



Changes in Cardiovascular Health Status and Risk of Sudden Cardiac Death in Older Adults

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Purpose: Cardiovascular health (CVH) status is associated with several cardiovascular outcomes; however, correlations between changes in CVH status and risk of sudden cardiac death (SCD) are unknown. We aimed to evaluate associations between changes in CVH status and risk of SCD and all-cause death in older adults.

Materials and Methods: We used data from the Korea National Health Insurance Service-Senior cohort database (2005–2012). Six metrics from the American Heart Association (smoking, body mass index, physical activity, blood pressure, total cholesterol, and fasting blood glucose) were used to calculate CVH scores. Changes in CVH status between two health checkups were categorized as low to low, low to high, high to low, and high to high.

Results: We included 105200 patients whose CVH status for an initial and follow-up health checkup (2-year interval) was available. During a median of 5.2 years of follow-up after a second health checkup, 688 SCDs occurred. Compared to patients with a persistent low CVH status, those with a consistently high CVH status had a reduced risk of SCD [adjusted hazard ratio (HR), 0.69; 95% confidence interval (CI), 0.56–0.86] and all-cause death (adjusted HR, 0.74; 95% CI, 0.69–0.78). The risk of all-cause death followed similar trends. However, an inconsistent linear relationship was observed for changes in CVH status and the risk of SCD, but not of all-cause death.

Conclusion: Maintaining a high CVH status was associated with future risks of SCD and all-cause death among an older adult population.

Key Words: Sudden cardiac death, mortality, health status index, elderly

INTRODUCTION

Sudden cardiac death (SCD) accounts for approximately 25% of the global mortality from cardiovascular disease (CVD).^{1,2} Despite the importance and societal burden of SCD, current

preventive strategies for SCD are limited. Several clinical risk factors, including hypertension, diabetes mellitus (DM), dyslipidemia, smoking, and physical inactivity are associated with SCD.^{3,4} Modification of these clinical factors might reduce the risk of SCD; hence, an effective risk stratification strategy is necessary. The American Heart Association (AHA) committee has developed a new concept of cardiovascular health (CVH) status and proposed metrics thereof that need to be monitored over time.⁵ The CVH metrics comprise seven modifiable behavioral and biologic metrics. Previous studies have revealed a strong association between CVH status and various cardiovascular outcomes.^{6–9} In a recent study conducted in Finland, a high CVH score at baseline substantially reduced the risk of SCD among middle-aged men.¹⁰ Although several studies have assessed the incidence of SCD,¹¹ limited data are available regarding the predictors of SCD in older adults.^{12–14} The effects of changes in CVH status, in particular, over time on

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SCD in older adults have not been widely studied. Therefore, we evaluated whether baseline CVH status and changes in CVH status over time are associated with a reduced risk of SCD in an older adult population. Further, we assessed whether individual CVH metrics are associated with a reduced risk of SCD. We also evaluated the risk of all-cause death with changes in CVH status.

MATERIALS AND METHODS

Data were collected from the Korea National Health Insurance Service (NHIS)-Senior, which contains data on 558147 individuals, accounting for approximately 10% of the entire older adult population aged ≥ 60 years in South Korea (about 5.1 million) from 2002.¹⁵ The NHIS-Senior database includes the following: sociodemographic and socioeconomic information, insurance status, health checkup data, and records of patient medical and dental history. These parameters were stratified to cover 12 years (2002–2013) and anonymized to protect the privacy of individuals included in this cohort study. This study was approved by the Institutional Review Board of Yonsei University Health System (4-2020-0703). The need for informed consent was waived. The NHIS-Senior database used in this study (NHIS-2016-2-171) is maintained by NHIS. The authors declare no conflicts of interest with NHIS.

Study population

From the NHIS-Senior database, 312736 patients who underwent health checkups between 2005 and 2012 and who had follow-up data until December 2013 were enrolled. Patients with a body mass index (BMI) < 18.5 kg/m² (n=15446), hypertrophic cardiomyopathy (n=686), a history of heart failure (n=25819), ischemic stroke or transient ischemic attack (n=29163), myocardial infarction (n=8874), hemorrhagic stroke (n=1075), venous thromboembolism (n=1430), malignancy (n=23432), and missing data on CVH metrics (n=5843) were excluded. The remaining 197241 participants were included for analysis (Fig. 1).

