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# Decreased risk of death from coronary heart disease amongst men with higher ‘femininity’ scores: a general population cohort study

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<b>Accepted</b>	30 January 2007
<b>Context</b>	At all ages men have higher rates of coronary heart disease (CHD) than women, although similar proportions of men and women eventually die of CHD. Gender differences in CHD incidence and mortality are often explained in relation to biological (hormonal) and behavioural risk factors (e.g. smoking), but psychological factors and broader social constructions of gender are rarely considered.
<b>Objective</b>	To examine the relationship between measures of gender role orientation at baseline in 1988 and mortality from CHD over 17 years (to June 2005).
<b>Design</b>	Prospective cohort study linked to national mortality reporting.
<b>Setting</b>	Socially varied mainly urban area centred on city of Glasgow in West Central Scotland, UK.
<b>Participants</b>	In total, 1551 participants (704 men and 847 women) aged 55 years took part in detailed interviews with nurses trained in survey methods in 1988. These included a wide range of measures of physical development and functioning, self reported health and health behaviour, personal and social circumstances and a measure of gender role orientation (yielding scores for ‘masculinity’ and ‘femininity’).
<b>Main outcome measures</b>	Mortality from CHD up to June 2005 (88 CHD deaths in men; 41 CHD deaths in women).
<b>Results</b>	After adjusting for smoking, binge drinking, body mass index, systolic blood pressure, household income and psychological well-being, higher ‘femininity’ scores in men were associated with a lower risk of CHD death (hazards ratio per unit increase in ‘femininity’ score 0.65, 95% CIs 0.48–0.87, $P=0.004$ ). No such relationship was observed amongst women. ‘Masculinity’ scores were unrelated to CHD mortality in either men or women.
<b>Conclusions</b>	These results suggest that social constructions of gender influence the risk of ill health, here death from CHD. Men who are less able to identify themselves with characteristics identified as ‘feminine’ or expressive (who have a more limited stereotypically masculine self-image) may be at increased risk of coronary disease. Further research on the link between social constructions of gender and health is needed.

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## Introduction

Gender differences in coronary heart disease (CHD) rates have been described as one of the most striking features of cardiovascular mortality in the 20th century,<sup>1</sup> 'enigmatic',<sup>2</sup> and 'one of the most interesting of all epidemiological questions'.<sup>3</sup> Although the ratio of male to female deaths has fluctuated to some degree over time, in the UK<sup>1</sup> and elsewhere<sup>4</sup> and CHD is increasingly recognized as a major cause of morbidity and mortality in women, men are still disadvantaged in experiencing or dying from CHD around 10 years earlier than women.<sup>5</sup> Despite advances in understanding CHD, and the recognition of major risk factors (notably cigarette smoking, diabetes, hyperlipidaemia and hypertension which are thought to be present in up to 80–90% of patients with CHD),<sup>5,6</sup> questions remain about why some people develop CHD and others do not, not least because many of the risk factors are present in people who have not developed CHD.<sup>7</sup> According to the Committee on Understanding the Biology of Sex and Gender Differences, 'none (of these questions) is more compelling than the differences between the sexes'<sup>8</sup> (p. 159). It is often assumed<sup>4</sup> that endogenous oestrogens provide a degree of cardioprotection to women prior to the menopause,<sup>8</sup> although, as Barrett-Connor has remarked, 'proof of this hypothesis has been surprisingly elusive'.<sup>3</sup> A protective effect of oestrogen cannot explain gender differences alone given the rapidly changing trends in CHD mortality by gender.<sup>4</sup> Further explanations for the patterns of CHD by gender have included other biological explanations (e.g. sex-linked inheritance, or central obesity), and classic risk factors such as smoking, alcohol consumption and HDL cholesterol. However, whilst differences in common CHD risk factors between men and women are clearly important, they do not appear to explain fully the differences.<sup>3,9</sup>

In recent years more attention has focused on whether masculinity, or particular practices of masculinity,<sup>10</sup> is detrimental to men's health.<sup>11–16</sup> It has been suggested that a sizeable portion of men's excess mortality over women is linked to masculine identity, men's roles and gendered patterns of socialization.<sup>9</sup> In relation to CHD, Helgeson suggests that traditional masculinity may both place men at greater risk for heart disease and influence their adjustment to heart disease, invoking men's supposed 'unwillingness to rely on others for assistance and inability to express emotions' (p. 69). However, she notes the shortage of empirical research in this area.<sup>9</sup>

