Estimating the changing burden of disease attributable to high sodium intake in South Africa for 2000, 2006 and 2012

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Background. Elevated sodium consumption is associated with increased blood pressure, a major risk factor for cardiovascular and chronic kidney disease.
Objectives. To quantify the deaths and disability-adjusted life years (DALYs) attributed to high sodium intake in persons aged ≥25 years in South Africa (SA) for 2000, 2006 and 2012.
Methods. Comparative risk assessment (CRA) methodology was used and population attributable fractions (PAFs) of high sodium intake, mediated through high blood pressure (BP), for cardiovascular and chronic kidney disease were estimated. This was done by taking the difference between the PAF for elevated systolic BP (SBP) based on the estimated SBP level in the population and the PAF based on the estimated SBP that would result if sodium intake levels were reduced to the theoretical minimum risk exposure level (1 g/day) according to population group and hypertension categories. A meta-regression based on data from nine national surveys conducted between 1998 and 2017 was used to estimate the prevalence of hypertension by age, sex and population group. Relative risks identified from international literature were used and the difference in PAFs was applied to local burden estimates from the second South African National Burden of Disease Study. Age-standardised rates were calculated using World Health Organization (WHO) standard population weights. The attributable burden was also estimated for 2012 using an alternative target of 2 g/day proposed in the National Strategic Plan for the Prevention and Control of Non-communicable Diseases (NSP).
Results. High sodium intake as mediated through high SBP was estimated to cause 8 071 (95% uncertainty interval (UI) 6 542 - 15 474) deaths in 2012, a drop from 9 574 (95% UI 8 158 - 16 526) in 2006 and 8 431 (95% UI 6 972 - 14 511) in 2000. In 2012, ischaemic heart disease caused the highest number of deaths in persons (*n*=1 832), followed by haemorrhagic stroke (*n*=1 771), ischaemic stroke (*n*=1 484) and then hypertensive heart disease (*n*=1 230). Ischaemic heart disease was the highest contributor to deaths for males (27%), whereas for females it was haemorrhagic stroke (23%). In 2012, 1.5% (95% UI 1.3 - 2.9) of total deaths and 0.7% (95% UI 0.6 - 1.2) of total DALYs were attributed to high sodium intake. If the NSP target of <2 g/day sodium intake had been achieved in 2012, ~2 943 deaths and 48 870 DALYs would have been averted.
Conclusion. Despite a slight decreasing trend since 2006, high sodium intake mediated through raised BP accounted for a sizeable burden of disease in 2012. Realising SA's target to reduce sodium intake remains a priority, and progress requires systematic monitoring and evaluation.

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The article in context

Evidence before this study. High dietary sodium consumption is a key driver of hypertension. There are no national data on dietary sodium intake in SA; however, limited older studies performed during the 2000s demonstrated daily intake of sodium to be between 2.4 and 4.8 g/day compared with recent studies that reported a mean sodium intake of 2.8 g/day in 2015, higher than the <2 g/day sodium (equivalent to 5 g/day salt) recommended by the WHO in 2012. A modelling study suggests that a modest reduction in the SA population's sodium intake of 0.85 g/day through selected processed foods would result in 7 400 fewer cardiovascular disease deaths and 4 300 fewer non-fatal strokes, with cost savings of up to ZAR300 million per year.
Added value of this study. This is the first study to produce comprehensive and comparable assessment of deaths and DALYs attributable to high sodium intake in SA, including an uncertainty analysis. Using a consistent approach, our study estimated the attributable burden

for 15 health-related outcomes for three time points, 2000, 2006 and 2012, due to high sodium intake. There has been a slight decline in mortality attributable to sodium since 2006.

Implications of the available evidence. Growing recognition of the role of high sodium intake led SA to legislate sodium restrictions in certain processed foods that came into effect in 2016. This study reiterates that the potential benefits of reducing sodium intake are considerable. The results suggest that the salt reduction policy could decrease the burden of cardiovascular disease by 2 943 deaths yearly and minimise health expenditure in thousands of households in SA. National programmes to monitor sodium consumption, including the content in the selected processed foods, need to be developed to inform strategies to reduce cardiovascular disease.

The burden of cardiovascular diseases continues to grow in South Africa (SA).[1,2] This increase is due to the high prevalence of hypertension, the upsurge in other risk factors such as obesity, and an ageing population.[3] Studies suggest that SA has the highest prevalence of hypertension in sub-Saharan Africa,[4,5] ranging from 42% to 54% among people 40 - 60 years old,[4] with a large number of people (48%) having uncontrolled high blood pressure (BP).[6]

High dietary sodium intake has been identified as an important factor contributing to the pathogenesis of hypertension and the increasing burden of cardiovascular diseases worldwide.[7-14] Unequivocal evidence[11,15,16] has highlighted the association between high dietary sodium intake and high BP. Reduction in sodium intake has been shown to be linked to a decrease in both BP levels and cardiovascular morbidity and mortality.[17,18] Although the evidence is clear that a reduction in high sodium intake leads to reduced BP and to cardiovascular benefit, there is some debate about how far sodium should be reduced to minimise cardiovascular diseases.[19,20]

According to the World Health Organization (WHO), average sodium intake globally is high, ranging from 3.6 to 6 g/day.[21,22] Based on evidence, a reduction of daily sodium consumption to <2 g/day has been recommended.[21,22] Since then, many countries and international organisations have advocated and recommended population-based intake targets. In 2017, the Global Burden of Diseases, Injuries, and Risk Factors Study (GBD) found high sodium intake to be a leading dietary risk factor, accounting for 3 million deaths and 70 million disability-adjusted life years (DALYs) a year globally (more than half of the diet-related deaths), owing to its strong association with cardiovascular disease.[23]

Prior to 2012, there were no national data on dietary sodium intake in SA. Local studies[24-26] have demonstrated relatively high daily intake of sodium, ranging between 2.4 and 4.8 g/day for different communities and population groups. A recent study, based on 24-hour urine excretion, conducted by Swanepoel *et al.*[27] in North West and KwaZulu-Natal provinces in three population groups (black African, white and Asian – see the disclaimer at the end of the article), reported that ~77% of South Africans consume >2 g/day of sodium. The same study found an average sodium intake of 2.8 g/day, which is lower than the levels reported previously[27] and seems to suggest that there has been a decrease in sodium intake.

