (SBP). The present study provides a robust examination of these relationships in Hispanic adolescents from a rural population for which data are scarce.

Methods: We compared PA levels, SBP and body mass categories (normal weight, overweight and obese) between non-Hispanic white and Hispanic adolescents (15 ± 0.1 yrs; n=983 males, 911 females) using odds ratio and path analyses.

Results: When groups (by gender & ethnicity) were categorized by body mass independent of SBP, prevalence of elevated SBP for obese compared to normal weight cohorts was 3.5- and 12-fold greater for non-Hispanic white males and females, respectively, and 2- and 3-fold greater for Hispanic males and females, respectively. When categorized by SBP independent of Body Mass Index (BMI), prevalence of obesity for adolescents with elevated SBP compared to normotensive cohorts was 3.5- and 6-fold greater for non-Hispanic white males and females, respectively, and 2-fold greater for both Hispanic males and females.

Conclusions: Path analyses suggest that both reduced PA and increased BMI are simultaneous predictors of the observed elevation in SBP. Odds ratio analyses revealed that 6+hr PA/wk reduced the probability of developing elevated SBP 3-fold in both genders independent of body mass category or ethnicity, identifying increased PA as a critical behavioral element to target to alleviate the consequences of obesity-related increases in SBP in young people regardless of ethnicity or gender.

Keywords: Cardiovascular disease prevention; Exercise; Health disparities; Hypertension; Metabolic syndrome; Obesity

Introduction

Obesity is closely associated with increased systolic blood pressure (SBP) in children [1,2] and young adults [3]. In young adults, the prevalence of hypertension and cardiovascular diseases (CVD) are disproportionately greater in ethnic and racial minorities [3]. However, this relationship is not consistent among children and adolescents. While higher blood pressure has been reported in African-American children than whites [4], other studies did not find racial [5] or ethnic [6] differences.

Although Hispanics had a significantly higher unadjusted relative risk of elevated blood pressure after each screening, no ethnic differences were detected in the prevalence of hypertension after adjusting for overweight suggesting that the primary contributing factor to the development of hypertension in these adolescents was overweight and not ethnicity (Hispanic) [6]. However, few studies have examined the relationship between body mass index (BMI) and elevated blood pressure in adolescents, especially those from

underrepresented or disadvantaged populations. Because BMI and SBP are used in the adults to predict the onset of cardiovascular complications [7], a closer examination of this relationship in adolescents, especially from disadvantaged or underserved populations, is warranted.

In rural Canadian adolescents, obesity was associated with pre-hypertension and hypertension, while overweight was only associated with hypertension in boys, and not girls [8] suggesting that rural overweight and obese males may be at a greater risk than their female counterparts for hypertension-related consequences. These gender differences among Hispanic adolescents in the US appear to have changed over the past decade. The prevalence for elevated arterial pressure for Hispanics increased significantly between the 1988-1994 and 1999-2002 measurement periods, but these increases were not different between males and females [9]. However, we recently demonstrated that overweight and obese adolescent females, independent of ethnicity or race, had almost 2-fold greater odds of developing pre-elevated and elevated blood pressures, respectively, than their male counterparts [10] suggesting that the risk of developing hypertension-related consequences in females may be greater than in males. This data also suggests that over the past decade,

at least in Hispanic adolescents, the risk has increased substantially. This is particularly important because obesity in children and adolescents is closely associated with negative cardiovascular outcomes in adulthood and considered a significant contributing risk factor of developing later-onset CVD [11-15].

Furthermore, the data on the relationships between physical activity (PA) and body mass and systolic blood pressure (SBP) in adolescents are scarce and incongruent. While a relationship between PA and SBP was not observed in adolescents [16], some evidence suggests an inverse relationship between the two variables [17-19]. However, examinations accounting for body mass in the relationship between PA and SBP in adolescents from at-risk populations are scarce if not non-existent.

Given that the blood pressure trends within adolescents can change within a 10 year period, further and more frequent examinations of SBP and body mass relationships among ethnic and racial minority groups are necessary, especially if these groups have been previously identified to be more susceptible to the development of later-onset elevated SBP. Unfortunately, blood pressure data from rural, Hispanic adolescents stratified by body mass category (i.e., normal weight, overweight and obese) are lacking. Therefore, we conducted more robust and comprehensive analyses of our SBP and body mass data [10,20] to better assess the effects of gender, ethnicity, body mass category and PA levels on increased SBP. The current study applied path analysis, an alternative modeling technique, which incorporates a system of hypothesized relationships among predictors. To the best of our knowledge, path analysis has not been used to assess the predictive nature of variables that contribute to elevated SBP, and it has the potential to provide novel insight to the relationships among predictors of SBP. We hypothesized that increased physical activity reduces the risk of developing pre-elevated and elevated SBP in non-Hispanic whites to a greater extent than in their Hispanic cohorts.

Methods

This study was reviewed and approved by the Institutional Review Board of the University of California, Merced. Details of the study subjects and protocols have been published elsewhere [10,20], but are included here briefly for completeness.

Subjects

The study population consisted of 613 non-Hispanic white (312 males, 301 females) and 1281 Hispanic (671 males, 610 females) adolescents (13–17 years). Merced Union High School District (MUHSD) is comprised of five high schools with a total enrollment of approximately 10,000 students. Students in MUHSD are predominately Hispanic/Latino (54%), followed by non-Hispanic white (24%), Asian/Hmong (15%), African-American (5%) and other (2%). This study population is representative of Merced County, which has a population of predominately Hispanic/Latino residents (53%), followed by non-Hispanic White (35%), Asian/Hmong (7%), African American (4%) and other (1%). The Hispanic population is predominately Mexican-American (94%). Analyses focused only on comparisons between non-Hispanic whites and Hispanics because of the lack of sufficient recruitment and enrollment of adolescents from other groups, which prohibited our ability to make any meaningful comparisons. The data were collected during the MUHSD annual health assessments and sport physicals. Prior to enrollment in the study, students were read the assent form in the presence of their

parents, allowed to read it for themselves, asked if they understood the procedures, and asked to sign the form if they agreed to participate. Parents were then asked to sign their parental consent form.

