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Childbearing history and late-life mortality: the Dubbo study of Australian elderly

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Abstract

Objective: to examine the association of parity with mortality in later life.
Design: a longitudinal, community-based study.
Setting: semi-rural town of Dubbo, NSW, Australia.
Subjects: a total of 1,571 women and 1,233 men 60 years and older first examined in 1988–89.
Outcome measures: all-cause and cause-specific mortality rates analysed over 16-year follow-up. Hazard ratios obtained from proportional hazards models employing conventional predictors, potential confounders and measure of parity.
Results: increasing parity in women was weakly associated with overweight, diabetes and hypertension. All-cause mortality fell progressively with increasing parity in women (hazard ratio and 95% confidence intervals): childless, 1.00; 1 child, 1.03 (0.75–1.43); 2 children, 0.83 (0.61–1.11); 3 children, 0.80 (0.60–1.08); 4 children, 0.91 (0.66–1.25); 5 children, 0.70 (0.49–1.01); 6+ children, 0.60 (0.43–0.85) (trend for parity $P < 0.002$). This result was similar whether or not hypertension,

diabetes and overweight were included in multivariate models adjusting for social variables and other confounders. The reduction in all-cause mortality was accompanied by a parallel reduction in deaths from cancer and respiratory conditions, while coronary heart disease mortality increased 60–111% in all parous women.
Conclusion: there was increased all-cause mortality in later life in childless women, accompanied by reduced mortality as parity increased. Underlying mechanisms are unclear but findings may have public health importance.

Keywords: *childbearing, parity, later-life mortality, cohort study*

Introduction

There is increasing interest in associations between women's childbearing history, parity in particular and late-life mortality [1–6]. Yet the reported findings are somewhat conflicting. Nulliparous women and those with 5+ children had increased mortality in England and Wales in those aged 50–79 years followed for a further 29 years [1]. Increased mortality was reported in childless women and those with 1 child in Israel in those aged 65–89 years followed for 9 years [2]. There was increased mortality in Norwegian women followed for 23 years to a final age range of 45–68 years who were childless or with 1 child, compared with those having 2 children [3]. They exhibited a negative association between higher degrees of parity and mortality [3]. There were similar findings in American women in those aged 45–59 years followed for a further 21 years [4]. A German study found no association of parity with mortality [5]. A systematic review of studies up to late 2003 also demonstrated conflicting findings—mortality declined with increasing parity in 12 historical cohorts, while in 8 contemporary cohorts the highest mortality was seen in nulliparous women and in those with >4 children [6].

Some of the contrasting information here may be related to differences in study design, population socio-demographics and the control of confounding factors. We have now examined the association of parity with selected health outcomes in an exclusively elderly cohort during 16 years of follow-up in subjects aged 60+ years at study entry [7–9].

Methods

Setting and study population

The Dubbo study is a prospective study of healthy ageing in an elderly Australian cohort first examined in 1988–89. All non-institutionalised residents of Dubbo, NSW born before 1930 were eligible: the participation rate was 73% (1,233 of 1,689 men and 1,572 of 2,171 women). Methods and measures have been described in detail previously [7].

The baseline examinations comprised standard demographic, psychosocial, physical disability, self-rated health and cardiovascular risk assessments. Each participant, man or woman, stated the number of children they had produced (parity data were unavailable in one woman). No other obstetric history was obtained. The medical examination included anthropometry, blood pressure, resting ECG, peak expiratory flow, 12-h fasting blood for measurement of serum lipids, lipoproteins and glucose. Prior coronary heart disease (CHD) and prior stroke were defined as before [7–9]. Diabetes mellitus was declared present on historical grounds (previous diagnosis or use of medications) or with fasting plasma glucose >7.0 mmol/l [10].

The study population was broadly representative of the Australian population born before 1930 by gender, age, employment, socio-economic status, housing tenure, tobacco use, mean blood pressure and other variables [11]. The Dubbo study has received approval from institutional ethics committees at St Vincent's Hospital Sydney, the University of NSW and the University of Western Sydney and all subjects gave informed, written consent.

Outcomes and data analysis

Hospitalisation and death records were monitored continuously over 16 years from September 1988 until September 2004, with postal surveys conducted every 2 years to confirm vital status. Records were coded according to the *International Classification of Diseases, 9th edition (Clinical Modification) (ICD-9) and 10th edition (Australian Modification) (ICD-10)*.