Based on the evidence of the harmful effects of high sodium intake, in 2013 the National Department of Health regulated the sodium content of selected processed food categories[28] that are commonly eaten, viz. bread, breakfast cereals, processed meat, fat and butter spread, soup powders and potato crisps. Furthermore, in its strategic plan (National Strategic Plan for the Prevention and Control of Non-communicable Diseases 2013 - 17 (NSP)), SA set a target for the population to reduce dietary salt intake by 30% by the year 2020 and recommended consumption of <2 g sodium (5 g salt) per day to prevent cardiovascular diseases.[29] For efficiency and effective monitoring, SA chose the legislation route of sodium reduction over a voluntary approach for the industries. The new legislation was implemented progressively in 2016 and 2019.[29]

The first South African Comparative Risk Assessment Study (SACRA1) in 2000,[30] conducted by the South African Medical Research Council to provide a systematic evaluation of the disease burden caused by 17 selected modifiable risk factors, did not include high sodium consumption. Since then, Bertram *et al.*[31] estimated that sodium intake in SA could be reduced by 0.85 g/day on average through salt regulations on processed foods, and ~7 400 cardiovascular deaths and 4 300 non-fatal strokes could be averted based on the 2008 information. However, information on the effects of sodium consumption, as well as the variations of these effects according to age, sex and population group over time, has not been reported. The study reported in this article aimed to estimate the number of deaths and DALYs attributable to high sodium consumption mediated through high BP in SA for 2000, 2006 and 2012 and estimate the burden that would be prevented if the national target to reduce the sodium intake was met.

Methods

Terminology

Some countries use the term 'sodium' in public health policy discourse, while others use the term 'salt'; the two terms are often used synonymously.[32] On a weight basis, salt comprises 40% sodium and 60% chloride, and 1 g sodium is equivalent to 2.5 g of salt.[22] In this article, we use the term sodium and the conversion of different units is as follows: 1 mmol of sodium = 23 mg of sodium or 1 g of sodium = 43.5 mmol of sodium.[32]

Comparative risk assessment

A comparative risk assessment (CRA) approach was undertaken following the GBD methodology for high sodium intake mediated through high systolic BP (SBP), updated in 2017.[23] For cardiovascular diseases and chronic kidney diseases, we calculated the fraction of disease burden attributable to high sodium intake, the population attributable fraction (PAF), as the difference between the proportion of disease burden associated with the current level of BP in the population and the proportion of disease burden if the actual population sodium intake (exposure) were shifted to a counterfactual scenario corresponding to the lowest risk (theoretical minimum risk exposure level, TMREL) of 1 g/day.[23,33] While there is uncertainty around the optimal level of sodium intake, the review by Mozaffarian *et al.*[33] indicated that a mean (standard deviation (SD)) level of 1.0 (0.1) g/day has the lowest risk for cardiovascular mortality.

The PAF was estimated in a two-step process. Firstly, for each disease outcome o and year y, we calculated the PAF for high SBP as:

$$PAF1 = \frac{\int_{50}^{250} RR_o(x)P1_y(x)dx - \int_{50}^{250} RR_o(x)P'(x)dx}{\int_{50}^{250} RR_o(x)P1_y(x)dx}$$

where $RR_o(x)$ is the relative risk (RR) for disease o as a function of the SBP x; $P1_y(x)$ is the population distribution of SBP in year y; and $P'(x)$ is the TMREL distribution for SBP, assumed uniform between 110

and 115 mmHg as applied in our estimation of the burden attributable to high BP.[34]

We then calculated the attributable fractions after removing the effects of high sodium in the same population:

$$PAF2 = \frac{\int_{50}^{250} RR_o(x)P2_y(x)dx - \int_{50}^{250} RR_o(x)P'(x)dx}{\int_{50}^{250} RR_o(x)P2_y(x)dx}$$

where $P2_y(x)$ is the distribution of SBP after the effects of the high sodium intake have been removed.

The effects of sodium consumption on SBP were determined using a linear model from the meta-analysis of 103 randomised control trials conducted by Mozaffarian *et al.*[33] The model provided shift values in average SBP according to age (older persons had larger reductions in SBP than younger people), population group (black African v. non-black African) and hypertensive v. normotensive persons (Table S1 in the appendix, https://www.samedical.org/file/1816), based on the TMREL target of 1 g of sodium per day (43.5 mmol/day).[35] We used the same SD as GBD 2017, calculated from the lower and upper confidence limits and assuming a normal distribution. In the SA context, we have considered white, coloured and Asian population groups in the non-black category, as salt sensitivity is considered to be a genetic factor associated with black Africans.[36,37] By calculating *PAF*1 – *PAF*2, we get the effect of high sodium intake on disease outcomes. For the sensitivity analysis using an alternative sodium intake target defined by the NSP, new shift values were calculated using the alternative target of 2 g/day (87 mmol/day) (Table S1 in the appendix: https://www.samedical.org/file/1816).

Estimates of SBP

As reported by Nojilana *et al.*,[34] we adopted a meta-regression approach for estimation of the mean and SD of the population distribution of SBP by sex, age (25 - 34, 35 - 44, 45 - 54, 55 - 64 and ≥65 years) and population group in SA. Data on individual SBP levels were extracted from nine national population surveys conducted in SA between 1998 and 2017, namely the three South Africa Demographic and Health Surveys (SADHSs),[38-40] the five waves of the National Income Dynamics Study (NIDS),[41-45] and the South African National Health and Nutrition Examination Survey 2012 (SANHANES-1).[46] We excluded the first of the multiple SBP readings included in the different datasets and those with implausible values (SBP <70 mmHg or >270 mmHg) and considered the average of the remaining readings as the participant's BP.[47] We used standard methods (weighted estimators with robust standard error) to recover survey-, age-, race- and sex-specific estimates of the mean and SD of SBP, taking into account the complex sampling design of each individual survey. Separately by sex and population group, we fit a series of generalised additive models with the estimates above as outcomes and year, age category and their interaction as predictors. In defining the structure of the models, we assumed a linear temporal trend for both mean SBP and its variance, but we left unspecified the shape of the relationship between each variable and age (modelled as a thin-plate spline).

We fit the models with R Statistical Software v. 3.6.0 (R Core Team, Austria), and weighted the input estimates according to the quality weight approach described by Doi *et al.*,[48] which combines in a principled manner information on the relative precision of the survey-specific estimates (as conveyed by their standard error) with information on the relative quality of the data sources[49] (summarised by the risk of bias score). The estimated coefficients were used to predict the values of the variable of interest (and their standard error) and recover sex-, age- and race-specific temporal trends in the mean and SD of SBP, used for the PAF estimation for 2000, 2006 and 2012.

We also estimated trends in the prevalence of hypertension, defined at individual level as having an SBP ≥140 mmHg and/or a diastolic BP ≥90 mmHg and/or being on (self-reported) antihypertensive treatment, to be able to calculate the shift in SBP for the counterfactual level of sodium intake. We used the same meta-regression approach described above both for cleaning data on diastolic BP (excluding readings <30 mmHg or >150 mmHg) and for modelling the relationship with time and age (Fig. S1 in the appendix: https://www.samedical.org/file/1816).

