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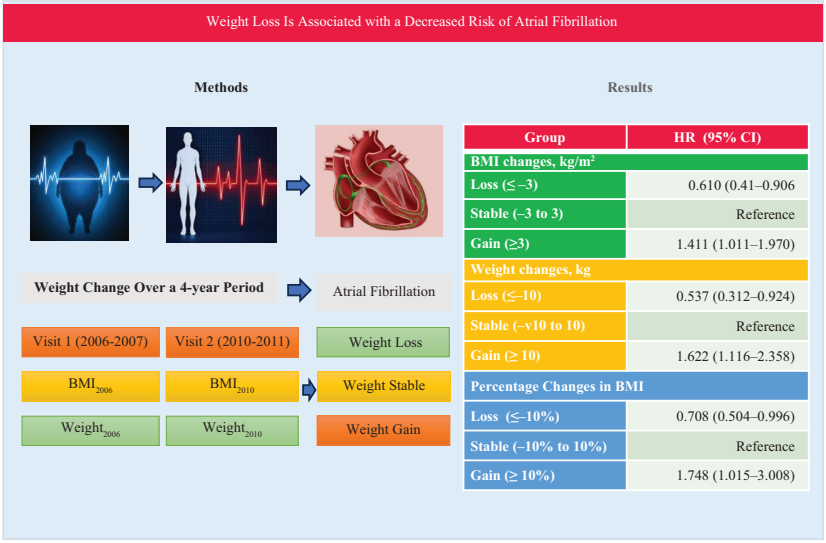
RESEARCH ARTICLE

Association Between Weight Change and the Risk of Atrial Fibrillation: Results from the Kailuan Cohort Study in China

Graphical Abstract



Highlights

- In the Kailuan cohort, 64,119 adults were followed for a median of 10 years.
- BMI loss ≤–3 kg/m² and weight loss ≤–10 kg were associated with lower incident AF risk.
- BMI gain ≥3 kg/m² and weight gain ≥10 kg were associated with higher AF risk.
- A dose–response pattern supports long-term weight management, especially among overweight or obese adults.

Authors

Hongmin Liu, Ying Liu, Oscar Hou In Chou, Gary Tse, Shouling Wu and Tong Liu

Correspondence

E-mail: liutong@tmu.edu.cn
or liutongdoc@126.com;
E-mail: drwusl@163.com

In brief

In this large Kailuan cohort study, long-term BMI and body weight loss were associated with a lower risk of incident atrial fibrillation, whereas weight gain was associated with a higher risk. These findings highlight the potential value of sustained weight management for atrial fibrillation prevention.

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Association Between Weight Change and the Risk of Atrial Fibrillation: Results from the Kailuan Cohort Study in China

Hongmin Liu[1,2,a], Ying Liu[1,a], Oscar Hou In Chou[3,4], Gary Tse[1,5,6], Shouling Wu[2] and Tong Liu[1]

[1]*Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular Disease, Department of Cardiology, Tianjin Institute of Cardiology, The Second Hospital of Tianjin Medical University, Tianjin 300211, China*
[2]*Department of Cardiology, Kailuan General Hospital, Tangshan 063001, China*
[3]*Division of Clinical Pharmacology and Therapeutics, Department of Medicine, School of Clinical Medicine, Li Ka Shing Faculty of Medicine, University of Hong Kong, Hong Kong SAR, China*
[4]*Diabetes Research Unit, Cardiovascular Analytics Group, PowerHealth Limited, Hong Kong SAR, China*
[5]*Kent and Medway Medical School, University of Kent and Canterbury Christ Church University, Canterbury, Kent, UK*
[6]*School of Nursing and Health Studies, Hong Kong Metropolitan University, Hong Kong, China*

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Abstract

Background: The long-term effects of sustained weight gain and loss on atrial fibrillation (AF) risk remain unclear. Herein, we examined associations between long-term weight change and incident AF.

Methods: We analyzed 64,119 AF-free participants from the Kailuan cohort whose height and weight were measured in 2006–2007 and 2010–2011. Weight change was assessed as changes in body mass index (BMI), body weight, and percentage weight changes. Incident AF was ascertained according to International Classification of Diseases codes and biennial electrocardiograms. Multivariable Cox models estimated hazard ratios (HRs) and 95% confidence intervals (CIs).