Covariates

Baseline comorbidities were identified from medical claims data and prescription medication information prior to the index date. To ensure diagnostic accuracy, the patients were considered to have comorbidities when the condition was mentioned as a diagnosis at discharge or had been confirmed at least twice in an outpatient setting. This was in line with previous studies using the NHIS database (Supplementary Table 1, only online).^{12,15–18} Economic status variables were categorized into low, intermediate, and high status based on the total amount of national health insurance premiums paid by an insured person in each year, which are proportional to the person's income.

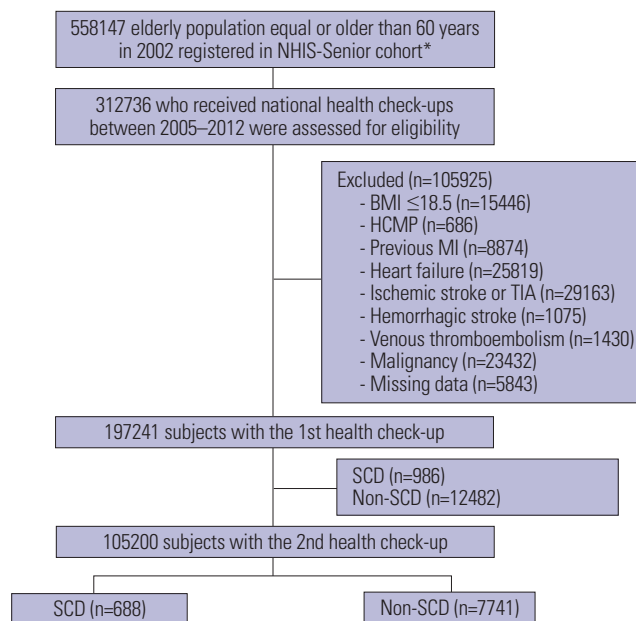


Fig. 1. Screening and study flowchart of the participants. *Korean National Health Insurance Service (NHIS)-Senior cohort. BMI, body mass index; HCM, hypertrophic cardiomyopathy; MI, myocardial infarction; TIA, transient ischemic attack; SCD, sudden cardiac death.

CVH

The AHA criteria were used to calculate CVH scores from six metrics (smoking, physical activity, BMI, blood pressure, fasting blood glucose concentrations, and blood total cholesterol) (Supplementary Table 2, only online). The scores for each metric ranged from 0 to 2 (0, poor metrics; 1, intermediate metrics; and 2, points for ideal metrics). Thus, the sum of CVH scores in an individual ranged from 0 to 12. Change in CVH scores was calculated and examined for participants whose CVH scores for an initial and follow-up health checkup were available. Based on the mean value of 6, changes in CVH status were categorized as consistent high (≥ 7 to ≥ 7), high to low (≥ 7 to ≤ 6), low to high (≤ 6 to ≥ 7), and consistent low (≤ 6 to ≤ 6). The median interval between the initial and follow-up health checkups was 2.0 years.

Ascertainment of SCD

In the NHIS-Senior database, we identified patients with SCD using the International Classification of Diseases, 10th revision codes I46.x (cardiac arrest) and I49.0 (ventricular fibrillation). To avoid erroneous inclusion of patients with non-cardiac death, we excluded patients with sudden death accompanied by respiratory arrest (R09.0 and R09.2), gastrointestinal bleeding (I85.0, K25.0, K25.4, K26.0, K26.4, K27.0, K27.4, and K92.0–K92.2), brain hemorrhage (I60.x–I62.x and S06.4–S06.6), septic shock (A41.9 and R57.2), pregnancy and delivery (O00–O99), diabetic ketoacidosis (E14.1), anaphylaxis (T78.2), and accidents including asphyxiation, drowning, poisoning, traffic accident, fall, and suicide (T71, T75.1, T36–T65, V01–V99, W00–W99, X60–X84, respectively). The positive predictive value of

our criteria of SCD was 80.2% (586 of 731), which suggests good diagnostic accuracy.¹²

Statistical analysis

Baseline characteristics of the participants were compared using descriptive statistics and are presented as median (interquartile interval) values for continuous variables and number (percentage) for categorical variables. Changes in CVH status based on 12-point CVH scores and the number of ideal metrics (0–6) were used as time-varying variables in Cox proportional hazards regression models. For each CVH metric, time-varying Cox regression analysis was performed. The rates of events were calculated by dividing the number of events by 1000 person-years at risk, with 95% confidence intervals (CIs) estimated using exact Poisson distributions. Competing risk analysis was performed using the Fine and Gray method,¹⁹ and sub-distribution hazard ratios (sHR) were estimated to determine the association of changes in CVH with SCD and all-cause death. Time-varying variables for change in CVH status were used to estimate hazard ratios (HR) in subgroup analysis. For each subgroup analysis for SCD and all-cause death, a *p* value for interaction was calculated, and a value of <0.05 denoted a statistically significant difference between the subgroups. The Kaplan-Meier method was used to compare the cumulative incidence difference (CID) from baseline to 72 months among the four CVH groups, with adjustment for covariates. Two-sided *p* values of <0.05 were considered significant. Statistical analyses were performed using SAS, version 9.4 (SAS Institute, Cary, NC, USA) and R, version 4.0.0 (R Foundation for Statistical Computing, Vienna, Austria).

