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Original Contribution

Spousal Loss and Cognitive Function in Later Life: A 25-year Follow-up in the AGES-Reykjavik Study

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The aim of this study was to investigate the associations between loss of a life partner and the development of dementia and decline in cognitive function in later life. We used an Icelandic cohort of 4,370 participants in the Age, Gene/Environment Susceptibility-Reykjavik Study who were living as married in 1978 (born in 1907–1935) and were either still married (unexposed cohort) or widowed (exposed cohort) at follow-up (in 2002–2006). We ascertained history of marital status and spouse's death by record linkage to the Registry of the Total Population, Statistics Iceland. The outcome measures were as follows: 1) dementia and mild cognitive impairment; and 2) memory, speed of processing, and executive function. During the observation period, 3,007 individuals remained married and 1,363 lost a spouse through death. We did not find any significant associations between loss of a spouse and our outcome variables, except that widowed women had poorer executive function (mean = −0.08) during the first 2 years after their husbands' deaths compared with still-married women (mean = 0.09). Our findings do not support the notion that the risk of dementia is increased following the loss of a spouse, yet women demonstrate a seemingly temporary decline in executive function following the death of a partner.

dementia; executive function; marital status; memory; psychological stress

Abbreviations: AGES, Age, Gene/Environment Susceptibility; ApoE, apolipoprotein E.

Neurodegenerative disorders leading to cognitive impairment have become one of the largest public health threats of modern times. Although risk factors remain to a large extent unknown, a growing body of research, both in animal models (1–3) and in humans (4–11), suggests an impact of psychological stress on cognitive impairment. Studies incorporating various measures of psychological distress (4–6) and psychiatric disorders (7–11) have reported an association with cognitive decline in older adulthood.

Loss of a spouse has been classified as one of the most stressful events a person can encounter (12). Indeed, widowed individuals have been shown to be at increased risk for psychological morbidity (13) and higher overall mortality (14) compared with married individuals. Animal models suggest that the introduction of a stressful environment impairs an animal's problem-solving skills (15–17) and reduces the volumes of important memory structures, such as the hippocampus (18, 19). Proposed mechanisms between stressful life events, such as bereavement, and cognitive decline in humans have accordingly been suggested to include hippocampal atrophy from stress-induced glucocorticoid secretion (20, 21) fueled by emotional trauma, as well as increased vulnerability to stress from diminished social support and interactions (22, 23).Yet relatively few studies have specifically investigated the association between marital status change and cognitive decline. Some have found increased risk of cognitive decline in widowhood (24–27), whereas others have not (28–30). In a recent population-based cohort study, Håkansson et al. (31) monitored changes in marital status and cognitive function in 1,449 Finns followed for an average of 21 years. They reported a markedly increased risk of cognitive impairment and Alzheimer's disease among those

widowed in midlife and at follow-up compared with those who lived with a partner during the time period. With the detailed information on dementia and cognitive function from the Age, Gene/Environment Susceptibility (AGES)-Reykjavik Study and complete ascertainment of marital status since 1978, we wanted to replicate the study by Håkansson et al. (31) in another setting. In contrast to previous attempts, the available registry information on the actual timing of spousal loss allowed us to explore the relationship between timing from the stressful life event and the risk of cognitive impairment. Our overarching aim was, therefore, to investigate the associations between loss of a life partner and the risk of dementia and decline in cognitive function.

METHODS

Study population

The basis of this study was the AGES-Reykjavik Study cohort. Details on the study design and the baseline AGES-Reykjavik assessments have been described elsewhere (32). Participants were born in 1907–1935, lived in Reykjavik in 1967, and were followed as a part of the Reykjavik Study from 1967 onward by the Icelandic Heart Association (33, 34). In 2002, a random sample of the surviving participants of the Reykjavik Study was invited to participate in the AGES-Reykjavik Study. A total of 5,764 participants completed the AGES-Reykjavik examination (in 2002–2006), which included a structured survey instrument, cognitive testing, and dementia ascertainment.