Results: Over a median 10.0 years' follow-up, 583 participants developed AF. Compared with stable BMI (−3 to 3 kg/m^2), BMI loss ≥3 kg/m^2 was associated with diminished AF risk (HR 0.61, 95% CI 0.41–0.91), whereas BMI gain ≥3 kg/m^2 was associated with elevated risk (HR 1.41, 95% CI 1.01–1.97). Similarly, weight loss ≥10 kg was associated with diminished AF risk (HR 0.54, 95% CI 0.31–0.92), whereas weight gain ≥10 kg was associated with elevated AF risk (HR 1.66, 95% CI 1.12–2.36). The findings were consistent across percentage-change definitions.

Conclusions: Long-term weight gain was associated with elevated incident AF risk, whereas sustained weight loss was associated with diminished risk, thus supporting the importance of long-term weight management for AF prevention.

Keywords: atrial fibrillation; weight change; body mass index; cohort study

[a]Hongmin Liu and Ying Liu contributed equally to this work.

Correspondence: **Tong Liu**, Department of Cardiology, Tianjin Key Laboratory of Ionic-Molecular Function of Cardiovascular Disease, Tianjin Institute of Cardiology, Second Hospital of Tianjin Medical University, No. 23 Pingjiang Road, Hexi District, Tianjin 300211, China, E-mail: liutong@tmu.edu.cn or liutongdoc@126.com; **Shouling Wu, PhD**, Department of Cardiology, Kailuan General Hospital, Tangshan 063001, China, E-mail: drwusl@163.com

Abbreviations: AF, atrial fibrillation; BMI, body mass index; BMI_{2006}, BMI at visit 1 (2006–2007); BMI_{2010}, BMI at visit 2 (2010–2011); CI, confidence interval; FBG, fasting blood glucose; HDL-C, high-density lipoprotein cholesterol; HR, hazard ratio; hs-CRP, high-sensitivity C-reactive protein; LDL-C, low-density lipoprotein cholesterol; $Weight_{2006}$, body weight at visit 1 (2006–2007); $Weight_{2010}$, body weight at visit 2 (2010–2011).

Introduction

The global burden of atrial fibrillation (AF) is rapidly increasing [1]. According to the Framingham Heart Study, the prevalence of AF in adults is currently estimated to be 2% to 4%, and has tripled over the past 50 years [2]. By 2050, at least 72 million individuals in Asia are projected to be diagnosed with AF, a number surpassing the combined total in Europe and the United States [3]. AF is a major cause of heart failure, stroke, and all-cause mortality [4, 5]. Recent studies have increasingly focused on modifiable risk factors to improve primary AF prevention. In parallel, machine learning and deep learning approaches have increasingly been applied to AF screening and risk prediction; however, those methods have been focused primarily on predictive performance rather than clarifying the long-term epidemiological associations between weight change and incident AF [6, 7].

Overweight and obesity are well-established causal risk factors for AF [8]. The Atherosclerosis Risk in Communities study identified obesity as the second leading risk factor for AF, after hypertension [9]. Several studies have shown that body mass index (BMI) change, particularly weight loss, might be associated with a decreased AF risk [10–12]. In the Swedish Obese Subjects cohort, substantial weight loss after bariatric surgery was associated with diminished AF incidence [13]. However, the evidence remains inconsistent. In community-based cohorts such as the HUNT and Atherosclerosis Risk in Communities studies, weight loss was not consistently associated with diminished AF risk and, in some analyses, was even associated with elevated risk [14, 15]. Therefore, in the general population, the relationship between weight change and AF might be more complex than that observed in clinical populations with obesity, and might partly reflect heterogeneity in study populations, adiposity measures, and the possibility of unintentional weight loss or reverse causation.

Overall, large-scale cohort studies on long-term weight change and incident AF remain limited, and previous studies have paid insufficient attention to sustained weight loss, direct comparisons between weight gain and weight loss, and potentially relevant BMI or weight-change thresholds for AF prevention. To address these gaps, we investigated the associations of obesity, BMI change, and body weight change with incident AF in the Kailuan cohort, and focused on whether sustained weight loss and weight gain might be associated with opposite directions of AF risk.

