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Loneliness, health, and mortality in old age: A national longitudinal study

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ABSTRACT

This study examined the relationship between loneliness, health, and mortality using a U.S. nationally representative sample of 2101 adults aged 50 years and over from the 2002 to 2008 waves of the Health and Retirement Study. We estimated the effect of loneliness at one point on mortality over the subsequent six years, and investigated social relationships, health behaviors, and health outcomes as potential mechanisms through which loneliness affects mortality risk among older Americans. We operationalized health outcomes as depressive symptoms, self-rated health, and functional limitations, and we conceptualized the relationships between loneliness and each health outcome as reciprocal and dynamic. We found that feelings of loneliness were associated with increased mortality risk over a 6-year period, and that this effect was not explained by social relationships or health behaviors but was modestly explained by health outcomes. In cross-lagged panel models that tested the reciprocal prospective effects of loneliness and health, loneliness both affected and was affected by depressive symptoms and functional limitations over time, and had marginal effects on later self-rated health. These population-based data contribute to a growing literature indicating that loneliness is a risk factor for morbidity and mortality and point to potential mechanisms through which this process works.

Introduction

Loneliness is a prevalent and serious social and public health problem (Hawkley & Cacioppo, 2010). Research on loneliness, conducted mostly in Western countries, has shown that at any given time, twenty to forty percent of older adults report feeling lonely (De Jong Gierveld & Van Tilburg, 1999; Savikko, Routasalo, Tilvis, Strandberg, & Pitkälä, 2005; Theeke, 2009; Walker, 1993), and from five to seven percent report feeling intense or persistent loneliness (Steffick, 2000; Victor, Scambler, Bowling, & Bond, 2005). Socially isolated individuals tend to feel lonely, but loneliness is not synonymous with being socially isolated. Loneliness can be thought of as perceived isolation and is more accurately defined as the distressing feeling that accompanies discrepancies between one's desired and actual social relationships (Pinquart & Sorenson, 2003). Prospective studies have shown that feelings of loneliness predict depressive symptoms (Cacioppo, Hawkley, & Thisted, 2010), impaired sleep and daytime dysfunction (Hawkley, Preacher, & Cacioppo, 2010), reductions in physical activity (Hawkley, Thisted, & Cacioppo, 2009), and impaired mental health and cognition (Wilson et al., 2007). At the biological level, loneliness is associated with increased vascular resistance (Cacioppo et al., 2002; Hawkley, Berntson, Burleson, & Cacioppo, 2003), increased systolic blood pressure (SBP) (Hawkley, Thisted, Masi, & Cacioppo, 2010), increased hypothalamic pituitary adrenocortical activity (Adam, Hawkley, Kudielka, & Cacioppo, 2006; Steptoe, Owen, Kunz-Ebrecht, & Brydon, 2004), under-expression of genes bearing anti-inflammatory glucocorticoid response elements (GREs), over-expression of genes bearing response elements for pro-inflammatory NF-κB/Rel transcription factors (Cole et al., 2007, Cole, Hawkley, Arevalo, & Cacioppo, 2011), and altered immunity (Kiecolt-Glaser et al., 1984; Pressman et al., 2005). Moreover, an increasing body of research shows that feelings of isolation and loneliness predict mortality (Patterson & Veenstra, 2010; Shiovitz-Ezra & Ayalon, 2010; Tilvis, Laitala, Routasalo, & Pitkälä, 2011).

In this study, we specify a conceptual model that examines several mechanisms that might account for the effects of perceived isolation (i.e., loneliness) on mortality, and we test this model using a sample of U.S. older adults. Our model posits that health – emotional, physical, and functional – is a proximal predictor of mortality and we therefore test for longitudinal effects of loneliness on health to determine whether health variables are plausible mediators of the loneliness effect on mortality. Previous research has tended to focus on the link between loneliness and specific

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diseases or health conditions, often in small, local samples. Most analyses have used cross-sectional data or examined changes in either loneliness or health, not their dynamic interactions. We add to this literature by (1) examining the link between loneliness and a number of general measures of health, (2) using a large, nationally representative sample, and (3) modeling the dynamic interactions between loneliness and health over time.

