

# Association between self-rated health and incident severe hypertension among men: a population-based cohort study

Shankar A, Wang J J, Rochtchina E, Mitchell P

## ABSTRACT

**Introduction:** Self-rated health (SRH) is a consistent predictor of cardiovascular disease and mortality. However, the intermediate biological mechanisms behind this association are not clear. We examined the longitudinal relationship between SRH and incident severe hypertension.

**Methods:** We studied a population-based cohort of 1,298 participants (mean age 62.5 years, range 49–84 years), at the baseline examination (1992–1994) residing in the Blue Mountains region, west of Sydney, Australia, and re-examined after five years (1997–1999). Main outcome-of-interest was incident severe hypertension (systolic blood pressure [BP] 160 mmHg or above, diastolic BP 100 mmHg or above, or a combination of self-reported hypertension diagnosis and use of antihypertensive medications) among baseline individuals without severe hypertension.

**Results:** Among men, those with fair/poor SRH had significantly higher odds of incident severe hypertension, compared to individuals with excellent SRH. Multivariable odds-ratio (OR) (95 percent confidence intervals [CI]) comparing fair/poor SRH to excellent SRH was 1.93 (1.04–3.56) (p-trend was 0.03). This association was not observed in women comparing fair/poor SRH to excellent SRH: OR 0.96, 95 percent CI 0.57–1.62 (p-trend was 0.70). Subgroup analyses stratified by age, smoking, body mass index, diabetes mellitus and BP categories, supported this male gender-specific pattern of association.

**Conclusion:** This data suggests an association between poor SRH and incident hypertension among men, but not among women. These results suggest that at least part of the previously-reported association between poor SRH and mortality may be mediated by its relation to incident severe hypertension.

**Keywords:** blood pressure, Blue Mountains Eye Study, hypertension, self-rated health, severe hypertension

*Singapore Med J 2008;49(11):860-867*

## INTRODUCTION

Subjective ratings of health (self-rated health [SRH]) are considered as main outcomes in clinical studies,<sup>(1)</sup> and global SRH rating is a standard for quality of life in national public health promotion strategies.<sup>(2)</sup> Poor SRH is a consistent predictor of cardiovascular disease<sup>(3-6)</sup> and mortality<sup>(7-9)</sup> across several populations, as well as hospital admission and nursing home placement in both middle-aged<sup>(10)</sup> and elderly<sup>(6,11,12)</sup> populations. However, biological mechanisms mediating the association between SRH and mortality are not clear. Several previous cross-sectional<sup>(6,13-16)</sup> and case-control<sup>(17,18)</sup> studies have reported an association between poor SRH and hypertension, and among hypertensive subjects, subjects with excellent SRH demonstrated better control of their hypertension.<sup>(19)</sup> The prospective association between SRH and incident hypertension has not been previously examined. In some studies, the association between poor SRH and vascular disease and mortality was stronger among men, compared to women.<sup>(4,9,20-23)</sup> It is not clear if there is any gender difference in the association between SRH and hypertension. In this report, we aim to examine the association between SRH and a five-year incidence of hypertension and severe hypertension among men and women in an older Australian population, after adjusting for smoking, alcohol intake, body mass index (BMI) and other important cardiovascular risk factors.

## METHODS

The Blue Mountains Eye Study (BMES) is a population-based cohort study of age-related eye diseases and other health outcomes in an older urban Australian population. Study details were described previously.<sup>(24)</sup> After a door-to-door census of residents living in two postcodes in the Blue Mountains region, west of Sydney, Australia, persons born before January 1, 1943, were invited to attend a detailed examination at a local hospital. Baseline examination was performed on 3,654 of 4,433 (82.4%)

Department of Community, Occupational, and Family Medicine, Yong Loo Lin School of Medicine, National University of Singapore, 16 Medical Drive, Singapore 117597

Shankar A, MD, PhD  
Associate Professor

Centre for Vision Research, Department of Ophthalmology and Westmead Millennium Institute, University of Sydney, Sydney, NSW 2145, Australia

Wang JJ, MBBS, PhD  
Senior Research Fellow

Rochtchina E, MSc  
Statistician

Mitchell P, MD, PhD  
Professor

Correspondence to:  
Dr Anoop Shankar  
Department of Community Medicine,  
West Virginia University School of  
Medicine,  
1 Medical Center Drive,  
Morgantown, WV 26506-9190,  
USA  
Tel: (1) 304 293 0199  
Fax: (1) 304 293 6685  
Email: ashankar@hsc.wvu.edu

eligible persons during 1992–1994.<sup>(25)</sup> During the five-year follow-up examination in 1997–1999, 2,334 (75.1%) of surviving participants were reexamined. This study followed the recommendations of Declaration of Helsinki and was approved by the Western Sydney Area Human Ethics Committee. Written informed consent was obtained from all participants.