Methods

Study Population

The Kailuan Study is a large-scale prospective cohort study based in Tangshan, Hebei, conducted by the Kailuan Group Company. Between July 2006 and October 2007, a total of 101,510 adult participants from the Kailuan community were enrolled in the first survey. Since then, comprehensive health assessments have been conducted biennially, including measurement of height and body weight, and use of standardized questionnaires capturing data on demographics; medical comorbidities; medication history; lifestyle factors such as physical activity, smoking status, and alcohol consumption; and laboratory test results. The detailed study design has been extensively described in previous publications [16]. In this study, participants completed both visit 1 (2006–2007) and the follow-up visit 2 (2010–2011), during which height and weight were measured, thus yielding a total of 64,866 individuals. The flowchart illustrating the study design is shown in Figure 1. Briefly, after exclusion of participants who had AF at visit 2 (n = 402), had cancer at visit 2 (n = 344), or died between visit 1 and visit 2 (n = 1), a total of 64,119 participants were included in the final analysis. The study was approved by the Ethics Committee of

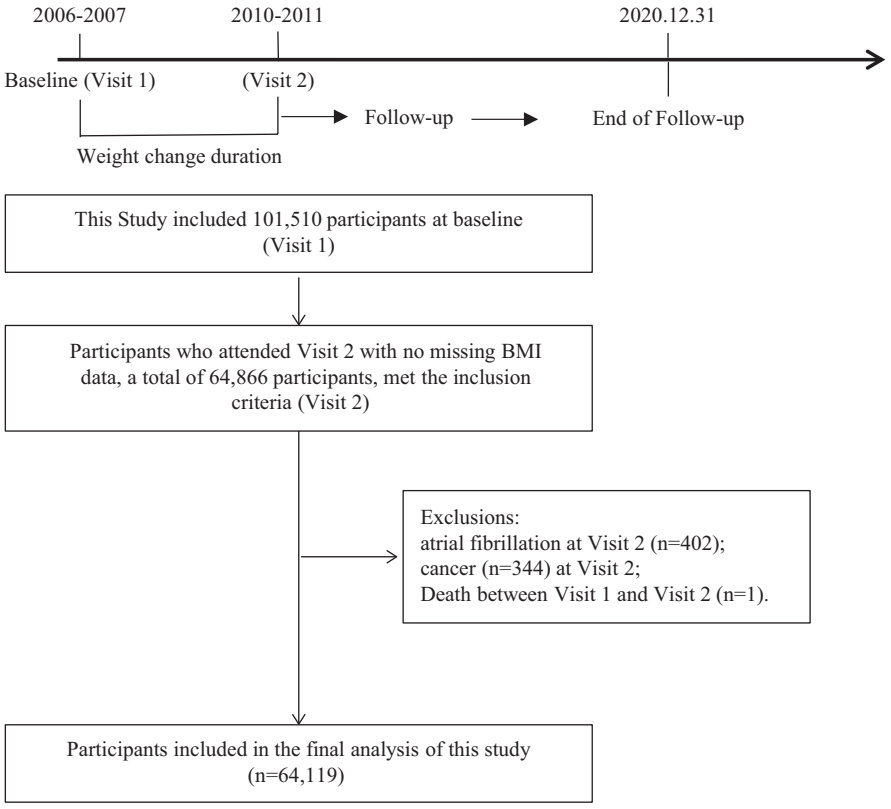

Figure 1 Flowchart of Study Participants.

Kailuan General Hospital ([2006] Medical Ethics Approval No. 5), and all participants provided informed consent to participate.

Assessment of Weight Change

In a clinical examination conducted by trained nurses, height and weight were measured with a calibrated RGZ−120 body weight scale, with participants barefoot and wearing light clothing. Height was recorded to the nearest 0.1 cm, and weight was recorded to the nearest 0.1 kg. BMI was calculated as the participants' weight divided by the square of their height (kg/m^2). BMI served as the primary reference index for weight loss, whereas body weight was considered a secondary measure.

BMI and body weight changes were calculated as follows:

$$\text{BMI}_{2006} = \text{BMI at visit 1 } (2006-2007)$$

$$\text{BMI}_{2010} = \text{BMI at visit 2 } (2010-2011)$$

$$\text{Weight}_{2006} = \text{body weight at visit 1 } (2006-2007)$$

$$\text{Weight}_{2010} = \text{body weight at visit 2 } (2010-2011)$$

$$\text{BMI change} = \text{BMI}_{2010} - \text{BMI}_{2006}$$

$$\text{Weight change} = \text{weight}_{2010} - \text{weight}_{2006}$$

The percentage weight loss was calculated with the following formula:

$$\text{Percentage of Weight Loss} = \left(\frac{\text{Weight changes}}{\text{Weight2006}}\right) \times 100\%$$

$$\text{Percentage of BMI Loss} = \left(\frac{\text{BMI changes}}{\text{BMI2006}}\right) \times 100\%$$

All participants were categorized into three groups according to their BMI changes: weight loss: BMI change ≤−3 kg/m^2; stable weight: −3 to 3 kg/m^2; and weight gain: BMI change ≥3 kg/m^2.