RESULTS

The baseline characteristics and CVH statuses of the study participants at the initial and follow-up health checkups are shown in Table 1. At baseline, the median patient age was 70 years. Of the total participants, 43.4% were men and 50.9% had hypertension. The median CVH score was 8 points at the first and second health checkups. In 71.4% participants, the CVH score was high at baseline. There were no differences in the baseline characteristics and CVH statuses between first and second health checkups.

Time-varying CVH status and risk of SCD and all-cause death

Over a median follow-up of 7.2 years after baseline, there were 1674 SCDs and 20223 all-cause deaths. Compared to low CVH status, high CVH status was associated with a lower risk of SCD (HR, 0.70; 95% CI, 0.63–0.78) and all-cause death (HR, 0.80; 95% CI, 0.78–0.82) after adjusting for age, sex, economic status, presence of coronary heart disease (CHD), hypertension, DM, anemia, and chronic obstructive pulmonary disease. For each ad-

Table 1. Patient Characteristics and CVH Status at Initial and Follow-Up Health Checkups

| Characteristics | Initial (n=197241) | Follow-up (n=105200) |
|--|--------------------|----------------------|
| Age (yr) | 70 (66, 74) | 70 (68, 72) |
| Men | 85415 (43.3) | 48416 (46.0) |
| Economic status | | |
| Low | 74508 (37.8) | 39692 (37.7) |
| Intermediate | 88097 (44.7) | 46821 (44.5) |
| High | 34636 (17.5) | 18687 (17.8) |
| Hypertension | 100437 (50.9) | 53471 (50.8) |
| Diabetes mellitus | 26894 (13.6) | 13783 (13.1) |
| Dyslipidemia | 58807 (29.8) | 31910 (30.3) |
| Coronary heart disease | 1734 (0.9) | 938 (0.9) |
| Chronic kidney disease | 1837 (0.9) | 928 (0.9) |
| Anemia | 31357 (15.9) | 15506 (14.8) |
| Hyperthyroidism | 4353 (2.2) | 2273 (2.2) |
| Hypothyroidism | 4732 (2.4) | 2520 (2.4) |
| Chronic obstructive pulmonary disease | 11862 (6.0) | 6149 (5.8) |
| Chronic liver disease | 39252 (19.9) | 21518 (20.5) |
| No. of ideal metrics* | 3 (2, 4) | 3 (2, 4) |
| No. of ideal CVH metrics* | | |
| Low, 0–1 | 25128 (12.7) | 13172 (12.5) |
| Moderate, 2–3 | 120381 (61.0) | 64042 (60.9) |
| High, 4–6 | 51732 (26.3) | 27986 (26.6) |
| 12-point CVH scores [†] | 8 (6, 8) | 8 (6, 9) |
| CVH status by 12-point CVH scores [†] | | |
| Low, 0–6 | 56327 (28.6) | 29320 (27.8) |
| High, 7–12 | 140914 (71.4) | 75880 (72.2) |

CVH, cardiovascular health.

The data are presented as a number (%) and median (interquartile). Non-parametric continuous variables that were evaluated using the Kolmogorov-Smirnov method were analyzed using a Mann-Whitney U test.

The CVH metrics included smoking, body mass index, physical activity, blood pressure, fasting plasma glucose, and plasma total cholesterol. [†]The continuous 12-point CVH score (range, 1–12; higher score indicating higher CVH) was calculated by assigning 0 (poor), 1 (intermediate), and 2 (ideal) points to each of the 6 metrics (listed in footnote) and summing them.

ditional time-varying point in the 12-point CVH scores (HR, 0.91; 95% CI, 0.88–0.93) and cardiovascular ideal metrics (HR, 0.93; 95% CI, 0.89–0.97), the risk of SCD decreased continuously. Similar results were seen for all-cause death (Table 2).

Subgroup analysis showed that the risk of SCD reduced consistently with higher CVH status, regardless of age, sex, CHD, hypertension, DM, and anemia. In patients with chronic obstructive pulmonary disease (COPD), high CVH status did not reduce the risk of SCD, compared with those without COPD (Fig. 2). In patients with age <75 years, male sex, absence of hypertension or COPD, high CVH status was significantly reduced the risk of all-cause death compared with their counterparts (Fig. 3).