SRH was assessed by asking a question with four possible answers: “For someone of your age, how would you rate your overall health—would you say it is excellent, good, fair, or poor?”<sup>(6)</sup> The baseline and the five-year follow-up examinations followed similar standardised protocols and included measurements of weight, height, pulse rate, systolic and diastolic blood pressures (BP) by a trained interviewer, administering a standardised questionnaire that collected information regarding participants’ demographic characteristics, cigarette smoking, alcohol intake, physical activity, medical histories and medications taken.

Fasting blood specimens were drawn from 3,222 participants (88.2%), centrifuged on-site and then couriered within the same day to Westmead Hospital, Sydney, Australia, for haematology and clinical biochemistry assessments. White blood cell (WBC) count was determined using a Coulter counter method. Reliability coefficients, based on blind replicate control data, ranged from 0.96 to 1.00. Total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C) and triglyceride (TG) concentrations were measured on a Reflotron reflectance photometric analyser (Boehringer Mannheim Diagnostics [currently Roche Diagnostics], Germany). Coefficients of variation for repeated measurements of plasma were 2.9% for TC, 3.2% for HDL-C and 1.4% for TG. Haematocrit and fasting plasma glucose were measured by spun microhaematocrit and hexokinase methods, respectively.

Age was defined as age at the baseline examination; education was categorised into beyond high school, high school and below; home ownership (yes, no) was categorised based on whether the subjects owned their house, flat, or independent living unit; BMI was calculated as weight (kg) divided by height<sup>2</sup> (m<sup>2</sup>); diabetes mellitus status was categorised using American Diabetes Association criteria, as follows: diabetes mellitus (diagnosis of diabetes mellitus by a physician and use of diabetic medications or fasting glucose levels at least 7.0 mmol/L [126 mg/dL]), fasting hyperglycaemia (fasting glucose levels 6.1 mmol/L [110 mg/dL] or above but less than 7.0 mmol/L [126 mg/dL]), normoglycaemia (fasting glucose levels below 6.1 mmol/L [110mg/dL]); cigarette

smoking was categorised into current (current smoker or had given up smoking less than 12 months before the study examination), former (positively answered to “have you ever smoked regularly before?” and had given up smoking at least 12 months before the study examination), and never smoked; alcohol intake was categorised into heavy (three or more drinks/day), moderate (less than three drinks/day), and never drank; physical inactivity (yes, no) was categorised with answering negatively or positively to “have you participated in any recreational exercise/walk in the last two weeks?”; and weekly aspirin intake was categorised as three times/week or more, and less than three times/week or none, by combining information on aspirin intake frequency in the questionnaire to obtain an adequate sample size in each category.

Details of BP measurement and prevalence of hypertension in this older Australian community have been described previously.<sup>(26)</sup> Briefly, trained observers recorded the systolic and diastolic BPs measured from the participant’s right arm with a mercury sphygmomanometer using a cuff size appropriate for the participant’s arm circumference, after he/she had been comfortably seated for at least ten min. Hypertension was defined as in the Seventh Report of the Joint National Committee (JNC7) on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure, as systolic BP of 140 mmHg or higher, diastolic BP of 90 mmHg or higher, or a combination of self-reported hypertension diagnosis and use of antihypertensive medications.<sup>(27)</sup> BP was also classified according to JNC7 BP stages (normal [systolic values < 120 mmHg and diastolic values < 80 mmHg], prehypertension [systolic values 120–139 mmHg or diastolic values 80–89 mmHg], stage 1 hypertension [systolic values 140–159 mmHg or diastolic values 90–99 mmHg] and stage 2 hypertension [systolic values ≥ 160 mmHg or diastolic values ≥ 100 mmHg]). For the current study, we defined severe hypertension as JNC7 stage 2 hypertension or higher, or a combination of self-reported hypertension diagnosis and use of antihypertensive medications. Normotensive individuals at baseline that developed hypertension according to these criteria at the five-year follow-up examination (1997–1999) were defined to have incident hypertension. Similarly, baseline individuals without severe hypertension that developed this outcome at the five-year follow-up examination were defined to have incident severe hypertension.

To examine the association between SRH and the five-year incidence of severe hypertension, the current study included 1,298 individuals free of severe hypertension at the baseline examination, from the

**Table I. Baseline characteristics of the cohort (1,298 subjects included in the analysis of incident severe hypertension) by self-rated health and gender.**