Every Icelandic citizen has a unique personal identification number, making it possible to link records between different sources of health registration and official records. The AGES-Reykjavik Study was linked to the database on marital status from Statistics Iceland (updated annually since 1978). Links were also made to the Icelandic Cause of Death Registry to gather information on dates of spousal deaths. Changes in marital status, including dates of spouses' deaths, were then ascertained from 1978 until entry into the AGES-Reykjavik Study (in 2002–2006). In the present study, we included only the AGES-Reykjavik Study participants who were living as married or as cohabitants in 1978 ($n = 4{,}722$) (Figure 1). Because variables measured at midlife (i.e., 50 years of age) by the Reykjavik Study were to be adjusted for in the statistical analyses, individuals who had lost a spouse after 1978 but before they came for their first Reykjavik Study visit when midlife measurements were performed were further excluded ($n = 24$), leaving 4,698 in the final analysis. The exposed group was defined as those who were living as married or as cohabitants in 1978 and who became widowed before entry into the AGES-Reykjavik Study. The reference group consisted of individuals who stayed married during the whole observation period (Figure 1).

Dementia and mild cognitive impairment

Dementia case ascertainment was a 3-step process in the AGES-Reykjavik Study (32). Depending on performance in the first 2 steps, a subset of individuals went on to a third step. This step included a neurological examination, further neuropsychological testing, and a proxy interview about medical history and social, cognitive, and daily functioning relevant to the diagnosis. A consensus diagnosis of dementia based on the *Diagnostic and Statistical Manual of Mental Disorders, Fourth Edition,* guidelines (35) was made by a panel that included a geriatrician, a neurologist, a neuropsychologist, and a neuroradiologist. The diagnosis of mild cognitive impairment was also made by the panel of specialists. The criterion was having deficit either in memory or in 1 other cognitive domain, or having deterioration in at least 2 cognitive domains without sufficiently severe cognitive function impairment or loss of instrumental activities of daily living to constitute dementia (36, 37).

Cognitive function

A battery of cognitive tests administered to the whole AGES-Reykjavik cohort included multiple tests of 3 cognitive domains. Composite scores of memory, speed of processing, and executive function were constructed on the basis of a theoretical grouping of tests, similar to those used in other population-based studies (38, 39). The details on the tests of each cognitive domain have been reported elsewhere (40, 41). Composite measures were computed by converting raw scores on each test to standardized z scores and averaging the z scores across the tests in each composite. The fit of the composites to the data has been reported elsewhere (41).

Covariates

The covariates included in the models were chosen according to their previously reported associations with cognitive function and dementia (42). These included the following: age, sex, apolipoprotein E (ApoE) E4 status, education, smoking, midlife hypertension, midlife physical activity, and midlife body mass index (weight (kg)/height $(m)^2$).

Statistical analysis

There are 5 outcome variables in this study, separated into 2 main groups. The first group includes dementia and mild cognitive impairment. The second group includes the following 3 outcomes of cognitive function: memory, speed of processing, and executive function.

We used multivariable logistic regression to estimate the relationship between spousal loss and dementia/mild cognitive impairment. We adjusted our models for age (continuous), sex, education (elementary school, secondary school, college education (upper secondary schooling), or university education), ApoE E4 status (any E4 vs. no E4 with E24 excluded), midlife hypertension (measured systolic blood pressure of 140 mm Hg or higher or diastolic blood pressure of 90 mm Hg or higher), smoking status (ever smoker vs. never smoker), midlife regular physical activity (yes or no), and midlife body mass index (as a continuous variable). We also examined the associations by sex and ApoE E4 status (any E4 vs. no E4), adjusting for other factors mentioned previously. In an additional analysis, we examined the associations by levels of leisure activity at follow-up (a summary score of leisurely active days per month in tertiles (41)).