Table 2. Time-Varying Cox Proportional Hazards Model for Incident SCD and All-Cause Death from the Initial Health Checkup (n=197241)

| | CVH status adjusted HR (95% CI)* | | | |
|---------------------------|-----------------------------------|------------------|---|-----------------------------|
| | CVH status by 12-point CVH scores | | Per 1-point increase in the 12-point CVH scores | Per additional ideal metric |
| | Low, ≤6 | High, ≥7 | | |
| SCD (n=1674) | 1 [Reference] | 0.70 (0.63–0.78) | 0.91 (0.88–0.93) | 0.93 (0.89–0.97) |
| All-cause death (n=20223) | 1 [Reference] | 0.80 (0.78–0.82) | 0.93 (0.92–0.94) | 0.97 (0.96–0.98) |

CI, confidence interval; CVH, cardiovascular health; HR, hazard ratio; SCD, sudden cardiac death.

*HRs and 95% CIs were estimated using Cox proportional hazards models stratified by a 2-year interval between the first and second health checkups as a time-varying variable, adjusted by age, sex, economic status, coronary heart disease, hypertension, diabetes mellitus, anemia, and chronic obstructive pulmonary disease over a median follow-up of 7.2 (interquartile range, 5.1–8.1) years starting from the first health checkup.

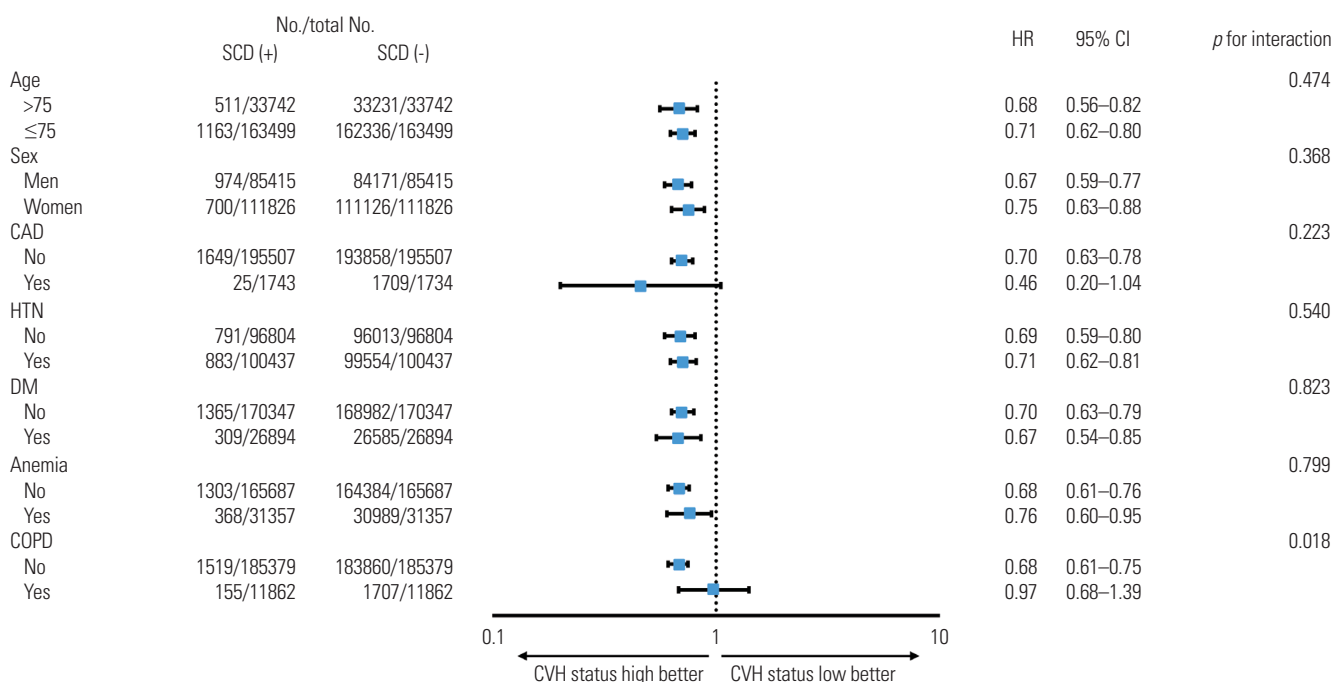


Fig. 2. Subgroup analysis of SCD by time-varying Cox proportional model per 1-point increase in the 12-point CVH scores. SCD, sudden cardiac death; CAD, coronary artery disease; HTN, hypertension; DM, diabetes mellitus; COPD, chronic obstructive pulmonary disease; HR, hazard ratio; CI, confidence interval, CVH, cardiovascular health.

