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This article is a corrected version of a now-retracted article published in SAMJ *in November 2025 (*S Afr Med J *2025;115(10):e3195. https://doi.org/10.7196/SAMJ.2025.v115i10.3195). The authors identified a methodological error in the hypertension case definition that materially affected the reported prevalence trend and its interpretation.*

Trends in hypertension prevalence among adults aged ≥40 years in Agincourt, South Africa (2014 - 2022)

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Background. Hypertension is a leading cause of cardiovascular disease. There are limited longitudinal data on temporal trends in hypertension prevalence in rural South Africa (SA).
Objectives. To analyse trends in hypertension prevalence and investigate its associated factors among adults aged ≥40 years in SA.
Methods. A retrospective observational secondary quantitative analysis of a sample from the Health and Aging in Africa: A Longitudinal Study of an INDEPTH Community in South Africa (HAALSI) was conducted. Data for the HAALSI study were collected over three waves during 2014, 2018 and 2021, with hypertension prevalence estimated on wave-specific cross-sections (n=4 936, 3 567 and 3 095, respectively; inverse-probability weighted to baseline), and within-person transitions estimated on a matched three-wave panel of n=2 612 (from 3 707 linked records), drawn from the existing Agincourt Health and Socio-demographic Surveillance System framework. Descriptive statistics were used to summarise sociodemographic data. A bivariate analysis and multivariable generalised estimating equation (GEE) model were applied to determine factors associated with hypertension prevalence, with significance set at p<0.05.
Results. Using the standard HAALSI case definition (measured blood pressure ≥140/90 mmHg or self-reported antihypertensive treatment), the overall prevalence of hypertension did not decline: it was 58.4% (95% confidence interval (CI) 57.0 - 59.8%) in wave 1, 53.9% in wave 2 and 63.1% in wave 3, indicating a stable-to-rising burden consistent with an ageing cohort. Panel hypertension prevalence increased from 60.5% to 61.7% from wave 1 to wave 3. The multivariable GEE analysis identified older age as the strongest predictor of hypertension, with odds increasing across age bands relative to those aged 40 - 49 years (50 - 59: adjusted odds ratio (aOR) 1.26; 60 - 69: aOR 1.92; 70 - 79: aOR 2.41; ≥80: aOR 3.08; all p≤0.008). High body mass index (BMI) was associated with increased odds (overweight aOR 1.37, obese aOR 1.84; both p<0.001), as was diabetes (aOR 1.25; p=0.003). Frequent fruit consumption was protective (aOR 0.86; p=0.025). After adjustment for age, education and employment were no longer significant, and smoking and alcohol were not associated with hypertension.
Conclusion. The study found that hypertension prevalence in Agincourt did not decline between waves 1 and 3: a decline previously observed in our retracted article was an artefact of a blood-pressure-only case definition that excluded treated, controlled individuals, and was corrected here using the standard HAALSI definition. High BMI, diabetes and older age were the key predictors of hypertension. The study highlights the need for targeted public health interventions, including regular hypertension screening, lifestyle modification and better management of associated factors. These efforts will be crucial in reducing hypertension-related morbidity and mortality in rural SA.

Keywords: Agincourt, hypertension, prevalence, risk factors, South Africa

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Hypertension is a chronic medical condition characterised by consistently elevated arterial pressure levels.[1] The condition can be classified into two categories: primary hypertension, which is idiopathic (unknown) and accounts for the majority of cases, and secondary hypertension, which results from an underlying medical condition such as kidney disease, endocrine disorders, or the use of certain medications (e.g. steroids).[2] Primary hypertension accounts for 90 - 95% of cases, and has no identifiable cause.[3] It is associated with various risk factors including age, genetic predisposition, dietary habits, physical inactivity and psychosocial stress.[2-4]

According to the American Heart Association, hypertension is diagnosed when blood pressure readings are consistently >130/80 mmHg.[5] Blood pressure is quantified in millimetres of mercury (mmHg) and recorded as two numbers: systolic blood pressure (SBP; the higher number), which gauges the pressure in the arteries during heart contractions, and diastolic blood pressure (DBP; the lower number), which gauges the pressure in the arteries during heart relaxation.[6] Furthermore, genetic predisposition is significant, with a family history of hypertension increasing the risk of the condition.[7] Globally, hypertension affects ~1.39 billion adults aged 30 - 79 years, with two-thirds of these living in low- and middle-income countries where healthcare systems often struggle to diagnose and manage hypertension effectively.[1,4] In sub-Saharan Africa (SSA), hypertension poses a considerable challenge, with an estimated 74.7 million living with the condition. This is expected to rise to 125.5 million by 2025.[8] Furthermore, it is predicted that hypertension in the SSA population will increase to 216.8 million by 2030.[8] This region is already strained by combatting infectious diseases such as malaria and HIV, and it has limited resources to address the growing burden of chronic conditions such as hypertension.[9]

In South Africa (SA), national surveys have reported a hypertension prevalence ranging from 38.4% in 2012 (the SA National Health and Nutrition Examination Survey) to 48.2% in 2016 (the Demographic and Health Survey).[10,11] Rural hypertension rates exceed those in most urban areas by >10%, with the 2017 Behavioural Risk Factor Surveillance System showing that 40% of participants in rural areas reported having hypertension, but 29.4% in urban areas.[12]

The Health and Aging in Africa: A Longitudinal Study of an INDEPTH Community in SA (HAALSI) provides important insight into hypertension prevalence and associated factors among older adults in rural SA. The HAALSI study uses a population-based survey to examine health, physical and cognitive function, ageing, cardiometabolic disease, associated factors and wellbeing among older men and women.[13]

The present study is relevant in light of the 2018 report on non-communicable diseases (NCDs) in SA, which highlights the growing burden of NCDs, including hypertension, in both urban and rural areas.[14] The 2018 NCD report identified hypertension as a significant contributor to mortality and morbidity, particularly in rural areas where access to healthcare is limited.[15]

The study aligns with the objectives of SA's National Strategic Plan for NCDs 2022 - 2027, which emphasises the need for comprehensive strategies to combat hypertension and other non-communicable diseases.[15] The plan outlines specific goals to improve the diagnosis, treatment and management of NCDs in resource-constrained areas.[15] By assessing hypertension trends and associated factors in a rural context, this study contributes to the broader national efforts to curb the rising burden of NCDs and improve health outcomes in SA.[15]

The study aligns with the Sustainable Development Goals (SDGs), particularly SDG 3 (good health and wellbeing) by contributing to the reduction of NCD mortality, and SDG 10 (reduced inequalities) by addressing health disparities in rural SA.[16] Strengthening public health interventions, community-based strategies and healthcare access will be essential to reduce the hypertension burden, and to ensure equitable management across all populations.

