Hypertension in Africa:
Redressing the burden of cardiovascular disease using cost-effective non-pharmacological approaches

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ABSTRACT

Hypertension may affect approximately one fifth or more of all adult South Africans. Despite the considerable evidence derived from economically developed countries to indicate the extent to which hypertension contributes to cardiovascular disease (CVD), it is only more recently that data has emerged from the African continent to support a contention that hypertension is the principal risk factor for CVD in African populations and that CVD accounts for a major proportion of deaths in the elderly and in younger adults in rural Africa. Active engagement in the harsh realities of managing this complex clinical trait should therefore be foremost on the minds of the healthcare sector in Africa. In this regard there are unique challenges.

In the present personal review we synthesise the evidence for or against the view that at a public health level, the answer to significantly reducing the burden of CVD produced by hypertension in African populations, may lie in something as simple as generating a healthier lifestyle. In this regard, we place recent evidence obtained from South African studies of the importance of modifiable cardiovascular risk factors related to hypertension, including salt intake and obesity, in the context of previously published evidence. We highlight the very recent and the first substantive evidence derived from an African community to show that salt intake indeed contributes to a significant portion of blood pressure (BP) variability in African populations, but this effect may be hidden because the impact is largely on central (aortic) rather than brachial BP.

We also discuss the increasing evidence to show that in African populations, the adverse effects of the epidemic of obesity that faces emerging communities is likely to account for a substantial proportion of cardiovascular risk not through marked effects on brachial BP, but through indirect effects by promoting the adverse effects of BP on the heart. In the present review we therefore argue that despite limited absolute effects of salt intake and obesity on brachial BP, a marked benefit could be gained by the BP effects of salt restriction and body weight reduction in African communities. SAHeart 2011; 8:28-36

INTRODUCTION

In economically developed countries there is considerable evidence for a continuous and positive relationship between blood pressure (BP) and both myocardial infarction and stroke.[1] The impact of BP on these catastrophic events extends well below currently accepted BP thresholds for the diagnosis of hypertension and the incremental risk for cardiovascular disease (CVD) with increasing BP is noted over a wide age range.[1] As reviewed by guideline committees for the diagnosis and management of hypertension,[2-4] numerous large clinical intervention studies have provided strong evidence to indicate that decreasing BP with a variety of anti-hypertensive agents substantially reduces the risk for cardiovascular events, thus establishing the role of BP as a key factor in the pathogenesis of CVD. More recent data derived from population or hospital-based studies conducted in economically developing populations including African populations, clearly demonstrate that in comparison to other cardiovascular risk factors, hypertension is the dominant risk factor for myocardial infarcts,[5] stroke[6-8] and heart failure of non-ischaemic origins[9] in these communities. As these conditions are the leading causes of death in the elderly and the third commonest cause of death in younger age groups in rural South Africa,[10] hypertension clearly accounts for a significant burden on health care in South Africa.

CONTROL OF BP IN AFRICA AND THE WORLD

Other than in Canada where 80 to 86% of hypertensives of the population of the province of Ontario have BP values that are controlled to target,[11] there is little evidence to suggest that health-care providers across continents are serious about controlling BP to currently accepted target levels (<140/90mm Hg).[2-4] Despite the fact that 21 to 59% of individuals within a wide range of populations have hypertension,[11-14] BP control rates are far below acceptable standards with 62 to 77% of hypertensives having an uncontrolled BP in the majority of developed countries.[12-15] The situation in economically emerging communities is far more disturbing, as 93% of black South African hypertensives may have a BP that is uncontrolled.[16] Hence, in countries where these national figures are available, hypertension is a neglected condition. Globally, the level of disregard for the management of this silent, but catastrophic condition is unimaginable.

AFRICAN HEALTHCARE SYSTEMS UNDER PRESSURE

It is now well accepted that Africa faces different healthcare challenges as compared to other continents because of a lack of resources and infrastructure and because of competition for healthcare resources through the burden of diseases of poverty, and the HIV/AIDS epidemic and its associated diseases. It is disappointing to say the least that in contrast to The Tobacco Products Act of 1993 and The Liquor Act of 2003 (act 59), which effectively limited the abuse of these substances, South Africa has not risen to the challenge of instituting cost-effective population interventions, which could considerably alleviate the burden of disease produced by BP effects.

Whilst at a national level in European countries, through pressure exerted at a number of levels, the food industry has reduced the salt content of their processed products, in countries such as South Africa this, to our knowledge, has only ever been discussed without concrete actions following suite. Furthermore, whilst education programmes have been instituted in a variety of European and North American countries at school and other levels to promote a decrease in calorie intake, or an increase in calorie expenditure, hence attempting to modify the emerging epidemic of obesity, there is no evidence at a national level in any African country that we are aware of, that these programmes are even being considered. We are dumbfounded by the national inertia in African countries in this regard, as by reducing BP at a national level using non-drug interventions could free up valuable health-care resources to further combat the HIV/AIDS epidemic and associated communicable diseases and prevent diseases of poverty. However, it is all very well being critical of healthcare providers if the evidence to support these notions in Africa is scanty.

