

# Relationship of blood pressure with some cardiovascular disease risk factors in a rural population of Plateau State, North Central Nigeria

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## ABSTRACT

**Background:** Hypertension is associated with certain cardiovascular disease (CVD) risk factors which vary from one place to the other depending on community sophistication. We decided to obtain the situation as it affects this rural Nigerian community to be in an evidence-based position to initiate individual and group prevention strategies. **Design:** Cross-sectional population survey. **Materials and Methods:** We surveyed for CVD risk factors among subjects 15 years and above in this rural community using a questionnaire requesting personal, medical and anthropometric information. One in three of them were randomly assigned to laboratory investigations. **Results:** Of the 840 subjects studied, 25% were males. The population mean age was 45.5 (18.2) standard deviation (SD), with 1.8% smokers and 4.1% using alcohol. Systolic blood pressure (SBP) correlated with age, body mass index (BMI), total cholesterol (TC) and uric acid (UA); while diastolic blood pressure (DBP) correlated with age, BMI, TC, UA and atherogenic index (AI). SBP and DBP improved with exercise but not salt intake. The local seasonings used in cooking had no impact on blood pressure. **Conclusion:** To reduce cardiovascular morbidity in this and probably other rural sub-Saharan African communities, BMI, TC, UA and salt intake in diet should be targeted for reduction. Physical activity should be encouraged. Interestingly, these fall into the sphere of healthy lifestyle which should be encouraged and re-inforced.

**Key words:** Blood pressure, cardiovascular diseases, prevention, risk factors, rural Nigeria

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## INTRODUCTION

High blood pressure is usually associated with certain cardiovascular disease (CVD) risk factors conjointly resulting in increased CVD morbidity.<sup>1</sup> These other CVD risk factors vary in proportion from one environment to another,<sup>2</sup> and depend on the level of sophistication of the community. Population-wide strategies to control CVD usually target for efficiency these risk factors which produce relevant intermediate diseases or the CVDs themselves.<sup>3</sup> Though some of the risk factors are non-modifiable, others are. When identified and worked upon, reduction in their rates especially hypertension which is

chief, gives rise to significant benefits.<sup>4</sup> Most works on these risk factors in our environment are hospital-based. A more lucid picture of the situation usually emerges if population surveys are done; as they would include those affected to degrees not warranting presentation to hospitals. Such studies are few in sub-Saharan Africa, especially in our locality. Despite sharing similar physical and cultural environments, group-level cultural variability and distinct genetic ancestry are known to result in different patterns of CVDs.<sup>5</sup> This reason prompted our desire to see how blood pressure relates to some common CVD risk factors in a rural population in North Central Nigeria. Findings would guide primary and secondary preventive efforts to reduce morbidity and mortality burdens of CVDs on the long run.

## MATERIALS AND METHODS

In 2008, our team re-surveyed two rural communities that had been studied 17 years previously as part of a national non-communicable diseases survey sponsored by the Federal Ministry of Health and Social Services in Nigeria. After getting ethical clearance from the research

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ethics committee of Jos University Teaching Hospital, we met with and got clearance from leaders of the area at various levels. Finally, we mobilized and got to the study sites where all subjects 15 years and above having been mobilized by their leaders at various levels came out for the survey. Subjects were made to sit in the order of their arrival to the study site as we had no control over that to remove any bias. Informed consent was then requested after explanation in English or subjects' mother tongue of what the study entailed. They were then registered and in the same order handed a copy of the study protocol and sample containers (given in the same order to one in every three subjects). They then moved to the next station where the history section was filled by research assistants. Thereafter they went through several other research stations where the various measurements were made. Three blood pressure measurements were obtained in stations 3, 5 and 7 by different trained assistants (Medical Registrars) after subjects had been sitting for about 5 minutes. The activities in intervening stations 4 (height and weight measurement) and 6 (waist and hip circumference measurements) ensured that there was at least 3 minutes in between blood pressure readings. Mercury (ACCOSSON BRAND) sphygmomanometers with appropriate-sized cuffs were used to measure blood pressure in the standard fashion. The mean of the last two measurements was calculated in each case and used for analysis. Weight was measured using a weighing scale on a firm flat surface to the nearest kilogram with subjects in light clothing only; while height was measured using stadiometer in metres to the nearest centimetre. Subjects stood with feet together without any head gear. Body mass index (BMI) was derived from weight (kilograms) and height (metres) using the formula  $w/h^2$ .

