The Journal of Prevention of Alzheimer's Disease 13 (2026) 100450

Contents lists available at ScienceDirect

The Journal of Prevention of Alzheimer's Disease

journal homepage: www.elsevier.com/locate/tjpad

Original Article

Trajectory of cognitive decline before and after incident heart failure among older adults: A 20-Year, population-based, prospective cohort study

Haibin Li [a,b,*], Frank Qian [c,*], Wuxiang Xie [d], Man Wang [e], Jianian Hua [f], Jiao Wang [g,h], Xinye Zou [i], Zhiyuan Wu [j], Xia Li [k], Deqiang Zheng [l], Xiuhua Guo [l], Hongjia Zhang [m,n,*]

[a] Department of Cardiology, Heart Center, Beijing Chaoyang Hospital, Capital Medical University, No. 8 Gongti South Road, Chaoyang District, 100020, Beijing, China
[b] Medical Research Center, Beijing Chaoyang Hospital, Capital Medical University, No. 8 Gongti South Road, Chaoyang District, 100020, Beijing, China
[c] Section of Cardiovascular Medicine, Boston Medical Center and Boston University Chobanian & Avedisian School of Medicine, 732 Harrison Ave 3rd Floor, Boston, MA 02118, USA
[d] Peking University Clinical Research Institute, Peking University Health Science Center, No. 8 Xishiku Street, Xicheng District, 100034, Beijing, China
[e] Department of Cardiology, China-Japan Friendship Hospital, East Street, Sakura Garden, Chaoyang District, 100029, Beijing, China
[f] Department of Neurology, The First Affiliated Hospital of Soochow University, No. 68 Jiyang West Road, 215600, Suzhou, China
[g] Center of Gerontology and Geriatrics, National Clinical Research Center for Geriatrics, West China Hospital, Sichuan University, No.37 Guoxue Lane, Wuhou District, 610041, Chengdu, China
[h] Department of Neurobiology, Care Sciences and Society, Karolinska Institutet, Tomtebodavägen 18A Floor 10, SE-171 65 Solna, Sweden
[i] Cambridge Public Health, University of Cambridge, Trumpington Street, Cambridge, CB20SR, UK
[j] Department of Nutrition, Harvard T.H. Chan School of Public Health, 677 Huntington Avenue, Boston, MA 02115, USA
[k] Department of Mathematics and Statistics, La Trobe University, Level 20 360 Collins Street, Melbourne VIC 3000, Australia
[l] Department of Epidemiology and Health Statistics, Capital Medical University School of Public Health, No. 10 Xitoutiao Street, Fengtai District, 100069, Beijing, China
[m] Department of Cardiovascular Surgery, Beijing Anzhen Hospital, Capital Medical University, No. 2 Anzhen Road, Chaoyang District, 100029, Beijing, China
[n] Beijing Lab for Cardiovascular Precision Medicine, No. 2 Anzhen Road, Chaoyang District, 100029, Beijing, China

ARTICLE INFO

Keywords:
Heart failure
Cognitive decline
Trajectory
Cohort study

ABSTRACT

Background: The magnitude of cognitive change before and after incident heart failure (HF) is unclear. We investigated whether incident HF is associated with changes in cognitive function at the time of diagnosis and accelerated trajectory in cognitive decline in the subsequent years.

Methods: We used data from the Health and Retirement Study, a nationally representative survey of US adults aged 50 years or older. Participants underwent a cognitive assessment at baseline (wave 5, 2000), and at least 1 other time point (from wave 6 [2002] to wave 15 [2020]). The outcomes were change in global cognition, memory, and executive function. Outcomes were standardized into *Z*-scores, with higher scores indicating better cognitive performance. Linear mixed-effects models estimated changes in cognition at the time of HF (change in the intercept) and the rate of cognitive change over the years after HF (change in the slope), after adjusting for pre-HF cognitive trajectories and potential confounders.

Results: We included 12 850 adults (mean [SD] age, 66.1 [9.4] years; 61.8 % women). Over a median follow-up of 16 years (interquartile range: 8 to 20 years), 1457 participants had incident HF. The annual rate of cognitive decline before HF diagnosis among individuals with incident HF was similar to that of participants who remained HF-free throughout follow-up. However, incident HF was associated with subsequent decreases in global cognition (−0.073 SD [95 % CI −0.109 to −0.038]), memory (−0.070 SD [95 % CI −0.108 to −0.032]), and executive function (−0.054 SD [95 % CI −0.092 to −0.016]) around the time of the HF diagnosis. Moreover,

* Corresponding authors.
E-mail addresses: lihb@mail.ccmu.edu.cn (H. Li), Frank.Qian@bmc.org (F. Qian), xiewuxiang@hsc.pku.edu.cn (W. Xie), wangman_liv@126.com (M. Wang), jnjnhua@foxmail.com (J. Hua), jiaowang0823@tmu.edu.cn (J. Wang), xz391@cam.ac.uk (X. Zou), zhiyuanwu@hsph.harvard.edu (Z. Wu), X.Li2@latrobe.edu.au (X. Li), dqzheng@ccmu.edu.cn (D. Zheng), statguo@ccmu.edu.cn (X. Guo), zhanghongjia722@ccmu.edu.cn (H. Zhang).

https://doi.org/10.1016/j.tjpad.2025.100450
Received 30 August 2025; Received in revised form 25 October 2025; Accepted 27 October 2025
Available online 1 January 2026

individuals with incident HF vs those without HF demonstrated faster and long-term declines in global cognition (−0.011 SD/year [95 % CI −0.018 to −0.004]) and executive function (−0.008 SD/year [95 % CI −0.015 to −0.001]), but not in memory (−0.006 SD/year [95 % CI −0.013 to 0.001]) over the years after HF compared with pre-HF slopes.
Conclusions: Incident HF was associated with subsequent decreases in cognitive function at the time of diagnosis and accelerated cognitive decline over the following years.

1. Introduction

The global burden of heart failure (HF) has increased dramatically in the past several decades, with 56.5 million people worldwide affected in 2021 [1–3]. The rising prevalence of HF can be attributed to both increasing population prevalence in certain cardiovascular risk factors, improved treatment and survival of coronary artery disease, and an overall aging population. A meta-analysis encompassing >300 000 participants revealed that >40 % of HF patients exhibited cognitive impairment [4]. Moreover, several population-based prospective studies showed that HF was associated with cognitive decline [5–8]. However, the magnitude of subsequent changes in cognition around the time of initial HF diagnosis as well as long-term post-diagnostic cognitive trajectory after controlling for the individual's pre-HF cognitive trajectory have not been well described in the existing literature [5–8]. Few studies had examined both the subsequent change in cognitive function at the time of the HF and the rate of cognitive decline over the years after HF simultaneously [9,10]. This association, however, yielded mixed results across different cognitive domains. It is also unclear whether the association of incident HF with cognitive decline varied by sex, similar to what a previous study has shown for myocardial infarction [11].