Despite the global and regional burden of hypertension and cardiovascular disease, and particularly that in rural SA, longitudinal studies assessing hypertension trends in rural SA remain limited. This study is one of the few that uses longitudinal, individual-level data from a population-based rural cohort to examine transitions in hypertension prevalence over time. Unlike many cross-sectional studies, it provides insights into how individual hypertension status changes over nearly a decade. This adds practical value for designing sustainable, rural-specific interventions in SA.

Methods

Study design

The study employed a retrospective observational quantitative analysis of secondary data extracted from the HAALSI study.[13]

Study setting

The current study analysed HAALSI data collected through the Agincourt Health and Demographic Surveillance System (AHDSS), a surveillance system operated by the South African Medical Research Council (SAMRC) and Wits Rural Public Health and Health Transitions Research Unit (Agincourt). Agincourt is located in rural Bushbuckridge, Mpumalanga, ~500 km north-east of Johannesburg. The research site comprises roughly 120 000 individuals residing in 21 000 households and 31 villages, spread over 450 km^2. One-third of the permanent population comprises former Mozambican refugees who immigrated to SA during the 1980s, and most of the population is Xitsonga-speaking. The study area consists of one community health centre and six government-run primary healthcare clinics. Referred patients usually use public transport to travel to one of three district hospitals (Mapulaneng, Matikwana and Tintswalo), which are 25 - 55 km away from the site (Fig. 1).[17]

Study population and sampling

The HAALSI study is a population-based survey implemented by the Harvard Center for Population and Development Studies and Agincourt, of the University of the Witwatersrand. HAALSI aims to examine and characterise a population of older men and women in rural SA concerning health, physical and cognitive function, ageing and wellbeing, in harmonisation with other health and retirement studies.

The study drew on individuals aged ≥40 years in the HAALSI cohort, surveyed over three waves (wave 1: 2014 - 2015; wave 2: 2018 - 2019; wave 3: 2021 - 2022). HAALSI employed random sampling from the AHDSS, and participants were linked across waves using a unique identifier assigned by the HAALSI platform.[13] Of the 5 059 individuals recruited at wave 1, 4 936 had a valid wave-1 hypertension status. The analysis used two distinct samples. Prevalence at each wave was estimated on the wave-specific cross-section; every participant with a valid hypertension measurement at that wave was included, irrespective of the inclusion in other

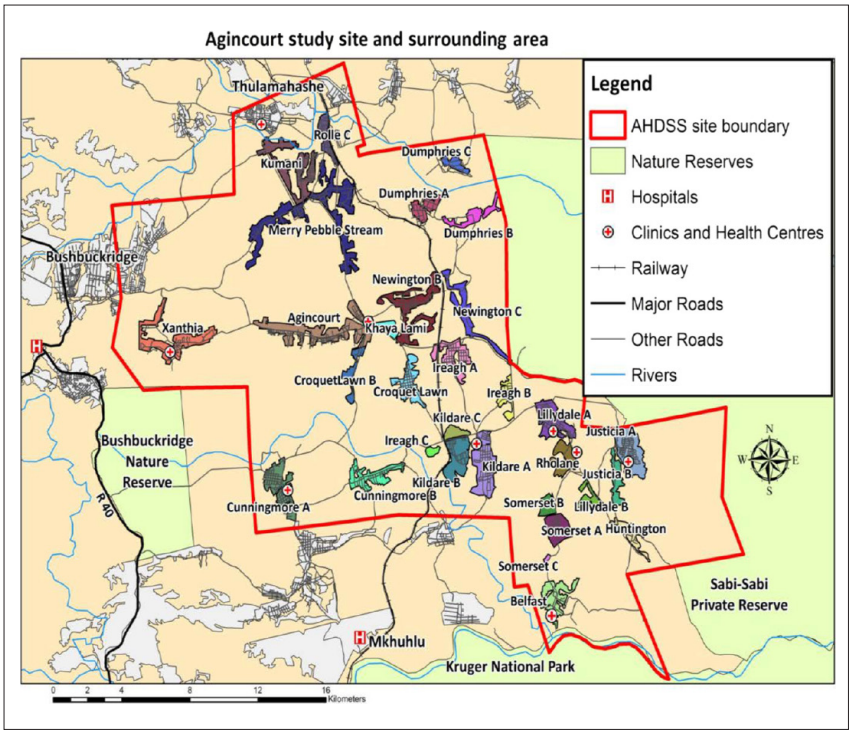


Fig. 1. Agincourt Health and Demographic Surveillance System (AHDSS) map.[17]

waves (n=4 936, 3 567 and 3 095 at waves 1, 2 and 3, respectively). These cross-sections shrink with attrition, and estimates were inverse-probability weighted so that each wave remained representative of the wave-1 population. Within-person transitions and the GEE model were estimated on the matched three-wave panel; 2 612 participants (of 3 707 with records linked across all waves) had a valid hypertension status at every wave. Since the transitions can only be observed among individuals seen at all three waves, prevalence within this panel is additionally reported as a sensitivity analysis to achieve a 95% statistical power for detecting significant differences, using a one-sided $p<0.05$ significance test. This calculation was conducted on G-Power statistical software incorporating an effect size of 0.1 and a precision level of 5%.