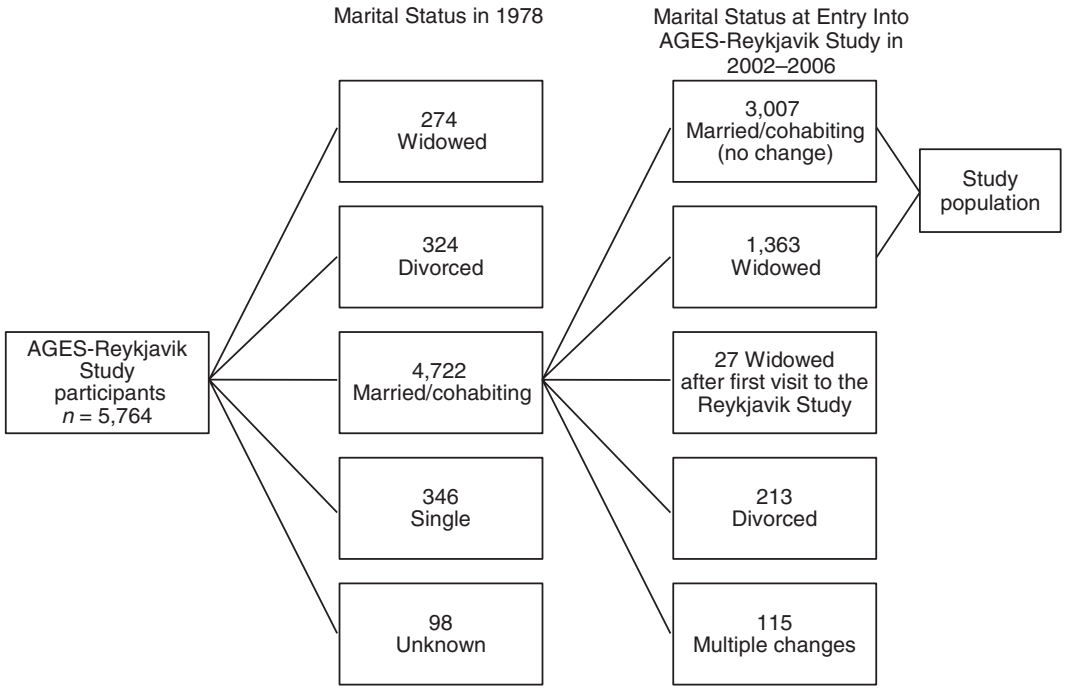

Figure 1. Change in marital status of Age, Gene/Environment Susceptibility (AGES)-Reykjavik Study participants from 1978 until entry into the study in 2002–2006. The "27 widowed after first visit to the Reykjavik Study" refers to individuals who lost a spouse after 1978 but before they came for their first Reykjavik Study visit when midlife measurements were performed.

We used linear regression to examine differences in cognitive composite scores between the exposed group and the reference group. The models were constructed the same way as the logistic regression models described above; however, we excluded from this analysis all individuals diagnosed with dementia ($n = 281$), and we also examined the associations by number of children (no child vs. 1 or more children). To illustrate the potential importance of depression on the studied associations, in additional analyses, we adjusted further for depression (as a continuous variable based on the Geriatric Depression Scale-15 (43)). We further studied the associations by sex, ApoE status, and leisure activity at follow-up.

To examine the associations between spousal loss and dementia, mild cognitive impairment, and cognitive function by time since loss, we calculated the time elapsed between the date of spousal death and the date of entry to the AGES-Reykjavik Study. We divided time since loss arbitrarily into a 4-level categorical variable as follows: 0–1.9 years, 2–4.9 years, 5–9.9 years, or ≥10 years. For dementia and mild cognitive impairment, we used multivariable logistic regression, and the models were constructed the same way as described above. For cognitive function, least squares means of composite scores were estimated for still-married and widowed participants in each time category. Only age was adjusted for in this model. The *F* test was used to test for overall differences in means among the time categories.

In a sensitivity analysis, we matched each exposed (widowed) individual to 2 nonexposed (married) individuals by sex and age in 1978 (plus/minus 1 year) by using the gmatch macro (44). We used multivariable logistic regression models to estimate the relationship between spousal loss and dementia/mild cognitive impairment, adjusting for the same covariates as before.