In general, physiological and behavioural risk factors for CHD have been more widely researched than psychosocial factors, although there has been interest in depression and anxiety, psychosocial conditions (particularly in the formal labour market), social support and 'type A' or 'hostile' personalities.<sup>17</sup> Helgeson has remarked on the failure to recognize the role that 'traditional masculinity plays in the development of (Type A) behaviour' (p. 755).<sup>18</sup> Hemingway and Marmot<sup>17</sup> proposed that psychosocial factors could be linked to CHD in

various ways: indirectly via health behavioural risk factors (such as smoking, alcohol and dietary consumption and physical activity); directly via acute or chronic physiological changes; and via uptake of medical care.

One additional psychological measure that has received little attention when studying gender differences in health<sup>19</sup> is gender role orientation (GRO). The originator of a widely used measure of GRO, Sandra Bem,<sup>20,21</sup> stimulated debate on androgyny (principally within psychology). Bem argued that 'psychologically "androgynous"' individuals (those who were able to be 'both instrumental *and* expressive, both assertive *and* yielding, both masculine *and* feminine',<sup>22</sup> (p. 634) emphasis in original) would have a 'sex role adaptability (that) enables them to engage in situationally effective behaviour without regard for its stereotype as masculine or feminine' (p. 634). She raises the possibility that 'the androgynous individual will some day come to define a new and more human standard of psychological health'. Although she did not extend this to other aspects of health or health-related behaviour, it is interesting to test empirically whether such relationships exist.

Helgeson asserts that masculinity is positively associated with psychological health, but negatively with health behaviour or physical health.<sup>9</sup> Others have hypothesized that protective health behaviours will be positively associated with both masculinity (instrumentality) and femininity (expressiveness).<sup>23</sup> However, studies which have examined health behaviours in relation to Bem's 'masculinity' and 'femininity' scores do not consistently support this hypothesis. In a general population sample in the UK, measures of 'masculinity' and 'femininity' were positively associated with current smoking amongst women born in the early 1950s (but not amongst women born in the early 1930s); for men, 'femininity' scores were positively associated with smoking in men born in the early 1930s but not amongst men 20 years younger, but 'masculinity' was not related to smoking at either age.<sup>24</sup> Amongst white-collar employees in the UK, masculinity but not femininity scores were positively associated with smoking in both men and women in two large organizations, a bank and a university, and heavy drinking was positively associated with masculinity but not femininity in both organizations.<sup>25</sup> A study comparing adults in Moscow and Toronto reported that masculinity was associated with lower alcohol use among men and higher use among women in Moscow, but that femininity was associated with higher alcohol use among women in Toronto.<sup>26</sup> We do not find the inconsistency of these results surprising; rather they illustrate the central importance of the broader (social, historical, political and cultural) context<sup>27</sup> and the complexity of the ways in which behaviours are linked to gender, as illustrated by historical research on smoking.<sup>28–30</sup>

Although measures of GRO have been widely used, examining the relationship between 'masculinity' and 'femininity' scores and a whole host of other outcomes related directly or indirectly to health (e.g. depression and anxiety, eating disorders, alcohol dependence, sexuality and sexual orientation,

body image, health risk taking), we have been able identify only one study (a population-based cohort study of cardiovascular disease amongst residents of Rancho Bernardo initially surveyed in 1972–74)<sup>31</sup> which has examined the relationship between these and CHD mortality. These unpublished data, presented by Barrett-Connor (Fig. 2),<sup>3</sup> suggest that ‘masculine’, ‘feminine’ and ‘androgynous’ traits were not related to fatal CHD, but this preliminary analysis was based on just 24 deaths. Here we investigate in a community-based sample whether measures of GRO are related to CHD mortality in men and women.

## Methods

Participants are from the West of Scotland Twenty-07 Study, a longitudinal study of social patterning of health in three age cohorts. Here we investigate mortality in the oldest cohort who were born in the early 1930s, and were aged around 55 years when first studied in 1988. Respondents were sampled from residents in the Central Clydeside Conurbation, a socially varied but mainly urban area centred on the city of Glasgow in the west of Scotland, UK. The initial sample size for the 1930s cohort was 1551. Baseline data collection comprised two detailed structured interviews, the second conducted by registered nurses trained in interview techniques, usually in the respondent’s home, and included a wide range of measures of self-reported health and health behaviour, physical development and functioning and personal and social circumstances. Interviews were supplemented by a self-completion questionnaire, mailed out in advance and collected by the nurse at the time of the interview, which included several standardized, well-validated measures.

