

Psychological Distress and the Development of Hypertension Over 5 Years in Black South Africans

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Alarming increases in the incidence of hypertension in many low- and middle-income countries are related to alcohol overuse. It is unclear whether alcohol overuse is a symptom of psychological distress. The authors assessed psychological distress in Africans and its relationship with a 5-year change in blood pressure (BP), independent of alcohol intake. The authors followed 107 Africans with optimal BP ($\leq 120/80$ mm Hg) (aged 35–75 years) over 5 years. Alcohol intake (self-report and serum γ -glutamyl transferase) and nonspecific psychological distress (Kessler Screening Scale for Psychological Distress [K6]) were assessed. The K6

predicted hypertension development ($P=.019$), and its individual component “nervous” increased a participant’s risk two-fold to become hypertensive (hazard ratio, 2.00 [1.23–3.26]). By entering K6 and γ -glutamyl transferase into multivariable-adjusted regression models for change in systolic BP, both were independently associated with change in systolic BP. Psychological distress and scoring high on being nervous predicted the development of hypertension over 5 years, independent of alcohol intake. *J Clin Hypertens (Greenwich)*. 2015;17:126–133. © 2014 Wiley Periodicals, Inc.

The importance of psychological distress in the development of cardiovascular disease (CVD) is underscored by an enormous amount of literature.¹ Over the past decade, studies integrating psychological distress into concrete physiological mechanisms in CVD development are providing convincing evidence for this link,² especially with elevated sympathetic nervous system activity at play.³ However, the biomedical community remains skeptical⁴ and often downplays the contributory role of or neglects to incorporate psychological distress in CVD research. This is probably the result of the challenges in accurately defining and assessing psychological distress exposure, which is mainly being performed by the use of psychological questionnaires.

The Kessler Screening Scale for Psychological Distress (K6), known to measure global nonspecific psychological distress, was proven to be reliable and valid in the US National Health Interview Surveys, as well as the World Health Organization World Mental Health Surveys.^{5,6} The good psychometric properties of this six-item scale across major sociodemographic subsamples make it ideal to use in general-purpose health surveys.

The relationship between psychological distress and CVD development may, however, be clouded by altered behaviors,^{4,7,8} which may have an independent effect on cardiovascular outcome.⁹ Changes in health behaviors occurring as adaptations or coping responses to

stressors, such as increased smoking or excessive alcohol use, provide an important pathway through which stressors influence CVD risk.⁴

We and others have demonstrated^{10–12} that alcohol intake (self-report or γ -glutamyl transferase) predicts hypertension development in black South Africans with low socioeconomic status. Furthermore, the average consumption per drinker in sub-Saharan Africa (19.5 L) is among the highest in the world.¹³ The pattern of drinking in South Africa is also one with high potential for causing health or social harm.¹⁴ Whether alcohol intake is a surrogate marker of psychological distress in this poverty-stricken population remains to be addressed. We therefore aimed to determine whether the short K6 screening scale for psychological distress (and its individual items) is predictive of 5-year elevation in blood pressure (BP) in 107 Africans, and whether such a relationship is dependent on alcohol intake.

METHODS

Study Population

This longitudinal study forms part of the multinational Prospective Urban and Rural Epidemiology (PURE) study.¹⁵ In the 2005 baseline data collection of the South African leg (North West Province), we were able to obtain BP data from 1995 African men and women (aged >35 years) from a sample of 6000 randomly selected households in rural and urban areas (Figure 1). We performed the first follow-up data collection in 2010 and included 1246 individuals (with BP data), wherein 233 died—giving a 74% successful follow-up rate.

This particular substudy included participants with optimal BP (≤ 120 mm Hg and/or 80 mm Hg) at baseline

