Long-term blood pressure trajectories and associations with age and body mass index among urban women in South Africa

Muchiri E Wandai, Samuel OM Manda, Jens Aagaard-Hansen, Shane A Norris

Abstract

Background: Blood pressure (BP) is known to increase inevitably with age. Understanding the different ages at which great gains could be achieved for intervention to prevent and control BP would be of public health importance.

Methods: Data collected between 2003 and 2014 from 1 969 women aged 22 to 89 years were used in this study. Growth curve models were fitted to describe intra- and inter-individual trajectories. For BP tracking, the intra-class correlation coefficient (ICC) was used to measure dependency of observations from the same individual.

Results: Four patterns were identified: a slow decrease in BP with age before 30 years; a period of gradual increase in midlife up to 60 years; a flattening and slightly declining trend; and another increase in BP in advanced age. These phases persisted but at slightly lower levels after adjustment for body mass index. Three groups of increasing trajectories were identified. The respective number (%) in the low, medium and highly elevated BP groups were 1 386 (70.4%), 482 (24.5%) and 101 (5.1%) for systolic BP; and 1 167 (59.3%), 709 (36.0%) and 93 (4.7%) for diastolic BP. The ICC was strong at 0.71 and 0.79 for systolic and diastolic BP, respectively.

Conclusion: These results show that BP preventative and control measures early in life would be beneficial for control later in life, and since increase in body mass index may worsen hypertension, it should be prevented early and independently.

Keywords: blood pressure, hypertension, body mass index, trajectory, intra-class correlation coefficient

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Hypertension is a major risk factor for non-communicable diseases (NCDs), especially stroke and heart attack,[1] and in South Africa, it was estimated to account for 19.0% of cardiovascular disease deaths in 2016.[2] The prevalence of hypertension in women aged 15 years and older was also estimated at 28.5%,[3] and increased steeply with age, with 84.0% of women aged 65 years and older having hypertension.[4] Urban populations have been shown to have a higher prevalence of hypertension compared with their rural counterparts,[3] and women in such dwellings have also been reported to have higher percentages than men.[5,6]

Accelerated global efforts for the prevention and control of NCDs began during the high-level meeting of heads of state and governments at the 66th session of the United Nations General Assembly in September 2011.[7] Following this, the South African Department of Health developed a strategy in line with the declaration in 2013, of which one of the 10 goals and targets was to 'reduce the prevalence of people with raised blood pressure by 20.0% by 2020 (through lifestyle and medication)'.[8]

Changes in both systolic (SBP) and diastolic blood pressure (DBP) with age are known to show an increasing trajectory that mostly starts between 30 and 40 years.[9-11] However, from approximately 50 years, DBP may plateau and thereafter start to decline.[10,11] An individual's BP trajectory can be used as an indicator for age-related vascular stiffening or for the existence of an underlying disease.[12]

Growth curves (trajectories) play an important role in life-course epidemiology,[13] and can be used in identifying groups of individuals at risk of developing high BP using known risk factors.[14] In addition, population subgroups with different BP trajectories can be useful in selecting people who might benefit most from intervention for the prevention of cardiovascular disease (CVD) risk.[15]

Although age is highly correlated with an increasing BP trajectory, some studies have however reported a decrease in SBP and DBP at the population level, especially for communities in the developed countries of western Europe, Australasia and North America,[16-18] and this decrease is usually attributed to lifestyle and pharmacological interventions.[18]

Understanding how individuals' BPs change and how fast these changes occur (intra-individual change) through their life course, and the patterns for people with different attributes (inter-individual differences in the intra-individual change) could be important in determining best methods for prevention at the

appropriate timing. The aim of this study was therefore to find out the association of age with long-term BP observations. The study had three specific objectives: (1) to identify groups of women with similar SBP and DBP between 22 and 89 years of age; (2) to find out how the trajectories are affected by body mass index (BMI); and (3) identify critical ages when intervention measures would be more appropriate in slowing down a steep upward trajectory.

Methods

Ethical approval (M170866) was granted by the Human Research and Ethics Committee of the University of the Witwatersrand, Johannesburg, South Africa.

