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ORIGINAL RESEARCH

Parent–Offspring Associations of Ideal Cardiovascular Health Metrics: Findings From the 2014 to 2021 Korea National Health and Nutrition Examination Survey

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BACKGROUND: Studies have reported the strength of cardiovascular health (CVH) metrics in parent–offspring relationships. This study aimed to describe the sex-specific associations between CVH in parents and adult offspring.

METHODS AND RESULTS: This study was conducted on the Korea National Health and Nutrition Examination Survey data set, which analyzed trios of mother–father–child, with the child's age from 20 to 39 years. To use the nature of sampling design, survey weighting was applied to all our analyses. Ideal CVH was defined as a cluster of at least 5 ideal individual CVH metrics. We examined the association between parents and their adult offspring regarding clustering CVH and individual CVH metrics through odds ratios and 95% CIs using multiple logistic regression with standard errors adjusted for within-family clustering. The study included 1267 married couples comprising 748 sons and 819 daughters. After adjusting for household income and offspring's sex, age, education, and alcohol consumption, an offspring with either parent attaining a nonideal CVH was 3.52 times more likely to have nonideal CVH. Fathers' nonideal CVH was significantly positively associated with the daughters' nonideal CVH. Maternal nonideal CVH was significantly positively associated with the son's nonideal CVH. When analyzing individual CVH metrics, ideal status in fathers or mothers reduced the likelihood of their offspring having a nonideal status.

CONCLUSIONS: This cross-sectional study showed positive and differential associations of CVH and its components between parents' and offsprings' nonideal status. Our hypothesis-generating results suggest the relevance of using CVH as a composite indicator in family-centered approaches and heart-health interventions.

Key Words: cardiovascular health ■ Korea National Health and nutrition examination survey ■ parent–offspring association

Cardiometabolic, behavioral, environmental, and social factors are major drivers of cardiovascular disease.[1] Therefore, in 2010, the American Heart Association (AHA) introduced a new concept, cardiovascular health (CVH) metrics, which was defined by a clustering of metabolic metrics (blood pressure [BP], total cholesterol [TC], and fasting blood glucose [FBG]) and health behaviors (physical activity [PA], body mass index [BMI], smoking, and diet).[2] One of the many purposes of creating this new measurement was to improve CVH in the population as a whole, in addition to continuing efforts at reducing cardiovascular disease incidence, which would contribute to the design and implementation of the upcoming cardiovascular disease and stroke prevention. According to the AHA, ideal CVH is the state of acquiring ideal status in all 7 metrics (nonsmoking, BMI <25 kg/m^2, PA at goal levels, having a diet consistent with the current guideline recommendations, untreated TC <200 mg/dL, untreated systolic/diastolic BP <120/80 mm Hg,

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CLINICAL PERSPECTIVE

What Is New?

- If a parent has poor cardiovascular health metrics, it is more likely that their child will also have poor cardiovascular health (CVH) metrics.
- The strength of this connection between parent and child's CVH may differ on the basis of sex and individual risk factors.

What Are the Clinical Implications?

- These findings underline the need for family-based interventions in managing CVH, highlighting the importance of considering familial context and individual traits in risk assessment and prevention.

Nonstandard Abbreviations and Acronyms

CVH	cardiovascular health
FBG	fasting blood glucose
KNHANES	Korea National Health and Nutrition Examination Survey
PA	physical activity
TC	total cholesterol

and FBG <100 mg/dL).[2] Ideal CVH is an independent protective factor for diabetes, atrial fibrillation, and heart failure.[3–5] It is also greatly associated with a lower risk of all-cause and cardiovascular deaths in adults.[6–9] Epidemiologic data from various nations have shown familial correlations in CVH[10–12] or individual CVH metrics, such as BMI.[13–15]

Family-based approaches, which target both caregivers and children, encourage communication within the family unit, address the structural and environmental conditions in which families live and operate, and are likely the most effective approach to promote CVH.[16] Children study and acquire health behavior from their caregivers in the shared-family context, especially during early childhood.[17] Researchers have demonstrated that CVH characteristics are passed down from parents to children through 3 possible mechanisms: pregnancy complications, heritability, and linked environmental propagation between generations.[10,18] However, the majority of studies investigating familial correlation in terms of CVH as a structure or some individual CVH metric focused only on the maternal and paternal effects on the offspring.[10,19] This study aimed to describe the sex-specific association between parents and their adult offspring with respect to CVH, using a Korean national representative survey. We also estimated the sex-specific association of the parent–offspring relationship for each CVH metric.

METHODS

Study Population

The data sets generated and analyzed during the current study are available in the Korea National Health and Nutrition Examination Survey (KNHANES) repository (https://knhanes.kdca.go.kr/knhanes/sub03/sub03_02_05.do).