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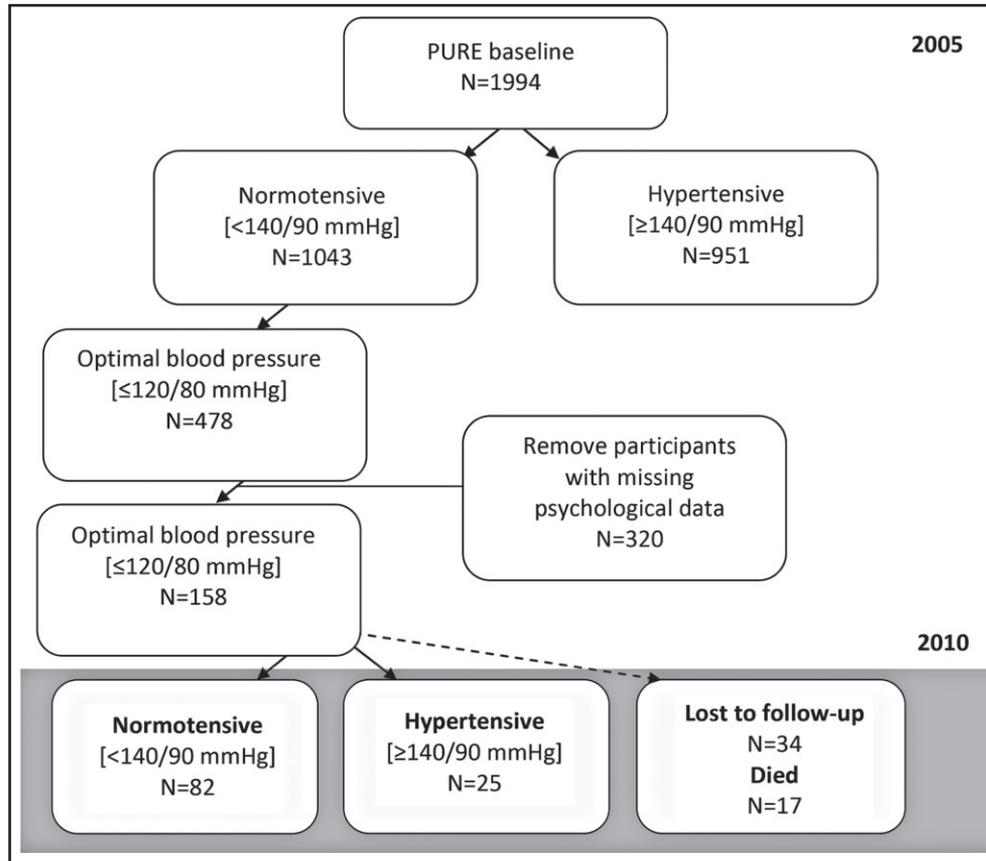


FIGURE 1. Outline of the study population. PURE indicates Prospective Urban and Rural Epidemiology study.

(N=478) and classified as either normotensive or hypertensive after 5-year follow-up.¹⁰ As shown in Figure 1, data on psychological distress were collected in a subsample (n=693) from the total PURE study, resulting in a total of 107 individuals with optimal baseline BPs, psychological distress data, and follow-up data.

Participants were fully informed about the objectives and procedures of the study prior to their first recruitment. Trained African fieldworkers fluent in both English and Setswana assisted and conveyed all information in the participants' home language. All participants gave written informed consent.

The study complied with all applicable requirements of US and international regulations, in particular the Helsinki declaration of 1975 (as revised in 2004) for investigation of human participants. The ethics committee of the North-West University (Potchefstroom Campus) approved the study.

Questionnaires

African fieldworkers interviewed participants using structured demographic, socioeconomic, lifestyle, and psychological questionnaires that were developed and standardized for the international PURE study.¹⁵ In a

subsample of 693 participants of the PURE study group, the K6 was used to measure global psychological distress.⁵ The scale was culture-sensitively translated into Setswana with implementation of the Brislin (1973) translation and back-translation method and the research committee approach for comparison. It was administered by trained Setswana-speaking fieldworkers. This six-item scale measures nonspecific psychological distress including symptoms of depression, anxiety, and restlessness.^{5,16} Respondents reported on a 5-point Likert scale (ranging from 1 to 5) how often during the preceding 30 days they felt each of the following: "so sad nothing could cheer you up," "nervous," "restless or fidgety," "hopeless," "that everything was an effort," "worthless." The scores for all items were then summed, ranging from 6 to 30, with a higher score reflecting a higher degree of psychological distress. A cutoff of 19 was considered probable serious mental illness.⁶

Anthropometric Measurements

Height, weight, and waist circumferences were measured by accredited anthropometrists using standardized methods with calibrated instruments (Precision Health Scale, A&D Company, Toshima, Tokyo; Leicester Height Measure, Seca, Birmingham, UK).

Cardiovascular Measurements

After a 10-minute rest period, BP measurements were performed in duplicate (5 minutes apart) on the right upper arm, while the participants were seated upright with the right arm supported at heart level. Systolic BP (SBP), diastolic BP (DBP), and heart rate were measured with the validated OMRON HEM-757 device (Omron Healthcare, Kyoto, Japan), and appropriate-sized cuffs were used for obese participants.

Blood Sampling

Participants were requested to fast for approximately 10 hours prior to sample collection in the mornings. A registered nurse obtained a blood sample with a sterile winged infusion set from the antebraial vein branches. Samples were prepared according to appropriate methods and stored at -80°C in the laboratory. In the rural area, samples were rapidly frozen and stored at -18°C (no longer than 5 days) until they could be transported to the laboratory facility and then stored at -80°C until analysis.

Biochemical Measurements

Fasting sodium fluoride (glucose) and serum samples for total and high-density lipoprotein cholesterol (HDL-C), γ -glutamyl transferase, and high-sensitivity C-reactive protein (CRP) were analyzed using two sequential multiple analyzers (Konelab 20i, Thermo Scientific, Vantaa, Finland; Cobas Integra 400 Plus, Roche, Switzerland). Glycosylated hemoglobin was determined onsite in EDTA-treated whole blood using the D-10 Hemoglobin Testing System (Bio-Rad Laboratories, Hercules, CA). We determined HIV status according to the protocol of the South African Department of Health by using the First Response rapid HIV card test (PMC Limited, Bangalore, India). The result was confirmed with the SD Bioline HIV 1/2 3.0 card test (Standard Diagnostics, Inc, Gyeonggi-do, Korea).