Loneliness and mortality

Our conceptual model posits that the influence of loneliness on mortality is attributable to the relationships between loneliness and social isolation, unhealthy behaviors, and poor health. Prospective epidemiological studies have shown that objectively indexed social isolation is a major risk factor for morbidity and mortality (House, Landis, & Umberson, 1988). Because feelings of loneliness are more prevalent and intense in socially isolated individuals, the mortality effects of loneliness may be explained, at least in part, by the higher likelihood of being more socially isolated among those feeling lonely. The effect of social isolation on health and mortality, in turn, has been attributed in part to the direct influence of friends and family on a person's health behaviors (e.g., exercise, adequate and regular rest) which influence physiology and health (House et al., 1988). To the extent that socially isolated individuals are more likely to engage in poor health behaviors, health behaviors may help explain their increased mortality risk. Two lines of evidence suggest otherwise, however. First, health behaviors in epidemiological and field studies have failed to explain the health effects of social isolation in humans (Hawkley, Thisted et al., 2010; Seeman, 2000). Second, non-human social animals subjected to social isolation are also at increased risk for early morbidity and mortality (reviewed in Cacioppo & Hawkley, 2009), suggesting that the effects of isolation extend beyond the social control of health behaviors exerted by concerned friends and family.

Health behaviors may help explain loneliness differences in mortality risk, however. Our theoretical model of loneliness holds that loneliness activates implicit hypervigilance for social threat in the environment (Cacioppo & Hawkley, 2009). Chronic activation of social threat surveillance diminishes executive functioning, and heightened impulsivity influences the tendency of individuals to engage in health behaviors that require self-control. Consistent with this notion, among middle- and older-age U.S. adults, loneliness was associated with a lower likelihood of engaging in physical activity and a faster decline in levels of physical activity participation over a two-year follow-up period (Hawkley et al., 2009).

Our conceptual model further posits that loneliness differences in mortality may be more directly explained by health, where health outcomes are the more proximal predictors of mortality. We therefore introduce emotional, physical, and functional health as additional mechanisms that may explain the association between loneliness and heightened risk of mortality. In this study, the emotional health outcome to be examined is depressive symptoms, the physical health outcome is self-rated health, and the functional health outcome is functional limitations. Justification for these outcomes as plausible mediators of loneliness differences in mortality is based on evidence showing that loneliness predicts these outcomes (as will be discussed below) and that these outcomes predict mortality (Ariyo et al., 2000; Everson, Roberts, Goldberg, & Kaplan, 1998; Idler & Benyamini, 1997; Okun, August, Rook, & Newsom, 2010).

Loneliness and emotional, physical, and functional health

Loneliness feels bad, as is evident by lower levels of well-being (Cacioppo et al., 2008) and higher levels of depressive symptoms (Nolen-Hoeksema & Ahrens, 2002) in lonelier individuals. Beyond cross-sectional associations between loneliness and depressive symptoms, loneliness leads to increases in depressive symptoms in longitudinal U.S. studies (Cacioppo et al., 2010; Hagerty & Williams, 1999; Wei, Russell, & Zakalik, 2005). Accordingly, we expect that loneliness will predict increases in depressive symptoms over time in our sample, and that this predictive role is necessary if depressive symptoms are to be considered plausible mechanisms through which loneliness affects mortality.

Loneliness is associated with poor physical health, and this relationship is effectively captured in the inverse association between loneliness and self-rated health (Segrin & Domschke, 2011; Stephens, Alpass, Towers, & Stevenson, 2011). Moreover, loneliness and increases in loneliness over time predict decrements in self-rated health (Nummela, Seppänen, & Uutela, 2011). We expect that loneliness will predict decreases in self-rated health over time in our sample, and that this predictive role is necessary if self-rated health is to be considered a plausible mechanism through which loneliness affects mortality.

Finally, we examine whether loneliness is related to functional limitations and increases in functional limitations over time. Physical activity is important in maintaining higher levels of physical functioning (Keysor, 2003; Lee & Park, 2006; Netuveli, Wiggins, Montgomery, Hildon, & Blane, 2008), and because lonely individuals are less likely to engage in physical activity than their non-lonely counterparts (Hawkley et al., 2009), they are more likely to experience the onset or worsening of limitations. Prior cross-sectional research has shown an association between loneliness and functional limitations (Greenfield & Russell, 2011; Prieto-Flores, Forjaz, Fernandez-Mayoralas, Rojo-Perez, & Martinez-Martin, 2011), but a plausible role for functional limitations in explaining loneliness differences in mortality would gain support if loneliness predicts increases in functional limitations while controlling for concurrent effects of functional limitations on loneliness.

Our analyses allow reciprocal relationships between loneliness and each health outcome, thus providing more rigorous assessments than previous studies of the causal directions between these variables. We hypothesize that loneliness negatively affects each later health outcome, even after accounting for the potential effects of poor health on later feelings of loneliness. All models linking loneliness and health take into account sociodemographic characteristics of the individual, including age, gender, race/ethnicity, education, and household income and assets, as well as measures of social isolation or, conversely, social relationships (marital status, the presence of relatives, and separately, friends in the neighborhood), and health behaviors (sleep quality, physical exercise, smoking history and current smoking).