SPSS Statistics for Windows, version 20.0, was used to perform the statistical analyses (IBM Corp., Armonk, New York). *P* values less than 0.05 were considered statistically significant. The study protocol was approved by the National Bioethics Committee of Iceland (Reykjavik, Iceland) and the Icelandic Data Protection Authority (Reykjavik, Iceland).

RESULTS

Table 1 shows the characteristics of the 4,370 participants in the AGES-Reykjavik Study who were married/cohabiting in 1978 and were either still married at entry into the AGES-Reykjavik Study (i.e., reference group; $n = 3,007$) or had lost a spouse before that (i.e., exposed group; $n = 1,363$). Compared with the still-married participants, widowed participants were more likely to be female, older, and less educated and less likely to have had regular physical activity in midlife and to have ever smoked. The number of children born to each participant was similarly distributed between these 2 groups.

Dementia and mild cognitive impairment

Overall, we found no association between loss of a spouse and risk of dementia or mild cognitive impairment in the full models (Table 2). When we examined associations within

Table 1. Characteristics of 4,370 Participants in the AGES-Reykjavik Study who Were Married/Cohabiting in 1978, Reykjavik, Iceland

Characteristic	Spouse Alive at Entry Into AGES-Reykjavik Study ($n = 3,007$)		Spouse Deceased Before Entry Into AGES-Reykjavik Study ($n = 1,363$)		*P* Value[a]
	No.	%	No.	%	
Male	1,652	54.9	342	25.1	<0.001
Age at entry, years	75.6 (5.2)[b]		79.4 (5.6)[b]		<0.001
Age category, years					<0.001
<69	362	12.0	52	3.7	
70–74	1,019	33.9	228	16.7	
75–79	893	29.7	375	27.5	
80–84	560	18.6	483	35.4	
≥85	173	5.8	225	16.5	
Education					<0.001
Primary	539	18.8	383	31.5	
Secondary	1,529	53.3	578	47.6	
College (upper secondary)	426	14.9	178	14.7	
University	372	13.0	76	6.3	
Without children	113	3.9	59	4.9	0.287
Depression (GDS-15 score ≥6)	171	6.1	129	10.1	<0.001
Mean BMI at midlife[c]	25.3 (3.4)[b]		25.5 (3.8)[b]		0.020
BMI category at midlife					0.006
<18.5 (Underweight)	18	0.6	14	1.0	
18.5–24.9 (Normal weight)	1,507	50.3	667	49.3	
25.0–29.9 (Overweight)	1,231	41.1	524	38.7	
≥30.0 (Obese)	242	8.1	148	10.9	
Hypertension at midlife[d]	1,029	34.2	519	38.1	0.014
Regular physical activity at midlife	1,035	34.4	395	29.0	<0.001
Ever smoker	1,728	58.9	629	48.5	<0.001
ApoE E4 carrier[e]	815	27.8	349	26.2	0.276
Time since loss of a spouse, years					
0–1.9			186	13.8	
2–4.9			223	16.6	
5–9.9			337	25.1	
≥10			597	44.5	
Cognitive status					<0.001
Normal	2,505	83.3	1,000	73.4	
Dementia	158	5.3	123	9.0	
Mild cognitive impairment	253	8.4	151	11.1	
Missing data	91	3.0	89	6.5	

Abbreviations: AGES, Age, Gene/Environment Susceptibility; ApoE, apolipoprotein E; BMI, body mass index; GDS-15, 15-item Geriatric Depression Scale.

[a] Differences between the exposed group (i.e., spousal loss group) and the reference group (i.e., still-married group) were assessed using the χ^2 test for categorical variables and 1-way analysis of variance for continuous variables.

[b] Values expressed as mean (standard deviation).

[c] BMI is weight (kg)/height $(m)^2$.

