Published in final edited form as:
Soc Sci Med. 2012 August ; 75(3): 526–530. doi:10.1016/j.socscimed.2012.04.004.

Transition to retirement and risk of cardiovascular disease: Prospective analysis of the US Health and Retirement Study

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Abstract

Transitioning from work to retirement could be either beneficial or harmful for health. We investigated the association between transition to retirement and risk of stroke and myocardial infarction (MI). We followed US Health and Retirement Study participants age 50+ working full-time for pay and free of major cardiovascular disease (n=5,422) in 1998 up to 10 years for transition to full retirement and self- or proxy-report of either stroke or MI (CVD; 665 events). We used discrete-time survival analysis to compare the CVD incidence for the fully retired versus the full-time working population. To distinguish short-term from long-term risks, we compared the association in the first year after retirement to estimates 2+ years after retirement.

In the full model adjusting for age, sex, childhood and adult SES, behavior, and co-morbidities, being retired was associated with elevated odds of CVD onset (OR=1.40, 95% CI: 1.04, 1.90) compared to those remaining in the full-time labor force. The odds ratio for CVD incidence within the first year of retirement was 1.55 (95% CI: 1.03, 2.33). From the second year post-retirement and thereafter, the retired had marginally elevated risk of CVD compared to those still working (OR=1.35; 95% CI: 0.96, 1.91). Although confidence intervals were wide for some subgroups, there were no significant interactions by sex or socioeconomic status. Results suggest that CVD risk is increased after retirement.

Keywords

stroke incidence; heart attack incidence; myocardial infarction; cardiovascular disease; social disparities; retirement; post-retirement; socioeconomic status; USA

Americans now spend more years in retirement than ever before. A major wave of retirement started in 2011, when the first Baby Boomers (born 1946–1964) began to turn 65

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(He, Sengupta, Velkoff, & DeBarros, 2005). By 2050, the older population is projected to number 86.7 million. The average age of retirement, which has been declining since the 1950s, has once again been falling again since 1990 after having leveled off during the mid-1970s through 1980s (Gendell, 2001). With the first wave of the Baby Boomers' retirement and spiraling health care costs for the elderly, the question of how retirement affects health is of vital importance.

Theoretically, it has been argued that retirement might have either good or bad effects on cardiovascular health (Kasl & Jones, 2000). Retirement is a lifecourse transition involving environmental changes that reshape health behaviors, social interactions, and psychosocial stressors. It is also a subjective developmental and social psychological change in identity and preferences (Dannefer, 1984). However, there is little consensus about the impact of the retirement transition on health outcomes. Existing evidence is conflicting, and reverse causality is difficult to rule out because of unobserved selection into retirement.

Empirical findings on the health risks of retirement have been inconsistent, variously reporting harmful, beneficial or no effects. Some early studies suggested that being retired had no deleterious effects on either physical or psychological health (Kasl, 1980; Minkler, 1981). However, those studies often ignored the complexity of the retirement transition, including issues of timing, previous health, and quality of the job (McGoldrick & Cooper, 1988). More current literature reports contradictory findings. A recent study in France showed that retirement was associated with improved self-perceived health for those in poor work environments and those with health complaints before retirement (Westerlund, Kivimäki, Singh-Manoux, Melchior, Ferrie, Pentti et al., 2009). Another recent Swedish study used recorded purchases of antidepressant medication to measure mental health; they found that retirement was associated with decreased anti-depressant usage (Oksanen, Vahtera, Westerlund, Pentti, Sjösten, Virtanen et al., 2011). Others found *adverse* associations, reporting increased musculoskeletal and CVD prevalence among retired Finnish men (Tuomi, Järvinen, Eskelinen, Ilmarinen, & Klockars, 1991), and significantly elevated risk of severe cardiovascular disease or cancer onset associated with retirement in England (Behncke, 2011). There are also studies reporting *no effect* on physical or mental health. One study found that retirement was associated with a reduction in mental and physical fatigue and depressive symptoms, particularly among people with chronic diseases, but retirement did not change the risk of major chronic disease onset (stroke, MI and coronary heart disease) (Westerlund, Vahtera, Ferrie, Singh-Manoux, Pentti, Melchior et al., 2010). These mixed results may be partially attributable to incomplete adjustment for differential selection into retirement, i.e., lack of comparability between retirees and those who continue to work at similar ages (Miah & Wilcox-Gök, 2007).