The KNHANES is a nationally representative cross-sectional survey conducted by the Korea Disease Control and Prevention Agency (KDCA) encompassing noninstitutionalized Korean citizens residing in South Korea. The recruitment for this survey adhered to a multistage clustered probability design, which was based on geographic area, sex, and age group using the most recent census data from the National Census Registry in South Korea. The survey process begins with choosing primary sampling units from about 200 000 geographically defined primary sampling units throughout the nation; for each primary sampling unit, final target households were sampled using systematic sampling. All family members aged ≥1 year residing together in the selected household, who met the entry requirements, were selected to participate in basic household interviews, health interviews, physical examinations, and nutrition-related surveys.[20,21] Before taking the questionnaires, each participant provided written informed consent. The KDCA administers the KNHANES, which has been authorized by their institutional review board (2013–12EXP–03–5C, 2018–01–03–P–A, 2018–01–03–C–A, 2018–01–03–2C–A, 2018–01–03–5C–A).

From the 2014 to 2021 KNHANES participants, we identified 6374 trios (offspring–father–mother) in which the offspring was aged at least 20 years. First, we excluded 992 trios where the offspring's age was ≥40 years; however, these participants were still considered in our sensitivity analysis. Subsequently, 2794 trios were removed from the study as any member of the trio had not fasted for at least 8 hours before blood sample collection. Additionally, we excluded 4 trios in which the mother or offspring was pregnant at the time of data collection. Moreover, 1017 trios were excluded due to missing data for at least 1 CVH metric in either the offspring, fathers, or mothers. Finally, 1567 trios (1267 married parents with 748 sons and 819 daughters) were included in the analysis (Figure S1).

Assessments of CVH Metrics

Participants were interviewed in person by trained medical staff and interviewers using standardized questionnaires to gather information about their demographics, medical history, health behaviors, and health care

usage. They also completed a food intake questionnaire that used the 24-hour recall method to report on their dietary habits. The KDCA used the latest nutrient data bank available for each year to calculate nutrient intake. Regarding smoking, participants were asked, "Do you currently smoke?," and provided information about their cessation time (the question was changed to "Do you currently smoke cigarettes?" from 2019 to 2021). Physical activity was assessed using the Global Physical Activity Questionnaire, which measures PA levels in 3 domains (work, transport, and leisure time) through 16 questions, including time spent in moderate-intensity activity, vigorous-intensity activity, and sitting for each domain. Anthropometric, blood, and urine measures were collected during on-site health examinations. Body weight and height were measured to the nearest 0.1 kg and 0.1 cm, respectively. After resting in a seated position, BP was measured 3 times in a row with a standard mercury sphygmomanometer (Baumanometer Wall Unit 33(0850); Baum Co., Inc., Copiague, NY). Serum TC and glucose levels were measured enzymatically after an 8-hour fast.[21]

The ideal CVH metrics in this study were based on AHA's Life's Simple 7 metrics but were defined with some modifications for the Korean population: (1) normal untreated BP (systolic/diastolic <120/80 mm Hg); (2) untreated TC <200 mg/dL; (3) untreated FBG level <100 mg/dL; (4) at least 150 minutes of moderate-intensity aerobic PA, 75 minutes of vigorous-intensity aerobic PA, or 150 minutes of a combination of moderate- and vigorous-intensity activity throughout the week; (5) nonsmoking (never or quit smoking >12 months); (6) BMI <23.0 kg/m^2 (followed by the World Health Organization's recommendation for the Asian population[22]); and (7) an ideal diet adapted from a previous Korean study.[23] An ideal diet had to meet at least 3 of the following requirements: fat intake <35% of the total energy intake, protein intake >15% of the total energy intake, carbohydrate intake <55% of the total energy intake, sodium intake <2300 g/day, and fiber intake >20 g/day.[23] The specific definition and assessment of CVH are available in Table S1.

Each CVH metric was coded as a binary variable, granting 1 point to the ideal category versus 0 points to other categories. The CVH score was calculated as the sum of all 7 metrics. Similar to others,[11,24,25] a nonideal CVH was considered if the CVH score was <5 points and vice versa for ideal CVH.

Assessment of Covariates

The following factors were collected from the offspring: age in years, sex, education, total household income per month, and alcohol intake. Age in years was considered a continuous variable. The highest level of formal education completed as of the date of the interview was defined as educational attainment. According to its distribution, this study categorized educational attainment into 2 levels: high school or lower and college graduation or higher. We categorized equivalized household income into 3 groups (low, middle, and upper), adjusted by the square root of household size. The alcohol consumption was directly derived from the questionnaire. It was coded as yes if the participant drank more than once per month over the past year and no if the participant had never drank or drank less than once per month over the past year.