Much of the reluctance to promote a healthier lifestyle in Africa may be derived from a distinct lack of substantial evidence to demonstrate the extent to which salt intake or obesity contributes toward the variation in BP at a population level on this continent. However, recent evidence has addressed some of these issues and concerns. In the following personal review we will discuss this evidence in the context of previous data originating from the African continent and other African populations.

SALT INTAKE AND HYPERTENSION IN GENERAL

Large cross-sectional studies indicate that dietary salt intake as indexed by increases in urinary Na^+ excretion or increases in urinary Na^+/K^+, is associated with BP.[17-20] Moreover, intervention studies have demonstrated that reductions in Na^+ intake decrease BP and the risk for hypertension.[21-26] As a consequence of these[17-26] and other findings, guidelines recommend that changes in salt intake should constitute part of lifestyle changes for the management of hypertension.[2-4] Nevertheless, it is important to note that the effect of salt intake on BP still generates controversy. In this regard, no long-term intervention studies have evaluated whether reductions in salt intake decrease cardiovascular mortality and morbidity. Moreover, although the Dietary Approaches to Stop Hypertension (DASH) trial provides robust evidence to show that reductions in Na^+ intake result in significant decreases in BP,[24,25] there are still a number of arguments against a strong role for salt in BP control. Indeed, the DASH study was conducted in a small study sample for a multicentre clinical trial (n = 412),[24,25] and other salt intervention studies were similarly conducted in relatively small study samples.[26] Furthermore, a meta-analysis of 57 trials derived from the Cochrane database with a considerable overall sample size (n = 9009), which included data from the DASH study, suggests that in Caucasians at least there is only a relatively small size effect on BP (-4.2 systolic [SBP]

and -2.0 diastolic [DBP] BP in hypertensives) of considerable reductions in salt intake.[26] These data are more in-line with the relatively small quantitative impact of salt intake on BP in the multicentre Intersalt study conducted in 52 centres across the world (n = 10 079).[16] Nevertheless, the relatively small quantitative effect of salt intake on BP may only apply to the dominant ethnic groups assessed in many of these studies and that is, in Caucasian populations. What is the evidence that African populations consume excessive salt in their diet and is there better evidence for a role of salt intake in contributing toward a marked proportion of BP variability at a population level in African groups?

SALT INTAKE IN AFRICA

In two studies with sample sizes of 110 to 291, the current evidence points to a considerably greater quantity of Na^+ being consumed by African populations than that recommended by guidelines,[27,28] even amongst hypertensives receiving antihypertensive medication.[28] Furthermore, at least in the South African context, hypertensives are not receiving advice on how to reduce Na^+ intake.[27,28] Extending the previous data obtained by our group from one of these studies,[28] Table 1 shows the mean 24-hour urinary Na^+ excretion values (an index of salt intake) in the largest database of 24-hour urinary salt excretion measurements obtained in African populations (n = 640). Clearly, in South Africa at least, in urban developing communities of African ancestry, a significant proportion (68%) of people are consuming Na^+ beyond the recommended daily allowance of 65mmol/day and 44.2% are consuming Na^+ at a level that exceeds the more liberal target of 100mmol/day. Moreover, treated hypertensives consume a similar amount of Na^+ as untreated hypertensives. There are many potential reasons why either healthcare professionals or patients are reluctant to take to heart the recommendation to reduce Na^+ intake. Importantly, what requires careful consideration is whether there is indeed significant evidence to suggest that salt intake accounts for a considerable proportion of an increased BP in Africa.

TABLE 1: Daily (24-hour) sodium (Na^+) intake as determined from 24-hour urinary Na^+ excretion, the proportion of people with 24-hour Na^+ intake above the recommended daily allowance of 65mmol/day, and the proportion of people with 24-hour Na+ intake above 100mmol/day in a community sample of African ancestry (African Programme on Genes in Hypertension) (see references[30,42,44-48] for study design and details).

	Urinary Na^+	% with urinary Na^+ excretion	
	Mean (±SD) (mmol/day)	>65 mmol/day	>100 mmol/day
All participants (n = 640)	105±73	68%	44%
Treated hypertensives (n = 156)	89±59	62%	37%
Untreated hypertensives (n = 154)	106±79	64%	42%
Obese* (n = 273)	103±68	69%	42%
Non-obese (n = 367)	107±77	68%	46%
Men (n = 222)	111±73	68%	50%
Women (n = 418)	102±73	68%	41%

**body mass index≥30kg/m²*

WHAT IS THE EVIDENCE TO SUPPORT A MAJOR EFFECT OF SALT INTAKE ON BP IN GROUPS OF AFRICAN ORIGINS?