To further explore weight change within a narrower range, we classified body weight changes as follows: weight loss: body weight change ≤−10 kg; stable weight: body weight change ranging from −10 kg to 10 kg; weight gain**:** body weight change ≥10 kg.

Additionally, participants were stratified into loss, stable, and gain groups according to the percentage change in body weight and BMI over a 4-year period. A threshold of −10% was used to define clinically meaningful weight loss, in agreement with prior studies suggesting that a ≥10% weight reduction is associated with a significant decrease in AF burden among individuals with overweight/obesity and AF [5, 11]. This threshold allows for meaningful group comparisons and reflects a target frequently recommended in weight management interventions.

Ascertainment of Outcomes

The primary outcome of this study was new-onset AF. Potential AF cases were identified with International Classification of Diseases, Tenth Revision (ICD-10) codes, specifically code I48. Trained physicians subsequently reviewed the participants' medical records to confirm diagnostic information. Additionally, all participants underwent a 10-second 12-lead electrocardiogram during biennial health examinations. The diagnostic criteria for AF have been described in detail previously [17]. In brief, AF was diagnosed on the basis of the following ECG criteria: (1) irregular R-R intervals, (2) absence of repeating P waves, and (3) irregular atrial activity. Mortality data were obtained from the Kailuan Social Security Registry, containing data for all Kailuan employees enrolled in both social and medical insurance programs. Follow-up began after the second survey (2010–2011), and participants were monitored for the occurrence of new-onset AF as the primary endpoint. For participants who did not develop AF, follow-up continued until death or until December 31, 2020.

Assessment of Other Potential Covariates

The candidate baseline variables presented in Table 1 were selected according to their widespread availability and use in previous studies. Covariates were collected during the first survey (2006 to 2007), and included age; sex (male or female); snoring during sleep (never, occasionally, or frequently); fatty food intake (less than once/week, one to three times/week, or more than three times/week); smoking status; alcohol consumption; and the presence of hypertension, diabetes, myocardial infarction (MI), stroke, and medication use. Current smokers were defined as individuals who had smoked at least one cigarette per day on average during the past year or had quit smoking within the past year, whereas non-smokers included those who had never smoked or had been smoke-free for more than 1 year, as well as individuals who smoked less than one cigarette per day on average during the past year. Current alcohol consumers were defined as individuals who consumed alcohol one to three times per day, regardless of alcohol content. Physical activity was categorized into three groups. Regular exercise was defined as engaging in physical activity for at least 20 minutes per session, at least four times per week. Individuals who exercised fewer than four times per week were classified as occasional exercise, whereas those who exercised less than once per week were classified as inactive physical activity.

All participants underwent blood tests after overnight fasting at each physical examination. The blood samples were analyzed with an auto-analyzer (Hitachi 747, Hitachi, Tokyo, Japan) on the day of blood collection. The tested biochemical indicators included low-density lipoprotein cholesterol (LDL-C), high-density lipoprotein cholesterol (HDL-C), high-sensitivity C-reactive protein (hs-CRP), and fasting blood glucose (FBG).

Diabetes mellitus was diagnosed according to the American Diabetes Association criteria, comprising a fasting glucose concentration ≥7 mmol/L on two separate examinations, use of glucose-lowering medications, or self-reported diabetes [18]. Hypertension was defined as systolic blood pressure ≥140 mmHg or diastolic blood pressure ≥90 mmHg, any use of anti-hypertensive agents, or self-reported history of hypertension, as previously described [19].

Statistical Analysis

Baseline characteristics were summarized as proportions for categorical variables and as means with standard deviations (SD) or medians with interquartile ranges for continuous variables, as appropriate. Comparisons of continuous variables across groups stratified by changes in BMI were conducted with one-way analysis of variance or the Kruskal-Wallis test, whereas differences in categorical variables were assessed with the chi-square test. The cumulative incidence of AF events was estimated with

Table 1 Baseline Characteristics of All Participants, Stratified by BMI Change Category.

