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Marital status and cardiovascular mortality: Behavioural, psychological distress and metabolic explanations.

Abstract

The intermediate processes through which the various unmarried states can increase the risk of subsequent cardiovascular disease (CVD) mortality are incompletely understood. An understanding of these processes and how they may vary by gender is important for understanding why marital status is strongly and robustly associated with subsequent cardiovascular disease. In a prospective study of 13,889 men and women (mean age 52.3, SD: 11.8 yrs, range 35-95, 56.1% female) without a history of clinically diagnosed CVD, we examined the extent to which health behaviours (smoking, alcohol, physical activity), psychological distress (General Health Questionnaire-12 item) and metabolic dysregulation (obesity levels, and the presence of hypertension and diabetes) account for the association between marital status and cardiovascular mortality. There were 258 cardiovascular deaths over an average follow up of 7.1 (SD=3.3) years. The risk of cardiovascular mortality was greatest in single, never married men and separated/divorced women compared with those that were married in gender stratified models that were adjusted for age and socioeconomic group. In models that were separately adjusted, behavioural factors explained up to 33% of the variance, psychological distress explained up to 10% of the variance and metabolic dysregulation up to 16% of the variance in the observed significant associations between marital status and cardiovascular mortality. Behavioural factors were particularly important in accounting for the relationship between being separated/divorced and cardiovascular mortality in both men and women (33% and 21% of variability, respectively). The present findings suggest that health behaviour, psychological distress and metabolic dysregulation data have varying explanatory power for understanding the observed relationship between CVD mortality and unmarried states.

SD = Standard Deviation; CVD = cardiovascular disease, GHQ =General Health Questionnaire, BMI =Body Mass Index.

Key words: marriage, cardiovascular disease, health behaviour, distress.

Introduction

There is good evidence that structural aspects of an individual's social relationships can predict all cause mortality (House, 2001; House, Landis & Umberson, 1988) and mortality from a range of clinical conditions across adulthood (Kaplan & Kronick, 2006), particularly conditions relating to cardiovascular disease (Brummett, Barefoot, Siegler, Clapp-Channing, Lytle, Bosworth et al., 2001; Lett, Blumenthal, Babyak, Strauman, Robins & Sherwood, 2005). This has been clearly demonstrated in the literature examining the relationship between marital status and health outcomes (Kiecolt-Glaser & Newton, 2001). All of the various unmarried states (being single never married, being separated/divorced and being widowed) have been associated with elevated mortality risks (Ikeda, Iso, Toyoshima, Fujino, Mizoue, Yoshimura et al. 2007; Johnson, Backlund, Sorlie & Loveless, 2000; Manzoli, Villari, Pirone & Boccia, 2007).

The two main explanations that have been proposed in accounting for these observations are social selection and social causation theory (Joung, van de Mheen, Stronks, van Poppel & Mackenbach, 1998). Although these are non-mutually exclusive explanations with respect to marriage and health, social selection usually refers to the selection of healthier individuals into marriage and unhealthy persons into unmarried states whereas social causation refers to the social/economic resources, sometimes referred to as the protective or social support consequences of marriage, and better health behaviours (this can also be a selection effect through assortive mating) that can accompany the married state and promote health and the harmful consequences of bereavement or marital dissolution experienced by widowed persons and the separated or divorced. Although social selection and social causation represent contrasting accounts at an ultimate level of explanation (Tinbergen, 1963), the proximate biobehavioural mechanisms are likely to be shared in social selection and social causation e.g. health behaviour, psychological distress, stress-related pathophysiological responses.

A range of studies have demonstrated that behavioural (Molloy, Perkins-Porras, Strike & Steptoe, 2008; Umberson, 1992), psychological distress (Kessler & Essex, 1982;

Umberson, Wortman & Kessler, 1992) and pathophysiological mechanisms (Uchino, 2006) that can influence morbidity and mortality from cardiovascular disease (CVD) are associated with various states of social isolation. In comparison with the other leading causes of mortality (e.g. cancer, respiratory conditions, infectious disease and external causes) theoretical models linking marital status with processes that are known to directly influence CVD mortality have been more completely outlined and tested e.g. cardiovascular reactivity (Kiecolt-Glaser & Newton, 2001). However there are few reliable estimates and comparisons of the extent to which these mechanisms can potentially explain the association between each of the unmarried states and risk of CVD mortality. The present study uniquely addresses this gap in this literature. This type of analysis is required to move our understanding of marriage and it's role in the pathogenesis of CVD forward, as there are potentially differing mechanisms, which may be more or less important in the various unmarried states (Kiecolt-Glaser & Newton, 2001). Although these three classes of mechanisms are clearly interdependent, a comparison of the separate explanatory power of these could inform what intervention strategies might be most effective in reducing the risk associated with being unmarried i.e. behaviour change, psychotherapy and biomedical intervention.