The study consisted of four measurement time points of data collection on African women dwelling in urban Soweto, Johannesburg, who were care givers of children in the Birth to Twenty Plus cohort study. The majority of the caregivers were the mothers, however a few of the participants included other close relatives such as sisters, aunts and grandmothers.

Data included four waves collected between 2003 and 2014. The four waves provided a sample of 1 969 individuals who had at least one wave of measurements for SBP, DBP, body weight and height, resulting in 4 554 observations. BP measurements were taken in a seated position after 30 minutes of seated rest. The SBP and DBP were measured twice on the right arm using a standard mercury sphygmomanometer and appropriately sized cuff. A final systolic/diastolic BP was calculated by taking the average of the BPs at each time point. Hypertension was defined as systolic/diastolic BP of more than 140/90 mmHg, and BMI was classified according to World Health Organisation as underweight (< 18.5 kg/m^2), normal weight (≥ 18.5– < 25 kg/m^2), overweight (≥ 25– < 30 kg/m^2) and obese (≥ 30 kg/m^2).

Statistical analysis

Multilevel (ML) growth-curve models (a technique to describe and explain an individual's change over time) were used to describe the intra-individual BP trajectories, and the inter-individual differences in the intra-individual changes with age were used as the time metric. Three models were used to describe the patterns of change. The first (model 1) had time effect as the only covariate, model 2 described the changes by adjusting for BMI, and model 3 built on model 2 by allowing effect of BMI to also vary randomly between individuals.

To estimate the mean SBP and DBP trajectories as a function of age, quadratic and cubic non-linear models were used in the analysis, as growth trajectories are known to take a variety of shapes other than linear,[19] mostly characterised by increases or decreases. Age was centred at the minimum age of 22 years to help in the interpretation of the models' intercepts. As BMI is generally known to increase with age, it was included as a time-varying covariate in the second stage of modelling to find out its effect on the BP trajectories. The intra-class correlation coefficient (ICC) was used to measure the degree of dependency among observations within an individual. A group-based trajectory model was used to identify distinct groups for the BPs.

Table 1. Mean and standard deviation for age, SBP, DBP and BMI at the four data-collection time points

Time point	*Number*	*Age (years)*	*SBP (mmHg)*	*DBP (mmHg)*	*BMI (kg/m^2)*
2003	1358	41.1 (7.9)	116.0 (20.1)	76.0 (12.4)	29.9 (6.4)
2005/6	1343	43.4 (8.0)	128.9 (21.6)	80.6 (13.5)	30.7 (7.0)
2007/9	854	45.6 (8.2)	133.5 (21.0)	86.8 (12.9)	30.5 (7.1)
2011/14	999	49.6 (5.7)	134.1 (21.9)	88.7 (12.6)	33.2 (7.2)

SBP, systolic blood pressure; DBP, diastolic blood pressure; BMI, body mass index.

Results

Summary measures for age, BP and BMI at the four time points are shown in Table 1. The mean age at the first occasion (2003) was 41.1 years, and 49.6 years in the fourth occasion. Systolic and diastolic BPs increased from one time point to another, but the increases became smaller with time, and remained almost unchanged, especially for SBP, between the third and fourth occasions. Mean BMI for the four occasions was high, at obesity level, and remained almost constant between the first and third waves, but increased by at least 2.5 kg/m^2 by the fourth occasion. There is an indication of constant variation in BP and BMI across the measurement occasions.

Table 2 shows the percentage of hypertension by BMI category at each data-collection time point. The percentage of subjects who had hypertension in 2003 was 16.7% and by 2014, this had increased to 47.1%. At each time point it was highest for those with an obese BMI, as expected. The greatest percentage increase was between the first and the second occasions (after approximately 2.4 years), where it almost doubled. For those with normal or overweight BMI statuses, the percentage of hypertension almost tripled between the first and fourth occasions. Between the third and fourth time points (approximately 5.3 years), the percentage of those with hypertension among participants in the obese category remained

Table 2. Hypertension percentages by BMI categories at the four data-collection time points