[d] Systolic blood pressure higher than 140 mm Hg or diastolic blood pressure higher than 90 mm Hg.

[e] ApoE E4 carriers have a higher risk of developing dementia. Because the ApoE E2 allele may play a protective role in dementia, ApoE E24 carriers were excluded.

Table 2. Multivariable Logistic Regression–Derived Odds Ratios for Associations Between Spousal Loss and Dementia/Mild Cognitive Impairment in the AGES-Reykjavik Study, 2002–2006

Subgroup	Mild Cognitive Impairment (*n* = 404 cases)								Dementia (*n* = 281 cases)							
	No.	%	Crude		Age Adjusted		Full Model[a]		No.	%	Crude		Age Adjusted		Full Model[a]	
			OR	95% CI	OR	95% CI	OR	95% CI			OR	95% CI	OR	95% CI	OR	95% CI
Total	3,909	100							3,786	100						
Spouse alive	2,758	69	1.00	Referent	1.00	Referent	1.00	Referent	2,663	70	1.00	Referent	1.00	Referent	1.00	Referent
Spouse deceased	1,151	31	1.50	1.21, 1.85	0.93	0.74, 1.18	0.99	0.76, 1.30	1,123	30	1.95	1.52, 2.50	1.06	0.81, 1.39	0.96	0.69, 1.34
Men																
Spouse alive	1,494	84	1.00	Referent	1.00	Referent	1.00	Referent	1,423	84	1.00	Referent	1.00	Referent	1.00	Referent
Spouse deceased	280	16	1.65	1.17, 2.32	1.08	0.75, 1.55	0.84	0.55, 1.27	267	16	2.04	1.37, 3.03	1.19	0.78, 1.82	0.86	0.52, 1.45
Women																
Spouse alive	1,264	59	1.00	Referent	1.00	Referent	1.00	Referent	1,240	59	1.00	Referent	1.00	Referent	1.00	Referent
Spouse deceased	871	41	2.06	1.50, 2.81	1.16	0.82, 1.64	1.11	0.77, 1.60	856	41	2.57	1.80, 3.68	1.17	0.79, 1.74	0.97	0.62, 1.51
ApoE E4 noncarriers																
Spouse alive	1,974	70	1.00	Referent	1.00	Referent	1.00	Referent	1,874	70	1.00	Referent	1.00	Referent	1.00	Referent
Spouse deceased	842	30	1.33	1.03, 1.71	0.85	0.64, 1.11	0.92	0.67, 1.26	814	30	2.08	1.51, 2.87	1.10	0.77, 1.55	0.92	0.61, 1.40
ApoE E4 carriers																
Spouse alive	715	71	1.00	Referent	1.00	Referent	1.00	Referent	718	72	1.00	Referent	1.00	Referent	1.00	Referent
Spouse deceased	287	29	1.96	1.30, 2.96	1.11	0.71, 1.74	1.19	0.72, 1.98	283	28	1.70	1.12, 2.56	0.93	0.59, 1.47	0.98	0.57, 1.68

Abbreviations: AGES, Age, Gene/Environment Susceptibility; ApoE, apolipoprotein E; CI, confidence interval; OR, odds ratio.

[a] Adjusted for age, sex, ApoE E4 status, education, smoking (ever or never), midlife hypertension, midlife regular physical activity (yes or no), and midlife body mass index (weight (kg)/height $(m)^2$).

strata of sex and ApoE E4 status, we remained unable to detect any associations. Nor did we detect any associations when stratifying by leisure activities at follow-up (Web Table 1, available at http://aje.oxfordjournals.org/).

Cognitive function

Compared with still-married individuals, widowed individuals did not have significantly different performance in any of the tests of cognitive function (memory, speed of processing, executive functioning) in the multivariable models (Table 3). When we adjusted further for depression, the results did not change significantly (data not shown). The association did not seem to differ by ApoE E4 status. However, when we examined the associations by sex, we found significantly lower scores of executive function for widowed women but not for widowed men (Table 3). Moreover, when we stratified the analyses by whether or not the individuals had any children, widowed individuals with no children appeared to have poorer executive function than still-married individuals with no children ($\beta = -0.356$, 95% confidence interval: −0.589, −0.123). After adjusting for age and all other covariates, we did not detect any associations between spousal loss and cognitive function within any strata of leisure activities at follow-up (Web Table 2).