Methods

Data mainly come from the 2002, 2004, 2006 waves of the Health and Retirement Study (HRS) although mortality data in 2008 were also used. The HRS is a U.S. nationally representative, longitudinal study of older adults composed of five birth cohorts that entered the study in different calendar years and the data are publicly available. The HRS began in 1992–93 as two separate samples: the original HRS focusing on 1931–41 birth cohorts and the Assets and Health Dynamics among the Oldest Old focusing on 1890–1923 birth cohorts. In 1998 the two samples were merged and two new samples, Cohort of Depression Age (1924–30 cohorts) and War Babies (1942–47 cohorts), were added, and in 2004, another new sample, Early Baby Boomers (1948–53 cohorts), was added, making the sample representative of those born in 1953 or before, approximately aged over 50 in 2004. Once they have

entered the study, respondents were re-interviewed every two years. The spouses were also interviewed irrespective of their age. The sample for each cohort was derived from the same stratified, multistage area probability design in which blacks, Hispanics, and Floridians were over sampled. The HRS now includes over 30,000 respondents. The initial cohort response rates ranged from 70 percent to slightly over 80 percent; re-interview rates for all cohorts at each wave have been between 92 and 95 percent (Health and Retirement Study, 2011).

Since its inception in 1992, the HRS has focused on the health, economics, and demographics of aging and the retirement process. The 2002 wave of HRS included a module on loneliness and 2190 respondents were randomly selected to answer the questions in this module. Among them 2101 respondents were aged 50 years and above and they constituted our analytical sample. In 2004, 1937 were re-interviewed. In 2006, 1815 were re-interviewed (including some who were not interviewed in 2004). Between 2002 and 2008, 303 respondents died. Loneliness questions were asked again of 1756 respondents in 2004 primarily during in-person interviews, and were asked again of 1620 respondents in 2006 in self-administered questionnaires that were left with the respondents upon the completion of an in-person core interview. Note that not all of the 2002 loneliness module respondents were selected to answer questions on loneliness in both 2004 and 2006.

Loneliness

In each wave, HRS asked how often the respondent feels (i) lack of companionship, (ii) left out, and (iii) isolated from others. This three-item loneliness scale was adapted from the standard measure of loneliness, the Revised UCLA Loneliness Scale, and it has been shown to have good internal consistency and both concurrent and discriminant validity (Hughes, Waite, Hawkley, & Cacioppo, 2004). The three-point response scale for each item ranges from "hardly ever or never" to "often." A loneliness scale was created by summing scores on the three items. It ranges from 3 to 9 with higher values indicating a greater degree of loneliness; Cronbach's alpha is .73 for 2002, .77 for 2004, and .81 for 2006.

Mortality

The study assesses mortality between 2002 and 2008. Mortality was determined through matching to the National Death Index or from contacts with household members through 2008. HRS tracking studies indicated a 98.8% validation of deaths (Health and Retirement Study, 2011). Month and year of death were released in a tracking file. Survival time was the number of months from date of 2002 interview to date of death. Of the 303 respondents who died, 9 had missing date of death and we used multiple imputation methods to assign survival time for these cases (Rubin, 1987).

Health outcomes

Depressive symptoms, self-rated health, and functional limitations were measured in 2002, 2004, and 2006. (1) Depressive symptoms. HRS includes a short version of the Center for Epidemiological Studies Depression Scale (CES-D) designed for telephone interviews with older respondents (Turvey, Wallace, & Herzog, 1999). Each item asked whether the respondent experienced a specific symptom in the past week (e.g., "I felt that everything I did was an effort"). Depression is conceptually related to but distinct from loneliness (Cacioppo et al., 2010). To reduce the overlap in measurement of the two concepts, we deleted the item in the CES-D that states "I felt lonely" from our depressive symptoms scale. We also deleted the item "sleep was restless" because quality of sleep was used as a separate covariate in our multivariate analysis. The number of depressive symptoms is a count of the affirmative responses from the remaining six items, with two items tapping positive affect reverse coded. (2) Self-rated health. Each respondent was asked to rate his or her physical health on a five-point scale from poor to excellent, providing a subjective assessment of his or her health status. (3) *Functional limitations*. The number of functional limitations was calculated by summing responses to twelve items assessing whether the respondent has any difficulty with specific forms of ambulation, such as walking a block and climbing a flight of stairs, or muscle movements, such as moving a large chair or picking up a dime. For the 371 respondents who indicated that they do not do certain activities, we used multiple imputation method to impute their values.