Relative risks

The cardiovascular diseases and chronic kidney disease outcomes associated with high sodium intake are given in Table 1, with the range of variation across age groups of the relative risks (RR) for each 10 mmHg increase above the TMREL for SBP by age group used in GBD 2017. Age-specific RRs are shown in Table S2 in the appendix (https://www.samedical.org/file/1816).

The shift in mean SBP attributed to high sodium intake was dependent on the prevalence of hypertension by population group. Fig. 1 shows the prevalence of hypertension by population group for 2000, 2006 and 2012. Variations can be observed among the population groups throughout the study period. The prevalence of hypertension was highest for the coloured population in 2012, followed by the Asian and white populations, while it had been highest for Asians in 2000.

Attributable burden and uncertainty analysis

The PAFs calculated for each disease outcome were then applied to the corresponding number of deaths and deaths and years of life lost (YLLs) due to premature mortality based on the second South African National Burden of Disease Study (SANBD2) for 2000, 2006 and 2012, which covers the whole country and was adjusted for underreporting of deaths.[50] The PAFs were also applied to DALYs extrapolated using the ratio of non-fatal burden to fatal burden from the GBD study. The total attributable burden was calculated by summing up the burden due to each individual disease outcome.

Age-standardised rates were calculated for the attributable deaths and DALYs for males, females and persons (≥25 years) using population estimates[51] and the WHO world standard population weights.[52]

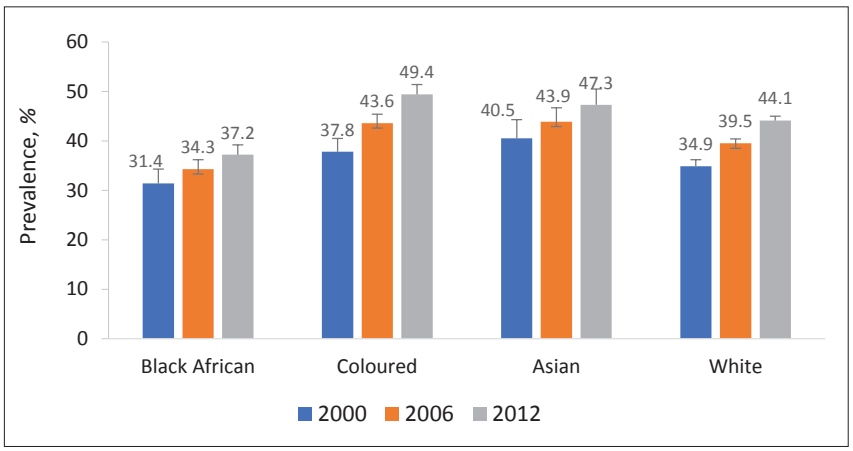

Fig. 1. Prevalence of hypertension among adults (≥25 years) by population group in South Africa for 2000, 2006 and 2012.

Table 1. RRs per 10 mmHg above the systolic blood pressure TMREL for cardiovascular and chronic kidney disease outcomes

Condition	ICD-10 code	RR range across age groups
Rheumatic heart disease	I00 - I09	1.104 - 1.631
Hypertensive heart disease	I11	1.619 - 2.862
Ischaemic heart disease	I20 - I25	1.266 - 1.972
Endocarditis and other cardiomyopathy	I30, I31, I33, I38 - I40, I42	1.128 - 1.755
Ischaemic stroke	G45 - G46.8, I63 - I63.9, I65 - I66.9, I67.2 - I67.8, I69.3 - I69.4	1.201 - 1.854
Haemorrhagic stroke	I60 - I62, I62.1 - I62.9, I67.0 - I67.1, I68.1 - I68.2, I69.0 - I69.2	1.279 - 2.134
Atrial fibrillation and flutter	I44, I45, I47, I48	1.134 - 1.76
Aortic aneurysm	I71	1.119 - 1.544
Peripheral vascular disease	I73	1.095 - 1.728
Other cardiovascular and circulatory diseases	I27, I28, I34 - I37, I72, I77 - I78, I80, I82 - I84, I86 - I89, I95 - I98	1.137 - 1.744
Chronic kidney disease due to hypertension, and glomerulonephritis	I12 - I13.9; N03 - N06	1.281*
Chronic kidney disease due to other and unspecified causes	N02, N07 - N08, N18	1.282*
Chronic kidney disease due to diabetes mellitus	E10.2, E11.2	1.283*

Source: Adapted from Global Burden of Disease 2017.[23]
RR = relative risk; TMREL = theoretical minimum risk exposure level.
*RR was the same for all ages and both sexes.

Uncertainty around the point estimates was calculated using Ersatz version 1.35,[53] which uses Monte Carlo simulation modelling techniques. This allows for uncertainty in the calculations to be reflected. Separately for each year, sex, age, population group and health outcome, we drew 2 000 random samples from the distributions of the parameters of the exposure distribution, the RR functions, TMREL and the shift values, and repeated the calculation of the PAFs, attributable deaths, YLLs, years lived with disability (YLDs), DALYs and age-standardised rates. We used the 2.5th and 97.5th percentiles as the bounds of the 95% uncertainty interval (UI). In drawing the samples, a normal distribution was specified for the mean and SD estimates of observed population distribution of SBP and distribution of SBP after the effects of the high sodium intake have been removed, as well as the shift values. For RR estimates we used the Ersatz function ErRelativeRisk.[54] The ErRelativeRisk function assumes a log-normal uncertainty distribution for the RR and introduces a correction to eliminate the upward bias in the mean of the randomly drawn values.

Results

Attributable burden

The overall PAFs, estimated attributable deaths and DALYs for each disease outcome are shown in Table 2. An estimated 8 431 (95% UI 6 972 - 14 511) deaths or 1.7% (95% UI 1.4 - 2.9) of total deaths were attributed to high sodium intake in 2000. This increased slightly in 2006 to 9 574 (95% UI 8 158 - 16 526) deaths or 1.4% (95% UI 1.2 - 2.4) of total deaths, and then decreased in 2012 to 8 071 (95% UI 6755 - 15 302) deaths or 1.5% (95% UI 1.3 - 2.9) of total deaths.

Female attributable deaths arising from high sodium intake as mediated through high BP were higher than for males (4 552 compared with 3 519) in 2012, a decrease from 2000, which was 4 603 for females and 3 828 for males.

Table 3 shows the proportion of attributable deaths due to high sodium intake out of all deaths, by population group. While the proportions were fairly similar for whites and Asians, they were consistently higher among Asians for both sexes throughout the study period and consistently lowest for black Africans throughout. The proportion of the total deaths (all ages) attributable to high sodium intake decreased in 2012 for all groups except black Africans.