Time since loss of a spouse

We found no association between time since loss of a spouse and the risk of dementia or mild cognitive impairment (Table 4). Figure 2 shows the mean scores of executive functioning by time since loss of spouse for women, adjusted for age at cohort entry. Widowed women who had lost a spouse during the previous 2 years had significantly worse executive function compared with still-married women ($P < 0.02$). No such difference was observed for men's executive function or for memory and speed of processing (data not shown).

Sensitivity analysis

To address potential differences in follow-up times between the exposed and unexposed cohorts, we performed a sensitivity analysis. We were able to match 771 nonexposed men (married) to 337 exposed men (widowers) and 1,096 nonexposed women (married) to 654 exposed women (widows). Hence, the "follow-up" times between 1978 and the AGES-Reykjavik Study visit were the same, or 25.2 and 25.1 years, respectively, for widowed and married participants. The results from the logistic regression on the matched data set did not differ from our original findings (Web Table 3).

DISCUSSION

Our study of 4,370 individuals who were married or living with a partner in 1978, whose cognitive status was carefully assessed from 2002 through 2006, does not support an association between loss of a spouse and risk of dementia. However, we did find that women who had been widowed for 2 years or less had significantly worse executive functioning compared with still-married women.

We are aware of only 1 similar population-based study on marital status and dementia in later life (31). In that Finnish study, being widowed was associated with a more than 2-fold greater risk of any cognitive impairment (dementia plus mild cognitive impairment) compared with still-married or cohabiting people. As mentioned previously, our main analysis did not appear to support these results. Moreover, in the same Finnish study, the risk for any cognitive impairment was even higher for those who were widowed at baseline (in midlife) and at reexamination (in late life). We accordingly performed additional analysis on even longer-term widowhood (i.e., up to 30 years), including individuals who were widowed at their first visit to the Reykjavik Study for midlife measurement (before 1978) and were therefore not included in the main analysis ($n = 221$). However, this additional analysis rendered similar results as we obtained in our main analysis, showing no association between long-term widowhood and the risk of dementia (data not shown). Thus, differential duration of widowhood is unlikely to explain the contradictory findings from these 2 studies. Moreover, the 2 studies had similar designs and recruitment procedures and the diagnostic criteria for dementia were similar, as was the ability to control for similar confounding factors. However, differences in findings from these 2 studies may pertain to differences in the average age of the study populations (76.8 vs. 71.1 years), as well as the fact that the present study had complete follow-up of marital status for 24–28 years through a national registry, whereas the former study assessed marital status at only 2 time points (in midlife and late life). In addition, there was a substantial difference in the study sizes (4,370 participants (6.4% with dementia) in the present study vs. 1,449 participants (3.9% with dementia) in the Finnish study).

Our results are supported by findings of previous studies reporting null association between widowhood and the risk of dementia. Helmer et al. (28) did not find an association between widowhood and diagnosed dementia in a French population–based cohort study of 3,675 individuals with an average of 5 years of follow-up. Fratiglioni et al. (22) did not find a statistically significant increased risk of diagnosed dementia associated with widowhood in a cohort study of 1,202 individuals with an average of 3 years of follow-up. However, the relatively short follow-up period might be a methodological shortcoming of these studies because there may be insufficient time for cognitive changes to manifest.