Measurements

Data for this study were extracted from the HAALSI website for wave 1, wave 2 and wave 3 surveys. The HAALSI survey tool was designed to collect data on participants' demographic characteristics, lifestyle factors and cardiometabolic health measurements. Demographic data included variables such as age, gender, education level, employment status and survey wave. Lifestyle factors included dietary habits, smoking status, alcohol consumption, physical activity and sedentary behaviour. Cardiometabolic measurements included body mass index (BMI), diabetes status and direct blood pressure measurements for hypertension assessment.

BMI was categorised as underweight (<18.5), normal weight (18.5 - 24.9), overweight (25 - 29.9) and obese (≥30) kg/m^2; the small morbidly obese group (>35) was combined with the obese group for analysis. High BMI was defined as ≥25, indicating excessive adipose tissue accumulation associated with increased health risks.[1] Hypertension data were based on direct blood pressure measurements taken during each survey. Participants were classified as hypertensive if they had mean SBP ≥140 mmHg or mean DBP ≥90 mmHg, or reported current use of antihypertensive treatment. All others were classified as normotensive. This treatment-inclusive definition is the standard case definition used across the HAALSI cohort, and was adopted to correct an earlier blood-pressure-only definition, which excluded treated individuals whose blood pressure was controlled, and therefore underestimated prevalence, increasingly so across waves as treatment coverage rose.[1,14]

Access to the HAALSI data was granted through a formal data use agreement, permitting the download of the longitudinal codebook and Stata (StataCorp, USA) data files in DTA format. Variables relevant to the current study, including demographic characteristics, lifestyle factors and cardiometabolic health data, were extracted and imported into a password-protected Excel spreadsheet (Microsoft, USA) for initial organisation and validation before analysis in Stata.

Data collection and analysis

The data collection process was conducted in a stepwise manner. Data were extracted from the HAALSI dataset across three waves, including measurements used to calculate the dependent variable (hypertension) and independent variables such as age, gender, education level, employment status, wave period, dietary habits, smoking status, alcohol consumption, physical activity, sedentary behaviour, BMI and diabetes status. Datasets from all three waves were merged using a pre-existing participant unique identifier automatically allocated by the HAALSI database to each unique participant to create a comprehensive dataset for analysis.

For the covariates entered into the adjusted models, complete cases were analysed rather than recoding missing values to a separate category. Employment was missing for a substantial proportion of panel person-waves, and was therefore not carried in the adjusted models. Blood pressure was not median-imputed. In our corrected analysis, participants missing all hypertension components at a given wave were excluded from that wave, since imputing central blood-pressure values would bias prevalence estimates, and contributed to the earlier error. Inverse-probability weights were applied to account for attrition across waves. The cleaned dataset was password-protected and saved as an Excel file (Microsoft, USA).

Data were imported into and analysed using Stata version 18 software (StataCorp, USA). Statistical analysis included analysing temporal trends in hypertension prevalence and associated risk factors, and 95% confidence intervals (CIs). Descriptive statistics, including counts and percentages, were used to summarise categorical data. Hypertension status was computed as a binary variable (0=normotensive, 1=hypertensive) based on systolic and diastolic blood pressure measurements categorised using the WHO guidelines (SBP ≥140 mmHg or DBP ≥90 mmHg). In this corrected analysis, this binary outcome additionally classified as hypertensive any participant reporting current antihypertensive treatment, in line with the standard HAALSI case definition.

To model the longitudinal associations, a GEE model was applied. GEEs were selected over mixed-effects models because they estimate population-averaged effects, which aligns with our aim of examining average trends across a rural population rather than individual-specific trajectories. An exchangeable working correlation structure was specified with robust standard errors; population-averaged estimates are robust to the choice of working correlation. Bivariate analysis (χ^2 tests) assessed associations between hypertension and independent variables per wave. Variables with a p-value ≤0.2 in bivariate analyses, or those significant in at least one wave, were included in the multivariable models. Variables were also selected based on prior literature and theoretical importance to ensure clinical relevance.[18]

Multicollinearity among candidate covariates was checked before model fitting. Two multivariable GEE models were fitted, both with a binary logit link and exchangeable working correlation: model A included the covariate set without age or sex, and model B additionally adjusted for age and sex (the primary model). Both models are reported as a pre-specified sequential adjustment: age is a common cause of hypertension and of educational attainment and employment in this cohort, so comparing the estimates before and after adjustment for age and sex distinguishes associations robust to demographic confounding factors (BMI and diabetes) from those largely attributable to it (education and employment), and aids comparison with earlier age-unadjusted studies. Adjusted odds ratios (aORs) with 95% CIs were reported for significant variables in the GEE models. Confounders such as gender and sedentary behavious were adjusted for the multivariable GEE model to control for potential bias. Sensitivity analyses were performed with alternative definitions of hypertension, including stage 1 (SBP 140 - 159 mmHg or DBP 90 - 99 mmHg) and stage 2 (SBP ≥160 mmHg or DBP ≥100 mmHg). The GEE model was stratified by subgroups, such as age group (<60 and ≥60), gender (male and female) and BMI category (underweight, normal weight, overweight and obese). The GEE model was assessed using quasi-likelihood under the independence model criterion. Significance was set at $p<0.05$.

Ethical approval

The study received ethics approval from the University of Pretoria Faculty of Health Sciences Research Ethics Committee before it was conducted (ref. no. 420/2024). The data use agreement was obtained from HAALSI for de-identified human subject data.

Results

Demographic characteristics

The wave-specific cross-section comprised 4 936, 3 567 and 3 095 participants at waves 1, 2 and 3, respectively (Table 1). In wave 1, 17.2% (*n*=849) of participants were aged 40 - 49 years, 27.1% (*n*=1 340) 50 - 59 years, 26.2% (*n*=1 291) 60 - 69 years, 18.1% (*n*=892) 70 - 79 years and 11.4% (*n*=564) ≥80 years. As the fixed cohort aged, this distribution shifted upward across waves: by wave 3, those aged 40 - 49 years had fallen to 6.7% (*n*=207), while those aged ≥80 years had risen to 16.9% (*n*=522). Females comprised 54.0% (*n*=2 663) of the wave 1 cross-section, rising to 59.3% (*n*=1 836) by wave 3. Employed participants decreased from 15.8% (*n*=782) at wave 1 to 9.2% (*n*=284) at wave 3, while those not working increased from 83.9% (*n*=4 142) to 89.8% (*n*=2 779). At wave 1, most participants had no formal education (45.6%, *n*=2 252); 34.1% (*n*=1 685) had some primary schooling (grades 1 - 7), 11.3% (*n*=556) some secondary schooling (grades 8 - 11) and 8.6% (*n*=426) had completed secondary schooling or higher (grade 12 or above).