Anthropometric definitions

Self-reported age, gender, and ethnicity of participants were attained from their annual health 120 assessment and sports physicals. Trained technicians and nurses measured each participant's height, weight, and SBP using standardized protocols [10, 20]. Overweight and obesity were defined according to BMI index cutoff points for age and gender published by the CDC [21]. Participants were classified as follows: BMI <5th percentile = "underweight," BMI \geq 5th percentile and <85th percentile = "normal weight," BMI \geq 85th and \leq 94.9th percentile = "overweight and BMI \geq 95th percentile = "obese" [22]. Only 2.8% of the adolescents measured were determined to be underweight and were removed from the analyses.

Systolic blood pressure

Blood pressure was classified using guidelines detailed in the Fourth Report on the Diagnosis, Evaluation, and Treatment of High Blood Pressure in Children and Adolescents [23]. Because the guidelines recommend multiple SBP measurements at different times to diagnosis pre-hypertension and hypertension, we used the terminology of Ostchega et al. [24], which is similar to that of Din-Dzietham et al. [9], to avoid any confusion with the diagnostic use of the terms that include "hypertension". Thus, a SBP measurement defined by SBP \geq 90th percentile (pre-hypertensive range) was termed "pre-elevated SBP" and measurements defined by SBP \geq 95th percentile (hypertensive range) was termed "elevated SBP" [23], and not intended to be used as a diagnostic term in the present study. SBP was measured using an automated oscillometric device (Omron HEM-780, Omron Healthcare, Bannockburn, IL) while subjects were seated. A mercury sphygmomanometer and extra-large cuff were used when the cuff of the automated device was not large enough to fit some of the subjects. Values were taken from a single reading, except on the rare occasions when an error was reported with the value and the measurement was re-taken. Falkner et al. demonstrated that a single blood pressure measurement in adolescents was sufficient to identify groups who are at a heightened risk for developing subsequent hypertension, illustrating the usefulness of a single measurement [25].

Estimated physical activity measures

As part of the self-report survey, participants were asked about a wide range of PA and screen behaviors, as previously described [20]. The survey was developed using the NHANES 2001-2002 survey as a template and modified for our purposes to facilitate time constraints as the survey was administered during the MUHSD physicals and annual health assessments. The survey had to be completed by each student within 20 minutes to accommodate the physicals and health assessments; therefore, we used an abbreviated survey that focused solely on physical activity and screen behavior times. Modifications included confining the time-frame of the participation to a typical week as opposed to the last 30 days and limited the answers to number of hours per week. Participants were able to indicate a range of hours per day and per week that they engaged in physical activity. An estimate of total physical activity (PA_T) was calculated and was used for statistical comparisons in the present study. Frequency distributions identified that a natural break point occurred at more or

less than 6 hr/wk of PA, which was used in the multinomial logistic regression analyses.

Statistical analysis

Means (\pm SD) were compared by analysis of variance (ANOVA) for ethnic groups, gender, and group \times gender differences. Regression slopes were compared by analysis of co-variance (ANCOVA). Prevalence rates according to gender and ethnicity were compared using chi-squared distribution statistics as previously described [10,26]. Prevalence values are presented as percentage. Age had no significant effect on any of the variables in the analyses, so data for each age were combined. Means and Regressions were considered significant at $p < 0.05$, and were performed using Statview software (SAS, Cary, NC). Categorical definitions of BMI were also used in our analyses because they offered an alternative approach to interpreting the data. In this study, we were primarily interested in the differences across discrete categories (i.e., “normal weight” vs. “overweight” vs. “obese”). Because of our use of categorical variables, logistic regressions were used to calculate adjusted odds ratios (OR). Non-Hispanic, white was the comparison group for analyses of ethnicity. Odds ratios were estimated using multinomial, logistic regression using Stata© (Stata, College Station, TX). After running a pooled logistic regression model of both male and female respondents, both gender and ethnicity effects were apparent. Models were then estimated using gender and ethnicity interaction terms. Gender was the most important characteristic in the multivariate models; therefore a strategy of running separate logistic regression with only males and only females was employed. Our logistic models used SBP as a dependent variable, and gender, ethnicity, body mass category and total PA as independent variables.

In general, statistical models that examine direct and indirect causes (or influences) among constructs can be used to delineate causal pathways among variables. These causal pathways can be simultaneously examined in the context of cross-sectional data by implementing a path analysis model. Specifically, dependencies among predictor variables are more closely examined with some predictors acting as mediator variables. A mediator variable is a third explanatory variable that can be used to capture the relationship between an independent and dependent variable. Within path analysis, it is hypothesized that the independent variable influences the mediator and then the mediator influences the dependent variable, or outcome. In general, mediator variables are used to further clarify or explain the relationship between the independent and dependent variables in a way that ordinary regression cannot. The path analysis model specifically uncovers the extent to which the mediator variable governs the relationship between the other two variables in the model. In order to better understand the phenomena influencing SBP, two different path analysis models were constructed to predict SBP as a means to better assess direct and indirect relationships among BMI, PA, and SBP. The first model looked at the direct impact that BMI and PA have on SBP. This first path model examined to what extent BMI and PA are predictors of SBP. The second path model assessed to what extent PA has as indirect effect on SBP through the mediating variable BMI. The notion of examining PA as an indirect predictor of SBP is a novel concept that has not been previously explored in the literature in relation to hypertension. The models were estimated for the full sample, a multi-group analysis for gender, a multi-group analysis for ethnic groups, and a multi-group analysis for gender \times ethnicity. Path coefficients were considered significant at $p < 0.05$, and the models were estimated using Mplus software program (version 7) [27].