Characteristics	Men (n = 588)				Women (n = 710)			
	Excellent	Good	Fair/poor	p-value	Excellent	Good	Fair/poor	p-value
Number at risk	158	327	103		185	405	120	
Age (years)	63.5	63.0	62.4	0.62	61.7	62.7	62.3	0.43
Education > high school level (%)	75.3	64.8	56.3	0.005	63.2	57.0	38.3	0.004
Home owner (%)	91.1	91.1	89.3	0.85	91.4	89.4	79.2	0.007
Smoking (%)								
Never	40.5	38.5	18.5	< 0.001	65.4	60.7	54.2	0.02
Former	46.8	46.5	49.5		23.8	26.2	21.7	
Current	12.7	15.0	32.0		10.8	13.1	24.2	
Alcohol intake (%)								
Nondrinker	25.3	25.1	25.2	0.04	35.1	34.6	50.0	0.0005
1–2 drinks/day	48.1	50.8	35.9		57.3	56.5	35.0	
≥ 3 drinks/day	26.6	24.2	38.8		7.6	8.9	15.0	
Body mass index (kg/m <sup>2</sup> )	25.5	25.7	26.8	0.006	25.1	25.6	26.8	0.004
Diabetes mellitus (%)	5.1	8.3	10.7	0.23	Nil	4.0	7.5	0.002
Physical inactivity (%)	26.0	28.1	41.8	0.02	25.4	27.2	44.2	0.006
Total cholesterol (mmol/L)	5.7	5.9	5.9	0.18	6.1	6.2	6.1	0.60
HDL cholesterol (mmol/L)	1.3	1.3	1.3	0.47	1.6	1.6	1.5	0.07
Triglyceride (mmol/L)	1.5	1.9	1.9	0.008	1.4	1.6	1.7	0.008
JNC7 BP stage (%)								
Normal BP	9.5	7.7	8.7	0.60	9.7	5.4	8.3	0.42
Prehypertension	70.9	75.8	68.9		73.0	76.8	74.2	
Stage I hypertension	19.6	16.5	22.3		17.3	17.8	17.5	
Systolic BP (mmHg)	134.1	134.3	135.6	0.61	133.5	135.9	135.9	0.09
Diastolic BP (mmHg)	80.0	80.4	80.7	0.76	78.7	80.0	80.2	0.09
Females only								
Menopausal status (%)					92.9	90.9	87.5	0.27
Used HRT before (%)					31.9	33.6	39.2	0.41

\*p-value for difference in the characteristic by self-rated health category based on analysis of variance or chi-square test, as appropriate  
HDL cholesterol: high density lipoprotein cholesterol; JNC7: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure; BP: blood pressure; HRT: hormone replacement therapy

2,334 individuals who participated in both the baseline and five-year follow-up examinations, after excluding those with missing baseline SRH information (n = 23), systolic or diastolic BP (n = 6), BMI (n = 23), and those with preexisting severe hypertension, including subjects with previous hypertension diagnosis and use of antihypertensive medications (n = 1,002). Similarly, to examine the association between SRH and the five-year incidence of hypertension, the current study included 679 hypertension-free individuals at the baseline examination from the 2,334 individuals who participated in both the baseline and five-year follow-up examinations, after excluding those with missing baseline SRH (n = 23), systolic or diastolic BP (n = 6), BMI (n = 23) and those with preexisting hypertension (n = 1,636). The above-mentioned categories of excluded subjects for incident hypertension and severe hypertension analyses are not mutually exclusive. We compared selected characteristics (similar to those listed in Table I) among subjects who were not followed-up (n = 1,320) to subjects who participated (n = 2,334) in the five-year follow-up examination. Compared to subjects who participated, those who did not participate in the five-year follow-up

examination were significantly older (p < 0.05), more likely to have been a smoker, less likely to have an above high-school education, had lower BMI, but more likely to have diabetes mellitus at the baseline examination. After five years of follow-up, there were 413 cases of incident severe hypertension and 380 cases of incident hypertension.

Baseline SRH was categorised into excellent, good, and fair/poor for the main analyses. We performed separate analyses for males and females. We used chi-square test and analysis of variance to compare the relationships of selected baseline characteristics by gender. We used multivariable logistic regression models to determine the odds-ratio (OR) and 95% confidence interval (CI) of the five-year incident severe hypertension (first outcome of interest) and five-year incident hypertension (second outcome of interest), controlling simultaneously for cardiovascular risk factors found, or potentially associated with incident hypertension in our study. In a subsidiary analysis, we examined a third outcome, longitudinal increase by at least one JNC7 BP stage over five years among study participants not taking antihypertensive medications, as medication

**Table II. Categories of self-rated health at baseline and five-year incidence of hypertension or severe hypertension.**

Self-rated health categories	5-year incident hypertension			5-year incident severe hypertension		
	No. at risk (cases)	Age-adjusted OR (95% CI)*	Multivariable OR (95% CI)†	No. at risk (cases)	Age-adjusted OR (95% CI)*	Multivariable OR (95% CI)†
<b>Men</b>						
Excellent	86 (45)	1 (referent)	1 (referent)	158 (35)	1 (referent)	1 (referent)
Good	179 (90)	0.91 (0.54–1.52)	0.91 (0.53–1.57)	327 (95)	1.44 (0.92–2.25)	1.44 (0.89–2.30)
Fair/poor	49 (30)	1.42 (0.70–2.91)	2.03 (0.91–4.55)	103 (37)	1.98 (1.14–3.43)	1.93 (1.04–3.56)
p-trend		0.20	0.09		0.01	0.03
<b>Women</b>						
Excellent	108 (68)	1 (referent)	1 (referent)	185 (64)	1 (referent)	1 (referent)
Good	203 (115)	0.73 (0.45–1.19)	0.57 (0.34–0.97)	405 (134)	0.90 (0.62–1.31)	0.76 (0.51–1.13)
Fair/poor	54 (32)	0.78 (0.40–1.55)	0.57 (0.26–1.23)	120 (48)	1.24 (0.77–2.00)	0.96 (0.57–1.62)
p-trend		0.72	0.49		0.49	0.70