			Blood pressure category		
	BMI		*Non-hypertensive*	*Hyper-tensive*	
Time point	*Category*	n *(%)*	n *(%)*	n *(%)*	*Total* n *(%)*
2003	Underweight	24 (1.8)	22 (91.7)	2 (8.3)	24 (100.0)
	Normal	300 (22.1)	263 (87.7)	37 (12.3)	300 (100.0)
	Overweight	405 (29.8)	349 (86.2)	56 (13.8)	405 (100.0)
	Obese	629 (46.3)	497 (79.0)	132 (21.0)	629 (100.0)
	Total	1358 (100.0)	1131 (83.3)	227 (16.7)	1358 (100.0)
2005/6	Underweight	19 (1.4)	12 (63.2)	7 (36.8)	19 (100.0)
	Normal	269 (20.0)	202 (75.1)	67 (24.9)	269 (100.0)
	Overweight	343 (25.5)	252 (73.5)	91 (26.5)	343 (100.0)
	Obese	712 (53.0)	467 (65.6)	245 (34.4)	712 (100.0)
	Total	1343 (100.0)	933 (69.5)	410 (30.5)	1343 (100.0)
2007/9	Underweight	10 (1.2)	5 (50.0)	5 (50.0)	10 (100.0)
	Normal	188 (22.0)	125 (66.5)	63 (33.5)	188 (100.0)
	Overweight	226 (26.5)	146 (64.6)	80 (35.4)	226 (100.0)
	Obese	430 (50.4)	212 (49.3)	218 (50.7)	430 (100.0)
	Total	854 (100.0)	488 (57.1)	366 (42.9)	854 (100.0)
2011/14	Underweight	7 (0.7)	4 (57.1)	3 (42.9)	7 (100.0)
	Normal	116 (11.6)	77 (66.4)	39 (33.6)	116 (100.0)
	Overweight	217 (21.7)	127 (58.5)	90 (41.5)	217 (100.0)
	Obese	659 (66.0)	320 (48.6)	339 (51.4)	659 (100.0)
	Total	999 (100.0)	528 (52.9)	471 (47.2)	999 (100.0)

BMI, body mass index. Due to approximation to one decimal place, some percentages may fall below or exceed 100% by 0.1.

Table 3. Average BP changes and shifts between categories at the four data-collection time points

Time points	Average years apart	BP category	n	BP category: Non-hypertensive	BP category: Hypertensive	Mean change (SD): SBP	Mean change (SD): DBP
2003 & 2005/6	2.4	Non-hypertensive	824	647 (78.5)	177 (21.5)	11.3 (19.3)	4.1 (12.1)
		Hypertensive	159	53 (33.3)	106 (66.7)		
		Total	983	700 (71.2)	283 (28.8)		
2005/6 & 2007/9	2.0	Non-hypertensive	512	354 (69.1)	158 (30.9)	4.5 (20.4)	6.4 (11.9)
		Hypertensive	237	72 (30.4)	165 (69.6)		
		Total	749	426 (56.9)	323 (43.1)		
2007/9 & 2011/14	5.3	Non-hypertensive	234	162 (69.2)	72 (30.8)	1.7 (19.0)	1.1 (11.2)
		Hypertensive	154	39 (25.3)	115 (74.7)		
		Total	388	201 (51.8)	187 (48.2)		
2003 & 2007/9	4.4	Non-hypertensive	493	303 (61.5)	190 (38.5)	16.4 (20.9)	10.9 (12.6)
		Hypertensive	99	26 (26.3)	73 (73.7)		
		Total	592	329 (55.6)	263 (44.4)		
2005/6 & 2011/14	7.3	Non-hypertensive	460	291 (63.3)	169 (36.7)	8.1 (22.3)	8.1 (12.6)
		Hypertensive	185	49 (26.5)	136 (73.5)		
		Total	645	340 (52.7)	305 (47.3)		
2003 & 2011/14	9.7	Non-hypertensive	549	326 (59.4)	223 (40.6)	19.1 (20.4)	12.6 (12.3)
		Hypertensive	96	15 (15.6)	81 (84.4)		
		Total	645	341 (52.9)	304 (47.1)		

almost unchanged (50.7% in the third occasion and 51.4% in the fourth occasion), even though the percentage of obesity increased by more than 15.6 points (50.4 to 66.0%) between the two occasions.