Waist circumference was taken using flexible tape at the level of the midpoint between the ribs and the iliac crest from the front in exhalation; and hip circumference measured at the point where the buttocks extended the most when viewed from the side, at the level of the greater trochanters of the femur. Waist and hip circumferences were not taken for pregnant women.

Urinalysis for protein and sugar were determined using appropriate dipstick method on site for those randomized for laboratory investigations. Venous blood was taken from the ante-cubital fossae of such subjects and put in appropriate specimen bottles and taken at the end of each day to the base laboratory for analysis by one of the investigators (IOI). Blood sugar, lipids and lipoprotein concentrations were measured using conventional enzymatic techniques. Serum creatinine was measured by the Jaffe's reaction and uric acid (UA) by Caraway method; while packed cell volume (PCV) was determined using the microhaematocrit centrifuge.

## Statistics

At the end, data were collated and analysed in the University of Jos, Computer Centre using Statistical Package of Social Sciences (SPSS) 17.0. Analysis of variance, student t test and Pearson's Correlation coefficients were used as appropriate to determine degree of difference or association between blood pressure and some CVD risk factors. A  $P$  value  $< 0.05$  was considered statistically significant.

## RESULTS

A total of 840 subjects were studied, 25.1% of whom were males and 74.9% females. They were aged between 16 and 104 years with a mean of  $45.5 \pm 18.2$ . Majority, 78.5% lived most of their last 5 years in the area while 19.3% although resident in the rural area in question at the time of the study lived most of their last 5 years in the urban areas. There were few smokers (1.8%) and not many (4.1%) admitted to using alcohol.

Systolic blood pressure (SBP) was found to be correlated significantly with BMI, total cholesterol (TC), age and serum uric acid ( $P = 0.01$ ); but not with pulse rate, high density lipoprotein cholesterol (HDL-C), blood sugar, PCV, serum creatinine and atherogenic index (AI) defined as quotient of TC and HDL-C. For diastolic blood pressure (DBP), there was significant correlation with BMI, TC, serum uric acid, age and AI; but not serum creatinine, PCV, blood sugar and HDL-C [Table 1].

There was a trend in association between alcohol use and SBP. Those who drank actually had lower mean SBP than those who did not (127.32 vs. 130.51), although the difference did not attain statistical significance ( $P = 0.499$ ). The small numbers did not permit any worthwhile scrutiny. Only 15 subjects (1.8%) smoked a number considered rather small; hence no further analysis was done.

Exercise was assessed by questions on physical activity in regular occupation and rated as mild, moderate or

**Table 1: Degree of correlation by blood pressure and various CVD risk factors**

CVD risk factor	SBP	DBP
Age	0.47**	0.32**
PR	-0.02	0.02
TC	0.24**	0.26**
HDLC	0.10	0.06
SUGAR	0.05	-0.07
PCV	-0.02	0.09
Cr	-0.03	0.06
UA	0.16**	0.14*
AI	0.12	0.17**