\* estimated from logistic regression model adjusted for age (years)

† estimated from multivariable logistic regression model adjusted for education (high school or below, above high school); home ownership (no, yes); smoking (current, former, never); alcohol intake (nondrinker, < 3 drinks/day, ≥ 3 drinks/day); body mass index (kg/m<sup>2</sup>); physical inactivity (yes, no); diabetes mellitus (absent, present), mean arterial pressure (mmHg); total cholesterol level (mmol/L); high-density lipoprotein cholesterol level (mmol/L); triglyceride level (mmol/L). Additionally adjusted for menopausal status (absent, present); and previous use of hormone replacement therapy (yes, no) among females.

intake could affect their BP level. We used two logistic regression models in our analysis: an age (years)-adjusted model, and a multivariable-adjusted model, additionally adjusting for education (high school or below, above high school), home ownership (no, yes), smoking (current, former, never), alcohol intake (nondrinker, < 3 drinks/day, ≥ 3 drinks/day), body mass index (kg/m<sup>2</sup>), physical inactivity (yes, no), diabetes mellitus (absent, present), mean arterial BP (mmHg), total cholesterol level (mmol/L), HDL-C level (mmol/L), and TG level (mmol/L); among women, we additionally adjusted for menopausal status (absent, present), and present/previous use of hormone replacement therapy (yes, no).

Logistic regression models with SRH categories as an ordinal variable were used to assess trends in risk. To examine the consistency of the association between SRH and incident severe hypertension, we performed analyses within subgroups of selected variables, including categories of age at baseline (62 years [median age] or below, above 62 years), current smoking categories (absent, present), BMI (< 25 kg/m<sup>2</sup>, ≥ 25 kg/m<sup>2</sup>), diabetes mellitus (absent, present), and categories of baseline BP according to the JNC7 on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure (normotensive [normal BP or prehypertension], stage 1 hypertension).<sup>(27)</sup> We also performed a supplementary analysis additionally adjusting for circulating WBC count ( $\times 10^9$  cells/L), a non-specific marker of inflammation, in the multivariable model. SAS version 9.2 (SAS Institute, Cary, NC, USA) was used for all analyses.

## RESULTS

The average age of the study subjects at the baseline

examination was 62.9 years among men and 62.4 years among women. Table I presents the baseline characteristics of the cohort subjects included in the incident severe hypertension analysis, by SRH categories and gender. For both men and women, individuals with fair/poor SRH were more likely to be current smokers, to consume three or more drinks/day, had higher BMI, more likely to be physically inactive, and had higher TG levels. Among women, those with fair/poor SRH were more likely to have diabetes mellitus, and lower HDL-C levels. Table II presents the separate results for the association between SRH categories and incident hypertension and severe hypertension among men and women. Among men, 52.5% (165/314) developed five-year incident hypertension, and among women, 58.9% (215/365) developed this outcome. In Table II, the association between fair/poor SRH and incident hypertension among men was in a positive direction, but failed to reach conventional levels of statistical significance ( $\alpha = 0.05$ ). The power for this analysis was only 72.5% in order to detect the observed OR of 2.03 in the fair/poor SRH category.

Among men, 28.4% (167/588) developed five-year incident severe hypertension and among women, 34.6% (246/710) developed this outcome. In Table II, overall, among men, compared to individuals with excellent SRH (referent category), those with fair/poor SRH had higher OR [95% CI] of incident severe hypertension in both the age-adjusted (1.98 [1.14–3.43]) and the multivariable-adjusted model (1.93 [1.04–3.56]). Corresponding models of trend were also statistically significant among men. In contrast, among women, there was no association between SRH and either incident hypertension or severe

**Table III. Categories of self-rated health at baseline and odds-ratio of five-year incident severe hypertension, within subgroups.**