Because cognitive function generally declines with aging, even in healthy elderly individuals, it is critical to measure pre-HF cognitive decline to accurately interpret the influence of HF on post-HF cognitive trajectories [12]. Therefore, we used longitudinal data from a large population-based cohort with multiple repeated cognitive assessment spanning 20 years to evaluate the trajectory of cognitive decline before and after incident HF. We hypothesized that HF is associated with subsequent decreases in cognitive function at the time of the event and faster cognitive decline during the years following the event.

2. Methods

2.1. Study population

The Health and Retirement Study (HRS) is a nationally representative, longitudinal cohort study of US community-dwelling adults aged ≥50 years [13]. Participants in the HRS have been interviewed biennially about their cognition, health conditions, and health behaviors until death or dropout since 1992 [14]. The HRS was approved by the University of Michigan Institutional Review Board (IRB Protocol: HUM00061128), and all participants gave written informed consent. The Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) reporting guideline for cohort studies was followed in the present study.

In the present study, we combined the 20-year HRS data from waves 5 (2000) to 15 (2020). The flowchart of participant selection is presented in **Supplemental Figure 1**. The baseline survey (wave 5) recruited 19 578 participants, of which 5595 individuals were excluded from the current study for the following reasons: age <50 years (n = 556), history of heart failure (n = 899), history of stroke (n = 1310), history of myocardial infarction (n = 888), had a confirmed diagnosis of memory-related disease (i.e., dementia) (n = 389), did not complete all the cognitive tests at baseline (n = 1165), or had missing covariate data (n = 388). We excluded those with stroke or myocardial infarction at baseline since they represent a clinically distinct group with a different pathogenesis of cognitive decline [11,15,16]. Furthermore, we also excluded participants who had no follow-up cognitive data from waves 6 to 15 (n = 1133). Therefore, the analytical sample of this study comprised 12 850 participants (4904 male and 7946 female) with complete baseline data and at least 1 reassessment of cognitive function.

2.2. Assessments of cognition

The primary outcome measure was change in global cognition. Secondary outcomes were change in memory and change in executive function. Details of cognitive assessments in HRS have been previously published [17,18]. Briefly, cognitive function was assessed at each wave with the modified Telephone Interview for Cognitive Status, which included a battery of 3 cognitive tests. First, memory function was measured by testing immediate and delayed recall of 10 unrelated words. A composite memory score (0–20 points) was calculated by summing the immediate and delayed recall scores. The memory test's validity and consistency are well-documented [19]. Second, executive function was evaluated using backward counting (0–2 points) and the Serial Sevens test (0–5 points), with a composite score (0–7 points) calculated by summing the scores from both tests. Higher scores indicate a better cognitive performance.

To allow for direct comparisons to be made across cognitive tests, Z score (i.e., mean of 0, SD of 1) for each of the 2 cognitive domains was calculated by subtracting the mean and dividing by the SD of cognition scores at wave 5. Then, we generated a composite global cognitive Z score for each participant by averaging the Z scores of the 2 cognitive domains and re-standardizing to wave 5 using the mean and SD of the global cognitive Z score at wave 5. A cognitive Z score of −1 at any given wave indicated that the score was 1 SD lower than the mean cognitive score at wave 5. This approach to processing cognitive test scores has been used and validated in previous studies [15,18].

2.3. Assessment of incident heart failure

In the HRS, the question used to assess incident HF was consistently administered, inquiring whether the survey participant had received a diagnosis of HF from a medical doctor within the past 2 years or since the time of their previous interview, in line with previous study [20,21]. The details of HF assessment have been provided in the **Supplemental Methods.** The agreement between self-report of HF and Medicare claims for HF diagnosis was 87.7 % (κ=0.34) [21]. We identified self-reported physician-diagnosed incident HF after wave 5 from HF diagnosis newly reported at waves 6 through 15. The HF diagnosis date was recorded as being between the date of the last interview and that of the interview reporting an incident HF (i.e., the midpoint between the 2 interviews).

2.4. Covariates

Covariates were factors that could influence HF and cognition and were measured at baseline. Demographics included sex (male or female), race and ethnicity (non-Hispanic White, non-Hispanic Black, non-Hispanic other, or Hispanic), education (less than high school or GED [General Educational Development], high-school graduate, some college, or college and above), and marital status (married/partnered or divorced/separate/widowed/single). Behavioral factors included smoking status (current, former, or never), alcohol consumption (yes or no) and engaging in vigorous physical activity (defined as exercising at

least 3 times per week). Biological factors included age in years, and body mass index (BMI), calculated as weight in kilograms divided by height in meters squared. Other biological factors included self-report of a physician diagnosis of diabetes, hypertension, lung disease, and cancer, with each categorized as a dichotomous variable (yes or no). In addition, depressive symptoms were defined a score of 3 or greater on the revised 8-item Center for Epidemiologic Studies Depression scale [22]. Functional limitation was defined as limitations in any of the five activities of daily living (dressing, bathing, eating, getting in/out of bed, and walking across a room) [23].

2.5. Statistical analysis

Data were summarized as n (%) for categorical variables and mean (SD) for continuous variables. Characteristics between individuals with and without incident HF were compared using a two-sample independent *t*-test or a χ^2 test as appropriate.

We used linear mixed-effects models with unstructured covariance to estimate the association of incident HF with changes in cognitive function. Random effects for intercept and slope were included to accommodate correlation of cognitive measures within participants over time and to allow participant-specific rates of cognitive change. We reported the coefficients and 95 % confidence intervals (CIs) derived using restricted maximum likelihood estimation. We analyzed each domain of cognitive function separately. Models were adjusted for age at baseline, sex, race/ethnicity, education, marital status, smoking status, alcohol consumption, participation in vigorous physical activity, body mass index, hypertension, diabetes, lung disease, cancer, depressive symptoms, and functional limitation.

To evaluate the pre-HF-diagnosis mean difference in the rate of change in cognitive function (SD/year) between participants who did and those who did not have an incident HF, Model A included incident HF (yes or no), time (years since baseline to incident HF or the end of follow-up), time × HF interaction, and covariates [15]. The time × HF interaction term was used to examine differential change associated with incident HF from baseline to incident HF or the end of follow-up (i.e., HF-free controls). To estimate the association of incident HF with cognitive function around the time of the HF diagnosis, Model B included a time-varying incident HF variable (changed from 0 to 1 on the date of incident HF), and time (years since baseline to the end of follow-up), and covariates [11,16]. The subsequent change in cognitive function around incident HF diagnosis (not observed) was estimated based on the fitted model [11,16]. To assess whether incident HF was associated with a faster rate of cognitive decline in the years following the HF event, Model C included the variables from Model B and added a time-after-HF covariate (years since an incident HF; 0 = before or at the time of HF) [11,16]. This variable indicates the rate of change in cognitive function (slope) after incident HF (i.e., post-HF-diagnosis).

