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Title: Association of socioeconomic status change between infancy and adolescence and blood pressure in South African young adults: Birth to Twenty Cohort

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Strengths and limitations of this study

- This present study is a prospective longitudinal cohort which is a rigorous study design with potential to infer causality.
- We employed an objective measure of blood pressure thereby increasing internal validity of the results.
- Only one ethnic group which comprises the majority of the cohort, was selected hence results may not be generalizable to other ethnic groups in South Africa.
- The analytical sample might compromise external validity of the results; however, the study sample was comparable to the excluded group with regards to SES in infancy and adolescence and anthropometry.

ABSTRACT

Objective: Social epidemiology models suggest that socioeconomic status (SES) mobility across the life course affects blood pressure. The aim of this study was to investigate the association between SES change between infancy and adolescence and blood pressure in young adults, and the impact of early growth on this relationship.

Setting: Data for this study was obtained from Birth to Twenty cohort Soweto, Johannesburg in South Africa.

Participants: The study included 838 black participants aged 18 years who had household SES measures in infancy and at adolescence, anthropometry at 0, 2, 4 and 18 years of age and blood pressure at age 18 years.

Methods: We computed SES change using asset-based household SES in infancy and during adolescence as an exposure variable, and blood pressure and hypertension status as outcomes. Multivariate linear and logistic regressions were used to investigate the associations between SES change from infancy to adolescence, and age-height-sex specific blood pressure and hypertension prevalence after adjusting for confounders.

Results: Compared to a persistent low SES, an upward SES change from low to high SES tertile between infancy and adolescence was significantly associated with lower systolic blood pressure (SBP) at age 18 years (β=-4.85; 95% CI -8.22 to -1.48; $p<0.01$; r^2=0.1804) after adjusting for SES in infancy, small-for gestational age (SGA) and weight gain. Associations between SES change and SBP were partly explained by weight gain between birth and age 18 years. There was no association between SES mobility and diastolic blood pressure, mean arterial pressure or hypertension status.

Conclusions: Our study confirms that upward SES change has a protective effect on systolic blood pressure by the time participants reach young adulthood. Socio-economic policies and interventions that address inequality may have the potential to reduce cardiovascular disease burden related to BP in later life.

BACKGROUND

Hypertension is a major public health problem and an independent modifiable risk factor for cardiovascular diseases, which is increasingly becoming a problem in low-to-middle income countries (LMICs).[1] Research has documented that socioeconomic status (SES) influences blood pressure (BP) with low SES being predictive of elevated blood pressure in children [2] and adulthood. [3, 4] In addition, early life factors like birth weight and weight gain may influence the SES change-BP relationship since children from low SES families are likely to be born small and at higher risk of excessive weight gain and high blood pressure.[5, 6]

Most of the evidence on social inequalities in blood pressure comes from longitudinal and cross sectional studies and assumes SES is quite stable over time. However, SES across an individual's lifespan is dynamic in nature especially in societies experiencing socio-political transitions like South Africa [7] , hence the SES-BP relationship might change even within short periods of time in the early life-course.[8]

There has been growing interest in a life course approach to social inequalities in hypertension epidemiology, owing to the evidence that high blood pressure in adulthood evolves from early life; hence the importance of early life environment as a factor influencing the development of hypertension. Life course approaches assume that an individual's health is influenced by dynamic biological and social exposures throughout a life span and that the exposures may not be static over the entire life course.[9] There are three major conceptual models proposed in life course social epidemiology: social origins (critical periods/latent effect) model, accumulation model and the social mobility model.[10, 11]

The social origins hypothesis states that early life is a critical period for biological programming where low SES plays a preeminent role in programming health, with children growing up in a low SES environment having raised BP,[12] independent of their SES in intervening years.[13] We have previously reported finding no relationship between SES in infancy and blood pressure in this cohort of South African adolescents in contrast to the social origins hypothesis.[14] The accumulation model proposes that persisting low SES is detrimental to health. Research on cardiovascular disease risk indicates that low SES in early life has an additive effect on risk factors like blood pressure.[15, 16] The social mobility model suggests that upward social mobility has a protective effect on hypertension risk while a downward SES change is deleterious to cardiovascular disease risk in adulthood. [17, 18] Hogberg and colleagues reported that intergenerational upward social mobility from low SES was associated with 18% reduction in hypertension risk in a Swedish Twin study of 12 030 adults.[19]

The social mobility model has been widely used in life course social epidemiology. However, there is limited literature on social mobility and hypertension, especially among children and adolescents, and most of the studies have concentrated on the intergenerational effect of social mobility on blood pressure using parental and participants' occupation or education to determine life course SES or have used later adulthood BP as an outcome. None of the studies adjusted for initial SES and weight gain, making it difficult to disentangle early life SES environmental effects and weight gain from social mobility effects. [11, 18-20]

Adolescence is a crucial developmental stage characterized by environmental and social changes, and the onset of hormonal and physiological factors that influence physical health outcomes like blood pressure.[21] The studies to date have focused on social mobility in high income countries, where less variability in experiences of SES over the early life-course exist compared to the dynamic SES environments of low and middle income countries.[22]

Post-apartheid South Africa has been undergoing a rapid social and political transition. The volatility of social environment in the post-apartheid era which has seen improvements in SES in previously disadvantaged black populations makes the Birth to Twenty prospective longitudinal cohort a unique and valuable resource to explore the social mobility hypothesis using blood pressure as an outcome which is highly sensitive to changing environments.

This study seeks to test the hypothesis that an upward SES change during childhood and adolescence would be associated with lower blood pressure in early adulthood. Therefore, this study aims to (1) examine the association between SES change and BP and hypertension risk at 18 years of age, and (2) explore whether the SES change-BP relationship is explained by birth outcomes and weight gain between birth and adolescence.

METHODS

Study design and participants

Data for this study came from the Birth to Twenty birth cohort (BT20) - a prospective longitudinal study of children born in Soweto, Johannesburg, South Africa in 1990. Details of recruitment and enrollment into the cohort study are outlined elsewhere.[23] Data for this study were collected at birth, and at ages 2, 4, 16 and 18 years. For the purpose of this study, only black children who had data on blood pressure during late adolescence (18 years), SES data in infancy and during adolescence, birth weight and gestational age, weight gain in infancy, mid-childhood and from mid-childhood to adolescence were included in the analysis (n=838). We only selected black children since they comprise the majority of the BT20 study (Figure 1). Ethics approval was obtained from University of Witwatersrand Human Research Ethics Committee (M130556). Informed consent was obtained from caregivers and participants gave their assent at all data collection time points before the participants turned 18 and their consent once they had turned 18 years of age.

Blood pressure assessment

Blood pressure was measured in triplicate using the Omron M6 (Kyoto, Japan) and an appropriate cuff size with participants in a seated position after an initial five minute rest, and a two minutes rest between each of the three measurements. An average of the second and third measurements was used for the analyses of systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate. The mean SBP and DBP were used to calculate mean arterial pressure (MAP) using the traditional formula: MAP = [(2 x diastolic) + systolic] / 3. [24] Hypertension risk was classified using the age, sex and height specific percentiles from the National High Blood Pressure Education Program Working Group on Hypertension control in Children and Adolescence, with hypertension being defined as $\geq 95^{th}$ percentile and non-hypertension as $<95^{th}$ percentile.[25]

Socioeconomic status change

We used physical asset-based household SES measures tool in infancy and at 16 years of age which utilized a validated standardised questionnaire based on the Demographic and Health survey for developing countries (available at: http://www.dhsprogram.com/). The selection of an asset-based household SES was inspired by the notion that assets are more dynamic and sensitive than other measures, like education and occupation, especially in previously disadvantaged populations undergoing rapid economic and social transition. The physical assets SES measures (for example television, car and refrigerator) were assessed by asking the caregiver or participant whether they had the asset in question (Yes/No). The physical asset scores were computed from all the 'YES' answers and were categorized into tertiles: low (1), medium (2) and high (3) for each of the two time points. Thereafter, nine categories of the social

mobility model were generated according to the literature and were defined as: low-low(11), low-medium(12), low-high(13), medium-low(21), medium-medium(22), medium-high(23), high-low(31), high-medium(32) and high-high(33). [26]

Potential confounders and mediators

Sex, gestational age and birth weight were included from data collected at birth. Weight and height at 2, 4 and 18 years were measured using standard procedures. Relative weight gain was defined as weight gain independent of height during infancy, at mid-childhood (2-4 years) and at adolescence to adulthood (4-18 years) and was computed as residuals obtained by regressing current weight on current height and previous weight and height to deal with the potential multi co-linearity between weight and height.[27] We also used SES in infancy as a covariate since it was a proxy for early life environment so that the SES change variable represents a true measure of social mobility. Because BP in children is age, sex and height specific, we adjusted for these three factors in all the models which included SBP, DBP and MAP. To assess alcohol and tobacco use during adolescence, participants at age 17 years were asked whether they had taken alcohol or smoked tobacco in the last month/ intake (No/Yes).

Statistical analyses

Chi square tests and t-tests were used to describe the study characteristics by sex and hypertension risk for categorical and continuous variables, respectively. Multiple linear regressions were used to assess the association between SES change SBP, DBP and MAP

adjusting for SES in infancy, birth weight and weight gain in infancy, mid-childhood and from mid-childhood to adulthood. We further adjusted the multivariate models for alcohol intake and baseline BP. Additional exploratory models were run for boys and girls separately (results not shown). We also computed the crude and adjusted odds ratios (and 95% confidence intervals) from logistic regressions for the association between SES change and hypertension risk. The statistical analysis were performed in STATA 13 with level of significance set at $p<0.05$ (two-tailed).

RESULTS

Descriptive statistics

Table 1 shows the study population characteristics by sex and hypertension risk (N=838; 48.0% boys). Boys were heavier at birth and at ages 2 and 4 years and taller at 2, 4 and 18 years than girls. Systolic blood pressure was significantly higher by 6 mmHg in boys than girls; on the contrary, girls had significantly higher DBP than boys at age 18 years. There were no sex differences with respect to all SES measures, gestational age, being born small for gestational age, weight at age 18 years and MAP.

Overall, 14.8% of the participants in the study sample were hypertensive (n=124) and 49.1% of these were boys. Table 1 comprises the sStudy characteristics in infancy and adolescence by sex and blood pressure status at age 18 years (n=838). Participants who were hypertensive were significantly 5.5kg heavier at age 18 years compared to their normotensive counterparts. No

major differences in hypertension risk with respect to SES change between infancy and adolescence, birth measures, weight and height in childhood and height at 18 years were observed.