One of the strongest recurrent findings in this literature on marital status and health has been the presence of gender differences in the relationship between marital status and health outcomes (Umberson, 1992). Being married is associated with greater protection for men compared to women, therefore gender stratified analysis have become commonplace in much of this work (Kaplan & Kronick, 2006; Scafato, Galluzzo, Gandin, Ghirini, Baldereschi, Capurso et al. 2008). Various explanations have been proposed for observed gender differences in the marriage-health relationship, namely gender differences in the social control of health behaviour, with women being more likely to control others health behaviour (Umberson, 1992) and the qualitative differences between men and women's support networks, with men more likely to rely on wife or partner as the main source of support, whereas women may have several close confidants. The extent of the differences between the intermediate processes between marital status and CVD mortality in men and women

has not to date been well charaterised. Therefore the analysis also aimed to examine gender differences in CVD mortality and potential intermediate mechanisms according to marital status.

We analysed data from the Scottish Health Survey (The Scottish Government Statistics, 2008) to address the following questions:(i) How much of the association between marital status and cardiovascular mortality can be explained by behavioural, psychological distress and metabolic dysregulation (ii) Does the relative contribution of behavioural, psychological distress and metabolic processes vary across the marital status categories i.e. being single never married, being separated/divorced and being widowed. In this study we eliminated individuals with previously clinically diagnosed CVD in order to assess the relationship between marital status and cardiovascular mortality in a population that were free from clinically confirmed CVD at baseline.

Methods

Sample

The Scottish Health Survey (SHS) is a periodic survey (typically every 3-5 years) that draws a nationally representative sample of the general population living in households. The sample was drawn using multistage stratified probability sampling with postcode sectors selected at the first stage and household addresses selected at the second stage. Different samples were drawn for each survey. The present analyses combined data from the 1995, 1998 and 2003 SHS in adults aged 35 yrs and older. The overall response rate ranged between 60-76% for the different survey years (The Scottish Government Statistics, 2008). Participants gave full informed consent to participate in the study and ethical approval was obtained from the London Research Ethics Council. Out of a total of 16,144 we excluded 1094 participants (7%) with a previous clinical history of CVD or cancer. There were 1151 participants with incomplete data (7%), therefore there was complete data available for 13,889 participants. This sample comprised the dataset for the present analysis.

Baseline assessment

Survey interviewers visited eligible households and collected data on demographics and health behaviours (physical activity, smoking, alcohol intake). There were 6 possible categories for marital status: 1. married, 2. co-habiting, 3. widowed, 4. divorced, 5. separated or 6 single and never married. For the purpose of this study 4 marital status categories were created namely 1. Married/co-habiting, 2. Single, never married, 3. Separated/divorced and 4. Widowed. On a separate visit nurses collected information on medical history, and took anthropometry variables (height, weight, waist circumference) from consenting adults. Detailed information on the survey method can be found elsewhere (The Scottish Government Statistics., 2008).

Predictor and outcome variables

Current mental health was assessed from the 12 item version of the General Health Questionnaire (GHQ-12), which is a measure of psychological distress devised for population studies. The questionnaire comprises twelve questions, asking informants about their general level of happiness, experience of depressive and anxiety symptoms, and sleep disturbance over the last four weeks. Interpretation of the answers is based on a four point response scale scored using a bimodal method (symptom present: 'not at all' = 0, 'same as usual' = 0, 'more than usual' = 1 and 'much more than usual' = 1). The GHQ-12 is a highly validated instrument and has been strongly associated with various psychological disorders such as depression and anxiety (Goldberg, Gater, Sartorius, Ustun, Piccinelli, Gureje et al. 1997). We used a score of ≥ 4 to define possible 'caseness' of psychological distress according to studies validating the GHQ-12 against standardised psychiatric interviews (Goldberg et al., 1997). Existing hypertension and diabetes was confirmed from self reported doctor's diagnosis, which is generally considered as being reliable (Colditz, Martin, Stampfer, Willett, Sampson, Rosner et al. 1986). Obesity was defined as body mass index ≥ 30 kg/m^2. Health behaviours were measured using self report questionnaires. Physical activity questions inquired about participation in the four weeks prior to the interview. Frequency, duration, and intensity of participation was assessed across three domains of activity: leisure time sports (e.g., cycling, swimming, running, aerobics, dancing, and ball sports such as football and tennis), walking for any purpose, and domestic physical activity (e.g., heavy