We investigated factors that may modify the effect of retirement on cardiovascular health, specifically risk of first stroke and myocardial infarction (MI). We hypothesized that higher individual- and family-level SES modifies the association between retirement and cardiovascular health. In this paper, we aim to address both reverse causation and effect modification by exploiting the extensive set of measures available in a nationally representative sample of Americans transitioning into retirement.

Methods

Data were drawn from the nationally representative, longitudinal Health and Retirement Study (HRS), described in detail elsewhere (Juster & Suzman, 1995). Our follow-up began in 1998, the earliest year when the sample was representative of all birth cohorts up to 1947. Biennial interviews (or proxy interviews for decedent participants) were conducted through 2008, by telephone or in person. We included HRS participants born 1900 to 1947 who

participated in the 1998 wave and follow them to 2008. Retention rates through 2008 were above 80%. The HRS was approved by the University of Michigan Health Sciences Human Subjects Committee and these analyses were determined exempt by Harvard School of Public Health Office of Human Research Administration.

In 1998, the age-eligible sample included 17,313 people; we excluded 482 individuals who already had diagnosed MI at baseline in 1998 and 979 with stroke in 1998; 10,322 individuals who were homemakers, disabled or not working full-time; 47 individuals missing information on key covariates; and 61 individuals with no follow-up information available in the 2000 interview. The final analytic sample included 5,422 individuals who were free of MI and stroke and working full-time in the paid workforce in 1998 (Table 1).

We defined new diagnoses of MI or stroke (fatal and non-fatal) as a primary cardiovascular disease (CVD) outcome. For first MI, participants were asked “In the past two years, have you had a heart attack or myocardial infarction?” Information on first stroke came from self- or proxy-report of a doctor’s diagnosis (“Has a doctor ever told you that you had a stroke?”). For participants who were unavailable for a direct interview (e.g., due to death), interviews were conducted with proxy informants, predominantly spouses (<15% for all 5 waves). Due to substantial missing data on month of diagnosis, we used year of diagnosis only to calculate survival.

Retirement status was classified as: not retired/working full-time (reference group), partially retired, or completely retired. Retirement status was time-updated, so participants in employment in 1998 could transition to partial or full retirement. The majority of those partially retired transition into fully-retired, and our estimates contrast the completely retired to those working full time, without presenting separate estimates for the partially retired.

We considered three racial/ethnic groups: non-Hispanic white (“whites”) and non-Hispanic black (“black“) and Hispanic groups. Socioeconomic status (SES) measures included self-reported years of completed education, per capita household income and wealth as of 1998. Values for income and wealth were equivalized by the number of household members by dividing household income by the square root of household size. Other covariates included baseline age, age-squared, gender, and birth in a southern state (Glymour & Manly, 2008; Luo & Waite, 2005), and parental maximum education (<8/≥8 years, higher value chosen between mother’s and father’s education).

Additional covariates included behavioral risk factors and co-morbidities (see Table 1). Behavioral risk factors consisted of smoking status, drinking status, body mass index (kg/m^2), and vigorous physical activity. Co-morbidities included self-reported diagnoses of hypertension and diabetes, depressive symptoms, self-rated health, limitations in activities of daily living (ADL) and instrumental activities of daily living (IADL). The effect estimates for CVD post-retirement was slightly attenuated in models using time-varying values for these variables, but findings were similar as for models that used baseline values. We present models using baseline values of these covariates to avoid controlling for potential mediators of the relationship between retirement and CVD.

We hypothesized that the risks of retirement might change as time since retirement elapsed; we categorized each person with respect to retirement status and years since retirement (0 in the retirement year, up to 10 years). We used information on the year of retirement only, because over 20% of the study population had missing information on month of retirement.

We used discrete-time survival analyses based on pooled logistic regression models to compute the incidence rates for CVD. Survival was defined as remaining event-free at each of 5 biennial interview waves (1998–2008). Respondents were censored after the first CVD

event or at the time of death. Analyses were conducted in SAS 9.3 (Cary, NC) PROC SURVEYLOGISTIC, correcting for the complex sample design. We applied the HRS sampling weights to make the population representative of the 1998 US community-dwelling population aged 50+ years.

The primary analysis assessed the main effect of retirement on risk of first stroke and MI. Initial models controlled for sex, baseline age, and time (years sinec baseline, (years since baseline)^2, and years since retirement). Supplemental models adjusted for childhood SES (parental education and Southern birth), adult SES (education), family-level SES (household income and wealth), and health behaviors and co-morbidities. Covariates were added sequentially to help identify the most important possible confounders.