Table 2 Study characteristics in infancy and adolescence by sex and blood pressure status at age 18 years (n=838)

Variables	All	Boys N (%)	Girls N (%)	P value	Non-Hypertensive N (%)	Hypertensive N (%)	P value
Socio economic status (Exposure)							
Household SES change between infancy and adolescence,%							
Low-low(ref)	255(30.4)	133(33.1)	122(28.0)	0.522	211(29.6)	44(17.3)	0.541
Low-medium	97(11.6)	45(11.2)	52(11.9)		81(11.3)	16(12.9)	
Low-high	35(4.2)	17(4.2)	18(4.1)		34(4.8)	1(0.81)	
Medium-low	99(11.8)	41(10.2)	58(13.3)		85(11.9)	14(11.3)	
Medium-Medium	71(8.5)	32(8.0)	39(8.9)		61(8.5)	10(8.1)	
Medium-high	43(5.1)	25(6.2)	18(4.1)		38(5.3)	5(4.0)	
High-low	78(9.3)	39(9.7)	39(8.9)		67(9.4)	11(8.9)	
High-Medium	81(9.7)	37(9.2)	44(10.1)		67(9.4)	14(12.0)	
High-high	79(9.4)	33(8.2)	46(10.6)		70(9.8)	9(7.3)	
Total	838	402(48.0)	436(52.0)		714(85.2)	124(14.8)	
Participant characteristics							
In childhood							
Gestational age, weeks (SD)	838	38(1.7)	38(1.8)	0.3736	38(1.7)	38(1.8)	0.8009
Birth weight ,g (SD)	838	3.1(0.5)	3.0(0.5)	**<0.01**	3.1(0.5)	3.1(0.5)	
Small-for-Gestational age(SGA),%							
No	743	348(86.6)	395(90.6)	0.066	639(89.5)	104(83.9)	0.068
Yes	95	54(13.4)	41(9.4)		75(10.5)	20(16.1)	
Weight at age 2,kg (SD)	838	11.6(1.5)	11.3(1.4)	**0.0177**	11.4(1.4)	11.5(1.5)	0.5112
Weight at age 4,kg(SD)	838	15.6(1.9)	15.2(2.0)	**<0.01**	15.3(2.0)	15.6(2.0)	0.0884
Height at age 2, cm(SD)	838	83.4(3.5)	82.5(3.2)	**<0.001**	83.0(3.3)	82.8(3.5)	0.4768

Height at age 4, cm(SD)	838	99.1(3.9)	98.6(3.8)	**0.0309**	98.8(3.9)	98.8(4.0)	0.854
In Adolescence							
Age, years(SD)	838	17.8(0.4)	17.8(0.4)	0.4521	17.8(0.4)	17.8(0.4)	0.2287
Weight at age 18, kg(SD)	838	59.8(10.2)	59.3(12.4)	0.6017	58.7(10.2)	64.2(15.5)	**<0.001**
Height at age 18,cm(SD)	838	170.6(8.2)	159.6(6.0)	**<0.001**	165.1(8.8)	163.5(9.9)	0.0685
Blood pressure measures at 18 years							
SBP, mmHg(SD)	838	121(10.6)	115(9.5)	**<0.001**	115(8.5)	131(11.2)	**<0.001**
DBP, mmHg(SD)	838	71(8.5)	72(8.5)	**0.0410**	70(6.9)	81(11.0)	**<0.001**
MAP, mmHg(SD)	838	87(8.2)	87(8.4)	0.1525	85(6.3)	99(8.3)	**<0.001**

Values are presented as mean (standard deviation) computed from a t-test for continuous variables or as N (%) for categorical variables obtained from a chi square test and Fischer's exact for N<5.

Determinants of blood pressure and hypertension status

In unadjusted analyses, SBP was significantly associated with change from low-to high SES between infancy and adolescence, sex, age, weight and height at 18 years, and relative weight gain independent of height at 0-2 and 4-18 years (Appendix 1). DBP was significantly associated with sex (higher in males), age and weight at age 18 years and weight gain from age 4 to 18 years. MAP was predicted by weight and height at 18 years, and weight gain from age 4 to 18 years. Hypertension risk was significantly associated with weight at 18 years and weight gain at ages 2-4 and 4 to 18 years.

Association between SES change and blood pressure and hypertension status

Multiple linear regression analyses of SES change characterized by nine subgroups and age-, sex- and height-adjusted SBP, DBP and MAP are presented in Table 2. SES change from low to high tertile was significantly associated with 4.8 mm Hg lower SBP compared to those who maintained a low SES profile between infancy and adolescence, adjusted for SES in infancy, SGA and weight gain between infancy and adulthood. The associations between DBP and MAP, and SES change were statistically insignificant in all the models.

Table 2 Multiple regression models for the relationship between SES change and SBP, DBP and MAP at 18 years of age in Urban Black South Africans.

Blood pressure measure	SBP						DBP						MAP					
	Model 1(n=838)			Model 2(n=838)			Model 1(n=838)			Model 2(n=838)			Model 1(n=838)			Model 2(n=838)		
Covariates	**B**	**95%CI**	**P value**	**β**	**95%CI**	**P value**	**β**	**95%CI**	**P value**	**β**	**95%CI**	**P value**	**β**	**95%CI**	**P value**	**β**	**95%CI**	**P value**
SES change																		
Low-low(ref)																		
Low-medium	-0.74	-3.08 to1.60	0.532	-0.38	-2.63 to 1.86	0.737	-0.52	-2.52 to 1.48	0.608	-0.33	-2.32 to 1.66	0.743	-0.62	-2.56 to1.33	0.532	-0.34	-2.24 to 1.55	0.723
Low-high	-5.10	-8.61 to-1.58	**<0.01**	-4.85	-8.22 to -1.48	**<0.01**	-2.41	-5.42 to 0.60	0.117	-2.27	-5.25 to 0.71	0.136	-2.99	-5.91 to 0.07	**0.045**	-2.81	-5.66 to 0.03	0.053
Medium-low	-0.52	-3.52 to 2.48	0.735	-0.69	-3.57 to 2.19	0.639	1.20	-1.37 to 3.77	0.358	1.09	-1.45 to 3.64	0.398	0.44	-2.05 to 2.94	0.725	0.34	-2.09 to 2.77	0.782
Medium-Medium	-1.77	-5.01 to1.48	0.285	-2.23	-5.35 to 0.89	0.16	-0.13	-2.91 to 2.64	0.925	-0.34	-3.10 to 2.42	0.811	-1.19	-3.88 to 1.51	0.388	-1.44	-4.07 to 1.19	0.282
Medium-high	-0.90	-4.64 to 2.83	0.634	-1.07	-4.66 to 2.51	0.557	-0.02	-3.22 to 3.18	0.99	-0.15	-3.33 to 3.02	0.925	-0.51	-3.61 to 2.60	0.749	-0.60	-3.63 to 2.43	0.696
High-low	-3.65	-7.79 to 0.48	0.083	-3.93	-7.90 to 0.04	0.062	-1.20	-4.74 to 2.34	0.505	-1.39	-4.90 to 2.13	0.439	-1.81	-5.24 to 1.62	0.302	-1.98	-5.33 to 1.37	0.247
High-Medium	-1.38	-5.50 to 2.73	0.51	-2.03	-5.98 to 1.91	0.312	1.36	-2.16 to 4.88	0.448	1.03	-2.45 to 4.53	0.56	0.39	-3.02 to 3.81	0.821	-0.60	-3.39 to 3.27	0.972
High-high	-3.47	-7.84 to 0.90	0.12	-3.41	-7.60 to 0.78	0.34	0.03	-3.71 to 3.77	0.989	0.00	-3.71 to 3.71	1.000	-1.41	-5.04 to 2.23	0.448	-1.35	-4.89 to 2.19	0.456
Sex	-4.03	-5.86 to -2.20	**<0.001**	-4.2	-5.98 to -2.42	**<0.001**	1.94	0.38 to 3.51	**0.015**	1.78	0.21 to 3.37	**0.026**	0.54	-0.98 to 2.06	0.486	0.47	-1.04 to 1.97	0.544
Participant age, years	2.49	0.69 to 4.30	**<0.01**	2.42	0.69 to 4.14	**<0.01**	-1.30	-2.84 to 0.25	0.1	-1.32	-2.85 to 0.21	0.092	-0.08	-1.58 to 1.43	0.921	-0.14	-1.60 to 1.32	0.853
Participant height, cm	0.17	0.06 to 0.28	**<0.01**	0.18	0.08 to 0.29	**<0.01**	0.07	-0.02 to 0.16	0.132	0.07	-0.02 to 0.16	0.131	0.12	0.02 to 0.21	**<0.01**	0.13	0.04 to 0.22	**<0.01**
Household SES in infancy	0.55	-0.46 to 1.55	0.285	0.64	-0.32 to 1.60	0.192	-0.15	-1.01 to 0.70	0.726	-0.10	-0.95 to 0.75	0.818	0.10	-0.73 to 0.93	0.821	0.17	-0.64 to 0.98	0.683
Small-for-Gestational age				0.87	-1.22 to 2.96	0.415				-0.16	-2.01 to 1.69	0.866				0.51	-1.25 to 2.28	0.571
Relative weight gain (0-2years)				1.06	0.38 to 1.74	**<0.01**				0.49	-0.12 to 1.09	0.114				0.65	0.07 to 1.22	**0.028**
Relative weight gain (2-4years)				0.65	0.02 to 1.27	**0.044**				0.29	-0.26 to 0.85	0.300				0.62	0.08 to 1.15	**0.023**
Relative weight gain (4-18years)				2.79	2.12 to 3.47	**<0.001**				1.28	0.68 to 1.87	**<0.001**				1.85	1.28 to 2.42	**<0.001**
Adjusted R^2 value	0.1053			0.1804			0.0064			0.0260			0.0076			0.0605		

[1]Model 1: adjusted for sex, current height, age, and household SES in infancy.

[2]Model 2: Model 1 + growth (SGA, relative weight gain in infancy and mid-childhood)[3]Baseline BP : SBP at 5 for SBP, DBP at 5 for the DBP and MAP at 5 for the MAP models, accordingly

Adjusted logistic regression models (Table 3) show no significant association between SES change from the low-high category and hypertension risk. Relative weight gain at 2-4 and 4-18 years predicted 30% and 66% increased odds of hypertension independent of SES change, SES in infancy, SGA and relative weight gain in infancy.

Table 3 Adjusted odds ratios of being hypertensive at 18 years in urban black South African children (n=838)

	Model 1			Model 2		
Covariates	**OR**	**95%CI**	**P value**	**OR**	**95%CI**	**P Value**
SES change between infancy and adolescence						
Low-low(ref)	1			1		
Low-medium	0.92	0.48 to 1.72	0.787	0.99	0.51 to 1.88	0.968
Low-high	0.14	0.02 to 1.04	0.055	0.14	0.02 to 1.04	0.055
Medium-low	0.61	0.27 to 1.42	0.255	0.57	0.24 to 1.34	0.197
Medium-Medium	0.61	0.25 to 1.52	0.290	0.53	0.21 to 1.36	0.186
Medium-high	0.49	0.16 to 1.50	0.213	0.47	0.15 to 1.48	0.198
High-low	0.51	0.16 to 1.64	0.259	0.46	0.14 to 1.56	0.214
High-Medium	0.65	0.21 to 2.02	0.455	0.51	0.16 to 1.65	0.262
High-high	0.38	0.11 to 1.37	0.140	0.36	0.10 to 1.33	0.125
Household SES in infancy	1.14	0.86 to 1.52	0.359	1.20	0.89 to 1.61	0.237
Small-for-Gestational age(SGA),%				1.33	0.75 to 2.33	0.328
Relative weight gain (0-2years)				1.18	0.96 to 1.45	0.119
Relative weight gain (2-4years)				1.31	1.08 to 1.58	**<0.01**
Relative weight gain (4 to18years)				1.65	1.35 to 2.04	**<0.001**
Pseudo R^2 value	0.0135			0.0630		

Model 1 adjusted for SES at baseline,

Model 2 model 1 +growth (SGA, relative weight gain in infancy and mi-childhood)

Furthermore, additional multivariate analyses of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years are presented in

Appendix 2. In these associations adjusting for alcohol intake and baseline blood pressure did not significantly alter the variance explained by the models.