Finally, cognitive trajectories before and after incident HF were described graphically. We calculated participant-specific (conditional) predicted values for each cognitive score over time for a 70-year-old White female with the average values of all covariates at baseline (high school education, married/partnered, never smoker, no alcohol use, no vigorous physical activity, BMI of 27.2 kg/m^2, without history of diabetes, hypertension, lung disease, cancer, and depressive symptoms, and no functional limitation) conditional on her experiencing or not experiencing an incident HF through the follow-up period (i.e., at year 5).

We conducted several sensitivity analyses to evaluate the robustness of our findings. First, to evaluate the modifying effects of sex on the association between incident HF and cognitive change, we used Z-test to compare the difference between the two regression coefficients from subgroup analysis [24]. Second, we restricted analyses of the subsequent cognitive change around incident HF diagnosis and post-HF-diagnosis cognitive decline to 1457 participants with an incident HF. Third, in parallel to the models fit to the entire study sample, we analyzed a subset of 9913 participants with at least 5 cognitive assessments. Fourth, to better understand the impact on each cognitive test's original scores, we replicated the analyses using the original cognitive scores. Finally, to investigate the potential impact of excluding patients with a history of myocardial infarction on our findings, we re-analyzed the data including the 667 participants with myocardial infarction at baseline, given the observed higher incidence of HF in this subgroup.

Data were analyzed from December 2024 to January 2025. Statistical analyses were performed using Stata, version 17.0 (StataCorp). Two-sided $P < 0.05$ were deemed to be statistically significant, unless otherwise specified.

3. Results

3.1. Baseline characteristics

A total of 12 850 participants (mean [SD] age, 66.1 [9.4] years) without HF, stroke, myocardial infarction, or memory-related disease at baseline were included in the analysis. Of these individuals, 77.9 % were White, 12.9 % were Black, 7.3 % were Hispanic, 38.2 % were men, and 61.8 % were women. Characteristics of included and excluded participants are summarized in **Supplemental Table 1.** During a median follow-up of 16 years (interquartile range: 8 to 20 years), 1457 (11.3 %) individuals developed incident HF. Table 1 presents baseline characteristics of study participants according to incident HF status. The participation patterns and sample size of this study population from wave 5 to 15 are shown in **Supplemental Table 2 to 4**. The median number of cognitive assessments were 8 (interquartile range: 5 to 11).

3.2. Trajectories of cognitive decline before HF diagnosis

The annual rate of cognitive decline before HF diagnosis in individuals experiencing a HF event was comparable to those in participants who remained HF-free throughout follow-up after multivariable adjustment (Table 2).

3.3. Subsequent change in cognition around HF diagnosis

As shown in the Table 3, incident HF was associated with subsequent decreases in global cognition (−0.102 SD; 95 % CI: −0.132 to −0.071 SD), memory (−0.088 SD; 95 % CI: −0.120 to −0.057 SD), and executive function (−0.076 SD; 95 % CI: −0.108 to −0.044 SD) around the HF event (Model B). Moreover, the association persisted after adjusting for the decline in cognition in the years following the HF event (Model C) (Table 3).

3.4. Long-term cognitive decline after HF diagnosis

In the years following HF diagnosis (Model C), global cognitive function declined significantly faster than it did before the HF event (decrease in slope after incident HF, −0.011 SD/year; 95 % CI: −0.018 to −0.004; $P = 0.002$), resulting in a net negative slope after incident HF (pre-HF slope: −0.044 SD/year; post-HF slope: −0.055 SD/year), after adjustment for follow-up time, the subsequent decrease in cognition around the time of the HF diagnosis, and baseline covariates (Table 3). Likewise, the rates of declines in executive function (−0.008 SD/year; 95 % CI: −0.015 to −0.001) were accelerated after HF diagnosis. The post-HF-diagnosis slope of memory also declined faster than its pre-HF diagnosis slope, although this difference did not achieve statistical significance. Fig. 1 presents the estimated trajectories of cognitive scores for an exemplar participant based on estimated parameters of Model C.

3.5. Sensitivity analyses

Stratified analysis by sex was shown in Table 2 (Model A), **Supplemental Table 5** (Model B), and **Supplemental Table 6** (Model C).

Table 1
Baseline Characteristics Between Participants Who Did and Did Not Have an Incident Heart Failure During Follow-up: Health and Retirement Study (HRS), 2000–2020.

Characteristic	Total (n = 12,850)	Heart Failure, No. (%)		P value
		No Incident (n = 11,393)	Incident (n = 1457)	
Demographics				
Age, mean (SD), y	66.1 (9.4)	65.8 (9.4)	68.1 (9.2)	<0.001
Sex				0.230
Male	4904 (38.2)	4327 (38.0)	577 (39.6)	
Female	7946 (61.8)	7066 (62.0)	880 (60.4)	
Race/ethnicity				0.210
Non-Hispanic White	10,012 (77.9)	8874 (77.9)	1138 (78.1)	
Non-Hispanic Black	1658 (12.9)	1454 (12.8)	204 (14.0)	
Non-Hispanic other*	237 (1.8)	214 (1.9)	23 (1.6)	
Hispanic	943 (7.3)	851 (7.5)	92 (6.3)	
Education				<0.001
Less than high school or GED	3418 (26.6)	2965 (26.0)	453 (31.1)	
High-school graduate	4222 (32.9)	3737 (32.8)	485 (33.3)	
Some college	2621 (20.4)	2331 (20.5)	290 (19.9)	
College and above	2589 (20.1)	2360 (20.7)	229 (15.7)	
Married/partnered	8737 (68.0)	7789 (68.4)	948 (65.1)	0.011
Health covariates				
Smoking status				<0.001
Current	5511 (42.9)	4951 (43.5)	560 (38.4)	
Former	5407 (42.1)	4737 (41.6)	670 (46.0)	
Never	1932 (15.0)	1705 (15.0)	227 (15.6)	
Alcohol consumption	6361 (49.5)	5746 (50.4)	615 (42.2)	<0.001
Vigorous physical activity	6079 (47.3)	5471 (48.0)	608 (41.7)	<0.001
BMI, mean (SD), Kg/m^2	27.2 (5.2)	27.0 (5.1)	28.6 (6.0)	<0.001
Diabetes	1501 (11.7)	1212 (10.6)	289 (19.8)	<0.001
Hypertension	5384 (41.9)	4583 (40.2)	801 (55.0)	<0.001
Lung disease	659 (5.1)	545 (4.8)	114 (7.8)	<0.001
Cancer	1277 (9.9)	1105 (9.7)	172 (11.8)	0.011
Depressive symptoms	2619 (20.4)	2253 (19.8)	366 (25.1)	<0.001
Functional limitation	1397 (10.9)	1160 (10.2)	237 (16.3)	<0.001
Baseline cognitive scores				
Global cognition, mean (SD)	15.8 (4.4)	15.9 (4.4)	15.3 (4.4)	<0.001
Memory, mean (SD)	10.3 (3.6)	10.3 (3.6)	9.9 (3.5)	<0.001
Executive function, mean (SD)	5.5 (1.8)	5.5 (1.8)	5.4 (1.8)	0.019

Abbreviations: BMI, body mass index (calculated as weight in kilograms divided by height in meters squared); GED, general educational development.
* Other race is a composite of Asian, Pacific Islander, American Indian, and Alaska Native.

