

# Self-Rated Health and Morbidity Onset Among Late Midlife U.S. Adults

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**Objectives.** Although self-rated health (SRH) is recognized as a strong and consistent predictor of mortality and functional health decline, there are relatively few studies examining SRH as a predictor of morbidity. This study examines the capacity of SRH to predict the onset of chronic disease among the late midlife population (ages 51–61 years).

**Method.** Utilizing the first 9 waves (1992–2008) of the Health and Retirement Study, event history analysis was used to estimate the effect of SRH on incidence of 6 major chronic diseases (coronary heart disease, diabetes, stroke, lung disease, arthritis, and cancer) among those who reported none of these conditions at baseline ( $N = 4,770$ ).

**Results.** SRH was a significant predictor of onset of any chronic condition and all specific chronic conditions excluding cancer. The effect was particularly pronounced for stroke.

**Discussion.** This research provides the strongest and most comprehensive evidence to date of the relationship between SRH and incident morbidity.

**Key Words:** Morbidity—Chronic disease—Self-rated health (SRH)—The Health and Retirement Study

SELF-RATED health (SRH), one of the most widely used survey measures of health, has been shown in numerous studies to be a strong and consistent predictor of mortality (Benyamini & Idler, 1999; DeSalvo, Bloser, Reynolds, He, & Muntner, 2006; Idler & Angel, 1990; Idler & Benyamini, 1997; Kaplan & Camacho, 1983; Mossey & Shapiro, 1982; Shadbolt, Barresi, & Craft, 2002; Walker, Maxwell, Hogan, & Ebly, 2004; Wolinsky & Johnson, 1992). Net of sociodemographic characteristics, multiple health risk factors (i.e., morbidity status, hospitalization, functional health, etc.), and physician-evaluated measures, most studies have found SRH to be a significant predictor of mortality (Idler & Benyamini, 1997). More recently, researchers have turned their attention to the predictive ability of SRH on other health outcomes. Although more limited than the research on SRH and mortality, studies in the United States and internationally have produced evidence that SRH is also a significant predictor of functional health declines (Idler & Benyamini, 1997; Idler & Kasl, 1995; Idler, Russell, & Davis, 2000; Lee, 2000; Martinez, Kasl, Gill, & Barry, 2010). The relationship between SRH and incident morbidity has also been explored, albeit on a much smaller scale. The small number of studies that have examined whether SRH predicts subsequent morbidity onset have produced equivocal results. The purpose of the study is to determine whether SRH predicts subsequent morbidity in a large, representative sample of U.S. middle-aged adults. We examine the effect of SRH on both a global measure of incident morbidity as well as incidence of six major chronic conditions among those with no previously

diagnosed chronic diseases. Our intention is to describe the relationship between SRH and incident morbidity more thoroughly than has previously been undertaken.

We believe that this study extends our understanding of the relationship between perceived and physical health in several important directions. Although there is considerable evidence linking SRH to subsequent declines in functional health and mortality, the evidence linking SRH to incident chronic disease is thin. To our knowledge, this study is the first to examine the extent to which SRH predicts the onset of chronic disease using data from a large, nationally representative study. By including multiple measures of chronic disease (six major chronic conditions and a global measure of chronic disease), we undertake the most comprehensive and systematic examination of the relationship between SRH and subsequent chronic disease to date. Previous studies have shown that SRH is linked to subsequent changes in physical health such as declines in physical functioning and mortality. Our research expands the connection between SRH and physical health to include chronic disease. Moreover, our research suggests that the relationship between SRH and physical health outcomes is evident in midlife as well as at older ages.

## *SRH and Morbidity in Contemporary Research*

SRH or subjective health assessments have been of interest to researchers for numerous decades. Nearly 40 years ago, Maddox and Douglass (1973) noted the usefulness of self-assessments relative to physician assessments of health

in large-scale studies when more objective measures were not practical. The capacity of SRH to predict subsequent mortality has also been documented by researchers for many years (see [Mossey & Shapiro, 1982](#)). Explanations for the link predictive effect of SRH on mortality have generally fallen into two categories. One group of explanations views SRH as a proxy for unmeasured aspects of health. A second alternative is that SRH measures awareness of risk factors for poor health outcomes (such as a family history of disease) and an assessment of available resources for avoiding or managing health problems (such as wealth, knowledge, or social support). The second category of explanation seems particularly compelling in light of the protective role that social and economic resources play in determining health, regardless of the disease or mechanisms involved ([Link and Phelan, 1995](#); [Phelan, Link, & Tehranifar, 2010](#)).