DISCUSSION

Main findings

We found that an upward mobility in SES was strongly associated with lower SBP at 18 years of age in contrast to remaining in a low SES profile between infancy and adolescence. This study highlights that the association between an upward social mobility and reduced SBP is not fully explained by growth trajectories in relative weight since the association remained significant even after controlling for growth. There was no association between SES change and DBP, MAP and hypertension risk.

Comparison with other studies

Our results are consistent with previous studies which reported that upward social mobility is related to reduced blood pressure. The Pitt county study of African American men aged 25 to 50 years at baseline in 1988 by James et al [18] reported that compared to the stable low SES group between childhood and adulthood, upward SES mobility between childhood and adulthood was associated with 47% reduction in hypertension risk using education, occupation and employment status to compute life course SES. Childhood SES data were collected retrospectively in this study thereby compromising internal validity of the findings. The Swedish study of twins born between 1926 and 1958 reported 16% lower odds in the upwardly mobile SES group compared

to the stable low SES group independent of familial factors.[19] This study used intergenerational SES measures based on parental and the offspring occupation as a measure for life course SES and self-reported hypertension status which is prone to information bias.

Contrary to our findings, a USA study conducted between 2002 and 2003 reported that children who experience an upward mobility trajectory in SES between 14 to 18 years of age had higher SBP compared to those who remained in the low SES profile. However, the results might have been influenced by the under-representation of low SES children in their study. [13] Hallal et al, [28] found no association between socioeconomic trajectories from birth to 11 years of age and SBP and DBP in 15 year old Brazilian adolescents born in 1993 using household income as an indicator of SES.

Possible explanation of the findings

Being small for gestational age had no independent effect on the association between SES change and SBP at 18 years implying that postnatal growth might be more important for programming of social gradients in blood pressure than prenatal growth. Social mobility effects on SBP are not fully explained by growth implying that a dynamic SES environment may influence blood pressure through additional mechanisms. Potential mechanisms through which an upward mobility in SES reduces blood pressure have been evaluated; including bio-behavioral factors and chronic stress. [29] An upward mobility in social class might imply that adolescents are protected from negative health behavior associated with poor households such as poor diet,

lower levels of physical activity, and higher prevalence of tobacco smoking or alcohol intake. However, in this study, adding alcohol use to the models did not alter the associations.

Association between SES change and blood pressure was significant for SBP but not DBP, implying that SBP might be more sensitive to environmental factors compared to DBP. Persistent low SES is a chronic stressor which is related to an increase in sympathetic nervous system reactivity and changes in vasculature which raises SBP.[30] High SBP may be an indicator of vascular dysfunction as a result of progressive stiffening of arterial walls or changes in the vasculature and it has been reported to be a stronger predictor of hypertension and cardiovascular diseases than DBP.[31]

Sex had a distinct independent relationship with SBP, DBP and hypertension risk. However, when the analyses were stratified by sex, the associations remained significant for boys (results not shown) in the SES change-SBP models only, implying that the protective effect of upward social mobility may be apparent in boys and not girls but this needs to be further explored with a larger sample size.

Strengths and limitations

These findings were based on a prospective birth cohort, thereby minimizing recall bias and having the potential to establish a causal relationship between life course SES and blood pressure. Asset based-SES measures are more sensitive measures for SES compared to education

and employment in LMICs since using schooling years for education might not take into account repeated years,[32] employment can be informal and transitory, and income and expenditure are notoriously difficult to assess without extensive validation from secondary sources.[33]

In contrast to previous studies on social mobility and hypertension which used self-reported measures of hypertension, we employed an objective measurement of blood pressure by trained research assistants. Furthermore, the study used both sexes in black urban South African adolescents from a rapidly transitioning urban environment which can be generalized to other African societies in transition. Sex, age and height adjusted blood pressure measures were used in the multivariate models since blood pressure in children and adolescents varies according to age, height and sex.[34] Unlike other studies, we adjusted for covariates to disentangle the effect of early life SES and weight gain on the SES change-BP relationship hence increasing the potential to infer causality.

There are a number of considerations that may pose as limitations. Firstly, we could not include other ethnic groups due to under-representation in the low SES group at the two time points; hence our findings may not be generalizable to the entire South African population. The proportion of hypertensive participants who were in the low-high SES change category was low and this might have resulted in underestimation of the upward social mobility-hypertension risk association resulting in marginal associations. Alcohol intake and tobacco use were self-reported hence we do not rule out reporting bias. There was potential for selection bias in the analytical sample, however, there were no significant differences between the black participants included

and those excluded from the study with regards to the key study variables thereby increasing the potential to generalize these findings.

Conclusions

Our study adds to a limited body of evidence concerning the protective effect of upward social mobility on blood pressure and shows an association between SES change in the early life-course from birth to adolescence and SBP in early adulthood. There is a need for replication of this study to assess its generalizability in other geographical settings and other ethnic groups. These study findings imply that national social and economic policies introduced in the post-apartheid era which seek to improve quality of life among previously disadvantaged black populations have the potential to reduce cardiovascular disease burden attributed to high blood pressure.

Acknowledgements

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Competing interests

None.

Contributors

JK, LSA and SAN conceived and developed the study design, objectives and analytic strategy. JK and PTP conducted the analysis and drafted the manuscript. PLG contributed to the SES data cleaning and the analytic strategy. JMP and SAN were responsible for data acquisition, revising the manuscript for critical intellectual content. All authors contributed to the interpretation of results, manuscript review and approved the final version.

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Ethics approval

This study was approved by the University of Witwatersrand Human Research Ethics Committee.

Provenance and peer review

Not commissioned, externally peer reviewed.

Data sharing statement

No additional data available.

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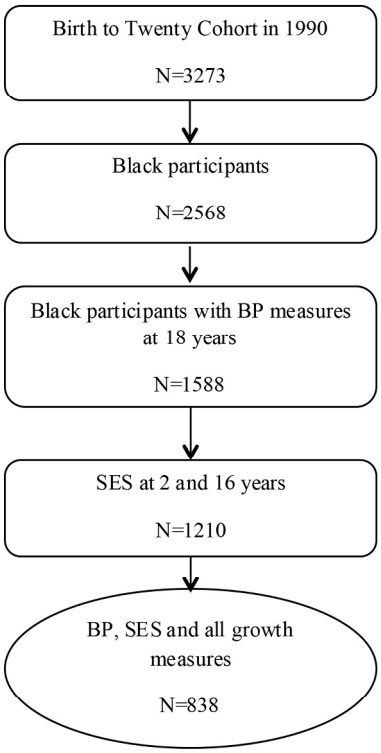

Figure 1 Flow chart of the study population with SES, growth and blood pressure at age 18 years

173x233mm (300 x 300 DPI)

Appendix 1 Bivariate analysis of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years (n=838)

	SBP			DBP			MAP			Hypertension risk		
Exposure variables	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**OR**	**95% CI**	**p value**
SES change												
Low-low (ref)										1		
Low-medium	-0.89	-3.34-to 1.56	0.474	-0.39	-2.38 to 1.60	0.702	-0.54	-2.47 to 1.40	0.586	0.95	0.51 to 1.77	0.865
Low-high	-4.94	-8.64-to -1.23	**<0.01**	-2.24	-5.26 to 0.77	0.144	-2.78	-5.71 to 0.14	0.062	0.14	0.02 to 1.06	0.057
Medium-low	-0.13	-2.56 to 2.30	0.916	1.12	-0.86 to 3.10	0.266	0.61	-1.31 to 2.53	0.534	0.79	0.41 to 1.52	0.478
Medium-Medium	-1.39	-4.15 to 1.36	0.321	-0.34	-2.58 to 1.90	0.765	-1.15	-3.33 to 1.03	0.301	0.79	0.37 to 1.65	0.526
Medium-high	0.50	-2.88 to 3.89	0.771	-0.23	-2.99 to 2.52	0.869	-1.16	-2.83 to 2.52	0.909	0.63	0.24 to 1.69	0.361
High-low	-2.29	-4.95 to 0.36	0.091	-1.69	-3.85 to 0.47	0.125	-1.63	-3.73 to 0.47	0.128	0.79	0.38 to 1.61	0.512
High-Medium	-0.23	-2.85 to 2.39	0.865	1.02	-1.11 to 3.15	0.348	0.63	-1.44 to 2.70	0.548	1.00	0.52 to 1.94	0.995
High-high	-2.31	-4.95 to 0.34	0.087	-0.41	-2.56 to 1.74	0.711	-1.21	-3.30 to 0.88	0.256	0.62	0.29t o 1.33	0.216
Participant characteristics												
Childhood												
Gestational age, weeks	0.01	-0.37 to 0.41	0.943	0.03	-0.28 to 0.35	0.836	0.03	-0.27 to 0.34	0.826	0.97	0.88 to 1.07	0.559
Birth weight, kg	0.40	-0.98 to 1.78	0.568	-0.12	-1.24 to 1.01	0.836	0.00	-1.09 to 1.09	0.999	0.96	0.67 to 1.40	0.861
Small-for-Gestational age(SGA),%												
No(ref)										1		
Yes	2.02	-0.16 to 4.19	0.069	-0.05	-1.83 to 1.74	0.96	0.76	-0.95 to 2.48	0.383	1.56	0.92 to 2.66	0.099
Adolescence												
Age, years	2.81	0.98 to 4.65	**<0.001**	-1.1	-2.61 to 0.40	0.15	0.11	-1.35 to 1.56	0.887	1.41	0.86 to 2.30	0.172
Sex												
Boys(ref)										1		
Girls	-6.10	-7.41 to -4.77	**<0.001**	1.19	0.07 to 2.31	**0.04**	-0.81	-1.90 to 0.27	0.142	1.00	0.69 to 1.45	0.99
Alcohol intake												
No										1		
Yes	-1.05	-2.40 to 0.31	0.131	-0.23	-1.38 to 0.93	0.701	-0.50	-1.61 to 0.61	0.378	0.81	0.57 to 1.16	0.259
Smoking												
No										1		
Yes	-1.29	-2.69 to 0.11	0.071	0.93	2.41 to 0.55	0.217	-1.06	-2.69 to 0.57	0.201	0.72	0.44 to 1.19	0.203
Weight at age 18yrs, kg	0.25	0.19 to 0.30	**<0.001**	0.12	0.07 to 0.17	**<0.001**	0.17	0.13 to 0.22	**<0.001**	1.04	1.02 to 1.06[3]	**<0.001**
Height at age 18yrs,cm	0.35	0.27 to 0.42	**<0.001**	0.00	-0.07 to 0.06	0.888	0.10	0.04 to 0.17	**<0.01**	0.99	0.97 to 1.01	0.236

Relative weight gain (0-2years)	0.87	0.15 to 1.59	**0.02**	0.45	-0.14 to 1.04	0.135	0.56	0.00 to 1.13	0.051	1.13	0.94 to 1.38	0.194
Relative weight gain (2-4years)	0.64	-0.02 to 1.30	0.058	0.12	-0.42 to 0.66	0.652	0.48	-0.04 to 1.00	0.068	1.28	1.07 to 1.55	**<0.01**
Relative weight gain (4-18years)	2.56	1.86 to 3.26	**<0.001**	1.29	0.71 to 1.87	**<0.001**	1.77	1.22 to 2.32	**<0.001**	1.59	1.30 to 1.93	**<0.001**

Appendix 2 Additional multivariate analyses of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years.

