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ORIGINAL RESEARCH

Omega-3 fatty acid intake and sudden cardiac arrest incidence in males and females : a case-control multicenter study

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Abstract

Background: Although numerous studies found an association between omega-3 fatty acids (FAs) and cardiovascular disease outcomes, the results remain controversial. Furthermore, research on the association with sudden cardiac arrest (SCA) is limited. This study aimed to determine the association of omega-3 FA intake with SCA risk according to sex. **Methods**: We conducted a multicenter case-control study at 17 hospitals in Korea from September 2017 to December 2023, including 1331 patients aged 19–79 years with out-of-hospital cardiac arrest with presumed cardiac etiology. Community-based controls were recruited at a 1:1 ratio after matching for age, sex, and urban residence level. The main exposure of interest was omega-3 FA intake. We compared the outcomes using multivariable logistic regression, with interaction terms included in the final model to assess whether sex modified the effect of omega-3 FA intake on SCA incidence. **Results**: Multivariable conditional logistic regression analysis revealed that omega-3 FA intake was associated with a reduced risk of SCA (adjusted odds ratio (OR) [95% confidence interval (CI)]: 0.52 [0.42–0.65]). Interaction analysis showed that omega-3 FA intake served as a preventive factor for SCA in males (OR [95% CI]: 0.47 [0.36–0.61]) but not in females (OR [95% CI]: 0.67 [0.44–1.01]). **Conclusions**: In the general population, omega-3 FA intake was associated with a reduced SCA incidence which was more pronounced in male patients. Early preventive strategies *involving* omega-3 FA supplementation, particularly for males, may help lower the SCA risk. **Clinical Trial Registration**: The study protocol is registered at ClinicalTrials.gov (NCT03700203).

Keywords

Omega-3 fatty acid; Heart arrest; Sex; Case-control study

1. Introduction

Sudden cardiac arrest (SCA) is a major public health problem characterized by low survival. In the USA, SCA is among the top 6 causes of death [1]. The global average SCA incidence in adults is 95.9 per 100,000 inhabitants per year. Survival to discharge rates are lower in Asia (2%) than in Europe (9%), North America (6%) or Australia (11%) [2]. Despite advances in technology and international guidelines for resuscitation, the increase in survival rates is limited and outcomes remain poor [3–5].

Nutritional support can impact the incidence of SCA by promoting overall health and addressing risk factors associated with cardiovascular disease (CVD), such as obesity, hypertension and diabetes [6, 7]. Among these, omega-3 fatty acids (FAs), such as eicosapentaenoic (EPA) and docosahexaenoic (DHA) acids, may reduce the risk of atherosclerotic cardiovascular disease (ASCVD) events through various mechanisms, including triglyceride (TG)-lowering, membrane-stabilizing, anti-thrombotic, anti-inflammatory and anti-arrhythmic effects [8–11]. These benefits could indirectly influence SCA incidence by stabilizing the heart rhythm and reducing the likelihood of cardiac events potentially causing SCA.

Numerous previous studies showed that omega-3 FA intake is associated with better CVD outcomes. However, findings are controversial due to discordant results among several studies and even meta-analyses [12–15]. Research on the relationship between omega-3 FA intake and SCA is also limited, requiring further investigation in this area.

Additionally, recent epidemiological studies have suggested that gender-specific factors may influence the risk and outcomes of CVD and SCA [16–18]. The growing awareness of the differential impact of biological sex on cardiac events indicates potential variations in the effects of intervention based on sex. Correspondingly, omega-3 FAs are believed to offer different health benefits in men and women due to metabolic differences, hormone interactions and genetic factors [19, 20].

We hypothesized that omega-3 FA intake influences the risk of SCA through several mechanisms. Furthermore, these effects may vary depending on sex. Therefore, this study aimed to determine the association of omega-3 FA intake with the SCA risk according to sex.

