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Research Article

Blame the parents? The association between parental longevity and successful ageing

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Blame the parents? The association between parental longevity and successful ageing

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Abstract

Research has suggested that children of long-lived parents might age more successfully than children of short-lived parents. The aim of this study is to contribute further to the understanding of the association between parental longevity and offspring's successful ageing. We used data from wave one of the English Longitudinal study of ageing (ELSA) to investigate the association between three measures of parental longevity and the respondents' cognitive and physical functioning, self-reported health and several chronic diseases. We found that parental lifespan, especially mother's lifespan, is positively associated with cognitive functioning at older age. Parental lifespan and mother's lifespan were also found to be associated with a decreased likelihood of occurrence of some chronic diseases such as pulmonary disease, coronary heart disease, hypertension and poor health.

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1. Introduction

Since Rowe and Kahn (1987) introduced the concept of 'successful ageing', research has pointed out the effect of a number of factors that influence ageing. Among such factors one can mention socioeconomic, environmental, lifestyle and genetic factors (Christensen et al. 2000; Frederiksen et al. 2002; Guralnik and Kaplan 1989; McGue and Christensen 2001; Strawbridge et al. 1996). Research has suggested that longevity might be hereditary (Frederiksen et al. 2002; Perls et al. 1999; 2002). Studies of twins and other populations have shown that genetic factors account for about 25-35 percent of the variance in lifespan (Herskind et al. 1996; Ljungquist et al.1998; McGue et al. 1993). Research has shown that parents and siblings of centenarians have longer life expectancies when compared to their birth cohorts (Perls et al. 1999; 2002; Hammond et al. 1971). Recent studies in the longevity of adopted children have reinforced the importance of biological parents in predicting the longevity of offspring (Petersen et al. 2005; Sorensen 1991; Sorensen et al. 1988). Recent research has also demonstrated that children of long-lived parents have an advantage compared to their counterparts with regards to inheritance of chronic disease (Lichtenstein et al. 2000; Marenberg et al. 1994; Poulsen et al.1999; Terry et al. 2004; 2007) as well as with regards to the timing of onset of chronic disease such as diabetes, heart disease and hypertension (Terry et al. 2004).

We hypothesise that children of long-lived parents age more successfully than others - suffer from fewer chronic disease and have a better physical and cognitive functioning at old age. In order to better understand how parental longevity could affect respondent's own successful ageing we test the following hypotheses:

1) The longevity of both parents can influence successful ageing of the offspring - to test this we use the mean lifespan of both parents.
2) The longevity of at least one of the parents can influence successful ageing of the offspring - to test this we use the maximum lifespan between mother's and father's lifespan
3) Mother's longevity is more important than father's longevity (or vice versa) in influencing successful ageing of the offspring - to test this hypothesis we use mother's lifespan and father's lifespan separately.

In addition we explore the extent to which the relationships between parental lifespan and successful ageing of the offspring are confounded not only by age and sex but also by alcohol consumption, smoking status, physical activity, total wealth and whether the parent has died or is still alive.

2. Methods

2.1 Data source

The data are from wave 1 of the English Longitudinal Study of Ageing (ELSA), a panel study where the same individuals are followed and re-interviewed every two years. The technical details and the primary analyses of wave one have been published (Marmot et al. 2003, also available at: http://www.ifs.org.uk/elsa/report.htm). The ELSA sample was drawn from people who had taken part in the Health Survey for England (HSE) in 1998, 2000 or 2001 and were born before March 1952. The HSE samples are selected to be representative of people living in private households in England. The first wave of ELSA took place in 2002-2003; a sample of 12,100 respondents was achieved. The analyses of this study are restricted to 11,234 core members, namely participants who were living in private household in England at the time the study was conducted (excluding partners). The survey comprised a personal face-to-face interview and a self-completion questionnaire. The study collects information on the personal, economic and social circumstances of ageing. Participants gave their informed consent to take part in the study. Ethical approval for ELSA was given by the London Multi-centre Research Ethics Committee[3].