The evidence linking SRH to mortality and declines in physical functioning has recently led some researchers to voice confidence in the capacity of SRH to predict subsequent morbidity. (For example, [Bailis, Segall, and Chipperfield \[2003\]](#) claim “Global self-evaluations of health have proven to be sensitive predictors of morbidity and mortality.” [p. 203], whereas [Riva, Gauvin, and Barnett \[2007\]](#) state that “SRH is a highly predictive measure of morbidity and mortality, independent of other medical, behavioural, or psychosocial factors . . .” [p. 853].) We agree that it is reasonable to assume such a relationship exists given the preponderance of evidence linking SRH to other health outcomes such as mortality and functional health declines. However, we found few studies that have attempted to systematically link SRH to morbidity onset. To our knowledge, only four prior studies have explicitly focused on SRH as a predictor of chronic disease incidence. [Shadbolt \(1997\)](#), the only one of these studies to employ a global measure of chronic disease, examined onset of any chronic disease in relation to SRH in a sample of Australian women. The duration of the study encompassed 4 years (1986/1987 to 1990) and found a significant relationship between SRH and chronic disease onset (net of age, region, and socioeconomic status) but only for those reporting fair or poor health initially. Of the three studies that explored cause-specific morbidity onset, two found significant evidence of a predictive effect of SRH. [Møller, Kristensen, and Hollnagel \(1996\)](#) examined the predictive ability of SRH on coronary heart disease (CHD) on a Danish cohort (born 1936) and discovered that SRH was a significant predictor of CHD incidence (fatal and nonfatal) even after controlling for many potential confounding factors. Another study, [Kaplan and colleagues \(1996\)](#), examined the association between SRH and cardiovascular mortality and morbidity among Finnish men. Although not a significant predictor of fatal and nonfatal myocardial infarctions, SRH did exhibit a significant association with intima-media thickness, forced expiratory volume, and maximal exercise capacity ([Kaplan et al., 1996](#)). Alternatively, [Pijls, Feskens, and Kromhout](#)

(1993) found no evidence that SRH (crude or adjusted) predicted chronic disease incidence (i.e., CHD, cancer, or lung disease/diabetes) for a Dutch cohort (born 1920–1930).

These studies fail to provide a conclusive depiction of the association between SRH and incident morbidity due to both the nature of the designs employed and the findings produced. [Pijls and colleagues \(1993\)](#), [Kaplan and colleagues \(1996\)](#), and [Shadbolt \(1997\)](#) all used samples that consist of only one gender and had a follow-up period of 5 or less years. [Møller and colleagues \(1996\)](#) included both genders and had the longest follow-up duration (16 years); their study is the best evidence of SRH being an independent predictor of morbidity onset. However, the study by [Møller and colleagues \(1996\)](#) only examined CHD. Only one of the four studies investigated the effect of SRH on incident morbidity using a global measure of chronic disease, whereas the other three examined specific chronic condition(s). Moreover, although three investigations found some evidence linking SRH to the onset of chronic disease, one failed to find any association.

Although the current literature does not provide definitive evidence that SRH is an independent predictor of morbidity incidence, we expect to find SRH to be a robust predictor of chronic disease onset. According to [Idler and Benyamini \(1997\)](#), SRH’s ability to predict health declines (especially mortality) has four possible interpretations: (a) SRH is more inclusive than other measures because it can measure pre-clinical symptoms, account for complex human judgments, and family history; (b) SRH accounts for trajectory, not just current health status; (c) SRH influences behaviors; and (d) SRH reflects potential resources such as social environment (e.g., social support) and within person resources (e.g., personality traits). These interpretations lay a conceptual foundation for the predictive capacity of SRH for multiple health outcomes including chronic disease onset.

Moreover, the relationship between SRH and functional health declines also suggests that SRH is an independent predictor of morbidity incidence. Chronic conditions are often the primary cause of functional limitation, especially among older adults ([Boult, Kane, Louis, Boult, & McCaffrey, 1994](#)); therefore, prior research linking SRH to functional limitation suggests a potential pathway between SRH and chronic disease. To illustrate, the Disablement Process (see [Verbrugge & Jette, 1994](#)) model of disability outlines the pathway from pathology (or underlying condition) to disability, and given that previous studies have demonstrated that SRH is a reliable predictor of functional limitation and disability onset ([Idler & Benyamini, 1997](#); [Idler et al., 2000](#); [Lee, 2000](#)), we anticipate that earlier stages of the disabling process including morbidity onset will be subject to self-assessments of health, especially diseases that have a well-defined course and symptomatology.

This research is interested in the ability of SRH to independently predict chronic disease onset, and therefore, it is important that the analyses control for other important

predictors of morbidity onset such as sociodemographic characteristics (i.e., age, gender, race/ethnicity, and education, income, and marital status) and risk factors (i.e., smoking status, physical activity, and body mass index [BMI]). Additionally, we elected to include health care access and utilization measures because the dependent variable relies on self-reports of physician (or other health care professional) diagnosed conditions. Our research contributes to the extant research in this area by examining whether SRH predicts chronic disease onset using both a global measure of chronic disease onset as well as measures of onset for six specific conditions. Additionally, this is the first study of which we are aware based on data from a large, nationally representative longitudinal survey of late midlife adults.