2. Methods

2.1 Study design, study setting and data sources

This retrospective multicenter case-control study utilized data from the phase III Cardiac Arrest Pursuit Trial with Unique Registration and Epidemiologic Surveillance (CAPTURES-III) project in Korea. This initiative, supported by the Korea Disease Prevention and Control Agency, aimed to identify SCA risk factors and assess determinants of favorable outcomes through extended follow-up. The initial phase of CAPTURES, conducted in 2014 across 27 emergency departments (EDs), was a hospital-based registration study previously described in the literature [21].

The CAPTURES-III study, conducted from September 2017 to December 2023, employed a prospective case-control design across 17 university hospital EDs. The study included patients who experienced SCA and were transported by emergency medical services (EMS) with resuscitation efforts, as well as those with a presumed cardiac cause, as determined by emergency physicians. Data collection included structured interviews on sociodemographic factors, health behaviors, psychological stress and comorbidities, alongside medical record reviews covering standardized Utstein reporting templates, laboratory results, cardiac assessments and short- and long-term outcomes. Blood samples were also collected to investigate potential biomarkers associated with SCA. Similar data collection methods were applied to community-based controls to facilitate comparative analyses.

Data from participating hospitals were centralized at the Data Quality Management Committee (DQMC), which performed quality assurance and statistical evaluations. Regular quality control meetings were held to provide feedback to study site coordinators. The study protocol is registered at ClinicalTrials.gov (NCT03700203).

2.2 Study population

The CAPTURES-II project enrolled EMS-treated SCA patients aged 20–79 years who had a presumed cardiac cause. Cases resulting from non-cardiac origins—such as trauma, drowning, poisoning, burns, asphyxia or hanging—were excluded. Additional exclusion criteria included terminal illness, hospice care, the presence of a “do not resuscitate” directive, pregnancy, and patients who lived alone or lacked reliable information sources. Patients with missing data regarding omega-3 FA intake or sex were also excluded.

Community-based controls were recruited from one metropolitan university hospital and two nonmetropolitan university hospitals. Specifically, controls from metropolitan areas were obtained from a center in Seoul, while nonmetropolitan controls were recruited from institutions in Wonju and Hwasun. Control participants were matched to cases in a 1:1 ratio based on age (in five-year intervals), sex and urbanization level (metropolitan *vs.* nonmetropolitan).

2.3 Variables and measurements

The primary exposure variable in this study was omega-3 FA intake, which was assessed through structured interviews with patients, community-based controls, or, in the case of SCA patients, their family members. Omega-3 FA intake was defined as consumption for a minimum of two weeks within the past year. The CAPTURES-II registry employed a uniform questionnaire for both cases and controls. Additional data collected included demographic details (age, sex, residence type (metropolitan *vs.* urban or rural), insurance status and education level), medical history (hypertension, diabetes, dyslipidemia, stroke and coronary artery disease), and health-related behaviors (body weight status, alcohol consumption, smoking habits and physical activity levels).

2.4 Statistical analysis

Baseline characteristics of SCA cases and controls were summarized using descriptive statistics. Categorical variables were analyzed using chi-square tests, whereas continuous variables with a normal distribution were evaluated using *t*-tests. Case-control matching was performed considering age, sex, and urbanization level (metropolitan *vs.* urban or rural). To assess the impact of omega-3 FA intake and sex on SCA risk, conditional logistic regression analysis was applied, adjusting for potential confounders identified through directed acyclic graph models. Adjusted odds ratios (aORs) and 95% confidence intervals (CIs) were calculated to determine statistical significance. Additionally, an interaction analysis was conducted to explore whether sex modified the effect of omega-3 FA intake on SCA incidence. All variables included in the final model were tested for multicollinearity, with no significant collinearity detected.

All statistical analyses were conducted using SAS version 9.4 (SAS Institute Inc., Cary, NC, USA). Statistical significance was set at a two-tailed p-value of < 0.05.

3. Result

3.1 Demographic findings

A total of 1331 SCA cases and 1331 community-based controls were enrolled in this analysis. Table 1 shows the characteristics of enrolled patients. The percentage of positive omega-3 FA intake history in SCA cases and controls was 14.4% (192/1331) and 28.3% (377/1331; $p < 0.001$), respectively.