2.2 Measurements

Parental age

During the interview people were asked whether one or both parents where still alive, and for each parent the actual age was asked; if the parent/s has died, the age at death was also asked. In addition, they were asked whether father or mother died from one of the following conditions: cancer, heart attack, stroke, other cardiovascular related illness, respiratory disease and none of these. We calculated mother's lifespan as the actual age of mother if still alive otherwise as the age at death; father's lifespan was calculated in the same way. We then calculated the mean parental lifespan as the average of mother's lifespan and father's lifespan; the maximum lifespan was calculated by taking the maximum between father's lifespan and mother's lifespan.

We excluded from the analyses 385 participants (3.5%) whose parents died before age 50 of unknown causes of death. We believe that these deaths are not related to the biology of ageing. Late deaths are the ones that we are interested since there is research

[3] International Review Board (IRB) number for the ethics approval of the ELSA study: IRB 00002380 and the MREC approvals for the two ELSA waves ref: MREC/04/ and MREC/01/2/91

that shows that longevity runs in the family (Gudmundsson et al. 2000; Perls et al. 1998; Schoenmaker et al. 2006).

Physical functioning

The measure of physical functioning used in this study is Activities of Daily Living (ADL-s). ELSA collects information on difficulties in Activities of Daily Living (ADL-s) through the health-assessment questionnaire (Steel et al. 2003). Respondents were shown a card and asked to report whether because of a health or memory problem, they have any difficulties doing any of the activities on the card. The following six ADL-s items were listed: dressing, walk across a room, bathing, eating, getting in and out of bed and using the toilet. From this information we derived a variable reporting whether the respondent has one or more ADL-s difficulties (0 ADL vs 1 and more ADLs).

Cognitive functioning

The cognitive processes that were assessed in ELSA include learning and memory (retrospective and prospective memory), word-finding ability, executive function, speed and processing and numerical ability. For each test summary cognitive performance measures were derived, and a global cognitive index (ranging score from 0 to 60, with higher scores indicating better cognitive functioning), which combines these summary measures, was used in this study (Steel et al. 2003).

Health

Participants were asked whether a doctor had ever told them that they suffered from various diseases, including diabetes, pulmonary disease (asthma and or chronic bronchitis), hypertension, cancer (excluding skin cancer), and heart disease. We included in the definition of heart disease any of the following: stroke, congestive heart failure, abnormal heart rhythm, heart murmur, angina and myocardial infarction.

In addition respondents were asked to rate their health, from excellent to poor. The variable was recoded into two categories, the first including excellent, very good and good health, and the second including fair and poor health.

Other variables

Potential confounding variables, such as smoking status, weekly alcohol consumption, physical activity and total-wealth (non pension) were used to adjust the multivariate analyses. Total non-pension wealth is defined as financial, plus physical (such as business wealth, land or jewellery), plus housing wealth minus debts; it represents a better measure of the permanent economic status of older people than income (Banks et al. 2003).

We have also derived a dummy variable that states whether the parent has died or is still alive.

2.3 Statistical Analyses

Variables measuring the parental lifespan (mean parental lifespan, the maximum lifespan between the father's and mother's lifespan, father's and mother's lifespan separately) were categorized in 10-year groups and used as continuous measures in multivariate analyses.

Separate multivariate linear regression models were run to explore the association between cognitive functioning and each of the parental lifespan variables.

Multivariate logistic regression was used with dichotomous variables, such as ADL-s (defined as no-ADL and 1 or more ADL-s), diabetes, pulmonary disease, hypertension, heart disease, cancer, self-reported health.

All regression analyses were initially adjusted for age and sex of participants; and then further adjusted for smoking habits, alcohol consumption, physical activity, total-wealth and for whether the parent has died or is still alive.

All analyses were done using Stata version 9.2, routinely using population weights to correct for non-response.