We distinguished the short-term risk of CVD associated with retirement from the long-term risk by comparing the hazard ratios during the first year after retirement to those during the second year and beyond. We then examined effect modification in analyses stratified by gender, race and SES (education, household income and household wealth), and provide formal tests of interaction terms for retirement and each hypothesized effect modifier.

Results

There were 665 self- or proxy-reports of first incidence of stroke or MI by 2008. Adjusted for age and sex, on average over 10 years of follow-up time, retirement was associated with elevated odds of CVD onset (OR=1.62, 95% CI: 1.20, 2.18) compared to the full-time working population (Table 2). In models adjusted for childhood SES, adult SES, family-level SES, behavioral risk factors and co-morbidities, retirement was still associated with significantly increased odds of CVD onset (OR=1.40, 95% CI: 1.04, 1.90). The elevated risk of CVD onset was most pronounced in the first year post-retirement, although confidence intervals overlapped for short- and long-term estimates. Within the first year of retirement, the odds of CVD onset were elevated in both the initial model (OR=1.74, 95% CI: 1.17, 2.60) and the fully adjusted model (OR=1.55, 95% CI: 1.03, 2.33). Two or more years post-retirement, retired status was associated with an attenuated OR of 1.57 (95% CI: 1.12, 2.21), which was non-significant after adding all covariates to the model (OR=1.35; 95% CI: 0.96, 1.91).

Cell counts in stratified analyses were small, leading to imprecise estimates of the short-term outcomes of retirement. We therefore conducted sensitivity analyses defining the first two years–instead of the first year–as short-term outcomes (results not shown). Although the contrast between short- and long-term was attenuated, short-term effect estimates for the first 2 years were still noticeably larger (OR=1.50; 95% CI: 1.10, 2.08) than the long-term (OR=1.32; 95% CI: 0.90, 1.95) in the full model, supporting the finding for a stronger risk in the first years of retirement.

Estimates from stratified analyses had large confidence intervals and were sometimes not significant, but there was no evidence of a significant interaction by gender or SES (Table 3). Interaction tests between gender and the time-since-retirement variable were not significant for either the short-term effect estimate (p=0.50) or the long-term effect estimate (p=0.70). In race-stratified models, we found that whites had 85% increased odds of CVD incidence in the first year post-retirement, (95% CI: 1.21, 2.83), whereas for blacks the effect estimate indicated a non-significant protective association (OR=0.79; 95% CI: 0.33, 1.91). However, the CIs were very wide and the interaction test by race was not significant. The risk of CVD associated with retirement seemed to be less consistent for individuals in the top and bottom 25% of the income distribution, but there was no evidence of a significant interaction by income or other SES measures (Table 3).

Discussion

In this large, nationally representative sample of Americans aged ≥50 years, retirement was associated with an elevated risk of cardiovascular disease (stroke or MI) compared to full-time employment. This elevated risk appeared to be more marked during the first year after retirement, although estimates were not significantly different from those for the second year and beyond. There was no evidence of a significant interaction by gender or SES in the association between retirement and CVD risk.

Although the HRS data are well-suited to conduct this work because of the availability of extensive health and financial data, there are limitations. The CVD outcomes in HRS were based on self- or proxy-reports, without medical record verification. However, prior analyses of self-reported stroke in HRS indicated that results were consistent with studies using medically verified events, suggesting that misreporting is not systematic (Glymour & Avendano, 2009). Extensive prior evidence suggests self-reports of major cardiovascular events correspond well with medical records (Bergmann, Byers, Freedman, & Mokdad, 1998; Carter, Barber, & Shaw, 2010). Also, we were not able to confirm that we established a clear temporal order when new events occurred in the same year as retirement. For instance, if someone had an event in January 2000 and retired in February 2000, our analyses would have treated the CVD as the consequence of retirement. We conducted sensitivity analyses, using both the month and year information to track the respondents to the month of their events to see if retirement date precedes the CVD event dates. Of the 46 events that occurred among the retired during the first year, over 70% had the retirement date preceding the CVD event date. The remaining 30% was missing the month information; therefore we were not able to confirm the temporal order.