housework, home improvement activities, manual and gardening work). Health behaviours were treated as categorical variables: physical activity was categorised into five groups according to frequency of any activity lasting at least 30 minutes (reference group no activity, <1/wk, 1-2 /wk, 3-4/wk, ≥5/wk); smoking was categorised into five groups (reference group never smoked, past smokers, current smokers <10 cigarettes/day, 10-20 cigarettes/day, >20 cigarettes/day); alcohol intake was quantified in units per week (1 unit = half pint beer, a small glass of wine, or a measure of spirits) and categorised into sex specific tertiles with the highest tertile representing hazardous levels (14+ units for women/ 21+ units for men).

The main outcome was cardiovascular mortality and all cause mortality. This information was obtained from a patient-based database of CVD hospital admissions and deaths (Information Services Division [ISD] Scotland) that was linked to the surveys. The ISD database has demonstrated 94% accuracy and 99% completeness when samples of computerized CVD records from the Scottish national database were compared with the original patient case notes. Classification of the underlying cause of death was obtained from the General Registrar Office for Scotland and was based on information collected from the death certificate together with any additional information provided subsequently by the certifying doctor. Mortality from cardiovascular causes was coded according to International Classification of Diseases - Version 9 (ICD-9) (390-459) and ICD-10 (I01-I99). Data on CVD hospital admissions were available between 1980 and September 2006 that allowed us to exclude 846 participants with existing CVD at baseline.

Statistical analysis

Logistic regression was used to examine associations between marital status categories and behavioural, psychological distress and metabolic risk factors. These models included adjustments for age. Cox proportional hazards models were used with months as the time scale to estimate the risk of cardiovascular and all cause mortality according to marital status. For participants who survived the data were censored to September 2006. The proportional hazards assumption was examined by comparing the cumulative hazard plots grouped on exposure, although no appreciable violations were noted. In the basic multivariate model we adjusted for potential confounders including age (continuous score**)** and socioeconomic

group using the Registrar General Classification (categories: I/II professional/intermediate, III skilled non-manual/ skilled manual, IV/V part-skilled/unskilled) as these two variables have known relationships to marital status e.g. the widowed are much older and the unmarried are more likely to be in lower socio-economic groups. In order to test the extent to which behavioural, psychological distress and metabolic dysregulation accounted for the association between marital status and CVD mortality, we grouped together CVD risk factors considered to potentially explain the association on an a priori basis. This included behavioural factors (physical activity, smoking, alcohol, treated as categorical variables), psychological distress (GHQ-12 treated as a continuous score), a metabolic dysregulation factor (body mass index, the presence of hypertension and diabetes, treated as categorical variables). We separately added these risk factors, one set at a time, into the basic model. Finally we performed a fully adjusted analysis that included all of the factors simultaneously. The proportion of CVD risk reduction explained by each set of factors was computed as follows: $(HR_{basic\ model} - HR_{adjusted})/ HR_{basic\ model} - 1) \times 100$. We used ANOVA tests to examine continuous variables across the marital categories. All analyses were performed using SPSS (version 14) and all tests of statistical significance were based on two-sided probability.

Results

The mean age of the sample was 52.3 years (SD: 11.8, range 35-95) and 56.1% were female. There were a total of 892 deaths, 258 (28.9%) were due to CVD and 353 (39.6%) were due to cancer over an average of 7.2 years of follow up. Coronary heart disease accounted for 65.1%, cerebrovascular diseases 26.7%, and aortic aneurysm 4.3%, of all cardiovascular deaths. At baseline, 65% of participants were married/co-habiting, 11 % were single, never married, 14.4% were separated/divorced and 9.5% were widowed.