Table 2 provides the characteristics of the study population according to omega-3 FA intake. The omega-3 FA intake group had a higher incidence of dyslipidemia than the control group (28.6% *vs.* 18.2%; $p < 0.001$). The omega-3 intake group also had higher frequencies of obesity (12.1% *vs.* 6.7%; $p < 0.001$) and vigorous activity (59.6% *vs.* 44.4%; $p < 0.001$) compared to controls. SCA incidence was significantly lower in the omega-3 intake group compared to controls (33.7% *vs.* 54.4%; $p < 0.001$).

TABLE 1. Characteristics of the out-of-hospital cardiac arrest case group and age-, sex-, and urbanization level–matched control group.

Variables	All N (%)	SCA cases N (%)	Community controls N (%)	*p*-value
All	2662 (100.0)	1331 (100.0)	1331 (100.0)	
Age, yr				
18–29	64 (2.4)	32 (2.4)	32 (2.4)	
30–39	122 (4.6)	61 (4.6)	61 (4.6)	
40–49	352 (13.2)	176 (13.2)	176 (13.2)	1.00
50–59	644 (24.2)	322 (24.2)	322 (24.2)	
60–69	760 (28.5)	380 (28.5)	380 (28.5)	
70–120	720 (27.0)	360 (27.0)	360 (27.0)	
Gender, female	652 (24.5)	326 (24.5)	326 (24.5)	1.00
Metropolis, yes	1348 (50.6)	674 (50.6)	674 (50.6)	1.00
Medical aid, yes	268 (10.1)	142 (10.7)	126 (9.5)	0.30
Education level, high	879 (33.0)	368 (27.6)	511 (38.4)	<0.001
Comorbidities				
Hypertension	1139 (42.8)	626 (47.0)	513 (38.5)	0.007
Diabetes mellitus	620 (23.3)	402 (30.2)	218 (16.4)	<0.001
Dyslipidemia	544 (20.4)	217 (16.3)	327 (24.6)	<0.001
Stroke	159 (6.0)	110 (8.3)	49 (3.7)	<0.001
Coronary artery disease	240 (9.0)	186 (14.0)	54 (4.1)	<0.001
Health-related behavior				
Obesity, yes	210 (7.9)	55 (4.1)	155 (11.6)	<0.001
Alcohol intake, yes	1729 (65.0)	760 (57.1)	969 (72.8)	0.004
Smoking				
Current smoker	751 (28.2)	476 (35.8)	275 (20.7)	
Ex-smoker	870 (32.7)	375 (28.2)	495 (37.2)	0.005
Non-smoker	1041 (39.1)	480 (36.1)	561 (42.1)	
Vigorous activity, yes	1269 (47.7)	408 (30.7)	861 (64.7)	<0.001
Omega-3 fatty acid intake, yes	569 (21.4)	192 (14.4)	377 (28.3)	<0.001

SCA: out-of-hospital cardiac arrest.

3.2 Main outcomes

Table 3 demonstrates the results of the multivariable conditional logistic regression model, including aORs (95% CIs) for SCA incidence with omega-3 FA intake. Omega-3 FA intake was associated with decreased SCA risk (aOR [95% CI]: 0.52 [0.42–0.65]).

3.3 Interaction analysis

We added an interaction term between omega-3 FA intake and sex to the fully adjusted model. Table 4 presents the ORs assessing the statistical interaction of SCA incidence with the multivariable conditional logistic regression analysis. Omega-3 FA intake was a preventive factor for SCA, although only in males (OR [95% CI]: 0.47 [0.36–0.61]) and not in females (OR [95% CI]: 0.67 [0.44–1.01]).

4. Discussion

This study provides evidence of an association between omega-3 FA intake and SCA incidence, with findings suggesting that this relationship may differ by sex. Interaction analysis indicated a significant association between omega-3 FA intake and reduced SCA risk in males, while no statistically significant effect was observed in females after adjusting for demographic factors, comorbidities, and lifestyle behaviors.