## METHOD

### Data

This research used data from the first nine waves (1992–2008) of the Health and Retirement Study (HRS), which is an ongoing nationally representative longitudinal survey of a U.S. late midlife cohort (born 1931–1941) and their spouses. The HRS is sponsored by the National Institute of Aging (grant number NIA U01AG009740) and the Social Security Administration (HRS, 2008). The initial interviews were conducted as structured face-to-face interviews, whereas subsequent interviews occurring every 2 years via telephone have been collected by the University of Michigan (HRS, 2008). The objectives of the HRS are to describe the lives of older midlife U.S. adults by gathering information about physical and mental health, finances, retirement, and family structure. Blacks/African Americans, Hispanics, and Florida residents were oversampled, and some proxy interviews were conducted after the death of a respondent; the proxy informant was the person “most familiar” with the respondent’s finances, health, and family, which was often the surviving spouse (HRS, 2008). The initial sample size of 12,654 respondents included 7,704 households. In 2004, it was estimated that 15.9% of the original HRS baseline sample had died. Additionally, the RAND HRS Data file (Version K), a user-friendly, longitudinal file created by the National Institute on Aging and Social Security Administration, was used to assist in data management and analysis (RAND, 2011).

### Dependent Variables

*Outcome of interest.*—The dependent variables for this research were dichotomous measures of (a) onset of any major chronic disease and (b) onset of specific major chronic diseases. All HRS participants were asked whether they had ever been diagnosed with six major chronic diseases (CHD, diabetes, stroke, lung disease, arthritis, and

cancer [i.e., any cancer or malignant tumor with the exception of skin cancer]). The wording of the specific questions was: “has a doctor ever told you that you have [name of specific condition].” This information was used to create a time-varying dichotomous indicator coded “1” if a respondent had ever been diagnosed with any of the six conditions and coded “0” otherwise (a global measure of chronic disease onset). A set of similar measures was created to reflect whether a respondent had been diagnosed any one of these specific conditions. These indicators were used to determine the timing of onset of any chronic condition and of onset of specific chronic conditions for those who had not previously been diagnosed with chronic disease.

*Risk group.*—The objective of this analysis was to determine whether SRH predicts initial onset of chronic disease. Accordingly, we defined the at-risk sample to consist of respondents who reported no chronic diseases and at the time of the baseline interview. Because the measure of chronic disease used by the HRS is based on physician diagnosis, it is possible that some respondents may have experienced onset of chronic disease, but had not received a diagnosis. To limit the number of such cases in the risk group, we impose a second criterion: those in the risk group must also be free of impairments with activities of daily living (ADLs) at the time of the baseline interview.

*Proxy interviews.*—For respondents who died (or were institutionalized) during the study, proxy interviews were conducted when possible with a relative to gather information on the circumstances of the respondent’s death. If a proxy interviewee stated that a respondent had been diagnosed with one of the major chronic diseases under investigation before their death then those respondents were recoded as having experienced onset. Among the at-risk respondents (i.e., respondents without any chronic condition or ADL impairment), 1,295 proxy interviews were available over the course of the study. On average, approximately 11% of the respondents per wave had proxy information.

### Independent Variables

The independent variable of interest was SRH. During each interview, respondents were asked to rate their health: “Would you say your health is excellent, very good, good, fair, or poor?” The original coding for this ordinal variable ranged from 1 (*excellent*) to 5 (*poor*). We retained the ordinal level of measure for this variable, but reversed the scale so that excellent health was the highest score (5) and poor health was the lowest (1). SRH was treated as a time-varying variable and was updated at the beginning of each observation interval. To determine whether linear coding was an appropriate parameterization, we compared models with linear coding of SRH to models with in which SRH

was represented by a series of dummy variables. Because the nonparametric (dummy variable) coding of SRH did not significantly improve model fit, we opted for the more parsimonious linear version of this variable.

Numerous covariates were included in the analysis including sociodemographic characteristics, health care access and utilization measures, and risk factors. Sociodemographic characteristics included age, gender, race and ethnicity, education, household income, and marital status. With the exception of household income and marital status, the sociodemographic characteristics were treated as time-fixed variables. A dichotomous dummy variable was created for gender, where female = 1. A four-category dummy variable was created for race and ethnicity with White (reference), Black, Hispanic, and other race as the categories. Education was measured as the amount of years of formal education. Household income was measured at the beginning of each interval and scaled by \$10,000. A four-category dummy variable was created for marital status with married/partnered (reference), divorced/separated, widowed, and never married as the categories; marital status was also assessed at the beginning of each interval.

Health care access and utilization was measured using three variables including health insurance coverage, doctor visits, and hospitalizations. All of the health care access and utilization measures were treated as time-varying variables. Health insurance coverage was determined by self-reports of having private insurance (personal or spousal), government insurance, or no insurance. Respondents with any type of private insurance were categorized as having “private insurance,” whereas those with government insurance, but no private insurance were categorized as having “government insurance,” and respondents reporting no private insurance or government insurance coverage were categorized as having “no insurance.” Both doctor visits and hospitalizations were self-reported and were measured at the beginning of the interval about the past 2 years (subsequent interval length).