Covariates

Our measures of social relationships include marital status, presence of relatives living nearby, and presence of friends living nearby. (1) Marital status. We compared respondents who are currently married with those who are separated, divorced, widowed, or never married. (2) *Relatives living nearby*. This variable was coded 1 if the answer is yes to the question: "Besides the people living here with you, do you have any relative in your neighborhood?" and 0 if the answer is no. (3) *Friends living nearby*. It was coded 1 if the answer is yes to the question: "Do you have any good friends living in your neighborhood?" and 0 if the answer is no.

Measures of health behaviors include sleep quality, physical exercise, current and past smoking. (1) Sleep quality. Respondents were asked how often they (i) "have trouble falling asleep," (ii) "have trouble with waking up during the night," (iii) "have trouble with waking up too early and not being able to fall asleep again," and (iv) "feel really rested when you wake up in the morning." The three-point response options to each item range from "most of the time" to "rarely or never." With the first three items reverse coded, the sleep quality scale is the sum of responses to the four items. It ranges from 4 to 12 with higher scores indicating poorer sleep quality; Cronbach's alpha is .65 in 2002. (2) *Physical exercise*. This measure was coded 1 if the answer is yes to the question whether on average over the last 12 months the respondent has participated in vigorous physical activity or exercise three times a week or more, and 0 if the answer is no. (3) *Smoking*. Respondents were asked whether they ever smoked cigarettes in their life time and for those who answered yes, whether they smoke cigarettes now. Based on this information, we grouped respondents into three categories: never smoked, past smoker, and current smoker. Multiple imputation was used to assign past smoking status for the 13 respondents who were missing on this information.

Sociodemographics include age, gender, race/ethnicity, education, household income and household assets. Age was measured in years. We distinguish three race/ethnicity categories: black, Hispanic, and white/others. Education was measured with the years of schooling completed. We use the total household income and household assets with missing data imputed by the HRS staff. Household income and household assets were log transformed to adjust for positive skew.

Statistical procedures

We weighted descriptive statistics by the sampling weight. The multivariate analyses were not weighted as research has shown that including variables related to sample selection in the regression produces unbiased coefficients without weights (Winship & Radbill, 1994). First, we conducted survival analysis to examine

the effect of loneliness in 2002 on mortality between 2002 and 2008. Parametric hazard models with Weibull distribution were used to estimate the hazard ratios and 95% confidence intervals. The parametric models produced more efficient estimates than semiparametric (Cox) hazard models (Cleves, Gould, Gutierrez, & Marchenko, 2008), and the Weibull distribution was the most efficient and parsimonious functional form for our sample data based on the Akaike Information Criterion. Preliminary analyses using Cox models showed essentially the same patterns. A series of four hierarchical Weibull hazard models was estimated using Stata Version 11. The first model included loneliness and sociodemographic covariates in 2002, the next three models sequentially added social relationships, health behaviors, and health variables in 2002. We added predictors in blocks of conceptually related variables because we were interested in identifying whether one or more variables within each block were uniquely and independently predictive of mortality. These additive models allowed us to examine the extent to which the effect of loneliness on mortality risk is explained by social relationships, health behaviors, and emotional, physical, and functional health. We performed Sobel tests with bootstrapping to test the significance of the indirect effects and to determine whether the effect of loneliness was significantly reduced with the addition of each block of explanatory variables (Preacher & Hayes, 2008). Because the assumption that indirect effects calculated by the product of coefficients method ($\alpha\beta$) and those calculated by the difference in coefficients method ($\tau - \tau'$) were not always identical in survival analysis (Tein & MacKinnon, 2003), we report results from both methods.

Next, we used cross-lagged path analysis to examine the relationships between loneliness and each health outcome. The survival analysis indicates whether loneliness affects mortality risk and the degree to which this effect is reduced when we hold constant measures of health status, possible mechanisms through which loneliness affects mortality. However, because loneliness and health status were measured at the same time, the causal directions between them cannot be established. For this reason we turned next to cross-lagged panel models and estimated autoregressive and cross-lagged paths, which allowed us to address reciprocal influences of loneliness and health simultaneously (Curran, 2000). These models were estimated with MPlus Version 5 (Muthén & Muthén, 1998–2007). Missing data were not imputed; rather, available data from all 2101 respondents were used in analyses. All models were estimated using Full Information Maximum Likelihood estimation with robust standard errors. The FIML method uses all information of the observed data, including mean and variance for the missing portions of a variable, given the observed portion(s) of other variables. FIML produces consistent and efficient estimates when the data are "missing at random" (MAR) and produces less biased estimates than other methods when the data deviate from MAR (Wothke, 1998). The degree of model fit was assessed with the chi-square goodness of fit statistic and the root-mean-square error of approximation (RMSEA). MacCallum, Browne, and Sugawara (1996) characterized a model with an RMSEA of .08 or less as an adequate fit; Hu and Bentler (1999) characterized a model with an RMSEA of .05 or less as a good fit and .10 or more as a poor fit.