As shown in table 1 there were significant age differences across the 4 marital categories for men and women ($p<0.01$). Table 1 therefore present age adjusted logistic regression models that examine the associations between marital status and behavioural, psychological distress and metabolic risk factors. There was no association between being unmarried and being physically inactive for men and women. All unmarried categories for

men and women were significantly more likely to smoke than married individuals. Separated/divorced and widowed men were more likely to engage in hazardous drinking. All unmarried categories for men and women were significantly more likely to experience psychological distress (GHQ $\geq$ 4). Separated/divorced men were more likely to have a diagnosis of hypertension. Single, never married men and women were more likely to have diabetes. Further details are provided in Table 1.

Table 2 demonstrates the gender stratified hazard ratios for all cause mortality and cardiovascular mortality in the unmarried versus the married groups. An age adjusted test for the interaction between marital status and gender showed that there was a significant interaction for all cause mortality (p= 0.025), however this was not observed for CVD mortality. All unmarried states were associated with a significantly higher risk of all cause mortality with the exception of separated/divorced women. All unmarried states were associated with a higher risk of cardiovascular mortality with the exception of widowed women. In general, the risk of death was higher for cardiovascular causes, especially in the case of single or widowed men, and separated/divorced women. In sensitivity analysis we restricted the analysis to participants who were greater than 50 and less than 80 given that a primary cause of CVD mortality is most typical of this age group. We found that the overall pattern of results did not change. For example in single, never married men (N=3,160, 124 CVD deaths) the hazard ratio for CVD mortality was 2.97 (95% CI 1.85-4.78) and for single, never married women (N=4,213, 93 CVD deaths) hazard ratio was 2.23 (1.17-4.24).

Table 3 presents the gender stratified analysis for the marital status and cardiovascular mortality with separate adjustments for health behaviours, psychological distress and metabolic factors. All unmarried groups had significantly higher risk of CV mortality relative to the married/co-habiting with the exception of widowed women in age and SES adjusted models. Table 3 present the details of the adjusted analysis for health behaviours (physical activity, smoking and alcohol), psychological distress and metabolic dysregulation (hypertension, diabetes and BMI). As there was not a significant association between being widowed and CVD mortality for women we did not investigate potential intermediate mechanisms in any more detail. The results show that inclusion of the health

behaviour data in the models was associated with attenuation in the strength of the relationship between marital status and CVD mortality for all categories of unmarried status for men, but only in the separated/divorced category for women. It is clear that inclusion of health behaviour data is associated with much greater attenuation in the observed relationship between being separated or divorced and CVD mortality than the two other unmarried categories. Including psychological distress in the models was associated with attenuation in the observed relationship between being unmarried and CVD mortality with attenuation ranging from 2.8% for single, never married women to 10.3% for separated/divorced women and between 5.2% for widowed men and 8.8% for separated/divorced men. Finally including metabolic dysregulation variables (presence of hypertension, diabetes and BMI) in the models was associated with a between 3.2% and 16% attenuation for women and between a 4.4% and 8.8% attenuation in the observed relationship between being unmarried and CVD mortality for unmarried men.

Discussion

The present data once again demonstrated the increased cardiovascular mortality risks for unmarried men and women. The associations observed in the present data were largely concordant with two recent population studies from the US (Kaplan & Kronick, 2006) and Japan (Ikeda et al., 2007) and several older studies (Ben-Shlomo, Smith, Shipley & Marmot, 1993; Ebrahim, Wannamethee, McCallum, Walker & Shaper, 1995; Johnson et al., 2000), suggesting that these relationships are highly robust across time and place. The unique contribution of the present study was to focus on the extent to which health behaviours, psychological distress and metabolic dysregulation can account for the association between the various unmarried categories and cardiovascular mortality risk. The present analyses show that between 16% and 39% of the variance in the observed relationships between being unmarried and CVD mortality can be accounted for by these variables. Health behaviour data emerged as being particularly important in explaining the observed relationship between being unmarried and CVD mortality among men. This is consistent with the social control hypothesis of marital relationships that argues that women are more likely to regulate men's health behaviour in marital relationship (Umberson, 1992).