Risk factors were also measured as time-varying variables and included BMI, smoking status, physical activity, and in the case of cause-specific morbidity onset models, the other chronic conditions. A four-category dummy variable was created for BMI, which utilized the RAND HRS file calculated (from self-reports of weight and height) BMI measure. The three categories included “underweight” (below 18.5), “healthy weight” (normal; 18.5–24.9), “overweight” (25–29.9), and “obese” (30 and above); these categories were generated using the Centers for Disease Control and Prevention (CDC) BMI guidelines (CDC, 2011). A three-category dummy variable was created for smoking status (i.e., never smoked, former smoker, and current smoker). This variable was constructed from two RAND HRS measures of whether respondents were “current” smokers or had “ever” smoked. Respondents were considered physically active if they participated in vigorous

exercise or sports multiple times per week. From Wave 1 through Wave 6 respondents were asked if they participated in vigorous activity three or more times per week. Beginning in Wave 7, respondents were asked about light, moderate, and vigorous physical activity with multiple frequency categories. Respondents who reported being vigorously physical active at least twice a week were considered physically active. Finally, for the cause-specific morbidity onset analysis, where an individual chronic condition was the outcome variable, the other chronic conditions were treated as risk factors.

### *Analytic Strategy*

The risk group selection criteria (described previously) were intended to identify the subsample of HRS respondents least likely to have any chronic conditions at the beginning of the study and resulted in a sample size of 4,770. The distribution of variables used in the analysis is shown in Table 1a. In general, the subsample used for this analysis differs from the full HRS sample in some notable ways. Because the selection criteria we employ are intended to identify respondents without chronic disease, the subsample has a higher mean SRH. The subsample used in this research is also younger than the full sample, and generally had overall better health measures including less health care utilization, more private insurance coverage, and higher levels of physical activity.

A series of discrete-time Cox proportional hazards models were estimated to ascertain the effect of SRH on likelihood of morbidity onset. The event of interest in these models was initial onset of chronic disease. For each of the eight possible observation intervals generated by the HRS baseline and follow-up interviews, we coded the event variable “1” onset of chronic disease occurred and “0” otherwise. Attrition from mortality or lost to follow-up was modeled as a competing event to chronic disease onset (the attrition analysis is not shown but is available from the authors upon request). Only those members of the risk group who had not previously experienced initial onset of chronic disease or attrition were included in each interval. Because the event under investigation in this analysis was *first* onset of chronic disease (see Table 1b for frequencies across waves), we did not permit reentry into the risk group. That is, once a member of the risk group experienced an initial onset of chronic disease, they were no longer considered at risk of that event. SRH, along with the other time-varying independent variables were measured at the beginning of each observation interval, whereas time-invariant independent variables were measured at the time of the baseline interview. The observation intervals were then pooled to conduct the analysis.

A global measure of chronic condition onset served as the dependent variable for the first series of models (shown in Table 2). The first model included only SRH



and a set of dummy variables representing observation intervals. To determine whether the effect of SRH on incident chronic disease persisted after controlling for factors known to influence the risk of morbidity, we introduced

Table 1a. Descriptive Statistics for Respondents Without Chronic Conditions or ADL Impairment at Baseline ( $N = 4,770$ )

	Distribution information <sup>a,b</sup>
Self-rated health	3.9 (1.0)
Sociodemographic characteristics	
Age	55.3 (3.1)
Gender (female = 1)	48.8%
Race/ethnicity	
White	73.1%
Black	15.0%
Other race	2.1%
Hispanic	9.9%
Educational attainment (# years)	12.4 (3.1)
Household income (scaled \$10,000)	5.3 (5.6)
Marital status	
Married/partnered	79.4%
Divorced/separated	12.2%
Widowed	4.9%
Never married	3.5%
Health care access and utilization	
Insurance coverage	
Private insurance	70.6%
Government insurance	7.3%
No insurance	22.1%
Visit doctor	70.6%
Hospitalizations	4.9%
Risk factors	
Body mass index	
Underweight	1.1%
Healthy weight	38.5%
Overweight	42.4%
Obese	18.1%
Smoking status	
Never smoked	39.3%
Former smoker	35.0%
Current smoker	25.6%
Physically active	22.7%

Note. ADL = activities of daily living.

<sup>a</sup>Percentage distributions are shown for categorical variables; means and (standard deviations) are shown for continuous variables.

<sup>b</sup>Results shown are unweighted.

sociodemographic characteristics in the second model, and included measures of health care access and utilization and health risk factors in the third model. In the second series of models (shown in Table 3), we examine the effect of SRH on the likelihood of onset of six specific chronic conditions. All independent variables described previously were included in each of these models. Additionally, to account for complex stratified sampling, the models were weighted by strata using PROC SURVEYLOGISTIC procedure and the WEIGHT and STRATA statement in *Statistical Analysis Software*. Robust standard errors were used to adjust for clustering at the individual level by employing the CLUSTER statement.