Stratified subgroups	Men			Women		
	Self-rated health		OR (95% CI)* comparing fair/ poor self-rated health vs. excellent	Self-rated health		OR (95% CI)* comparing fair/ poor self-rated health vs. excellent
	Excellent	Fair/poor		Excellent	Fair/poor	
	No. at risk (no. of severe hypertension cases)	No. at risk (no. of severe hypertension cases)		No. at risk (no. of severe hypertension cases)	No. at risk (no. of severe hypertension cases)	
Age at baseline (years)						
≤ 62	76 (14)	58 (21)	2.63 (1.08–6.39)	104 (30)	66 (25)	1.05 (0.51–2.18)
> 62	82 (21)	45 (16)	1.42 (0.59–3.43)	81 (34)	54 (23)	0.87 (0.39–1.90)
Current smoking						
No	138 (30)	70 (28)	2.12 (1.05–4.25)	165 (56)	91 (39)	1.01 (0.57–1.81)
Yes	20 (5)	33 (9)	1.33 (0.31–5.61)	20 (8)	29 (9)	0.57 (0.14–2.30)
Body mass index (kg/m <sup>2</sup> )						
< 25	69 (16)	35 (11)	1.64 (0.58–4.70)	97 (29)	44 (16)	1.03 (0.44–2.41)
≥ 25	89 (19)	68 (26)	2.08 (1.01–4.28)	88 (35)	76 (32)	0.93 (0.47–1.85)
Diabetes mellitus						
Absent	150 (33)	92 (32)	1.75 (0.96–3.19)	185 (64)	111 (41)	0.88 (0.51–1.50)
Present	8 (2)	11 (5)	9.05 (0.52–156.5)	Nil	9 (7)	NA
JNC7 BP categories <sup>‡</sup>						
Normotensive	86 (12)	49 (11)	1.98 (0.72–5.45)	108 (27)	54 (15)	0.95 (0.40–2.22)
Stage I hypertension	72 (23)	54 (26)	1.69 (0.77–3.71)	77 (37)	66 (33)	1.00 (0.58–1.71)

\* estimated from multivariable logistic regression model adjusted for age (years); education (high school or below, above high school); home ownership (no, yes); smoking (current, former, never); alcohol intake (nondrinker, < 3 drinks/day, ≥ 3 drinks/day); body mass index (kg/m<sup>2</sup>); physical inactivity (yes, no); diabetes mellitus (absent, present); mean arterial pressure (mmHg); total cholesterol level (mmol/L); high-density lipoprotein cholesterol level (mmol/L); triglyceride level (mmol/L). Additionally adjusted for menopausal status (absent, present); and previous use of hormone replacement therapy (yes, no) among females.

<sup>‡</sup>JNC7: The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: normotensive (normal BP (systolic BP < 120 and diastolic BP < 80 mmHg); prehypertension (systolic BP 120–139 or diastolic BP 80–89 mmHg); stage I hypertension (systolic BP 140–159 or diastolic BP 90–99 mmHg).

hypertension. In Table III, we examined the OR (95% CI) of incident severe hypertension comparing fair/poor SRH vs. excellent SRH with subgroups of age (≤ 62 years, > 62 years), current smoking (absent, present), BMI (< 25 kg/m<sup>2</sup>, ≥ 25 kg/m<sup>2</sup>), diabetes mellitus (absent, present), and JNC7 BP categories (normotensives, stage I hypertension). Overall, among men, the observed positive association between fair/poor SRH and incident severe hypertension in the whole cohort was consistently present within these subgroups; however, the ORs within some subgroups failed to reach conventional levels of statistical significance, probably due to small sample size. In contrast, among women, the ORs for the association between SRH and incident severe hypertension generally hovered around the null.

In a subsidiary analysis, to examine if the observed association between fair/poor SRH and incident severe hypertension was explained by inflammatory markers, we repeated the multivariable models with additional adjustment for circulating WBC count ( $\times 10^9$  cells/L). The results were attenuated, but essentially similar. For example, among men, compared to individuals with excellent SRH (referent), the OR (95% CI) of incident

severe hypertension was 1.43 (0.88–2.34) among those with good SRH, and 1.80 (0.99–3.27) among those with fair/poor SRH, *p*-trend = 0.056. We also examined the association between SRH categories and longitudinal increase by at least one JNC7 BP stage over five years (*n* = 395) among study participants not taking antihypertensive medications at baseline or follow-up (*n* = 1,140); the results were essentially similar. Among men, compared to individuals with excellent SRH (referent), the OR (95% CI) of increase by ≥ 1 JNC7 BP stage was 1.51 (0.99–2.32) among those with good SRH, and 1.71 (1.00–2.93) among those with fair/poor SRH, *p*-trend = 0.04. Among women, compared to individuals with excellent SRH (referent), the OR (95% CI) of increase by ≥ 1 JNC7 BP stage was 0.78 (0.55–1.13) among those with good SRH, and 1.04 (0.65–1.66) among those with fair/poor SRH, *p*-trend = 0.91.

## DISCUSSION

In the Blue Mountains cohort of older Australians, compared to individuals reporting excellent SRH, we found that fair/poor SRH was positively associated with five-year incident severe hypertension among men, but

not among women. This association was independent of smoking, alcohol intake, BMI, and other related risk factors. Among men, the OR of incident severe hypertension increased in a dose-dependent manner with lower/worsening SRH categories and the association was consistently present in subgroup analyses stratified by age, smoking, BMI, diabetes mellitus, and JNC7 BP categories. The findings from this long-term follow-up study of older, community-dwelling Australians contribute to the current understanding of the predictive relation between SRH and cardiovascular disease<sup>(3-5)</sup> and mortality.<sup>(7-9)</sup> This data suggests that development of severe hypertension, a strong, independent predictor of cardiovascular disease and mortality,<sup>(27)</sup> may be one of the biological mechanisms mediating between low SRH and mortality.