With respect to cognitive function, Van Gelder et al. (25) studied 1,042 men and found, after 10 years of follow-up, a greater decline in cognitive scores (on the Mini–Mental State Examination) among men who had lost a spouse during the study period than among those who remained married. Mousavi-Nasab et al. (26) found a greater decline in episodic memory among widowed individuals than among married individuals over a 5-year period. Aartsen et al. (24) followed 1,144 individuals for 6 years and found a greater decline in memory among those who lost a spouse compared with those whose spouses remained alive. Taken together, these findings are, to some extent, in line with ours, indicating that loss of a spouse may affect cognitive function, at least temporarily. However, in addition to the possibility of random findings, it is unclear why some studies suggested a

Table 3. Associations Between Spousal Loss and Cognitive Performance From Linear Regression in the AGES-Reykjavik Study, 2002–2006

Subgroup by Cognitive Function	No.	%	Crude		Age Adjusted		Full Model[a]	
			β	95% CI	β	95% CI	β	95% CI
Memory	3,728	100						
All with spouse alive	2,635	71	0.00	Referent	0.00	Referent	0.00	Referent
All with spouse deceased	1,093	30	−0.156	−0.218, −0.094	0.069	0.009, 0.129	−0.035	−0.096, 0.026
Men								
Spouse alive	1,422	84	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	266	16	−0.236	−0.341, −0.130	−0.051	−0.152, 0.050	−0.002	−0.103, 0.099
Women								
Spouse alive	1,213	60	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	827	40	−0.338	−0.417, −0.260	−0.075	−0.153, 0.004	−0.044	−0.122, 0.034
ApoE E4 noncarrier								
Spouse alive	1,887	70	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	797	30	−0.167	−0.241, −0.094	0.051	−0.020, 0.122	−0.050	−0.121, 0.021
ApoE E4 carrier								
Spouse alive	688	71	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	277	29	−0.117	−0.242, 0.008	0.139	0.022, 0.257	0.014	−0.104, 0.132
Speed of processing	3,826	100						
All with spouse alive	2,709	71	0.00	Referent	0.00	Referent	0.00	Referent
All with spouse deceased	1,117	29	−0.212	−0.264, −0.160	−0.007	−0.056, 0.042	−0.017	−0.065, 0.031
Men								
Spouse alive	1,464	84	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	272	16	−0.313	−0.410, −0.216	−0.119	−0.210, −0.029	−0.061	−0.146, 0.025
Women								
Spouse alive	1,245	60	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	845	40	−0.273	−0.336, −0.210	−0.040	−0.102, 0.023	−0.002	−0.061, 0.056
ApoE E4 noncarrier								
Spouse alive	1,926	70	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	816	30	−0.209	−0.269, −0.149	−0.016	−0.073, 0.041	−0.022	−0.077, 0.033
ApoE E4 carrier								
Spouse alive	717	72	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	280	28	−0.198	−0.305, −0.090	0.048	−0.052, 0.149	0.006	−0.094, 0.105
Executive function	3,865	100						
All with spouse alive	2,739	71	0.00	Referent	0.00	Referent	0.00	Referent
All with spouse deceased	1,126	29	−0.211	−0.256, −0.166	−0.074	−0.119, −0.030	−0.044	−0.090, 0.002
Men								
Spouse alive	1,483	84	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	274	16	−0.228	−0.316, −0.139	−0.073	−0.157, 0.010	−0.032	−0.115, 0.050
Women								
Spouse alive	1,256	60	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	852	40	−0.223	−0.277, −0.169	−0.086	−0.142, −0.030	−0.069	−0.125, −0.014
ApoE E4 noncarrier								
Spouse alive	1,949	70	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	821	30	−0.219	−0.272, −0.166	−0.094	−0.147, −0.042	−0.055	−0.109, −0.001
ApoE E4 carrier								
Spouse alive	723	72	0.00	Referent	0.00	Referent	0.00	Referent
Spouse deceased	284	28	−0.190	−0.279, −0.100	−0.012	−0.098, 0.073	−0.009	−0.097, 0.079

Abbreviations: AGES, Age, Gene/Environment Susceptibility; ApoE, apolipoprotein E; CI, confidence interval.