## RESULTS

In the first part of the analysis, a series of discrete-time Cox proportional hazards models were used to examine the effect of SRH and other factors on the likelihood of experiencing onset of first chronic disease (any of the following: arthritis, cancer, CHD, diabetes, lung disease, or stroke). The results of this analysis are shown in Table 2. The first model included only SRH and a series of dummy variables representing the observation intervals. The effect of SRH on onset of first chronic disease is highly significant ( $p < .001$ ). Each unit increase in SRH (higher scores indicate better health) corresponded to a hazard ratio of 0.74 indicating that those with higher SRH were considerably less likely to experience subsequent chronic disease. In Model 2, sociodemographic variables were introduced to determine the extent to which the predictive effect of SRH on chronic disease onset might be a consequence of these compositional factors. The coefficient for SRH changed little and remained significant. Additionally, age, race/ethnicity, and marital status were associated with chronic disease incidence: older respondents were more likely to experience onset of a chronic condition, whereas Hispanic and other race respondents were less likely to experience onset. Compared with married/partnered respondents, divorced/separated and widowed respondents were less likely to experience chronic disease onset. Measures of health care utilization and access and health risk factors were included

Table 1b. Descriptive Statistics<sup>a,b</sup> of Initial Onset for Chronic Conditions by Wave

	Wave 2 (1994)	Wave 3 (1996)	Wave 4 (1998)	Wave 5 (2000)	Wave 6 (2002)	Wave 7 (2004)	Wave 8 (2006)	Wave 9 (2008)
Any chronic condition	11.53% (550)	12.68% (479)	12.60% (379)	13.58% (329)	15.55% (303)	17.64% (266)	18.55% (218)	21.23% (186)
Arthritis	7.39% (352)	8.06% (321)	8.07% (271)	7.47% (213)	8.76% (214)	9.58% (197)	9.94% (175)	11.38% (167)
Cancer	1.01% (48)	1.03% (44)	1.45% (57)	1.95% (70)	2.61% (86)	2.82% (84)	2.69% (74)	3.22% (81)
CHD	1.55% (74)	1.64% (70)	1.86% (72)	2.33% (82)	3.08% (99)	3.68% (106)	3.67% (97)	3.82% (91)
Diabetes	1.34% (64)	1.87% (80)	1.53% (59)	2.07% (73)	3.13% (101)	3.08% (89)	3.34% (89)	4.32% (104)
Lung disease	0.84% (40)	0.58% (25)	0.71% (28)	0.72% (26)	1.21% (41)	1.65% (51)	1.35% (39)	1.84% (49)
Stroke	0.40% (19)	0.58% (25)	0.50% (20)	0.74% (27)	0.77% (26)	1.22% (38)	1.02% (30)	0.88% (24)

Note. CHD = coronary heart disease.

<sup>a</sup>Percentage distributions are shown (with frequencies).

<sup>b</sup>Results shown are unweighted.

in the third model to determine whether the relationship between SRH and subsequent chronic disease could be explained by variation in health that had not yet resulted in chronic disease. The effect of SRH attenuated slightly compared with the previous two models (hazard ratio = 0.76) but remained highly significant ( $p < .001$ ). Age remained significant in Model 3. Gender became significant with the introduction of health care access and utilization measures and risk factors. Women were more likely to experience chronic disease onset compared with men. Hispanic respondents continued to be less likely to experience chronic disease onset in Model 3, whereas the relationship among other race respondents and chronic disease onset was no longer significant. Divorced/separated and widowed also remained significant in the full model. Those who had visited a doctor or were hospitalized during the 2 years prior to the beginning of the observation interval were more likely to subsequently report a chronic condition. Being overweight or obese and being a smoker (both current and former smokers) increased the risk of chronic condition onset. In all three models, a general trend appeared with the observational intervals; over the duration of the study, the likelihood of chronic disease onset increased.

The second part of the analysis was designed to determine whether SRH was associated with the risk of onset of six specific chronic conditions. Accordingly, we estimated a series of six models to determine whether SRH acted as a predictor of the initial onset of six major chronic diseases: arthritis, cancer, CHD, diabetes, lung disease, and stroke. The results of these models are shown in Table 3. The same sets of covariates were used in these models with one exception. To account for the possibility that a respondent developed some other condition prior to the onset of the condition being examined, we included indicators of other conditions in this series of models. For all conditions with the exception of cancer, positive self-ratings of health were associated with a decreased likelihood of onset. SRH had a particularly strong effect on the risk of reporting stroke (hazard ratio = 0.65), whereas SRH was not significantly associated with risk of initial onset of cancer.