However the findings clearly indicate that the explanatory power of health behaviour data varies greatly depending on the unmarried category for both men and women. In men health behaviour data has a relatively lower explanatory value for the CVD mortality risk associated with being single, never married and with being widowed compared with the risk associated with being separated or divorced and in women health behaviour data is only of value in explaining the CVD risk associated with being separated or divorced.

Health behaviours emerged as a particularly important variable in understanding the cardiovascular mortality risk associated with being separated/divorced for both men and women. Health behaviour explained 33% of the association observed between being separated/divorced and cardiovascular mortality in men and 21% of this association in women suggesting that poor health behaviour has significant explanatory power for understanding differences in health outcomes in this group. The observed key role of health behaviour in accounting for cardiovascular mortality is in line with a previous population study in the Netherlands examining self-rated health (Joung, Stronks, van de & Mackenbach, 1995). The results also confirm that a large part of the association between being unmarried and mortality can not be explained by the key health behaviours, aspects of psychological distress and metabolic dysregulation as measured in this study. In women in particular the data suggests that the increased CVD mortality risk associated with being single never married is not accounted for by health behaviour or emotional distress by any appreciable amount. As prior evidence supports direct influences of marriage on a range of biological markers (Kiecolt-Glaser & Newton, 2001), such as immune and endocrine processes, it is possible that these direct processes may be relatively more important for women than men when compared with indirect processes through health behaviour and psychological distress in accounting for the elevated CVD risk associated with being a single, never married women. The present data would provide some support for this contention. However, it is important to note that we did not assess other potentially important psychobiological indicators, such as inflammatory markers, hemodyamic and autonomic nervous system functioning, and lipid profiles, which have known relationships with other psychosocial variables and CVD outcomes. In addition there are some limitations to the statistical power of

the analysis for women in particular, as there are a low number of events for some unmarried categories e.g. single, never married women, which can limit the reliability of the estimates.

The lack of association between being separated/divorced and all cause mortality and being widowed and cardiovascular mortality in women in the present study supports the findings of a previous study that found no association between being separated/divorced and widowed and all cause mortality in women (Cheung, 2000). This finding is also compatible with the argument in this literature that women benefit less from the presence of a marital relationship and that marital disruption is more damaging for men than women (Kiecolt-Glaser & Newton, 2001). The higher risk of cardiovascular disease in men and women compared with non-cardiovascular death in the present data suggesting that marriage might may be particularly related to mechanisms specifically affecting CVD risk. One key set of behaviours that may be relevant and have been shown to be related to marital status and relationship quality are secondary prevention behaviours following the onset of a condition related to CVD e.g. cardiac rehabilitation attendance (Molloy, Hamer, Randall & Chida, 2008) and medication adherence (Molloy et al., 2008), however such measures were not available in the present study.

As we have acknowledged in the introduction the three classes of mechanism that are examined in this analysis are highly interdependent. For example it is clear that hazardous drinking can be a risk factor for the development of subsequent psychological distress and obesity or diabetes. This can make statistical models with simultaneous adjustment of interdependent processes very difficult to interpret and we would encourage researchers in this area to consider the issue of over-adjustment in statistical models that attempt to identify important intermediate mechanisms in the marital status-health relationship, which can be obscured if analysis are not driven by a specific research question that is theoretically informed by conceptual models that consider the interdependencies of intermediate processes. Future work should also consider in more detail the interactions between these processes and marital status.

There are several limitations to the current study which should be acknowledged. As data on marital status was collected at one time point, we were unable to look at the

influence of marital transitions (Ebrahim et al., 1995) on cardiovascular mortality or the influence of CVD events on marital transitions. This would have allowed a more conclusive analysis about issues relating to health selection and social causation. In relation to this we did not have information on the number of years married or the time since or number of separation(s)/divorce(s) or bereavement(s). This information is important in that it represents time since and intensity of exposure to a protective or deleterious social conditions (Zhang, 2006). The data set did not have any measures of marital quality for the married participants. Several studies have shown that the quality of the marital relationship can contribute to increased risk for CVD (De Vogli, Chandola & Marmot, 2007; Eaker, Sullivan, Kelly-Hayes, D'Agostino, Sr. & Benjamin, 2007). Several of the measures, including smoking and physical activity, were assessed by self-report at one time point only, which precludes a formal mediation analysis as the temporal relationship between marital status and intermediate mechanisms cannot be established. More precise and repeated assessment of these variables would have allowed for a more formal and compelling mediational analysis. More generally the study is also subject to the usual limitations of survey methodology e.g. certain groups may be underrepresented (the homeless, prisoners, psychiatric hospital residents), and while the data linkage process has been validated it remains imperfect e.g. deaths that happen outside of the UK and that are not registered will not be detected. Finally the distinction between marriage and co-habitation status was not investigated in the present analysis (Scafato, Galluzzo, Gandin, Ghirini, Baldereschi, Capurso et al. 2008). While this is an important related issue it was viewed to be beyond the scope of the current research questions.