Data Preparation

The purpose of this process is to link each offspring with their father and mother. First, participants living in 1-generation families were excluded. Then, 2 separate data sets were created for fathers and mothers, using 2 variables indicating the identification numbers of the participant's biological father and mother in the original data set. Finally, the 3 data sets were merged by family identification number, and duplicate observations were checked and removed.

Statistical Analysis

Characteristics of participants (sociodemographic status, lifestyle factors, and physical and laboratory measurements) by sex or ideal/nonideal CVH status were recorded and presented. Chi-square test and independent *t*-test were used to compare statistical differences for categorical and continuous variables, respectively.

To avoid the violation of independence assumptions in the regression analyses described below, all standard errors were adjusted for within-family clustering because some families had multiple children in the data.[26] Additionally, the associations between parental CVH and offspring CVH were adjusted for various factors, including household income, offspring's sex, age in years, education, and alcohol use. To assess the strength of these associations, weighted odds ratios were calculated from weighted logistic regression (SAS PROC SURVEYLOGISTIC; more details in Data S1). First, we examined the adjusted associations between paternal/maternal/parental CVH and their offsprings' CVH. Subsequently, these associations were stratified on the basis of the sex of the offspring. Next, to determine if there was any effect modification by offspring sex in all parent–offspring associations, the interaction term (exposure × offspring sex) was incorporated into the fully adjusted regression models. Finally, weighted logistic regression analyses were conducted to explore the relationship between each component of paternal/maternal CVH (BP, TC, FBG, PA, BMI, smoking, and diet) and the corresponding component of son's/daughter's CVH.

Originally, the AHA recommended the use of the CVH definition for individuals without a history of cardiovascular disease. However, we wanted to investigate the parent–offspring CVH association in the general population, not just in the healthy population; in our primary analysis, we did not exclude participants diagnosed with any cardiovascular disease. To account for unknown variations that could occur due to having patients with cardiovascular disease as our study participants, we conducted sensitivity analyses for the association of parent–offspring clustering CVH. In the first sensitivity analysis, patients with cardiovascular disease were recorded as having nonideal CVH regardless of their CVH scores. In the second sensitivity analysis, 125 trios, involving cardiovascular disease, were excluded. Cardiovascular disease and cardiovascular risk factors tend to occur later in life. Therefore, in the third sensitivity analysis, compared with the primary analysis, we did not exclude trios whose offspring were at least 40 years old to validate whether parent–offspring CVH would be robust even when the offspring were older. All analyses were performed using SAS version 9.4 (SAS Institute, Cary, NC), and statistical significance was defined as a 2-sided *P* value of <0.05.

RESULTS

Characteristics of the Study Population

The study population comprised 1267 parents with 748 sons and 819 daughters. General characteristics of the study population according to sex are shown in Table 1. The weighted median ages of sons and daughters were similar (24.7 [22.2–28.3] and 24.2 [21.5–28.0] years, respectively). The sons had higher BMI, FBG, TC, triglycerides, systolic BP, diastolic BP, fat intake, protein intake, carbohydrate intake, sodium intake, and fiber intake than daughters (*P*<0.001 for all except TC). Overall, daughters exhibited a significantly higher weighted mean CVH score than sons (5.3 versus 4.3; *P*<0.001). Additionally, 18.8% of the daughters and 51.3% of the sons had nonideal CVH (*P*<0.001). Similar to their offspring, fathers had higher BMI, FBG, triglycerides, systolic BP, diastolic BP, fat intake, protein intake, carbohydrate intake, sodium intake, and fiber intake, but lower TC intake than mothers. The parental weighted prevalence of nonideal CVH was higher than that in their offspring (82.4% in fathers and 65.9% in mothers).

Table 2 shows the characteristics of the offspring according to their CVH status. Offspring who had nonideal CVH were older than those with ideal CVH (weighted median ages were 25.7 [22.6–29.9] versus 23.9 [21.6–27.2] years; *P*<0.001). However, there were no significant differences between offspring with nonideal and ideal CVH in terms of education level, household income, or alcohol use.