We cross-tabulated the number of children (coded as 0–6+) with baseline variables (those used in an earlier analysis [8, 9]). The independent prediction of selected baseline variables by parity was examined in multiple logistic models which also included significant predictors and potential confounders [9]. Point estimates and 95% confidence intervals for the prediction of a variable were calculated from the regression coefficients (presented as odds ratio, a measure of relative risk).

All-cause and cause-specific mortality rates were calculated. The independent prediction of these outcomes by parity was examined in Cox proportional hazards models. In respect of all-cause mortality, initial models included only confounders (defined in the Results), subsequent models included potential intermediary predictors. Point estimates and 95% confidence intervals for the risk of an event were calculated from the regression coefficients (presented as the hazard ratio, a measure of relative risk). For categorical variables, the lowest or opposite category served as the reference group. The proportional hazards model assumes constant relative hazard over the length of follow-up and this was confirmed by a log-minus-log hazard plot

demonstrating parallel curves over all categories of parity. Statistical analysis was conducted using IBM SPSS 18.

Results

Cross-sectional data at study entry

The following baseline variables were cross-tabulated with the categories of parity: age, cigarette smoking, any alcohol intake, body mass index (BMI), serum lipids and lipoproteins, diabetes, hypertension, peak expiratory flow, prior CHD or stroke, atrial fibrillation, depression score, physical activities of daily living and self-rated health. Only age, hypertension, diabetes and BMI showed possible associations with parity and the data are presented in Table 1. With the exception of women with 6+ children, the mean age was lower in those at higher parity. Average BMI rose with increasing parity, as did the prevalence of diabetes and hypertension in women with 4+ children.

Prediction of BMI, diabetes and hypertension by parity was explored in separate multiple logistic models controlling for age and other variables and the findings are presented in Table 1. The odds ratios only reached significance for hypertension in women with 6+ children.

All-cause mortality in women

All-cause mortality rate declined with increasing parity (Table 2). It was highest in nulliparous women, progressively falling until women had 3+children. All-cause mortality findings in proportional hazards models in relation to parity are presented in Table 2. In a model controlling only for age, hazard ratios were lower at high degrees of parity but did not reach statistical significance. In a model which then included other significant predictors of mortality, but excluded hypertension, diabetes and BMI (the 'confounder model'), the hazard ratios for all-cause mortality fell progressively with increasing parity beyond 1 child, reaching statistical significance in those with 6+ children, where the risk of death was 40% lower than in nulliparous women (test of trend for parity $P < 0.004$).

The prediction of all-cause mortality by parity was not materially changed if the 'confounder model' now included hypertension or diabetes or BMI (separate models in Table 2). A final model incorporating all variables showed little change from the 'confounder model', even with the inclusion of hypertension, diabetes and BMI (Table 2). Test of trend for parity in this final model was highly significant ($P < 0.002$).

The relationship of parity to all-cause mortality in women is demonstrated in the hazard curves in Figure 1. These curves demonstrate a broad hierarchy of reducing mortality with increasing parity in the final multivariate model which controls for the contribution of other predictors and potential confounders.

The final proportional hazards model included the following variables in addition to parity (hazard ratio and 95% CI): age, 1.10 (1.08–1.11); BMI, 0.99 (0.97–1.00); any alcohol intake, 0.79 (0.67–0.93); current cigarette smoking, 1.76 (1.36–2.27); low peak expiratory flow, 2.10 (1.66–2.67); increased physical disability, 1.65 (1.32–2.08); poor self-rated health, 1.26 (1.01–1.56); diabetes, 2.00 (1.54–2.59); hypertension, 1.49 (1.22–1.81) and atrial fibrillation, 2.23 (1.46–3.40). Additional variables listed above in the cross-sectional analysis did not approach statistical significance and were excluded from all-cause mortality models.

Cause-specific mortality in women

Cause-specific mortality rates are presented in Table 3 for the predominant groupings. The pattern of decline in all-cause mortality with parity was accompanied by parallel reductions in deaths from cancer and respiratory conditions and 'other' causes of death. In multivariate models employing the final list used in Table 2, this was statistically significant only in a consistent manner for deaths from other causes. CHD mortality was generally increased in all parous women, statistically so in many groups, who had an increase in the range 60–111% compared with nulliparous women.