	SBP[1](n=655)				DBP[1](n=655)				MAP[1](n=655)				Hypertension risk[2](n=653)			
	β	95% (CI)		p value	β	95% (CI)		p value	β	95% (CI)		p value	Odds Ratio	95% (CI)		P value
SES change																
Low-low(ref)																
Low-medium	-1.35	-4.19	1.49	0.350	-0.60	-3.12	1.92	0.639	-0.86	-3.27	1.55	0.482	0.61	0.28	1.34	0.215
Low-high	-4.78	-8.92	-0.65	**0.024**	-0.34	-4.02	3.33	0.855	-1.77	-5.28	1.73	0.321	0.27	0.06	1.23	0.091
Medium-low	-0.85	-4.38	2.67	0.634	0.98	-2.16	4.11	0.540	0.35	-2.64	3.34	0.820	0.56	0.22	1.45	0.232
Medium-Medium	-3.64	-7.59	0.32	0.071	-1.69	-5.21	1.82	0.344	-2.37	-5.72	0.99	0.166	0.45	0.15	1.35	0.153
Medium-high	1.07	-3.14	5.28	0.619	1.19	-2.56	4.93	0.533	1.15	-2.43	4.72	0.528	0.64	0.20	2.05	0.458
High-low	-4.28	-9.26	0.71	0.093	-1.06	-5.49	3.38	0.640	-2.15	-6.38	2.08	0.319	0.43	0.10	1.77	0.243
High-Medium	-0.99	-5.89	3.90	0.691	2.81	-1.54	7.16	0.204	1.53	-2.62	5.69	0.469	0.46	0.12	1.75	0.254
High-high	-3.54	-8.76	1.68	0.184	0.99	-3.66	5.63	0.676	-0.52	-4.95	3.91	0.818	0.50	0.12	2.13	0.351
Current participant age, yrs	2.45	0.26	4.64	**0.028**	-1.03	-2.98	0.91	0.298	0.14	-1.71	2.00	0.879				
Current participant height, cm	0.08	-0.05	0.21	0.227	-0.02	-0.13	0.10	0.761	0.01	-0.09	0.12	0.808				
Baseline BP at 5 yrs	0.13	0.07	0.19	**0.000**	0.18	0.09	0.27	**0.000**	0.16	0.08	0.24	**0.000**	1.38	0.84	2.29	0.204
Household SES in infancy	0.29	-0.95	1.53	0.650	-0.39	-1.49	0.71	0.489	-0.16	-1.21	0.89	0.770	1.05	0.74	1.48	0.782
Current alcohol intake	-0.71	-2.31	0.90	0.386	0.13	-1.29	1.56	0.854	-0.16	-1.52	1.20	0.822	0.84	0.54	1.32	0.454
Sex	-4.98	-7.17	-2.78	**0.000**	1.26	-0.71	3.22	0.210	-0.82	-2.69	1.05	0.390				
small for gestational age(SGA)	2.00	-0.45	4.45	0.109	0.45	-1.73	2.63	0.687	0.93	-1.15	3.01	0.379	1.87	1.05	3.32	**0.033**
Relative weight gain (0-2years)	1.19	0.35	2.03	**0.005**	0.53	-0.22	1.27	0.166	0.75	0.04	1.46	**0.039**	1.13	0.90	1.43	0.301
Relative weight gain (2-4years)	0.35	-0.38	1.09	0.348	0.32	-0.33	0.97	0.340	0.32	-0.31	0.94	0.319	1.21	0.98	1.48	0.072
Relative weight gain (4-18years)	3.43	2.62	4.24	**0.000**	1.38	0.65	2.10	**0.000**	2.06	1.37	2.75	**0.000**	1.61	1.28	2.03	**0.000**
	R^2=0.2014				R^2=0.0544				R^2=0.0823				Pseudo R^2=0.0631			

[1]Model adjusted for BP measure and SES at baseline, alcohol intake, height and age at 18yrs, sex, growth (SGA, relative weight gain in infancy and mi-childhood)

[2]Model adjusted for BP measure and SES at baseline, alcohol intake at 18yrs, growth (SGA, relative weight gain in infancy and mi-childhood)

JULIANA KAGURA Reporting Checklist for manuscript submission entitled:
"Association of socioeconomic status change between infancy and adolescence and blood pressure in South African young adults: Birth to Twenty Cohort"

STROBE Statement—checklist of items that should be included in reports of observational studies

	Item No	Recommendation
Title and abstract	1	(*a*) Indicate the study's design with a commonly used term in the title or the abstract**(page1 line 2)**
		(*b*) Provide in the abstract an informative and balanced summary of what was done and what was found**(page 3and 4)**
Introduction		
Background/rationale	2	Explain the scientific background and rationale for the investigation being reported **(page 4 to 6)**
Objectives	3	State specific objectives, including any pre-specified hypotheses **(page 6 line 11 to 15)**
Methods		
Study design	4	Present key elements of study design early in the paper **(page 6)**
Setting	5	Describe the setting, locations, and relevant dates, including periods of recruitment, exposure, follow-up, and data collection **(pages 6-7)**
Participants	6	(*a*) *Cohort study*—Give the eligibility criteria, and the sources and methods of selection of participants. Describe methods of follow-up **(pages 6-7)** *Case-control study*—Give the eligibility criteria, and the sources and methods of case ascertainment and control selection. Give the rationale for the choice of cases and controls *Cross-sectional study*—Give the eligibility criteria, and the sources and methods of selection of participants
		(*b*) *Cohort study*—For matched studies, give matching criteria and number of exposed and unexposed *Case-control study*—For matched studies, give matching criteria and the number of controls per case
Variables	7	Clearly define all outcomes, exposures, predictors, potential confounders, and effect modifiers **(pages 8 and 9)**. Give diagnostic criteria **(not applicable),** if applicable
Data sources/ measurement	8*	For each variable of interest, give sources of data and details of methods of assessment (measurement) **(pages 8 and 9)**. Describe comparability of assessment methods if there is more than one group **(not applicable)**
Bias	9	Describe any efforts to address potential sources of bias **(the excluded and analytical sample were compared with regards to key study variables: page 20 line39 to 46)**
Study size	10	Explain how the study size was arrived at **(page 7 figure 1)**
Quantitative variables	11	Explain how quantitative variables were handled in the analyses **(page 8 and 9).** If applicable, describe which groupings were chosen and why**(hypertension status: page 8 for MAP and hypertension risk)**
Statistical methods	12	(*a*) Describe all statistical methods, including those used to control for confounding

		(page 9 -10)
		(*b*) Describe any methods used to examine subgroups and interactions **(not applicable)**
		(*c*) Explain how missing data were addressed **(those with missing data were excluded: page 6 and 7)**
		(*d*) *Cohort study*—If applicable, explain how loss to follow-up was addressed **(page 20 line 39 to 46)** *Case-control study*—If applicable, explain how matching of cases and controls was addressed *Cross-sectional study*—If applicable, describe analytical methods taking account of sampling strategy
		(*e*) Describe any sensitivity analyses
Results		
Participants	13*	(a) Report numbers of individuals at each stage of study—eg numbers potentially eligible, examined for eligibility, confirmed eligible, included in the study, completing follow-up, and analysed **(page 7 figure 1)**
		(b) Give reasons for non-participation at each stage **(reasons were generalised not specific for each stage: page 6 -7)**
		(c) Consider use of a flow diagram **(page 7 figure 1)**
Descriptive data	14*	(a) Give characteristics of study participants (eg demographic, clinical, social) and information on exposures and potential confounders **(table 1, page 11)**
		(b) Indicate number of participants with missing data for each variable of interest **(those with missing data were excluded from the beginning: page 6 and 7)**
		(c) *Cohort study*—Summarise follow-up time (eg, average and total amount)**(page 6)**
Outcome data	15*	*Cohort study*—Report numbers of outcome events or summary measures over time **(table 1 page 11)**
		Case-control study—Report numbers in each exposure category, or summary measures of exposure
		Cross-sectional study—Report numbers of outcome events or summary measures
Main results	16	(*a*) Give unadjusted estimates and, if applicable, confounder-adjusted estimates and their precision (eg, 95% confidence interval). Make clear which confounders were adjusted for and why they were included **(table 2, 3 and appendix 2 (adjusted) and appendix 1(unadjusted))**
		(*b*) Report category boundaries when continuous variables were categorized **(table 1, 2 and 3, appendices)**
		(*c*) If relevant, consider translating estimates of relative risk into absolute risk for a meaningful time period**(not applicable. no relative risk reported rather odds ratios)**
Other analyses	17	Report other analyses done—eg analyses of subgroups and interactions, and sensitivity analyses **(not applicable)**
Discussion		
Key results	18	Summarise key results with reference to study objectives **(page 17)**
Limitations	19	Discuss limitations of the study, taking into account sources of potential bias or imprecision Discuss both direction and magnitude of any potential bias **(Page 20 line 17-22).**
Interpretation	20	Give a cautious overall interpretation of results considering objectives, limitations, multiplicity of analyses, results from similar studies, and other relevant evidence (pages 18 and 19
Generalisability	21	Discuss the generalisability (external validity) of the study results **(page 20 line 10-12)**
Other information		

Funding	22	Give the source of funding and the role of the funders for the present study **(page 22)** and, if applicable, for the original study on which the present article is based **(not applicable)**

*Give information separately for cases and controls in case-control studies and, if applicable, for exposed and unexposed groups in cohort and cross-sectional studies.

Note: An Explanation and Elaboration article discusses each checklist item and gives methodological background and published examples of transparent reporting. The STROBE checklist is best used in conjunction with this article (freely available on the Web sites of PLoS Medicine at http://www.plosmedicine.org/, Annals of Internal Medicine at http://www.annals.org/, and Epidemiology at http://www.epidem.com/). Information on the STROBE Initiative is available at www.strobe-statement.org.