Statistical Analyses

We compared the characteristics of participants (means and proportions) by independent *t* test and the chi-square test, respectively. Variables with a non-Gaussian distribution were logarithmically transformed. We used Pearson correlation coefficients to determine the intercorrelations between K6 and the six scale items. We also employed Pearson correlations to determine relationships between the 5-year percentage change in BPs (% SBP and %DBP) with measures of alcohol intake and the K6. Analyses of variance and covariance were used to plot and compare K6 quintiles against %SBP before and after adjustments for confounders. Using Cox regression analyses, we determined multivariable-adjusted hazard ratios for the development of hypertension after 5 years for the K6 and individual scale items. Lastly, we investigated independent associations of K6 and γ -glutamyl transferase with %SBP by using multiple linear regressions.

RESULTS

We included 107 black men and women who had optimal BP at baseline ($<120/80$ mm Hg) and with complete datasets at baseline and follow-up (Figure 1). Of these individuals, 23% developed BP in the hypertensive range after 5 years, indicating a $31.6\% \pm 17.4\%$ increase in SBP during this period (Table I). Thirteen percent of those who remained normotensive had K6 scores exceeding the cutoff of 19 for probable serious mental illness, compared with 33% from those who became hypertensive. This was also evident by an elevated K6 score ($P=.005$), as well as certain scale items, namely “nervous” ($P=.010$) and “restless or fidgety” ($P=.037$) for the participants who became hypertensive. With respect to measures of alcohol intake, we found that γ -glutamyl transferase concentrations were higher in the hypertensive group ($P=.002$), supported by self-reported alcohol intake ($P=.058$).

When viewing the performance of the K6 screening scale, we found that the internal reliability of the K6 was poor in this study, with a Cronbach α of 0.48. We evaluated the intercorrelations of K6 and its six scale items (Table II) and found that K6 correlated well with all scale items, but, in general, the intercorrelations between the six items were weak and thus reflected in a low Cronbach α that is based on internal homogeneity. Most of the subsequent statistical analyses were thus performed for K6 as well as the individual scale items.

In line with our aim we assessed the relationship between %BP, alcohol intake, and measures of psychological distress (K6 and its scale items) (Table III). We found that %SBP correlated best with K6 total score ($r=0.35$; $P=.001$) (Figure 2) and the nervous scale item ($r=0.24$; $P=.013$) and also with γ -glutamyl transferase ($r=0.22$; $P=.028$). Self-reported alcohol intake correlated positively with K6 ($r=0.23$; $P=.027$).

We plotted quintiles of K6 against the 5-year change in %SBP before and after adjustments for age, waist circumference, baseline SBP, and γ -glutamyl transferase (Figure 2). A significant trend ($P \leq .001$) was shown with %SBP, before and after adjustments. The fifth K6 quintile exceeded the cutoff of 19 and presented a % SBP higher than those of K6 quintiles 1, 2, and 3.

In multivariable-adjusted Cox proportional hazard models (Figure 3), we determined the separate contributory roles of K6 and each of the six scale items to hypertension development (normotensive vs hypertensive at follow-up). We found that although K6 predicted hypertension significantly ($P=.019$), scoring high on being nervous increased a participant's risk two-fold in becoming hypertensive over the 5-year period (hazard ratio, 2.00 [1.23–3.26]).

Finally, in multivariable regression analyses (Table IV), we evaluated the independent associations of %SBP with either K6 (model 1) or γ -glutamyl transferase (model 2) in the model. In both models, either K6 ($P=.001$) or γ -glutamyl transferase ($P=.019$) was independently associated with %SBP. When both

TABLE I. Baseline Characteristics of Participants With Optimal Blood Pressure ($\leq 120/80$ mm Hg), Remaining Normotensive ($<140/90$ mm Hg), or Becoming Hypertensive (≥ 140 and/or 90 mm Hg) After 5 Years (N=107)