Changes in trends

In wave 1, 23% (*n*=1 042) participants consumed soft drinks once daily, while 28% (*n*=1 867) consumed them twice. By wave 3, the highest frequency (35.7%) shifted to zero consumption (*n*=1 106). In the matched panel (*N*=2 612), daily fruit intake was reported by 8.0% in wave 1, 6.5% in wave 2 and 3.9% in wave 3. Daily vegetable intake was low throughout (5.6% in wave 1, declining to 2.3% in wave 3), with most participants reporting infrequent consumption. Daily alcohol consumption (coded as 1) declined over the waves, from 3% (*n*=124) in wave 1 to 2% (*n*=72) of participants in wave 3. Most participants reported minimal alcohol use, with the proportion in the 'less than once per month' category (coded as 5) decreasing from 5% (*n*=177) in wave 1 to 1% (*n*=33) in wave 3, suggesting a reduction in occasional drinking over time. Most participants in all waves reported 'unknown' alcohol quantities. Among those who disclosed quantities, the proportion consuming 1 - 2 drinks (coded as 2) increased to 6% (*n*=211) in wave 3 from 1% (*n*=8) in wave 1.

The corrected Fig. 2 illustrates the changes in the key health and behavioural variables across three waves. The health and behavioural outcomes of the study population changed over the three waves. Under the corrected HAALSI definition (wave-specific cross-section, inverse-probability weighted), hypertension prevalence was 58.4% (95% CI 57.0 - 59.8%) in wave 1, 53.9% in wave 2 and 63.1% in wave 3, a shallow dip, then a rise, rather than a decline. In the corrected panel, overweight prevalence was broadly stable, dipping slightly in wave 2 (30.1% in wave 1, 29.1% in wave 2 and 30.3% in wave 3) and obesity stable-to-rising (32.6% - 33.0%), with underweight low (3.9% - 4.8%) across waves. Self-reported diabetes rose from 9.7% in wave 1 to 19.7% in wave 3, and ever smoking declined from 18.1% to 7.8%.

Changes in hypertension status

Table 2 summarises the changes in hypertension status from wave 1 to wave 2 and wave 2 to wave 3. Under the corrected definition, the largest group was consistently hypertensive at both wave pairs (45.2%). Apparent transitions from hypertensive to normotensive were

Table 1. Wave-specific cross-section demographic characteristics summary

Characteristic	Wave 1, *n*=4 936 (%)	Wave 2, *n*=3 567 (%)	Wave 3, *n*=3 095 (%)
Population size	4 936	3 567	3 095
Age group, years			
40 - 49	849 (17.2)	410 (11.5)	207 (6.7)
50 - 59	1 340 (27.1)	862 (24.2)	701 (22.6)
60 - 69	1 291 (26.2)	1 019 (28.6)	933 (30.1)
70 - 79	892 (18.1)	757 (21.2)	732 (23.7)
≥80	564 (11.4)	519 (14.6)	522 (16.9)
Gender			
Male	2 273 (46.0)	1 543 (43.3)	1 259 (40.7)
Female	2 663 (54.0)	2 024 (56.7)	1 836 (59.3)
Employment			
Employed	782 (15.8)	547 (15.3)	284 (9.2)
Not working	4 142 (83.9)	3 004 (84.2)	2 779 (89.8)
Education			
No formal education	2 252 (45.6)	1 580 (44.3)	1 354 (43.7)
Some primary	1 685 (34.1)	1 243 (34.8)	1 108 (35.8)
Some secondary	556 (11.3)	424 (11.9)	355 (11.5)
Secondary or more	426 (8.6)	311 (8.7)	270 (8.7)

Table 2. Changes in hypertension status

Hypertension status	Wave 1 - wave 2*	Proportion, %	Wave 2 - wave 3†	Proportion, %
Consistently hypertensive	1 180	45.2	1 181	45.2
Consistently normotensive	776	29.7	746	28.6
Hypertensive to normotensive	401	15.4	254	9.7
Normotensive to hypertensive	255	9.8	431	16.5
Total	2 612	100	2 612	100

Pearson $\chi^2(3)=78.7$, $p<0.001$.

substantially smaller than originally reported (15.4% for wave 1 to 2, and 9.7% for wave 2 to 3); the earlier excess reflected treated, controlled individuals being misclassified as normotensive. From wave 2 to wave 3, more individuals moved into hypertension (16.5%) than out of it (9.7%). Proportions are included alongside absolute values. These changes were statistically significant (Pearson χ^2 $p<0.001$ for both transitions).

Multivariable GEE analysis

Two population-averaged GEE models are reported (Table 3). Model A includes the original covariate set (education, BMI, diabetes, smoking, alcohol and diet) without adjustment for age or sex; model B additionally adjusts for age and sex and is the primary model. In the fully adjusted model B, obesity (aOR 1.84, 95% CI 1.60 - 2.12), overweight (aOR 1.37, 1.22 - 1.55), diabetes (aOR 1.25, 1.08 - 1.45) and older age (≥80 years, aOR 3.08, 2.39 - 3.99, with a graded increase from age 50 - 59) were independently associated with higher odds of hypertension, and frequent fruit consumption was protective (aOR 0.86, 0.76 - 0.98). The crude association with lower education was fully attenuated after adjustment for age (no formal education aOR 0.96, 0.79 - 1.16), indicating age confounding; sex, smoking and alcohol were not independently associated. Employment was examined but, as the data were missing for a substantial proportion of the panel, was not carried in the adjusted model; it was non-significant when included. The table summarises the significant variables in one or more waves of the bivariate analysis and multivariable GEE analysis identifying factors associated with hypertension, with wave 1 as a reference.