There was no difference by sex in the association between incident HF and the subsequent decrease in cognition around the time of the HF diagnosis or post-HF slope change in cognition (P for interaction >0.05). When we restricted the analyses to the 1457 participants with incident HF, individuals with HF experienced an subsequent decrease in global cognition after the index HF event (−0.084 SD; 95 % CI: −0.122 to −0.046), and demonstrated faster declines over the years after the diagnosis of HF (−0.013 SD/year; 95 % CI: −0.021 to −0.005) (Fig. 2 **and Supplemental Table 7)**. Results with a subset of participants who completed at least five cognitive assessments (n = 9913) were similar **(Supplemental Table 8 and 9)**. Results using the original cognitive scores were directionally similar to those based on Z scores **(Supplemental Table 10 and 11)**. In the analyses that included patients with history of myocardial infarction (n = 13 517), the results were consistent with our main analyses and the post-HF-diagnosis annual decline in memory became significant (−0.008 SD/year; 95 % CI: −0.015 to −0.001), after adjusting for baseline covariates and history of myocardial infarction **(Supplemental Table 12 and 13)**.

Table 2
Pre-HF-Diagnosis Mean Difference in Rate of Change in Cognitive Z Score (SD/Year) Between Participants Who Did and Did Not Have an Incident HF (Model A).

	Mean Difference (95 % CI) in Rate of Change*	P value	P value for Interaction#
Global cognitive Z score			
Male	0.004 (−0.003 to 0.011)	0.245	0.273
Female	−0.001 (−0.007 to 0.005)	0.733	Ref.
Total	0.001 (−0.004 to 0.006)	0.650	
Memory Z score			
Male	−0.0001 (−0.007 to 0.007)	0.972	0.699
Female	−0.002 (−0.008 to 0.004)	0.529	Ref.
Total	−0.001 (−0.006 to 0.004)	0.642	
Executive function Z score			
Male	0.005 (−0.002 to 0.012)	0.161	0.161
Female	−0.002 (−0.007 to 0.004)	0.601	Ref.
Total	0.001 (−0.004 to 0.005)	0.675	

Abbreviations: HF, heart failure.
* Using participants who did not have an incident HF as the reference group, after adjusting for baseline age, sex (when analyzing total data), race/ethnicity, education, marital status, smoking status, alcohol consumption, vigorous physical activity, body mass index, hypertension, diabetes, lung disease, cancer, depression symptoms, and functional limitation.
# P value for interaction was calculated using Z test.

3.6. Nonresponse analyses

Of the 13 983 participants who had complete data at baseline, 1133 (8.1 %) were excluded from this study because they had no follow-up cognitive data. These 1133 participants had poorer cognitive scores than those included in this study. Moreover, participants excluded from the study exhibited a higher prevalence of comorbidities, such as diabetes, hypertension, lung disease, depressive symptoms, and functional limitations **(Supplemental Table 14)**.

4. Discussion

In this large, nationally representative cohort of individuals ≥50 years from the US, incident HF was associated with subsequent decreases in global cognition, memory, and executive function cognition at the time of the event, after accounting for individuals' cognitive trajectory before and after the event. Moreover, we found that incident HF was associated with accelerated and persistent declines in global cognition and executive function over the years after HF diagnosis, independent of established sociodemographic, behavioral, and vascular

Table 3
Adjusted Changes in Cognitive Function After Incident HF Diagnosis Among All 12 850 Individuals.

Variable	Model B Coefficient (95 % CI)	*P* value	Model C Coefficient (95 % CI)	*P* value
Global cognitive Z score				
Baseline slope without incident HF, per y	−0.044 (−0.045 to −0.043)	<0.001	−0.044 (−0.045 to −0.043)	<0.001
Subsequent change in cognitive score around incident HF diagnosis	−0.102 (−0.132 to −0.071)	<0.001	−0.073 (−0.109 to −0.038)	<0.001
Change in cognitive slope after incident HF, per y	Not included		−0.011 (−0.018 to −0.004)	0.002
Age, per y	−0.034 (−0.036 to −0.033)	<0.001	−0.034 (−0.036 to −0.033)	<0.001
Intercept	1.615 (1.464 to 1.765)	<0.001	1.612 (1.462 to 1.763)	<0.001
Log restricted-likelihood	−100,728.16		−100,728.18	
Memory Z score				
Baseline slope without incident HF, per y	−0.045 (−0.046 to −0.044)	<0.001	−0.045 (−0.046 to −0.044)	<0.001
Subsequent change in cognitive score around incident HF diagnosis	−0.088 (−0.120 to −0.057)	<0.001	−0.070 (−0.108 to −0.032)	<0.001
Change in cognitive slope after incident HF, per y	Not included		−0.006 (−0.013 to 0.001)	0.097
Age, per y	−0.045 (−0.046 to −0.044)	<0.001	−0.045 (−0.046 to −0.044)	<0.001
Intercept	2.124 (1.988 to 2.259)	<0.001	2.123 (1.987 to 2.258)	<0.001
Log restricted-likelihood	−107,059.99		−107,063.30	
Executive function Z score				
Baseline slope without incident HF, per y	−0.023 (−0.024 to −0.022)	<0.001	−0.023 (−0.024 to −0.022)	<0.001
Subsequent change in cognitive score around incident HF diagnosis	−0.076 (−0.108 to −0.044)	<0.001	−0.054 (−0.092 to −0.016)	0.005
Change in cognitive slope after incident HF, per y	Not included		−0.008 (−0.015 to −0.001)	0.033
Age, per y	−0.012 (−0.014 to −0.011)	<0.001	−0.012 (−0.014 to −0.011)	<0.001
Intercept	0.646 (0.486 to 0.805)	<0.001	0.644 (0.485 to 0.804)	<0.001
Log restricted-likelihood	−100,728.16		−107,886.34	

Abbreviations: HF, heart failure.
For linear mixed-effects models, model B included a time-varying incident HF variable to estimate the association of incident HF with decline in cognitive function (intercept) at the time of the event (value changed from 0 to 1 at the time of incident HF). Model C included covariates in model B and added a time-after-HF covariate to estimate the association of incident HF with decline in cognitive function (slope) over the years following the event. Covariates in model B included a random intercept, calendar time, time-varying incident HF, and baseline values of age, sex, race/ethnicity, education, marital status, smoking status, alcohol consumption, vigorous physical activity, body mass index, hypertension, diabetes, lung disease, cancer, depression symptoms, and functional limitation.

risk factors.