Variables	Total N = 64,119	Loss (BMI change ≤−3, kg/m²) N = 4319	Stable (BMI change >−3 to <3 kg/m²) N = 55,422	Gain (BMI change ≥3, kg/m²) N = 4378	P value
Age (years), mean ± SD	49.6 ± 11.9	50.3 ± 12.4	49.5 ± 11.8	49.3 ± 13.5	0.003
Male sex, n (%)	49,670 (77.5)	3309 (76.6)	43,231 (78.0)	3130 (71.5)	<0.001
Change duration (years), median (IQR)	3.96 (3.73–4.31)	3.92 (3.72–4.29)	3.95 (3.73–4.30)	4.06 (3.78–4.37)	<0.001
Physical activity, n (%)					<0.001
Inactive	5315 (8.61)	291 (6.97)	4707 (8.82)	317 (7.58)	
Occasional	47,839 (77.5)	3384 (81.1)	41,122 (77.1)	3333 (79.7)	
Regular	8567 (13.9)	500 (12.0)	7536 (14.1)	531 (12.7)	
Snoring, n (%)					<0.001
Never	39,802 (64.5)	2749 (65.8)	33,986 (63.7)	3067 (73.3)	
Occasional	13,742 (22.3)	839 (20.1)	12,219 (22.9)	684 (16.4)	
Frequent	8205 (13.3)	588 (14.1)	7186 (13.5)	431 (10.3)	
Current alcohol consumer, n (%)	10,804 (17.3)	565 (13.4)	9644 (17.9)	595 (14.2)	<0.0001
Current smoker, n (%)	18,690 (30.0)	1035 (24.6)	16,582 (30.7)	1073 (25.6)	<0.001
Fatty food, n (%)					<0.001
<1 time/week	5143 (8.34)	312 (7.47)	4537 (8.51)	294 (7.04)	
1–3 times/week	51,162 (82.9)	3589 (86.0)	44,026 (82.5)	3547 (84.9)	
>3 times/week	5393 (8.74)	274 (6.56)	4781 (8.96)	338 (8.09)	
Fasting blood glucose (mmol/L), mean ± SD	5.41 ± 1.57	5.62 ± 1.76	5.40 ± 1.53	5.41 ± 1.87	<0.001
Heart rate (beats/min), mean ± SD	73.4 ± 9.88	74.1 ± 10.11	73.3 ± 9.81	73.4 ± 10.4	<0.001
Low-density lipoprotein cholesterol (mmol/L), mean ± SD	2.30 ± 0.91	2.35 ± 0.89	2.31 ± 0.90	2.17 ± 0.98	<0.001
High-density lipoprotein cholesterol (mmol/L), mean ± SD	1.55 ± 0.40	1.58 ± 0.40	1.55 ± 0.40	1.57 ± 0.42	<0.001
hs-CRP (mg/L), median (IQR)	0.77 (0.29–2.16)	0.96 (0.38–2.69)	0.75 (0.29–2.10)	0.76 (0.25–2.60)	<0.000
Myocardial infarction, n (%)	658 (1.06)	43 (1.03)	582 (1.09)	33 (0.78)	0.326
Stroke, n (%)	1042 (1.63)	92 (2.13)	899 (1.62)	51 (1.16)	0.009
Hypertension, n (%)	26,436 (41.4)	2295 (53.4)	22,506 (40.8)	1635 (37.5)	<0.001
Diabetes mellitus, n (%)	7738 (12.1)	689 (16.0)	6515 (11.8)	534 (12.2)	<0.001
Anti-diabetic agents, n (%)	1348 (2.19)	131 (3.15)	1123 (2.11)	94 (2.25)	<0.001
Anti-hypertensive agents, n (%)	7097 (11.5)	634 (15.2)	6080 (11.4)	383 (9.11)	<0.001
Anti-lipidemic agents, n (%)	580 (0.90)	56 (1.30)	492 (0.89)	32 (0.73)	0.006

Abbreviations: BMI, body mass index; hs-CRP, high-sensitivity C-reactive protein; IQR, interquartile range; SD, standard deviation.

the Kaplan-Meier method, and differences between groups were evaluated with the log-rank test. AF incidence rates were expressed as the number of new cases per 10,000 person-years of follow-up.