The evidence to support a relationship between salt intake and BP at a population level in Africa has, until recently, been almost non-existent. Three studies (n = 110, 169 and 291) have failed to show relationships between urinary salt excretion and BP in black South Africans.[27-29] Moreover, in the highly cited Intersalt study showing a relationship between urinary salt excretion and BP in an analysis of data obtained from 52 centres across the world (n = 10 079), the only data included in the analysis from Africa were from Zimbabwe (n = 195) and Kenya (n = 176) and the relationship between salt intake and BP in these population samples was either absent (Kenya) or weak at best (Zimbabwe).[21] Is there evidence from intervention studies to suggest that salt intake could contribute to a considerably greater proportion of BP variability in groups of African descent?

Importantly, in the DASH study, reductions in Na^+ intake were particularly effective in people of black African ancestry (57% of the sample),[24,25] with a mean change in SBP of -8.1mm Hg in this ethnic group[25] and the meta-analysis of all studies with groups of black African descent similarly indicated that SBP decreased by -6.4mm Hg on a low Na^+ diet.[26] However, the meta-analysis[26] suggested that there was insufficient data to draw conclusions on the impact of decreases in Na^+ intake on BP in groups of African descent. Indeed, as compared to data from the 8 487 Caucasians that could be included in the meta-analysis, data from only 522 participants of black African descent could be included.[26]

Despite some evidence in favour of a greater effect of Na^+ reduction on BP in groups of African descent as compared to alternative groups, neither the DASH,[24,25] nor any other study included in the recent meta-analysis[26] evaluated the effect of a reduced Na^+ intake on participants from the African continent. Although this may appear to be a trivial omission in the eyes of some, it is well recognised that the capacity to reduce BP on a low Na^+ diet differs enormously from individual to individual, possibly because of differences in the renal handling of salt. This may occur to the extent that whilst in some individuals BP decreases considerably in response to a reduced Na^+ intake, in others BP may even increase on a low Na^+ diet. Is this a factor that needs careful consideration? Indeed, our group in collaboration with colleagues on the European continent, has provided evidence to indicate that the genetic factors that contribute toward the renal handling of salt are markedly different in black African populations in South Africa as compared to European populations.[30] The obvious question therefore is what explains the previous evidence to show a lack of relationship between urinary indices of salt intake and BP in groups of African ancestry in South Africa?[27-29]

NEW EVIDENCE FOR SALT EFFECTS ON BP IN GROUPS OF AFRICAN ANCESTRY

There are many experts who believe that despite measures of urinary Na^+ excretion being substantially better measures of salt intake than dietary recall, a lack of relationship between urinary indices of salt intake and BP in Africa[27-29] may reflect the inability of these measures to fully capture actual salt intake. However, we have recently hypothesised that the main effect of salt intake is on pulse pressure (SBP – DBP), the dynamic component of BP and that this effect is possibly best detected in the aorta (central BP).[31] This hypothesis is derived from previous data to indicate that despite reductions in Na^+ intake producing marked SBP effects, either no significant effect[26] or only a comparatively modest effect[25] on DBP may occur.

Our recent data[30] provide clear evidence in 635 randomly recruited participants from the African Programme on Genes in Hypertension (APOGH) to show that in an urban community of African ancestry (South West Township of Gauteng [SOWETO]), a urinary index of salt intake is strongly and independently related to central (aortic) SBP and pulse pressure. The independent relationship between a urinary index of salt intake and brachial artery pressures in this study was weak at best and no relationship with DBP was noted.[31] The difference in the impact of salt intake on central aortic and peripheral brachial BP can be attributed to the marked differences in the pathophysiological determinants of brachial and aortic pulse pressure and SBP. What are the implications of these data for South Africa and possibly Africa as a whole?