These results contribute to a broader understanding of the complex interplay between dietary factors and sex-specific cardiovascular risk. This knowledge can inform future strategies aimed at reducing SCA incidence, particularly through nutritional interventions. Given the observed protective effect in males, dietary recommendations emphasizing omega-3 FA intake may be particularly beneficial for this group.

Previous studies evaluated the potential cardiovascular benefits of omega-3 FAs with divergent conclusions [22, 23].

TABLE 2. Characteristics of the study population according to the omega-3 fatty acid intake.

Variables	All N (%)	Omega-3 fatty acid intake		*p*-value
		Yes N (%)	No N (%)	
All	2662 (100.0)	569 (100.0)	2093 (100.0)	
Case-control				
SCA cases	1331 (50.0)	192 (33.7)	1139 (54.4)	<0.001
Community controls	1331 (50.0)	377 (66.3)	954 (45.6)	
Age, yr				
18–29	64 (2.4)	8 (1.4)	56 (2.7)	
30–39	122 (4.6)	20 (3.5)	102 (4.9)	
40–49	352 (13.2)	64 (11.2)	288 (13.8)	0.041
50–59	644 (24.2)	131 (23.0)	513 (24.5)	
60–69	760 (28.5)	172 (30.2)	588 (28.1)	
70–120	720 (27.0)	174 (30.6)	546 (26.1)	
Gender, female	652 (24.5)	161 (28.3)	491 (23.5)	0.023
Metropolis, yes	1348 (50.6)	293 (51.5)	1055 (50.4)	0.651
Medical aid, yes	268 (10.1)	56 (9.8)	212 (10.1)	0.844
Education level, high	879 (33.0)	213 (37.4)	666 (31.8)	0.013
Comorbidities				
Hypertension	1139 (42.8)	258 (45.3)	881 (42.1)	0.161
Diabetes mellitus	620 (23.3)	123 (21.6)	497 (23.7)	0.292
Dyslipidemia	544 (20.4)	163 (28.6)	381 (18.2)	<0.001
Stroke	159 (6.0)	27 (4.7)	132 (6.3)	0.163
Coronary artery disease	240 (9.0)	54 (9.5)	186 (8.9)	0.661
Health-related behavior				
Obesity, yes	210 (7.9)	69 (12.1)	141 (6.7)	<0.001
Alcohol intake, yes	1729 (65.0)	379 (66.6)	1350 (64.5)	0.352
Smoking				
Current smoker	751 (28.2)	135 (23.7)	616 (29.4)	
Ex-smoker	870 (32.7)	194 (34.1)	676 (32.3)	0.030
Non-smoker	1041 (39.1)	240 (42.2)	801 (38.3)	
Vigorous activity, yes	1269 (47.7)	339 (59.6)	930 (44.4)	<0.001

SCA: out-of-hospital cardiac arrest.

TABLE 3. Multivariable conditional logistic regression analysis of omega-3 fatty acid intake for out-of-hospital cardiac arrest.

SCA incidence	SCA cases/Community controls	Model 1			Model 2			Model 3		
	n/n	cOR	95% CI		aOR	95% CI		aOR	95% CI	
Omega-3 fatty acid intake (−)	1139/954									
Omega-3 fatty acid intake (+)	192/377	0.43	0.35	0.52	0.44	0.36	0.54	0.52	0.42	0.65

SCA: out-of-hospital cardiac arrest; cOR: crude odds ratio; aOR: adjusted odds ratio; CI: confidence interval.
Model 1: adjusted for age, gender, and urbanization level.
Model 2: model 1 plus hypertension, diabetes mellitus, dyslipidemia, stroke, and ischemic heart disease.
Model 3: model 2 plus obesity, alcohol intake, smoking and physical activity.

TABLE 4. Interaction analysis for SCA incidence of omega-3 fatty acid intake and sex.