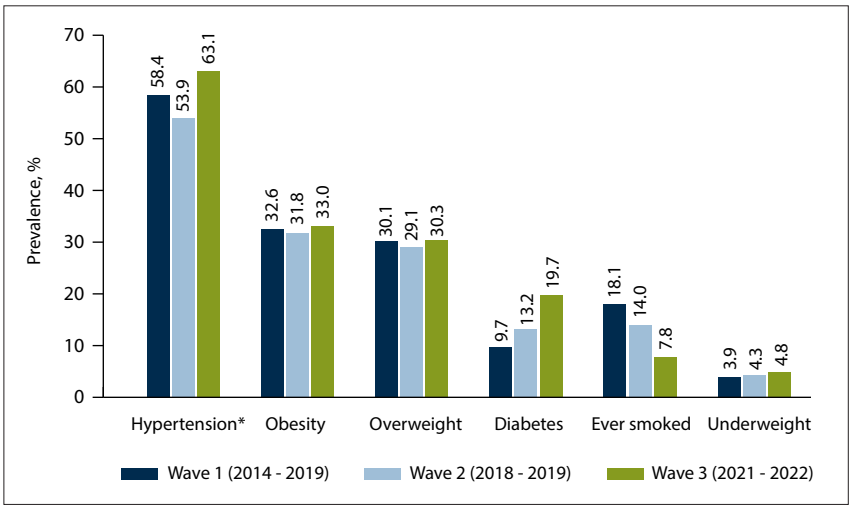


*Fig. 2. Changes in health outcome trends across waves. *Hypertension = inverse probability-weighted wave-specific cross-section; others = matched panel (N=2 612).*

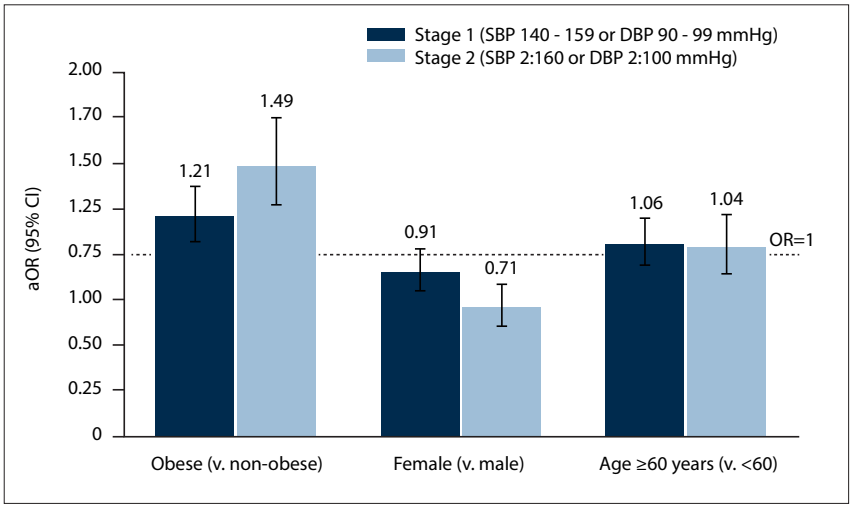


Fig. 3. Subgroup adjusted odds ratios (aORs) by hypertension stage (sensitivity analysis). (CI = confidence interval; SBP = systolic blood pressure; DBP = diastolic blood pressure.)

Sensitivity analysis

Fig. 3 illustrates the sensitivity analysis by subgroups. Adjusted ORs were estimated using GEE: the principal subgroups of interest were obesity, sex and age ≥60 years. Stage 1 hypertension is defined as SBP 140 - 159 mmHg or DBP 90 - 99 mmHg, and stage 2 hypertension as SBP ≥160 mmHg or DBP ≥100 mmHg. The dashed line at OR=1 represents no increased odds. In the corrected subgroup analysis, obese individuals had significantly higher odds of both stage 1 (OR 1.25, 95% CI 1.13 - 1.39) and stage 2 (OR 1.45, 95% CI 1.28 - 1.65) hypertension, and older adults (≥60 years) had higher odds of both stage 1 (OR 1.12, 95% CI 1.02 - 1.24) and stage 2 (OR 1.20, 95% CI 1.06 - 1.36) hypertension. Females had lower odds of both stage 1 (aOR 0.91, 95% CI 0.80 - 1.03) and stage 2 hypertension (aOR 0.71, 95% CI 0.60 - 0.84).

Discussion

The study's objectives were to examine temporal trends of hypertension prevalence and its associated factors among adults aged ≥40 years in Agincourt HDSS from 2014 to 2022. After correcting the case definition to include treated individuals, this study found no genuine decline in hypertension prevalence; the previously observed decline was an artefact of a blood-pressure-only definition. Prevalence was stable-to-rising across waves, consistent with an ageing cohort. In the corrected models, high BMI, diabetes and older age were the strongest predictors of hypertension; associations with education and employment attenuated after adjustment for age, indicating age confounding.

This analysis is one of the few to leverage repeated measures from a rural SA cohort. It allows us to observe within-individual transitions in hypertension status – an advantage over prior cross-sectional surveys. These findings support public health policy that is responsive to longitudinal risk patterns rather than snapshots.

The improvements in awareness, screening and treatment coverage in Agincourt increased the proportion of treated, controlled individuals over time. Under a blood-pressure-only definition, this produced an artefactual decline in measured prevalence, whereas the corrected treatment-inclusive definition shows a stable-to-rising burden. The corrected wave 1 prevalence (58.4%) matches the published HAALSI baseline, which used the same treatment-inclusive definition. In an independent HAALSI analysis using the identical definition, prevalence was essentially unchanged between baseline and

Table 3. Population-averaged GEE (matched panel, *N*=2 612)