Individual CVH metrics and the risk of SCD and all-cause death

Supplementary Table 3 (only online) shows time-varying hazards of individual CVH metrics. An increase in scores toward the ideal CVH status afforded a significantly reduced risk of SCD. Compared with poor CVH scores, ideal CVH scores for smoking, physical activity, blood pressure, fasting glucose, and total cholesterol were significantly associated with 39%, 18%, 20%, 34%, and 18% reductions in the risk of SCD, respectively. The associations remained significant for the risk of all-cause death. However, ideal BMI was not associated with a reduced risk of SCD or all-cause death. The findings of Cox proportional regression analysis based on baseline CVH metrics were similar to those of the time-varying analysis. However, the effects were weaker on SCD, especially in smoking, blood pressure, fasting glucose, and total cholesterol components (Supplementary Table 4, only online).

Changes in CVH status, SCD, and all-cause death

During the median follow-up of 5.2 years after the second health checkup, there were 688 SCDs and 7741 all-cause deaths. Baseline characteristics and CVH status of all patients with SCD and all-cause death are presented in Supplementary Table 5 (only online).

Table 3 shows the results of incidence rate ratio and competing risk analysis in participants whose CVH scores for an initial and follow-up health checkup were available. Based on baseline CVH status, 27.9% and 72.1% participants were divided into the low- and high-level groups, respectively. At baseline, compared to participants in the low group, those in the high group had a lower risk of SCD (adjusted sHR, 0.82; 95% CI, 0.70–0.97) and all-cause death (adjusted sHR, 0.82; 95% CI, 0.70–0.97), respectively. The incidence rates also showed lower risks of SCD (adjusted HR, 0.81; 95% CI, 0.69–0.95) and all-cause death (adjusted HR, 0.80; 95% CI, 0.78–0.85). To assess the association between change in CVH status and risk of SCD,

we constructed four groups using a cutoff score of 6 points. The consistent low status group was used as the reference. While the CVH status of 15.9% participants improved from low to

high, the CVH status of 9.5% participants deteriorated from high to low. Of the 74.6% participants with a stable CVH status, 11.9% had a consistent low CVH status, and 62.7% had consis-