As expected, a number of other covariates significantly influenced onset of specific conditions. Older age was associated with increased risk of CHD and lung disease. Women had higher risk for arthritis than men, but lower risk for cancer, CHD, and diabetes. A number of race and ethnicity variations in risk of chronic disease were observed. Compared with Whites, both Black and Hispanic respondents had higher risk of diabetes. Additionally, Blacks had lower risk for CHD and lung disease. The risk of arthritis, CHD, and lung disease were lower for Hispanics than Whites. The only significant effect of more education was to lower the risk of lung disease, whereas more household income lowered the risk of stroke. However, more household income was associated with greater risk of arthritis. Divorced/separated and widowed respondents were less

likely to experience onset of arthritis, whereas widowed respondents were more likely to experience onset of diabetes. Health care utilization affected a number of conditions; a recent hospitalization, prior to the beginning of the interval, was associated with a greater chance of reporting arthritis, cancer, and CHD. Among behavioral risk factors, smoking status not surprisingly had the broadest effects. Compared with nonsmokers, both current and former smokers had much greater risk of reporting lung disease and CHD. The effect of smoking on the risk of incident lung disease was particularly dramatic (hazard ratios of 3.13 for former smokers and 8.31 for current smokers). Former smokers also had a higher risk for arthritis, whereas current smokers had a higher risk of stroke and cancer. Other behavioral risk factors also influenced chronic condition onset: being overweight or obese was associated with elevated risk of arthritis and diabetes, whereas being overweight was associated with increased risk of CHD. Engaging in regular physical activity was associated with a lower risk of diabetes and lung disease.

Among the other chronic conditions, respondents with diabetes were less likely to experience onset of arthritis. Both diabetes and lung disease were associated with greater likelihood of CHD onset. Similarly, respondents with arthritis or CHD were at a greater risk of experiencing lung disease onset. Lung disease greatly reduced a respondent's risk of stroke. Although there was some inconsistency (e.g., lung disease and stroke) across the models, there was a loose trend of increasing risk of experiencing a cause-specific condition over the duration of the study—this was especially true for cancer, CHD, and diabetes.

## DISCUSSION

This study set out to systematically examine the capacity of SRH to predict morbidity onset in a large, nationally representative sample of late midlife U.S. adults. Our results suggest that SRH is a significant independent predictor of global morbidity onset and cause-specific morbidity onset, excluding cancer, even after controlling for important sociodemographic characteristics, health care access and utilization, and risk factors. Cancer was the sole condition where SRH was not a significant predictor of onset in the full model. This may be a result of cancer being broadly defined as any type of cancer (with the exception of skin cancer), where the course of the disease may vary greatly depending on type and stage of diagnosis. Relative to the other chronic conditions in these analyses, arthritis was the most prevalent newly diagnosed chronic disease, which may raise questions about the usefulness of a global measure of morbidity. Nonetheless, this research provides evidence that SRH is a key predictor of morbidity incidence.

In relation to mortality, there is a large body of empirical data that confirms the predictive power of SRH. For

Table 2. Cox Proportional Hazard Ratios of Morbidity Onset, by Self-Rated Health, Sociodemographic Characteristics, Health Care Access and Utilization, Risk Factors, and Time<sup>a</sup>

	Model 1	Model 2	Model 3
Self-rated health	0.74***	0.73***	0.76***
Sociodemographic characteristics			
Age	—	1.02**	1.03***
Gender (female = 1)	—	1.03	1.10*
Race/ethnicity			
White (ref.)	—	—	—
Black	—	1.01	0.94
Hispanic	—	0.75**	0.76**
Other race	—	0.66*	0.73
Education (# years)	—	0.99	0.99
Household income (scaled \$10,000)	—	1.00	1.00
Marital status			
Married/partnered	—	—	—
Divorced/separated	—	0.85*	0.85*
Widowed	—	0.84*	0.82*
Never married	—	0.89	0.93
Health care access and utilization			
Insurance coverage			
Private insurance (ref.)	—	—	—
Government insurance	—	—	0.93
No insurance	—	—	0.94
Visit doctor	—	—	1.17*
Hospitalizations	—	—	1.32***
Risk factors			
Body mass index	—	—	—
Under weight	—	—	1.17
Healthy weight (ref.)	—	—	—
Over weight	—	—	1.27***
Obese	—	—	1.81***
Smoking status			
Never smoked (ref.)	—	—	—
Former smoker	—	—	1.16**
Current smoker	—	—	1.29***
Physically active	—	—	1.04
Time			
Interval 1 (ref.)	—	—	—
Interval 2	1.14	1.14	1.12
Interval 3	1.13	1.13	1.12
Interval 4	1.18*	1.19*	1.16
Interval 5	1.49***	1.51***	1.48***
Interval 6	1.62***	1.64***	1.62***
Interval 7	1.80***	1.84***	1.89***
Interval 8	2.01***	2.05***	2.14***
Intercept	−0.79***	−1.84***	−2.99***
Likelihood ratio	309.53***	386.77***	513.20***
Degrees of freedom	8	18	28

Note. ADL = activities of daily living.

<sup>a</sup>Risk group = no chronic conditions or ADL impairment at baseline ( $N = 4,770$ ).