	Normotensive at Follow-Up	Hypertensive at Follow-Up	P Value
No	82	25	
Age, y	47.8 \pm 8.36	46.9 \pm 8.25	.61
Women, No. (%)	56 (68.2)	19 (76.0)	.46
Anthropometry			
Height, m	1.60 \pm 0.08	1.59 \pm 0.08	.56
Weight, kg	59.1 \pm 14.6	65.1 \pm 19.9	.10
Body mass index, kg/m ²	23.3 \pm 6.25	25.9 \pm 8.30	.086
Waist circumference, cm	75.4 \pm 12.3	81.1 \pm 14.3	.053
Blood pressure			
Baseline SBP, mm Hg	106 \pm 10.1	109 \pm 7.28	.12
Baseline DBP, mm Hg	70.6 \pm 6.78	73.4 \pm 4.98	.059
Follow-up SBP, mm Hg	116 \pm 12.8	143 \pm 15.3	<.001
Follow-up DBP, mm Hg	77.4 \pm 7.85	95.0 \pm 7.68	<.001
% Change in SBP, mm Hg	9.68 \pm 12.3	31.6 \pm 17.4	<.001
% Change in DBP, mm Hg	10.5 \pm 14.3	30.2 \pm 15.3	<.001
Biochemical measurements			
Glucose, mmol/L	4.87 (3.60–5.90)	4.79 (2.80–7.10)	.77
Glycated hemoglobin, %	5.58 (4.50–6.50)	5.71 (4.90–6.70)	.44
C-reactive protein, mg/L	2.17 (0.10–48.1)	3.52 (0.31–24.5)	.24
HDL cholesterol, mmol/L	1.39 \pm 0.50	1.60 \pm 0.74	.12
γ -glutamyl transferase, U/L	37.8 (16.6–200)	66.1 (24.6–366)	.002
Self-reported alcohol intake, No. (%)	20 (24.4)	11 (44.0)	.058
Self-reported tobacco use, No. (%)	42 (51.2)	15 (62.5)	.33
Antihypertensive medication, No. (%)	0 (0)	4 (16.0)	<.001
HIV infected, No. (%)	7 (8.54)	2 (8.00)	.93
Education, No. (%)	51 (63.7)	14 (58.3)	.63
Psychological distress: K6 score			
So sad nothing could cheer you up	16.1 \pm 3.28	18.3 \pm 3.45	.005
Nervous	2.68 \pm 1.10	3.17 \pm 1.13	.062
Restless or fidgety	2.69 \pm 0.96	3.28 \pm 1.06	.010
Hopeless	2.57 \pm 1.07	3.08 \pm 0.99	.037
That everything was an effort	2.58 \pm 0.99	2.88 \pm 1.05	.20
Worthless	3.18 \pm 1.08	3.32 \pm 1.07	.57
	2.40 \pm 1.19	2.52 \pm 1.19	.66

Abbreviations: DBP, diastolic blood pressure; HDL, high-density lipoprotein; HIV, human immunodeficiency virus; K6, Kessler Screening Scale for Psychological Distress; SBP, systolic blood pressure. Data are presented as arithmetic mean \pm standard deviation or geometric mean (5th and 95th percentile intervals) for logarithmically transformed variables.

K6 and γ -glutamyl transferase were entered into the model (model 3), significance for γ -glutamyl transferase became weaker ($R^2=0.21$; $\beta=0.18$; $P=.058$), whereas K6 remained significant ($R^2=0.21$; $\beta=0.30$; $P=.002$). After adding antihypertensive medication into the model (model 4), the contribution of γ -glutamyl transferase became nonsignificant ($P=.14$). We replaced K6 with the scale item “nervous” in model 5 and also found a significant contribution of this to the model ($\beta=0.20$; $P=.029$), with γ -glutamyl transferase remaining nonsignificant ($P=.14$). In model 6, we inserted the interaction of K6* γ -glutamyl transferase into the model and found that the contribution was stronger ($\beta=0.35$; $P=.001$) than for either of the separate components (models 1–4). A similar stronger result was found for “nervous”* γ -glutamyl transferase ($\beta=0.27$; $P=.004$). In additional models (not shown), we also tested the other five

scale items of K6, but none showed a significant contribution to %SBP.

In sensitivity analyses, we also repeated all multiple regression models by not using forward stepwise regression, but forcing all independent variables into the model, and found similar results. Additional analyses were also performed where γ -glutamyl transferase was replaced with self-reported alcohol intake in the regression models. We found that self-reported alcohol intake did not reach statistical significance (model 2: $R^2=0.10$; $\beta=0.15$; $P=.13$) as was originally found for γ -glutamyl transferase.

DISCUSSION

In 2014, the highest ever reported hypertension prevalence by a nationally representative survey of people aged 50 and older was reported for South Africa, at

TABLE II. Single Linear Regression Analyses Between K6 Score and Scale Items

	K6	So sad nothing could cheer you up	Nervous	Restless or fidgety	Hopeless	That everything was an effort
So sad nothing could cheer you up	r=0.36 P<.001	–				
Nervous	r=0.56 P<.001	r=0.09 P=.35	–			
Restless or fidgety	r=0.58 P<.001	r=0.03 P=.77	r=0.28 P=.004	–		
Hopeless	r=0.61 P<.001	r=0.07 P=.51	r=0.13 P=.20	r=0.22 P=.026	–	
That everything was an effort	r=0.58 P<.001	r=0.11 P=.28	r=0.09 P=.39	r=0.09 P=.36	r=0.31 P=.001	–
Worthless	r=0.50 P<.001	r=–0.14 P=.17	r=0.06 P=.57	r=0.18 P=.070	r=0.23 P=.018	r=0.19 P=.058

Abbreviation: K6, Kessler Screening Scale for Psychological Distress. Bold values denote statistical significance ($P<.05$).