There are however several notable strengths to the present study including the large, community-based representative sample that excluded those with clinically confirmed CVD at baseline. The prospective and retrospective data linkage to National Health Service databases in Scotland represents a unique resource to examine the relationship between key psychosocial variables such as marital status and subsequent health outcomes, while controlling for variables related to previous clinical diagnoses. The analysis presents for the first time precise estimates of the extent to which key behavioural, emotional and metabolic

variables can partly explain the observed relationship between marital status categories and cardiovascular mortality for men and women. This work adds to the growing and increasingly influential body of evidence demonstrating the key role of structural social network phenomena such as marital relationships in understanding health behaviours and disease at the population level (Christakis & Fowler, 2007; Christakis & Fowler, 2008; Iwashyna & Christakis, 2003). The present findings can guide future work attempting to unravel the key proximate mechanisms that can explain the relationship between marital status and CVD morbidity and mortality. In terms of practical application the findings would support approaches that emphasis health behaviour change in those that may be at risk of conditions related to CVD because of their martial status, particularly men, as these variables appear to have the greatest explanatory power in accounting for the marriage-CVD relationship.

Table 1. Age adjusted logistic regression models for marital status and behaviour, psychological distress and metabolic dysregulation in healthy participants† stratified by gender

		Behavioural			Distress	Metabolic	
		Physical Inactivity	**Current Smoker**	**Hazardous Alcohol**	**Psychological Distress**	**Obesity (BMI >30)**	**Hypertension**
	N	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)	OR (95% CI)
Men							
Married	4272	1.00	1.00	1.00	1.00	1.00	1.00
Single	788	0.92 (0.79 – 1.08)	1.77 (1.51 – 2.07)	1.06 (0.90 – 1.25)	1.50 (1.22 – 1.86)	1.06 (0.88 – 1.27)	1.18 (0.98 – 1.43)
Sep/Div	746	1.11 (0.95 – 1.31)	2.70 (2.35 – 3.24)	1.76 (1.50 – 2.07)	2.45 (2.02 – 2.97)	0.73 (0.60 – 0.90)	1.35 (1.22 – 1.63)
Widowed	295	0.99 (0.77 – 1.27)	1.59 (1.22– 2.08)	1.37 (1.05 – 1.80)	2.05 (1.49 – 2.84)	0.84 (0.62 -1.14)	0.96 (0.74 – 1.26)
Women							
Married	4757	1.00	1.00	1.00	1.00	1.00	1.00
Single	743	1.13 (0.96 – 1.33)	1.41 (1.19 – 1.67)	1.01 (0.70 – 1.43)	1.29 (1.05 – 1.59)	1.15 (0.95 – 1.39)	1.06 (0.87 – 1.28)
Sep/Div	1258	1.05 (0.92 – 1.20)	2.45 (2.16 – 2.79)	1.15 (0.88 – 1.51)	2.35 (2.03 – 2.72)	1.07 (0.92 – 1.24)	1.06 (0.90 – 1.24)
Widowed	1030	1.09 (0.94 – 1.27)	2.03 (1.73 – 2.39)	0.56 (0.34 – 0.92)	1.77 (1.46 – 2.15)	1.09 (0.92 – 1.30)	0.93 (0.79 – 1.09)

…..Table 1 continued.

		Diabetes	Age (Mean SD)
	N	OR (95% CI)	
Men			
Married	4272	1.00	51.82 (11.25)
Single	788	1.48 (1.00 – 2.19)	48.94 (11.30)
Sep/Div	746	1.15 (0.74 – 1.78)	50.09 (9.93)
Widowed	295	1.10 (0.67 – 1.81)	66.07 (11.66)
Women			
Married	4757	1.00	51.19 (10.85)
Single	743	1.83 (1.24 – 2.70)	50.46 (12.59)
Sep/Div	1258	1.18 (0.79 – 1.74)	48.91 (9.87)
Widowed	1030	0.97 (0.67 – 1.39))	65.73 (10.48)

†Participants with previous hospitalisation for CVD excluded from all analyses.