IMPLICATIONS FOR AFRICA OF THE IMPACT OF SALT INTAKE ON CENTRAL (AORTIC) BP

Even in the context of the limitation of being unable to fully capture the impact of salt intake on BP from 24-hour urinary salt excretion data, our data nevertheless provide the first convincing evidence that abnormalities in salt intake are indeed independently associated with BP in groups of African ancestry in Africa.[31] Although our study was an association study, and hence only implies a cause-effect relationship, the sample size (n = 635) and the probability values ($p<0.0001$) for an association between a urinary index of salt intake and central BP demonstrated in this study[31] cannot be considered to occur by chance. Thus, the effects of salt intake on BP may have previously been underestimated in African populations.[27-29] The robust relationships between a urinary index of salt intake and central aortic BP in groups of African ancestry[31] is consistent with the higher lymphocyte Na^+ content previously reported to occur in hypertensives as compared to normotensives of African descent.[32] From the data obtained in the APOGH study,[31] we suggest that salt intake will increase aortic SBP 1.45 times more than it will brachial BP. Hence, if we assume that a reduction in Na^+ intake of 100mmol/day (3.7g Na^+ per day which equates to 9.2g or about 4 teaspoons of table salt per day) reduces brachial SBP by -8.1mm Hg in groups of African descent as suggested by the DASH study,[25] then presumably aortic SBP may decrease by as much as 11.8mm Hg in response to a decrease in Na^+ intake. Only intervention studies will be able to assess this hypothesis.

The second implication of the finding that salt intake is related to aortic SBP, with minimal effects on brachial BP in the APOGH

study,[31] is that there is now increasing evidence that central pressures predict cardiovascular events and damage beyond brachial artery BP.[33-37] Therefore, there is a distinct possibility that an excess salt intake produces cardiovascular damage that exceeds that predicted by conventional BP measurements. Is there evidence for similar effects of aortic beyond brachial BP on cardiovascular damage in African populations? In this regard, we have demonstrated in the APOGH study that factors which contribute toward central pressures are indeed associated with left ventricular mass independent of brachial pressure.[38] As there is no question that left ventricular mass predicts cardiovascular outcomes beyond brachial BP, we assume that this evidence translates into outcomes as well. Thus, we believe our data provide convincing support for a need to reduce Na^+ intake to achieve optimal BP values in groups of African ancestry in South Africa. Nevertheless, intervention studies are required to provide a higher level of evidence to support these data.

The third implication of the finding that salt intake affects aortic SBP, with minimal effects on brachial BP in the APOGH study,[31] relates to potential therapy. In this regard, there is little evidence for a strong central pulse pressure lowering effect of any currently recommended anti-hypertensive agent beyond that produced by decreases in static BP (mean arterial pressure). Thus, presently there may be no real alternative to treating Na^+ induced increases in central BP independent of static pressures, but to reduce Na^+ intake.

A word of caution is required regarding the implications of the findings that salt intake is related to aortic SBP, with minimal effects on brachial BP in the APOGH study.[31] These results do not suggest that central aortic BP measurements should replace brachial artery BP measurements when assessing the effects of reduction in salt intake on BP. The use of costly technology such as that required to measure aortic BP is unlikely to improve adherence to anti-hypertensive (including lifestyle) treatment. Furthermore, whether this technology is cost-effective is a question that requires considerably more research to answer. The finding that salt intake is related to aortic SBP, with minimal effects on brachial BP,[31] may nevertheless lend a degree of confidence in the efficacy of the effect of Na^+ restriction on BP when only small decreases in brachial BP occur.

OBESITY AND HYPERTENSION IN GENERAL

There is now substantial evidence from population-based studies with large study samples (n = 10 969 to 15 063) conducted out of Africa in favour of obesity being a major determinant of BP and the development of hypertension.[39,40] Indeed, the odds of developing hypertension are 1.7 to 3.4 times greater in obese individuals as compared to lean individuals.[40] Further, there is considerable evidence also obtained out of Africa to indicate that weight reduction results in decreases in BP. In a meta-analysis of a number of weight reduction studies, with a total sample size of 4 874 participants, the estimated decrease in BP that will occur for every 5.1kg loss of weight over 16 months was 3.5 to 4.5mm Hg.[41] Moreover, in the Atherosclerosis Risk in Communities (ARIC) study involving 3 245 participants, a 1.5 to 2.0 fold chance of hypertension remission was reported to occur for every 1kg decrease in body weight over 9 years.[42]

WHAT IS THE EVIDENCE TO SUPPORT A MAJOR EFFECT OF OBESITY ON BP IN AFRICA?

Despite the emerging epidemic of obesity in black African populations in South Africa,[43] the size effect of excess adiposity on ambulatory (an assessment of BP that discounts alerting responses, includes 24-hour BP profiles and excludes observer bias) and conventional BP in African populations has only recently been reported on by our group in the APOGH study in 300 participants, 65% of whom were either overweight or obese and 42% of whom were hypertensive.[44] In this regard, after multiple adjustments, every 15cm (one standard deviation) increase in waist circumference (the index of adiposity most closely related to BP) was associated with a 4.04mm Hg increase in 24-hour systolic BP and a 4.33mm Hg increase in 24-hour diastolic BP.[44] Moreover, after multiple adjustments, every 15cm (one standard deviation) increase in waist circumference was associated with a 3.35mm Hg increase in conventional SBP and a 3.27mm Hg increase in conventional DBP.[44] It should be obvious that the impact of large changes in central adiposity, only produce modest effects on BP.