We estimated separate cross-lagged path models for loneliness and each health outcome. All models include sociodemographic characteristics, measures of social relationships, and health behaviors as covariates. Our theoretical models assume that prospective relationships between variables are stable over time. These assumptions were modeled by applying equality constraints to the autoregressive and cross-lagged paths, thereby imposing "stationarity" on the relationships among variables in the model. We also assumed that the 2-year prospective effects of covariates on loneliness, depressive symptoms, self-rated health, and functional limitations did not differ from one time point to another. Therefore equality constraints were applied to each of these covariates over the two 2-year intervals. Correlations between variables and residuals at a given time were also estimated.

Results

Descriptive statistics

Weighted descriptive statistics of the variables are presented in Table 1. The average score of the loneliness scale was quite similar in 2002 and 2004, but increased between 2004 and 2006 ($t = 9.56$, $p < .001$). The latter increase may be in part due to the change in data collection method from in-person interviews in 2004 to self-administered questionnaires in 2006. There were no significant changes in the average number of depressive symptoms over the 4-year period. Self-rated health had deteriorated from 2002 to 2006 ($t = -3.85$, $p < .001$) and the number of functional limitations

Table 1
Descriptive statistics of the HRS sample.

Variables	Year 2002		Year 2004			Year 2006		
	Mean/%	SD	Mean/%	SD	*N*	Mean/%	SD	*N*
Loneliness (3–9)	3.84	1.33	3.80	1.32	1675	4.29	1.53	1540
Depressive symptoms (0–6)	.95	1.43	.89	1.44	1902	.90	1.42	1765
Self-rated health (1–5)	3.35	1.08	3.28	1.08	1936	3.21	1.07	1813
Functional limitations (0–12)	2.58	2.71	2.74	2.85	1937	3.09	3.03	1815
Married %	76.4							
Relatives living nearby %	31.8							
Friends living nearby %	70.3							
Sleep quality poor (4–12)	6.41	2.00						
Physical exercise %	45.7							
Past smoker %	44.3							
Current smoker %	13.5							
Age (50–98)	67.08	9.16						
Female %	59.6							
Black %	7.6							
Hispanic %	5.1							
Education (0–17)	12.67	2.83						
Household income (log) (0–15)	10.57	1.09						
Household assets (log) (0–17)	11.71	2.73						

Note: $N = 2101$ in Year 2002. The numbers are weighted.

had increased ($t = 4.95$, $p < .001$). Intraclass correlation, which indicates within-subject stability, was .51 for depressive symptoms, .67 for self-rated health, and .76 for functional limitations.

Loneliness and mortality

Table 2 presents results from the analysis of mortality between 2002 and 2008. Net of sociodemographic characteristics, feeling lonely in 2002 was associated with increased mortality risk over the subsequent six years (OR = 1.14, 95% CI = 1.06, 1.23) (Table 2, Model I). When social relationships were added in Model II, the change in the coefficient for loneliness was not statistically significant and none of the social relationship measures were associated with mortality risk. A test of mediation using the product of coefficients method showed that neither the total indirect effect nor the indirect effect through each of the social relationship variable was statistically significant. When health behaviors were added in Model III, the coefficient for loneliness did not decrease significantly, although physical exercise and past and current smoking were all associated with mortality in the expected direction. A test of mediation using the product of coefficients method showed that neither the total indirect effect nor the indirect effect through each of the health behavior variables was statistically significant.

The next model (Model IV) added measures of health. The effect of loneliness became only marginally significant when depressive symptoms, self-rated health, and functional limitations were added jointly (OR = 1.07, 95% CI = .99, 1.17), and this attenuation was marginally significant at $p < .1$. Among these health measures, self-rated health and functional limitations each had a significant independent effect on mortality risk. A test of mediation using the product of coefficients method showed that the total indirect effect through all three health variables was significant ($B = .05$, 90% CI = .02, .08, $p < .05$), and the individual indirect effects of self-rated health ($B = .04$, 90% CI = .02, .06, $p < .001$) and functional limitations ($B = .02$, 90% CI = .01, .03, $p < .01$) were also significant.