Table 2. Age adjusted Cox regression models for marital status and mortality in healthy participants† stratified by gender

		All cause death		CVD death	
	N	Deaths	HR (95% CI)	Deaths	HR (95% CI)
Men					
Married	4272	231	1.00	66	1.00
Single	788	91	2.52 (1.97 – 3.21)	31	3.02 (1.97 – 4.63)
Sep/Div	746	84	2.25 (1.75 – 2.90)	23	2.04 (1.25 – 3.34)
Widowed	295	65	1.83 (1.37 – 2.45)	25	2.51 (1.53 – 4.10)
Women					
Married	4757	182	1.00	40	1.00
Single	743	53	1.66 (1.22 – 2.26)	15	1.99 (1.10 – 3.62)
Sep/Div	1258	54	1.28 (0.94 – 1.73)	23	2.59 (1.55 – 4.33)
Widowed	1030	132	1.38 (1.08 – 1.76)	35	1.37 (0.84 – 2.23)

†Participants with previous hospitalisation for CVD excluded from all analyses.

Table 3 Adjusted analyses for the association between marital status and CVD death *(% attenuation in relationships by adjustments)*[1].

	Deaths/N	Model 1 HR (95% CI)	Model 2 HR (95% CI)	Model 3 HR (95% CI)	Model 4 HR (95% CI)	Fully adjusted HR (95% CI)
Men						
Married	66/ 4272	1.00	1.00	1.00	1.00	1.00
Single	31/ 788	2.71 (1.76 – 4.17) *(15%)*	2.55 (1.65 – 3.93) *(9.4%)*	2.70 (1.75 – 4.16) *(8.1%)*	2.56 (1.66 – 3.97) *(8.8%)*	2.44 (1.57 –3.78) *(15.8%)*
Sep/Div	23/ 746	1.91 (1.17 – 3.14) *(13%)*	1.61 (0.97 – 2.68) *(33%)*	1.83 (1.11 – 3.01) *(8.8%)*	1.87 (1.13 – 3.08) *(4.4%)*	1.56 (0.93 – 2.63) *(38.5%)*
Widowed	25/ 295	2.34 (1.43 – 3.83) *(11%)*	2.17 (1.33 – 3.56) *(12.7%)*	2.27 (1.39 – 3.72) *(5.2%)*	2.27 (1.38 – 3.73) *(5.2%)*	2.11 (1.29 – 3.47) *(17.2%)*
Women						
Married	40/ 4757	1.00	1.00	1.00	1.00	1.00
Single	15/ 743	2.06 (1.13 – 3.76) *(no attenuation)*	2.06 (1.13 – 3.75) *(no attenuation)*	2.03 (1.11 – 3.71) *(2.8%)*	1.83 (1.00 – 3.37) *(16%)*	1.84 (1.00 –3.38) *(17.2%)*
Sep/Div	23/ 1258	2.55 (1.52 – 4.28) *(3%)*	2.22 (1.32 – 3.73) *(21.2%)*	2.39 (1.42 – 4.02) *(10.3)*	2.50 (1.50 – 4.20) *(3.2%)*	2.13 (1.26 – 3.61) *(27.1%)*
Widowed	35/ 1030	1.35 (0.83 – 2.20) *(not applicable)*	1.24 (0.76 – 2.03) *(not applicable)*	1.28 (0.78 – 2.10) *(not applicable)*	1.35 (0.82 – 2.20) *(not applicable)*	1.20 (0.73 – 1.97) *(not applicable)*

Model 1 adjusted for age, SES
Model 2 adjusted for age, SES + health behaviours (physical activity, smoking, alcohol)
Model 3 adjusted for age, SES + distress (GHQ-12)
Model 4 adjusted for age, SES + metabolic dysregulation (doctor diagnosed hypertension, diabetes, and BMI).
[1]The proportion of CVD risk reduction explained by each set of factors was computed as follows: $(HR_{basic\ model} - HR_{adjusted})/ HR_{basic\ model} - 1) \times 100$

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