It may be argued that the relatively limited impact of excessive adiposity on BP reported on in the APOGH study[44] may reflect an inadequate sample size (n = 300). Therefore, extending these previous data on relationships between indices of adiposity and

conventional or ambulatory BP from our group.[44] Table 2 shows the impact of increases in either waist circumference or body mass index on conventional or 24-hour BP evaluated in a much larger study sample of randomly recruited participants of African descent in the APOGH with 24-hour ambulatory BP values that met with pre-specified quality control criteria (n = 657). Importantly, these data represent the largest study conducted so far assessing the impact of an excess adiposity on BP in African populations. Even with a significant study sample for an ambulatory BP study, in South Africa at least the independent impact of adiposity on BP in groups of African descent is remarkably small. These data raise the question of exactly how effective major efforts at reducing excess adiposity in Africa would be at achieving BP related decreases in cardiovascular risk at a population level. In this regard, despite the data from APOGH (Table 2), there are a number of reasons why we believe that promoting weight loss will substantially reduce BP related cardiovascular risk, hypotheses that nevertheless still need to be further explored. The following discussion outlines these arguments.

GENETIC MODIFIERS OF OBESITY EFFECTS ON BP

We have recently demonstrated that the impact of an excessive adiposity on BP may in fact be genetically pre-determined.[45] Thus, it is possible that in certain pre-identified obese individuals, weight loss programmes may have profound effects on BP and that as long as these as opposed to alternative individuals are targeted for weight loss programmes, far more substantial effects of weight loss on decreases in BP could be produced than those estimated at a community level by our group. At present, in further studies we are attempting to identify the impact of body mass index or waist circumference on conventional and 24-hour BP at a community level in genotype-specific groups and to identify the exact mechanism of this genetic effect on obesity-BP relations. Importantly however, weight loss studies are required in genotype-specific groups to provide convincing evidence of these effects.

BLOOD PRESSURE AND AN EXCESS ADIPOSITY ACT IN SYNERGY TO ACCOUNT FOR CARDIOVASCULAR DAMAGE

There is now substantial evidence to indicate that obesity contributes toward cardiovascular outcomes and damage beyond both BP and alternative cardiovascular risk factors. This topic goes beyond the present review. Importantly, the additive effects of obesity on cardiovascular damage in other populations is now well supported by evidence obtained in African communities[5] including the APOGH study.[46-49] However, we have also recently considered the possibility that the deleterious effects of obesity occur in synergy with BP. Is there evidence to support this notion?

TABLE 2: Impact of one standard deviation (SD) increase in waist circumference (WC) or body mass index (BMI) on multivariate adjusted conventional or 24-hour systolic (SBP) or diastolic (DBP) blood pressure in a community sample of African ancestry (African Programme on Genes in Hypertension) (see references[30,42,44-48] for study design and details). Importantly, in this study 44.6% had central (abdominal) obesity and 42.5% had a BMI≥30kg/m^2.

	One SD WC	BP effect*	One SD BMI	BP effect*
All participants				
Conventional SBP (n = 1029)	16.3cm	2.31mm Hg	8.03kg/m^2	2.52mm Hg
Conventional DBP (n = 1029)	16.3cm	2.33mm Hg	8.03kg/m^2	2.26mm Hg
24-hour SBP (n = 657)	15.5cm	2.55mm Hg	7.68kg/m^2	2.61mm Hg
24-hour DBP (n = 657)	15.5cm	1.15mm Hg	7.68kg/m^2	0.84mm Hg
Participants not receiving antihypertensive therapy, 29.6% of whom were hypertensive				
Conventional SBP (n = 787)	15.7cm	2.69mm Hg	7.57kg/m^2	2.76mm Hg
Conventional DBP (n = 787)	15.7cm	2.82mm Hg	7.57kg/m^2	3.07mm Hg
24-hour SBP (n = 533)	15.1cm	2.77mm Hg	7.30kg/m^2	2.80mm Hg
24-hour DBP (n = 533)	15.1cm	1.06mm Hg	7.30kg/m^2	1.05mm Hg

**Calculated from the ß-coefficient (slope) of the relationship between either WC or BMI and BP. All data are adjusted for age, sex, regular smoking, regular alcohol consumption, diabetes mellitus or an HbA1c>6.1%, menopausal status in women and antihypertensive therapy (only in all participants).*