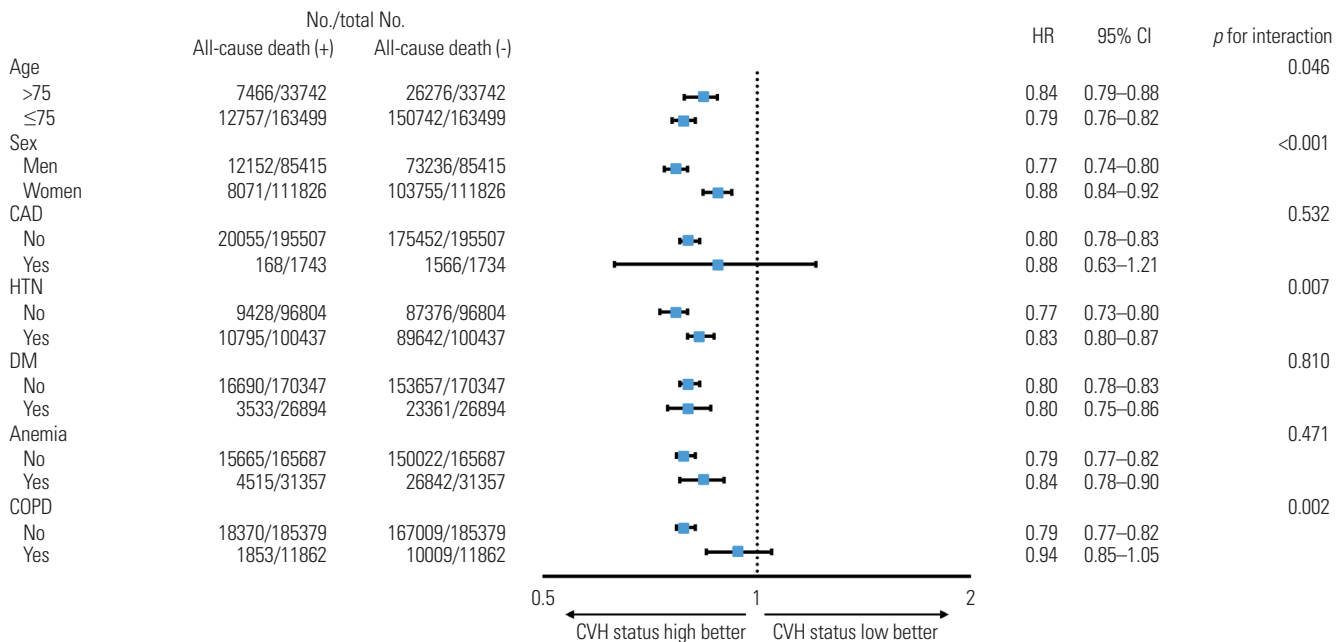


Fig. 3. Subgroup analysis of all-cause death by time-varying Cox proportional model per 1-point increase in the 12-point CVH scores. SCD, sudden cardiac death; CAD, coronary artery disease; HTN, hypertension; DM, diabetes mellitus, COPD, chronic obstructive pulmonary disease; HR, hazard ratio; CI, confidence interval, CVH, cardiovascular health.

Table 3. Incident SCD, All-Cause Death, and Sub-Distribution Hazard Model for the Competing Risk of CVH Status Changes between Unital and Follow-Up Health Checkups (n=105200)

| | Incidence rate per 1000 person-years (95% CI) | Adjusted HR [†] | p value | Competing analysis model sHR [‡] | p value |
|--------------------------|---|--------------------------|---------|---|---------|
| SCD (n=688) | | | | | |
| Baseline CVH status* | | | | | |
| Low (n=29320) | 0.12 (0.10–0.13) | [Reference] | | [Reference] | |
| High (n=75880) | 0.09 (0.08–0.10) | 0.81 (0.69–0.95) | 0.012 | 0.82 (0.70–0.97) | 0.019 |
| CVH status change* | | | | | |
| Low to low (n=12550) | 0.13 (0.11–0.16) | [Reference] | | [Reference] | |
| Low to high (n=16770) | 0.11 (0.09–0.13) | 0.89 (0.69–1.14) | 0.357 | 0.87 (0.67–1.12) | 0.270 |
| High to low (n=9983) | 0.14 (0.12–0.17) | 1.10 (0.84–1.44) | 0.483 | 1.12 (0.86–1.46) | 0.400 |
| High to high (n=65897) | 0.08 (0.07–0.09) | 0.69 (0.56–0.86) | <0.001 | 0.69 (0.55–0.85) | <0.001 |
| All-cause death (n=7741) | | | | | |
| Baseline CVH status* | | | | | |
| Low (n=29320) | 1.34 (1.29–1.39) | [Reference] | | [Reference] | |
| High (n=75880) | 1.00 (0.97–1.02) | 0.80 (0.78–0.85) | <0.001 | 0.81 (0.77–0.85) | <0.001 |
| CVH status change* | | | | | |
| Low to low (n=12550) | 1.46 (1.39–1.54) | [Reference] | | [Reference] | |
| Low to high (n=16770) | 1.24 (1.18–1.31) | 0.87 (0.82–0.94) | <0.001 | 0.88 (0.82–0.95) | 0.014 |
| High to low (n=9983) | 1.21 (1.13–1.29) | 0.86 (0.79–0.93) | <0.001 | 0.83 (0.77–0.91) | <0.001 |
| High to high (n=65897) | 0.96 (0.93–0.99) | 0.74 (0.69–0.78) | <0.001 | 0.74 (0.70–0.79) | <0.001 |

CI, confidence interval; CVH, cardiovascular health; HR, hazard ratio; sHR, sub-distribution hazard ratio; SCD, sudden cardiac death. Change in CVH status was computed over a median time interval of 2.0 (interquartile range 1.7–2.3) years in participants who were free from SCD and all-cause death within this time interval.

*The two statuses were defined as CVH score low (those with 12-point CVH score ≤6 at baseline) and high (≥7 baseline) as used in the Framingham Offspring Study, [†]HRs and 95% CIs were estimated using Cox proportional hazards model with a median follow-up time of 5.2 (interquartile range 4.1–6.3) years from the second health checkup. HRs were adjusted for age, sex, economic status, coronary heart disease, hypertension, diabetes mellitus, anemia, and chronic obstructive pulmonary disease, [‡]sHRs and 95% CIs were estimated using Cox proportional hazards model using the Fine and Gray method for competing risk. HRs were adjusted for age, sex, economic status, coronary heart disease, hypertension, diabetes mellitus, anemia, and chronic obstructive pulmonary disease.

tent high CVH status over time. Participants who had high CVH status throughout the study period had a lower risk of SCD (adjusted sHR, 0.69; 95% CI, 0.55–0.85) and all-cause death (adjusted sHR, 0.74; 95% CI, 0.70–0.79). This group also had a lower risk of incident SCD (adjusted HR, 0.69; 95% CI, 0.56–0.86) and all-cause death (adjusted HR, 0.74; 95% CI, 0.69–0.78) than the consistent low CVH status group.