Characteristic	Wave	Hypertension prevalence, *n* (%)	Bivariate analysis (χ^2)			Multivariable analysis (GEE)		
			OR	95% CI	*p*-value	aOR	95% CI	*p*-value
Age, years								
40 - 49	1	193/464 (41.6)	Ref	-	-	Ref	-	-
	2	109/304 (35.9)	Ref	-	-			
	3	70/166 (42.2)	Ref	-	-			
50 - 59	1	436/748 (58.3)	1.96*	1.55 - 2.48	0.000	1.26*	1.06 - 1.49	0.008
	2	283/620 (45.6)	1.50*	1.13 - 1.99	0.005			
	3	260/560 (46.4)	1.19	0.84 - 1.69	0.333			
60 - 69	1	479/747 (64.1)	2.51*	1.98 - 3.18	0.000	1.92*	1.58 - 2.32	0.000
	2	466/813 (57.3)	2.40*	1.83 - 3.16	0.000			
	3	497/776 (64.0)	2.44*	1.74 - 3.44	0.000			
70 - 79	1	327/457 (71.6)	3.53*	2.68 - 4.65	0.000	2.41*	1.95 - 2.98	0.000
	2	366/562 (65.1)	3.34*	2.50 - 4.47	0.000			
	3	444/650 (68.3)	2.96*	2.08 - 4.19	0.000			
≥80	1	146/196 (74.5)	4.10*	2.83 - 5.94	0.000	3.08*	2.39 - 3.99	0.000
	2	211/313 (67.4)	3.70*	2.65 - 5.16	0.000			
	3	341/460 (74.1)	3.93*	2.71 - 5.70	0.000			
Sex								
Male	1	605/1 040 (58.2)	Ref	-	-	Ref	-	-
	2	529/1 040 (50.9)	Ref	-	-			
	3	616/1 040 (59.2)	Ref	-	-			
Female	1	976/1 572 (62.1)	1.18*	1.00 - 1.38	0.045	1.11	0.96 - 1.29	0.163
	2	906/1 572 (57.6)	1.31*	1.12 - 1.54	0.001			
	3	996/1 572 (63.4)	1.19*	1.01 - 1.40	0.034			
Employment								
Not working	1	1 361/2 190 (62.1)	Ref	-	-	Ref	-	-
	2	1 251/2 219 (56.4)	Ref	-	-			
	3	1 503/2 372 (63.4)	Ref	-	-			
Employed	1	215/416 (51.7)	0.65*	0.53 - 0.80	0.000	0.99	0.86 - 1.15	0.935
	2	179/381 (47.0)	0.69*	0.55 - 0.85	0.001			
	3	98/216 (45.4)	0.48*	0.36 - 0.64	0.000			
Education								
No formal education	1	731/1 152 (63.5)	Ref	-	-	Ref	-	-
	2	659/1 152 (57.2)	Ref	-	-			
	3	747/1 152 (64.8)	Ref	-	-			
Some primary (1 - 7 years)	1	568/943 (60.2)	0.87	0.73 - 1.04	0.131	-	-	-
	2	529/943 (56.1)	0.96	0.80 - 1.14	0.611			
	3	586/943 (62.1)	0.89	0.74 - 1.06	0.201			
Some secondary (8 - 11 years)	1	166/298 (55.7)	0.72*	0.56 - 0.94	0.014	-	-	-
	2	143/298 (48.0)	0.69*	0.53 - 0.89	0.004			
	3	170/298 (57.0)	0.72*	0.56 - 0.93	0.013			
Secondary or more (≥12 years)	1	111/212 (52.4)	0.63*	0.47 - 0.85	0.002	-	-	-
	2	99/212 (46.7)	0.66*	0.49 - 0.88	0.005			
	3	107/212 (50.5)	0.55*	0.41 - 0.74	0.000			
BMI category								
Normal	1	417/848 (49.2)	Ref	-	-	Ref	-	-
	2	349/798 (43.7)	Ref	-	-			
	3	377/709 (53.2)	Ref	-	-			
Underweight	1	46/99 (46.5)	0.90	0.59 - 1.36	0.610	0.88	0.68 - 1.15	0.345
	2	29/98 (29.6)	0.54*	0.34 - 0.85	0.008			
	3	50/107 (46.7)	0.77	0.51 - 1.16	0.214			

...continued

Table 3. (continued) Population-averaged GEE (matched panel, *N*=2 612)

Characteristic	Wave	Hypertension prevalence, *n* (%)	Bivariate analysis (χ^2)			Multivariable analysis (GEE)		
			OR	95% CI	*p*-value	aOR	95% CI	*p*-value
	2	373/667 (55.9)	1.63*	1.33 - 2.01	0.000			
	3	405/675 (60.0)	1.32*	1.07 - 1.63	0.011			
Obese	1	574/827 (69.4)	2.34*	1.92 - 2.86	0.000	1.84*	1.60 - 2.12	0.000
	2	453/728 (62.2)	2.12*	1.73 - 2.60	0.000			
	3	482/736 (65.5)	1.67*	1.35 - 2.07	0.000			
Diabetes								
No	1	1 305/2 211 (59.0)	Ref	-	-	Ref	-	-
	2	995/1 944 (51.2)	Ref	-	-			
	3	1 229/2 085 (58.9)	Ref	-	-			
Yes	1	199/276 (72.1)	1.79*	1.36 - 2.37	0.000	1.25*	1.08 - 1.45	0.003
	2	264/394 (67.0)	1.94*	1.54 - 2.43	0.000			
	3	383/527 (72.7)	1.85*	1.50 - 2.29	0.000			
Ever smoked								
No	1	1 314/2 138 (61.5)	Ref	-	-	Ref	-	-
	2	1 244/2 233 (55.7)	Ref	-	-			
	3	1 472/2 380 (61.8)	Ref	-	-			
Yes	1	267/471 (56.7)	0.82	0.67 - 1.00	0.055	1.07	0.90 - 1.26	0.449
	2	182/363 (50.1)	0.80*	0.64 - 1.00	0.048			
	3	114/200 (57.0)	0.82	0.61 - 1.09	0.177			
Ever alcohol								
No	1	939/1 555 (60.4)	Ref	-	-	Ref	-	-
	2	999/1 773 (56.3)	Ref	-	-			
	3	1 287/2 068 (62.2)	Ref	-	-			
Yes	1	642/1056 (60.8)	1.02	0.87 - 1.19	0.834	1.09	0.97 - 1.22	0.169
	2	425/818 (52.0)	0.84*	0.71 - 0.99	0.037			
	3	297/510 (58.2)	0.85	0.69 - 1.03	0.097			
Daily fruit								
Infrequent	1	1 343/2 199 (61.1)	Ref	-	-	Ref	-	-
	2	1 185/2 178 (54.4)	Ref	-	-			
	3	1 400/2 225 (62.9)	Ref	-	-			
Frequent	1	230/399 (57.6)	0.87	0.70 - 1.08	0.198	0.86*	0.76 - 0.98	0.025
	2	237/405 (58.5)	1.18	0.95 - 1.47	0.127			
	3	175/325 (53.8)	0.69*	0.54 - 0.87	0.002			
Daily vegetable								
Infrequent	1	1 281/2 147 (59.7)	Ref	-	-	Ref	-	-
	2	1 062/1 944 (54.6)	Ref	-	-			
	3	1 327/2 151 (61.7)	Ref	-	-			
Frequent	1	297/460 (64.6)	1.23	1.00 - 1.52	0.051	0.91	0.81 - 1.01	0.086
	2	360/640 (56.2)	1.07	0.89 - 1.28	0.475			
	3	250/404 (61.9)	1.01	0.81 - 1.25	0.943			