Results

Body mass index and systolic blood pressure

With one exception, mean BMI and SBP values between ethnicity and genders were not different when the groups were categorized by their body mass classifications (i.e., normal weight, overweight and obese) (Table 1). The lone exception was a 6.2% and 4.9% greater mean BMI and SBP, respectively, in obese, non-Hispanic white females compared to their Hispanic cohorts (Table 1).

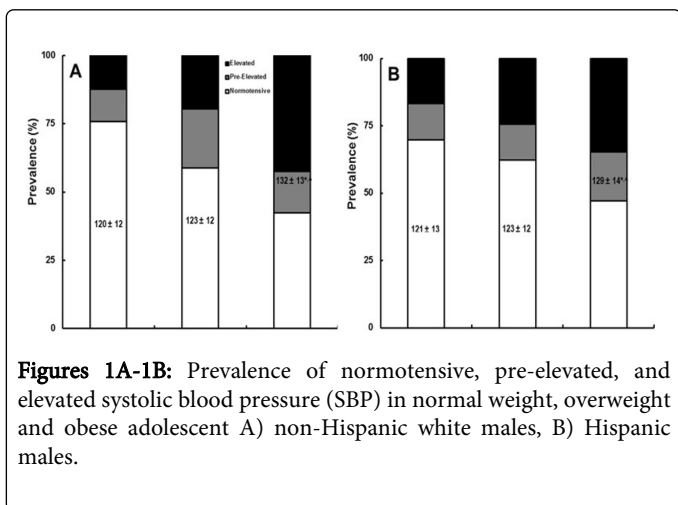
	n	Non-Hispanic White	n	Hispanic
Age	499	15 (0.7)	1003	15 (0.7)
Males				
BMI (kg/m²)				
Normal Weight	145	20.1 (1.9)	251	20.4 (1.9)
Overweight	51	25.1 (1.0)	98	24.8 (1.0)
Obese	66	32.0 (4.8)	170	31.6 (3.9)
SBP (mmHg)				
Normal Weight	145	120 (12)	251	121 (13)
Overweight	51	123 (12)	98	123 (12)
Obese	66	132 (13)	170	129 (14)
Females				
BMI (kg/m²)				
Normal Weight	156	20.4 (2.0)	307	20.5 (1.8)
Overweight	44	25.6 (1.2)	85	25.0 (0.8)
Obese	37	34.0 (6.0)	92	31.9 (4.9)*
SBP (mmHg)				
Normal Weight	156	110 (9)	307	109 (10)
Overweight	44	115 (9)	85	115 (13)
Obese	37	123 (11)	92	117 (9)†

Table 1: Mean (\pm SD) age, body mass index (BMI), and systolic blood pressure (SBP) for normal weight, overweight and obese adolescents categorized by gender and ethnicity. * $p < 0.05$ and † $p < 0.01$ vs. non-Hispanic whites

Obesity prevalence and elevated systolic blood pressure categorized by body mass

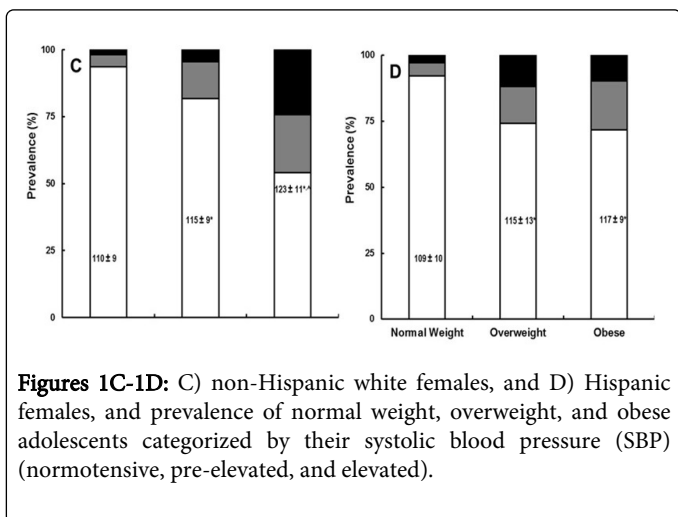
When groups (by gender & ethnicity) were categorized by body mass independent of SBP, the prevalence of elevated SBP for obese compared to normal weight cohorts was 3.5- and 12-fold greater for non-Hispanic white males and females, respectively, and 2- and 3-fold greater for Hispanic males and females, respectively (Figure 1A-D). Furthermore, the mean SBP for obese adolescents regardless of gender and ethnicity was greater ($p < 0.05$) than that for normal weight adolescents (Figure 1A-D). The prevalence of normotensive non-Hispanic white and Hispanic males decreased in overweight ($p = 0.016$)

and obese ($p < 0.0001$) cohorts, with reciprocal increases in elevated SBP in those body mass categories (Figure 1A and 1B).