Table 3 shows the mean change in BP and BMI between two observation periods. Between the first two time points, average SBP and DBP per individual changed by about 11.3 and 4.1 mmHg, respectively but that change decreased substantially between the third and fourth time points to about 1.7 and 1.1 mmHg, respectively. The majority remained in their BP status between observation times but with time (age), the percentage remaining in the hypertension state increased, while the percentage remaining in the normotensive state decreased.

Between the first two occasions when the average age at both times was below 45 years, the percentage of those who were normotensive who transited to the hypertensive state was 21.5%, while the percentage for the counter-transition was 33.3%, or a net recovery of 11.8%. By the fourth occasion when the average age was about 50 years, those initially in the non-hypertensive state who transited to hypertensive state were 40.6%, but the counter-transition (recovery) rate was 15.6%, or a net increase of 25.0% to the hypertensive state.

Fig. 1 and Table 4 show how the progression of BP evolves. A crest (inverted U-shape) marks a negative change (decrease) while a trough (U-shape) marks a positive change (increase). Fig. 1 however shows that the 95% confidence band for ages above 70 years are quite large and therefore the shape in that age group could be spurious. The first crest occurs around 25 years of age and is indicated by negative coefficients (–1.8 for SBP and –1.5 for DBP) for age (Table 4), and the second around 60 years, and is shown by negative coefficients (–0.004 for SBP and –0.003 for DBP) for age cubed. Similarly, the troughs are identifiable around ages 30 and 70 years and marked by the positive coefficients for age squared and age raised to the power of four (Table 4).

Model 1 describes the BP trajectories without accounting for the effect of BMI, while models 2 and 3 show the adjusted

Table 4. Coefficients (95% confidence interval) for the three models predicting SBP and DBP trajectories with standard deviations (SD) for the random effects

BP	Variables	Model 1	Model 2	Model 3
Systolic	Fixed effects			
	Intercept	116.2 (106.7, 125.7)	105.5 (95.7, 115.3)	105.0 (95.1,114.9)
	Age	–1.7 (–3.3, –0.1)	–1.7 (–3.4, –0.1)	–1.8 (–3.4, –0.2)
	Age^2	0.18 (0.08, 0.27)	0.17 (0.08, 0.27)	0.17 (0.08 ,0.27)
	Age^3	–0.004 (–0.006, –0.002)	–0.004 (–0.006, –0.002)	–0.004 (–0.006, –0.002)
	Age^4	0.00003 (0.00001, 0.00005)	0.00003 (0.00001, 0.00005)	0.00003 (0.00001, 0.00005)
	BMI		0.39 (0.29, 0.49)	0.41 (0.30, 0.52)
	Random effects			
	SD (intercept)	14.5 (13.7, 15.2)	14.4 (13.7, 15.1)	24.3 (18.4, 32.2)
	SD (BMI)			0.65 (0.43, 0.96)
	Correlation (BMI, intercept)			–0.82 (–0.91, –0.66)
	SD (residual)	15.5 (15.1, 15.9)	15.4 (15.0, 15.8)	15.2 (14.8, 15.7)
	ICC	0.47 (0.43, 0.50)	0.47 (0.43, 0.50)	0.72 (0.59, 0.82)
Diastolic	Fixed effects			
	Intercept	75.7 (69.8, 81.5)	64.5 (58.5, 70.4)	63.3 (57.2, 69.3)
	Age	–1.4 (–2.4, –0.4)	–1.4 (–2.4, –0.4)	–1.5 (–2.4, –0.5)
	Age^2	0.15 (0.09, 0.21)	0.14 (0.08, 0.20)	0.14 (0.08, 0.20)
	Age^3	–0.004 (–0.005, –0.002)	–0.003 (–0.005, –0.002)	–0.003 (–0.005, –0.002)
	Age^4	0.00003 (0.00001, 0.00004)	0.00002 (0.00001, 0.00004)	0.00002 (0.00001, 0.00004)
	BMI		0.41 (0.35, 0.47)	0.46 (0.39, 0.53)
	Random effects			
	SD (intercept)	9.4 (8.9, 9.9)	9.1 (8.6, 9.5)	17.5 (14.2, 21.7)
	SD (BMI)			0.52 (0.41, 0.67)
	Correlation (BMI, intercept)			–0.87 (–0.92, –0.79)
	SD (residual)	9.4 (9.1, 9.6)	9.3 (9.0, 9.5)	9.0 (8.8, 9.3)
	ICC	0.50 (0.47, 0.53)	0.49 (0.46, 0.52)	0.79 (0.71, 0.85)

SBP, systolic blood pressure; DBP, diastolic blood pressure; ICC, intraclass correlation coefficient.