Associations Between Parental and Offspring CVH Metrics

Table 3 presents the primary analysis results of the association between parents' and offspring's clustering CVH. Overall, parental nonideal CVH was associated with offspring's nonideal CVH. After adjusting for household income and offspring's sex, age, education, and alcohol consumption, an offspring with either parent attaining nonideal CVH was 3.52 times more likely to have nonideal CVH compared with others (adjusted odds ratio [aOR], 3.52 [95% CI, 1.88–6.58]). Stratifying by offspring sex, sons were 3.17 times (aOR, 3.17 [95% CI, 1.51–6.63]) and daughters were 5.07 times (aOR, 5.07 [95% CI, 1.22–21.08]) more likely to have nonideal CVH if either parent had nonideal CVH. Fathers' nonideal CVH was significantly positively associated with daughters' nonideal CVH (aOR, 2.97 [95% CI, 1.59–5.55]). In contrast, the maternal nonideal CVH was significantly positively associated with the son's nonideal CVH (aOR, 1.48 [95% CI, 1.04–2.12]). Father–son and mother–daughter CVH associations showed similar patterns of association, although they did not reach statistical significance. Furthermore, interaction analysis was conducted to explore whether the parent–offspring CVH associations differed significantly between sons and daughters. However, the results did not reveal statistically significant interactions.

Parent–offspring CVH associations were robust in all sensitivity analyses, including (1) the change in CVH's definition, as any patient with cardiovascular disease was considered as having nonideal CVH regardless of their CVH scores; (2) exclusion of patients with cardiovascular disease; and (3) nonexclusion of trios whose offspring were aged at least 40 years (Figure). See Tables S2 through S4 for further details.

Associations Between Parental and Offspring Individual CVH Metrics

The associations between the parental and offspring individual CVH metrics are shown in Table 4. When analyzing individual metrics, similar to the results observed previously with clustering CVH, having ideal status in fathers or mothers would reduce the likelihood of having the corresponding nonideal status in their offspring. Looking more closely at each metric, the covariate-adjusted parent–offspring BP, TC, FBG, BMI, and diet were positively significant in all 4 parent–offspring pairs. For example, sons and daughters, respectively, would be 2.05 times (aOR, 1.83 [95% CI, 1.26–2.66) and 4.93 times (aOR, 4.93 [95% CI, 2.48–9.79]) more likely to have nonideal BP if their fathers had nonideal BP. In other CVH metrics, we could not find significant covariate-adjusted associations with father–daughter PA, mother–son PA, father–daughter smoking, and mother–son smoking.

Table 1. Characteristics of Study Participants

Variables	Father (n=1267)	Mother (n=1267)	Offspring (n=1567)		
			Son (n=748)	Daughter (n=819)	*P* value*
Age, y	54.5 (51.6–58.0)	52.7 (49.4–56.6)	24.7 (22.2–28.3)	24.2 (21.5–28.0)	–
Education attainment					
High school and lower	737 (56.4)	890 (68.6)	399 (55.1)	304 (38.1)	<0.001
College graduation or higher	527 (43.6)	377 (31.4)	349 (44.8)	515 (61.9)	
Household income level					
Low	158 (11.8)	158 (11.9)	93 (13.7)	96 (10.8)	0.294
Middle	223 (17.8)	223 (18.2)	125 (17.2)	142 (17.6)	
Upper	885 (70.4)	885 (69.8)	530 (69.2)	580 (71.6)	
Weight, kg	71.2±9.8	59.2±8.6	75.6±14.7	57.2±11.6	<0.001
Waist circumference, cm	87.4±7.9	80.2±8.6	84.4±11.1	72.5±9.2	<0.001
BMI, kg/m^2	24.7±2.9	23.8±3.3	24.6±4.3	21.9±4.1	<0.001
FBG, mg/dL	107.1±23.7	99.5±21.0	92.3±16.1	88.8±10.1	<0.001
HbA_{1c}, %	5.9±0.8	5.7±0.7	5.4±0.6	5.3±0.3	0.009
TC, mg/dL	190.9±36.8	203.5±37.8	182.1±31.4	178.7±29.1	0.036
HDL cholesterol, mg/dL	46.9±10.7	55.0±12.3	50.0±11.5	58.8±12.1	<0.001
LDL cholesterol, mg/dL	114.4±33.3	124.8±34.4	110.3±27.6	103.8±25.7	<0.001
Triglyceride, mg/dL	162.4±125.5	120.9±77.1	121.0±91.9	82.1±50.1	<0.001
SBP, mm Hg	121.3±14.2	119.4±17.0	116.0±11.4	105.9±9.5	<0.001
DBP, mm Hg	79.8±9.2	76.7±9.7	75.2±9.2	69.8±8.0	<0.001
Total energy intake, kcal	2312.5±869.9	1654.8±625.4	2467.9±1100.3	1790.1±788.2	<0.001
Fat intake, g	48.6±32.2	37.0±23.6	69.5±48.5	51.9±34.3	<0.001
Protein intake, g	83.3±37.6	59.6±28.3	93.6±52.8	67.8±35.5	<0.001
Carbohydrate intake, g	336.8±125.2	266.1±109.4	327.4±137.2	247.9±107.2	<0.001
Sodium intake, g	4.2±2.2	2.9±1.7	4.1±2.3	2.9±2.0	<0.001
Fiber intake, g	30.9±15.8	26.8±15.0	24.9±15.4	18.7±9.5	<0.001
CVH score	3.2±1.4	3.9±1.4	4.3±1.4	5.3±1.1	<0.001
Nonideal BP	897 (69.7)	704 (54.5)	314 (41.8)	109 (12.2)	<0.001
Nonideal TC	671 (54.1)	824 (63.9)	216 (28.5)	169 (21.5)	0.003
Nonideal FBG	703 (54.8)	460 (34.9)	110 (13.8)	55 (6.2)	<0.001
Nonideal PA	671 (52.4)	697 (54.3)	259 (33.4)	355 (43.1)	<0.001
Nonideal BMI	599 (72.8)	692 (53.3)	469 (61.5)	229 (27.6)	<0.001
Nonideal smoking	386 (32.6)	18 (1.7)	294 (37.9)	48 (5.8)	<0.001
Nonideal diet	616 (47.8)	629 (49.1)	394 (53.4)	435 (53.1)	0.891
Nonideal CVH	1046 (82.4)	854 (65.9)	397 (51.3)	154 (18.8)	<0.001