TABLE III. Single Linear Regression Analyses of 5-Year Percentage Change in Blood Pressure and Measures of Alcohol Intake With K6 Score and Scale Items

	%SBP	%DBP	Self-Reported Alcohol Intake	γ -Glutamyl Transferase
K6	r=0.35 P=.001	r=0.22 P=.029	r=0.23 P=.027	r=0.14 P=.19
So sad nothing could cheer you up	r=0.15 P=.12	r=0.14 P=.17	r=0.08 P=.40	r=0.04 P=.69
Nervous	r=0.24 P=.013	r=0.20 P=.039	r=0.11 P=.28	r=0.10 P=.34
Restless or fidgety	r=0.10 P=.33	r=0.05 P=.60	r=0.14 P=.16	r=0.08 P=.45
Hopeless	r=0.14 P=.15	r=–0.03 P=.78	r=0.02 P=.86	r=0.02 P=.88
That everything was an effort	r=0.15 P=.13	r=0.10 P=.31	r=0.12 P=.24	r=0.08 P=.46
Worthless	r=0.22 P=.028	r=0.12 P=.24	r=0.14 P=.16	r=0.11 P=.28
Self-reported alcohol intake, no/yes	r=0.15 P=.12	r=0.15 P=.13	–	r=0.32 P=.001
γ -glutamyl transferase (log), U/L	r=0.22 P=.028	r=0.24 P=.018	r=0.32 P=.001	–

Abbreviations: DBP, diastolic blood pressure; K6, Kessler Screening Scale for Psychological Distress; SBP, systolic blood pressure. Bold values denote statistical significance ($P<.05$).

78%.¹² This highlights the urgency to address hypertension in low- and middle-income countries where awareness, treatment, and control are poor.^{12,17} In agreement with our previous results,^{10,11} Lloyd-Sherlock and colleagues¹² indicate that excessive alcohol use is a particularly important health behavior relating directly to the development of hypertension in low- and middle-income countries. Although proactive public health interventions at a population level are advocated to curb the hypertension epidemic,^{18,19} we need to understand why certain health behaviors, such as excessive alcohol use, are driving the hypertension epidemic. It is generally accepted that in sub-Saharan

Africa, the overuse of alcohol accompanying urbanization is the result of ease of access, but our findings indicate that excessive alcohol use may also be a symptom of psychological distress in this community.

We found that despite poor internal reliability of the K6 in this population, both the K6 as a measure of nonspecific psychological distress and the individual scale item referring to nervousness, predicted a 5-year elevation in BP. Of significance in this context is that these associations occurred independently of γ -glutamyl transferase—a marker of alcohol use.⁹ When markers of psychological distress were not taken into account, we found that alcohol use did predict hypertension