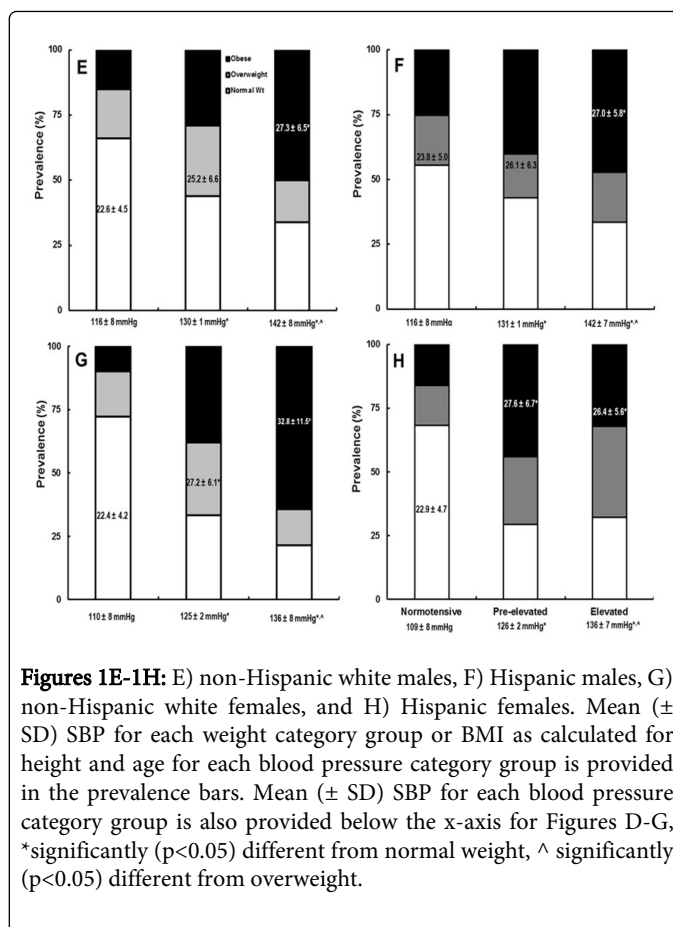


Figures 1A-1B: Prevalence of normotensive, pre-elevated, and elevated systolic blood pressure (SBP) in normal weight, overweight and obese adolescent A) non-Hispanic white males, B) Hispanic males.

Prevalence's between non-Hispanic and Hispanic males for each body mass category were not different ($p > 0.05$) (Figure 1A vs. 1B). The prevalence of normotensive non-Hispanic white and Hispanic females decreased in overweight ($p < 0.0001$) and obese ($p < 0.0001$) cohorts, with reciprocal increases in elevated SBP in those body mass categories (Figure 1C and 1D). Prevalence's between non-Hispanic and Hispanic females for each body mass category were not different ($p > 0.05$), with the exception of obese non-Hispanic females, which had a greater ($p < 0.0001$) prevalence of elevated SBP and lower ($p < 0.0001$) prevalence of normal SBP than Hispanic females (Figures 1C vs. 1D). Mean SBP was 5.5% greater in overweight adolescent females regardless of ethnicity compared to their normal weight cohorts (Figures 1C and 1D).



Figures 1C-1D: C) non-Hispanic white females, and D) Hispanic females, and prevalence of normal weight, overweight, and obese adolescents categorized by their systolic blood pressure (SBP) (normotensive, pre-elevated, and elevated).



Figures 1E-1H: E) non-Hispanic white males, F) Hispanic males, G) non-Hispanic white females, and H) Hispanic females. Mean (\pm SD) SBP for each weight category group or BMI as calculated for height and age for each blood pressure category group is provided in the prevalence bars. Mean (\pm SD) SBP for each blood pressure category group is also provided below the x-axis for Figures D-G, *significantly ($p < 0.05$) different from normal weight, ^ significantly ($p < 0.05$) different from overweight.

Obesity prevalence and elevated systolic blood pressure categorized by blood pressure

When groups (by gender & ethnicity) were categorized by SBP independent of BMI, the prevalence of obesity for adolescents with elevated SBP compared to normotensive cohorts was 3.5- and 6-fold greater for non-Hispanic white males and females, respectively, and 2-fold greater for both Hispanic males and females (Figure 1E-H). Furthermore, the mean BMI for adolescents with elevated SBP regardless of gender and ethnicity was greater ($p < 0.05$) than that for normotensive adolescents (Figure 1E-H). The prevalence of normal weight non-Hispanic white and Hispanic males decreased in pre-elevated ($p < 0.01$) and elevated ($p < 0.0001$) cohorts, with reciprocal increases in obesity in those blood pressure categories (Figure 1E and 1F). The prevalence of obese category was greater ($p < 0.001$) in Hispanic than non-Hispanic white males in the normotensive and pre-elevated blood pressure Categories, but not the elevated SBP category (Figure 1E vs. 1F). The prevalence of normal weight non-Hispanic white and Hispanic females decreased in pre-elevated ($p < 0.0001$) and obese ($p < 0.0001$) cohorts, with reciprocal increases in obesity in those SBP categories (Figure 1G and 1H). Prevalences between non-Hispanic and Hispanic females for each blood pressure category were not different ($p > 0.05$), with the exception of non-Hispanic females in the elevated SBP category, which had a nearly 2-fold greater ($p < 0.0001$) prevalence of obesity than Hispanic females (Figure 1G vs. 1H). Mean BMI was approximately 20% greater in adolescent females with pre-elevated SBP regardless of ethnicity compared to their normotensive cohorts (Figure 1G and 1H).

Regression analyses: Body Mass Index vs. Systolic Blood Pressure

Collectively, BMI demonstrated strong, positive and highly significant ($p < 0.0001$) correlations with SBP for all groups. The intercepts for males (non-Hispanic whites: $y = 103 + 0.87x$; $R = 0.385$ vs. Hispanics: $y = 107 + 0.70x$; $R = 0.292$) were greater ($p < 0.01$) than their female cohorts (non-Hispanic whites: $y = 91 + 0.92x$; $R = 0.499$ vs. Hispanics: $y = 95 + 0.70x$; $R = 0.330$). None of the slopes among the four groups were different ($p > 0.10$). Multinomial regression analyses demonstrated that: 1) ethnicity (Hispanic) had no significant impact on the development of pre-elevated or elevated SBP, 2) being overweight or obese had the most significant and greatest impact on developing pre-elevated and elevated SBP in females than in males, and 3) six or more hours of physical activity per week had a significant effect on ameliorating pre-elevated and elevated SBP in both males and females, with more pronounced effects on elevated SBP (Table 2).