Kaplan-Meier curves and cumulative incidence rates throughout the study period are shown in Fig. 4. At 72 months, the cumulative incidence of SCD was lower in the consistent high group than in the consistent low group (CIDs, -0.003%; 95% CI, -0.006–-0.001), high to low group (CIDs, -0.004%; 95% CI, -0.007–-0.001), and low to high group (CIDs, -0.001%; 95% CI, -0.006–0) (Fig. 4A). The cumulative incidence rate of all-cause death was lower in the consistent high group than in the consistent low group (CIDs, -0.032%; 95% CI, -0.041–-0.023), high to low group (CIDs, -0.013%; 95% CI, -0.022–-0.004), and low to high group (CIDs, -0.013%; 95% CI, -0.022–-0.014). The high to low group had a lower cumulative incidence of all-cause death (CIDs, -0.019%; 95% CI, -0.032–-0.007) than the consistent low group, different from the pattern seen for SCD (Fig. 4B).

DISCUSSION

In this study, a consistently high CVH status was associated with a lower risk and incident rate ratio of SCD than a consistently low CVH status. Similarly, in time-varying Cox regression analysis, a high CVH score was associated with a lower risk of SCD than a low CVH score. These results were consistent regardless of age, sex, presence of CHD, hypertension, DM, and anemia. Furthermore, a consistently high CVH status was as-

sociated with a lower risk and incident rate ratio of all-cause death than a consistently low CVH status.

CVH and elderly population

Several reports have described the beneficial effects of an ideal CVH status on health-related outcomes in middle-age patients; however, there are limited data available for older adults.²⁰⁻²³ According to the studies that are available, ideal CVH scores for older individuals is 2–3 times lower than that for younger individuals. Gaye, et al.⁸ analyzed the trend of ideal CVH status among an older French population and showed that a higher CVH status was highly beneficial in lowering the risk of mortality and vascular events. Mozaffarian, et al.²⁴ reported that combined lifestyle factors, including physical activity, diet, smoking, alcohol consumption, BMI, and waist circumference, were associated with the incidence of new-onset DM. Both these studies had a 9- to 10-year follow-up period and evaluated the longitudinal effect of CVH. Although only four risk factors were used, several studies in older adult populations have shown that the ideal CVH level can increase survival and also reduce the risk of death after CVD events.^{25,26} Our study shows consistent evidence regarding the association between baseline CVH status and risk of SCD and all-cause death in older adults.

Changes in CVH status, SCD, and all-cause death

Multiple clinical risk factors have been found to be associated with SCD in previous studies.^{3,4,27} A recent study conducted in Finland reported that the risk of SCD is higher in physically inactive patients with CHD.²⁸ The risk of SCD was also increased in highly active patients, compared to that in active patients. Another recent study conducted in Finland showed that base-