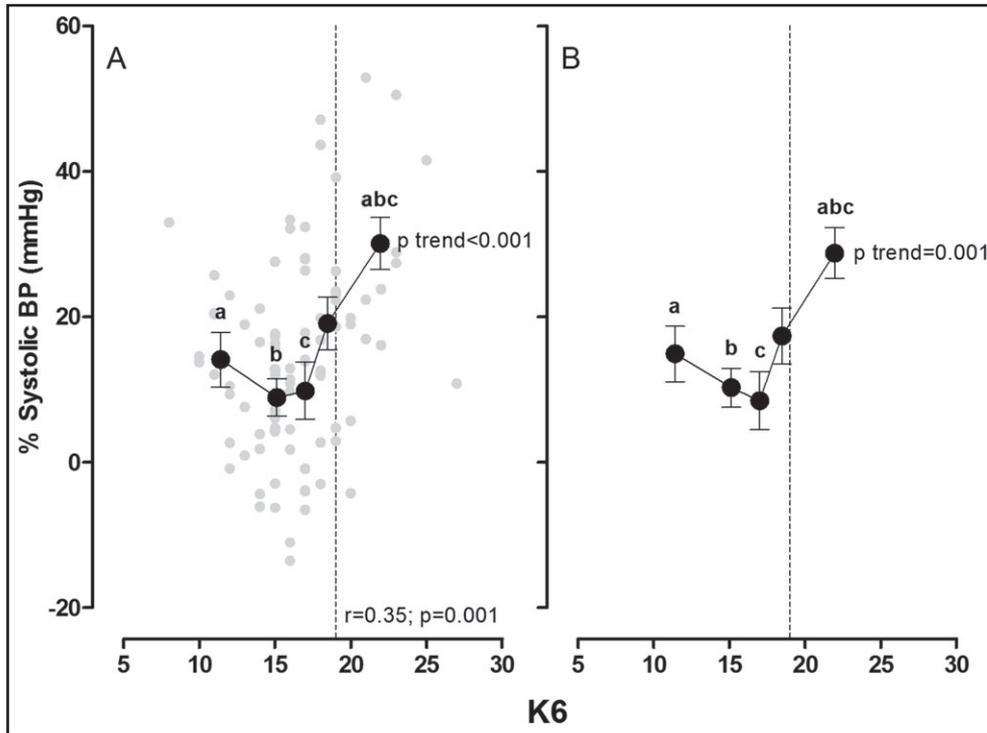


FIGURE 2. Total score of Kessler Screening Scale for Psychological Distress (K6) against 5-year percentage change in systolic blood pressure before (A) and after adjustments (B) for age, waist circumference, baseline systolic blood pressure (BP), and γ -glutamyl transferase. Dashed lines represent the K6 cutoff of 19 for probable serious mental illness. Quintiles with the same superscript letters differ significantly ($P < .05$).

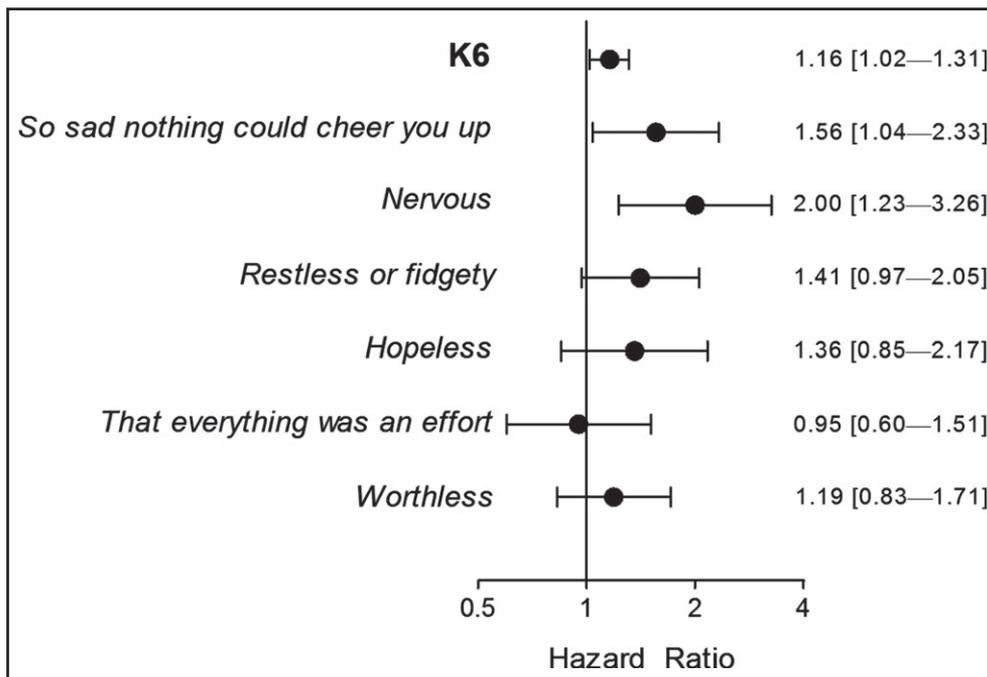


FIGURE 3. Multivariable-adjusted hazard ratios (95% confidence interval) for the development of hypertension after 5 years for the Kessler Screening Scale for Psychological Distress (K6) and each individual scale item. Each model includes the following covariables: age, waist circumference, baseline systolic blood pressure, high-density lipoprotein cholesterol, γ -glutamyl transferase, fasting glucose, and HIV status.

TABLE IV. Forward Stepwise Multivariable Regression Analyses With Percentage Change in SBP as Dependent Variable

	Percentage of SBP Over 5 Years		
	Adjusted R^2	β (SE)	P Value
Model^a 1: K6	0.18		
K6		0.33 (0.10)	.001
Model 2: γ -glutamyl transferase	0.13		
γ -glutamyl transferase (log), U/L		0.23 (0.09)	.019
Model 3: K6 + γ -glutamyl transferase	0.21		
K6		0.30 (0.10)	.002
γ -glutamyl transferase (log), U/L		0.18 (0.10)	.058
Model 4: K6 + γ -glutamyl transferase + antihypertensive medications	0.22		
K6		0.26 (0.10)	.010
γ -glutamyl transferase (log), U/L		0.15 (0.10)	.14
Model 5: <i>Nervous</i> + γ -glutamyl transferase + antihypertensive medications	0.20		
<i>Nervous</i>		0.20 (0.10)	.029
γ -glutamyl transferase (log), U/L		0.14 (0.10)	.14
Model 6: K6* γ -glutamyl transferase + antihypertensive medications	0.24		
K6* γ -glutamyl transferase (log), U/L		0.35 (0.10)	.001
Model 7: <i>Nervous</i> * γ -glutamyl transferase + antihypertensive medications	0.22		
<i>Nervous</i> * γ -glutamyl transferase (log), U/L		0.27 (0.10)	.004
Abbreviations: K6, Kessler Screening Scale for Psychological Distress; SBP, systolic blood pressure. Bold values denote statistical significance ($P < .05$). ^a All models include the following covariables: age, waist circumference, glycated hemoglobin (log), high-density lipoprotein cholesterol, HIV status, and baseline SBP.			

development, thereby confirming other studies.^{20,21} However, this result became nonsignificant when psychological distress was included in the statistical model. The interrelatedness of alcohol overuse as a behavioral change occurring as an adaptation or coping response to psychological distress⁴ emerged when we evaluated the interaction of alcohol intake and psychological distress in our prediction model, and found that this interaction term predicted BP elevation stronger than either a measure of psychological distress or alcohol intake alone.