Percentage of deaths by disease outcome

Fig. 2 shows the percentage of deaths attributable to high sodium by disease outcome and by sex for 2000, 2006 and 2012. Attributable deaths increased in 2006 and declined slightly in 2012. Females have always had higher proportions than males for cardiovascular disease-related deaths, except for ischaemic heart disease and ischaemic stroke. The figure shows that the top four causes of deaths in males were similar to those in females with variations in ranking. In 2012, ischaemic heart disease was a leading contributor to deaths in males, accounting for 27.2%, followed by haemorrhagic stroke (20.3%). In females, haemorrhagic stroke was the leading cause, accounting for 23.2% of deaths, followed by ischaemic stroke (20.5%). Hypertensive heart disease was common in both males and females; however, it was higher in females (17.6%) compared with males (12.2%). Furthermore, other cardiomyopathy contributed more than chronic kidney disease in males.

Fig. 3 shows that, nationally, cardiovascular diseases contributed more to attributable deaths than chronic kidney disease in both males and females in 2000 and 2012. However, the contribution of chronic kidney disease increased in 2012 in both genders and across all age groups. Male deaths due to cardiovascular diseases and chronic kidney diseases peaked around the working age groups (45 - 69 years old) and then began to decline from ≥70 years, whereas female deaths showed an increase in both these groups of conditions as age increased.

Age-standardised death and DALY rates attributable to high sodium intake

National age-standardised death rates per 100 000 population increased from 2000 to 2006 to 56 and 47 for males and females, respectively, then dropped in 2012 to 43 and 45 (Fig. 4). This drop was more notable in the Asian population than in black Africans. Coloureds and whites also had much smaller age-standardised death rates in 2012, but similar rates in 2000 and 2006 (Fig. 5).

Age-standardised DALYs also increased in 2006 and dropped in 2012 nationally for both males and females. This was again more notable in the Asian population, as well as the coloured and white populations. The age-standardised DALY rates of the black African group also decreased slightly over the study period.

Table 2. Deaths and DALYs attributable to high sodium intake in South Africa for 2000, 2006 and 2012

	Males			Females			Persons		
Disease outcome	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n***	**DALYs, *n***
2000									
Ischaemic heart disease	9.6	1 213	20 605	9.5	1 098	17 857	9.5	2 311	38 462
Haemorrhagic stroke	9.4	778	14 279	9.0	1 071	19 049	9.2	1 850	33 328
Ischaemic stroke	9.1	554	8 373	8.6	859	12 588	8.8	1 414	20 961
Hypertensive heart disease	7.6	460	7 533	7.0	737	11 615	7.2	1 198	19 148
Other cardiomyopathy	7.5	258	5 257	7.1	208	4 306	7.3	466	9 563
Other cardiovascular and circulatory diseases	7.9	171	3 583	7.7	226	4 446	7.8	396	8 029
Chronic kidney disease due to hypertension	8.0	171	3 684	8.2	175	3 706	8.1	346	7 390
Chronic kidney disease due to other and unspecified causes	7.9	80	1 730	7.6	76	1 815	7.7	156	3 546
Rheumatic heart disease	6.0	20	518	5.7	42	1 161	5.8	62	1 680
Chronic kidney disease due to glomerulonephritis	7.0	30	1 325	6.6	23	1 226	6.8	53	2 551
Aortic aneurysm	7.7	34	524	7.3	26	424	7.5	61	948
Chronic kidney disease due to diabetes mellitus	8.1	12	283	8.1	18	413	8.1	30	696
Atrial fibrillation and flutter	7.5	16	596	7.2	25	784	7.3	42	1 379
Endocarditis	7.5	17	355	6.7	9	183	7.2	25	538
Peripheral vascular disease	5.9	14	234	5.7	9	148	5.8	23	381
Total attributable burden	-	**3 828**	**68 880**	-	**4 603**	**79 721**	-	**8 431**	**148 601**
(95% UI)		(3 305 - 6 067)	(60 002 - 107 281)		(3 605 - 8 611)	(64 945 - 137 568)		(6 972 - 14 511)	(127 569 - 242 710)
% of total burden	-	**1.4**	**0.7**	-	**1.9**	**0.8**	-	**1.7**	**0.8**
(95% UI)		(1.2 - 2.3)	(0.6 - 1.1)		(1.5 - 3.6)	(0.7 - 1.5)		(1.4 - 2.9)	(0.7 - 1.3)
2006									
Ischaemic heart disease	9.6	1 414	24 238	9.4	1 232	20 378	9.5	2 646	44 617
Haemorrhagic stroke	9.2	832	14 945	9.1	1 199	21 270	9.1	2 031	36 215
Ischaemic stroke	9.0	605	9 052	8.6	993	14 462	8.8	1 598	23 514
Hypertensive heart disease	7.2	451	7 074	7.0	855	13 230	7.1	1 305	20 304
Other cardiomyopathy	7.6	284	5 903	7.2	264	5 792	7.4	548	11 695
Other cardiovascular and circulatory diseases	8.2	238	5 170	8.2	264	5 655	8.2	502	10 825
Chronic kidney disease due to hypertension	8.0	176	3 959	7.7	255	5 603	7.8	431	9 562
Chronic kidney disease due to other and unspecified causes	7.8	104	2 384	7.5	85	2 200	7.7	189	4 584
Rheumatic heart disease	7.1	40	1 961	6.5	30	1 847	6.8	70	3 807
Chronic kidney disease due to glomerulonephritis	6.1	21	567	5.8	35	907	5.9	55	1 474
Aortic aneurysm	7.7	33	482	7.3	19	288	7.5	51	770

Continued ...

Table 2. (continued) Deaths and DALYs attributable to high sodium intake in South Africa for 2000, 2006 and 2012