In the APOGH study in a group of African ancestry, we have demonstrated that marked synergy between indices of adiposity (waist circumference and skin-fold thickness) and either conventional, aortic or 24-hour SBP or pulse pressure is independently associated with left ventricular mass and mean wall thickness.[50] These synergistic effects were noted after multivariate adjustments including adjustments for the individual adiposity indices and haemodynamic variables.[50] These synergistic effects translated into a greater impact of SBP or pulse pressure on left ventricular mass and mean wall thickness in participants with an increased as compared to a normal waist circumference.[50] Indeed, for equivalent increases in SBP, as compared to participants with a normal waist circumference, participants with a high waist circumference had a 3 to 4 times greater increase in left ventricular mass after adjustments for confounders.[50] Moreover, left ventricular mass was only increased in participants with both an increased waist circumference and an increased SBP, whilst participants with just an increased BP did not have an increased left ventricular mass.[50]

Although in the APOGH we were able to show distinct synergy between obesity and BP as a determinant of left ventricular mass,[50] this was a cross-sectional study and hence we are unable to draw conclusions regarding cause and effect. In order to provide further proof of principle, we therefore performed a prospective study in an animal model of hypertension.[51] In this regard, a dietary intervention that promoted visceral obesity similarly resulted in a marked further increase in left ventricular weight in hypertensive, but no effect on left ventricular weight in normotensive rats, the consequence being that in the hypertensive rats, cardiac dilatation and pump dysfunction occurred.[51] These data lend further support for the concept that an excess adiposity may enhance the adverse effects of BP on cardiovascular damage. Thus, although obesity may not account for a significant proportion of the variability of BP in groups of African ancestry, the purpose of targeting obesity may be to decrease the adverse impact of BP on the heart. Under these circumstances it is possible that even modest BP reductions achieved by weight loss may translate into major benefits on cardiovascular damage. However, weight loss studies are required to test this hypothesis.

SUMMARY AND CONCLUSIONS

Despite the poor BP control that occurs in communities of African ancestry in Africa,[16] and the possibility that improved drug treatment may not occur because of therapeutic inertia by healthcare providers and competing healthcare resources, there is little evidence for actions being taken at a national level to reduce BP related cardiovascular damage through an improved lifestyle. However, criticism should not be levelled until all the facts are available. The present review argues from recent strong data that there is now indeed significant evidence to suggest that modifying salt intake and promoting weight reduction may reduce cardiovascular risk related to BP in urban, developing communities of African descent and these benefits may occur well beyond that predicted by conventional (brachial artery) BP measurements alone. Indeed, the available evidence suggests that the deleterious effects of salt intake and central obesity on BP related cardiovascular damage may not be easily discernable from conventional BP measurements.

The time is right for initiating large (at least comparable with the DASH study in the sample size), controlled intervention studies designed to reduce salt intake and decrease body weight in groups of African descent in Africa in order to assess the extent to which these interventions may reduce BP related cardiovascular risk. In this regard, these studies will not be of significant value if small study samples are employed, as convincing evidence is required in a study that represents a significant proportion of the black African population. The ideal would be a study that assesses cardiovascular outcomes, but with the present low quality reporting of causes of death in many areas of South Africa, this approach is likely to be of little value.