Data represent Pearson correlation co-efficient; \* $P < 0.05$ , \*\* $P < 0.01$ ;  
CVD – Cardiovascular disease; SBP – Systolic blood pressure; DBP – Diastolic blood pressure; PR – Pulse rate; TC – Total cholesterol; HDLC – High density lipoprotein cholesterol; PCV – Packed cell volume; Cr – Serum creatinine; UA – Uric acid; AI – Atherogenic index

high. It was judged high if the activity left subject feeling out of breath and sweaty, moderate if it left the subject slightly out of breath and warm but not sweaty and mild if it did not result in running out of breath.<sup>6</sup> In mild and moderate degrees of physical activity, both SBP and DBP were inversely related to a statistically significant level. ( $F = 20.3, P = 0.000$  and  $F = 5.73, P = 0.03$ ), respectively. When subjected to multiple comparison (mild, moderate and severe), whereas subjects admitting moderate and severe degrees of physical activity in their regular occupation had increasingly significant differences in mean SBP compared with mild degree; that between moderate and severe was not significant. The same trend was observed for physical activity and DBP [Table 2]. With regard to leisure time physical activity, there was also an inverse significant relationship; SBP  $F = 12.93, P = 0.000$  and DBP  $F = 3.42, P = 0.03$ . Multiple comparison yielded differences as shown in [Table 3]. High-level leisure activity paradoxically but curiously gave higher mean blood pressure values than mild and moderate levels.

For diet, those who ate food preserved with salt, added extra salt after cooking and those who liked their food well-salted had significantly higher SBP ( $t = 2.704, P = 0.007$ ;  $t = 4.795, P = 0.000$ ;  $t = 2.83, P = 0.005$ , respectively). Regarding DBP, those who ate food preserved with salt, added extra salt after cooking and liked food well-salted had significantly higher values as well ( $t = 2.698, P = 0.007$ ;  $t = 3.953, P = 0.000$ ;  $t = 2.459, P = 0.014$ , respectively). Use of 'Kanwa' rock salt as food seasoning did not affect SBP ( $t = 0.018, P = 0.986$ ) or DBP ( $t = 0.292, P = 0.77$ ). 'Mai-shanu' or animal fat believed to be high in saturated fat and also used as food seasoning did not affect SBP ( $t = 1.311, P = 0.19$ ) or DBP ( $t = 0.098, P = 0.922$ ).

For TC, HDL-C and AI, use of 'Mai-shanu' as food seasoning caused a rise, reduction and rise, respectively. The difference in mean values between those who used the seasoning and those who did not failed to attain statistical

significance ( $t = 0.843, P = 0.4$ ;  $t = 0.765, P = 0.445$ ;  $t = 0.726, P = 0.469$ , respectively).

## DISCUSSION

Blood pressure both SBP and DBP correlated positively to a statistically significant extent with BMI. This is not surprising and has been the experience in several studies. Dudina *et al.* [on behalf of the Systematic Coronary Risk Evaluation (SCORE) investigators] posited that a single unit rise in BMI was associated with a 1.14 mmHg rise in SBP.<sup>7</sup> Doll *et al.* also found that both SBP and DBP rose with BMI<sup>8</sup> across all populations. Individuals who are obese tend to have hyper insulinaemia which in various ways is related to elevated blood pressure. There was also a positive significant correlation with total cholesterol, an experience shared by Akuyan *et al.* working in Zaria, Nigeria.<sup>9</sup> As shown in the SCORE investigation referred to above,<sup>8</sup> a 0.055 mmol/l increase in total cholesterol resulted in a 1.14 mmHg rise in SBP.

Age was found to positively correlate with SBP and DBP significantly. This is a common finding and is attributed to arterial stiffness consequent upon structural alterations in the arterial wall.<sup>10</sup> With ageing, the arteries lose elasticity because of medial degeneration and sclerosis.<sup>11</sup> Excess salt intake either as preference for food preserved with salt or adding extra salt on table after cooking resulted in a significant increase in both SBP and DBP. There has been a general consensus on this relationship; leading to steps in many countries to reduce dietary salt as a population control strategy.<sup>12</sup> Earlier studies in Nigeria also support the observation<sup>13</sup> just as the International study of salt and blood pressure.<sup>14</sup> Excess sodium expands blood volume, thus increasing pre load and cardiac output. BP rises as a result. Other mechanisms include increasing vascular reactivity<sup>15</sup> and contractility.<sup>16</sup> Another CVD risk factor that correlated positively with SBP and DBP here was serum uric acid. Findings in this area have been inconsistent. Whereas some studies see UA as a disease marker<sup>17</sup> being high because of renal changes induced by hypertension and drugs used in treating it; others surmise that it is an independent risk factor for development of hypertension.<sup>18</sup> Our findings here agree with that of Kansui *et al.*<sup>19</sup> in Japan that showed UA correlating significantly with blood pressure whether or not such individuals were on anti hypertensives or uricosuric drugs.