In the UK, the National Health Service Central Registers provide a reliable and virtually complete facility, which initiates an automatic report of the date and cause of any deaths occurring in ‘flagged’ cohorts. This is a standard approach for ascertaining mortality in the UK. This analysis reports mortality follow-up to June 2005. For all deaths, a copy of the death certificate was available. For our primary analyses, those with codes 410 to 414.9 in ICD9 and I20 to I25.9 in ICD10 for the main cause of death were coded as CHD deaths. In supplementary analyses, we also coded any mention of CHD on the death certificate as a CHD death, although we expected this more inclusive outcome to reveal a weaker relationship with risk factors for CHD.

### Measurement of ‘masculinity’, ‘femininity’ and androgyny

‘Masculinity’ and ‘femininity’ scores were measured using the Short Form of the Bem Sex Role Inventory (BSRI), a widely used self-complete measure of GRO.<sup>20,24,32</sup> The BSRI is premised on the assumption that masculinity and femininity are both conceptually and empirically independent. It relies on an individual’s endorsement (on a scale from 1—‘never or almost never true’ to 7—‘always or almost always true’) of a series of qualities, which had been judged in the USA to be culturally characteristic of either males or females. This measure has been validated in Scotland.<sup>33</sup> The ‘masculinity’ and ‘femininity’ scores are each the mean of ratings of 10 items

(‘masculinity’—defend my own beliefs, independent, assertive, strong personality, forceful, have leadership abilities, willing to take risks, dominant, willing to take a stand, aggressive; ‘femininity’—affectionate, sympathetic, sensitive to the needs of others, understanding, compassionate, eager to soothe hurt feelings, warm, tender, loves children, gentle). As an inspection of missing data showed that cases with any missing data mostly either missed all or only one or two items, masculinity and femininity scores were calculated for subjects who answered eight or more of the 10 items. ‘Masculinity’ and ‘femininity’ scores were available for 1415 (91.2%) and 1417 (91.4%) of the 1551 participants, respectively.

The ‘masculinity’ and ‘femininity’ scores were generally normally distributed.<sup>19</sup> The internal reliability of both scales was high (Cronbach’s alpha: masculinity scale 0.84; femininity scale 0.87). As expected, correlations between the two scores are low ( $r=0.07$ ).

There has been considerable discussion about how best to score the BSRI and to assess whether people are ‘androgynous’ or ‘sex-typed’ on the basis of their BSRI scores.<sup>32</sup> Bem recommended a ‘median-split’ method yielding four distinct groups depending on whether individuals scored above (high) or below (low) the median on the ‘masculinity’ and ‘femininity’ scales: ‘androgynous’ [high ‘femininity’ score (F), high ‘masculinity’ score (M)]; ‘masculine’ (high M, low F), ‘feminine’ (high F, low M) or ‘undifferentiated’ (low M, low F). However, we do not favour this method since it loses much of the information on variation within the population, and is prone to misclassification with many people’s scores differing little from the median.<sup>32</sup> Hence, our primary analyses use the masculinity and femininity scores as continuous variables, testing for an interaction between the two scales as an indicator of ‘androgyny’. However, we have chosen also to include results from the median-split four-category method for comparability with the only other (unpublished) data on GRO and CHD mortality (the analysis of the Rancho Bernardo cohort described above).<sup>3</sup> Here, as one method advocated by Bem,<sup>20</sup> we have calculated the median value for our population by weighting the sample to give equal numbers of men and women. This is another disadvantage to using the median split method since the measure is effectively standardized to our study population rather than to some generic, generalizable group. We could have used cut-offs recommended in the manual for the BSRI, but these are based on a non-representative sample of US college students collected in the early 1970s. This seems an inappropriate reference group given age and cultural differences and historical changes in gender roles. Scores greater than or equal to the median score are classified as ‘high’, all others as ‘low’. We use the ‘androgynous’ group as the comparator group when assessing the relationship with CHD mortality since Bem consistently theorized (albeit with respect to psychological health) that this is the group which should be in the best health, for both men and women.