3. Results

Table 1 shows the sample characteristics of participants in the study. 72.6% reported that both parents died, 22.8% has one parent still alive and 4.7% has both parents still alive. The mean parental lifespan was 74.1, significantly lower than the maximum between mother's lifespan and father's lifespan ($p<0.001$). The lifespan of mother was significantly higher ($p<0.001$) than the lifespan of father (Table 1).

Table 1: Sample characteristics

	% (95%CI)	N
Males	46.5 (45.8; 47.1)	4,945
Females	53.5 (52.9; 54.2)	5,895
Both parents dead	72.6 (71.6; 73.5)	7,893
One parent alive	22.8 (21.9; 23.7)	2,467
Both parents alive	4.7 (4.3; 5.1)	480
Diabetes	7.2 (6.8; 7.8)	813
Pulmonary disease	15.8 (15.1; 16.5)	1,765
Hypertension	37.2 (36.3; 38.1)	4132
CHD	20.3 (19.6; 21.2)	2,264
Cancer	6.3 (5.8; 6.8)	695
Fair or poor health	26.7 (25.7; 27.7)	2,924
1 or more ADLs	21.9 (21.0; 22.7)	2,397
	Mean (s.e.)	**N**
Age of participants	65.2 (0.13)	10,980
Lifespan of mother	77.2 (0.15)	6,716
Lifespan of father	73.1 (0.17)	5,344
Mean lifespan of parents	74.1 (0.13)	4,956
Max lifespan between mother and father lifespan	80.0 (0.14)	4,956
Mean of global cognitive function score	33.7 (0.11)	10,246

s.e: Standard error of the mean

Table 2 reports the odds ratios for the association between ADL-s and each of the parental lifespan variables. In the age and sex-adjusted model, the odds of having one and more ADL-s, were significantly lower for every 10-year increase in the mean parental lifespan, in the maximum lifespan between mother's and father's lifespan and in mother's lifespan. However, when the models were adjusted for other variables, the associations were no longer significant.

Table 2: Odds for the association between ADLs and parental lifespan variables

	OR	95%CI	p-value
Model 1[a]			
Mean parental lifespan (10 yrs)	0.90	(0.84; 0.97)	<0.010
Max parental lifespan (10 yrs)	0.92	(0.85; 0.99)	<0.050
Mother's lifespan (10 yrs)	0.93	(0.87; 0.98)	<0.010
Father's lifespan (10 yrs)	0.98	(0.92; 1.03)	0.398
Model 2[b]			
Mean parental lifespan (10 yrs)	0.98	(0.90; 1.06)	0.565
Max parental lifespan (10 yrs)	1.00	(0.91; 1.09)	0.938
Mother's lifespan (10 yrs)	0.98	(0.92; 1.04)	0.505
Father's lifespan (10 yrs)	1.00	(0.94; 1.06)	0.940

a Adjusted for age and sex only
b Adjusted for age, sex, alcohol, smoking status, physical activity, total wealth and whether parent is still alive or dead

Table 3 reports the un-standardised regression coefficients for the association between cognitive functioning and each of the parental lifespan variables. In the age and sex adjusted model, for every 10-year increase in mean parental lifespan, there is a significant increase in the global cognitive score equal to 0.85, while per 10-year increase in the maximum lifespan between mother's and father's lifespan the increase in the global cognitive score is 0.97. When we looked at association of cognitive functioning with father's lifespan and with mother's lifespan, we found that the increase in the global cognitive score was higher for the mother's lifespan (10 years increase) than for father's lifespan. When further adjusted for covariates, only father's lifespan

was no longer significantly associated with the cognitive function index, while the other variables remained significant, although decreased in magnitude.