Regarding exercise (whether occupational or leisure), there was a significant association with blood pressure both SBP and DBP. When graded into mild, moderate or severe, there was reduction in BP as the exercise severity rose. However, severe occupational physical activity did not produce any significant reduction in BP over that for moderate degree. This is also the experience of Eicher *et al.*<sup>20</sup> who found that antihypertensive effect of

**Table 2: Multiple comparison of blood pressure and occupational exercise degrees**

Occupational exercise degree	SBP	P	DBP	P
Mild vs. moderate	11.41 (1.96)	***	2.67 (0.94)	**
Mild vs. high	15.53 (3.53)	***	4.59 (1.69)	**
Mod vs. high	4.13 (3.38)	0.22 (NS)	1.92 (1.69)	0.235 (NS)

Data represent difference in means (SE); SBP – Systolic blood pressure; DBP – Diastolic blood pressure; NS – Not significant; \*\*\* $P < 0.01$ ; \*\*\*\* $P < 0.001$

**Table 3: Multiple comparison of blood pressure and leisure exercise degrees**

Leisure exercise degree	SBP	P	DBP	P
Mild vs. moderate	9.49 (1.87)	***	2.32 (0.89)	**
Mild vs. high	4.19 (4.95)	0.39	0.82 (2.35)	0.726
Mod vs. high	-5.30 (4.92)	0.28	-1.49 (2.34)	0.524

Data represent differences in means (SE); \*\* $P < 0.01$ ; \*\*\* $P < 0.001$



exercise occurred in a dose-dependent fashion. Regular physical exercise acting as a basic component of life style modification reduces major cardiovascular risk factors chief of which is hypertension. Exercise is known to reduce blood pressure. It reduces sympathetic activity, reduces angiotensin II levels, improves lipid profile and produces weight loss, all lowering blood pressure.<sup>21</sup> It can also delay progression to hypertension, a response said to be related to decrease in plasma levels of proinflammatory cytokines and norepinephrine, reduction in oxidative stress and diminished activation of the nuclear factor kappa-light-chain-enhancer of activated B cells (nuclear factor KB) system.<sup>22</sup> Whereas severe occupational physical activity did not produce a significant reduction in blood pressure over moderate degree; severe leisure physical activity recorded blood pressure values higher than moderate degree. Why this would be the case is interesting but difficult to fathom; and would need research specifically designed for that to unravel. However, Schultz *et al.*<sup>23</sup> had shown though in rats that excessive exercise especially in untreated hypertensives can have deleterious effects on cardiac function.

We found in this study a reduction in SBP which did not reach statistical significance between those who drank and those who did not. Most of those who admitted to drinking (71%) were mild drinkers and may be at the nadir of the J-shaped relationship between alcohol and blood pressure.<sup>24</sup> Osibogun<sup>13</sup> had also found in his study that those who drank had lower blood pressures. This does not imply encouragement of heavy alcohol intake or advising non drinkers to start; as beyond a certain level, blood pressures rise with increasing amounts of alcohol.

In conclusion, for this sub-Saharan rural community BMI, TC, age, serum uric acid and high salt intake are related to blood pressure and would be favourable points to manipulate for population control. Age is a non-modifiable cardiovascular disease risk factor but the older individuals get, the more careful they should be with their diet to forestall rises in BMI, TC and serum uric acid.

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