Data presented as mean±standard, median (interquartile range), or number (percentage) of subjects. All values have been calculated reflecting sample weights.

BMI indicates body mass index; BP, blood pressure; CVH, cardiovascular health; DBP, diastolic blood pressure; FBG, fasting blood glucose; HbA_{1c}, glycated hemoglobin; HDL, high-density lipoprotein; LDL, low-density lipoprotein; PA, physical activity; SBP, systolic blood pressure; and TC, total cholesterol.

*Independent *t*-test or chi-square test for the difference between son and daughter.

DISCUSSION

This study investigated a relatively large number of Korean family trios to study the strength of parent–offspring associations regarding ideal CVH. Overall, in the clustering CVH, offspring with either parent having nonideal CVH were more likely to have nonideal CVH than the others; daughters whose fathers had nonideal CVH were most likely to have nonideal CVH. Our study showed positive but potentially differential associations of CVH and its component between parents and offspring. Parent–offspring BP, FBG, BMI, and diet were positively significant in all 4 pairs of parent–offspring relationships.

Several studies have demonstrated that cardiovascular risk factors can be inherited across

Table 2. Characteristics of the Offspring by Their CVH Status

Characteristics	Nonideal CVH (n=551)	Ideal CVH (n=1016)	*P* value*
Age, y	25.7 (22.6–29.9)	23.9 (21.6–27.2)	–
Sex			
Male	397 (77.5)	351 (43.1)	<0.001
Female	154 (22.5)	665 (56.9)	
Individual education attainment			
High school and lower	253 (48.1)	450 (47.3)	0.788
College graduation or higher	298 (51.9)	566 (52.7)	
Father's education attainment			
High school and under	343 (61.3)	549 (53.5)	0.005
College graduation or higher	204 (38.7)	467 (46.5)	
Mother's education attainment			
High school and lower	404 (72.1)	694 (67.8)	0.101
College graduation or higher	147 (27.9)	322 (32.2)	
Household income level			
Low	70 (13.4)	119 (11.8)	0.186
Middle	103 (19.5)	164 (16.1)	
Upper	378 (67.1)	732 (72.1)	
Alcohol consumption			
No	189 (33.4)	387 (36.6)	0.241
Yes	362 (66.6)	629 (63.4)	

Data presented as median (interquartile range), or number (percentage) of subjects. All values have been calculated reflecting sample weights.
CVH indicates cardiovascular health.
*Independent *t*-test or chi-square test.

generations,[13,19,26–31] highlighting the potential of parental nonideal CVH to predict nonideal CVH in their offspring.[10,18] However, there have been limited investigations into the sex-specific associations of CVH between parents and their children. The Framingham Heart Study examined the association of parental CVH with the time to onset of cardiovascular disease in offspring, reporting 4 parent–offspring relationships. They found that ideal CVH in parents was protective for their offspring's cardiovascular disease-free survival.[11] They reported a significant association between maternal CVH and the time to onset of cardiovascular disease in daughters.[11] In contrast, our study did not find a significant association between mothers' and daughters' CVH. It is possible that the inclusion of younger daughters (aged 20–40 years) in our study may have masked the relationship between mothers and daughters, as cardiovascular risk factors develop later in life and are more frequent in women. Even after including offspring aged at least 40 years and their parents, the mother–daughter CVH association remained insignificant in our study. Moreover, the Framingham Heart Study suggested a stronger association between mothers and offspring compared with fathers and offspring. However, in our analysis, we observed a greater strength of association (represented by the odds ratio) between fathers and their offspring's CVH compared with mothers and their offspring's CVH. Another study examining metabolic syndrome associations found a similarity between mother–offspring and father–offspring associations.[26] However, they employed differential statistical testing to draw their conclusions. The ongoing controversy surrounding this topic[11,26] necessitates further exploration using more robust research methodologies, such as prospective cohort or clinical trial studies. Despite the differences in findings, the strong familial correlation in CVH suggests the importance of involving the entire family in the prevention and management of cardiovascular disease. Health care professionals can adopt family-focused approaches in their practice, such as family-based counseling and support services that promote lifestyle modifications, including smoking cessation, regular exercise, and healthier eating habits. By incorporating these strategies into the broader health care strategy, we can potentially enhance the effectiveness of cardiovascular disease management on a population level.