	Female	Confidence Interval	Male	Confidence Interval
Pre-Elevated				
Normal Weight	1		1	
Overweight	1.01 -0.28	0.34 - 1.43	0.19 -2.62	0.04 - 0.69
Obese	1.38*** - 0.29	0.75 - 1.91	1.77*** -0.22	0.36 - 2.20
White	1		1	
Hispanic	0.2 - 0.26	0.13 - 0.68	0.31 -0.21	0.13 - 0.68
Physical Activity hr/wk	6+	0.38* - 0.18	0.28* -0.14	0.07 - 0.58
Elevated				
Normal Weight	1		1	
Overweight	1.64*** - 0.28	1.09 - 2.10	1.49*** -0.21	0.16 - 1.97
Obese	2.50*** - 0.28	1.67 - 2.80	1.30*** -0.17	1.05 - 1.72
White	1		1	
Hispanic	0.15 - 0.25	0.04 - 0.36	0.13 -0.16	0.08 - 0.37
Physical Activity hr/wk	6+	0.04* - 0.02	0.10* - 0.54	0.03 - 0.25
Pseudo R2	0.08		0.04	
Chi2	90.68		74.45	

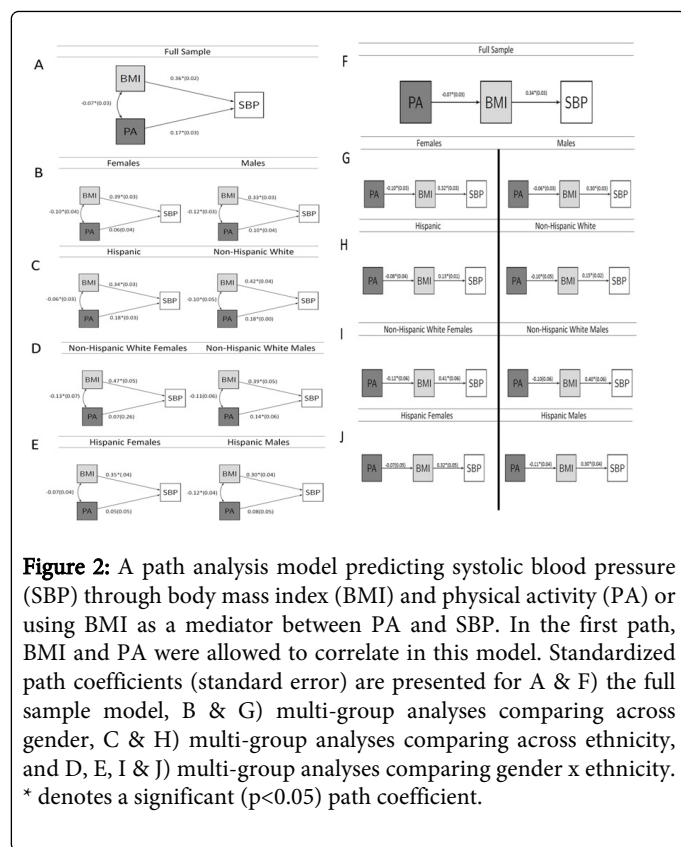
Table 2: Odds ratio (standard error) from multinomial, logistic regression of pre-elevated and elevated systolic blood pressure (SBP) by gender, * $p < 0.05$, ** $p < 0.01$, *** $p < 0.001$

Path analyses

Two different path models were estimated to help explain the complex relationships among BMI, PA and SBP. Additionally, each of these models were estimated through a multi-group modeling framework in order to uncover any gender and/or ethnic group differences underlying the way BMI and PA influence SBP.

The first model illustrated a causal relationship that specified BMI and PA as correlated, direct predictors of SBP (i.e., PA and BMI are predicted to have direct influence on SBP) (Figure 2A). Specifically, the path joining BMI and PA together illustrates that: 1) these predictors are correlative, and 2) the causal paths leading from BMI and PA to SBP indicate that SBP is a function of the two simultaneous predictors. BMI ($\beta = 0.36$) and PA ($\beta = 0.17$) were both significant ($p < 0.05$), positive predictors of SBP in the full sample model (Figure 2A). Furthermore, BMI and PA were significantly ($p < 0.05$) and negatively ($r = -0.07$) correlated with one another indicating that lower PA was correlated with higher BMI levels. A multi-group analysis comparing gender indicated that all causal relationships were comparable across males and females except that PA was not a significant ($p > 0.10$) predictor of SBP for females (Figure 2B). By collapsing gender and comparing across ethnicities, causal relationships were comparable. Specifically, BMI and PA were significant ($p < 0.05$) predictors for SBP (Figure 2C). Similarly, BMI and PA were significantly ($p < 0.05$) and negatively correlated (non-Hispanic whites: $r = -0.10$; Hispanics: $r = -0.06$). Differences across gender and ethnicity (i.e., comparing Hispanic females, Hispanic Males, non-Hispanic white females, and non-Hispanic white males) were not consistently significant. BMI and PA were only significantly ($p < 0.05$) and negatively correlated for Hispanic males ($r = -0.12$) and non-Hispanic white females ($r = -0.13$) (Figure 2D and 2E). Only PA was a significant ($p < 0.05$) and positive ($\beta = 0.14$) predictor of SBP for non-Hispanic white males (Figure 2D). Results for Hispanic males and females, and non-Hispanic white females demonstrated that only BMI significantly ($p < 0.05$) predicted SBP (Figure 2D and 2E).