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REFERENCES

1. Lewington S, Clarke R, Qizilbash N, et al. Prospective Studies Collaboration. Age-specific relevance of usual blood pressure to vascular mortality: a meta-analysis of individual data for one million adults in 61 prospective studies. Lancet 2002;360:1903-1913.
2. Mancia G, De Backer G, Dominiczak A, et al. 2007 Guidelines for the Management of Arterial Hypertension: The Task Force for the Management of Arterial Hypertension of the European Society of Hypertension (ESH) and of the European Society of Cardiology (ESC). J Hypertens 2007;25,1105-1187.
3. Chobanian AV, Bakris GL, Black HR, et al. Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. National Heart, Lung, and Blood Institute; National High Blood Pressure Education Program Coordinating Committee. Seventh report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure. Hypertension 2003;42:1206-1252.
4. Williams B, Poulter NR, Brown MJ, et al. British Hypertension Society. Guidelines for management of hypertension: report of the fourth working party of the British Hypertension Society, 2004-BHS IV. J Human Hypertens 2004;18:139-185.
5. Steyn K, Sliwa K, Hawken S, et al. INTERHEART Investigators in Africa. Risk factors associated with myocardial infarction in Africa: the INTERHEART Africa study. Circulation 2005;112:3554-3561.
6. O'Donnell MJ, Xavier D, Liu L, et al. on behalf of the INTERSTROKE investigators. Risk factors for ischaemic and intracerebral haemorrhagic stroke in 22 countries (the INTERSTROKE study): a case-control study. Lancet 2010;376: 112-123.
7. Mensah GA. Epidemiology of stroke and high blood pressure in Africa. Heart 2008;94:697-705.
8. Connor MD, Modi G, Warlow CP. Differences in the nature of stroke in a multiethnic urban South African population: The Johannesburg hospital stroke register. Stroke 2009;40:355-362.
9. Stewart S, Wilkinson D, Hansen C, et al. Predominance of heart failure in the Heart of Soweto Study cohort: emerging challenges for urban African communities. Circulation 2008;118:2360-2367.
10. Tollman SM, Kahn K, Sartorius B, et al. Implications of mortality transition for primary healthcare in rural South Africa: a population-based surveillance study. Lancet 2008;372:893-901.
11. McInnis NH, Fodor G, Lum-Kwong MM, et al. Antihypertensive medication use and blood pressure control: A community-based cross-sectional survey (ON-BP). Am J Hypertens 2008;21:1210-1215.
12. Cutler JA, Sorlie PD, Wolz M, et al. Trends in hypertension prevalence, awareness, treatment, and control rates in United States adults between 1988 – 1994 and 1999 – 2004, Hypertension 2008;52:818-827.
13. Wolf-Maier K, Cooper RS, Kramer H, et al. Hypertension treatment and control in five European countries, Canada, and the United States. Hypertension. 2004;43:10-17.
14. Lloyd-Jones DM, Evans JC, Levy D. Hypertension in adults across the age spectrum: current outcomes and control in the community. JAMA 2005;294: 466-472.
15. Hertz RP, Unger AN, Cornell JA, et al. Racial disparities in hypertension prevalence, awareness, and management. Arch Intern Med. 2005;165:2098-2104.
16. Steyn K, Gaziano TA, Bradshaw D, et al. South African Demographic and Health Coordinating Team. Hypertension in South African adults: results from the Demographic and Health Survey, 1998. J Hypertens. 2001;19:1717-1725.
17. Intersalt Cooperative Research Group. Intersalt: an international study of electrolyte excretion and blood pressure. Results for 24-hour urinary sodium and potassium excretion. BMJ 1988;297:319-328.
18. Liu Z. Dietary sodium and incidence of hypertension in the Chinese population: a review of nationwide surveys. Am J Hypertens 2009;22:929-933.
19. Hajjar IM, Grim CE, George V, et al. Impact of diet on blood pressure and age-related changes in blood pressure in the US population: analysis of NHANES III. Arch Intern Med 2001;161:589-593.
20. Buyck JF, Blacher J, Kesse-Guyot E, et al. Differential associations of dietary sodium and potassium intake with blood pressure: a focus on pulse pressure. J Hypertens 2009;27:1158-1164.
21. The Trials of Hypertension Prevention Collaborative Research Group. Effects of weight loss and sodium reduction intervention on blood pressure and hypertension incidence in overweight people with high-normal blood pressure. The Trials of Hypertension Prevention, phase II. Arch Intern Med 1997;157: 657-667.
22. Whelton PK, Appel LJ, Espeland MA, et al. for the TONE Collaborative Research Group. Sodium reduction and weight loss in the treatment of hypertension in older persons: a randomised controlled trial of non-pharmacological interventions in the elderly (TONE). JAMA 1998;279:839-846.
23. He J, Whelton PK, Appel LJ, et al. Long-term effects of weight loss and dietary sodium reduction on incidence of hypertension. Hypertension 2000;35:544-549.
24. Sacks FM, Svetkey LP, Vollmer WM, et al. DASH-Sodium Collaborative Research Group. Effects on blood pressure of reduced dietary sodium and the Dietary Approaches to Stop Hypertension (DASH) diet. N Engl J Med 2001;344:3-10.
25. Vollmer WM, Sacks FM, Ard J, et al. DASH-Sodium Trial Collaborative Research Group. Effects of diet and sodium intake on blood pressure: subgroup analysis of the DASH-sodium trial. Ann Intern Med 2001;135:1019-1028.
26. Jurgens G, Graudal JG. Effects of low sodium diet versus high sodium diet on blood pressure, renin, aldosterone, catecholamines, cholesterols, and triglyceride. Cochrane Database of Systematic Reviews 2008; Issue 1. Art. No: CD004022. DOI:10.1002/14651858.CD004022.pub2.
27. Charlton KE, Steyn K, Levitt NS, et al. Ethnic differences in intake and excretion of sodium, potassium, calcium and magnesium in South Africans. Eur J Cardiovasc Prev Rehabil. 2005;12:355-362.
28. Maseko M, Majane HOI, Milne JF, et al. Salt intake in an urban, developing South African community. Cardiovasc J of S Afr 2006;17:186-191.
29. Hoosen S, Seedat YK, Bhigjee AI, et al. A study of urinary sodium and potassium excretion rates among urban and rural Zulus and Indians. J Hypertens 1985;3: 351-358.
30. Bochud M, Staessen JA, Maillard M, et al. Ethnic differences in proximal and distal tubular sodium reabsorption are heritable in black and white populations. J Hypertens 2009;27:606-612.
31. Redelinghuys M, Norton GR, Scott L, et al. Relationship between urinary salt excretion and pulse pressure and central aortic hemodynamics independent of steady state pressure in the general population. Hypertension 2010;56:584-590.

