SUPPLEMENTAL MATERIAL

Data S1.

Supplemental Methods

Assessment of covariates

Education years, BMI, smoking (never/ex/current), and alcohol use (none/>1 drink per week) were recorded during the initial interview. Total nighttime sleep duration was estimated from activity recordings using the same algorithm for daytime napping estimation except that sleep estimation was rendered during 9 PM and 7 AM. Sleep fragmentation was estimated using an actigraphy-based method that calculates the probability of having an arousal (e.g., a non-zero activity count) after a long (\sim 5 min) period of rest (i.e., sleep).³³ Frailty is a composite indicator based on five dichotomized frailty components (BMI, fatigue, gait, grip strength, and physical activity). Participants were categorized as having frailty if three or more frail components presented. ³⁴ The Rosow-Breslau scale was used to assess mobility disability. Participants were categorized as having mobility disability if they reported needing help for at least one item.³⁵ Motor function was assessed by a composite score covering 10 motor performance tests. Medications (taken/not taken) were inspected and coded using the Medi-Span system (Medi-Span, Inc.)³⁶ Presence/absence of comorbidities at baseline was determined at interview. We coded for presence of urinary conditions (urinary incontinence/spasms, benign prostatic hypertrophy, diuretic use, or associated medications) that may confound results related to nighttime awakening, fragmented rest and increased likelihood for daytime napping. Participants were considered to have diabetes, hypertension, or thyroid disease if they were taking medications or

endorsed a diagnosis on interview. Depressive symptoms were assessed with a 10-item version of the Center for Epidemiologic Studies-Depression Scale and results were square root transformed because of right skewness. Cognition was assessed using a composite score representing global cognition constructed from z-scores of 19 cognitive tests. 37

We also calculated the number of nighttime awakenings based on the sleep episodes estimated from actigraphy. Interestingly, the number of nighttime awakenings was negatively correlated, instead of being positively correlated, with nap duration and nap frequency (Fig. S1), and it was not significantly associated incident HF (results not reported). The calculated number of nighttime awakenings was positively correlated with the sleep fragmentation index (Fig. S1). Considering that the sleep fragmentation index was priorly linked to incident HF in the same cohort,¹⁹ we included the sleep fragmentation index instead of the number of awakenings in the adjusted models (models B).

Supplemental Results

Consistent results obtained using incident heart failure (HF) jointly determined by self-report and medication

To improve our predictive models, we also further parsed through the associations using a combination of any two HF-related medications and self-report HF. Using this criterion to define incident HF, the initial models revealed positive associations between longer and more frequent daytime napping and risk of HF (Tables S1 and S2). Specifically, for each 1 SD increase in the square root transformed nap duration, the hazard ratio (HR) was 1.42 [95% confidence interval (CI): 1.13-1.83; *p* = 0.004]. The HR was 1.48 (95% CI: 1.15-1.90; *p* =

0.003) for each 1 SD increase in the square root transformed nap frequency. In both models, age was not associated with incident HF which might be due to a power issue, and this makes it irrelevant to directly compare the effects of daytime napping duration or frequency with that of age.

The results were consistent after separately adjusted for sleep, comorbidities, and cardiovascular diseases/risk factors. With fully adjusted models, the associations of nap duration and nap frequency with incident HF were still significant, with 1-SD increase in the square root transformed napping duration and 1-SD increase in the square root transformed napping frequency being corresponding to an HR of 1.68 (95% CI: 1.17-2.39; *p* = 0.005) and of 1.66 (95% CI: 1.16-2.35; *p* = 0.006) in the risk of developing incident HF.

Sensitivity analyses within cognitively intact participants showed consistent results

Sensitivity analyses were done by including participants who were cognitively intact at baseline (N = 837; female: 655) to further reduce recall bias on self-report HF incidence due to cognitive decline. Among them, 72 developed incident HF. Results were consistent with analyses based on the complete set. As shown in Table S4, for each 1-SD increase in the square root transformed nap duration, the HR was 1.39 (95% CI: 1.09-1.75; *p* = 0.009). The HR was 1.45 (95% CI: 1.14-1.83; *p* = 0.003) for 1-SD increase in the square root transformed nap frequency.

Consideration of reduced physical activity level

We assessed total daily activity using actigraphy in terms of activity counts per day. To avoid collinearity between napping characteristics and total daily activity (Figure S2), we dichotomized napping duration and frequency based on their medians, and fitted Cox proportional hazards models by including the dichotomized variables and square root transformed total daily activity (due to right skewness) adjusted for age, sex, and education. Consistently, results demonstrated increased risk of HF in frequent nappers (Table S3; HR 2.00 (95% CI: 1.16-3.44; *p* = 0.012) while total daily activity became not significant although it was by itself(for each 1-SD decrease in the square root transformed total daily activity: HR $= 1.35$; 95% CI: 1.06-1.70; $p = 0.013$). However, neither napping duration nor total daily activity was significant which may still be a consequence of collinearity between them although dichotomization was done. Further studies are warranted to better elucidate their relationships.

Table S1. Daytime nap duration, covariates, and incident heart failure jointed determined by self-report and medication.

*Results for 1-unit increase. †Results for 1-SD increase.

Model A is the core model adjusted for age, sex, and years of education. Model B, C, and D all build upon model A by additionally including nighttime sleep factors (B), co-morbidities (C), and cardiovascular diseases and risk factors (D), respectively. Model E is the full model with all covariates adjusted.

Table S2. Daytime nap frequency, covariates, and incident heart failure jointed determined by self-report and medication.

*Results for 1-unit increase. †Results for 1-SD increase.

Model A is the core model adjusted for age, sex, and years of education. Model B, C, and D all build upon model A by additionally including nighttime sleep factors (B), co-morbidities (C), and cardiovascular diseases and risk factors (D), respectively. Model E is the full model with all covariates adjusted.

Table S3. Dichotomized napping characteristics and incident heart failure.

*Results for 1-unit increase.

CI = confidential interval; HR = hazard ratio

Table S4. Sensitivity analyses results within subjects who were cognitively intact at baseline.

*Results for 1-unit increase. †Results for 1-SD increase.

Table S5. Nighttime sleep duration and incident heart failure.

*Results for 1-unit increase.

CI = confidential interval; HR = hazard ratio

Table S6. Objective measures of daytime napping duration, nighttime sleep duration,

and incident heart failure.

*Results for 1-unit increase. †Results for 1-SD increase.

Table S7. Objective measures of daytime napping duration, longer nocturnal sleeper,

and incident heart failure.

*Results for 1-unit increase. †Results for 1-SD decrease.

Figure S1. Number of awakenings and its correlations with sleep fragmentation index, nap duration, and nap frequency.

Figure S2. Napping characteristics highly correlated with total daily activity.