	Males			Females			Persons		
Disease outcome	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n***	**DALYs, *n***
Chronic kidney disease due to diabetes mellitus	7.6	17	892	7.3	29	935	7.4	46	1 827
Atrial fibrillation and flutter	6.0	25	405	5.9	16	251	6.0	41	656
Endocarditis	8.4	13	280	8.3	19	423	8.4	32	703
Peripheral vascular disease	7.6	17	353	6.8	10	219	7.2	27	572
Total attributable burden	-	**4 270**	**77 665**	-	**5 303**	**93 460**	-	**9 574**	**171 125**
(95% UI)		(3 750 - 6 687)	(68 863 - 117 951)		(4 324 - 9 966)	(79 200 - 161 180)		(8 158 - 16 526)	(149 120 - 277 008)
% of total burden	-	**1.2**	**0.6**	-	**1.6**	**0.7**	-	**1.4**	**0.7**
(95% UI)		(1.1 - 1.9)	(0.6 - 1.0)		(1.3 - 3.0)	(0.6 - 1.2)		(1.2 - 2.4)	(0.6 - 1.1)
2012									
Ischaemic heart disease	7.3	957	16 492	7.5	875	14 474	7.4	1 832	30 967
Haemorrhagic stroke	8.3	714	12 305	8.5	1 057	17 901	8.4	1 771	30 206
Ischaemic stroke	8.1	552	8 139	8.0	932	13 314	8.1	1 484	21 453
Hypertensive heart disease	6.6	428	6 461	6.6	802	12 171	6.6	1 230	18 632
Other cardiomyopathy	7.5	283	6 476	7.5	299	7 596	7.5	581	14 072
Other cardiovascular and circulatory diseases	7.0	218	4 506	6.7	187	4 026	6.8	405	8 532
Chronic kidney disease due to hypertension	7.3	123	3 076	7.1	174	4 278	7.2	297	7 354
Chronic kidney disease due to other and unspecified causes	7.2	109	2 815	7.0	96	2 936	7.1	205	5 750
Rheumatic heart disease	6.7	33	2 338	6.3	21	2 399	6.5	54	4 738
Chronic kidney disease due to glomerulonephritis	6.9	19	1 131	6.7	30	1 049	6.8	49	2 181
Aortic aneurysm	5.5	13	336	5.4	25	712	5.5	38	1 048
Chronic kidney disease due to diabetes mellitus	5.3	21	332	4.9	15	250	5.1	36	582
Atrial fibrillation and flutter	5.8	21	312	5.7	12	193	5.7	33	505
Endocarditis	7.1	13	307	7.2	19	522	7.2	31	830
Peripheral vascular disease	7.0	15	311	6.5	8	166	6.8	23	477
Total attributable burden	-	**3 519**	**65 337**	-	**4 552**	**81 988**	-	**8 071**	**147 326**
(95% UI)		(3 069 - 5 990)	(57 345 - 104 757)		(3 567 - 9 434)	(68 439 - 149 455)		(6 755 - 15 302)	(128 435 - 252 817)
% of total burden	-	**1.3**	**0.6**	-	**1.8**	**0.8**	-	**1.5**	**0.7**
(95% UI)		(1.1 - 2.2)	(0.6 - 1.0)		(1.4 - 3.7)	(0.7 - 1.4)		(1.3 - 2.9)	(0.6 - 1.2)

DALYs = disability-adjusted life years; AF = attributable fraction based on the numbers of attributable deaths; UI = uncertainty interval.

Table 3. Number of attributable deaths due to high sodium intake and the proportion of all-age deaths by population group in South Africa for 2000, 2006 and 2012

Year	Black African, *n* (%)	Coloured, *n* (%)	White, *n* (%)	Indian/Asian, *n* (%)	National, *n* (%)
Male					
2000	2 569 (1.2)	291 (1.7)	791 (3.6)	177 (3.6)	3 828 (1.4)
2006	2 847 (1.0)	330 (1.6)	858 (3.5)	235 (3.9)	4 270 (1.2)
2012	2 680 (1.2)	204 (1.0)	502 (2.1)	133 (2.6)	3 519 (1.3)
Female					
2000	3 277 (1.6)	343 (2.5)	835 (4.1)	148 (4.0)	4 603 (1.9)
2006	3 863 (1.3)	390 (2.3)	870 (3.9)	180 (4.0)	5 303 (1.6)
2012	3 696 (1.7)	233 (1.4)	517 (2.4)	106 (2.5)	4 552 (1.8)

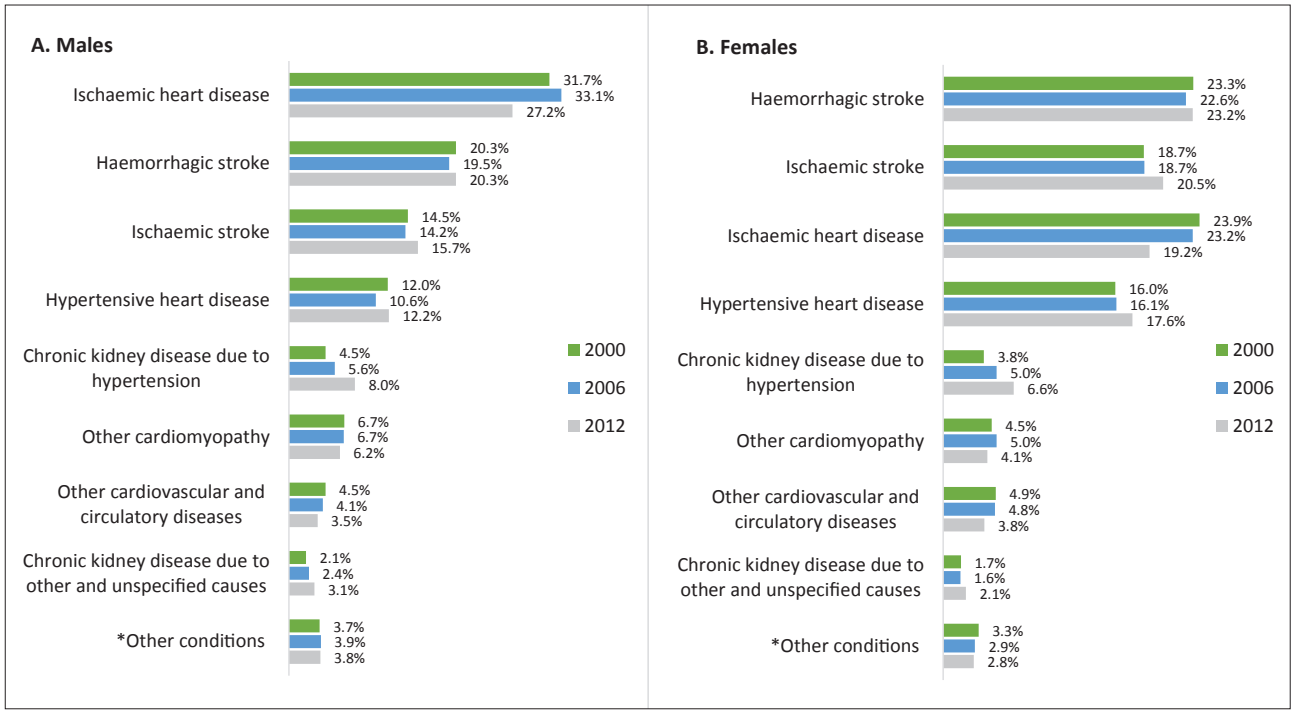

*Fig. 2. Percentage of deaths attributable to high sodium intake by disease outcome for (A) males and (B) females aged ≥25 years in South Africa for 2000, 2006 and 2012. (*Other conditions include chronic kidney disease due to glomerulonephritis, peripheral vascular disease, aortic aneurysm, atrial fibrillation and flutter, endocarditis, chronic kidney disease due to diabetes mellitus, and rheumatic heart disease.)*

Using the NSP target of reducing sodium intake to 2 g/day for 2012 would lower the attributable deaths by 2 823 for females and 2 304 for males, or a total of 5 128 fewer attributable deaths for persons (Table 4).