REFERENCES

32. Hoosen S, Seedat YK, Bhigjee AI. A study of urinary and intracellular sodium and potassium, renin, aldosterone and hypertension in Black and Indians in Natal. Cardiovasc Drugs and Therapy 1990;4:363-365.
33. Safar M, Blacher J, Pannier B, et al. Central pulse pressure and mortality in end-stage renal disease. Hypertension. 2002;39:735-738.
34. Roman MJ, Devereux RB, Kizer JR, et al. Central pressure more strongly relates to vascular disease and outcome than does brachial pressure: the Strong Heart Study. Hypertension. 2007;50:197-203.
35. London GM, Blacher J, Pannier B, et al. Arterial wave reflections and survival in end-stage renal failure. Hypertension. 2001;38:434-438.
36. Chirinos JA, Zambrano JP, Chakko S, et al. Aortic pressure augmentation predicts adverse cardiovascular events in patients with established coronary artery disease. Hypertension. 2005;45:980-985.
37. Williams B, Lacy PS, Thom SM, et al. The CAFE Investigators, for the Anglo-Scandinavian Cardiac Outcomes Trial (ASCOT) Investigators. Differential impact of blood pressure-lowering drugs on central aortic pressure and clinical outcomes: Principal results of the Conduit Artery Function Evaluation (CAFE) study. Circulation. 2006;113:1213-1225.
38. Libhaber E, Woodiwiss AJ, Libhaber C, et al. Gender-specific brachial artery blood pressure-independent relationship between pulse wave velocity and left ventricular mass index in a group of African ancestry. J Hypertens 2008;26: 1619-1628.
39. Zhu S, Heymsfield SB, Toyoshima H, et al. Race-ethnicity specific waist circumference cut-offs for identifying cardiovascular disease risk factors. Am J Clin Nutr 2005;81:409-415.
40. Harris MM, Stevens J, Thomas N, et al. Associations of fat distribution and obesity with hypertension in a bi-ethnic population: the ARIC Study. Atherosclerosis Risk in Communities Study. Obes Res 2000;8:516-524.
41. Neter JE, Stam BE, Kok FJ, et al. Influence of weight reduction on blood pressure: a meta-analysis of randomised controlled trials. Hypertension 2003;42:878-884.
42. Juhaeri, Stevens J, Chambless LE, et al. Associations of weight loss and changes in fat distribution with the remission of hypertension in a bi-ethnic cohort: the Atherosclerosis Risk in Communities Study. Prev Med 2003;36:330-339.
43. Puoane T, Steyn K, Bradshaw D, et al. Obesity in South Africa: the South African demographic and health survey. Obes Res. 2002;10:1038-1048.
44. Majane OHI, Norton GR, Maseko MJ, et al. The association of waist circumference with ambulatory blood pressure is independent of alternative adiposity indices. J Hypertens 2007;25:1798-1806.
45. Tiago AD, Samani NJ, Candy GP, et al. Angiotensinogen gene promoter region variant modifies body size-ambulatory blood pressure relations in hypertension. Circulation. 2002;106:1483-1487.
46. Norton GR, Maseko M, Libhaber E, et al. Is pre-hypertension an independent predictor of target organ changes in young-to-middle aged persons of African descent? J Hypertens 2008;26:2279-2287.
47. Libhaber CD, Norton GR, Majane OHI, et al. Contribution of central and general adiposity to abnormal left ventricular diastolic function in a community sample with a high prevalence of obesity. Am J Cardiol 2009;104:1527-1533.
48. Woodiwiss AJ, Libhaber CD, Majane OHI, et al. Obesity promotes left ventricular concentric rather than eccentric geometric remodelling and hypertrophy independent of blood pressure. Am J Hypertens 2008;21:1144-1151.
49. Majane OHI, Woodiwiss AJ, Maseko MJ, et al. Impact of age on the independent association of adiposity with pulse-wave velocity in a population sample of African ancestry. Am J Hypertens 2008;21:936-942.
50. Norton GR, Majane OHI, Libhaber E, et al. The relationship between blood pressure and left ventricular mass index depends on an excess adiposity. J Hypertens. 2009;27:1873-1883.
51. Majane OHI, Vengethasamy L, du Toit EF, et al. Dietary-induced obesity hastens the progression from concentric cardiac hypertrophy to pump dysfunction in spontaneously hypertensive rats. Hypertension 2009;54:1376-1383.