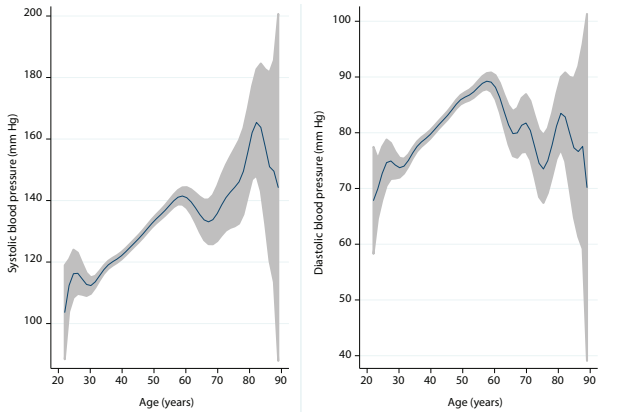

Fig. 1. SBP and DBP trajectories with 95% confidence interval (greyed) by age.

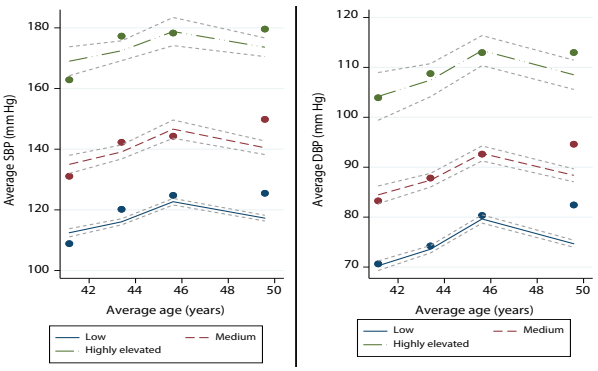

Fig. 2. Observed and BMI-adjusted SBP and DBP trajectories by average age at data-collection time points.

changes. The biggest impact of the BMI adjustment was on the intercepts, shifting each BP's mean trajectory downwards (116.2 to 105.0 mmHg for SBP, and 75.7 to 63.3 mmHg for DBP). Model 2, as shown in Table 4, includes a random effect in the baseline values (intercept) to address the variability in starting point for each individual in the sample. Model 3 in addition to model 2, allowed a random BMI effect (slope) for each individual to explain variability in the slope (change by BMI).

The estimated SBP and DBP for the whole sample at 22 years of age was 105.0 and 63.3 mmHg, respectively, and each individual value varied randomly around these baseline values with 24.3 mmHg standard deviations for SBP and 17.5 mmHg for DBP. Therefore 95% of the estimated individual's BPs at age 22 years lies between $105.0 \pm 1.96 \times 24.3 = (57.4–152.6)$ for SBP and $63.3 \pm 1.96 \times 17.5 = (29.0–96.7)$ for DBP. The estimated increases in BP for every 1 kg/m^2 increase in BMI are 0.41 mmHg for SBP and 0.46 mmHg for DBP for the total sample. But the individual slopes (BMI effect) vary randomly around these values with standard deviations of 0.65 mmHg for SBP and 0.52 mmHg for DBP. Therefore the 95% CI for BMI slopes lie between $0.41 \pm 1.96 \times 0.65 = (–0.86–1.68)$ for SBP and $0.46 \pm 1.96 \times 0.52 = (–0.56–1.48)$ for DBP.