Our results suggest an association between retirement and increased cardiovascular disease risk. This association seems to be more pronounced immediately following the retirement transition, and attenuates in the second year and beyond. One explanation of this pattern is that there is an endogenous relationship between retirement and cardiovascular health—those who are not feeling well select into retirement rather than retirement causing the illness. For example, it is possible that people opt to retire when they begin to feel prodromal symptoms of cardiovascular disease. Thus, we cannot exclude the possibility that our findings reflect a process of reverse causation. Since randomization of retirement is infeasible, the strongest evidence on the causal effects of retirement is likely to arise from quasi- or natural-experiments, for example based on changes in mandatory retirement ages, pension eligibility ages, or mandatory early retirement offers. Our findings are consistent with two prior studies in England exploiting legal changes in statutory retirement ages (Behncke, 2011; Dave, Rashad, & Spasojevic, 2006) and spousal retirement status (Behncke, 2011; Dave et al., 2006). They are less consistent with a recent study exploiting statutory retirement ages in European countries and showing positive health effects of retirement (Coe & Zamarro, 2010), but this study did not specifically focus on cardiovascular outcomes. Our analysis focuses on CVD as the endpoint and controlled for potential mediating/modifying factors.

To summarize the prior literature, whether retirement is linked to changes in health status appears to vary according to the health outcome studied (mental health versus physical health) as well as by socioeconomic position (the individual's working conditions prior to retiring). Our findings suggest a higher risk of cardiovascular disease in the immediate post-retirement period, but we cannot exclude reverse causation. On the other hand, the lower bounds of our estimates do not support a beneficial effect of retirement on cardiovascular health. Given the ongoing policy debates in many countries over changing the statutory

retirement age, our study highlights the need for further research to clarify the causal relationships between retirement and health.

Acknowledgments

The authors gratefully acknowledge financial support from the following funding sources: JRM is supported by the National Research Service Award (NRSA) training grant (T32-HL098048-01); MA is supported by a grant from the European Research Council (263684)and a Eur-fellowship from the Erasmus University; MMG is supported by American Heart Association (10SDG2640243).

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Table 1

Sample Characteristics of Study Participants at Baseline, by Gender (weighted): Health and Retirement Study, United States, 1998.

	Overall		Women		Men	
	n/mean	%	n/ mean	%	n/mean	%
n	5,422	100.0	2,926	54.0	2,496	46.0
Mean years of follow-up	8.6		8.7		8.4	
Total person-years of follow-up	46,530		25,532		20,998	
Core Demographic Variables						
Mean age at enrollment (SD)	58	(5.7)	58	(5.6)	58	(5.8)
Race/ethnicity*						
White	4,126	76.1	2,173	74.3	1,953	78.3
Hispanic	414	7.6	195	6.7	219	8.8
Black	763	14.1	497	17.0	266	10.7
Southern birth state	1,867	34.4	1,062	36.3	805	32.3
Adult SES Risk Factors						
Highest parental education (SD)	10	(4.3)	10	(4.2)	10	(4.4)
Mean years of education (SD)	13	(2.9)	13	(2.7)	13	(3.2)
Median income in $1,000s (IQR)	52	(23.5)	450	(20.8)	60	(24.7)
Median wealth in $1,000s (IQR)	128	(82.0)	117	(77.1)	141	(89.2)
Behavioral Risk Factors						
Vigorous Activity (≥3/wk)	2,870	53.0	1,410	48.2	1,460	58.6
Overweight (30-<35 kg/m^2)	2,200	40.6	1,003	34.3	1,197	48.0
Obese (≥35 kg/m^2)	1,303	24.0	685	23.4	618	24.8
Moderate alcohol use (1–2 drinks/2 wks)	1,029	19.0	491	16.8	538	21.6
Heavy alcohol use (≥3 drinks/2 wks)	958	17.7	341	11.7	617	24.7
Current Smoker	1,126	20.8	572	19.6	554	22.2
Past Smoker	3,262	60.2	1,519	51.9	1,743	69.8
ADL (≥1 limitation)	300	5.5	178	6.1	122	4.9
IADL (≥1 limitation)	158	2.9	85	2.9	73	2.9
Chronic Cardiovascular Conditions						
CES-D^b ≥ 3	996	18.4	623	21.3	373	14.9

	Overall		Women		Men	
	n/mean	%	n/ mean	%	n/mean	%
Fair/Poor Self-Assessed Health	832	15.3	470	16.1	362	14.5
Hypertension	1,980	36.5	1,055	36.1	925	37.1
Diabetes	502	9.3	246	8.4	256	10.3
Heart Attack	66	13.4	18	8.7	48	16.8
Stroke	48	0.9	24	0.8	24	1.0

Note: IQR=inter-quartile range; SD=standard deviation.

[a] Categories do not sum to 100% because of rounding.

[b] Measured with a modified 8-item Center for Epidemiologic Studies Depression (CES-D) scale.

Table 2

Hazard Ratios for First Incident Cardiovascular Disease on Average, Immediate (during first year) and Long-term (second year and beyond) Post-retirement: Health and Retirement Study, United States, 1998–2008.