Table 3: Regression coefficients for the association between cognitive functioning and parental lifespan variables

	Unst. Coef.	95%CI	p-value
Model 1[a]			
Mean parental lifespan (10 yrs)	0.85	(0.62; 1.08)	<0.001
Max between mother's and father's lifespan (10 yrs)	0.97	(0.73; 1.21)	<0.001
Mother's lifespan (10 yrs)	0.53	(0.35; 0.70)	<0.001
Father's lifespan (10 yrs)	0.32	(0.14; 0.50)	<0.001
Model 2[b]			
Mean parental lifespan (10 yrs)	0.42	(0.20; 0.65)	<0.001
Max parental lifespan (10 yrs)	0.53	(0.31; 0.76)	<0.001
Mother's lifespan (10 yrs)	0.28	(0.11; 0.44)	<0.001
Father's lifespan (10 yrs)	0.14	(-0.03; 0.31)	0.117

a Adjusted for age and sex only
b Adjusted for age, sex, alcohol, smoking status, physical activity, total wealth and whether parent is still alive or dead

Table 4 reports the odds ratios for the association between self-reported diseases and each of the parental lifespan variables. Respondents were less likely to have diabetes for every 10-year increase in mean parental lifespan, in the maximum lifespan between mother's and father's lifespan and in the father's lifespan. In the fully adjusted model, only the odds ratio for having diabetes, per 10-year increase in the mean parental lifespan, remained significantly low. In both models (age and sex adjusted and in the fully adjusted) the likelihood of having pulmonary disease decreased for every 10-year increase in each of the lifespan variables, with the exception of father's lifespan. Parental lifespan variables were all significantly associated with hypertension in both models. The odds of having CHD decreased significantly per 10-year increase in each of the lifespan variables. The significant associations remained after adjusting for other covariates, with the exception of the association between CHD and father's lifespan which was no longer significant (Table 4). Cancer was not associated with any of the parental lifespan variables, in both models. For every 10-year increase in all parental lifespan variables there was between 6% and 18% decrease in the odds of reporting fair or poor health. However, when the models were fully adjusted, the associations remained significant only for mean parental lifespan and for mother's lifespan (Table 4).

Table 4: Odds ratios for the association between self-reported diseases and health and parental lifespan variables

	Mean parental lifespan (10 yrs)			Max between mother's and father's lifespan (10 yrs)			Mother's lifespan (10 yrs)		
Model 1[a]	***OR***	***95%CI***	***p***	***OR***	***95%CI***	***p***	***OR***	***95%CI***	***p***
Diabetes	**0.83**	**(0.75; 0.92)**	**<0.001**	**0.87**	**(0.78; 0.97)**	**<0.050**	0.93	(0.86; 1.01)	0.090
Pulmonary disease	**0.86**	**(0.79; 0.94)**	**<0.001**	**0.87**	**(0.80; 0.94)**	**<0.001**	**0.89**	**(0.84; 0.95)**	**<0.001**
Hypertension	**0.85**	**(0.80; 0.91)**	**<0.001**	**0.89**	**(0.83; 0.95)**	**<0.001**	**0.93**	**(0.88; 0.97)**	**<0.001**
CHD	**0.86**	**(0.80; 0.93)**	**<0.001**	**0.86**	**(0.79; 0.93)**	**<0.001**	**0.92**	**(0.87; 0.97)**	**<0.010**
Cancer	1.02	(0.89; 1.16)	0.814	1.00	(0.87; 1.14)	0.976	1.00	(0.91; 1.10)	0.966
Fair or poor health	**0.82**	**(0.76; 0.88)**	**<0.001**	**0.83**	**(0.77; 0.90)**	**<0.001**	**0.88**	**(0.83; 0.92)**	**<0.001**
Model 2[b]									
Diabetes	**0.89**	**(0.80; 1.00)**	**<0.050**	0.93	(0.83; 1.04)	0.216	0.97	(0.89; 1.06)	0.481
Pulmonary disease	**0.91**	**(0.84; 1.00)**	**<0.050**	**0.91**	**(0.84; 0.99)**	**<0.050**	**0.91**	**(0.86; 0.97)**	**<0.010**
Hypertension	**0.90**	**(0.84; 0.96)**	**<0.001**	**0.91**	**(0.85; 0.98)**	**<0.050**	**0.95**	**(0.90; 0.99)**	**<0.050**
CHD	**0.91**	**(0.84; 0.98)**	**<0.010**	**0.89**	**(0.82; 0.96)**	**<0.010**	**0.95**	**(0.90; 1.00)**	**<0.050**
Cancer	1.01	(0.88; 1.15)	0.930	0.99	(0.87; 1.14)	0.932	0.99	(0.90; 1.09)	0.857
Fair or poor health	**0.92**	**(0.85; 1.00)**	**0.050**	0.93	(0.86; 1.01)	0.100	**0.93**	**(0.88; 0.99)**	**<0.010**

a Adjusted for age and sex only

b Adjusted for age, sex, alcohol, smoking status, physical activity, total wealth and whether parent is still alive or dead