Given that PA was hypothesized to have an impact on SBP within a multi-group modeling perspective, a second causal model was developed that introduced an indirect relationship between PA and SBP. This second causal model introduced BMI as a variable that mediates the relationship between PA and SBP (Figure 2F). Specifically, PA was modeled as a direct predictor of BMI, which in turn would be a proximate predictor of SBP. The full sample results indicated that lower PA was a significant ($p < 0.05$) predictor of higher BMI ($\beta = -0.07$), and higher BMI was a significant ($p < 0.05$) predictor of higher SBP ($\beta = 0.34$) (Figure 2F). However, this predictive pattern was not consistent across all groups in the multi-group analyses (Figures 2G-J). Specifically, PA was a significant ($p < 0.05$) and negative predictor for BMI only for non-Hispanic, white females ($\beta = -0.12$) (Figure 5D) and Hispanic males ($\beta = -0.11$) (Figure 2J).



Discussion

While the prevalence of hypertension among adult ethnic and racial minorities is disproportionately higher, the present study suggests that overweight and obesity, independent of ethnicity, is the primary contributing factor to increased SBP in rural adolescents. Furthermore, the impact of overweight and obesity on pre-elevated and elevated SBP was greater in females, regardless of ethnicity, than in males. These alarming results suggest that females in rural, disadvantaged populations may be at a greater risk for developing hypertension-related metabolic consequences than males. Because obesity is associated with insulin resistance, type II diabetes, and elevated blood pressure in children and adolescents [28-30], these children and adolescents are at a greater risk for later-onset hypertension-related CV risks, which may befall adolescents in this study population, especially females.

While collectively the prevalence of pre-high blood pressure (HBP) and HBP in children and adolescents are on the rise [25], the trends are disproportionately greater for African-Americans and Hispanics, but the increases by gender within those groups are not consistent [9]. For example, the increase in the prevalence of pre-HBP was greater in African-American females than males, but greater for Hispanic males than females [9] suggesting that gender contributes differentially to the early development of pre-elevated blood pressure depending on ethnicity and race. Furthermore, the prevalence of HBP for African-Americans did not increase. However, this prevalence for Hispanics increased significantly, but was not different between males and females [9] suggesting that Hispanics are at a greater risk for developing HBP than other groups and that this risk is independent of gender. While this may have been the case 10 years ago, the data from

the present study would suggest otherwise. Obesity, independent of ethnicity, was a greater contributing factor to the odds of developing elevated blood pressure (equivalent measure to HBP in Din-Dzietham et al. [9]) in females than in males in the present study. This OR data suggests that females, especially overweight and obese, are at greater risks for developing later-onset hypertension-related morbidities than males, regardless of ethnicity, which did not appear to be the case 10 years ago. Furthermore, while data for adults indicates that males are at a greater risk for CVD mortality and have a higher prevalence of hypertension [31-33], an important finding of this study suggests an opposite trend for adolescents. Obese adolescent females, regardless of ethnicity, may be more susceptible to the development of elevated SBP. Unfortunately, the causal mechanisms cannot be elucidated here; however, our previous [20] and current study suggest that the lack of sufficient physical activity is a critical contributing factor.

The regression analyses in the present study demonstrate that the increase in BMI is significantly and positively associated with increased SBP across the range of BMI values measured suggesting that increased adiposity is a contributing factor to the elevation in SBP for non-Hispanic white and Hispanic male and female adolescents. The higher intercepts for regressions for both non-Hispanic white and Hispanic males than their female cohorts and the parallel slopes among all groups indicates that males, regardless of ethnicity, present with higher SBP for any given BMI. Unfortunately, comparable regression data for children or adolescents are scarce, but estimated slopes (BMI vs. SBP) obtained from a study of Swiss children (mean age 12) were approximately 1.7 for males and 1.0 for females [34]. In the present study, all slopes were less than 1.0 indicating that the incremental increases in SBP for any BMI value is lower in US adolescents compared to the children in the Swiss study [35].

Collectively, the linear and multinomial, logistic regression analyses provide strong evidence for the impact of increased BMI on increased pre- and elevated SBP independent of gender and ethnicity, and the causal relationship analyses would corroborate this contention. More importantly, the causal relationship analyses substantiate the OR data demonstrating that 6+ hours of PA per week can significantly reduce pre- and elevated SBP in adolescents, which corresponds well with the American Heart Association's recommendation of 60 minutes of physical activity per day (i.e., NFL Play 60 Challenge). And while the relationships between elevated BMI and reduced PA may be inherent, the first model developed here (Figure 2) resulted in more consistent interactions among BMI, PA and SBP suggesting that regardless of the proximate factors that reduce PA levels and increase BMI, the two variables are virtually indistinguishable as they relate to increased SBP in adolescents, regardless of gender or ethnicity. Thus, unlike most studies, an important finding of the present study is that the increase in elevated SBP in adolescents is independent of ethnicity and gender suggesting that the implementation of strategies that increase PA levels should have global ramifications and not just regional and/or isolated to one particular group or gender. Ultimately, the disparities in cardiovascular disease that are currently recognized among adults may disappear suggesting that behavioral factors independent of culture or society may have a greater impact on the development of hypertension-related disorders later in life. Also, as to the best of our knowledge, this is the first study to apply a path analysis approach to better assess the impact of the contributing variables on blood pressure in adolescents. Nonetheless, the application of path analysis to SBP/BMI/PA dataset from adolescents was novel and unique and revealed many important relationships that may have otherwise remained undiscovered.