	On Average		*During first year*		*Second Year & Beyond*	
	Events Among Retirees	**HR (95% CI)**	**Events Among Retirees**	**HR (95% CI)**	**Events Among Retirees**	**HR (95% CI)**
Model 1: Adjusted for demographic characteristics[a]	165	1.62 (1.20 – 2.18)	46	1.74 (1.17 – 2.60)	119	1.57 (1.12 – 2.21)
Model 2: Model 1 + Childhood SES[b]	165	1.59 (1.17 – 2.15)	46	1.72 (1.15 – 2.59)	119	1.54 (1.09 – 2.18)
Model 3: Model 2 + Adult (individual) SES[c]	165	1.57 (1.16 – 2.12)	46	1.69 (1.13 – 2.53)	119	1.52 (1.07 – 2.15)
Model 4: Model 3 + Family-level SES[d]	165	1.59 (1.18 – 2.13)	46	1.70 (1.14 – 2.53)	119	1.54 (1.10 – 2.17)
Model 5: Model 4 + Behavioral Risk Factors[e]	165	1.53 (1.14 – 2.05)	46	1.66 (1.11 – 2.48)	119	1.48 (1.06 – 2.08)
Model 6: Model 5 + Co-morbidities[f]	165	1.40 (1.04 – 1.90)	46	1.55 (1.03 – 2.33)	119	1.35 (0.96 – 1.91)

All models are adjusted for both linear and quadratic terms of age at baseline.

Reference group for all models are full-time working population.

[a] Demographic characteristics include sex and race.

[b] Childhood SES includes southern birth and parental education.

[c] Adult individual SES includes education.

[d] Family-level SES includes household income and wealth.

[e] Behavioral risk factors include BMI, drinking, smoking, ADL, IADL and vigorous activity (≥3 times/wk).

[f] Co-morbidities include self-reported diagnoses of depression, hypertension, diabetes and self-assessed health.

Table 3

Hazard Ratios for First Incident Cardiovascular Disease on Average Post-retirement, Stratified by Gender, Race and SES: Health and Retirement Study, United States, 1998–2008.

	On Average		First Year		Second Year +	
	Events Among Retirees	HR 95% CI	Events Among Retirees	HR 95% CI	Events Among Retirees	HR 95% CI
Gender[a]						
Male	93	1.40 (0.95 – 2.07)	30	1.42 (0.82 – 2.47)	63	1.39 (0.90 – 2.16)
Female	72	1.44 (0.92 – 2.25)	16	1.74 (0.89 – 3.40)	56	1.34 (0.82 – 2.19)
Race[b]						
White	125	1.40 (1.01 – 1.94)	40	1.85 (1.21 – 2.83)	85	1.24 (0.84 – 1.81)
Black	30	1.63 (0.78 – 3.39)	4	0.79 (0.33 – 1.91)	26	1.92 (0.88 – 4.21)
Education[c]						
≤High School	107	1.55 (1.04 – 2.31)	26	1.67 (0.98 – 2.85)	81	1.61 (0.98 – 2.34)
>High School	58	1.13 (0.74 – 1.72)	20	1.45 (0.79 – 2.66)	38	1.02 (0.59 – 1.77)
Wealth[c]						
<25th percentile	68	1.84 (1.15 – 2.95)	16	1.75 (0.87 – 3.51)	52	1.87 (1.08 – 3.23)
25th–75th percentile	76	1.27 (0.86 – 1.87)	23	1.53 (0.88 – 2.65)	53	1.16 (0.75 – 1.81)
>75th percentile	21	1.13 (0.59 – 2.16)	7	1.55 (0.52 – 4.65)	14	0.00 (0.47 – 2.15)
Income[c]						
<25th percentile	41	1.21 (0.60 – 2.46)	10	1.24 (0.50 – 3.12)	31	1.20 (0.51 – 2.58)
25th–75th percentile	88	1.82 (1.30 – 2.55)	24	2.09 (1.28 – 3.43)	64	1.71 (1.09 – 2.57)
>75th percentile	36	0.91 (0.53 – 1.57)	12	0.89 (0.39 – 2.01)	24	0.92 (0.51 – 1.64)

Reference group for all models are full-time working population.

[a] Adjusted for years since retirement, age, race, southern birth, parental education, education, income, wealth, health behaviors and co-morbidities.

[b] Adjusted for years since retirement, age, sex, southern birth, parental education, education, income, wealth, health behaviors and co-morbidities.

[c] Adjusted for years since retirement, age, race, sex, southern birth, parental education, health behaviors and co-morbidities.