Cox proportional hazards regression models were used to estimate hazard ratios (HRs) and 95% confidence intervals (CIs) for the association between weight change and AF risk. For the Cox regression models, parameters were estimated with partial likelihood methods, and the proportional hazards assumption for all covariates was assessed with the supremum test with 1000 resampling iterations in PROC PHREG. The details of the models were as follows:

Model 1 was adjusted for baseline BMI (for analyses of BMI change) or baseline body weight (for analyses of weight change), both assessed at visit 1 (2006–2007), as continuous variables.

Model 2 included additional adjustments for age (continuous), sex, smoking status (current or non-smoker), alcohol consumption (current or non-alcohol consumer), physical activity (inactive or active), fatty food intake (less than once/week, one to three times/week, or more than three times/week), snoring frequency (never, occasionally, or frequently), heart rate (continuous), FBG (continuous), LDL-C (continuous), HDL-C (continuous), hs-CRP (continuous), hypertension (yes or no), diabetes (yes or no), MI (yes or no), and stroke (yes or no), all assessed at visit 1 (2006–2007).

Model 3 was further adjusted for use of antihypertensive, lipid-lowering, and antidiabetic agents (yes or no), all assessed at visit 1 (2006–2007).

Model 4 was based on model 3, except that heart rate, FBG, LDL-C, HDL-C, and hs-CRP were modeled as concurrent changes between visit 1 and visit 2 rather than as values assessed at visit 1.

Furthermore, recognizing the effects of mortality on AF during follow-up, we performed a competing risk analysis for mortality with the Fine–Gray subdistribution hazard model. We performed the following sensitivity analyses to evaluate the robustness of our findings, as follows: (1) participants within the first 2 years of follow-up were excluded to minimize the potential for reverse causation; (2) participants who had experienced an MI at the second visit or during follow-up were excluded; (3) participants diagnosed with cancer during follow-up were also excluded; (4) to further address potential reverse causation associated with unintentional weight loss, participants with underweight (body mass index <18.5 kg/m^2), hypertension, diabetes, or estimated glomerular filtration rate <45 mL/min/1.73 m^2 at visit 1, as well as those with heart failure at visit 2, were excluded; and (5) baseline comorbidity burden was additionally defined as the number of major comorbid conditions present at visit 1, including hypertension, diabetes, MI, stroke, heart failure, and reduced kidney function, and the analyses were repeated after exclusion of participants with ≥2 or ≥3 baseline comorbidities.

Additional sensitivity analyses were performed by using alternative definitions of BMI change, including a narrower reference range (−1 to 1 kg/m^2), a five-category classification (≤−3; −3 to −1; −1 to 1; 1 to 3; and ≥3 kg/m^2), and percentage changes in BMI.

To better align potential confounders with the start of risk follow-up, we additionally performed a supplementary analysis in which all time-varying covariates were updated to visit 2, whereas sex and baseline body mass index (for BMI change analyses) or baseline body weight (for weight change analyses) was retained from visit 1.

Supplementary sensitivity analyses were additionally performed among 63,297 participants with repeated waist circumference measurements, in whom waist circumference change was used as an alternative marker of adiposity change. The association between waist circumference change and incident AF was examined with Cox regression models.

We conducted subgroup and interaction analyses stratified by age, sex, hypertension status, baseline overweight/obesity, and physical activity to assess whether these factors modified the association between weight loss and AF.

To assess the effects of baseline weight on AF, we categorized participants into four groups according to baseline BMI: underweight (BMI <18.5 kg/m^2), normal weight (18.5–24 kg/m^2), overweight (24–28 kg/m^2), or obesity (BMI ≥28 kg/m^2) [20]. Using the normal weight group as the reference, we analyzed AF risk across BMI categories.

Restricted cubic spline models with three knots at the 25th, 50th, and 75th percentiles were used to explore the patterns of associations between BMI and AF risk.

All potential covariates had <10% missing data (Supplementary Table 1). Because the proportion

of missing data for anti-lipidemic agents exceeded 5%, the missing indicator method was applied, and missing values were categorized as unknown.

All statistical analyses were performed in SAS 9.4 (SAS Institute; Cary, NC). Two-sided tests were conducted, and a $P < 0.05$ was considered statistically significant.

Results

Baseline Characteristics

The participants' baseline characteristics by BMI change category are presented in Table 1. The participants had an average age of 49.6 ± 11.9 years, and an age range of 18–98 years. A total of 49,670 (77.5%) participants were men.