Among the 7 metrics of CVH, our study discovered significant positive correlations of 4 pairs of parent–offspring, regarding BP, BMI, TC, FBG, BMI, and diet.

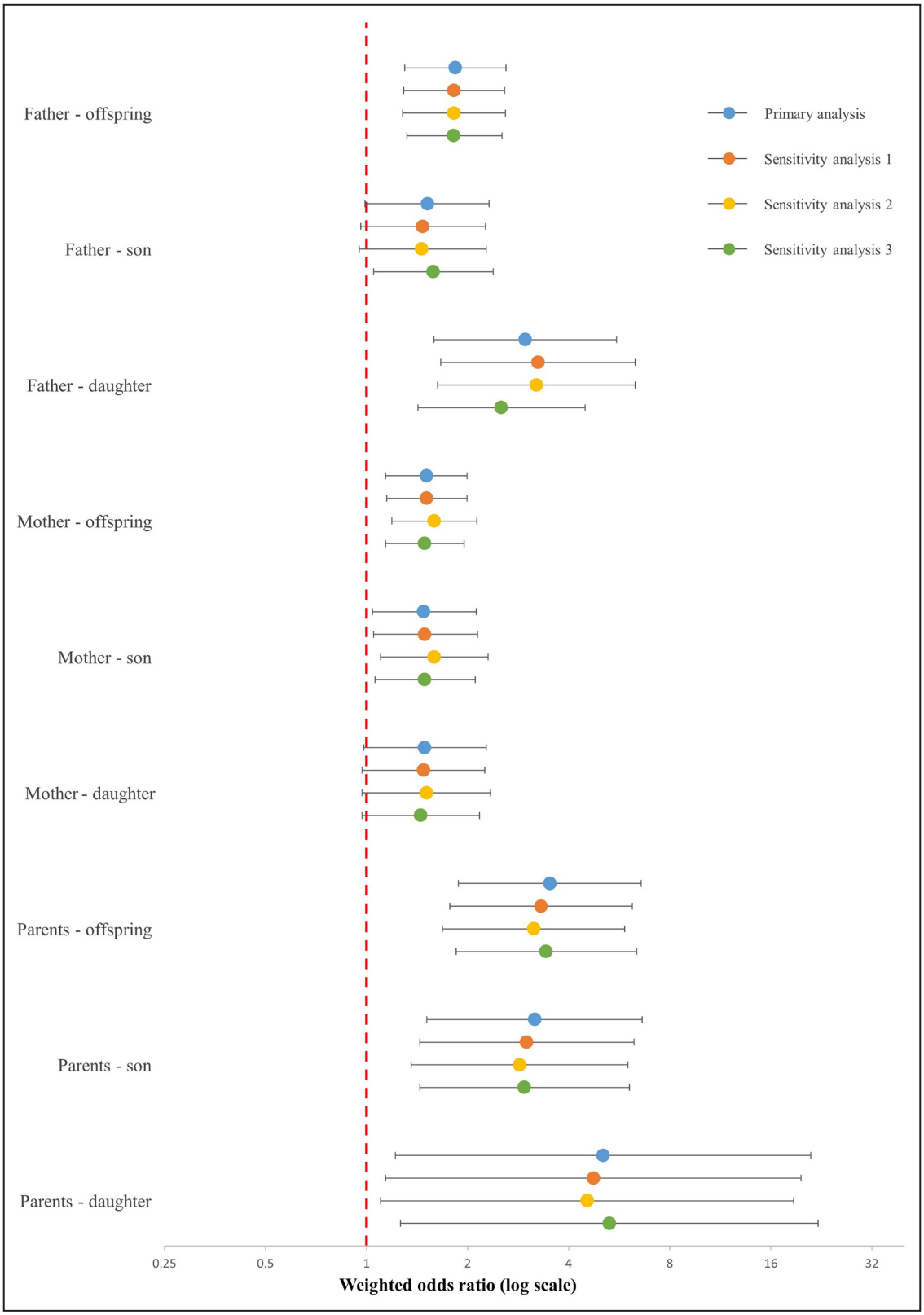
Table 3. Associations Between Parental and Offspring CVH