### Covariates

Of the major established risk factors for CHD, we included smoking, alcohol use, blood pressure, body mass index (BMI) and socio-economic status as covariates. Respondents were

categorized as never smokers, ex-smokers and current smokers using self-reports of smoking history. We use a measure of binge drinking, rather than total consumption of alcohol, as an indicator of alcohol consumption as this is most consistently shown to be related to mortality.<sup>34,35</sup> We have defined binge drinking as consumption of 10+ units on a single occasion in the last week in men, and 7+ units in women.<sup>36,37</sup> Blood pressure was measured in the sitting position, in the right arm after a five-minute rest, using a Hawksley random zero sphygmomanometer and an adult size cuff (with inflatable bladder 35 × 12 cm). Two readings of systolic and diastolic phase 5 were recorded to the nearest even number; the average of the two systolic measurements is used in this analysis. BMI was calculated as weight (kg)/height (m)<sup>2</sup>. Height was measured to the nearest centimetre using Nivotoise stadiometers, respondents standing without shoes, heels against the wall and head in the Frankfort plane. Weight (clothed) was measured to the nearest 0.5 kg by portable electronic scales calibrated at the local trading standards office. Household income was used as an indicator of socio-economic status. No measures of hyperlipidaemia are available because blood samples were not taken. No adjustment was made for age because of the very limited age range of participants, nor for ethnicity as the sample was predominantly white, reflecting the relative lack of ethnic diversity in this part of the UK.

Although psychological well-being is often not included as a covariate in studies of CHD, we have included it here because it has been shown to be related both to CHD mortality<sup>17</sup> and to masculinity scores, including in the Twenty-07 Study.<sup>19,27</sup> Here we have measured psychological well-being using the 12-item version of the General Health Questionnaire (GHQ),

a well-validated and commonly used measure in community-based studies.<sup>38</sup> Scores on each item range from 0 to 3, Likert scores (range 0–36) are used.

### Statistical analysis

Frequencies or descriptive statistics for the predictor variables and covariates, and descriptive statistics by gender of the masculinity and femininity scores were obtained. *T*-tests were used to test for gender differences in the mean masculinity and femininity scores. Univariate statistics were used to investigate relationships between the masculinity and femininity scores and the covariates. A series of Cox regression models was run separately for males and females to assess the relationship between CHD mortality and (i) 'masculinity' and 'femininity' scores, (ii) the BSRI fourfold classification and (iii) each of the covariates. Initially, univariate relationships were assessed. A Cox regression model testing for an interaction between the 'masculinity' and 'femininity' scores was also run. Finally, models were run including both the 'masculinity' and 'femininity' scores together (or the BSRI 'median-split' 4-fold classification), adjusted for smoking, binge drinking, blood pressure, BMI, occupational social class and GHQ score. On theoretical grounds, the 'androgynous' group was taken as the reference category in the 'median-split' analyses (as Bem argued that they should have the best psychological health, and we have extrapolated this to wider health).

### Results

Over a quarter (28.3%, 439 deaths) of the cohort died from all causes during follow-up (from baseline interview in 1988 to

**Table 1** Masculinity and femininity scores according to covariates by gender

	Men ( <i>n</i> = 704)			Women ( <i>n</i> = 847)		
	<i>n</i>	%	Mean (SD)	<i>n</i>	%	Mean (SD)
Masculinity score	646	–	4.50 (0.92)	769	–	4.08 (0.97)
Femininity score	646	–	5.30 (0.77)	771	–	5.63 (0.76)
Median-split						
'Androgynous'	153	23.7	–	171	22.2	–
'Masculine'	223	34.6	–	108	14.0	–
'Feminine'	84	13.0	–	273	35.5	–
'Undifferentiated'	185	28.7	–	217	28.2	–
Covariates						
Smoking						
<i>Never smoker</i>	164	23.3	–	347	41.0	–
<i>Ex-smoker</i>	180	25.6	–	165	19.5	–
<i>Current smoker</i>	360	51.1	–	335	39.6	–
Alcohol						
<i>Binge drinking</i>	200	32.9		31	4.7	
<i>Not</i>	408	67.1		628	95.3	
Household income	704	–	171.31 (102.94)	847	–	149.02 (84.46)
BMI (kg/m <sup>2</sup> )	657	–	26.04 (3.68)	795	–	26.02 (4.91)
Systolic blood pressure (mm Hg)	662	–	138.20 (21.26)	797	–	135.99 (20.06)
GHQ score	651	–	10.98 (4.86)	778	–	11.94 (5.35)

12 June 2005). As expected CHD was the most prevalent cause of death and CHD mortality was higher amongst men; 12.5% of men (88 deaths) and 4.8% of women (41 deaths) had died with CHD as the major cause of death.