Participants were categorized into three groups according to BMI change. The weight loss group (BMI change ≤−3 kg/m^2, N = 4319) showed a decrease in mean BMI from 28.45 ± 3.55 kg/m^2 in 2006 to 23.54 ± 3.27 kg/m^2 in 2010. The stable weight group (BMI change >−3 to <3 kg/m^2) exhibited minimal fluctuations in mean BMI, from 24.95 ± 3.25 kg/m^2 in 2006 to 25.00 ± 3.21 kg/m^2 in 2010. The weight gain group (BMI change ≥3 kg/m^2) experienced an increase in mean BMI from 23.22 ± 3.28 kg/m^2 in 2006 to 27.94 ± 3.60 kg/m^2 in 2010 (Supplementary Table 2).

The weight loss group, defined by a BMI change ≤−3 kg/m², had the highest baseline BMI, weight, age, FBG, HR, LDL-C, and hs-CRP among the three BMI change groups.

This group also had the highest proportions of participants with frequent snoring, stroke, hypertension, diabetes, and use of anti-diabetic, antihypertensive, and lipid-lowering medications among the three BMI change groups. In contrast, this group had the lowest proportions of smokers, alcohol consumers, and individuals with a high-fat diet, but the highest proportion of individuals engaging in occasional physical activity among the three groups (all $P < 0.01$, Table 1).

Associations of BMI and Weight Changes with Atrial Fibrillation Incidence and Risk

During a median follow-up of 10.0 years (interquartile range: 9.6–10.3 years), 583 participants were diagnosed with new-onset AF, and 6402 participants experienced all-cause mortality. The incidence density of new-onset AF progressively increased across the weight loss, stable weight, and weight gain groups. Specifically, the incidence densities were 7.62 (5.36–10.8), 9.54 (8.74–10.4), and 10.6 (7.92–14.3) per 10,000 person-years, respectively (Table 2). However, no significant difference was observed in cumulative AF incidence among the three groups, according to Kaplan-Meier analysis (Supplementary Figure 1). For the weight change categories, the incidence densities were 5.83 (3.45–9.85) per 10,000 person-years for the weight loss group (body weight ≤−10 kg), 9.54 (8.77–10.4) for the stable weight group (−10 to 10 kg), and 11.4 (8.09–16.0) for the weight gain group (body weight ≥10 kg). After adjustment for confounding covariates (model 3), weight loss, defined as BMI change ≤−3 kg/m², was associated with a 39.0% lower risk of AF than stable BMI change (>−3 to <3 kg/m²) (HR: 0.610, 95% CI: 0.410–0.906). Similarly, weight loss defined as body weight change ≤−10 kg was associated with a 46.3% lower risk of AF than stable body weight change (>−10 to <10 kg) (HR: 0.537, 95% CI: 0.312–0.924). In contrast, BMI gain ≥3 kg/m² was associated with a 41.1% higher risk of AF than stable BMI change (HR: 1.411, 95% CI: 1.011–1.970), and body weight gain ≥10 kg was associated with a 62.2% higher risk of AF than stable body weight change (HR: 1.622, 95% CI: 1.116–2.358) (Table 2).

We further analyzed the effects of percentage changes in BMI and body weight on AF risk. After adjustment for confounding covariates (model 3), with respect to the stable group, a 10% decrease in BMI was associated with a 29.2% lower AF risk (HR: 0.708, 95% CI: 0.504–0.996), whereas a 10% decrease in body weight was associated with a 44.8% lower AF risk (HR: 0.552, 95% CI: 0.354–0.861) (Table 3).

The proportional hazards assumption was generally satisfied in the main Cox model, and no evidence of violation for the main exposure variables in the supremum test was found (Supplementary Table 3).

Sensitivity Analysis

A series of sensitivity analyses were performed to examine the associations of weight changes with AF risk (Figure 2). The results remained consistent

Table 2 Association of Changes in BMI and Body Weight with Atrial Fibrillation Risk.