Our findings suggest that in the efforts to curb the increasing burden of hypertension in South Africa, we need to be aware that environmental or other social factors in poverty-stricken areas may have an impact on increased psychological distress, which may hamper interventions to improve health behaviors. With alcohol use reflecting psychological distress,²² one may argue that the roots of psychological distress require further

investigation. In the South African environment, many factors may contribute to anxiety, nervousness, and elevated psychological distress, where the latest figures indicate that 25% of South Africans are unemployed.²³ Moreover, South Africa is a country experiencing a quadruple burden of epidemic infectious diseases, a rise in noncommunicable diseases, a heavy burden of perinatal and maternal disorders, injury, and violence.²⁴ Rapid urbanization of individuals seeking employment may further enhance social isolation. This could be particularly important since previous studies have found social isolation and the lack of quality social support to be independent risk factors for CVD.²⁵ Although studies reporting the link between psychological distress and CVD are very limited in Africa, a cross-sectional study in school teachers in South Africa also reported higher psychological distress in hypertensive teachers, as well as a tendency to develop left ventricular hypertrophy.²⁶ The first point of action may thus be to address the wide-ranging roots of psychological distress as a predictor of hypertension development. However, more research within the affected communities is needed to pinpoint the critical areas requiring intervention.

Addressing health behaviors without addressing the potential underlying psychological distress may seem counterproductive, but interventions such as tax increases for tobacco control were highly effective in South Africa, where a 33% reduction in tobacco use was found after substantial tax increases in the 1990s.²⁷ Focusing directly on interventions to reduce alcohol abuse may also be effective, but it may be a more daunting task because of the popularity of home-brewed alcohol particularly in poverty-stricken communities.²⁸ The burden of alcohol abuse may therefore be a significant challenge to address.

STUDY STRENGTHS AND LIMITATIONS

The findings of this study should be interpreted within the context of its strengths and limitations. Firstly, participants were selected from specific communities and may not be representative of the whole country. Because of our specific subsample within the larger study having complete data on psychological distress, we realize that selection bias cannot be ruled out. Our sample size was relatively small but the dataset for all individuals were complete, spanning the 5-year follow-up and thereby addressing the urgent need for longitudinal studies in Africa.²⁹ We recommend that K6 be validated in larger samples in Africa, as a result of its potential usefulness as shown by the present study, specifically with respect to its association with hypertension. The usefulness of K6 in this population is an aspect that warrants further research in light of the low internal reliability and may reflect culture-specific expressions of distress. The items indicating symptoms of psychological distress as reflected in the six-item scale may indeed all be tapping on psychological distress (depression, anxiety, self-esteem) but cannot necessarily achieve homogeneity and thus the scale cannot be truly

unidimensional. The factors that could affect the Cronbach α value, but cannot be isolated in this case, include language of administration, scale length, and homogeneity of items. Although the scale was translated and administered by Setswana-speaking fieldworkers, some concepts may be difficult to translate. Finally, although our results were consistent after multiple adjustments, we cannot exclude residual confounding.

CONCLUSIONS

We found that nonspecific psychological distress and scoring high on being “nervous” over the past 30 days significantly predicted the development of hypertension in a black population, independent of alcohol intake. Our results support the notion that hypertension development is primarily enhanced by psychological distress and that excessive alcohol use may be a coping mechanism further exacerbating the situation. Strategies to curb the epidemic of alcohol abuse should not be undertaken in isolation, but need to incorporate the potential role of global psychological distress factors and early identification and treatment of possible serious mental illness for prevention of further health complications.