As expected, men had higher scores on the 'masculinity' scale than women (mean 4.50 vs 4.08,  $P < 0.001$ ) and lower scores on the 'femininity' scale (mean 5.30 vs 5.63,  $P < 0.001$ ) (Table 1). More men than women were classified as 'masculine' (35% vs 14%), and fewer as 'feminine' (13% vs 36%), but similar proportions of men and women were classified as 'androgynous' (24% vs 22%) and 'undifferentiated' (29% vs 28%). Around a quarter of the sample (27% men, 26% women) were more socio-economically advantaged (head of household in a professional or intermediate job), although the reported household income was lower for women than men. More women than men were never smokers (41% vs 23%) and fewer were current smokers (40% vs 51%). Binge drinking in the previous week was much higher in men (33%) than women (5%). Mean GHQ scores were marginally higher in women (11.94, 5.35 SD) than men (10.98, 4.86 SD), whereas the reverse was apparent for systolic blood pressure with slightly elevated values in men [138.02 (21.26 SD) vs 135.99 (20.06 SD)]. Mean BMI did not differ between the sexes. Univariate analyses (data not shown) revealed expected relationships between covariates and CHD death amongst men: a significantly increased hazard ratio (HR) for CHD death associated with being a current smoker (HR = 1.80, 95% CIs 1.33–2.44,  $P < 0.000$ ) and having poorer psychological well-being (HR per unit increase in GHQ score = 1.06, 95% CIs 1.02–1.11,  $P = 0.002$ ). A trend towards higher risk with increasing systolic blood pressure (HR per unit increase = 1.01, 95% CIs 1.00–1.02,  $P = 0.08$ ) was apparent. Perhaps owing to the lower number of deaths, none of the univariate risk ratios were statistically significant for

women although there was some evidence of a relationship between current smoking and CHD mortality (HR = 1.48, 95% CIs 0.98–2.22,  $P = 0.06$ ).

Table 2a shows the relationship between the continuous 'masculinity' and 'femininity' scores for CHD death and all covariates separately for men and women. Similar results are presented in Table 2b for the median-split 4-fold classification.

In men, higher 'femininity' scores were associated with a lower risk of CHD death: the unadjusted HR for each unit increase in femininity score was 0.68 (95% CIs 0.50–0.91,  $P = 0.011$ ) (Table 3). This was little changed following adjustment for other risk factors (HR per unit in femininity score 0.65, 95% CIs 0.48–0.87,  $P = 0.004$ ). No relationship was observed between 'femininity' and CHD in women. There was no relationship between 'masculinity' score and CHD mortality in either men or women (unadjusted HRs 0.99, 95% CIs 0.76–1.30,  $P = 0.950$ ) for men, 0.90, 95% CIs 0.62–1.33,  $P = 0.608$  for women). These ratios altered little when adjusting for other potential risk factors (smoking, binge drinking, BMI, socio-economic status and psychological well-being). There was no evidence of interaction and between 'masculinity' and 'femininity' score for either men or women.

Various sensitivity analyses were performed. First, as 13.6% of men and 22.2% of women had missing data on the binge-drinking variable, the unadjusted and adjusted models were run without binge drinking. Unadjusted and adjusted HRs were very similar to those reported in Table 3 (adjusted HRs: men, masculinity 1.14 (0.89–1.45), femininity 0.69 (0.53–0.90); women, masculinity 0.89 (0.63–1.27), femininity 1.25 (0.78–2.02). Secondly, all models were rerun with any mention of CHD on the death certificate as the outcome. In general, there was a small attenuation in the HRs for men (adjusted risk ratios, masculinity 1.04 (0.81–1.32), femininity 0.75 (0.57–0.98)) and minor discrepancies for