The correlation between the random intercepts and BMI random slopes is strongly inversely proportional (–0.82 for SBP and –0.87 for DBP) implying that higher values of baseline BP at the individual level were associated with a relatively smaller effect of BMI on BP, and vice versa. By including a random BMI effect, the ICC (correlation among observations within an individual) increased from 0.47 (model 2) to 0.72 (model 3) for SBP and from 0.49 to 0.79 for DBP. In addition, the variability in the random intercepts also increased from 14.4 (model 2) to 24.3 (model 3) for SBP, and from 9.1 (model 2) to 17.5 (model 3) for DBP.

There were three distinct groups from the 1 969 individuals (Fig. 2). The low-BP trajectory group comprised the majority of the study participants (70.4% for SBP and 59.3% for DBP), and showed a trajectory that was initially in the normotensive state but gently rose to the pre-hypertensive region. A medium BP trajectory (24.5% for SBP and 36.0% for DBP) had an initial average BP in the pre-hypertensive state that gradually rose to the hypertensive level, while the third group of about 5.0% had highly elevated increasing BPs throughout. Individuals in the medium and highly elevated BP groups were most likely to be aged 40 years and above at baseline, while most of those in the low-BP group were 40 years and below initially.

Discussion

This study examined BP life-course trajectories for a period of approximately 10 years in women from an urban setting in South Africa, and how these trajectories were related to changes in BMI. The trajectories were initially characterised by a short instance of decreasing SBP and DBP with age up to around 30 years (Fig. 1, Table 4). Although our sample for ages below 30 years was small, which could have affected the shape of the trajectories, other studies have shown small downward changes in BP in early adulthood, which could be associated with capacity for vascular repair or adaptations.[20-22] Possible vascular repair could be a reason for our results showing that the percentage of subjects recovering from the hypertensive to the normotensive state was highest (Table 3) in the relatively younger ages.

A second phase of rapid increase in the trajectories began from around 30 up to 60 years. This closely mirrors data from the Framingham Heart Study, which showed that SBP increased continuously between 30 and 84 years, but for DBP the continuous increase was between 30 and 49 years.[23] Increasing SBP and DBP are associated with increased peripheral vascular resistance up to around 50 years, while large arterial stiffness leads to the steeper rise in SBP after 50 years.[24] Studies have shown that younger women have less stiff arteries compared with men of a similar age,[25,26] but increased stiffness occurs after menopause. Attention for control of BP in post-menopausal women would therefore reduce the risk for cerebrovascular and cardiovascular events.[24] Understanding the variation in midlife BP trajectories, and factors associated with this acceleration, may be important in understanding the risk of development and the prevention of CVD, and to implement strategies for lowering BP, as per the National Strategic Plan.[8]

A third phase from around 60 years of age showed a flattening or slightly decreasing trajectory for both BPs (Fig. 1, Table 4). Similar studies have shown a decreasing trend for SBP at ages > 65 years,[27] and for DBP from > 55 years.[28] A decline in BP in old

age has been linked to deteriorating health.[29,30] Decreasing SBP with age has been associated with dementia, depression, polypharmacy (use of a large number of medications) and increased number of co-morbidities.[31,32] A steeper decrease in both SBP and DBP has been associated with a diagnosis of diabetes,[33] and an increase in all-cause and cardiovascular mortality.[34] However, longer-term decreases in BP have also been shown to occur with or without the presence of hypertension, heart failure, atrial fibrillation or stroke,[35] implying that decreasing BP could be due to low cardiac output, a feature of ageing.[36] The deceleration and decline in BP in old age is also associated with use of antihypertensive medication,[22] which we did not account for in this study.

The results of the three group-based trajectories show that the averages for both BPs were either at the pre-hypertensive or hypertensive levels for the medium and highly elevated BP groups. The proportion of women in these two groups of trajectories aged 45 years or above was more than those in the lower BP trajectory group. Persistently elevated BP and hypertension trajectories have been associated with increased incidence of atrial fibrillation, with the associations being stronger in women than men.[37] It has also been associated with a higher risk of subclinical renal damage (SRD) since evidence shows that the higher the levels of SBP in early life, the higher the urinary albumin-to-creatinine ratio and risk of SRD later in life.[38] Some studies have suggested that individuals with high BP, especially SBP in midlife, are at a higher risk of arterial stiffening.[39,40] Evidence has shown that continuously high BP for years is closely correlated with subclinical atherosclerosis,[41] intima–media thickness and left ventricular mass index.[42]