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References

1. Dimsdale JE. Psychological stress and cardiovascular disease. *J Am Coll Cardiol.* 2008;51:1237–1246.
2. Ferketich AK, Binkley PF. Psychological distress and cardiovascular disease: results from the 2002 National Health Interview Survey. *Eur Heart J.* 2005;26:1923–1929.
3. Lambert EA, Lambert GW. Stress and its role in sympathetic nervous system activation in hypertension and the metabolic syndrome. *Curr Hypertens Rep.* 2011;13:244–248.
4. Cohen S, Janicki-Deverts D, Miller GE. Psychological stress and disease. *JAMA.* 2007;298:1685–1687.
5. Kessler RC, Andrews G, Colpe LJ, et al. Short screening scales to monitor population prevalences and trends in non-specific psychological distress. *Psychol Med.* 2002;32:959–976.
6. Kessler RC, Green JG, Gruber MJ, et al. Screening for serious mental illness in the general population with the K6 screening scale: results from the WHO World Mental Health (WMH) survey initiative. *Int J Methods Psychiatr Res.* 2010;19(suppl 1):4–22.
7. Pickering TG. Mental stress as a causal factor in the development of hypertension and cardiovascular disease. *Curr Hypertens Rep.* 2001;3:249–254.
8. Twisk JW, Snel J, Kemper HC, van Mechelen W. Changes in daily hassles and life events and the relationship with coronary heart disease risk factors: a 2-year longitudinal study in 27–29-year-old males and females. *J Psychosom Res.* 1999;46:229–240.
9. Tsai J, Ford ES, Li C, Zhao G. Past and current alcohol consumption patterns and elevations in serum hepatic enzymes among US adults. *Addict Behav.* 2012;37:78–84.
10. Schutte AE, Schutte R, Huisman HW, et al. Are behavioural risk factors to be blamed for the conversion from optimal blood pressure to hypertensive status in Black South Africans? A 5-year prospective study. *Int J Epidemiol.* 2012;41:1114–1123.
11. Zatu MC, van Rooyen JM, Loots du T, et al. Self-reported alcohol intake is a better estimate of 5-year change in blood pressure than biochemical markers in low resource settings: the PURE study. *J Hypertens.* 2014;32:749–755.
12. Lloyd-Sherlock P, Beard J, Minicuci N, et al. Hypertension among older adults in low- and middle-income countries: prevalence, awareness and control. *Int J Epidemiol.* 2014;43:116–128.
13. Roerecke M, Obot IS, Patra J, Rehm J. Volume of alcohol consumption, patterns of drinking and burden of disease in sub-Saharan Africa. *Afr J Drug Alcohol Stud.* 2008;7:1–17.
14. Obot IS. Alcohol use and related problems in sub-Saharan Africa. *Afr J Drug Alcohol Stud.* 2006;5:17–26.
15. Teo K, Chow CK, Vaz M, et al. The Prospective Urban Rural Epidemiology (PURE) study: examining the impact of societal influences on chronic noncommunicable diseases in low-, middle-, and high-income countries. *Am Heart J.* 2009;158:1–7.
16. Kessler RC, Barker PR, Colpe LJ, et al. Screening for serious mental illness in the general population. *Arch Gen Psychiatry.* 2003;60:184–189.
17. Damasceno A, Azevedo A, Silva-Matos C, et al. Hypertension prevalence, awareness, treatment, and control in mozambique: urban/rural gap during epidemiological transition. *Hypertension.* 2009;54:77–83.
18. Ogah OS, Rayner BL. Recent advances in hypertension in sub-Saharan Africa. *Heart.* 2013;99:1390–1397.
19. Peer N, Steyn K, Lombard C, et al. A high burden of hypertension in the urban black population of Cape Town: the cardiovascular risk in Black South Africans (CRIBSA) study. *PLoS ONE.* 2013;8:e78567.
20. Jousilahti P, Rastenyte D, Tuomilehto J. Serum gamma-glutamyl transferase, self-reported alcohol drinking, and the risk of stroke. *Stroke.* 2000;31:1851–1855.
21. Lucas DL, Brown RA, Wassef M, Giles TD. Alcohol and the cardiovascular system: research challenges and opportunities. *J Am Coll Cardiol.* 2005;45:1916–1924.
22. Markman GI, Larimer ME, Neighbors C. The relationship among alcohol use, related problems, and symptoms of psychological distress: gender as a moderator in a college sample. *Addict Behav.* 2004;29:843–848.
23. The World Bank. Unemployment, total (% of total labor force) (modeled ILO estimate). <http://data.worldbank.org/indicator/SL.UEM.TOTL.ZS>. 15-5-2014. Ref Type: Electronic Citation.
24. Mayosi BM, Flisher AJ, Lalloo UG, et al. The burden of non-communicable diseases in South Africa. *Lancet.* 2009;374:934–947.
25. Das S, O’Keefe JH. Behavioral cardiology: recognizing and addressing the profound impact of psychosocial stress on cardiovascular health. *Curr Hypertens Rep.* 2008;10:374–381.
26. Mashele N, van Rooyen JM, Malan L, Potgieter JC. Cardiovascular function and psychological distress in urbanised black South Africans: the SABPA study. *Cardiovasc J Afr.* 2010;21:206–211.
27. Baleta A. Africa’s struggle to be smoke free. *Lancet.* 2010;375:107–108.
28. Onya H, Tessera A, Myers B, Flisher A. Community influences on adolescents’ use of home-brewed alcohol in rural South Africa. *BMC Public Health.* 2012;12:642.
29. Holmes MD, Dalal S, Volmink J, et al. Non-communicable diseases in sub-Saharan Africa: the case for cohort studies. *PLoS Med.* 2010;7:e1000244.