Title: Association of socioeconomic status change between infancy and adolescence and blood pressure in South African young adults: Birth to Twenty Cohort

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Strengths and limitations of this study

- This present study is a prospective longitudinal cohort which is a rigorous study design with potential to infer causality.
- We employed an objective measure of blood pressure thereby increasing internal validity of the results.
- Only one ethnic group which comprises the majority of the cohort, was selected hence results may not be generalizable to other ethnic groups in South Africa.
- The analytical sample might compromise external validity of the results; however, the study sample was comparable to the excluded group with regards to SES in infancy and adolescence and anthropometry.

ABSTRACT

Objective: Social epidemiology models suggest that socioeconomic status (SES) mobility across the life course affects blood pressure. The aim of this study was to investigate the association between SES change between infancy and adolescence and blood pressure in young adults, and the impact of early growth on this relationship.

Setting: Data for this study was obtained from Birth to Twenty cohort Soweto, Johannesburg in South Africa.

Participants: The study included 838 black participants aged 18 years who had household SES measures in infancy and at adolescence, anthropometry at birth, age 2, 4 and 18 years and blood pressure at age 18 years.

Methods: We computed SES change using asset-based household SES in infancy and during adolescence as an exposure variable, and blood pressure and hypertension status as outcomes. Multivariate linear and logistic regressions were used to investigate the associations between SES change from infancy to adolescence, and age-height-sex specific blood pressure and hypertension prevalence after adjusting for confounders.

Results: Compared to a persistent low SES, an upward SES change from low to high SES tertile between infancy and adolescence was significantly associated with lower systolic blood pressure (SBP) at age 18 years (β=-4.85; 95% CI -8.22 to -1.48; $p<0.01$; r^2=0.1804) after adjusting for SES in infancy, small-for gestational age (SGA) and weight gain. Associations between SES change and SBP were partly explained by weight gain between birth and age 18 years. There was no association between SES mobility and diastolic blood pressure or hypertension status.

Conclusions: Our study confirms that upward SES change has a protective effect on systolic blood pressure by the time participants reach young adulthood. Socio-economic policies and interventions that address inequality may have the potential to reduce cardiovascular disease burden related to BP in later life.

BACKGROUND

Hypertension is a major public health problem and an independent modifiable risk factor for cardiovascular diseases, which is increasingly becoming a problem in low-to-middle income countries (LMICs).[1] Research has documented that socioeconomic status (SES) influences blood pressure (BP) with low SES being predictive of elevated blood pressure in children [2] and adulthood. [3, 4] In addition, early life factors like birth weight and weight gain may influence the SES change-BP relationship since children from low SES families are likely to be born small and at higher risk of excessive weight gain and high blood pressure.[5, 6]

Most of the evidence on social inequalities in blood pressure comes from longitudinal and cross sectional studies and assumes SES is quite stable over time. However, SES across an individual's lifespan is dynamic in nature especially in societies experiencing socio-political transitions like South Africa [7] , hence the SES-BP relationship might change even within short periods of time in the early life-course.[8]

There has been growing interest in a life course approach to social inequalities in hypertension epidemiology, owing to the evidence that high blood pressure in adulthood evolves from early life; hence the importance of early life environment as a factor influencing the development of hypertension. Life course approaches assume that an individual's health is influenced by dynamic biological and social exposures throughout a life span and that the exposures may not

be static over the entire life course.[9] There are three major conceptual models proposed in life course social epidemiology: social origins (critical periods/latent effect) model, accumulation model and the social mobility model.[10, 11]

The social origins hypothesis states that early life is a critical period for biological programming where low SES plays a preeminent role in programming health, with children growing up in a low SES environment having raised BP,[12] independent of their SES in intervening years.[13] We have previously reported finding no relationship between SES in infancy and blood pressure in this cohort of South African adolescents in contrast to the social origins hypothesis.[14] The accumulation model proposes that persisting low SES is detrimental to health. Research on cardiovascular disease risk indicates that low SES in early life has an additive effect on risk factors like blood pressure.[15, 16] The social mobility model suggests that upward social mobility has a protective effect on hypertension risk while a downward SES change is deleterious to cardiovascular disease risk in adulthood. [17, 18] Hogberg and colleagues reported that intergenerational upward social mobility from low SES was associated with 18% reduction in hypertension risk in a Swedish Twin study of 12 030 adults.[19]

The social mobility model has been widely used in life course social epidemiology. However, there is limited literature on social mobility and hypertension, especially among children and adolescents, and most of the studies have concentrated on the intergenerational effect of social mobility on blood pressure using parental and participants' occupation or education to determine life course SES or have used later adulthood BP as an outcome. None of the studies adjusted for initial SES and weight gain, making it difficult to disentangle early life SES environmental effects and weight gain from social mobility effects. [11, 18-20]

Adolescence is a crucial developmental stage characterized by environmental and social changes, and the onset of hormonal and physiological factors that influence physical health outcomes like blood pressure.[21] The studies to date have focused on social mobility in high income countries, where less variability in experiences of SES over the early life-course exist compared to the dynamic SES environments of low and middle income countries.[22]

Post-apartheid South Africa has been undergoing a rapid social and political transition. The volatility of social environment in the post-apartheid era which has seen improvements in SES in previously disadvantaged black populations, makes the Birth to Twenty prospective longitudinal cohort a unique and valuable resource to explore the social mobility hypothesis using blood pressure as an outcome which is highly sensitive to changing environments.

This study seeks to test the hypothesis that an upward SES change during childhood and adolescence would be associated with lower blood pressure in early adulthood. Therefore, this study aims to (1) examine the association between SES change and BP and hypertension risk at 18 years of age, and (2) explore whether the SES change-BP relationship is explained by birth outcomes and weight gain between birth and adolescence.

METHODS

Study design and participants

Data for this study came from the Birth to Twenty birth cohort (BT20) - a prospective longitudinal study of children born in Soweto, Johannesburg, South Africa in 1990. Details of recruitment and enrollment into the cohort study are outlined elsewhere.[23] Data for this study were collected at birth, and at 2, 4, 16 and 18 years. For the purpose of this study, only black children who had data on blood pressure during late adolescence (18 years), SES data in infancy

and during adolescence, birth weight and gestational age, weight gain in infancy, mid-childhood and from mid-childhood to adolescence were included in the analysis (n=838). We only selected black children since they comprise the majority of the BT20 study (Figure 1~~Figure 1~~). Ethics approval was obtained from University of Witwatersrand Human Research Ethics Committee (M130556). Informed consent was obtained from caregivers and participants gave their assent at all data collection time points before the participants turned 18 and their consent once they had turned 18 years of age.

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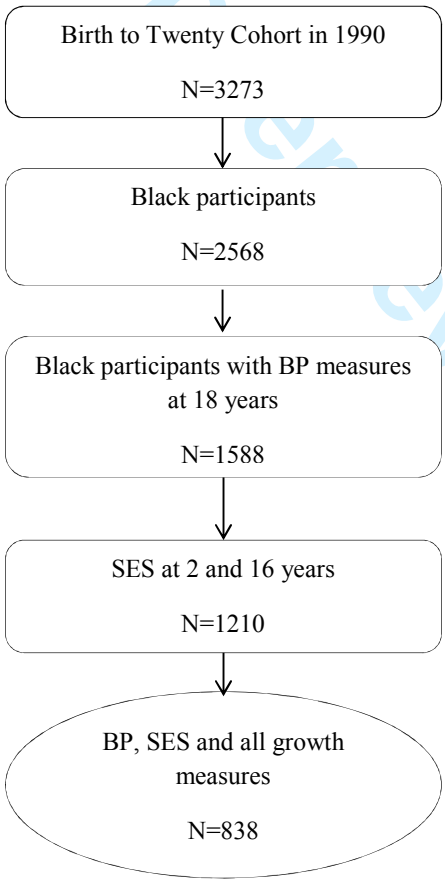

Figure 1 Flow chart of the study population with SES, growth and blood pressure at age 18 years

Blood pressure assessment

Blood pressure was measured in triplicate using the Omron M6 (Kyoto, Japan) and an appropriate cuff size with participants in a seated position after an initial five minute rest, and a two minutes rest between each of the three measurements. An average of the second and third measurements was used for the analyses of systolic blood pressure (SBP), diastolic blood pressure (DBP) and pulse rate. The mean SBP and DBP were used to calculate mean arterial pressure (MAP) using the traditional formula: MAP = [(2 x diastolic) + systolic] / 3. [24] Hypertension risk was classified using the age, sex and height specific percentiles from the National High Blood Pressure Education Program Working Group on Hypertension control in Children and Adolescence, with hypertension being defined as ≥95th percentile and non-hypertension as <95th percentile.[25]

Socioeconomic status change

We used physical asset-based household SES measures tool in infancy and at 16 years of age which utilized a validated standardised questionnaire based on the Demographic and Health survey for developing countries (available at: http://www.dhsprogram.com/). The selection of an asset-based household SES was inspired by the notion that assets are more dynamic and sensitive than other measures, like education and occupation, especially in previously disadvantaged populations undergoing rapid economic and social transition. The physical assets SES measures (for example television, car and refrigerator) were assessed by asking the caregiver or participant whether they had the asset in question (Yes/No). The physical asset scores were computed from all the 'YES' answers and were categorized into tertiles: low (1), medium (2) and high (3) for each of the two time points. Thereafter, nine categories of the social

mobility model were generated according to the literature and were defined as: low-low(11), low-medium(12), low-high(13), medium-low(21), medium-medium(22), medium-high(23), high-low(31), high-medium(32) and high-high(33). [26]

Potential confounders and mediators

Sex, gestational age and birth weight were included from data collected at birth. Weight and height at 2, 4 and 18 years were measured using standard procedures. Relative weight gain was defined as weight gain independent of height during infancy, at mid-childhood (2-4 years) and at adolescence to adulthood (4-18 years) and was computed as residuals obtained by regressing current weight on current height and previous weight and height to deal with the potential multi co-linearity between weight and height.[27] We also used SES in infancy as a covariate since it was a proxy for early life environment so that the SES change variable represents a true measure of social mobility. Because BP in children is age, sex and height specific, we adjusted for these three factors in all the models which included SBP, DBP and MAP. To assess alcohol and tobacco use during adolescence, p~~P~~articipants at age 17 years were asked whether they had taken alcohol or smoked tobacco in the last month/ intake (No/Yes).

Statistical analyses

Chi square tests and t-tests were used to describe the study characteristics by sex and hypertension risk for categorical and continuous variables, respectively. Multiple linear regressions were used to assess the association between SES change ~~and age, sex and height specific~~ SBP, DBP and MAP adjusting for SES in infancy, birth weight and weight gain in infancy, mid-childhood and from mid-childhood to adulthood. We further adjusted the multivariate models for alcohol intake and baseline BP (appendix 2). Additional exploratory

models were run for boys and girls~~,~~ separately (results not shown)~~.~~ We also computed the crude and adjusted odds ratios (and 95% confidence intervals) from logistic regressions for the association between SES change and hypertension risk. The statistical analysis were performed in STATA 13 with level of significance set at $p<0.05$ (two-tailed).