	Omega-3 fatty acid intake		
	No	Yes	*p* for interaction
	OR (95% CI)	OR (95% CI)	<0.001
Male	1.00	0.47 (0.36–0.61)	
Female	1.00	0.67 (0.44–1.01)	

SCA: out-of-hospital cardiac arrest; OR: odds ratio; CI: confidence interval.

While large-scale studies such as ASCEND (A Study of Cardiovascular Events in Diabetes) and VITAL (Vitamin D and Omega-3 Trial) did not establish a statistically significant reduction in major cardiovascular events with omega-3 FA intake, both trials reported a decrease in vascular mortality and myocardial infarction (MI). Conversely, the REDUCE-IT (Reduction of Cardiovascular Events with Icosapent Ethyl-Intervention Trial) study demonstrated a 25% relative reduction in composite cardiovascular endpoints, including CVD mortality, non-fatal MI, non-fatal stroke, coronary revascularization, and unstable angina [24]. Several other studies have similarly reported a protective role of omega-3 FAs in reducing SCA risk [24, 25].

The biological mechanisms underlying the association between omega-3 FA intake and SCA may *involve* multiple pathways. Omega-3 FAs are known to exert antihypertensive effects by decreasing systemic vascular resistance and enhancing endothelial function through increased nitric oxide production. These processes contribute to improved arterial elasticity and flow-mediated vasodilation. Furthermore, omega-3 FAs produce specialized pro-resolving mediators (SPMs), such as resolvins and neuroprotectins, which actively regulate inflammation. Resolvin E1 (RvE1) mitigates leukocyte infiltration and cytokine production, thereby reducing inflammatory responses, while RvD1 promotes the transition to anti-inflammatory macrophage activity to prevent myocardial fibrosis.

Another key mechanism is the role of omega-3 FAs in stabilizing cardiac cell membranes. Docosahexaenoic acid (DHA), a primary omega-3 FA component, helps prevent arrhythmias by modulating sodium ion channel activity in myocardial cells. Additionally, omega-3 FA supplementation has been linked to decreased cardiovascular disease events, reduced hospitalization rates for heart failure and lower overall mortality. Although further research is needed, omega-3 FAs also appear to lower triglyceride concentrations through enhanced clearance of very-low-density lipoprotein (VLDL) particles and reduced hepatic synthesis of triglycerides.

Despite these proposed mechanisms, this study had limitations in directly addressing all aspects of omega-3 FA effects. For instance, the specific composition of omega-3 FAs (*e.g.*, EPA *vs.* DHA) and their plasma and tissue levels were not measured. This limits the ability to confirm the biological plausibility of the observed protective effects against SCA. Additionally, the study did not evaluate the duration or dosage of omega-3 FA intake, which could significantly influence its cardiovascular benefits. These gaps underscore the need for future studies to incorporate detailed biochemical and longitudinal data to better understand the mechanistic role of omega-3 FAs in SCA prevention.

Moreover, the multivariable analysis results indicating that omega-3 FA intake reduces SCA risk were maintained only in males. Possible reasons for this include physiological and metabolic differences between sexes. First, men and women have different hormonal regulation regarding cardiovascular diseases. Particularly, estrogen may have a cardioprotective effect in women, potentially reducing the additional benefits of omega-3 FAs. Second, the metabolic processing of omega-3 FAs may vary by sex, possibly causing a more pronounced cardioprotective effect in men. Lastly, men and women may have different dietary patterns, lifestyles, and exposure to cardiovascular risk factors, potentially contributing to the observed sex differences in the association between omega-3 FA intake and reduced SCA risk.

Moreover, the observed sex-specific differences may be partially explained by the protective effect of omega-3 fatty acids on improving HDL cholesterol function. Males, who are more likely to have low HDL cholesterol levels compared to females, may derive greater cardiovascular benefits from omega-3 fatty acid intake through mechanisms that enhance HDL functionality. These findings align with previous studies suggesting that omega-3 fatty acids improve lipid metabolism, particularly HDL-related pathways, in males.