The impact of incident HF on cognitive trajectory before the event has not been previously investigated. Compared with individuals who remained free of HF, we did not observe a significant change in the slope of pre-HF-diagnosis cognitive decline in participants who later had an incident HF, suggesting potential adaptive or compensatory mechanisms for cerebral and/or vascular protection prior to apparent clinical manifestations of HF, and in turn not manifesting with a degree of cognitive impairment that can be detected using the methods applied in the current study. Another potential explanation is that the cognitive assessments employed in this study lacked the sensitivity required to detect the subtle cognitive deficits present prior to the HF event. More research is needed on this, particularly leveraging imaging approaches to assess for subclinical cerebral ischemia and injury.

The present study added to previous research by describing the subsequent change in cognitive function around the time of the HF diagnosis. We observed significant subsequent decreases in global cognition, memory, and executive function among participants with HF diagnosis. This suggests that a new HF diagnosis and the associated hemodynamic changes and vascular injury may be sufficient to cause cognitive changes in the short-term [25]. Findings from the ASPREE (ASPirin in Reducing Events in the Elderly) study also suggested that hospitalization for HF was associated with an subsequent decrease in processing speed but not global cognition [9]. Differences in our results may be attributable to discrepancies in the ascertainment and clinical severity of HF, the cognitive measures used, and length of follow-up.

We quantified the magnitude of post-HF cognitive decline after controlling for an individual's pre-HF cognitive decline. Among participants with an incident HF diagnosis, decline in global cognition was faster compared with before HF by 0.013 SD per year, and that global cognition declined by 0.084 SD after HF, and this was consistent across all cognitive measures. While a change of −0.013 SD over 1 year appears small, the cumulative effect was more substantial. The combined subsequent decreases and long-term effect of HF on cognition was 0.097 SD after 1-year post-HF, equivalent to 2.2 years of cognitive aging in individuals without HF. Prior studies indicated that a decline of 0.5 SD or more from baseline has been defined as clinically meaningful decline [26,27]. Therefore, for the significantly accelerated cognitive decline observed in the years following HF, the global cognition in approximately 14 years would correspond to a decline of 0.5 SD. Taken together, our findings suggest that careful monitoring for cognitive function in participants with an incident HF diagnosis is warranted in the years following their diagnosis, particularly given the increasing incidence and prevalence of heart failure.

Following a HF diagnosis, we observed a significant acceleration in executive function decline, in contrast to memory, which has been observed in prior studies [10,28]. This difference is likely because memory, which relies on the hippocampus, is more resilient to HF-related pathophysiological changes such as cerebral hypoperfusion, oxidative stress, and inflammation. Executive function, on the other hand, is dependent on the prefrontal cortex, an area with high metabolic needs and a particular susceptibility to hypoperfusion and cerebral microemboli [29]. It is possible that interventions for HF, such as medication adjustments and lifestyle modifications, may help protect memory function. Furthermore, the decline in memory might necessitate a longer exposure to the underlying HF disease processes than what was captured in our study's follow-up period. It is conceivable that our study may have been underpowered to detect changes in the memory domain alone, and larger prospective studies with longer follow-up are warranted. Additionally, the assessment tools used for memory may be less adept at detecting subtle, early changes compared to those used for executive function.

We also found that male, on average, had less of a decline in global cognition and executive function than female over the years after HF, although this difference was not statistically significant. Previous studies have shown significant disparities in the incidence of HF and cognitive

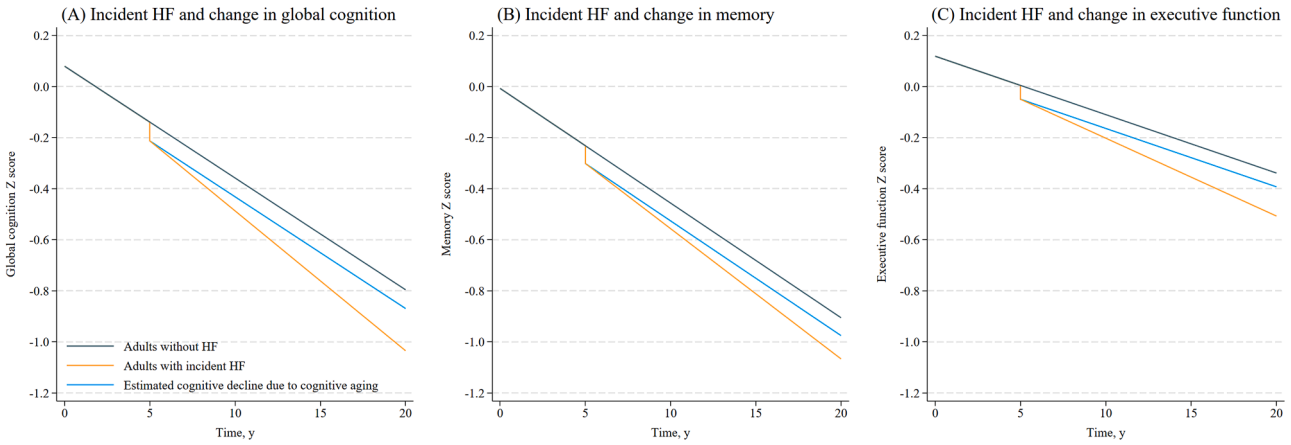

Fig. 1. Estimated values of cognition for the association between incident heart failure (HF) and change in global cognition, memory, and executive function around incident HF diagnosis and in the years following the HF event among 12 850 participants.
Participant-specific (conditional) predicted values of cognition were calculated for a 70-year-old White female with the average values of all covariates at baseline (high school education, married/partnered, never smoker, no alcohol use, no vigorous physical activity, BMI of 27.2 kg/m^2, without history of diabetes, hypertension, lung disease, cancer, and depressive symptoms, and no functional limitation).