RESULTS

Descriptive statistics

Table 1 shows the study population characteristics by sex and hypertension risk (N=838; 48.0% boys). Boys were heavier at birth and at ages 2 and 4 years and taller at 2, 4 and 18 years than girls. Systolic blood pressure was significantly higher by 6 mmHg in boys than girls; on the contrary, girls had significantly higher DBP than boys at age 18 years. There were no sex differences with respect to all SES measures, gestational age, being born small for gestational age, weight at age 18 years and MAP.

Overall, 14.8% the participants in the study sample were hypertensive (n=124) and 49.1% of these were boys. Table 1 comprises the sStudy characteristics in infancy and adolescence by sex and blood pressure status at age 18 years (n=838)~~tudy characteristics in infancy and adolescence by sex and blood pressure status at age 18 years (n=838)~~. Participants who were hypertensive were significantly 5.5kg heavier at age 18 years compared to their normotensive counterparts. No major differences in hypertension risk with respect to SES change between infancy and adolescence, birth measures, weight and height in childhood and height at 18 years were observed.

Table 1 Study characteristics in infancy and adolescence by sex and blood pressure status at age 18 years (n=838)

Variables	All	Boys N (%)	Girls N (%)	P value	Non-Hypertensive N (%)	Hypertensive N (%)	P value
Socio economic status (Exposure)							
Household SES change between infancy and adolescence,%							
Low-low(ref)	255(30.4)	133(33.1)	122(28.0)	0.522	211(29.6)	44(17.3)	0.541
Low-medium	97(11.6)	45(11.2)	52(11.9)		81(11.3)	16(12.9)	
Low-high	35(4.2)	17(4.2)	18(4.1)		34(4.8)	1(0.81)	
Medium-low	99(11.8)	41(10.2)	58(13.3)		85(11.9)	14(11.3)	
Medium-Medium	71(8.5)	32(8.0)	39(8.9)		61(8.5)	10(8.1)	
Medium-high	43(5.1)	25(6.2)	18(4.1)		38(5.3)	5(4.0)	
High-low	78(9.3)	39(9.7)	39(8.9)		67(9.4)	11(8.9)	
High-Medium	81(9.7)	37(9.2)	44(10.1)		67(9.4)	14(12.0)	
High-high	79(9.4)	33(8.2)	46(10.6)		70(9.8)	9(7.3)	
Total	838	402(48.0)	436(52.0)		714(85.2)	124(14.8)	
Participant characteristics							
In childhood							
Gestational age, weeks (SD)	838	38(1.7)	38(1.8)	0.3736	38(1.7)	38(1.8)	0.8009
Birth weight ,g (SD)	838	3.1(0.5)	3.0(0.5)	**<0.01**	3.1(0.5)	3.1(0.5)	
Small-for-Gestational age(SGA),%							
No	743	348(86.6)	395(90.6)	0.066	639(89.5)	104(83.9)	0.068
Yes	95	54(13.4)	41(9.4)		75(10.5)	20(16.1)	
Weight at age 2,kg (SD)	838	11.6(1.5)	11.3(1.4)	**0.0177**	11.4(1.4)	11.5(1.5)	0.5112
Weight at age 4,kg(SD)	838	15.6(1.9)	15.2(2.0)	**<0.01**	15.3(2.0)	15.6(2.0)	0.0884
Height at age 2, cm(SD)	838	83.4(3.5)	82.5(3.2)	**<0.001**	83.0(3.3)	82.8(3.5)	0.4768
Height at age 4, cm(SD)	838	99.1(3.9)	98.6(3.8)	**0.0309**	98.8(3.9)	98.8(4.0)	0.854
In Adolescence							

Age, years(SD)	838	17.8(0.4)	17.8(0.4)	0.4521	17.8(0.4)	17.8(0.4)	0.2287
Weight at age 18, kg(SD)	838	59.8(10.2)	59.3(12.4)	0.6017	58.7(10.2)	64.2(15.5)	**<0.001**
Height at age 18,cm(SD)	838	170.6(8.2)	159.6(6.0)	**<0.001**	165.1(8.8)	163.5(9.9)	0.0685
Blood pressure measures at 18 years							
SBP, mmHg(SD)	838	121(10.6)	115(9.5)	**<0.001**	115(8.5)	131(11.2)	**<0.001**
DBP, mmHg(SD)	838	71(8.5)	72(8.5)	**0.0410**	70(6.9)	81(11.0)	**<0.001**
MAP, mmHg(SD)	838	87(8.2)	87(8.4)	0.1525	85(6.3)	99(8.3)	**<0.001**

Values are presented as mean (standard deviation) computed from a t-test for continuous variables or as N (%) for categorical variables obtained from a chi square test and Fischer's exact for N<5.

Determinants of blood pressure and hypertension status

In unadjusted analyses, SBP was significantly associated with change from low-to high SES between infancy and adolescence, sex, age, weight and height at 18 years, and relative weight gain independent of height at 0-2 and 4-18 years (~~Table 2~~). DBP was significantly associated with sex (higher in males), age and weight at age 18 years and weight gain from age 4 to 18 years. Mean arterial pressure (MAP) was predicted by weight and height at 18 years, and weight gain from age 4 to 18 years. Hypertension risk was significantly associated with weight at 18 years and weight gain at ages 2-4 and 4 to 18 years.

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Table 2 Bivariate analysis of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years (n=838)

	SBP			DBP			MAP			Hypertension risk		
Exposure variables	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**OR**	**95% CI**	**P value**
SES change	-	-	-	-	-	-	-	-	-	-	-	-
Low-low (ref)	1	-	-	1	-	-	1	-	-	1	-	-
Low-medium	-0.89	-3.34 to 1.56	0.474	-0.39	-2.38 to 1.60	0.702	-0.54	-2.47 to 1.40	0.586	0.95	0.51 to 1.77	0.865
Low-high	-4.94	-8.64 to -1.23	**<0.01**	-2.24	-5.26 to 0.77	0.144	-2.78	-5.71 to 0.14	0.062	0.14	0.02 to 1.06	0.057
Medium-low	-0.13	-2.56 to 2.30	0.916	1.12	-0.86 to 3.10	0.266	0.61	-1.31 to 2.53	0.534	0.79	0.41 to 1.52	0.478
Medium-Medium	-1.39	-4.15 to 1.36	0.321	-0.34	-2.58 to 1.90	0.765	-1.15	-3.33 to 1.03	0.301	0.79	0.37 to 1.65	0.526
Medium-high	0.50	-2.88 to 3.89	0.771	-0.23	-2.99 to 2.52	0.869	-1.16	-2.83 to 2.52	0.909	0.63	0.24 to 1.69	0.361
High-low	-2.29	-4.95 to 0.36	0.091	-1.69	-3.85 to 0.47	0.125	-1.63	-3.73 to 0.47	0.128	0.79	0.38 to 1.61	0.512
High-Medium	-0.23	-2.85 to 2.39	0.865	1.02	-1.11 to 3.15	0.348	0.63	-1.44 to 2.70	0.548	1.00	0.52 to 1.94	0.995
High-high	-2.31	-4.95 to 0.34	0.087	-0.41	-2.56 to 1.74	0.711	-1.21	-3.30 to 0.88	0.256	0.62	0.29t o 1.33	0.216
Participant characteristics	-	-	-	-	-	-	-	-	-	-	-	-
Childhood	-	-	-	-	-	-	-	-	-	-	-	-
Gestational age, weeks	0.01	-0.37 to 0.41	0.943	0.03	-0.28 to 0.35	0.836	0.03	-0.27 to 0.34	0.826	0.97	0.88 to 1.07	0.559
Birth weight, kg	0.40	-0.98 to 1.78	0.568	-0.12	-1.24 to 1.01	0.836	0.00	-1.09 to 1.09	0.999	0.96	0.67 to 1.40	0.861
Small-for-Gestational age(SGA),%	-	-	-	-	-	-	-	-	-	-	-	-
No(ref)	1	-	-	1	-	-	1	-	-	1	-	-
Yes	2.02	-0.16 to 4.19	0.069	-0.05	-1.83 to 1.74	0.96	0.76	-0.95 to 2.48	0.383	1.56	0.92 to 2.66	0.099
Adolescence	-	-	-	-	-	-	-	-	-	-	-	-
Age, years	2.81	0.98 to 4.65	**<0.001**	-1.1	-2.61 to 0.40	0.15	0.11	-1.35 to 1.56	0.887	1.41	0.86 to 2.30	0.172
Sex	-	-	-	-	-	-	-	-	-	-	-	-
Boys(ref)	1	-	-	1	-	-	1	-	-	1	-	-
Girls	-6.10	-7.41 to -4.77	**<0.001**	1.19	0.07 to 2.31	**0.04**	-0.81	-1.90 to 0.27	0.142	1.00	0.69 to 1.45	0.99
Weight at age 18, kg	0.25	0.19 to 0.30	**<0.001**	0.12	0.07 to 0.17	**<0.001**	0.17	0.13 to 0.22	**<0.001**	1.04	1.02 to 1.06[2]	**<0.001**
Height at age 18,cm	0.35	0.27 to 0.42	**<0.001**	0.00	-0.07 to 0.06	0.888	0.10	0.04 to 0.17	**<0.01**	0.99	0.97 to 1.01	0.236
Relative weight gain (0-2years)	0.87	0.15 to 1.59	**0.02**	0.45	-0.14 to 1.04	0.135	0.56	0.00 to 1.13	0.051	1.13	0.94 to 1.38	0.194
Relative weight gain (2-4years)	0.64	-0.02 to 1.30	0.058	0.12	-0.42 to 0.66	0.652	0.48	-0.04 to 1.00	0.068	1.28	1.07 to 1.55	**<0.01**
Relative weight gain (4-18years)	2.56	1.86 to 3.26	**<0.001**	1.29	0.71 to 1.87	**<0.001**	1.77	1.22 to 2.32	**<0.001**	1.59	1.30 to 1.93	**<0.001**

Association between SES change and blood pressure and hypertension status

Multiple linear regression analyses of SES change characterized by nine subgroups and age-, sex- and height-adjusted SBP, DBP and MAP are presented in Table ~~Table 3~~. SES change from low to high tertile was significantly associated with 4.8 mm Hg lower SBP compared to those who maintained a low SES profile between infancy and adolescence, adjusted for SES in infancy, SGA and weight gain between infancy and adulthood. The associations between DBP and MAP, and SES change were statistically insignificant in all the models.