Not surprisingly, mortality risk was higher for individuals who were older, male, and with lower household income and assets. Education was not associated with mortality risk in Model I when household income and assets were controlled for, but its effect on mortality risk became positive and significant once health measures were added in Model IV. This indicates a statistical suppression effect that reflects correlations between education and health. Specifically, education was associated with better health, but when income/assets and health advantages of education were held constant, education predicted greater risk of mortality.

Loneliness and emotional, physical, and functional health

Fig. 1 shows the cross-lagged relationships between loneliness and depressive symptoms, self-rated health, and functional limitations while controlling for sociodemographic covariates, social relations, and health behaviors. The results support stationary processes and in each case fit the data adequately. The RMSEA was .048 (90% CI = .042, .054) for depressive symptoms, .060 (90% CI = .054, .066) for self-rated health, and .051 (90% CI = .045, .058) for functional limitations. The 2-year cross-lagged effect of loneliness on depressive symptoms was significant ($B = .132$, $p < .001$), and the 2-year cross-lagged effect of depressive symptoms on loneliness was also significant ($B = .113$, $p < .001$). Thus these results provide evidence for a reciprocal relationship between loneliness and depressive symptoms. We also see a similar reciprocal relationship between loneliness and functional limitations. The 2-year cross-lagged effect of loneliness on functional limitations was significant ($B = .086$, $p < .01$), as was the 2-year cross-lagged effect of functional limitations on loneliness ($B = .036$, $p < .001$). The 2-year cross-lagged effect of loneliness on self-rated health was marginally significant ($B = -.019$, $p < .10$), and the 2-year cross-lagged effect of self-rated health on loneliness was significant ($B = -.068$, $p < .01$).

Discussion

This study replicates and extends a growing body of prospective research showing that loneliness has adverse consequences for

Table 2
Hazard ratios of the Weibull models of mortality from 2002 to 2008.

Variables	Model I		Model II		Model III		Model IV	
	Hazard ratio	95% CI	Hazard ratio	95% CI	Hazard ratio	95% CI	Hazard ratio	95% CI
Loneliness in 2002	1.14***	[1.06, 1.23]	1.13**	[1.05, 1.22]	1.12**	[1.03, 1.21]	1.07+	[.99, 1.17]
Married			.91	[.69, 1.21]	.93	[.70, 1.23]	.87	[.65, 1.15]
Relatives living nearby			.92	[.72, 1.17]	.92	[.72, 1.17]	.93	[.72, 1.19]
Friends living nearby			.86	[.66, 1.11]	.95	[.73, 1.24]	.91	[.70, 1.19]
Sleep quality poor					1.02	[.96, 1.08]	.94+	[.89, 1.00]
Physical exercise					.60***	[.47, .78]	.79+	[.61, 1.04]
Past smoker[a]					1.37*	[1.06, 1.78]	1.30+	[1.00, 1.69]
Current smoker[a]					1.57*	[1.06, 2.32]	1.46+	[.98, 2.17]
Depressive symptoms in 2002							.98	[.89, 1.07]
Self-rated health in 2002							.65***	[.57, .75]
Functional limitations in 2002							1.08**	[1.02, 1.13]
Age	1.09***	[1.08, 1.10]	1.09***	[1.07, 1.10]	1.09***	[1.07, 1.10]	1.08***	[1.07, 1.10]
Female	.49***	[.39, .62]	.48***	[.38, .61]	.50***	[.39, .65]	.50***	[.38, .64]
Black[a]	.90	[.62, 1.30]	.90	[.62, 1.32]	.90	[.62, 1.31]	.78	[.54, 1.13]
Hispanic[a]	1.09	[.69, 1.73]	1.09	[.69, 1.74]	1.06	[.67, 1.69]	1.02	[.64, 1.61]
Education	1.02	[.98, 1.06]	1.02	[.98, 1.06]	1.02	[.98, 1.07]	1.05*	[1.01, 1.09]
Household income (log)	.81***	[.73, .89]	.82***	[.74, .91]	.83***	[.74, .92]	.86**	[.77, .96]
Household assets (log)	.95**	[.91, .99]	.95*	[.92, .99]	.96*	[.92, 1.00]	.97	[.93, 1.01]
Chi-square	273.76		276.19		300.77		378.87	
df	8		11		15		18	

Note: $N = 2101$. Results are unweighted. Sobel test of the change in coefficient of loneliness from Model I to Model II is −.005 with bias corrected 90% CI = [−.021, .012], total mediated effect through social relationship variables is .005 with 90% CI = [−.004, .018]; the change in coefficient of loneliness from Model II to Model III is −.012 with 90% CI = [−.030, .007], total mediated effect through health behavior variables is .014 with 90% CI = [−.002, .035]; the change in coefficient of loneliness from Model III to Model IV is −.041 with 90% CI = [−.076, −.001], total mediated effect through health variables is .051 with 90% CI = [.016, .082].
+$p < .1$, *$p < .05$, **$p < .01$, ***$p < .001$.
[a] Reference categories are "Never smoked" and "White or Others."