\* $p \leq .05$ . \*\* $p \leq .01$ . \*\*\* $p \leq .001$ .

example, a meta-analysis of published studies from 1966 to 2003 demonstrated a nearly twofold increase risk of mortality for respondents with “poor” SRH versus those with “excellent” SRH (DeSalvo et al., 2006). Since the discovery that SRH is a reliable predictor of subsequent mortality, additional research has been conducted to attempt to explicate

the relationship. Generally, researchers agree that SRH is a multifaceted measure and that criteria for self-assessments may vary; however, some researchers have attempted to identify the major factors contributing to individual self-assessments. In a number of studies, findings suggested that physical health (e.g., absence/presence of disease, physical functioning, etc.), mental health, and health behaviors had the greatest influence on health self-assessment (Krause & Jay, 1994; Manderbacka, 1998; Manderbacka, Lundberg, & Martikainen, 1999; Singh-Manoux et al., 2006). These studies incorporate ideas laid out by the epidemiological model of SRH, where the emphasis is on SRH as a proxy.

Recently, the common sense model of SRH has highlighted the possibility that more subjective measures (e.g., subjective somatic experiences, social comparisons, time, identity, controllability, personality traits, etc.) account for the persistence of SRH as a predictor of mortality especially after controlling for physical and mental health status (Benyamini, Leventhal, & Leventhal, 2003; Idler, Leventhal, McLaughlin, & Leventhal, 2004; Mora, DiBonaventura, Idler, Leventhal, & Leventhal, 2008). A particularly noteworthy study divided respondents into two subgroups: (a) individuals with no diagnosable diseases and (b) individuals with circulatory system disease, and discovered that individuals with circulatory system disease who classified their health as “poor” or “fair” significantly predicted subsequent mortality adjusted for other risk factors, whereas those with no diagnosable diseases did not (Idler et al., 2004). The authors suggested that the respondents in the unhealthy group were exposed to additional knowledge about their health status, which enabled the respondents to make comparisons and judgments based on experience (Idler et al., 2004). Accordingly, Quesnel-Vallée (2007) argued that the results from the study of Idler and colleagues (2004) were the “most convincing illustration of the salience of [the] individual experiential process” (p. 1162). Our study suggests a broader application of this notion, at least in the case of chronic disease. The risk group in our research was comprised respondents who had never been diagnosed with any of the six chronic conditions we examined and who reported no ADL impairment. Even among those in this relatively healthy group, SRH was a strong and consistent predictor morbidity onset. In the study of Idler and colleagues (2004), the respondents were subjected to physician examinations and were deemed free of any diagnosable condition; our risk group did not have such rigorous screening and it is possible that the differences in the two studies arise from measurement.

On the other hand, it is also possible that the discrepancy in our results compared with the study of Idler and colleagues (2004) is due to the different outcome measured. Idler and colleagues (2004) examined mortality, whereas we examined morbidity onset. There is a possibility that the predictive relationship between SRH and mortality works through morbidity. A potential pathway for the SRH and subsequent

Table 3. Cox Proportional Hazard Ratios of Morbidity Onset, by Self-Rated Health, Sociodemographic Characteristics, Health Care Access and Utilization, Risk Factors, and Time<sup>a</sup>

	Arthritis	Cancer	CHD	Diabetes	Lung disease	Stroke
Self-rated health	0.80***	0.94	0.80***	0.82***	0.74***	0.65***
Sociodemographic characteristics						
Age	1.02	1.03	1.06***	1.01	1.05*	1.04
Gender (female = 1)	1.56***	0.66***	0.67***	0.77**	0.83	0.74
Race/ethnicity						
White (ref.)	—	—	—	—	—	—
Black	0.95	0.83	0.64**	1.33*	0.36***	1.44
Hispanic	0.74**	0.78	0.61**	1.48**	0.49**	0.89
Other race	0.73	0.90	0.75	1.65	0.57	1.48
Education (# years)	0.99	1.02	1.01	0.99	0.95*	1.01
Household income (scaled)	1.01*	1.00	0.99	0.99	0.99	0.94*
Marital status						
Married/partnered (ref.)	—	—	—	—	—	—
Divorced/separated	0.80*	1.03	0.99	1.14	1.41	0.63
Widowed	0.74**	1.36	0.88	1.34*	0.95	1.00
Never married	1.12	1.22	1.07	0.98	0.93	0.45
Health care access and utilization						
Insurance coverage						
Private insurance (ref.)	—	—	—	—	—	—
Government insurance	1.03	0.96	1.03	1.03	0.88	1.20
No insurance	1.01	1.07	0.80	0.98	0.76	0.83
Visit doctor	1.15	1.18	1.23	0.96	1.12	0.85
Hospitalization	1.25**	1.29*	1.35**	1.00	1.14	1.20
Risk factors						
Body mass index						
Underweight	1.01	1.38	1.41	2.04	0.87	1.29
Healthy weight (ref.)	—	—	—	—	—	—
Overweight	1.28***	1.14	1.44***	3.13***	0.93	0.85
Obese	1.57***	1.00	1.25	6.68***	1.02	0.80
Smoking status						
Never smoked (ref.)	—	—	—	—	—	—
Former smoker	1.18*	1.23	1.21*	1.12	3.13***	1.07
Current smoker	1.03	1.41*	1.78***	1.08	8.31***	1.88**
Physically active	1.09	0.94	1.06	0.80*	0.67**	0.86
Other chronic conditions						
Arthritis	—	0.89	0.99	0.93	1.61**	0.93
Cancer	0.98	—	0.90	0.76	1.28	1.00
CHD	1.09	1.07	—	1.22	1.69**	1.33
Diabetes	0.78*	1.10	1.41*	—	0.75	1.48
Lung disease	1.09	1.44	1.53*	1.18	—	0.34*
Stroke	0.98	0.73	1.53	0.88	0.57	—
Time						
Interval 1 (ref.)	—	—	—	—	—	—
Interval 2	1.10	0.91	1.04	1.44	0.74	1.48
Interval 3	1.05	1.47	1.15	1.28	0.98	1.23
Interval 4	0.90	2.03***	1.39	1.58*	0.87	1.63
Interval 5	1.16	2.60***	1.77**	2.60***	1.59	1.87
Interval 6	1.20	2.77***	2.11***	2.34***	2.08**	2.69**
Interval 7	1.28*	2.74***	2.02***	2.48***	1.41	2.34*
Interval 8	1.39*	3.03***	1.96***	3.41***	1.76	1.85
Intercept	-2.89***	-6.16***	-7.05***	-4.84***	-6.91***	5.69***
Likelihood ratio	448.18***	366.77***	519.93***	673.31***	552.93***	416.81***
Degrees of freedom	33	33	33	33	33	33