The unadjusted trajectories reflect the added effect of ageing and the influence of other life-course risk factors such as BMI. Generally, the effect of BMI on BP trajectories affected the baseline value (intercept) more than the rate of change (slope). This could be an indication that the effect of BMI on BP is as a result of BMI increases that usually and rapidly take place early in life. Accelerated weight gain and increased BMI in childhood and early adult life increase the risk of elevated BP and the development of hypertension in later life.[43,44]

Surveys in South Africa have shown that the average BMI for women by age 30 years is more than 28.0 kg/m^2,[45-49] which is in the upper range of the overweight level. The majority of the women in this study were at least 30 years old, and the average BMI at the first encounter was 29.9 kg/m^2. An increase in BMI could lead to arterial stiffness, which may cause the development of higher BP levels.[50] Higher SBP levels reflect the stiffening of the arterial walls in areas exposed to increased pressure,[51] while coronary perfusion of the myocardium may be related to DBP.[52]

Allowing the effect of BMI to vary by individual brought about three more important findings. First, the standard deviation of the random intercepts increased, an indication that baseline BPs varied greatly by individual. This was evident from the clear distinction in the group trajectories, which did not interact at any time point. Second, the results showed that the effect of BMI on BP changes was higher in women who initially were in the normotensive status (low BP group), and the effect progressively became less in those initially in the pre-hypertensive (medium BP group) and hypertensive (highly elevated BP) states.

The third issue relates to the correlation between measurements from the same individual, which became more pre-eminent after allowing the effect of BMI to be specific to each individual. A stronger correlation can be helpful in tracking those likely to have persistently high BP. Tracking of a characteristic is the stability of a certain feature over time or its predictability based on earlier measurements.[53,54] Tracking the stability of BPs is of considerable public health interest because those at high risk of developing hypertension could be identified at an early stage,[55] through screening. The influence of change in BMI on BP tracking emphasises the importance of weight control at an early age. Maintenance of normal weight gain in childhood may prevent clustering of hypertension and CVD risk factors in adulthood.[56]

The ideal trajectory for BP is one with minimal fluctuations within the normotensive ranges across all ages. Favourable (less steep trajectory) BP trends are attributed to socially patterned and modifiable BP-related exposures such as lifestyle and diet.[57,58] Few studies from isolated communities such as forager-farmers, have shown minimal age-related BP increases in comparison to Western societies.[59] These communities however have adopted a predominantly vegetarian diet with very low salt content, a physically active lifestyle, and very low or non-existent obesity levels.[59-61] Individuals who undergo an urban migration from one of these isolated communities have been found to adapt quickly to BP profiles of their adopted communities.[60]

The main limitation to the study was that we were unable to account for subjects on antihypertensive medications, which could also have contributed to the decreasing BPs with increasing BMI in higher age groups, and the majority of the women (> 70.0%) having SBP in the lowest of the three trajectory groups. Nonetheless, controlling BMI for this and similar populations should be prioritised as it could be beneficial in many ways and possibly cheaper for BP control than medication alone. Another limitation was that the sample size for women above 60 years at any time point was small and this was the reason for the volatile trajectory in this age range.

Notwithstanding, the major strength was in using repeated BP and anthropometric measurements, which helped in analysing long-term trends in BP changes as they were influenced by age and BMI. This could be useful in guiding clinical practitioners to focus on population segments with particular risk profiles. Another strength was the study result showing that the effect of BMI on elevation of BP was not similar for all individuals, and this could help in clinical practice by designing individualised interventions.

Conclusions

Three subgroups of increasing SBP and DBP trajectories were identified, with the majority of the women in each BP type falling in the lowest group, which on average was initially in the normotensive state. The effect of BMI on the BP trajectory for age was highest in women who initially had relatively lower (mostly in the normotensive state) initial BPs. This BMI effect gradually dropped in tandem with increasing initial BP. The study also showed that steep increasing trajectories could be avoided if preventative interventions are implemented between 30 and 40 years of age from when the BP starts to increase steeply. Follow-up study is required to find out if these trajectories would be similar to findings from a larger and more diverse nationally representative sample.

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