Mortality in men

Mortality and 'parity' were similarly evaluated in men in the full proportional hazards model. There were 704 deaths

Table 1. Baseline data and multivariate prediction by number of children (parity) in 1,571 women

	0 ($n = 140$)	1 ($n = 159$)	2 ($n = 322$)	3 ($n = 361$)	4 ($n = 236$)	5 ($n = 157$)	6+ ($n = 196$)	All women ($n = 1,571$)
Mean age (years)	72.3	71.1	69.2	69.3	68.7	68.3	70.0	69.6
Mean BMI (kg/m^2)	24.9	25.2	25.0	25.7	26.5	26.4	27.5	25.8
Odds ratio (95% CI)	1.00	0.93 (0.50–1.74)	0.60 (0.34–1.06)	0.83 (0.49–1.43)	1.11 (0.63–1.95)	0.76 (0.40–1.42)	1.37 (0.78–2.42)	
Diabetes (%)	6.5	2.5	6.6	6.4	8.7	9.7	12.2	7.5
Odds ratio (95% CI)	1.00	0.37 (0.10–1.34)	1.32 (0.53–3.32)	1.10 (0.44–2.76)	1.31 (0.52–3.32)	1.53 (0.58–4.09)	1.27 (0.50–3.21)	
Hypertension (%)	59.3	62.9	62.4	55.1	64.0	59.2	74.5	61.9
Odds ratio (95% CI)	1.00	1.36 (0.81–2.30)	1.57 (0.99–2.49)	1.16 (0.74–1.81)	1.56 (0.95–2.54)	1.28 (0.75–2.17)	1.95 (1.16–3.30)	

Continuous variables are means, categorical variables are rate/100. Hypertension was defined as blood pressure ≥160 and/or ≥95 mm or using medication. The multivariate prediction of BMI, diabetes and hypertension by parity was tested in logistic models which included all potential predictors (or confounders) listed in the Results. BMI was evaluated as >80th percentile versus lower.

(57% of all men) and there was modest evidence of reduced all-cause mortality with increasing number of children. But this finding did not approach statistical significance (hazard ratio and 95% CI): childless, 1.00; 1 child, 0.88 (0.64–1.21); 2 children, 0.92 (0.70–1.20); 3 children, 0.90 (0.69–1.17); 4 children, 0.95 (0.72–1.27); 5 children, 0.87 (0.61–1.24); 6+ children, 0.83 (0.60–1.17) (test of trend for parity not significant $P < 0.5$). There were 222 CHD deaths (18% of all men), but there was no significant association with number of children, certainly no suggestion of the excess CHD mortality seen in women (data not shown).

Discussion

Although the recent literature does contain some discordant reports on parity and late-life mortality [1–6], we have clearly shown increased all-cause mortality in nulliparous women, accompanied by reduced all-cause mortality as the degree of parity increases. These effects were independent of other conventional risk factors [9], especially hypertension, diabetes and BMI, each of which did show some association with parity in cross-sectional analysis. The multivariate models in Table 2 suggest that the overall relationship between all-cause mortality and parity is not mediated via hypertension, diabetes or overweight. Despite a significant increase in CHD mortality with increasing parity, the overall reduction in all-cause mortality with increasing parity is associated with parallel reductions in deaths from cancer, respiratory diseases and other causes. Because of the small number of deaths due to cancer or respiratory causes in each parity group and the lack of autopsy confirmation, we are unable to identify with any certainty which specific cancer or respiratory conditions may have changed with differing parity. The 'other causes' group is too diverse to enable any conclusions.

Our findings with all-cause mortality are consistent with those from Norway where women and men were followed for 23 years to a final age range of 45–68 years [3]. This study recorded 7.2 million person-years of follow-up in women, the Dubbo study 19,500 person-years. Women and men in Norway showed a significant excess of all-cause mortality in those who were childless or had only 1 child compared with those with 2 children, while women with 3+ children had significantly reduced all-cause mortality. A similar but non-significant trend at high degrees of parity was noted in Norwegian men. Israeli women and men showed a similar mortality relationship as in Norway [2], a finding also suggested in Dubbo men and women. Other studies have also reported that nulliparous women have increased all-cause mortality compared with parous women [1, 2, 4]. It has been suggested that women in Norway do not carry a higher parity 'penalty', similarly in Dubbo Australian women, yet women in England and Wales do carry such a penalty [12]. Although the respective countries have varying systems of social support, conflicting findings cannot be easily resolved, but there may be other important differences in study design or confounding by unspecified factors.