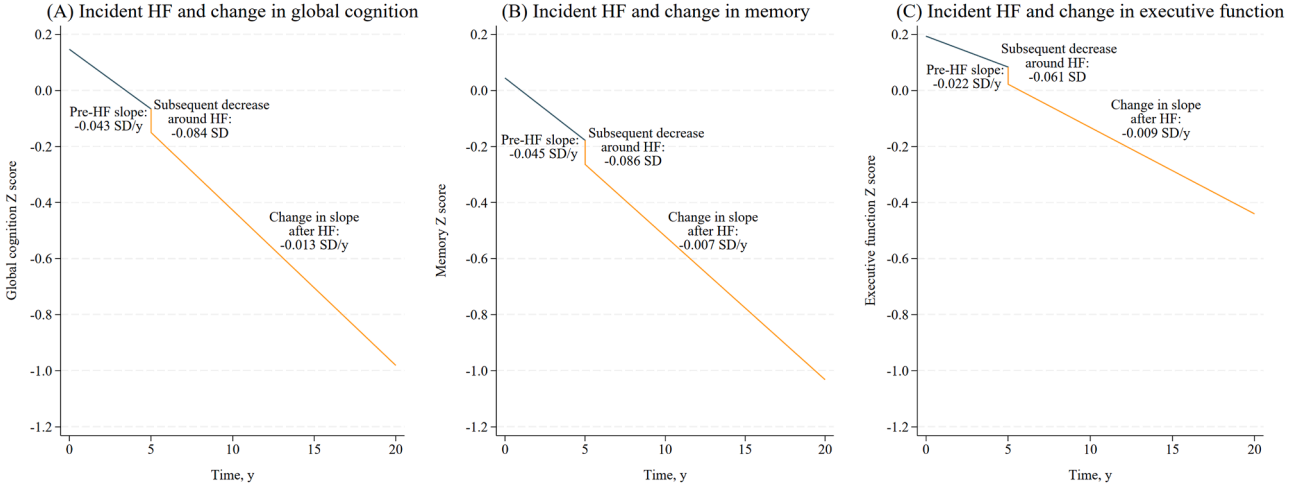

Fig. 2. Estimated values of cognition for the association between incident heart failure (HF) and change in global cognition, memory, and executive function around incident HF diagnosis and in the years following the HF event among in 1457 participants with incident HF.
Participant-specific (conditional) predicted values of cognition were calculated for a 70-year-old White female HF patient with the average values of all covariates at baseline (high school education, married/partnered, never smoker, no alcohol use, no vigorous physical activity, BMI of 27.2 kg/m^2, without history of diabetes, hypertension, lung disease, cancer, and depressive symptoms, and no functional limitation).

decline between male and female, indicating that sex likely plays a crucial role in these health outcomes [30,31]. Therefore, managing and treating HF-associated cognitive decline in older adults requires an individualized strategy, though this requires further research.

Incident HF may cause long-term cognitive decline through several mechanisms. First, insufficient cardiac output associated with HF could cause substantial reduced cerebral blood flow and cerebrovascular reactivity, leading to cerebral hypoperfusion, cerebral grey matter atrophy, and white matter lesions, culminating in neurodegeneration [25,32,33]. Other mechanisms that may be relevant include a pro-inflammatory state, hemodynamic swings, and microvascular dysfunction [34–36]. Lastly, another important mechanism linking HF to cognitive decline involves the shared vascular risk factors [37–39]. Notably, our results remained robust when we controlled for several vascular risk factors in the statistical analysis.

The clinical relevance for our findings with respect to dementia prevention are three-fold. Firstly, our findings corroborate and expand upon prior observations of a high prevalence of cognitive impairment among individuals with HF. By highlighting the time course over which cognitive decline occurs in this population, we have identified crucial windows during which increased clinical surveillance and monitoring should be instituted, should our findings be replicated in future studies. Secondly, the association between HF and subsequent decreases in cognitive function and long-term cognitive decline suggests important pathways, such as systemic inflammation, oxidative stress, endothelial and microvascular dysfunction, subclinical cerebral emboli, and global cerebral hypoperfusion [40]. Thirdly, our identification of accelerated rate of cognitive decline after HF diagnosis is of clinical importance. While the mechanisms for this finding remains to be elucidated, it is conceivable that given the high comorbidity burden among individuals with HF, subsequent cognitive decline may trigger a vicious cascade of difficulty with adherence to medical therapy, increased disability and frailty, and worsening social isolation that further accelerates cognitive decline [41].

Our study has several strengths. First, analyses were based on a nationally representative community-based cohort, with a large sample size and long follow-up, providing greater statistical power to estimate the association between incident HF and cognitive decline. Second,

using repeated longitudinal cognitive assessments over time, we accounted for pre-HF cognitive decline and subsequent cognitive changes after HF to disentangle the association between HF and long-term cognitive decline.

This study has several limitations. First, the use of self-reported physician-diagnosed incident HF might lead to a misclassification of HF cases and could have biased our findings to a minor extent. Nevertheless, previous studies have validated self-reported measures as accurate diagnostic tools for health conditions, particularly cardiovascular diseases, demonstrating reliability across diverse populations [15,20,42–45]. Although the agreement between self-reported HF and claims-based diagnosis was 87.7 % in HRS, self-reported HF is highly specific (99.2 %) but not as sensitive (25.2 %) as clinically validated HF diagnosis [21]. The low sensitivity (potential undercounting of true HF cases) is more likely to bias our findings towards the null, as individuals with undiagnosed or milder heart failure in the comparator group is also expected experience declines in cognitive function. Second, we were unable to conduct stratified analysis by HF severity and type because these data were unavailable. Third, there is a possibility of selection bias, as 1133 participants (8.1 %) with complete baseline data were excluded from the study because they lacked follow-up cognitive data. We also performed nonresponse analyses, which showed that our study sample was healthier than the initial sample of HRS participants. Selective attrition may lead to underestimation of cognitive decline because participants with worse cognition at baseline or after incident HF may have increased mortality and be more likely to drop out of the study. Fourth, high rates of attrition—a common occurrence in longitudinal studies involving older adults—led to the dropout of participants who were older, had more severe health issues, and exhibited lower cognitive function. In addition, we used linear mixed-effects models that are robust to handle missing data, using all available cognitive measurements before and after HF diagnosis and performed additional sensitivity analyses among individuals with $\geq$5 cognitive assessments that demonstrated consistent results. Fifth, although several key confounders were adjusted for and robust results were demonstrated across several sensitivity analyses, unmeasured covariates might still have led to confounding bias, including *APOE* status, atrial fibrillation, left ventricular ejection fraction, and serum biomarkers (i.e., NT-proBNP). Lastly, the HRS mainly included White participants, limiting the generalizability of our findings to other population.

5. Conclusion

In this cohort study, incident HF was associated with subsequent decreases in global cognition, memory, and executive function at the time of the event. Furthermore, incident HF demonstrated an accelerated and persistent cognitive decline in the years following the HF event. The findings suggest the importance of surveilling cognitive function in older adults with HF.

Ethics approval and consent to participant

The HRS was approved by the University of Michigan Institutional Review Board (IRB Protocol: HUM00061128), and all participants gave written informed consent.

Declaration of generative AI and AI-assisted technologies in the writing process

No generative AI or AI-assisted technologies were used in the preparation of this manuscript.

Funding

This work was supported by funding from the National Natural Science Foundation of China (72474142, 82103942), the Capital's Funds for Health Improvement and Research (CFH2024-2G-2036), the Clinical Research Incubation Project, Beijing Chao-Yang Hospital, Capital Medical University (CYFH202310), the Beijing Nova Program (20250484858), and the Talent development plan for the future in Medical-Engineering Integration by BRA-CDCHE and ZTA (MBRC0012025021). Frank Qian is supported by the T32HL125232 Multidisciplinary Training Program in Cardiovascular Epidemiology.