Though previous cross-sectional<sup>(6,13-16)</sup> and case-control<sup>(17,18)</sup> studies have reported an association between poor SRH and prevalent hypertension, to our knowledge, this is the first prospective investigation of this relationship. Our finding of an association between SRH and five-year incident severe hypertension shows high internal validity, as indicated by: the independence of the association from traditional risk factors, evidence suggesting a dose-response trend in the magnitude of the association, the consistency of these findings across stratified sub-groups, as well as in the magnitude of the association. In the current study, by comparison to the results for incident severe hypertension, the association between SRH and incident hypertension failed to reach conventional levels of statistical significance due to the limited sample size (power < 80%); approximately 70% of the baseline participants of this elderly cohort had  $\geq$  stage 1 hypertension and had to be excluded from the incident hypertension analysis. Nevertheless, the overall results for incident hypertension analysis are in agreement with the results for incident severe hypertension analysis, where we had adequate sample size, and suggest a positive, albeit non-significant, association among men, but not among women.

SRH has been shown to be closely related to social integration and adaptability to psychosocial stress.<sup>(28,29)</sup> Plausible explanations for the observed association between SRH and incident severe hypertension include the reported relationship between poor SRH and related negative psychosocial variable cluster to the metabolic syndrome,<sup>(30)</sup> low testosterone levels,<sup>(31)</sup> increased insulin resistance and markers of inflammation,<sup>(31-33)</sup> disordered sleep and sleep apnoea,<sup>(34-37)</sup> and increased cortisol and low adrenal androgen levels.<sup>(28,37-39)</sup> The observed lack of

association between SRH and incident severe hypertension among women in the current study is analogous to the reported weaker/lack of association between SRH and other cardiovascular outcomes and mortality among women, compared to men.<sup>(4,9,20-23)</sup> Also, several studies have reported gender differences in the association between many of the above-mentioned markers and hypertension and other vascular diseases, including the role of androgens and androgen receptor,<sup>(40)</sup> insulin resistance and insulin-like growth factor,<sup>(41-44)</sup> fibrinogen and markers of inflammation,<sup>(45,46)</sup> and cortisol and adrenal androgen levels.<sup>(38,39,47,48)</sup> A previous report from the Framingham Heart Study had reported that lack of adaptability to psychosocial stress, in relation to anger and anxiety, were associated with hypertension among men, but not among women.<sup>(49)</sup> Furthermore, a recent animal model on the effect of long-term psychosocial stressors on morbidity and mortality also suggest evidence for similar gender difference.<sup>(50)</sup>

The main advantages of our study included its stable general population sample base, longitudinal follow-up, and the use of standardised protocols for exposure and outcome assessment. Several study limitations also need to be considered while interpreting our findings. BP levels were based on a single reading at both surveys. This could have resulted in misclassification of hypertension status (likely to be overestimation). However, previously-reported hypertension prevalence,<sup>(26)</sup> incidence,<sup>(51)</sup> and trend over time<sup>(26)</sup> from our cohort were comparable to other older general population samples, though higher than in some middle-aged samples.<sup>(45)</sup> Furthermore, any misclassification was likely to be non-differential in nature and should bias the estimates of association towards the null. It is possible that the observed association between fair/poor SRH and incident severe hypertension in the current study could be explained by unmeasured psychosocial factors such as depression. Finally, it is possible that our results were biased by selective survival of the cohort. However, as poor SRH is related to decreased survival,<sup>(7-9)</sup> selective survival was likely to underestimate our findings.

In conclusion, findings from our study support the hypothesis of an association between fair/poor SRH and incident severe hypertension among men, in comparison to excellent SRH. These results suggest that at least part of the reported association between SRH and mortality may be mediated by its relation to incident severe hypertension. Assessment of SRH in clinical practice may provide additional information regarding a subject's cardiovascular health. Future analyses from larger cohort

studies among women are required to further investigate the lack of association among women.

## ACKNOWLEDGEMENT

This study was supported in part by the Australian National Health and Medical Research Council, Canberra, Australia (project grant ID 974159 and 211069).