Parental CVH	Either son or daughter			Son			Daughter		
	No. of people	No. (%) of nonideal CVH	OR (95% CI)*	No. of people	No. (%) of nonideal CVH	OR (95% CI)†	No. of people	No. (%) of nonideal CVH	OR (95% CI)†
Father									
Ideal	277	73 (26.6)	1.00	125	57 (42.9)	1.00	152	16 (8.9)	1.00
Nonideal	1290	478 (39.2)	1.84 (1.30–2.60)	623	340 (52.9)	1.52 (0.99–2.32)	667	138 (21.1)	2.97 (1.59–5.55)
	P for interaction=0.077								
Mother									
Ideal	517	143 (29.9)	1.00	242	103 (42.4)	1.00	275	40 (14.5)	1.00
Nonideal	1050	408 (40.5)	1.51 (1.14–1.99)	506	294 (55.8)	1.48 (1.04–2.12)	544	114 (21.0)	1.49 (0.98–2.27)
	P for interaction=0.780								
Parents									
Both ideals	105	14 (15.8)	1.00	46	12 (26.3)	1.00	59	2 (4.9)	1.00
Either nonideal	1462	537 (38.6)	3.52 (1.88–6.58)	702	385 (53.0)	3.17 (1.51–6.63)	760	152 (20.0)	5.07 (1.22–21.08)
	P for interaction=0.598								

Descriptive data presented as number (percentage) of subjects. All values have been calculated reflecting sample weights.
CVH indicates cardiovascular health; and OR, odds ratio.
*Adjusted for household income, offsprings' sex, age, education level, and alcohol use.
†Adjusted for household income, offsprings' age, education level, and alcohol use.

There is growing scientific evidence supporting these results.[13–15,29,32–35] The association of father–daughter's smoking and mother–son's smoking were not statistically significant, but they were all positive. This is similar to a recent Finnish study that found a significant association between parental high-stable activity and greater levels of self-reported PA in adult offspring of both sexes.[36] Our study also found a higher association of smoking in sex-concordant pairs, which is consistent with a study in Amsterdam (odds ratio, 3.16 [95% CI, 2.12–4.51] in sex-concordant pairs versus 1.73 [95% CI, 1.15–2.59] in sex-discordant pairs; *P* for interaction=0.017).[37] In addition, our study did not show statistically significant results for father–daughter and mother–son associations. This might be because Korean women are more socially discouraged and restricted from smoking than men.[38]

Several pathways have been hypothesized as possible grounds for the transmission of phenotypes from parents to their children. On the one hand, high cholesterol, BP, BMI, and blood glucose are all risk factors for cardiovascular disease that are explained in part by heredity and environmental and behavioral variables.[27,39] Scientists have discovered several sex-specific genetic elements that are engaged in regulating certain metabolic syndrome features.[40–42] For example, adipose mitochondrial functions, which produce energy at the cellular level to support a variety of metabolic pathways, including triglyceride synthesis, gluconeogenesis, and fatty acid reesterification, are elevated in women and are strongly associated with adiposity, insulin resistance, and plasma lipids.[42] Recent studies have reported preliminary findings from metabolic impact of sex chromosomes.[43,44] A mouse study found that those with 2 X chromosomes displayed higher food intake, body weight, and increased adipose tissue content compared with mice with XY chromosomes. These effects may be influenced by X chromosome inactivation and differential genomic imprinting of genes inherited from both the mother and father.[43] On the other hand, the shared environment of offspring and parents is where offspring initially observe and learn about health behaviors. Role modeling of health behaviors by primary caregivers (fathers and mothers), particularly for young infants, has been proven to be a substantial contributor to the parent–offspring concordance of selected behaviors.[17,35,45] In addition, our study showed that there can be sex-specific adoption of parental behaviors, such as PA, eating behavior, and smoking, and this can also cause the differential association of clustering CVH that we found. For example, the higher odds ratio between mother and daughter in terms of smoking discovered in our analysis could reflect a stressful family environment, which could possibly put more burden on the women. Compared with the contribution of clinical metrics (Table 4), our finding suggests that



the shared environmental factors experienced by the offspring who were at least 20 years old and residing with their families during the survey may predominantly mediate the observed associations.[30]

Strengths and Limitations

This study had several limitations. First, because the study design was cross-sectional, caution should be exercised when interpreting causal relationships