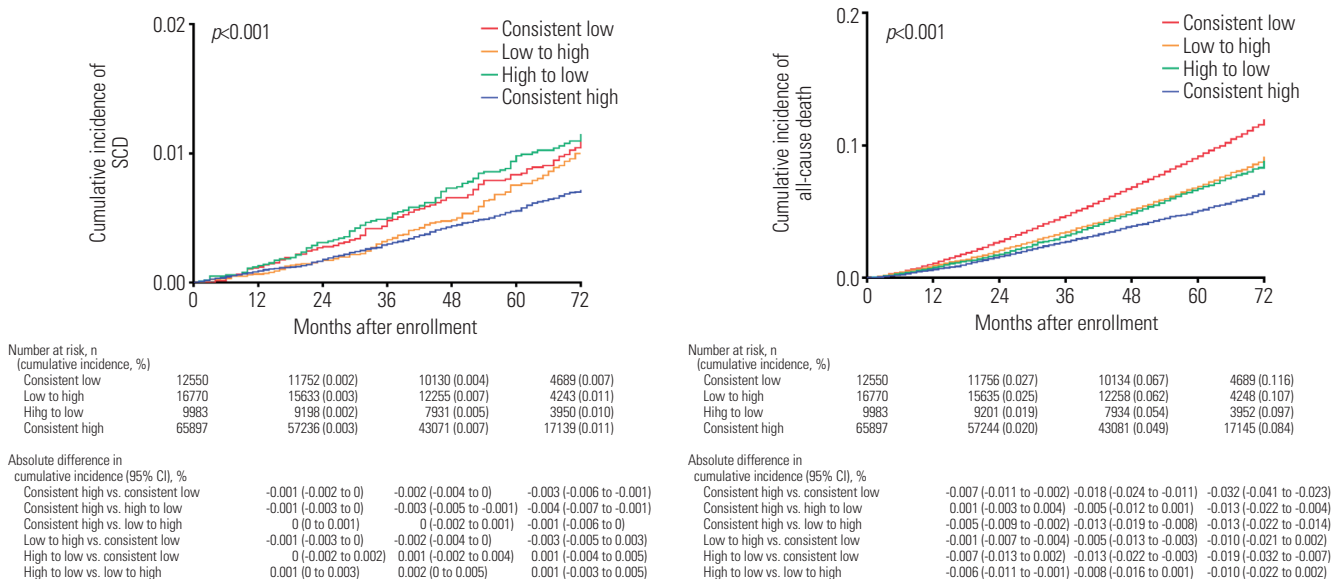


Fig. 4. Kaplan-Meier curves and cumulative incidences of (A) sudden cardiac death (SCD) and (B) all-cause death according to change in cardiovascular health status between the first and second health checkups.

line CVH status was strongly and linearly associated with the future risk of SCD and all-cause death.¹⁰ In that study, ideal physical activity did not show an independent association with the risk of SCD, compared to poor physical activity, because the study included participants with high physical activity, which itself might be a cause of sudden death.²⁹ However, in our study, ideal physical activity, which included vigorous activity, was related to lower risks of both SCD and all-cause death. In addition, our study evaluated changes in CVH status more accurately and had a larger sample size (n=105200) than previous studies. The Framingham Offspring study evaluated the trends in CVH status over a 20-year period and assessed the association between change in CVH status with subclinical and clinical CVD.⁹ They observed a decrease in ideal CVH status over 20 years, which increased the odds of subclinical disease and the risk of CVD and all-cause death. Otherwise, Sloten, et al.³⁰ reported that there was an inconsistent linear relationship between the direction of change in CVH status and the risk of CVD. Our results showed that CVH changes of low to high and high to low did not have reduced the risk of SCD, compared to consistently low CVH. In regards to all-cause death, individuals with these change in CVH had a reduced risk. This emphasizes the importance of maintaining a healthy lifestyle to prevent SCD among older adults, compared to the all-cause death, relatively.⁹ This inconsistent linear relationship was observed in our results for the risk of SCD, but not of all-cause death.

Our study included a large number of participants in a sample representative of the older Korean adult population. We explicitly accounted for the competing risk of all-cause death, thus identifying the utility of CVH change, which was enriched for SCD and all-cause death. We studied the CVD population with an intermediate risk for SCD, where clinically meaningful stratification of risk is more likely to be observed. Majority of SCDs occur in subjects without a known heart disease; thus, risk prediction might be helpful, although it is considered a greater challenge. The prediction of SCD is essential for the identification and prevention of patients at risk. Although an ejection fraction <35% or 30% is commonly considered as a major risk factor, <10% patients with myocardial infarction develop an ejection fraction <35%³¹ and most SCDs occur in patients with an ejection fraction >35%.¹ We excluded subjects who were diagnosed with heart failure at baseline, and this approach represents the larger population who require assessment for the prediction of SCD.

Limitations

This study has several limitations. First, in studies using administrative databases, coding inaccuracies can lead to errors. We used a definition that has already been validated in previous studies to minimize this possibility. Second, since the study design was observational and health examination of the subjects was conducted at different hospitals and clinics, the pos-

sibility of unmeasured confounders cannot be ruled out. Third, we used only six metrics of CVH and excluded the diet metrics. There are no data from nationally representative samples that allows for adequate quantification of caloric expenditure; hence, inclusion of diet metrics can increase the risk of bias. Therefore, it would be interesting to investigate how accurately the diet component can be measured in this cohort. Fourth, participants in our study who survived until at least 65 years of age could be healthier and have higher socioeconomic status, compared to their counterparts. Finally, all-cause death was not classified into further categories. We, therefore, might have overestimated the benefit of high CVH status over time in this study.

In older adults, maintaining a high CVH status is significantly associated with a lower risk of SCD and all-cause death. These findings may help risk stratification for the prediction of SCD and all-cause death.

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AUTHOR CONTRIBUTIONS

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