Data sharing

The HRS is publicly available to access and download at https://hrs.isr.umich.edu/data-products.

CRediT authorship contribution statement

Haibin Li: Writing – review & editing, Writing – original draft, Visualization, Methodology, Investigation, Funding acquisition, Formal analysis, Conceptualization. **Frank Qian:** Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization. **Wuxiang Xie:** Writing – review & editing, Supervision, Methodology, Conceptualization. **Man Wang:** Writing – review & editing. **Jianian Hua:** Writing – review & editing, Methodology. **Jiao Wang:** Writing – review & editing, Methodology, Conceptualization. **Xinye Zou:** Writing – review & editing. **Zhiyuan Wu:** Writing – review & editing, Investigation. **Xia Li:** Writing – review & editing, Supervision, Software, Methodology. **Deqiang Zheng:** Writing – review & editing, Supervision, Methodology. **Xiuhua Guo:** Writing – review & editing, Supervision. **Hongjia Zhang:** Writing – review & editing, Supervision, Investigation, Conceptualization.

Declaration of competing interests

None.

Acknowledgments

We appreciate efforts made by the original data creators, depositors, copyright holders, and funders of the data collection and their contributions for access of data from the HRS (waves 5–15). The HRS was funded by the NIA (U01AG009740) and the Social Security Administration, and was performed at the Institute for Social Research, University of Michigan.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.tjpad.2025.100450.

References

[1] Savarese G, Becher PM, Lund LH, Seferovic P, Rosano GMC, Coats AJS. Global burden of heart failure: a comprehensive and updated review of epidemiology. Cardiovasc. Res. 2022;118(17):3272–87.
[2] Khan MS, Shahid I, Bennis A, Rakisheva A, Metra M, Butler J. Global epidemiology of heart failure. Nat Rev Cardiol 2024;21(10):717–34.
[3] Roth GA, Dorsey H, Decleene N, Razo C, Stark B, Johnson C, TGBoDS Collaborators. The global burden of heart failure: a systematic analysis for the Global Burden of Disease Study 2021. Eur. Heart J. 2023;44(Supplement_2).
[4] Yap NLX, Kor Q, Teo YN, Teo YH, Syn NL, Evangelista LKM, Tan BY, Lin W, Yeo LL, Kong WK, Chong YF, Wong RC, Poh KK, Yeo TC, Sharma VK, Chai P, Chan MY, Goh FQ, Sia CH. Prevalence and incidence of cognitive impairment and dementia in heart failure - a systematic review, meta-analysis and meta-regression. Hellenic J Cardiol 2022;67:48–58.
[5] Bressler J, Knopman DS, Sharrett AR, Gottesman RF, Penman A, Chang PP, Rosamond WD, Boerwinkle E, Mosley TH. Incident heart failure and cognitive decline: the Atherosclerosis Risk in Communities Study. J Card Fail 2017;23(1):47–55.
[6] Hammond CA, Blades NJ, Chaudhry SI, Dodson JA, Longstreth Jr WT, Heckbert SR, Psaty BM, Arnold AM, Dublin S, Sitlani CM, Gardin JM, Thielke SM, Nanna MG, Gottesman RF, Newman AB, Thacker EL. Long-term cognitive decline after newly