Figure. Sensitivity analyses of association between parental cardiovascular health and offspring cardiovascular health. Estimates in all models were adjusted for household income, offspring's sex (except for models for son or daughter), age, education level, and alcohol use. Primary analysis indicates estimates from Table 3 for comparison with sensitivity analyses. Sensitivity analysis 1 indicates estimates from Table S2, where any patients with cardiovascular disease were considered as having nonideal cardiovascular health no matter what their cardiovascular health scores were. Sensitivity analysis 2 indicates estimates from Table S3, where we excluded patients with cardiovascular disease. Sensitivity analysis 3 indicates estimates from Table S4, where we did not exclude any trios whose offspring were aged at least 40 years.

from our findings. Second, it should be noted that information on PA and diet was gathered through a self-reported questionnaire, which could have exaggerated optimum diet and exercise habits because people are known to be overoptimistic when self-reporting such qualities.[46,47] Additionally, the criteria for a healthy diet were arbitrary and did not fully align with the AHA definition of an ideal diet. However, we adapted guidelines from a previous Korean study,[23] following the Dietary Approaches to Stop Hypertension framework,[48] to analyze the available KNHANES data. Third, although the major known covariates were carefully controlled in the model, the findings may be partly explained by unknown residual confounding factors, such as neighborhood health index, health care, and quality, which are not considered in our study. Fourth, the response rate of the KNHANES was ≈80%,[20] and there were some instances of missing data for key variables. This is an important factor to consider when interpreting the results of our study. Fifth, we found some statistically significant differences between excluded and included participants among quantifiable characteristics (Table S5); the selection bias caused by our choosing eligible participants and the limited number of mothers with nonideal smoking status may restrict the generalizability of the results; and future studies with larger sample sizes or longitudinal designs would be beneficial to gain more comprehensive insights into this aspect of the study. Finally, because there was no information on genetic markers and the offspring shared a household setting with their parents at the time of data collection, it was impossible to distinguish the exact contributions between genes and the environment. Further investigation is required in the future.

Despite these limitations, this study had several strengths. First, we explored the association of parent–offspring CVH as a composite among Korean families. These findings corroborate those of other studies and suggest that in the future, family-targeted interventions using CVH as an evaluation indicator can have a group impact on all family members,[10,11,16,26,29] particularly capturing the attention of health policy makers in South Korea. This prompts the need for enacting familial strategies aimed at achieving the goal of the National Health Plan for Cardiovascular Disease. Besides, general practitioners can use parental CVH as a part of cardiovascular disease risk assessment in patients. Second, the study population was derived from a large representative survey with a population-based design. A representative sample was drawn from a wide age-ranged population, and data were gathered through questionnaires and clinical tests. Outsourced data link the KNHANES to the family registry as well as death events, which allows researchers to analyze how familial CVH correlations impact cardiovascular disease incidence and death in the future.

This cross-sectional study showed positive and differential associations of CVH and its components between parents' and offspring's nonideal status. Our hypothesis-generating results highlight the relevance of using CVH as a composite indicator in family-centered screening or interventions.

Table 4. Odds Ratios From the Associations of Parental–Offspring Individual CVH Metrics

Nonideal status in CVH metrics	Father's CVH		Mother's CVH	
	Son's CVH	Daughter's CVH	Son's CVH	Daughter's CVH
Blood pressure	1.83 (1.26–2.66)	4.93 (2.48–9.79)	1.69 (1.2–2.38)	2.22 (1.35–3.65)
Total cholesterol	1.54 (1.07–2.22)	1.64 (1.12–2.39)	2.75 (1.74–4.33)	2.19 (1.44–3.34)
Fasting blood glucose	1.82 (1.12–2.97)	3.34 (1.54–7.22)	1.79 (1.11–2.88)	3.52 (1.83–6.78)
Physical activity	1.57 (1.12–2.19)	1.23 (0.91–1.66)	1.15 (0.82–1.61)	1.64 (1.21–2.23)
Body mass index	2.25 (1.59–3.17)	2.22 (1.44–3.42)	2.01 (1.45–2.80)	2.99 (2.09–4.26)
Smoking	1.93 (1.31–2.83)	0.93 (0.48–1.82)	1.6 (0.58–4.46)	7.48 (1.95–28.72)
Diet	1.41 (1.03–1.92)	1.81 (1.34–2.45)	1.43 (1.04–1.96)	1.65 (1.22–2.24)

Weighted odds ratios were all adjusted for household income, offspring's age, education level, and alcohol use.
CVH indicates cardiovascular health.

ARTICLE INFORMATION

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Disclosures

None.

Supplemental Material

Data S1.

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