[a] Adjusted for age, sex, ApoE E4 status, education, smoking (ever or never), midlife hypertension, midlife regular physical activity (yes or no), and midlife body mass index (weight (kg)/height $(m)^2$). Individuals diagnosed with dementia were excluded ($n = 281$).

Table 4. Multivariable Logistic Regression–Derived Odds Ratios[a] for the Associations Between Time Since Loss of Spouse and Dementia/Mild Cognitive Impairment in 4,189 Participants in the AGES-Reykjavik Study, 2002–2006

Years Since Loss of Spouse	No.	%	Mild Cognitive Impairment ($n = 406$ Cases)						Dementia ($n = 281$ Cases)					
			Crude		Age Adjusted		Full Model[b]		Crude		Age Adjusted		Full Model[b]	
			OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI	OR	95% CI
0–1.9	179	4	1.20	0.73, 2.00	0.82	0.49, 1.39	0.72	0.39, 1.31	1.39	0.77, 2.51	0.84	0.45, 1.54	0.81	0.40, 1.64
2–4.9	210	5	1.59	1.04, 2.44	1.06	0.68, 1.66	0.97	0.59, 1.61	1.42	0.82, 2.46	0.88	0.49, 1.56	0.66	0.32, 1.34
5–9.9	310	7	1.25	0.84, 1.86	0.82	0.54, 1.23	0.94	0.60, 1.47	2.20	1.49, 3.26	1.26	0.83, 1.91	1.28	0.79, 2.08
≥10	574	14	1.65	1.25, 2.18	0.97	0.72, 1.30	1.13	0.80, 1.59	2.04	1.48, 2.81	1.01	0.82, 1.42	0.94	0.61, 1.45

Abbreviations: AGES, Age, Gene/Environment Susceptibility; CI, confidence interval; OR, odds ratio.

[a] Reference group is those with spouse alive ($n = 2,916$).

[b] Adjusted for age, sex, apolipoprotein E E4 status, education, smoking (ever or never), midlife hypertension, midlife regular physical activity (yes or no), and midlife body mass index (weight (kg)/height $(m)^2$).

potential impact of psychological stress on memory, whereas our results pointed to executive function.

Our findings of decreased cognitive performance for childless widowed individuals compared with childless still-married individuals have, to our knowledge, not been reported before. Although we remain cautious when interpreting these results because the number of people without children was small, it is indeed possible that when faced with stressful life events such as the loss of a spouse, the social support from an adult child could potentially protect against the development of cognitive decline. Social support—an important buffer of psychological distress—has repeatedly been reported to reduce the risk of cognitive decline in older people (45–47).

Our study has several qualities that address methodological shortcomings of earlier studies. An important strength of this study was the large population-based cohort and its detailed assessment of dementia and cognitive function, along with other health profile indicators. The richness of information on the outcome allowed us to obtain a more thorough understanding of various aspects associated with cognitive impairment. Furthermore, record linkage to Statistics Iceland permitted complete follow-up for marital status change for over 25 years. Thus, we had a unique opportunity to explore potential associations between this relatively common life stressor (i.e., spousal bereavement) on the development of cognitive impairment. In addition, information on exposure (loss of a life partner) was collected totally independently from the outcome ascertainment (dementia, mild cognitive impairment, and cognitive function). Moreover, we were able to account for important confounding factors, (e.g., ApoE genotype) in our analyses.

Nevertheless, our study has limitations. The study population is a cohort of older people who survived long enough to enter the study. Thus, it is possible that our findings are affected by survival bias, particularly if the individuals most severely affected by spousal loss die or are too frail at follow-up for cognitive assessment. Such bias would result in lower observed point estimates. We cannot exclude the possibility that such differential survival may affect our results. In summary, no overall association between spousal loss and development of dementia was suggested. However, we found that spousal loss may temporarily reduce executive function in women.

Figure 2. Mean scores of executive functioning by time since loss of a spouse for women, adjusted for age at entry into the Age, Gene/Environment Susceptibility (AGES)-Reykjavik Study in 2002–2006. F test $P = 0.019$.

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