## REFERENCES

1. Detmar SB, Muller MJ, Schornagel JH, Wever LD, Aaronson NK. Health-related quality-of-life assessments and patient-physician communication: a randomized controlled trial. *JAMA* 2002; 288:3027-34.
2. US Department of Health and Human Services. *Healthy People 2010. With Understanding and Improving Health and Objectives for Improving Health*. 2nd ed. Washington DC: US Government Printing Office, November 2000-2006.
3. Moller L, Kristensen TS, Hollnagel H. Self rated health as a predictor of coronary heart disease in Copenhagen, Denmark. *J Epidemiol Community Health* 1996; 50: 423-8.
4. Emmelin M, Weinehall L, Stegmayr B, et al. Self-rated ill-health strengthens the effect of biomedical risk factors in predicting stroke, especially for men -- an incident case referent study. *J Hypertens* 2003; 21: 887-96.
5. Kubzansky LD, Kawachi I, Spiro A, III, et al. Is worrying bad for your heart? A prospective study of worry and coronary heart disease in the Normative Aging Study. *Circulation* 1997; 95: 818-24.
6. Wang JJ, Smith W, Cumming RG, Mitchell P. Variables determining perceived global health ranks: findings from a population-based study. *Ann Acad Med Singapore* 2006; 35: 190-7.
7. Idler EL, Benyamini Y. Self-rated health and mortality: a review of twenty-seven community studies. *J Health Soc Behav* 1997; 38: 21-37.
8. Borawski EA, Kinney JM, Kahana E. The meaning of older adults' health appraisals: congruence with health status and determinant of mortality. *J Gerontol B Psychol Sci Soc Sci* 1996; 51:S157-70.
9. McCallum J, Shadbolt B, Wang D. Self-rated health and survival: a 7-year follow-up study of Australian elderly. *Am J Public Health* 1994; 84: 1100-5.
10. Wannamethee G, Shaper AG. Self-assessment of health status and mortality in middle-aged British men. *Int J Epidemiol* 1991; 20: 239-45.
11. Weinberger M, Damell JC, Tierney WM, et al. Self-rated health as a predictor of hospital admission and nursing home placement in elderly public housing tenants. *Am J Public Health* 1986; 76: 457-9.
12. Schoenfeld DE, Malmrose LC, Blazer DG, Gold DT, Seeman TE. Self-rated health and mortality in the high-functioning elderly -- a closer look at healthy individuals: MacArthur field study of successful aging. *J Gerontol* 1994; 49: M109-15.
13. Barger SD, Muldoon MF. Hypertension labelling was associated with poorer self-rated health in the Third US National Health and Nutrition Examination Survey. *J Hum Hypertens* 2006; 20: 117-23.
14. Bardage C, Isacson DG. Hypertension and health-related quality of life. an epidemiological study in Sweden. *J Clin Epidemiol* 2001; 54: 172-81.
15. Mena-Martin FJ, Martin-Escudero JC, Simal-Blanco F, et al. Health-related quality of life of subjects with known and unknown hypertension: results from the population-based Hortega study. *J Hypertens* 2003; 21: 1283-9.
16. Lawrence WF, Fryback DG, Martin PA, Klein R, Klein BE. Health status and hypertension: a population-based study. *J Clin Epidemiol* 1996; 49: 1239-45.
17. Erickson SR, Williams BC, Gruppen LD. Perceived symptoms and health-related quality of life reported by uncomplicated hypertensive patients compared to normal controls. *J Hum Hypertens* 2001; 15: 539-48.
18. Milne BJ, Logan AG, Flanagan PT. Alterations in health perception and lifestyle in treated hypertensives. *J Chronic Dis* 1985; 38: 37-45.
19. Hong TB, Oddone EZ, Dudley TK, Bosworth HB. Subjective and objective evaluations of health among middle-aged and older veterans with hypertension. *J Aging Health* 2005; 17: 592-608.
20. House JS, Robbins C, Metzner HL. The association of social relationships and activities with mortality: prospective evidence from the Tecumseh Community Health Study. *Am J Epidemiol* 1982; 116: 123-40.
21. Helmer C, Barberger-Gateau P, Letenneur L, Dartigues JF. Subjective health and mortality in French elderly women and men. *J Gerontol B Psychol Sci Soc Sci* 1999; 54: S84-92.
22. Spiers N, Jagger C, Clarke M, Arthur A. Are gender differences in the relationship between self-rated health and mortality enduring? Results from three birth cohorts in Melton Mowbray, United Kingdom. *Gerontologist* 2003; 43: 406-11.
23. Deeg DJ, Kriegsman DM. Concepts of self-rated health: specifying the gender difference in mortality risk. *Gerontologist* 2003; 43: 376-86.
24. Attebo K, Mitchell P, Smith W. Visual acuity and the causes of visual loss in Australia. The Blue Mountains Eye Study. *Ophthalmology* 1996; 103: 357-64.
25. Mitchell P, Smith W, Attebo K, Wang JJ. Prevalence of age-related maculopathy in Australia. The Blue Mountains Eye Study. *Ophthalmology* 1995; 102: 1450-60.
26. Chua B, Rochtchina E, Mitchell P. Temporal changes in the control of blood pressure in an older Australian population. *J Hum Hypertens* 2005; 19: 691-6.
27. Chobanian AV, Bakris GL, Black HR, et al. The Seventh Report of the Joint National Committee on Prevention, Detection, Evaluation, and Treatment of High Blood Pressure: the JNC 7 report. *JAMA* 2003; 289: 2560-72.
28. Kristenson M, Olsson AG, Kucinskiene Z. Good self-rated health is related to psychosocial resources and a strong cortisol response to acute stress: the LiVicordia study of middle-aged men. *Int J Behav Med* 2005; 12: 153-60.
29. Barger SD. Do psychological characteristics explain socioeconomic stratification of self-rated health? *J Health Psychol* 2006; 11: 21-35.
30. Meyer JM, Nasrallah HA, McEvoy JP, et al. The Clinical Antipsychotic Trials Of Intervention Effectiveness (CATIE) Schizophrenia Trial: clinical comparison of subgroups with and without the metabolic syndrome. *Schizophr Res* 2005; 80: 9-18.
31. Nilsson PM, Moller L, Solstad K. Adverse effects of psychosocial stress on gonadal function and insulin levels in middle-aged males. *J Intern Med* 1995; 237: 479-86.
32. Janszky I, Lekander M, Blom M, Georgiades A, Ahnve S. Self-rated health and vital exhaustion, but not depression, is related to inflammation in women with coronary heart disease. *Brain Behav Immun* 2005; 19: 555-63.
33. Lekander M, Elofsson S, Neve IM, Hansson LO, Uden AL. Self-rated health is related to levels of circulating cytokines. *Psychosom Med* 2004; 66: 559-63.
34. Weaver EM, Kapur V, Yueh B. Polysomnography vs self-reported measures in patients with sleep apnea. *Arch Otolaryngol Head Neck Surg* 2004; 130: 453-8.
35. Akashiba T, Kawahara S, Akahoshi T, et al. Relationship between quality of life and mood or depression in patients with severe obstructive sleep apnea syndrome. *Chest* 2002; 122: 861-5.
36. Rocha FL, Uchoa E, Guerra HL, et al. Prevalence of sleep complaints and associated factors in community-dwelling older people in Brazil: the Bambui Health and Ageing Study (BHAS). *Sleep Med* 2002; 3: 231-8.

