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Research Article

Stimulus response of blood pressure in black and white young individuals helps explain racial divergence in adult cardiovascular disease: The Bogalusa Heart Study

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Abstract

Blood pressure (BP) is a highly variable physiologic trait with short-term and long-term fluctuations within the same individual at different time points. The burden of BP on the cardiovascular (CV) system has been studied in terms of multiple cross-sectional BP measurements at rest, response of BP to stresses, and long-term longitudinal variability of BP. Observations from childhood are available extending into early middle age in the biracial (black-white) population of Bogalusa, Louisiana. Left ventricular mass index was used to illustrate damaging effects on the CV system by both resting BP levels and fluctuations. Long-term BP variability reflecting intermittent and repeated variability was shown to have a greater effect in blacks. The childhood BP response to several stressors was found to be greater in blacks. These observations suggest that, although at rest a greater vagal effect occurs in blacks, they show a greater response when reacting to a stimulus. This, along with aspects such as carbohydrate-insulin metabolism or other biochemical/physiological differences, may account for the greater acceleration of CV atherosclerosis in blacks. The racial contrasts suggest, in part, that effects of lipoproteins may be greater in whites, whereas the effects of excess BP levels and variability of BP and Na^+-K^+ intake and diet as well as other environmental effects result in more CV damage in blacks. The strong association of hemodynamic measures with anatomic, metabolic, and environmental factors emphasizes the need to begin prevention of risk factors at an early age. Taken together, understanding racial (black-white) contrasts to stress contribute to both prevention and treatment of hypertension, especially for black males. J Am Soc Hypertens 2011;5(4):230–238.

Keywords: Blood pressure; hypertension; ethnicity; black/white contrasts; stimulus response; sympathetic activity.

Introduction

The etiology of essential hypertension is multifactorial in nature, involving an interplay of genes and a host of other factors from lifestyle to environment. Despite decades of research, specific genetic and pathophysiologic mechanisms mediating the elevation of blood pressure (BP) are not completely understood.[1]

BP is a highly variable physiologic trait that increases with age and body growth in childhood and has 2 aspects: (1) different levels among individuals at one time point (among-individual variability) and (2) short-term and long-term fluctuations within the same individual at different time points (within-individual variability).[2,3] Studies have shown that wide variability of BP over a long-term period or 24 hours is associated with severity of end-organ damage and an increase of subsequent cardiovascular (CV) and cerebrovascular events, even after adjusting for the mean BP levels.[4,5]

Recently, long-term BP variability, as well as 24-hour monitoring, has received increasing attention because of its practical implications in the prevention and treatment

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of hypertension and related disorders.[4–11] However, whether long-term BP variability, especially beginning in childhood, is a more powerful parameter than their levels per se in predicating cardiac/cerebral/renal diseases is unknown, particularly in blacks compared with whites. Further, studies in childhood among blacks and whites over time may provide clues to mechanisms of BP control with important implications for treatment and prevention.

Observations in Childhood

The within-individual variability has been studied in terms of 24 hours by ambulatory monitoring but also can be measured over seconds (beat-to-beat changes related to heart rate).[8] Children with higher casual levels of BP, but not classified as hypertension, had a greater percentage of high levels persisting over 24 hours by ambulatory measurements comprising a greater BP load. Further, the decrease (nondipping) that occurs at night is less in blacks than in whites, even in childhood.[12–14] We found that a 2 series observation of casual measurements may misclassify a child as having hypertension and that a series of 4 to 6 observations with replicate measurements is needed to establish hypertension in children. Importantly, a parental history of hypertension has an effect as expected.[13] Observations of children in the top quintile show an increase in cardiac size by left ventricular wall thickness in systole, even adjusted for body surface area, ponderosity, race, and gender.[15] Echocardiographic observations also show racial differences in hemodynamic parameters in childhood, eg, higher cardiac output in white males and higher peripheral resistance in blacks (Figure 1).[16]

Further studies conducted on children and adolescents selected at high, medium, and low levels provided additional clues to mechanisms of BP control.[17,18] Based on diastolic BP (fourth phase), children 7 to 15 years old were grouped into 5 strata: 1 (low BP) to 5 (high BP), representing all children of the extreme 2% for each strata 1 and 5; 70% random sampling fraction of the next 4% to 9% for strata 2 and 4; and 3% to 8% random sampling fraction of the remaining children for stratum 3, for each race-gender specific group. Strata 1 and 2 were combined to form the low stratum; strata 3 the medium stratum; and strata 4 and 5 the high stratum. Black/white and gender contrasts were observed, as shown in Table 1. Of particular interest are the higher BP levels and slower heart rate in black children, and lower renin levels and less of a relation of BP levels to body weight and fatness in blacks. These observations also showed lower 24-hour urinary excretion of K^+ in blacks, and implicated the renin-angiotensin system and dietary sodium intake as predictors of childhood BP levels and development of hypertension in blacks. The faster heart rates, a greater relation of BP to body fatness in white children, and higher glucose and lower insulin levels suggested more metabolic and adrenergic influences on BP levels in white versus black subjects; electrolyte exposure and state of the renin-angiotensin system have more influence among black subjects.

The notion of a greater parasympathetic role in childhood BP in blacks with slower heart rate was challenged with observations from the Coronary Artery Risk Development in Young Adults (CARDIA) study showing faster heart rates in blacks.[19] Further, observations from Calhoun and coworkers[20] and Victor and coworkers[21] indicated increased sympathetic activity in black adults with hypertension by leg peroneal nerve impulse studies. Our heart rate variability studies earlier on children and adolescents in Bogalusa, Louisiana, indicated more sympathetic

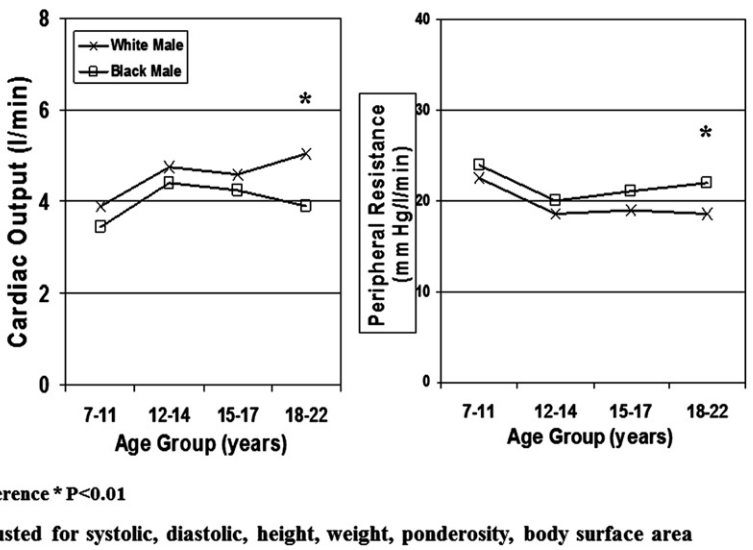

Figure 1. Echocardiographic studies show greater cardiac output in white boys and greater peripheral resistance in black boys. Reprinted with permission.[16]