Discussion

In 2012, a high sodium intake in SA was estimated to cause 8 071 (95% UI 6 542 - 15 474) deaths and 147 326 (95% UI 128 435 - 252 817) DALYs. The burden attributable to high sodium intake increased between 2000 and 2006 and then dropped in 2012 when measured in absolute numbers, as well as age-standardised rates. This is in the context of a rapidly changing cause of death profile in SA,[55] with the rapid increase of HIV/AIDS until 2006 and the decrease following the roll-out of antiretroviral treatment in the mid-2000s, together with differing trends in non-communicable diseases (NCDs) including a decline in the rate of cardiovascular diseases since 2003.[1] The present study found that over time, kidney disease is contributing more to the disease burden due to high sodium, while cardiovascular disease is decreasing. From 2000 to 2012, ischaemic heart disease accounted for the largest number of deaths from cardiovascular diseases in SA, attributed to the SBP-raising effect of high sodium intake, followed by haemorrhagic stroke. Attributable deaths and DALYs for women were consistently higher than for men throughout the period.

It is interesting to note that based on the age-standardised mortality rates, there has been a 22 - 23% decline in deaths attributable to high sodium intake between 2006 and 2012, even before the introduction of salt reduction regulations. This can be seen in a study by Newson *et al.*,[56] which indicated that nearly 30% of the participants had already decreased their salt intake over the previous 6 months. It is possible that improved diagnosis of hypertension through primary healthcare resulted in patients being guided to reduce salt intake, as well as a population shift in the use of salt with growing awareness of 'Use salt sparingly', as promoted and implemented nationally in the food-based dietary guidelines since the early 2000s.[57-59] Reducing salt intake is a recommendation in the national hypertension guidelines,[60] but there is little information about its implementation. While the decline in sodium intake suggested by Swanepoel *et al.*[27] appears to be confirmed by the WHO Study on Global AGEing and Adult Health (SAGE)[6] conducted in 2016, which

Table 4. Attributable deaths and DALYs in South Africa for 2012 if the NSP target* were met

	Males			Females			Persons		
Disease outcome	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n***	**DALYs, *n***	**AF, %**	**Deaths, *n*†**	**DALYs, *n***
Ischaemic heart disease	2.4	316	2 101	2.9	332	4 242	2.6	648	6 343
Haemorrhagic stroke	2.8	242	2 829	3.1	391	5 079	3.0	633	7 907
Ischaemic stroke	3.0	207	1 331	3.3	378	1 301	3.2	585	2 632
Hypertensive heart disease	2.3	150	91	2.5	299	217	2.4	449	308
Chronic kidney disease due to hypertension	2.7	101	928	2.8	113	1 432	2.7	213	2 359
Other cardiomyopathy	2.3	72	86	2.4	67	54	2.3	139	140
Other cardiovascular and circulatory diseases	2.5	43	117	2.7	65	92	2.6	108	209
Chronic kidney disease due to other and unspecified causes	2.5	38	680	2.6	36	769	2.6	74	1 449
Atrial fibrillation and flutter	2.7	8	3770	2.8	12	6 059	2.8	20	9 829
Chronic kidney disease due to glomerulonephritis	2.2	11	2 058	2.2	7	2 606	2.2	18	4 664
Peripheral vascular disease	2.0	8	101	1.9	6	65	2.0	14	166
Rheumatic heart disease	1.7	4	0	1.8	8	0	1.8	12	0
Aortic aneurysm	2.0	7	438	2.1	4	432	2.1	12	869
Chronic kidney disease due to diabetes mellitus	2.4	4	868	2.6	7	982	2.5	11	1 850
Endocarditis	2.2	5	4 858	2.4	3	5 027	2.3	8	9 885
Total attributable burden	-	**1 214**	**20 346**	-	**1 729**	**28 524**	-	**2 943**	**48 870**
(95% UI)		(892 - 3 734)	(15 306 - 60 159)		(841 - 6 961)	(18 406 - 100 037)		(1 916 - 10 547)	(34 910 - 157 862)
% of total burden	-	**0.4**	**0.2**	-	**0.7**	**0.3**	-	**0.6**	**0.2**
(95% UI)		(0.3 - 1.4)	(0.2 - 0.6)		(0.3 - 2.8)	(0.2 - 1.0)		(0.4 - 2.0)	(0.2 - 0.8)
Difference between national estimated attributable burden for 2012 based on TMREL and NSP target	-	2 304	44 991	-	2 823	53 465	-	5 128	98 456

DALYs = disability-adjusted life years; NSP = National Strategic Plan for the Prevention and Control of Non-communicable Diseases;[29] AF = attributable fraction based on the numbers of attributable deaths; UI = uncertainty interval; TMREL = theoretical minimum risk exposure level.
*2 g/d (87 mmol/d).
†Deaths ranked by persons (highest to lowest).

found a median sodium intake of 2.7 g/day, there are limited data on sodium intake during the study period. Data from Wave 3 of SAGE demonstrated a drop in salt intake following the interim phase of mandatory sodium legislation between 2015 (pre) and 2018 - 19 (post) of 1.15 g/day (p=0.028). No further studies have been conducted following the implementation of the stricter phase of salt targets (July 2019).[61]

Despite the decrease in age-standardised attributable death rates, the present study estimated that in 2012, 1.5% (n=8 071) of total deaths and 0.7% (n=147 325) of DALYs were attributed to the SBP-raising effect of high sodium intake among adults aged ≥25 years. The attributable burden was higher for females than for males, accounting for 1.8% (n=4 552) of female deaths and 0.8% (n=91 988) of female DALYs, whereas the figures for males were 1.3% (n=3 519) of deaths and 0.6% (n=65 337) of DALYs. Our country estimate is higher than the GBD 2019[62] estimates for SA, which may result from a different approach to account for the large uncertainty in the TMREL, but are somewhat lower than the global estimates from GBD 2019 that 3.3% of global deaths and 1.7% of global DALYs were attributable to high intake of sodium. The populations of countries such as China, Japan and Thailand have extremely high sodium intake, resulting in this being the leading dietary risk factor there.[14]

Our study confirmed that, despite the decrease in burden attributable to high sodium intake observed between 2006 and 2012, if the NSP target were to be met, there would be a further reduction in disease burden. We estimated that 2 943 deaths and 48 870 DALYs for both males and females could potentially have been prevented if the set target had been met (Table 4). Furthermore, modelling by Watkins *et al.*[63] demonstrates that successful reduction in sodium intake will result in cost savings of up to ZAR300 million a year in healthcare costs, as well as reducing impoverishment by preventing out-of-pocket spending and catastrophic health expenditure affecting the middle class.[64] In addition to legislation to address non-discretionary sodium intake, the promotion of consumer awareness and education through mass media campaigns targeting the general population and vulnerable groups have shown significant positive change in behaviour regarding sodium intake in other settings.[65,66] However, some research has indicated that this approach is costly.[65]