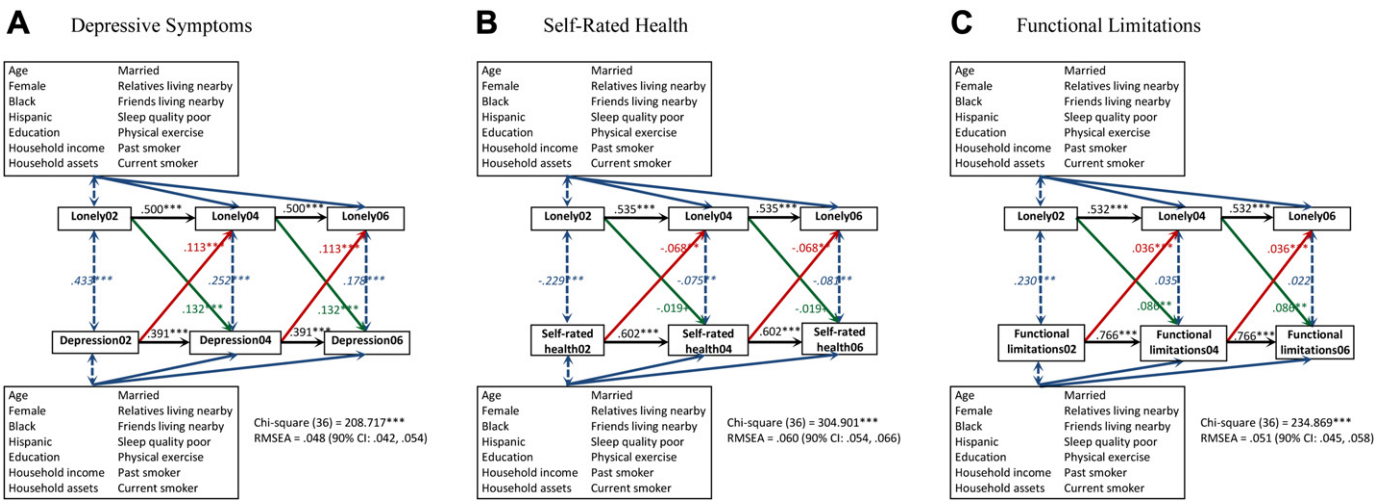

Fig. 1. Cross-lagged relationship between loneliness and health net of sociodemographic covariates, social relationships, and health behaviors. Italicized estimates are standardized covariances (i.e., correlations) and the remaining estimates are unstandardized coefficients.+$p < .1$, $^{*}p < .05$, $^{**}p < .01$, $^{***}p < .001$.

health and mortality. In a large, population-based national sample of older Americans, we replicated prior research by showing that feelings of loneliness are associated with increased mortality risk. Net of sociodemographic characteristics, social relationships, and health behaviors, older adults with the highest levels of loneliness were 1.96 times more likely to die within six years than those with the lowest levels of loneliness.

Several studies exist on the mechanisms that may explain the association between loneliness and mortality. Sugisawa, Liang, and Liu (1994) reported that the effect of loneliness on mortality over a 3-year period among older adults in Japan was fully explained by chronic diseases, functional status, and self-rated health. Patterson and Veenstra (2010) found that the relationship between loneliness and mortality fell to non-significance when social relationships and health behaviors were taken into account. Shiovitz-Ezra and Ayalon (2010) found that the effect of loneliness on mortality remained statistically significant after controlling for medical status, functional impairment, and depression. The apparent inconsistencies may be attributable to differences in the mechanisms that were investigated and the measures that were included. Shiovitz-Ezra and Ayalon (2010), for instance, did not include health behaviors or social relationships in their analysis. In the present study, we included measures of each of these mechanisms. In addition, we used a time sensitive model for survival analysis while some previous studies used a simple "died-didn't die" dichotomy (e.g., Patterson & Veenstra, 2010). Moreover, unlike other studies, we explicitly tested the significance of mediated (i.e., indirect) effects and the resulting change in the magnitude of the direct effect of loneliness. Our results showed that the three health outcomes combined to affect changes in the risk of dying later, and that taking the health variables into account resulted in a small decline in the association between loneliness and mortality. The results of the mediational analyses further indicated that functional status and self-rated health, but not depressive symptoms, constitute proximal mechanisms through which loneliness affects later mortality.