The present study demonstrates that the prevalence of elevated SBP after a single measurement was 14.8%, with the highest prevalence (15.3%) among Hispanic adolescents. This is consistent with previous research where the prevalence of elevated blood pressure was 19.4% overall, and the highest prevalence among Hispanics (25%) after the first screening [6]. However, when groups (by gender & ethnicity) were categorized by BMI independent of SBP, the prevalence of elevated SBP was greater for obese, non-Hispanic white males and females (42% and 24% vs. 35% and 10%, respectively) than for Hispanic males and females, which is higher than previously reported values for a large metropolitan sample population [35]. Although we report values based on a single measurement, we are cognizant of the fact that multiple measurements over time provide a better diagnostic value. Nonetheless, it has previously been shown that the strongest determinant of hypertension was BMI percentile, regardless of whether it was after the first screening or the third screening [6] suggesting that the current data produced from a single screening is robust and applicable. Thus, the current study extends the previous findings [6] and further implicates reduced PA levels as a contributing culprit in the progression of pre- and elevated SBP in adolescents, regardless of ethnicity or gender.

Limitations

Despite the strengths of the current findings, we recognize the limitations of the results, which necessitate cautious interpretation. Understanding the importance of repeated BP measurements to accurately diagnose hypertension in children and adolescents [23], the use of a single blood pressure reading has been successfully applied in adolescents to identify groups who are at a heightened risk for developing subsequent hypertension. Such data illustrate the usefulness of a single measurement in hypertension-related research [25]. We also recognize that objectively measured PA in children and adolescents is more accurate than self-reported values [17,36,37]; however, the fact that our data are in excellent agreement with these previous studies suggests that our findings are reliable and that these self-reported data provide some interpretative value.

Perspectives

The present study demonstrates that when groups (by gender & ethnicity) were categorized by body mass independent of blood pressure category, the prevalence of elevated SBP for obese adolescents was consistently greater than that compared to normal weight cohorts, regardless of ethnicity or gender. This is important because previous studies have demonstrated ethnicity and gender effects in children and adults [11,12,38,39]. Independent of the difference in age, this inconsistency of ethnicity and gender effects suggests that the impact of obesity on blood pressure among adolescents is now so severe that disparities are no longer detectable and that the ultimate contributing factors go well beyond ethnicity, culture and gender, but may lie in common, high-risk behaviors such as lack of physical activity and poor eating habits. The early development of elevated blood pressure increases the risk of these adolescents for hypertension-related morbidities as adults and emphasizes the need for early intervention and more frequent blood pressure screening among children and adolescents [37,40]. Ultimately, these outcomes should ameliorate the potential for overweight and obese adolescents of manifesting CV consequences as adults. Furthermore, the path analyses further implicate both reduced physical activity and increased BMI as contributing factors to the elevation in SBP. The odds ratio analyses

revealed that 6+ hr PA/wk reduced the probability of developing elevated SBP in both genders independent of body mass category or ethnicity suggesting that increased PA is a critical behavioral element to target to alleviate the consequences of obesity-related increases in SBP in young people regardless of ethnicity or gender.

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References

1. Sorof JM, Poffenbarger T, Franco K, Bernard L, Portman RJ (2002) Isolated systolic hypertension, obesity, and hyperkinetic hemodynamic states in children. *J Pediatr* 140: 660-666.
2. Babinska K, Kovacs L, Janko V, Dallos T, Feber J (2012) Association between obesity and the severity of ambulatory hypertension in children and adolescents. *J Am Soc Hypertens* 6: 356-363.
3. Winkleby MA, Robinson TN, Sundquist J, Kraemer HC (1999) Ethnic variation in cardiovascular disease risk factors among children and young adults: Findings from the third national health and nutrition examination survey, 1988-1994. *JAMA* 281: 1006-1013.
4. Dekkers JC, Snieder H, Van Den Oord EJ, Treiber FA (2002) Moderators of blood pressure development from childhood to adulthood: a 10-year longitudinal study. *J Pediatr* 141: 770-779.
5. Rosner B, Prineas R, Daniels SR, Loggie J (2000) Blood pressure differences between blacks and whites in relation to body size among US children and adolescents. *Am J Epidemiol* 151: 1007-1019.
6. Sorof JM, Lai D, Turner J, Poffenbarger T, Portman RJ (2004) Overweight, ethnicity, and the prevalence of hypertension in school-aged children. *Pediatrics* 113: 475-482.
7. Saydah S, Bullard KM, Cheng Y, Ali MK, Gregg EW, et al. (2014) Trends in cardiovascular disease risk factors by obesity level in adults in the United States, NHANES 1999-2010. *Obesity* (Silver Spring).
8. Salvadori M, Sontrop JM, Garg AX, Truong J, Suri RS, et al. (2008) Elevated blood pressure in relation to overweight and obesity among children in a rural Canadian community. *Pediatrics* 122: e821-827.
9. Din-Dzietam R, Liu Y, Bielo MV, Shamsa F (2007) High blood pressure trends in children and adolescents in national surveys, 1963 to 2002. *Circulation* 116: 1488-1496.
10. Rodriguez R, Mowrer J, Romo J, Aleman A, Weffer SE, et al. (2010) Ethnic and gender disparities in adolescent obesity and elevated systolic blood pressure in a rural US population. *Clin Pediatr (Phila)* 49: 876-884.
11. Freedman DS, Dietz WH, Srinivasan SR, Berenson GS (1999) The relation of overweight to cardiovascular risk factors among children and adolescents: the Bogalusa Heart Study. *Pediatrics* 103: 1175-1182.
12. Freedman DS, Khan LK, Dietz WH, Srinivasan SR, Berenson GS (2001) Relationship of childhood obesity to coronary heart disease risk factors in adulthood: the Bogalusa Heart Study. *Pediatrics* 108: 712-718.
13. Must A, Strauss RS (1999) Risks and consequences of childhood and adolescent obesity. *Int J Obes Relat Metab Disord* 23 Suppl 2: S2-11.
14. Serdula MK, Ivery D, Coates RJ, Freedman DS, Williamson DF, et al. (1993) Do obese children become obese adults? A review of the literature. *Prev Med* 22: 167-177.
15. Field AE, Cook NR, Gillman MW (2005) Weight status in childhood as a predictor of becoming overweight or hypertensive in early adulthood. *Obes Res* 13: 163-169.
16. Boreham C, Twisk J, Neville C, Savage M, Murray L, et al. (2002) Associations between physical fitness and activity patterns during