**Table 2a** Relationships between masculinity and femininity scores, CHD death and covariates by gender

	Men ( <i>n</i> = 704)				Women ( <i>n</i> = 847)			
	Masculinity score		Femininity score		Masculinity score		Femininity score	
	<i>n</i>		<i>n</i>		<i>n</i>		<i>n</i>	
<b>CHD death<sup>a</sup></b>								
<i>Died from CHD</i>	79	4.57 (0.95)	79	5.12 (0.81)	38	3.97 (0.92)	38	5.76 (0.78)
<i>Others</i>	567	4.49 (0.92)	567	5.33 (0.76)	731	4.09 (0.97)	733	5.63 (0.76)
<b>Smoking<sup>a</sup></b>								
<i>Never</i>	148	4.50 (0.93)	149	5.21 (0.71)	316	4.08 (0.96)	316	5.64 (0.79)
<i>Ex</i>	170	4.50 (0.85)	170	5.28 (0.76)	152	4.21 (0.94)	152	5.55 (0.79)
<i>Current</i>	328	4.50 (0.96)	327	5.36 (0.81)	301	4.01 (0.99)	303	5.67 (0.71)
<b>Binge drinking<sup>a</sup></b>								
<i>Yes</i>	183	4.54 (0.92)	182	5.36 (0.79)	30	4.04 (1.06)	30	5.60 (0.75)
<i>No</i>	372	4.51 (0.93)	373	5.27 (0.77)	566	4.10 (0.95)	567	5.65 (0.74)
<b>BMI<sup>b</sup></b>	640	0.16 (0.000)	640	0.04 (0.268)	764	0.03 (0.378)	766	0.08 (0.028)
<b>Blood pressure<sup>b</sup></b>	644	0.04 (0.343)	644	0.03 (0.463)	765	-0.06(0.084)	767	0.09 (0.017)
<b>GHQ<sup>c</sup></b>	635	-0.08(0.041)	635	-0.16(0.000)	748	-0.15(0.000)	748	-0.03(0.487)
<b>Income<sup>c</sup></b>	646	0.16 (0.000)	646	-0.13(0.001)	769	0.16 (0.000)	771	0.04 (0.305)

<sup>a</sup> Mean (standard deviation) masculinity and femininity scores.

<sup>b</sup> Correlation ( $P$ -value).

<sup>c</sup> Spearman's rho ( $P$ -value).

**Table 2b** Relationship between BSRI 4-fold classification, CHD death and covariates by gender

	Men (n = 704)								Women (n = 847)							
	Androgynous		Masculine		Feminine		Undifferentiated		Androgynous		Masculine		Feminine		Undifferentiated	
	n	%	n	%	n	%	n	%	n	%	n	%	n	%	n	%
<b>CHD death<sup>a</sup></b>																
Dead CHD	15	9.8	37	16.6	10	11.9	17	9.2	9	5.3	3	2.8	15	5.5	11	5.1
Others	138	90.2	186	83.4	74	88.1	168	90.8	162	94.7	105	97.2	259	94.5	206	94.9
<b>Smoking<sup>a</sup></b>																
Never	36	23.5	62	27.8	17	20.2	33	17.8	68	39.8	46	42.6	115	42.1	87	40.1
Ex	42	27.5	58	26.0	18	21.4	52	28.1	38	22.2	25	23.1	42	15.4	47	21.7
Current	75	49.0	103	46.2	49	58.3	100	54.1	65	38.0	37	34.3	116	42.5	83	38.2
<b>Binge drinking<sup>a</sup></b>																
Yes	37	28.2	62	32.3	27	32.1	56	35.2	6	4.9	4	4.9	13	6.0	7	4.0
No	94	71.2	130	67.7	45	62.5	103	64.8	116	95.1	78	95.1	203	94.0	170	96.0
<b>BMI<sup>b</sup></b>	151	25.3 (3.8)	222	26.3 (3.5)	83	25.7 (4.1)	184	26.3 (3.6)	169	25.8 (5.0)	108	25.4 (4.0)	272	26.0 (5.0)	215	26.3 (5.0)
<b>Blood pressure<sup>b</sup></b>	153	135.7 (18.1)	222	138.6 (20.7)	84	140.2 (24.2)	185	138.5 (22.8)	171	137.5 (18.8)	108	132.1 (18.3)	271	137.3 (20.7)	215	135.5 (20.9)
<b>GHQ<sup>c</sup></b>	152	11 (6)	217	10 (4)	82	9.5 (6.25)	183	9 (5)	167	11 (5)	105	10 (4)	267	12 (6)	209	11 (5)
<b>Income<sup>c</sup></b>	153	144.5 (112.5)	223	184.6 (149.2)	84	134.2 (115.9)	185	150 (108.9)	171	120 (99.5)	108	144.5 (110.7)	273	134.0 (94.0)	217	120 (99.5)

<sup>a</sup> Percentage;  
<sup>b</sup> Mean (SD);  
<sup>c</sup> Median (interquartile range).