Furthermore, this study did not account for potential interactions between omega-3 FAs and other medications, such as aspirin or COX inhibitors, which could alter their anti-inflammatory or anti-arrhythmic properties. This oversight could affect the interpretation of results and calls for careful consideration in future research.

In this study, omega-3 FA intake was associated with decreased SCA incidence, with this effect maintained only in males. Hence, males with omega-3 FA intake may be at a lower risk for SCA than the average population. This study provides a theoretical basis for the importance of omega-3 FA supplementation for males to reduce SCA risk. However, further research on the appropriate levels of omega-3 FA intake based on sex is needed.

This study has several limitations. First, as a case-control study, it is susceptible to biases inherent to observational research. Recall bias may have influenced the accuracy of reported risk factors due to reliance on participant or family member recollections. Additionally, the selection bias in the control group could have affected comparability, potentially impacting the robustness of our findings. Misclassification of matching variables may have introduced further inconsistencies, potentially influencing risk estimations. Second, this study lacked detailed data on certain risk factors, such as parental history of SCA, duration of heart disease, smoking history, and exercise intensity. Third, self-reported historical data on patient risk factors may have been subject to over- or underestimation, as they were not cross-verified with objective data sources, such as health insurance records. The absence of secondary data validation may have led to discrepancies in assessing study outcomes. Fourth, although matching was

conducted based on age, sex and urbanization level, residual selection bias cannot be entirely excluded. Fifth, we did not collect comprehensive details regarding omega-3 FA intake, including specific dosages, duration and subtypes of omega-3 FA consumed. Additionally, reliance on caregiver reports in the case group may have led to inaccuracies in dietary intake assessment. Sixth, the study assumed a cardiac etiology for SCA cases unless a definitive non-cardiac cause was identified. This assumption may have led to misclassification of some cases, potentially affecting study results. Seventh, investigators were aware of the study hypotheses, which may have introduced an element of bias in data collection and interpretation. Eighth, this study was based on data collected more than three years ago. As such, it may not fully reflect recent changes in clinical practice, dietary patterns, or public health interventions that could influence omega-3 fatty acid intake and sudden cardiac arrest risk. The age of the data should be considered when interpreting the generalizability and current applicability of the findings.

Despite these limitations, this study provides valuable insights into the relationship between omega-3 FA intake and SCA risk, highlighting the importance of sex-specific differences. Further research is needed to refine dietary recommendations and clarify the mechanisms underlying these associations.

5. Conclusions

This study found that omega-3 fatty acid (FA) intake was associated with a reduced incidence of sudden cardiac arrest (SCA) in males but not in females, suggesting a sex-specific effect potentially related to hormonal, metabolic, and lipid profile differences. Biological mechanisms such as antihypertensive, anti-inflammatory, and anti-arrhythmic actions, as well as improved lipid metabolism, may underlie these findings. Given these results, targeted dietary recommendations and omega-3 FA supplementation could be particularly beneficial for men at higher cardiovascular risk, although further prospective research is needed to confirm these associations and refine sex-specific prevention strategies.

AVAILABILITY OF DATA AND MATERIALS

Data were obtained from the Korea Disease Control and Prevention Agency.

AUTHOR CONTRIBUTIONS

EJ and JYM—formal analysis. JYM and JHK—investigation. HHR—methodology. EJ and HHR—resources; supervision. JYM—writing-original draft. EJ—writing-review and editing. All authors have read and agreed to the published version of the manuscript.

ETHICS APPROVAL AND CONSENT TO PARTICIPATE

This study was approved by the Institutional Review Board (IRB) of Chonnam National University Hospital (IRB No. 2017–285), and the requirement for informed consent was waived by the Institutional Review Board of Chonnam National University Hospital. This study followed the Strengthening the Reporting of Observational Studies in Epidemiology (STROBE) guidelines.

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CONFLICT OF INTEREST

The authors declare no conflict of interest.

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