37. Adler NE, Epel ES, Castellazzo G, Ickovics JR. Relationship of subjective and objective social status with psychological and physiological functioning: preliminary data in healthy white women. *Health Psychol* 2000; 19:586-92.
38. Berr C, Lafont S, Debuire B, Dartigues JF, Baulieu EE. Relationships of dehydroepiandrosterone sulfate in the elderly with functional, psychological, and mental status, and short-term mortality: a French community-based study. *Proc Natl Acad Sci U S A* 1996; 93:13410-5.
39. Barrett-Connor E, Goodman-Gruen D. The epidemiology of DHEAS and cardiovascular disease. *Ann N Y Acad Sci* 1995; 774:259-70.
40. Reckelhoff JF, Zhang H, Srivastava K, Granger JP. Gender differences in hypertension in spontaneously hypertensive rats: role of androgens and androgen receptor. *Hypertension* 1999; 34:920-3.
41. Unden AL, Elofsson S, Brismar K. Gender differences in the relation of insulin-like growth factor binding protein-1 to cardiovascular risk factors: a population-based study. *Clin Endocrinol (Oxf)* 2005; 63:94-102.
42. Falkner B, Hulman S, Kushner H. Gender differences in insulin-stimulated glucose utilization among African-Americans. *Am J Hypertens* 1994; 7:948-52.
43. Polderman KH, Gooren LJ, Asscheman H, Bakker A, Heine RJ. Induction of insulin resistance by androgens and estrogens. *J Clin Endocrinol Metab* 1994; 79:265-71.
44. Tamaya-Mori N, Uemura K, Iguchi A. Gender differences in the dietary lard-induced increase in blood pressure in rats. *Hypertension* 2002; 39:1015-20.
45. Folsom AR, Peacock JM, Nieto FJ, et al. Plasma fibrinogen and incident hypertension in the Atherosclerosis Risk in Communities (ARIC) Study. *J Hypertens* 1998; 16:1579-83.
46. de Simone G, Devereux RB, Roman MJ, et al. Gender differences in left ventricular anatomy, blood viscosity and volume regulatory hormones in normal adults. *Am J Cardiol* 1991; 68:1704-8.
47. Greenspan SL, Rowe JW, Maitland LA, oon-Dyke M, Elahi D. The pituitary-adrenal glucocorticoid response is altered by gender and disease. *J Gerontol* 1993; 48:M72-7.
48. Traustadottir T, Bosch PR, Matt KS. Gender differences in cardiovascular and hypothalamic-pituitary-adrenal axis responses to psychological stress in healthy older adult men and women. *Stress* 2003; 6:133-40.
49. Markovitz JH, Matthews KA, Kannel WB, Cobb JL, D'Agostino RB. Psychological predictors of hypertension in the Framingham Study. Is there tension in hypertension? *JAMA* 1993; 270:2439-43.
50. Hermes GL, Rosenthal L, Montag A, McClintock MK. Social isolation and the inflammatory response: sex differences in the enduring effects of a prior stressor. *Am J Physiol Regul Integr Comp Physiol* 2006; 290:R273-82.
51. Smith W, Wang JJ, Wong TY, et al. Retinal arteriolar narrowing is associated with 5-year incident severe hypertension: the Blue Mountains Eye Study. *Hypertension* 2004; 44:442-7.

## Looking to hit that sales target?

Advertise with the **Singapore Medical Journal.**

*The voice of academic medicine in Singapore and Southeast Asia since 1960*

To advertise, please contact: **Li Li Loy**, Advertising Executive  
 Mobile: **9634 9506** Tel: **6223 1264 ext 23** Email: **lili@sma.org.sg**