**Table 3** Hazards ratios (95% confidence interval) for the relation of a unit increase in masculinity and femininity scores with CHD mortality by gender

	Men		Women	
	Unadjusted hazard ratio P value	Adjusted hazard ratio+ P value	Unadjusted hazard ratio P value	Adjusted hazard ratio+ P value
Masculinity score	0.99 (0.76–1.30) 0.950	1.05 (0.80–1.38) 0.721	0.90 (0.62–1.33) 0.608	0.99 (0.67–1.46) 0.958
Femininity score	0.68 (0.50–0.91) 0.011	0.65 (0.48–0.87) 0.004	1.17 (0.71–1.95) 0.537	1.18 (0.70–2.01) 0.529

+ Adjusted for smoking, binge drinking, BMI, systolic BP, household income and psychological well-being (GHQ score).

**Table 4** Hazards ratios (95% confidence interval) for the relation of a BSRI 4-fold classification with CHD mortality by gender

	Men		Women	
	Unadjusted hazard ratio (confidence interval) (P value)	Adjusted hazard ratio+ (confidence interval) (P value)	Unadjusted hazard ratio (confidence interval) (P value)	Adjusted hazard ratio+ (confidence interval) (P value)
‘Androgynous’	1.00	1.00	1.00	1.00
‘Masculine’	1.58 (1.08–2.31) (0.018)	1.69 (1.15–2.47) (0.007)	0.79 (0.32–1.95) (0.611)	0.91 (0.37–2.25) (0.832)
‘Feminine’	1.00 (0.55–1.81) (0.988)	0.90 (0.49–1.65) (0.736)	1.23 (0.69–2.18) (0.484)	1.14 (0.63–2.05) (0.666)
‘Undifferentiated’	0.70 (0.43–1.14) (0.149)	0.71 (0.43–1.16) (0.165)	0.98 (0.52–1.87) (0.962)	1.03 (0.54–1.97) (0.928)

+ Adjusted for smoking, binge drinking, BMI, systolic BP, household income and psychological well-being (GHQ score).

women (adjusted HRs, masculinity 1.15 (0.83–1.59), femininity 1.21 (0.78–1.88)).

For the purposes of comparability with results from the Rancho Bernardo cohort, Table 4 reports analyses using the median split method. In comparison with men who were classified as ‘androgynous’ (i.e. had ‘high’ masculinity and ‘high’ femininity scores), those who were classified as

‘masculine’ (i.e. had ‘high’ ‘masculinity’ and ‘low’ ‘femininity’ scores) had a higher risk of CHD mortality (unadjusted HR = 1.58, 95% CIs 1.08–2.31, P = 0.018). After adjustment for other risk factors, the HR increased somewhat (Adjusted HR = 1.69, 95% CIs 1.15–2.47, P = 0.007). However, the men classified as ‘feminine’ (i.e. high ‘femininity’ and low ‘masculinity’) and ‘undifferentiated’ (i.e. low ‘femininity’ and low

'masculinity') did not differ from the 'androgynous' men in CHD risk. No differences in CHD risk were seen between the groups amongst women.

## Comment

Although there has been increasing attention in recent years to men's health,<sup>39–41,11,12,15</sup> some focusing specifically on men's experience of heart disease,<sup>9,18,42–44</sup> empirical research linking experiences of masculinities to health is limited. It is ironic that, although there has been a predominance of male subjects in research on heart disease (until recently at least),<sup>45</sup> earlier research on heart disease was gender neutral, failing to recognize the ways in which gendered expectations and experiences are socially constructed and may contribute to men's increased risk of heart disease.

Our results showed a relationship between lower levels of 'femininity', or expressive traits, and increased CHD mortality in men but not women, in a cohort in which we saw the expected relationship between key traditional risk factors<sup>46</sup> and CHD mortality. Scores on a 'masculinity', or instrumental dimension did not relate to CHD risk in either men or women. These results, if replicated in other studies, raise a number of questions about why higher levels of stereotypically feminine or expressive traits are associated with lower CHD risks in men. In this analysis, we have been best able to address the first of the pathways proposed by Hemingway and Marmot (that psychosocial factors are linked to CHD indirectly via health behavioural risk factors). However, whilst we have adjusted for two important health behavioural risk factors (smoking and binge drinking), we were not able to control for other such factors (e.g. diet, general orientation towards health maintenance, etc). Similarly, we do not have the measures in this study to address any direct pathways via acute or chronic physiological changes; studies with more biological measures are required to investigate such pathways. Similarly, a different design would be required to investigate how instrumentality and expressiveness relate to the uptake of medical care, although it is perhaps relevant to note that a study of college students in the US reported the 'unexpected' finding that men with high 'femininity' scores (as measured by the BSRI) were more concerned about health risks than other men or women.<sup>47</sup>