Table 2. All-cause mortality incidence/1,000 person-years and multivariate prediction by parity in 1,571 women followed for 16 years

	0 ($n = 140$)	1 ($n = 159$)	2 ($n = 322$)	3 ($n = 361$)	4 ($n = 236$)	5 ($n = 157$)	6+ ($n = 196$)	All women ($n = 1,571$)
All-cause mortality rate	51.2	47.1	34.1	31.3	33.5	28.2	32.4	35.1 ($n = 683$)
Age	1.00	1.10 (0.81–1.50)	0.92 (0.69–1.21)	0.82 (0.62–1.07)	1.02 (0.76–1.38)	0.86 (0.61–1.20)	0.85 (0.62–1.16)	
Age + confounders[a]	1.00	1.01 (0.73–1.39)	0.91 (0.68–1.22)	0.86 (0.64–1.14)	0.94 (0.69–1.29)	0.73 (0.51–1.05)	0.65 (0.47–0.90)	
Age + confounders + hypertension	1.00	0.98 (0.71–1.35)	0.85 (0.64–1.14)	0.83 (0.62–1.10)	0.89 (0.65–1.22)	0.70 (0.49–1.00)	0.60 (0.43–0.83)	
Age + confounders + diabetes	1.00	1.05 (0.76–1.44)	0.88 (0.65–1.18)	0.82 (0.62–1.10)	0.93 (0.68–1.28)	0.71 (0.49–1.01)	0.63 (0.45–0.88)	
Age + confounders + BMI	1.00	1.02 (0.74–1.40)	0.92 (0.68–1.23)	0.87 (0.65–1.16)	0.96 (0.70–1.31)	0.75 (0.52–1.07)	0.66 (0.47–0.92)	
Full model, all variables	1.00	1.03 (0.75–1.43)	0.83 (0.61–1.11)	0.80 (0.60–1.08)	0.91 (0.66–1.25)	0.70 (0.49–1.01)	0.60 (0.43–0.85)	

The hazard ratio (95% CI) was adjusted for the variables as indicated.
The multivariate prediction of all-cause mortality by parity used Cox models which progressively incorporated variables as indicated in the table.
[a]Confounders included: alcohol intake, smoking, peak expiratory flow, physical disability, self-rated health and atrial fibrillation. Hypertension was defined as blood pressure ≥160 and/or ≥95 mm or using medication. BMI was a continuous variable. The reference group for parity was 0 children.

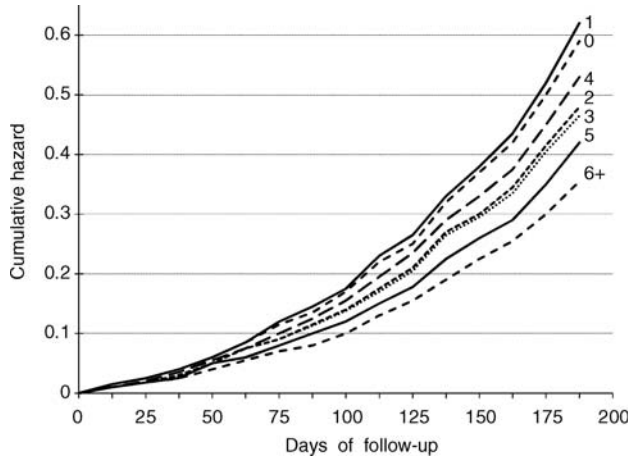

Figure 1. Hazard curves for all-causes mortality by parity in the full proportional hazards model.

Based on broadly similar mortality patterns in men and women in relation to parity in Norway and Israel, investigators have speculated that this might be the effect of reproductive behaviour on non-pregnancy-related pathways, especially lifestyle and other biosocial interactions [2, 3, 13]. Some support for the influence of social pathways comes from studies of parity and late-life morbidity. In a cohort of high parity (6+ children), African-American women 50 years and older followed for 11 years, they manifested worsening self-rated health compared with white women [14]. A study of West German women 50 years and older followed for 23 years found that high parity (4+ children) was associated with better self-rated health, an association not found in East German women followed for 17 years [5].

Cause-specific mortality data have recently been reported for the Norwegian cohort [13]. The excess mortality noted in those who were childless or with only 1 child related to nearly all-causes of death. High parity women (4 + children) manifested increased cervical cancer deaths. Excess cardiovascular mortality was recently reported in Israeli women 65–89 years who were childless or with 1 child, while there was reduced reproductive cancer mortality at high degrees of parity [15]. The Dubbo findings stand in contrast with increasing CHD mortality with increasing parity (Table 3), but our numbers in each parity group are smaller than in other studies. We have searched in our cross-sectional entry data for associations of parity with morbidity likely to be associated with pregnancy. We observed a suggestion of increasing overweight, diabetes and hypertension with increasing parity, but the findings did not reach statistical significance (Table 1). This finding may partly explain increased CHD mortality with increasing parity, while the overall decline in all-cause mortality was accounted for by falls in respiratory and cancer deaths (Table 3). Notably absent was any clear association between parity and self-rated health, physical disability or depression score. This suggests that surviving women with a mean age around 70 years are not carrying excess morbidity from