GEE = generalised estimating equation; OR = odds ratio; CI = confidence interval; aOR = adjusted OR; Ref = reference; BMI = body mass index.
Bivariate (χ^2) and multivariable GEE analysis of factors associated with hypertension, matched panel (*N*=2 612), corrected HAALSI definition. Wave 1 (2014 - 2015) is the reference wave.
Variables were retained if $p \leq 0.2$ in the bivariate analysis at any wave. aORs are population-averaged (GEE, exchangeable independence working correlation). Statistically significant results ($p<0.05$) are marked with an asterisk.
Model A uses the original covariate set without age or sex; model B adds age and sex. ORs >1 indicate increased odds of hypertension. Education and employment lose significance in Model B, indicating age confounding.

4-year follow-up (58.4% v. 59.8%), despite a significant fall in mean SBP (−9.5 mmHg) and marked improvements in hypertension awareness, treatment and control over 2014 - 2019. This is precisely the pattern expected under a treatment-inclusive definition: as more individuals are treated and their blood pressure controlled, they remain classified as hypertensive, so prevalence stays stable even as blood pressure falls. A blood-pressure-only definition, by contrast, reclassifies these controlled individuals as normotensive, and therefore manufactures an artefactual decline – which is the error corrected here.[13,19]

These trends align with SA's national Strategic Plan for Non-Communicable Disease (2022 - 2027), which emphasises early detection and lifestyle modification to reduce hypertension-related morbidity.[15]

Older age was the strongest predictor of hypertension in the corrected model, with a clear gradient relative to the 40 - 49-year group: 50 - 59 years, aOR 1.26, 95% CI 1.06 - 1.49; 60 - 69 years, aOR 1.92, 95% CI 1.58 - 2.32; 70 - 79 years, aOR 2.41, 95% CI 1.95 - 2.98; and ≥80 years, aOR 3.08, 95% CI 2.39 - 3.99. This gradient is consistent with existing literature reporting that hypertension prevalence increases with age.

A study conducted in Ghana showed that there was a 53.72% hypertension prevalence rate among older adults, and the trend increased with increasing age.[20] A study

in China showed a continuous rise in SBP from age 35 to 79 years, and a concurrent early increase in the risk of high DBP after age 50 - 65 years.[21] The high SBP risk progresses more rapidly in the early lifetime in women, compared with the lifetime thereafter.[21] Older adults are more likely to develop hypertension due to age-related physiological changes, such as increased arterial stiffness and endothelial dysfunction.[22] These factors contribute to elevated blood pressure by impairing the ability of blood vessels to regulate blood flow.[21] These results underscore the importance of implementing age-targeted interventions, including regular health screenings, lifestyle modifications and medication management, to prevent and manage hypertension in older populations.

High BMI was a strong, dose-dependent predictor of hypertension in the corrected model: overweight (aOR 1.37, 95% CI 1.22 - 1.55) and obese (aOR 1.84, 95% CI 1.60 - 2.12) individuals had higher odds of hypertension than those of normal weight, while underweight was not significantly associated (aOR 0.88, 95% CI 0.68 - 1.15). This is consistent with existing literature.[23] A study showed that there is a linear correlation between body weight and BMI with blood pressure, where an increase in body weight of 1 kg increased SBP by 0.725 mmHg and DBP by 0.318 mmHg. In addition, an increase in BMI of 1 kg/m^2 was followed by an increase in SBP and DBP of 1.6 mmHg and 0.834 mmHg, respectively. Obesity is a well-known risk factor for hypertension, likely due to increased vascular resistance and sympathetic nervous system activity.[24] Studies suggest that even modest weight loss can significantly lower blood pressure, highlighting the importance of maintaining a healthy weight through lifestyle intervention.[25]

In the corrected model, ever consuming alcohol was not significantly associated with hypertension (aOR 1.09, 95% CI 0.97 - 1.22, p=0.169). Although alcohol was not independently associated with hypertension in this cohort after adjustment, the wider literature nonetheless links heavy consumption to raised blood pressure. Previous studies have demonstrated that high alcohol frequency (≥3 standard drinks per day) is associated with and predictive of hypertension; a reduction in alcohol frequency is associated with a significant dose-dependent lowering of mean SBP and DBP.[26] Excessive alcohol intake can elevate blood pressure by affecting vascular tone and increasing sympathetic nervous system activity. High-dose alcohol ≥13 hours after consumption has been found to increase SBP by 3.7 mmHg, DBP by 2.4 mmHg, and heart rate (HR) by 2.7 bpm (moderate certainty evidence for all).[27] Studies have shown that there is a causal association between high alcohol frequency and risk of hypertension, especially an alcohol intake of >12 g/d. These are consistent with recommendations to avoid or limit alcohol intake.[28] Given the cultural acceptance of alcohol use in many rural communities, measures to limit harmful alcohol use remain relevant to broader cardiovascular prevention, even though alcohol was not an independent predictor of hypertension in this analysis.