The limitations of this study include the sparsity of the national data on dietary

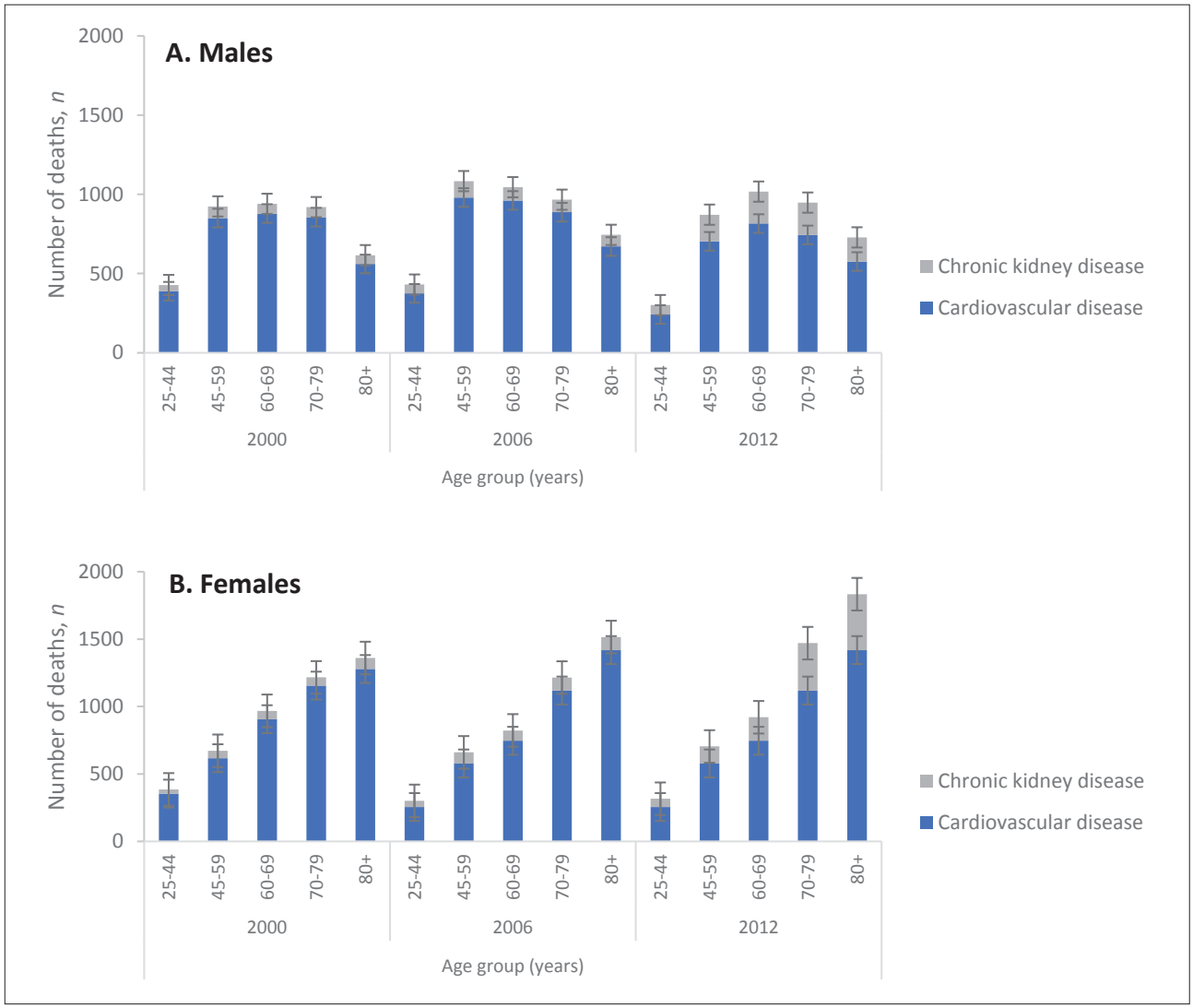

Fig. 3. Age distribution of high sodium attributable deaths for (A) males and (B) females in South Africa for 2000, 2006 and 2012.

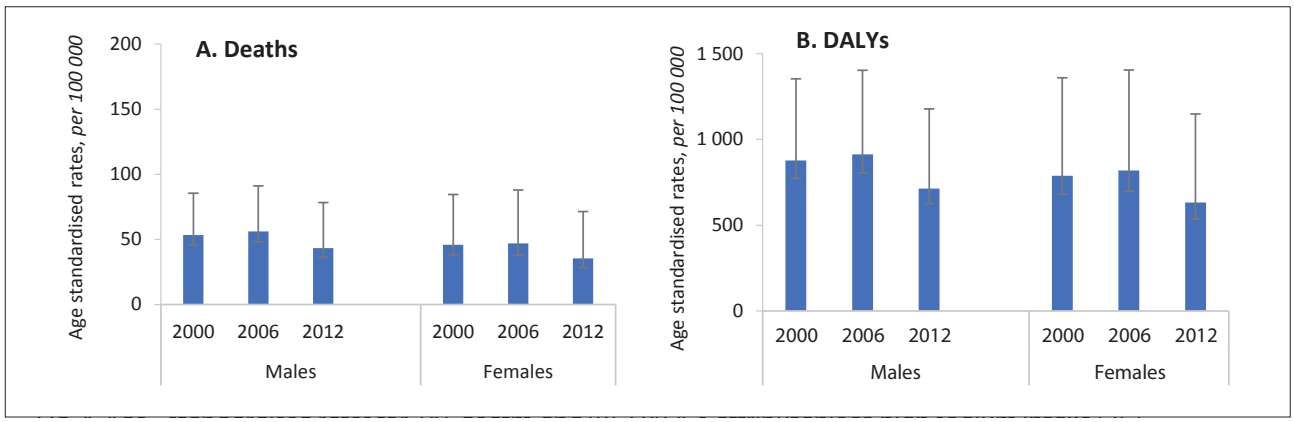

Fig. 4. Age-standardised rates for (A) deaths and (B) DALYs attributable to high sodium intake (≥25 years), by sex in South Africa for 2000, 2006 and 2012.

sodium intake to calculate mean sodium intake for SA. It was therefore not possible to estimate the contribution of stomach cancer that could be attributed to high sodium intake, as this requires an estimate of national sodium intake. This lack of data highlights the need to establish national surveillance and monitoring systems for sodium intake.[67] Our data depended on extrapolating the quantitative effects of high sodium intake on BP, and the GBD RR values for systolic BP on cardiovascular diseases were directly obtained from Cochrane studies and the GBD study (mainly meta-analysis), which does not necessarily represent the risk of cardiovascular disease outcomes for the SA population. However, the extrapolation of GBD RR from the meta-analysis may not affect the results of longitudinal comparison within the same region, as well as horizontal comparison in a population with