diagnosed heart failure: longitudinal analysis in the CHS (Cardiovascular Health Study). Circ Heart Fail 2018;11(3):e004476.
[7] Sterling MR, Ringel JB, Safford MM, Goyal P, Khodneva Y, McClure LA, Durant RW, Jacob AE, Levitan EB. Trajectory of cognitive decline after incident heart failure hospitalization: findings from the REGARDS Study. J Am Heart Assoc 2024;13(17):e032986.
[8] Jung M, Apostolova LG, Gao S, Burney HN, Lai D, Saykin AJ, Pressler SJ. Association of heart failure with cognitive decline and development of mild cognitive impairment and dementia. J Cardiovasc Nurs 2024;39(3):E80–5.
[9] Vishwanath S, Hopper I, Wolfe R, Polekhina G, Reid CM, Tonkin AM, Murray AM, Shah RC, Storey E, Woods RL, McNeil J, Orchard SG, Nelson MR, Steves CJ, Ryan J. Cognitive trajectories and incident dementia after a cardiovascular event in older adults. Alzheimers Dement 2023;19(8):3670–8.
[10] Shore S, Li H, Zhang M, Whitney RT, Gross AL, Bhatt AS, Nallamothu BK, Giordani B, Briceno EM, Sussman JB, Gutierrez J, Yaffe K, Griswold ME, Johansen MC, Lopez OL, Gottesman RF, Sidney S, Heckbert SR, Rundek T, Hughes TM, Longstreth Jr WT, Levine DA. Trajectory of cognitive function after incident heart failure. Circ Heart Fail 2025;18(3):e011837.
[11] Johansen MC, Ye W, Gross A, Gottesman RF, Han D, Whitney R, Briceño EM, Giordani BJ, Shore S, Elkind MSV, Manly JJ, Sacco RL, Fohner A, Griswold M, Psaty BM, Sidney S, Sussman J, Yaffe K, Moran AE, Heckbert S, Hughes TM, Galecki A, Levine DA. Association between acute myocardial infarction and cognition. JAMA Neurol 2023;80(7):723–31.
[12] Nyberg L, Lovden M, Riklund K, Lindenberger U, Backman L. Memory aging and brain maintenance. Trends Cogn Sci 2012;16(5):292–305.
[13] Sonnega A, Faul JD, Ofstedal MB, Langa KM, Phillips JW, Weir DR. Cohort profile: the Health and Retirement Study (HRS). Int J Epidemiol 2014;43(2):576–85.
[14] Fisher GG, Ryan LH. Overview of the Health and Retirement Study and introduction to the special issue. Work, Aging and Retirement. 2017;4(1):1–9.
[15] Xie W, Zheng F, Yan L, Zhong B. Cognitive decline before and after incident coronary events. J Am Coll Cardiol 2019;73(24):3041–50.
[16] Levine DA, Galecki AT, Langa KM, Unverzagt FW, Kabeto MU, Giordani B, Wadley VG. Trajectory of cognitive decline after incident stroke. JAMA 2015;314(1):41–51.
[17] Crimmins EM, Kim JK, Langa KM, Weir DR. Assessment of cognition using surveys and neuropsychological assessment: the Health and Retirement Study and the Aging, Demographics, and Memory Study. J Gerontol B Psychol Sci Soc Sci 2011;(Suppl 1):i162–71. 66 Suppl 1.
[18] Li C, Zhu Y, Ma Y, Hua R, Zhong B, Xie W. Association of cumulative blood pressure with cognitive decline, dementia, and mortality. J Am Coll Cardiol 2022;79(14):1321–35.
[19] Cheke LG, Clayton NS. Do different tests of episodic memory produce consistent results in human adults? Learn Mem 2013;20(9):491–8.
[20] Mahmood A, Ray M, Dobalian A, Ward KD. Ahn S. Insomnia symptoms and incident heart failure: a population-based cohort study. Eur Heart J 2021;42(40):4169–76.
[21] Gure TR, McCammon RJ, Cigolle CT, Koelling TM, Blaum CS, Langa KM. Predictors of self-report of heart failure in a population-based survey of older adults. Circ Cardiovasc Qual Outcomes 2012;5(3):396–402.
[22] Andresen EM, Malmgren JA, Carter WB, Patrick DL. Screening for depression in well older adults: evaluation of a short form of the CES-D (Center for Epidemiologic Studies Depression Scale). Am J Prev Med 1994;10(2):77–84.
[23] Katz S. Assessing self-maintenance: activities of daily living, mobility, and instrumental activities of daily living. J Am Geriatr Soc 1983;31(12):721–7.
[24] Altman DG, Bland JM. Interaction revisited: the difference between two estimates. BMJ 2003;326(7382):219.
[25] Havakuk O, King KS, Grazette L, Yoon AJ, Fong M, Bregman N, Elkayam U, Kloner RA. Heart failure-induced brain injury. J Am Coll Cardiol 2017;69(12):1609–16.
[26] Wolinsky FD, Unverzagt FW, Smith DM, Jones R, Stoddard A, Tennstedt SL. The ACTIVE cognitive training trial and health-related quality of life: protection that lasts for 5 years. J Gerontol A Biol Sci Med Sci 2006;61(12):1324–9.
[27] Norman GR, Sloan JA, Wyrwich KW. Interpretation of changes in health-related quality of life: the remarkable universality of half a standard deviation. Med Care 2003;41(5):582–92.
[28] Connors EJ, Hauson AO, Barlet BD, Sarkissians S, Stelmach NP, Walker AD, Nemanim NM, Greenwood KL, Chesher NJ, Wollman SC, Allen KE, Hall MG. Flora-Tostado C. Neuropsychological assessment and screening in heart failure: a meta-analysis and systematic review. Neuropsychol Rev 2021;31(2):312–30.
[29] Moroni F, Ammirati E, Hainsworth AH, Camici PG. Association of white matter hyperintensities and cardiovascular disease: the importance of microcirculatory disease. Circ Cardiovasc Imaging 2020;13(8):e010460.
[30] Lam CSP, Arnott C, Beale AL, Chandramouli C, Hilfiker-Kleiner D, Kaye DM, Ky B, Santema BT, Sliwa K, Voors AA. Sex differences in heart failure. Eur Heart J 2019;40(47):3859–68.
[31] Levine DA, Gross AL, Briceño EM, Tilton N, Giordani BJ, Sussman JB, Hayward RA, Burke JF, Hingtgen S, Elkind MSV, Manly JJ, Gottesman RF, Gaskin DJ, Sidney S, Sacco RL, Tom SE, Wright CB, Yaffe K, Galecki AT. Sex differences in cognitive decline among US adults. JAMA Netw Open 2021;4(2).
[32] Villringer A, Laufs U. Heart failure, cognition, and brain damage. Eur Heart J 2021;42(16):1579–81.
[33] Goh FQ, Kong WKF, Wong RCC, Chong YF, Chew NWS, Yeo TC, Sharma VK, Poh KK, Sia CH. Cognitive impairment in heart failure-a review. Biology (Basel) 2022;11(2).
[34] Redwine LS, Hong S, Kohn J, Martinez C, Hurwitz BE, Pung MA, Wilson K, Pruitt C, Greenberg BH, Mills PJ. Systemic inflammation and cognitive decrements in patients with stage B heart failure. Psychosom Med 2022;84(2):133–40.
[35] de la Torre JC. Hemodynamic instability in heart failure intensifies age-dependent cognitive decline. J Alzheimers Dis 2020;76(1):63–84.
[36] Hillier E, Covone J, Fischer K, Chen HY, Hafyane T, Friedrich MG. Microvascular dysfunction as a possible link between heart failure and cognitive dysfunction. Circulation-Heart Failure 2023;16(11).
[37] Uijl A, Koudstaal S, Vaartjes I, Boer JMA, Verschuren WMM, van der Schouw YT, Asselbergs FW, Hoes AW, Sluijs I. Risk for heart failure: the opportunity for prevention with the American Heart Association's life's simple 7. JACC Heart Fail 2019;7(8):637–47.
[38] Samieri C, Perier MC, Gaye B, Proust-Lima C, Helmer C, Dartigues JF, Berr C, Tzourio C, Empana JP. Association of cardiovascular health level in older age with cognitive decline and incident dementia. JAMA 2018;320(7):657–64.
[39] Zlokovic BV, Gottesman RF, Bernstein KE, Seshadri S, McKee A, Snyder H, Greenberg SM, Yaffe K, Schaffer CB, Yuan C, Hughes TM, Daemen MJ, Williamson JD, González HM, Schneider J, Wellington CL, Katusic ZS, Stoeckel L, Koenig JI, Corriveau RA, Fine L, Galis ZS, Reis J, Wright JD, Chen J. Vascular contributions to cognitive impairment and dementia (VCID): a report from the 2018 National Heart, Lung, and Blood Institute and National Institute of Neurological Disorders and Stroke Workshop. Alzheimers Dement 2020;16(12):1714–33.
[40] Li M, Zhang J, Hu Y, Zhang Y. Cognitive impairment in patients with heart failure: physiopathology, clinical implications, and therapeutic considerations. J Cardiol 2025;85(6):465–72.
[41] Goyal P, Didomenico RJ, Pressler SJ, Ibeh C, White-Williams C, Allen LA, Gorodeski EZ, Members HSSC. Cognitive impairment in heart failure: a Heart Failure Society of America Scientific Statement. J Card Fail 2024;30(3):488–504.
[42] Dhingra R, Gaziano JM, Djousse L. Chronic kidney disease and the risk of heart failure in men. Circ Heart Fail 2011;4(2):138–44.
[43] Heliovaara M, Aromaa A, Klaukka T, Knekt P, Joukamaa M, Impivaara O. Reliability and validity of interview data on chronic diseases. The Mini-Finland Health Survey. J Clin Epidemiol 1993;46(2):181–91.
[44] Glymour MM, Avendano M. Can self-reported strokes be used to study stroke incidence and risk factors? Evidence from the Health and Retirement Study. Stroke 2009;40(3):873–9.
[45] Camplain R, Kucharska-Newton A, Loehr L, Keyserling TC, Layton JB, Wruck L, Folsom AR, Bertoni AG, Heiss G. Accuracy of self-reported heart failure. The Atherosclerosis Risk in Communities (ARIC) Study. J Card Fail 2017;23(11):802–8.