Odds ratios in bold are statistically significant

4. Discussion

In this study we looked at parental lifespan as well as mean parental lifespan, the maximum lifespan between mother's and father's lifespan to asses whether the longevity of one or both parent's could influence the health of the off-spring at old age.

Some previous studies have reported the association between parental longevity and successful ageing of offspring. Also the fact that some diseases show familial risk patterns has been shown before (Hammond et al. 1971). Vaillant (1991) found that ancestral longevity was strongly predictive of chronic disease at age 60. Frederiksen and colleagues (2002) reported that parental lifespan was positively associated with offspring's physical and cognitive functioning as well as with avoidance of some of the common chronic diseases. More recently Terry and colleagues (2007) analysed data from the Framingham Study and found that individuals with long-lived parents have advantageous cardiovascular risk profiles in middle age compared to those whose parents died at younger ages.

We found that parental lifespan is positively associated with physical functioning at older age when the model was adjusted only for age and sex. For every 10 year increase either in the mean parental lifespan, or in the maximum lifespan between mother's and father's lifespan, there was a significant reduction in odds of having one or more ADL-s. However these results lose the significance after adjusting for other factors such as smoking, alcohol, physical activity and total wealth. This shows that not accounting for the confounding effect of these variables can lead to overestimate the relationships between parental lifespan and physical functioning. We also found that parental lifespan is positively associated with better cognitive functioning and with a decreased likelihood of occurrence of some chronic diseases such as diabetes, pulmonary disease, coronary heart disease and hypertension.

While previous studies have shown contradictory findings regarding offspring survival and parental longevity (Abbott et al. 1978; Rosengren et al. 2002), in our study we found several significant differences linked to the gender of the parent. Mother's lifespan was associated with decreased likelihood of occurrence of pulmonary disease, CHD and fair and poor health. Mother's lifespan was also associated with better cognitive functioning. This suggests that maternal longevity in more important for some aspects of the successful ageing of the offspring.

The paper's most important strength is that it uses a wide range of variables from a large national representative study giving a comprehensive account of how parental longevity might relate to health and successful ageing. To the best of our knowledge this is the first time that such a study has been conducted in the UK. In addition it is the first study to include possible confounders in the models exploring the relationships between parental longevity and offspring's successful ageing. We found that non-

inclusion of such confounders in the statistical models could lead to overestimate some of these relationships.

A possible limitation is that the information on parental age at death was self-reported by the respondents and as such it could contain errors. We also are aware that while the genomic expression influences the risk of several diseases, there are other ways in which parents can influence their offspring's health and longevity. Parents and offspring share similar socioeconomic environment and behavioural patterns which influence health to some extent. More importantly some of the parental influence on offspring starts in-utero where mother's health and nourishment has been shown to affect offspring's health later in life (Barker 1998; Poulton et al. 2002). Although we have adjusted for several covariates, we did not have information on childhood circumstances, parental age at conception, sibling-related information as well as information about grandparents. The extent to which the lack of this information can influence our results could not be assessed in this study.

5. Conclusions

We conclude that parental lifespan and more importantly mother's lifespan is positively associated with better cognitive and to physical functioning at older ages as well as with decreased likelihood of some chronic diseases and poor health. The study reinforces the need for further research which combines genetic and epidemiologic information and which will further contribute to a better understanding of successful ageing, such as survival to old age without adverse health effects or decline of physical and cognitive function.

6. Acknowledgements

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