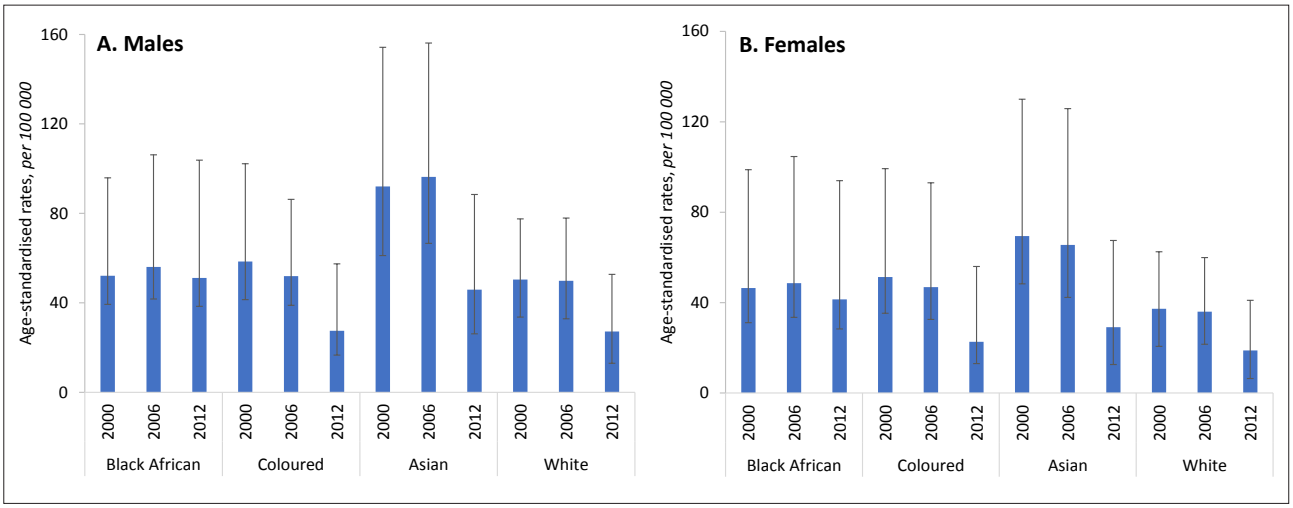

Fig. 5. Age-standardised death rates by population group for (A) males and (B) females in South Africa for 2000, 2006 and 2012.

similar characteristics. Although many of these dietary RRs have been adjusted for the major confounders (e.g. age, sex, smoking and physical activity), the possibility of residual confounding cannot be excluded. Despite the literature reporting uncertainty regarding the sodium intake level considered to represent no health risk,[68] our study has not allowed for this uncertainty. Instead, we estimated the attributable burden against the lowest level (1 g/day) and then assessed a second scenario based on meeting the NSP target (2 g/day). The impact of simultaneously increasing potassium while lowering sodium was not investigated in the current analysis, since it was beyond the scope of the study.

The strength of this study includes the use of publicly available nationally representative surveys on the prevalence of hypertension that include both self-reported information on use of medication and standard measurement of systolic blood pressure and diastolic blood pressure disaggregated by age, sex and population group over time. The use of GBD methodology allows comparisons under similar conditions. The UIs of the results incorporate the uncertainty of many inputs, including the uncertainty of risk exposure and RRs, but exclude some sources such as the uncertainty of the burden estimate. Furthermore, a degree of caution is necessary when interpreting results from a modelling approach that is dependent on multiple assumptions.

Tracking sodium intake and the prevalence of hypertension are an essential component of NCD surveillance and will contribute to an understanding of the effectiveness of salt legislation, as well as provide information needed to develop policies and programmes to reduce the impact of high BP. However, the national NCD surveillance system is currently underdeveloped. Ideally, sodium intake should be monitored at a population level using 24-hour urinary excretion, considered the 'gold standard' from a methodological perspective because it captures intake from salt added at the table and during cooking, in addition to that in foods consumed. The logistics of collecting such data from a nationally representative sample of the population are onerous. Neither food frequency questionnaires nor spot urine samples, which would be much easier to collect, are recommended.[69] Sentinel surveillance may be a more practicable approach.

It will also be important to monitor the sodium content of processed foods to ensure that the regulations are implemented. These data can also be used to update food composition tables. Since it is not feasible to monitor every food product on the market, the Centers for Disease Control recommends a system of sentinel food surveillance.[70] This will involve selecting a list of processed foods and a complete nutrient profile for each product listed to provide baseline assessments for sodium consumption. Finally, research directed towards a better understanding of the salt taste mechanism may lead to the identification of more effective salt substitutes, other flavour enhancers, or methods to trigger the salt taste sensation using smaller amounts of sodium chloride. Health promotion activities should aim to improve the sodium-to-potassium ratio through increased intake of vegetables and fruit.[71,72] It has been found that a major proportion of South Africans do not consume enough potassium.[27,37]

Conclusion

This study has highlighted that high sodium intake resulted in a sizeable burden of disease in 2012. Sodium reduction regulations, implemented in phases in 2016 and 2019, are an effective strategy to help prevent and control hypertension and cardiovascular diseases, and can be expected to contribute towards a reduction of the attributable burden. The high discretionary salt intake (41%)[24] by South Africans means that a population awareness campaign in parallel with legislation is essential. In addition, further research to understand how food labelling policies can be best used in SA is important. Surveillance and monitoring activities should be integrated into planned surveys and NCD surveillance to assess whether the intended outcomes of the salt regulations have been achieved. Our study highlights the need for nationally representative population-based data on sodium intake. We found that several national surveys have measured BP and collected data on hypertension, which we have used to track trends and provide limited demographic disaggregation; however, the surveys need to apply a standardised methodology and to be conducted regularly. Efforts to provide contextualised national estimates for the trends in high BP and hypertension and sodium intake should be a priority to monitor progress on the NCD targets.

Disclaimer. The population group classification used in this article is based on self-reporting according to apartheid-era groups defined by the Population Registration Act of 1950, i.e. black African, coloured, Indian/Asian and white. This classification is used because it has important correlates with lifestyle, culture and socioeconomic conditions that impact on health and health-related behaviours. The authors do not subscribe to this classification for any other purpose.

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Author contributions. Conceived and designed the study: BN, RP, DB, VPvW. Analysed the data: NA, AC. Prepared data for analysis: AC. Interrogated and interpreted results: all. Drafted manuscript: BN, NA, VPvW, DB. Critical review of manuscript: all. Senior authors: DB, VPvW, RP. Agreed to final version: all.

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Conflicts of interest. None.

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