Frequent fruit consumption was associated with lower odds of hypertension in the corrected model (aOR 0.86, 95% CI 0.76 - 0.98), consistent with previous studies. A systematic review indicated that a high intake of fruit and vegetables, particularly fruit, was associated with a reduced risk of hypertension.[29] In our study, vegetable consumption was not significantly associated with hypertension after adjustment. There is a need for targeted public health intervention to promote healthier dietary habits for both fruit and vegetable consumption.

In unadjusted analyses, individuals with no formal education had higher odds of hypertension, but this association attenuated to non-significance after adjustment for age, indicating confounding by age. The crude association is nonetheless consistent with studies showing that lower education is associated with poorer health outcomes and limited access to health information.[30] A nationwide longitudinal study conducted in China on the association of education status with the risk of hypertension and its control revealed that individuals with higher education levels exhibited a significantly lower hypertension prevalence and decreased SBP and DBP.[30] A study conducted in the USA also found similar results among older US adults: those with less education were more likely to experience hypertension and uncontrolled BP than those with more schooling.[31] A study conducted in SA also found that a high level of education was inversely related to the odds of hypertension prevalence.[32]

Employment was protective in unadjusted analyses, but this association also attenuated after adjustment for age. Where observed, a protective effect of employment may operate through increased physical activity, better access to healthcare resources and social support.[33] Previous studies found that employment was protective of hypertension, with 70% lower odds of hypertension among those employed compared with those not in any form of employment.[33]

Diabetes was significantly associated with hypertension in the corrected model (aOR 1.25, 95% CI 1.08 - 1.45, p=0.003), reversing the earlier null finding. Diabetes shares common risk factors with hypertension, such as insulin resistance and obesity, which contribute to their interconnectedness.[34] A study has shown that the prevalence of hypertension among type 2 diabetes patients was 59.5%.[35] Stage 1 hypertension was the most common (30.95%).[35] Diabetic individuals' odds of hypertension were highest among the age group 50 - 60 years, with aOR=2.5.[35] Patients with longer duration of type 2 diabetes had aOR 1.16, and patients with poor glycaemic control had aOR=3.0.[35] A study conducted in Ethiopia found that hypertension was prevalent in over half of diabetic patients. The relationship between hypertension and diabetes highlights the need for integrated care strategies to manage both conditions effectively, particularly in rural areas.[36]

Although smoking was not significant within the study population, it is a well-established associated factor for hypertension and cardiovascular disease. A study conducted in China showed that heavy smokers of machine-rolled cigarettes had an elevated hypertension risk compared with non-smokers (HR 1.50, 95% CI 1.05 - 2.16).[37] The interaction effects of heavy smoking-heavy drinking patterns increased future hypertension risk, with an adjusted HR of 2.58 (95% CI 1.06 - 6.33).[37]

The present study highlights the need for comprehensive hypertension prevention and management strategies in rural SA, to expand hypertension screening programmes, promote healthy diets, physical activity and weight loss management, implement community-based education on the risks of excessive alcohol consumption and improve literacy and health knowledge during clinic visits.

Study limitations

The study analysed secondary data, which has its limitations. These include having no control over data collection. Other significant diseases, specifically chronic kidney disease, were not included as captured variables, as they are not present in the HAALSI dataset. This omission affects the comprehensiveness of the analysis regarding hypertension risk factors. There is an absence of data on secondary causes of hypertension within the HAALSI datasets, such as secondary hypertension, which can be caused by conditions such as hypo-/hyperthyroidism, coarctation of the aorta, hyperparathyroidism, vasculitis, autoimmune diseases, pheochromocytoma, Cushing's disease/syndrome, renal artery stenosis, glomerulonephritis, nephrotic syndrome and drug-induced hypertension (e.g. due to steroids or

sympathomimetics). The exclusion of these variables from our analysis means that our findings are primarily focused on primary hypertension, and do not fully capture the complexity of hypertension in the studied population. This limitation could lead to an underestimation or misattribution of the causes of elevated blood pressure, particularly in cases where secondary hypertension is present but not diagnosed or recorded in the datasets.

A key limitation of the original analysis was the use of a blood-pressure-only case definition (SBP ≥140 mmHg or DBP ≥90 mmHg), which excluded individuals whose blood pressure was controlled by treatment, and therefore underestimated hypertension prevalence, increasingly so across waves as treatment coverage rose. This has been corrected in the present analysis by adopting the standard HAALSI definition, which additionally classifies as hypertensive any participant reporting current antihypertensive treatment. A further consideration is that the matched three-wave panel (*N*=2 612) comprises individuals who survived and remained in the cohort across all three waves. Because attrition selects towards younger, healthier participants, the panel is somewhat older and higher-risk than the full baseline sample, which explains why panel prevalence (60.5% at wave 1) slightly exceeds the full-sample baseline (58.4%). The matched panel is nonetheless the appropriate sample for a within-person transitions analysis, since individual-level movement between hypertensive and normotensive states can only be estimated among participants observed at every wave.

Conclusion

The study analysed temporal trends of hypertension prevalence and its associated risk factors among adults aged ≥40 years in Agincourt HDSS, Mpumalanga Province, SA. Using the standard HAALSI case definition, the study found that hypertension prevalence did not decline between waves 1 and 3 (58.4%, 53.9% and 63.1%, respectively). The previously reported decline, and the apparent mass transition from hypertensive to normotensive, were artefacts of a blood-pressure-only case definition that excluded treated, controlled individuals. In the corrected models, high BMI, diabetes and older age were the strongest predictors of hypertension, while associations with education and employment attenuated after adjustment for age. In the corrected subgroup analysis, obese individuals had significantly higher odds of both stage 1 and stage 2 hypertension. Older age ≥60 years had significantly higher odds of stage 1 or stage 2 hypertension. Females had lower odds of both stage 1 and stage 2 hypertension.

Future studies should focus on increasing access to hypertension screenings and promoting lifestyle modifications in rural areas, to further reduce the burden of hypertension as the leading cause of cardiovascular disease.

Data availability. The data used in this study are publicly available and can be accessed from the HAALSI dataset at https://haalsi.org.

Declaration. None.

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