Mortality analysis showed that health outcomes assessed at the same time as loneliness helped explain the effect of loneliness on mortality, but did not permit evaluation of the causal direction between loneliness and health. Using cross-lagged models, we found that loneliness predicted increases in depressive symptoms, modest decreases in self-rated health and increases in functional limitations over two years even when the reciprocal effects of these health measures on loneliness were taken into account. These findings are consistent with a causal direction that implicates decrements in emotional and physical health as mechanisms through which loneliness leads to increases in mortality risk.

Possessing a richness of social attachments and friends protects against mortality (House et al., 1988), and generally signifies lower levels of loneliness (Hawkley et al., 2008; Pinquart & Sorenson, 2003). Ancillary analyses confirmed that respondents who were married and who had more friends living nearby were less lonely (not shown), but we found that these objective characteristics of respondents' social lives did not explain much of the effect of loneliness on mortality risk. This is consistent with Patterson and Veenstra (2010) which, in a much larger sample of more than 6500 adults followed over a 34-year period, found only a modest effect of marital status and no effect of number of close friends and relatives on mortality risk independent of a significant effect of loneliness.

We posited that social relationships may influence mortality to the extent that family and friends exert social control by encouraging and supporting salubrious health behaviors. At the same time, we noted that health behaviors are insufficient to account for the mortality effects of the existence (or not) of social relationships in humans and are not plausible explanations for mortality effects in socially isolated non-human social animals. The effects of loneliness on self-control (Cacioppo & Hawkley, 2009), on the other hand, suggest that health behaviors may differ as a function of loneliness and help explain mortality differences. This reasoning did not find support, however. Although health behaviors influenced mortality as expected, health behaviors (physical exercise, smoking) did not explain loneliness differences in mortality.

The fact that loneliness continues to predict health outcomes when health behaviors are held constant suggests that loneliness alters physiology at a more fundamental level. The sizeable remaining effect of loneliness on mortality in our final model allows for additional explanatory mechanisms. Prior research indicates that loneliness increases vascular resistance (Hawkley et al., 2003), increases SBP (Hawkley, Thisted et al., 2010), alters hypothalamic pituitary adrenocortical activity (Adam et al., 2006), diminishes sleep salubrity (Hawkley, Preacher et al., 2010), alters gene transcription (Cole et al., 2007, 2011), and diminishes immunity (Pressman et al., 2005). Future research should include efforts to examine how these physiological processes contribute to the effect of loneliness on mortality.

This study used a 3-item composite index of loneliness which has been shown to have good validity and reliability (Hughes et al., 2004). This measure is an important improvement over previous studies on the loneliness–mortality relationship that measured loneliness with a single item asking respondents whether and/or how often they felt lonely. Specifically, our 3-item measure avoids use of the term "lonely" or "loneliness" and thus avoids much of the stigma associated with and consequent underestimation of loneliness. Nevertheless, the fact that mean loneliness levels were higher in 2006 than in 2002 and 2004, and that this difference corresponded to a change from an interview-based to a self-administered questionnaire leaves open the possibility that stigma may have resulted in an underestimation of loneliness in the interview-based data. Future waves of HRS, in which loneliness will continue to be assessed by self-administered questionnaire, will allow testing the degree to which the relationships reported in this study are robust to questionnaire format.

Furthermore, even though we analyzed the reciprocal associations of loneliness with depressive symptoms, self-rated health, and functional limitations, arguably other factors, such as sleep quality and physical activities, could be both cause and effect of loneliness and health. Future research needs to construct more complex models to gain a better understanding of the dynamics of these relationships.

Our study contributes to a growing literature indicating that loneliness is a risk factor for morbidity and mortality and it points to potential mechanisms through which this process works. Whether loneliness has similar health effects and operates through similar mechanisms in non-Western countries as was observed in the U.S. remains to be studied, and the importance of such research is highlighted by the fact that loneliness is as or more prevalent in non-Western countries as in the United States. In China, for example, a national survey conducted in 2000 found that 29.8% of older adults were lonely (Yang & Victor, 2008); in rural China, as many as 78.1% of the older adults were reportedly moderately or intensely lonely (Wang et al., 2011). The potential implications for health and longevity are great in a world characterized by societal unrest and threats to the integrity of people's sense of interpersonal and collective connectedness.

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