As far as we are aware, only one other study has examined whether 'masculinity' and 'femininity' are related to CHD mortality.<sup>3</sup> Barrett-Connor reported a preliminary analysis of CHD deaths in men and women in the Rancho Bernardo cohort in the USA, and found no association between 'masculinity', 'femininity' and 'androgenicity' (also measured using the BSRI using the median-split method) after 6 years of follow-up. However, this analysis was based on a small number of deaths (14 in men and 10 in women). When using the 'median-split' method in our analyses, the increased risk associated with low 'femininity' scores was confined to men who had high 'masculinity' scores (i.e. the 'masculine' men). The 'feminine' men (high F, low M) and the 'undifferentiated' men (low F, low M) did not differ from the androgynous men (high M, high F). Thus, the two methods of analysing the relationship between GRO and CHD mortality are only partially consistent.

Both suggest that GRO is not associated with CHD risk in women. The 'continuous' method suggests that higher femininity scores are protective in men (irrespective of masculinity scores since the interaction term was not significant). However, the 'median-split' method suggests that lower 'femininity' scores were only detrimental when men also had high 'masculinity' scores, possibly equating with a more 'extreme' version of masculinity. Helgeson has reported a relationship between 'negative or extreme masculinity' (also referred to as 'unmitigated agency') and heart attack severity amongst US men (not mediated by type A behaviour, poor health practices or impaired social networks)<sup>18</sup> and suggests that there is 'emerging evidence that aspects of masculine identity and male socialization are related to men's risk for coronary heart disease' (p. 68).<sup>9</sup>

Our study has a number of strengths. It is a community-based general population sample suggesting a high degree of generalizability of our findings. We have: accurate, independent reports of deaths; validated and widely used measures of GRO and psychological well-being; and directly measured height, weight and blood pressure. The study also has limitations. First, because we are comparing men and women of the same age, we have relatively few CHD deaths in women and therefore reduced statistical power for detecting relationships between any risk factors and CHD mortality. Secondly, we do not have measurements of some important CHD risk factors such as HDL cholesterol. Thirdly, had our sample size been large enough to test more robustly for interactions between the 'masculinity' and 'femininity' scores there may not have been discrepancies between the two methods.

As we have noted elsewhere,<sup>48</sup> theorists of masculinity<sup>10</sup> have stressed the complex and multifaceted nature of masculinity, or masculinities. Here we have used an instrument which taps into some dimensions of stereotypical masculinity and femininity, but clearly has limitations.<sup>32</sup> The BSRI allows people to rate themselves against 'some' aspects contributing to common cultural notions of hegemonic (or dominant) forms of masculinity (which might be health-damaging, such as being willing to take risks and being aggressive) and femininity, but is inevitably limited. Hoffman and Borders argue that there is now a challenge to think of new models for conceptualizing masculinity and femininity 'in non-stereotypical ways', and that the BSRI may 'best be used to examine one's instrumentality and expressiveness, providing that the importance attached to one's classification is minimized and providing that instrumentality and expressiveness are described as human traits rather than as synonyms for masculinity and femininity' (p. 53).<sup>32</sup>

Our results present some empirical support for theories that social constructions of gender are important for health, and suggest that this is an important area to study further. We believe that it is important to explore ways in which societal constructions of gender may explain sex differences in longevity, and in specific diseases like CHD which contribute substantially to premature mortality and to inequalities in health by gender (and other social characteristics). We hope that these results, which suggest that men who are less able to identify themselves with characteristics that have been identified as being 'feminine' or expressive (and thus perhaps subscribe to a more limited and stereotypically



**KEY MESSAGES**

What is already known:

- Men have higher rates of CHD than women.
- Established risk factors for CHD do not fully explain these gender differences.
- The role of social constructions of gender as a potential risk factor for CHD has been little examined.

What this article adds:

- In the largest study to date, men who are less able to identify themselves with characteristics stereotypically defined as 'feminine' or 'expressive' may experience increased risk of CHD.
- This relationship persisted after adjusting for smoking, binge drinking, BMI, systolic blood pressure, household income and psychological well-being.

'masculine' range of behaviours) are at an increased risk of dying of CHD, will stimulate discussion and further research into the links between the social construction of gender and health.

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