Table 3. Cause-specific mortality incidence/1,000 person-years and multivariate prediction by parity in 1,571 women followed for 16 years

	0 ($n = 140$)	1 ($n = 159$)	2 ($n = 322$)	3 ($n = 361$)	4 ($n = 236$)	5 ($n = 157$)	6+ ($n = 196$)	All women ($n = 1,571$)
CHD rate, hazard ratio (95% CI)	8.8,1.00	15.5, 2.07 (1.04–4.12)	13.9, 2.11 (1.11–3.98)	10.3, 1.60 (0.84–3.05)	13.1, 2.10 (1.08–4.09)	9.9, 1.47 (0.70–3.07)	13.6, 1.34 (0.68–2.66)	12.2 ($n = 237$)
Ischaemic stroke rate, hazard ratio (95% CI)	3.8, 1.00	5.0, 2.15 (0.63–7.36)	1.3, 0.65 (0.17–2.53)	2.4, 1.30 (0.40–4.23)	2.3, 1.46 (0.39–5.48)	0.5, —	3.3, 1.66 (0.47–5.84)	2.4 ($n = 47$)
Other CVD rate, hazard ratio (95% CI)	5.7, 1.00	6.7, 1.36 (0.54–3.41)	2.8, 0.63 (0.24–1.63)	4.5, 0.96 (0.41–2.24)	5.0, 1.56 (0.63–3.87)	3.5, 0.91 (0.32–2.64)	3.3, 0.71 (0.25–1.99)	4.3 ($n = 83$)
All cancer rate, hazard ratio (95% CI)	9.5, 1.00	7.8, 0.83 (0.39–1.75)	6.3, 0.72 (0.37–1.42)	4.7, 0.55 (0.28–1.11)	3.3, 0.39 (0.17–0.93)	4.9, 0.63 (0.28–1.45)	4.5, 0.44 (0.19–1.01)	5.5 ($n = 107$)
Respiratory rate, hazard ratio (95% CI)	8.2, 1.00	3.9, 0.45 (0.17–1.23)	3.5, 0.46 (0.21–1.05)	2.8, 0.47 (0.21–1.06)	4.7, 0.69 (0.30–1.59)	3.5, 0.52 (0.19–1.44)	2.9, 0.33 (0.13–0.88)	3.9 ($n = 75$)
Other causes, hazard ratio (95% CI)	13.9, 1.00	8.3, 0.66 (0.33–1.31)	4.8, 0.41 (0.21–0.78)	6.0, 0.48 (0.27–0.88)	4.4, 0.39 (0.19–0.82)	4.9, 0.38 (0.17–0.85)	4.5, 0.23 (0.10–0.53)	6.1 ($n = 118$)

The multivariate prediction of cause-specific mortality used Cox models incorporating the full variable list in Table 2.

earlier childbearing. Our findings vis-a-vis reduced respiratory mortality are intriguing, given that reduced peak expiratory flow and current cigarette smoking significantly predicted all-cause mortality. Does this suggest that increasing parity is associated with better peak expiratory flow and a lower smoking prevalence? Such associations could not be confirmed in the baseline data.

The present study has strengths and limitations. The cohort does comprise most of the community-dwelling women in one town, they were well documented in 1988–89 and mortality follow-up was virtually complete. On the other hand, the present analyses were not originally envisaged and other important and potentially predictive data such as age at first birth, birth intervals or pregnancy complications were not collected, while we have essentially studied women who were 'survivors'. However, the number of deaths has been large enough to enable firm conclusions about all-cause mortality and parity.

Since high degrees of parity are now relatively uncommon in Western societies, with the exception of certain religious groups, the finding of reduced all-cause mortality and increasing parity is not of immediate practical application to public health. However, this finding will have underlying mechanisms which deserve further elucidation, potentially adding to the quality of maternal health.

Key points

- In a cohort of women followed for 16 years from age 60+ years, childless women or those with 1 child had increased all-cause mortality compared with those at higher degrees of parity.
- All-cause mortality was reduced with increasing degrees of parity.
- This was a genuine association present after adjustment for social variables, other potential confounders and health-related phenotypes.
- The reduction in all-cause mortality was accompanied by a parallel reduction in deaths from cancer and respiratory conditions, but also by a parallel increase in CHD mortality.
- The findings with respect to all-cause mortality confirm results of studies in Norway and Israel, but contrast with findings from England and Wales and elsewhere.

Conflict of interest

None declared.

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