Notes. ADL = activities of daily living; CHD = coronary heart disease.

<sup>a</sup>Risk group = no chronic conditions or ADL impairment at baseline ( $N = 4,770$ ).

\* $p \leq .05$ . \*\* $p \leq .01$ . \*\*\* $p \leq .001$ .



mortality and/or functional limitations may arise from the robustness of the SRH and morbidity relationship. Our findings suggest that SRH is an extremely sensitive measure, which supports Jylhä's (2009) model of individual health evaluation. Jylhä (2009) argues that SRH has a biological basis as well as a cognitive component and attempts to unify both the social and biological explanations of SRH. Further evidence of Jylhä's proposed model comes from a study that examined biomarkers and SRH; the authors observed a graded relationship for SRH and multiple biomarkers (i.e., blood levels of albumin, white blood cell count, hemoglobin, high-density lipoprotein cholesterol, and creatinine; Jylhä, Volpato, & Guralnik, 2006). Jylhä's (2009) model highlights the sensitivity of SRH and offers an explanation of why SRH significantly predicts subsequent mortality, functional limitations, or morbidity after adjusting for other risk factors. We controlled for sociodemographic characteristics, health care access and utilization, and other health risk factors, and SRH continued to be a significant predictor of morbidity onset among a relatively healthy group of late midlife U.S. adults. It is obvious that individuals are very good at making health assessments; however, there is still much to learn about SRH, and we suggest that, in the future, researchers explore the potential pathway of the SRH and subsequent mortality/functional limitation as a function of SRH and morbidity onset. Additionally, we encourage researchers to investigate the sensitivity of SRH and morbidity onset in the context of biological and social factors.

The findings of this study should be viewed in light of potential limitations that stem from the measurement of the outcome variable (onset of chronic disease). Onset of chronic disease, the event of interest, is based on respondent reports of physician diagnosis (questions about chronic diseases use the wording "has a doctor ever told you that you have" a specific condition). Because some respondents may not use or have access to health care, chronic disease is likely underreported. The potential consequences of this measurement strategy on the analyses are twofold. First, there may be respondents identified as being at risk of first onset of chronic disease (i.e., those who were free of chronic disease) who suffered from a disease but had not been diagnosed by a physician. We attempted to minimize the extent of this misclassification by selecting only respondents who had not been diagnosed with any of the six chronic conditions asked about in the HRS and who were also free of any ADL disability. Second, there may have been respondents who reported experiencing onset of chronic disease during the study interval, but in actuality did not have the recounted disease. Prior literature has noted that self-reports of common chronic conditions have strong agreement with medical records (Haapanen, Miilunpalo, Pasanen, Oja, & Vuori, 1997; Simpson et al., 2003). Another major limitation is that the global measure is conceptualized to measure first onset of chronic disease. We cannot establish whether another chronic disease, not included in the six used, was

experienced by a respondent previously; however, the six chronic conditions utilized in this study are the most common and encompass a large portion of chronic morbidity incidence. Information collected from proxy interviews was used to establish chronic disease onset for deceased respondents who had previously reported not having a specific condition. Although proxy information introduces reliability concerns, proxy interviewees were the person most familiar with the respondent's health and finances; therefore, the information provided, typically by a surviving spouse, was from an intimate source. Furthermore, sensitivity analyses omitting respondents with proxy information yielded similar results. Finally, like all longitudinal analyses, attrition due to lost to follow-up and mortality was a potential source of selection bias; therefore, attrition was included in the analyses as a competing event. As expected, respondents with higher ratings of SRH were less likely to attrite, which suggests that respondents with low SRH were underestimated in our analyses. Despite these limitations, this study provides the strongest and most comprehensive evidence to date of a relationship between SRH on the onset of chronic disease.

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