Adjusted logistic regression models (Table 3~~Table 4~~) show no significant association between SES change from the low-high category and hypertension risk. Relative weight gain at 2-4yrs and 4-18 years predicted 30% and 66% increased odds of hypertension independent of SES change, SES in infancy, SGA and relative weight gain in infancy. Adjusting for alcohol intake and baseline BP did not alter the associations (see appendix 2)

Table 2 Multiple regression models for the relationship between SES change and SBP, DBP and MAP at 18 years of age in Urban Black South Africans.

Blood pressure measure	SBP						DBP						MAP					
	Model 1(n=838)			Model 2(n=838)			Model 1(n=838)			Model 2(n=838)			Model 1(n=838)			Model 2(n=838)		
Covariates	B	95%CI	P value	β	95%CI	P value	β	95%CI	P value	β	95%CI	P value	β	95%CI	P value	β	95%CI	P value
SES change																		
Low-low(ref)	ǂ			ǂ			ǂ			ǂ			ǂ			ǂ		
Low-medium	-0.74	-3.08 to1.60	0.532	-0.38	-2.63 to 1.86	0.737	-0.52	-2.52 to 1.48	0.608	-0.33	-2.32 to 1.66	0.743	-0.62	-2.56 to1.33	0.532	-0.34	-2.24 to 1.55	0.723
Low-high	-5.10	-8.61 to-1.58	**<0.01**	-4.85	-8.22 to -1.48	**<0.01**	-2.41	-5.42 to 0.60	0.117	-2.27	-5.25 to 0.71	0.136	-2.99	-5.91 to 0.07	**0.045**	-2.81	-5.66 to 0.03	0.053
Medium-low	-0.52	-3.52 to 2.48	0.735	-0.69	-3.57 to 2.19	0.639	1.20	-1.37 to 3.77	0.358	1.09	-1.45 to 3.64	0.398	0.44	-2.05 to 2.94	0.725	0.34	-2.09 to 2.77	0.782
Medium-Medium	-1.77	-5.01 to1.48	0.285	-2.23	-5.35 to 0.89	0.16	-0.13	-2.91 to 2.64	0.925	-0.34	-3.10 to 2.42	0.811	-1.19	-3.88 to 1.51	0.388	-1.44	-4.07 to 1.19	0.282
Medium-high	-0.90	-4.64 to 2.83	0.634	-1.07	-4.66 to 2.51	0.557	-0.02	-3.22 to 3.18	0.99	-0.15	-3.33 to 3.02	0.925	-0.51	-3.61 to 2.60	0.749	-0.60	-3.63 to 2.43	0.696
High-low	-3.65	-7.79 to 0.48	0.083	-3.93	-7.90 to 0.04	0.062	-1.20	-4.74 to 2.34	0.505	-1.39	-4.90 to 2.13	0.439	-1.81	-5.24 to 1.62	0.302	-1.98	-5.33 to 1.37	0.247
High-Medium	-1.38	-5.50 to 2.73	0.51	-2.03	-5.98 to 1.91	0.312	1.36	-2.16 to 4.88	0.448	1.03	-2.45 to 4.53	0.56	0.39	-3.02 to 3.81	0.821	-0.60	-3.39 to 3.27	0.972
High-high	-3.47	-7.84 to 0.90	0.12	-3.41	-7.60 to 0.78	0.34	0.03	-3.71 to 3.77	0.989	0.00	-3.71 to 3.71	1.000	-1.41	-5.04 to 2.23	0.448	-1.35	-4.89 to 2.19	0.456
Sex	-4.03	-5.86 to -2.20	**<0.001**	-4.2	-5.98 to -2.42	**<0.001**	1.94	0.38 to 3.51	**0.015**	1.78	0.21 to 3.37	**0.026**	0.54	-0.98 to 2.06	0.486	0.47	-1.04 to 1.97	0.544
Participant age, yrs	2.49	0.69 to 4.30	**<0.01**	2.42	0.69 to 4.14	**<0.01**	-1.30	-2.84 to 0.25	0.1	-1.32	-2.85 to 0.21	0.092	-0.08	-1.58 to 1.43	0.921	-0.14	-1.60 to 1.32	0.853
Participant height, cm	0.17	0.06 to 0.28	**<0.01**	0.18	0.08 to 0.29	**<0.01**	0.07	-0.02 to 0.16	0.132	0.07	-0.02 to 0.16	0.131	0.12	0.02 to 0.21	**<0.01**	0.13	0.04 to 0.22	**<0.01**
Household SES in infancy	0.55	-0.46 to 1.55	0.285	0.64	-0.32 to 1.60	0.192	-0.15	-1.01 to 0.70	0.726	-0.10	-0.95 to 0.75	0.818	0.10	-0.73 to 0.93	0.821	0.17	-0.64 to 0.98	0.683
Small-for-Gestational age				0.87	-1.22 to 2.96	0.415				-0.16	-2.01 to 1.69	0.866				0.51	-1.25 to 2.28	0.571
Relative weight gain (0-2years)				1.06	0.38 to 1.74	**<0.01**				0.49	-0.12 to 1.09	0.114				0.65	0.07 to 1.22	**0.028**
Relative weight gain (2-4years)				0.65	0.02 to 1.27	**0.044**				0.29	-0.26 to 0.85	0.300				0.62	0.08 to 1.15	**0.023**
Relative weight gain (4-18years)				2.79	2.12 to 3.47	**<0.001**				1.28	0.68 to 1.87	**<0.001**				1.85	1.28 to 2.42	**<0.001**
Adjusted R^2 value	0.1053			0.1804			0.0064			0.0260			0.0076			0.0605		

[1]Model 1 adjusted for sex, current height, age, and household SES in infancy.

[2]Model 2 Model 1 + growth(SGA, relative weight gain in infancy and mid-childhood)

[3]Baseline BP : SBP at 5 for SBP, DBP at 5 for the DBP and MAP at 5 for the MAP models, accordingly

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Table 3 Adjusted odds ratios of being hypertensive at 18 years in urban black South African children (n=838)

	Model 1			Model 2		
Covariates	**OR**	**95%CI**	**P value**	**OR**	**95%CI**	**P Value**
SES change between infancy and adolescence						
Low-low(ref)	1			1		
Low-medium	0.92	0.48 to 1.72	0.787	0.99	0.51 to 1.88	0.968
Low-high	0.14	0.02 to 1.04	0.055	0.14	0.02 to 1.04	0.055
Medium-low	0.61	0.27 to 1.42	0.255	0.57	0.24 to 1.34	0.197
Medium-Medium	0.61	0.25 to 1.52	0.290	0.53	0.21 to 1.36	0.186
Medium-high	0.49	0.16 to 1.50	0.213	0.47	0.15 to 1.48	0.198
High-low	0.51	0.16 to 1.64	0.259	0.46	0.14 to 1.56	0.214
High-Medium	0.65	0.21 to 2.02	0.455	0.51	0.16 to 1.65	0.262
High-high	0.38	0.11 to 1.37	0.140	0.36	0.10 to 1.33	0.125
Household SES in infancy	1.14	0.86 to 1.52	0.359	1.20	0.89 to 1.61	0.237
Small-for-Gestational age(SGA),%				1.33	0.75 to 2.33	0.328
Relative weight gain (0-2years)				1.18	0.96 to 1.45	0.119
Relative weight gain (2-4years)				1.31	1.08 to 1.58	**<0.01**
Relative weight gain (4 to18years)				1.65	1.35 to 2.04	**<0.001**
Pseudo R^2 value	0.0135			0.0630		

Model 1 adjusted for SES at baseline,

Model 2 model 1 +growth (SGA, relative weight gain in infancy and mi-childhood)

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DISCUSSION

Main findings

We found that an upward mobility in SES was strongly associated with lower SBP at 18 years of age in contrast to remaining in a low SES profile between infancy and adolescence. This study highlights that the association between an upward social mobility and reduced SBP is not fully explained by growth trajectories in relative weight since the association remained significant even after controlling for growth. There was no association between SES change and DBP, MAP and hypertension risk.

Comparison with other studies

Our results are consistent with previous studies which reported that upward social mobility is related to reduced blood pressure. The Pitt county study of African American men aged 25 to 50 years at baseline in 1988 by James et al [18] reported that compared to the stable low SES group between childhood and adulthood, upward SES mobility between childhood and adulthood was associated with 47% reduction in hypertension risk using education, occupation and employment status to compute life course SES. Childhood SES data were collected retrospectively in this study thereby compromising internal validity of the findings. The Swedish study of twins born between 1926 and 1958 reported 16% lower odds in the upwardly mobile SES group compared to the stable low SES group independent of familial factors.[19] This study used intergenerational SES measures based on parental and the offspring occupation as a measure for life course SES and self-reported hypertension status which is prone to information bias.

Contrary to our findings, a USA study conducted between 2002 and 2003 reported that children who experience an upward mobility trajectory in SES between 14 to 18 years of age have higher SBP compared to those who remained in the low SES profile. However, the results might have been influenced by the under-representation of low SES children in their study. [13] Hallal et al, [28] found no association between socioeconomic trajectories from birth to 11 years of age and SBP and DBP in 15 year old Brazilian adolescents born in 1993 using household income as an indicator of SES.

Possible explanation of the findings

Being small for gestational age had no independent effect on the association between SES change and SBP at 18 years implying that postnatal growth might be more important for

programming of social gradients in blood pressure than prenatal growth. Social mobility effects on SBP are not fully explained by growth implying that a dynamic SES environment may influence blood pressure through additional mechanisms. Potential mechanisms through which an upward mobility in SES reduces blood pressure have been evaluated; including bio-behavioral factors and chronic stress. [29] An upward mobility in social class might imply that adolescents are protected from negative health behavior associated with poor households such as poor diet, lower levels of physical activity, and higher prevalence of tobacco smoking or alcohol intake. However, in this study, adding alcohol or tobacco use to the models did not alter the associations.

ssociation between SES change and blood pressure was significant for SBP but not DBP, implying that SBP might be more sensitive to environmental factors compared to DBP. Persistent low SES is a chronic stressor which is related to an increase in sympathetic nervous system reactivity and changes in vasculature which raises SBP.[30] High SBP may be an indicator of vascular dysfunction as a result of progressive stiffening of arterial walls or changes in the vasculature and it has been reported to be a stronger predictor of hypertension and cardiovascular diseases than DBP.[31]

Sex had a distinct independent relationship with SBP, DBP and hypertension risk. However, when the analyses were stratified by sex, the associations remained significant for boys in the SES change-SBP models only, implying that the protective effect of upward social mobility may be apparent in boys – and not girls but this needs to be further explored with a larger sample size.

Strengths and limitations

These findings were based on a prospective birth cohort, thereby minimizing recall bias and having the potential to establish a causal relationship between life course SES and blood pressure. Asset based-SES measures are more sensitive measures for SES compared to education and employment in LMICs since using schooling years for education might not take into account repeated years,[32] employment can be informal and transitory, and income and expenditure are notoriously difficult to assess without extensive validation from secondary sources.[33]

In contrast to previous studies on social mobility and hypertension which used self-reported measures of hypertension, we employed an objective measurement of blood pressure by trained research assistants. Furthermore, the study used both sexes in black urban South African adolescents from a rapidly transitioning urban environment which can be generalized to other African societies in transition. Sex, age and height adjusted blood pressure measures were used in the multivariate models since blood pressure in children and adolescents varies according to age, height and sex.[34] Unlike other studies, we adjusted for covariates to disentangle the effect of early life SES and weight gain on the SES change-BP relationship hence increasing the potential to infer causality.