- adolescence and cardiovascular risk factors in young adulthood: the Northern Ireland Young Hearts Project. *Int J Sports Med* 23: 22.
17. Mark AE, Janssen I (2008) Dose-response relation between physical activity and blood pressure in youth. *Med Sci Sports Exerc* 40: 1007-1012.
 18. Boreham CL, Twisk J, van Mechelen W, Savage M, Strain J, et al. (1999) Relationships between the development of biological risk factors for coronary heart disease and lifestyle parameters during adolescence: The Northern Ireland Young Hearts Project. *Public Health* 113: 7-12.
 19. Maximova K, O'Loughlin J, Paradis G, Hanley JA, Lynch J (2009) Declines in physical activity and higher systolic blood pressure in adolescence. *Am J Epidemiol* 170: 1084-1094.
 20. Rodriguez R, Weffer SE, Romo J, Aleman A, Ortiz RM (2011) Reduced Physical Activity Levels Associated with Obesity in Rural Hispanic Adolescent Females. *Childhood Obesity* 7: 194-205.
 21. Kuczmarski RJ, Ogden CL, Guo SS, Grummer-Strawn LM, Flegal KM, et al. (2002) 2000 CDC Growth Charts for the United States: methods and development. *Vital Health Stat* 11: 1-190.
 22. Barlow SE; Expert Committee (2007) Expert committee recommendations regarding the prevention, assessment, and treatment of child and adolescent overweight and obesity: summary report. *Pediatrics* 120 Suppl 4: S164-192.
 23. National High Blood Pressure Education Program Working Group on High Blood Pressure in Children and Adolescents (2004) The fourth report on the diagnosis, evaluation, and treatment of high blood pressure in children and adolescents. *Pediatrics* 114: 555-576.
 24. Ostchega Y, Carroll M, Prineas RJ, McDowell MA, Louis T, et al. (2009) Trends of elevated blood pressure among children and adolescents: data from the National Health and Nutrition Examination Survey 1988-2006. *Am J Hypertens* 22: 59-67.
 25. Falkner B, Gidding SS, Portman R, Rosner B (2008) Blood pressure variability and classification of prehypertension and hypertension in adolescence. *Pediatrics* 122: 238-242.
 26. Janssen I, Katzmarzyk PT, Boyce WF, King MA, Pickett W (2004) Overweight and obesity in Canadian adolescents and their associations with dietary habits and physical activity patterns. *J Adolesc Health* 35: 360-367.
 27. Muthén LK, Muthén BO (1998-2012) *Mplus user's guide*. (7th edn), Los Angeles: Muthén & Muthén.
 28. Lurbe E, Torro I, Aguilar F, Alvarez J, Alcon J, et al. (2008) Added impact of obesity and insulin resistance in nocturnal blood pressure elevation in children and adolescents. *Hypertension* 51: 635-641.
 29. Goran MI, Ball GD, Cruz ML (2003) Obesity and risk of type 2 diabetes and cardiovascular disease in children and adolescents. *J Clin Endocrinol Metab* 88: 1417-1427.
 30. Caballero AE, Bousquet-Santos K, Robles-Osorio L, Montagnani V, Soodini G, et al. (2008) Overweight Latino Children and Adolescents Have Marked Endothelial Dysfunction and Subclinical Vascular Inflammation in Association With Excess Body Fat and Insulin Resistance. *Diabetes Care* 31: 576-582.
 31. Kalin MF, Zumoff B (1990) Sex hormones and coronary disease: a review of the clinical studies. *Steroids* 55: 330-352.
 32. Lemieux S, Després JP, Moorjani S, Nadeau A, Thériault G, et al. (1994) Are gender differences in cardiovascular disease risk factors explained by the level of visceral adipose tissue? *Diabetologia* 37: 757-764.
 33. Regitz-Zagrosek V, Lehmkuhl E, Weickert MO (2006) Gender differences in the metabolic syndrome and their role for cardiovascular disease. *Clin Res Cardiol* 95: 136-147.
 34. Chioloro A, Cachat F, Burnier M, Paccaud F, Bovet P (2007) Prevalence of hypertension in schoolchildren based on repeated measurements and association with overweight. *J Hypertens* 25: 2209-2217.
 35. McNiece KL, Poffenbarger TS, Turner JL, Franco KD, Sorof JM, et al. (2007) Prevalence of hypertension and pre-hypertension among adolescents. *J Pediatr* 150: 640-644, 644.
 36. Brage S, Wedderkopp N, Ekelund U, Franks PW, Wareham NJ, et al. (2004) Features of the metabolic syndrome are associated with objectively measured physical activity and fitness in Danish children: the European Youth Heart Study (EYHS). *Diabetes Care* 27: 2141-2148.
 37. Knowles G, Pallan M, Thomas GN, Ekelund U, Cheng KK, et al. (2013) Physical activity and blood pressure in primary school children: a longitudinal study. *Hypertension* 61: 70-75.
 38. Rosner B, Cook N, Portman R, Daniels S, Falkner B (2009) Blood pressure differences by ethnic group among United States children and adolescents. *Hypertension* 54: 502-508.
 39. Carson AP, Howard G, Burke GL, Shea S, Levitan EB, et al. (2011) Ethnic differences in hypertension incidence among middle-aged and older adults: the multi-ethnic study of atherosclerosis. *Hypertension* 57: 1101-1107.
 40. Chen X, Wang Y (2008) Tracking of blood pressure from childhood to adulthood: a systematic review and meta-regression analysis. *Circulation* 117: 3171-3180.