There are a number of considerations that may pose as limitations. Firstly, we could not include other ethnic groups due to under-representation in the low SES group at the two time points; hence our findings may not be generalizable to the entire South African population. The proportions of the hypertensive participants who were in the low-high SES change category was low and this might have resulted in underestimation of the upward social mobility-hypertension risk association resulting in marginal associations. Alcohol intake and tobacco use were self-

reported hence we do not rule out reporting bias. There was potential for selection bias in the analytical sample, however, there were no significant differences between the black participants included and those excluded from the study with regards to the key study variables thereby increasing the potential to generalize these findings.

Conclusions

Our study adds to a limited body of evidence concerning the protective effect of upward social mobility on blood pressure and shows an association between SES change in the early life-course from birth to adolescence and SBP in early adulthood. There is a need for replication of this study to assess its generalizability in other geographical settings and other ethnic groups. These study findings imply that national social and economic policies introduced in the post-apartheid era which seek to improve quality of life among previously disadvantaged black populations have the potential to reduce cardiovascular disease burden attributed to high blood pressure. ~~The period between infancy and adolescence might be a crucial window of opportunity for interventions targeting hypertension by improving household SES.~~

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Competing interests

None.

Contributors

JK, LSA and SAN conceived and developed the study design, objectives and analytic strategy. JK and PTP conducted the analysis and drafted the manuscript. PLG contributed to the SES data

cleaning and the analytic strategy. JMP and SAN were responsible for data acquisition, revising the manuscript for critical intellectual content. All authors contributed to the interpretation of results, manuscript review and approved the final version.

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Ethics approval

This study was approved by the University of Witwatersrand Human Research Ethics Committee.

Provenance and peer review

Not commissioned, externally peer reviewed.

Data sharing statement

No additional data available.

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Appendix 1 Bivariate analysis of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years (n=838)

	SBP			DBP			MAP			Hypertension risk		
Exposure variables	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**β**	**95% CI**	**p value**	**OR**	**95% CI**	**p value**
SES change												
Low-low (ref)										1		
Low-medium	-0.89	-3.34-to 1.56	0.474	-0.39	-2.38 to 1.60	0.702	-0.54	-2.47 to 1.40	0.586	0.95	0.51 to 1.77	0.865
Low-high	-4.94	-8.64-to -1.23	**<0.01**	-2.24	-5.26 to 0.77	0.144	-2.78	-5.71 to 0.14	0.062	0.14	0.02 to 1.06	0.057
Medium-low	-0.13	-2.56 to 2.30	0.916	1.12	-0.86 to 3.10	0.266	0.61	-1.31 to 2.53	0.534	0.79	0.41 to 1.52	0.478
Medium-Medium	-1.39	-4.15 to 1.36	0.321	-0.34	-2.58 to 1.90	0.765	-1.15	-3.33 to 1.03	0.301	0.79	0.37 to 1.65	0.526
Medium-high	0.50	-2.88 to 3.89	0.771	-0.23	-2.99 to 2.52	0.869	-1.16	-2.83 to 2.52	0.909	0.63	0.24 to 1.69	0.361
High-low	-2.29	-4.95 to 0.36	0.091	-1.69	-3.85 to 0.47	0.125	-1.63	-3.73 to 0.47	0.128	0.79	0.38 to 1.61	0.512
High-Medium	-0.23	-2.85 to 2.39	0.865	1.02	-1.11 to 3.15	0.348	0.63	-1.44 to 2.70	0.548	1.00	0.52 to 1.94	0.995
High-high	-2.31	-4.95 to 0.34	0.087	-0.41	-2.56 to 1.74	0.711	-1.21	-3.30 to 0.88	0.256	0.62	0.29t o 1.33	0.216
Participant characteristics												
Childhood												
Gestational age, weeks	0.01	-0.37 to 0.41	0.943	0.03	-0.28 to 0.35	0.836	0.03	-0.27 to 0.34	0.826	0.97	0.88 to 1.07	0.559
Birth weight, kg	0.40	-0.98 to 1.78	0.568	-0.12	-1.24 to 1.01	0.836	0.00	-1.09 to 1.09	0.999	0.96	0.67 to 1.40	0.861
Small-for-Gestational age(SGA),%												
No(ref)										1		
Yes	2.02	-0.16 to 4.19	0.069	-0.05	-1.83 to 1.74	0.96	0.76	-0.95 to 2.48	0.383	1.56	0.92 to 2.66	0.099
Adolescence												
Age, years	2.81	0.98 to 4.65	**<0.001**	-1.1	-2.61 to 0.40	0.15	0.11	-1.35 to 1.56	0.887	1.41	0.86 to 2.30	0.172
Sex												
Boys(ref)										1		
Girls	-6.10	-7.41 to -4.77	**<0.001**	1.19	0.07 to 2.31	**0.04**	-0.81	-1.90 to 0.27	0.142	1.00	0.69 to 1.45	0.99
Alcohol intake												
No										1		
Yes	-1.05	-2.40 to 0.31	0.131	-0.23	-1.38 to 0.93	0.701	-0.50	-1.61 to 0.61	0.378	0.81	0.57 to 1.16	0.259
Smoking												
No										1		
Yes	-1.29	-2.69 to 0.11	0.071	0.93	2.41 to 0.55	0.217	-1.06	-2.69 to 0.57	0.201	0.72	0.44 to 1.19	0.203

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Weight at age 18yrs, kg	0.25	0.19 to 0.30	**<0.001**	0.12	0.07 to 0.17	**<0.001**	0.17	0.13 to 0.22	**<0.001**	1.04	1.02 to 1.06[3]	**<0.001**
Height at age 18yrs,cm	0.35	0.27 to 0.42	**<0.001**	0.00	-0.07 to 0.06	0.888	0.10	0.04 to 0.17	**<0.01**	0.99	0.97 to 1.01	0.236
Relative weight gain (0-2years)	0.87	0.15 to 1.59	**0.02**	0.45	-0.14 to 1.04	0.135	0.56	0.00 to 1.13	0.051	1.13	0.94 to 1.38	0.194
Relative weight gain (2-4years)	0.64	-0.02 to 1.30	0.058	0.12	-0.42 to 0.66	0.652	0.48	-0.04 to 1.00	0.068	1.28	1.07 to 1.55	**<0.01**
Relative weight gain (4-18years)	2.56	1.86 to 3.26	**<0.001**	1.29	0.71 to 1.87	**<0.001**	1.77	1.22 to 2.32	**<0.001**	1.59	1.30 to 1.93	**<0.001**

Appendix 2 Additional multivariate analyses of factors associated with blood pressure and hypertension risk in urban South African black participants aged 18 years.

	SBP[1](n=655)				DBP[1](n=655)				MAP[1](n=655)				Hypertension risk[2](n=653)			
	β	95% (CI)		p value	β	95% (CI)		p value	β	95% (CI)		p value	Odds Ratio	95% (CI)		P value
SES change																
Low-low(ref)																
Low-medium	-1.35	-4.19	1.49	0.350	-0.60	-3.12	1.92	0.639	-0.86	-3.27	1.55	0.482	0.61	0.28	1.34	0.215
Low-high	-4.78	-8.92	-0.65	**0.024**	-0.34	-4.02	3.33	0.855	-1.77	-5.28	1.73	0.321	0.27	0.06	1.23	0.091
Medium-low	-0.85	-4.38	2.67	0.634	0.98	-2.16	4.11	0.540	0.35	-2.64	3.34	0.820	0.56	0.22	1.45	0.232
Medium-Medium	-3.64	-7.59	0.32	0.071	-1.69	-5.21	1.82	0.344	-2.37	-5.72	0.99	0.166	0.45	0.15	1.35	0.153
Medium-high	1.07	-3.14	5.28	0.619	1.19	-2.56	4.93	0.533	1.15	-2.43	4.72	0.528	0.64	0.20	2.05	0.458
High-low	-4.28	-9.26	0.71	0.093	-1.06	-5.49	3.38	0.640	-2.15	-6.38	2.08	0.319	0.43	0.10	1.77	0.243
High-Medium	-0.99	-5.89	3.90	0.691	2.81	-1.54	7.16	0.204	1.53	-2.62	5.69	0.469	0.46	0.12	1.75	0.254
High-high	-3.54	-8.76	1.68	0.184	0.99	-3.66	5.63	0.676	-0.52	-4.95	3.91	0.818	0.50	0.12	2.13	0.351
Current participant age, yrs	2.45	0.26	4.64	**0.028**	-1.03	-2.98	0.91	0.298	0.14	-1.71	2.00	0.879				
Current participant height, cm	0.08	-0.05	0.21	0.227	-0.02	-0.13	0.10	0.761	0.01	-0.09	0.12	0.808				
Baseline BP at 5 yrs	0.13	0.07	0.19	**0.000**	0.18	0.09	0.27	**0.000**	0.16	0.08	0.24	**0.000**	1.38	0.84	2.29	0.204
Household SES in infancy	0.29	-0.95	1.53	0.650	-0.39	-1.49	0.71	0.489	-0.16	-1.21	0.89	0.770	1.05	0.74	1.48	0.782
Current alcohol intake	-0.71	-2.31	0.90	0.386	0.13	-1.29	1.56	0.854	-0.16	-1.52	1.20	0.822	0.84	0.54	1.32	0.454
Sex	-4.98	-7.17	-2.78	**0.000**	1.26	-0.71	3.22	0.210	-0.82	-2.69	1.05	0.390				
small for gestational age(SGA)	2.00	-0.45	4.45	0.109	0.45	-1.73	2.63	0.687	0.93	-1.15	3.01	0.379	1.87	1.05	3.32	**0.033**
Relative weight gain (0-2years)	1.19	0.35	2.03	**0.005**	0.53	-0.22	1.27	0.166	0.75	0.04	1.46	**0.039**	1.13	0.90	1.43	0.301
Relative weight gain (2-4years)	0.35	-0.38	1.09	0.348	0.32	-0.33	0.97	0.340	0.32	-0.31	0.94	0.319	1.21	0.98	1.48	0.072
Relative weight gain (4-18years)	3.43	2.62	4.24	**0.000**	1.38	0.65	2.10	**0.000**	2.06	1.37	2.75	**0.000**	1.61	1.28	2.03	**0.000**
	R^2=0.2014				R^2=0.0544				R^2=0.0631				Pseudo R^2			

[1]Model adjusted for BP measure and SES at baseline, alcohol intake, height and age at 18yrs, sex, growth (SGA, relative weight gain in infancy and mi-childhood)

[2]Model adjusted for BP measure and SES at baseline, alcohol intake at 18yrs, growth (SGA, relative weight gain in infancy and mi-childhood)