Group	Events/ total	Incidence rate (per 10,000 person-years)	Model 1 HR (95% CI)	Model 2 HR (95% CI)	Model 3 HR (95% CI)
*BMI change, kg/m2					
Loss (≤−3)	31/4319	7.62 (5.36–10.8)	0.570 (0.393–0.828)‡	0.605 (0.407–0.898)‡	0.610 (0.410–0.906)‡
Stable (−3 to 3)	508/55,422	9.54 (8.74 −10.4)	Reference	Reference	Reference
Gain (≥3)	44/4378	10.6 (7.92–14.3)	1.314 (0.963–1.793)	1.437 (1.034–1.998)‡	1.411 (1.011–1.970)‡
†Weight change, kg					
Loss (≤−10)	14/2544	5.83 (3.45–9.85)	0.459 (0.268–0.787)‡	0.533 (0.309–0.917)‡	0.537 (0.312–0.924)‡
Stable (−10 to 10)	536/58,476	9.54 (8.77–10.4)	Reference	Reference	Reference
Gain (≥10)	33/3099	11.4 (8.09–16.0)	1.331 (0.936–1.895)	1.614 (1.111–2.346)‡	1.622 (1.116–2.358)‡

Abbreviations: BMI, body mass index; BMI change, $BMI_{2010} - BMI_{2006}$; weight change, $weight_{2010} - weight_{2006}$; HR, hazard ratio; CI, confidence interval.

*Model 1 was adjusted for BMI at baseline (2006).

†Model 1 was adjusted for body weight at baseline (2006).

Model 2 was further adjusted for age (continuous), sex, smoking status (current or non-smoker), alcohol consumption (current or non-alcohol consumer), physical activity (inactive or active), fatty food intake (<1 time/week, 1–3 times/week, or >3 times/week), snoring frequency (never, occasionally, or frequently), heart rate (continuous), fasting blood glucose (continuous), low-density lipoprotein cholesterol (continuous), high-density lipoprotein cholesterol (continuous), high-sensitivity C-reactive protein (continuous), hypertension (yes or no), diabetes (yes or no), myocardial infarction (yes or no), and stroke (yes or no), and was based on model 1.

Model 3 was further adjusted for anti-diabetic agents (yes or no), anti-hypertensive agents (yes or no), and lipid-lowering agents (yes or no), and was based on model 2.

‡$P < 0.05$.

after exclusion of participants within the first 2 years of follow-up, as well as those with MI at the second visit or during follow-up. Additionally, exclusion of participants with cancer during follow-up did not affect the findings. To account for the competing risk of mortality, we used a competing risk model to evaluate the relationship between weight loss and AF. The results were similar to the main findings (all P for trend <0.01).

In Model 4, replacement of baseline heart rate, FBG, LDL-C, HDL-C, and hs-CRP with their concurrent changes between visit 1 and visit 2 did not materially alter the associations of BMI change or weight change with incident AF (Supplementary Table 4).

To further address potential reverse causation associated with unintentional weight loss, we excluded participants with underweight, hypertension, diabetes, or an estimated glomerular filtration rate <45 mL/min/1.73 m^2 at visit 1, as well as those with heart failure at visit 2. We also repeated the analyses after exclusion of participants with a high baseline comorbidity burden (≥2 or ≥3 major comorbid conditions). Across these analyses, the inverse association between BMI loss and incident AF remained materially unchanged, with HRs ranging from 0.600 to 0.720 and all trend tests remaining statistically significant (Supplementary Table 5).

The results were also generally robust to alternative BMI change definitions. A more pronounced inverse association was observed for substantial BMI loss, whereas moderate BMI changes showed no clear association with AF risk (Supplementary Tables 6 and 7). In the supplementary analysis with covariates assessed at visit 2 (2010–2011), the overall association pattern remained consistent with that in the main analysis, thus further supporting the robustness of the findings (Supplementary Table 8).

In a supplementary analysis of 63,297 participants with repeated waist circumference measurements,

Table 3 Associations of Percentage Changes in BMI and Body Weight with Risk of New-Onset Atrial Fibrillation.

	Percentage changes in anthropometric measures			
	Loss	Stable	Gain	P for trend
Percentage change in BMI	≤−10%	−10% to 20%	≥20%	-
Events/total	45/5490	522/57,087	16/1542	-
Incidence rate (per 10,000 person-years)	8.70 (6.50–11.7)	9.52 (8.74–10.4)	11.1 (6.77–18.0)	-
Model 1*	0.723 (0.530–0.988)‡	Reference	1.603 (0.968–2.656)	0.022
Model 2	0.701 (0.498–0.985)‡	Reference	1.717 (0.998–2.955)	0.017
Model 3	0.708 (0.504–0.996)‡	Reference	1.748 (1.015–3.008)‡	0.017
Percentage change in weight	≤−10%	−10% to 30%	≥30%	-
Events/total	26/3981	549/59,529	8/609	-
Incidence rate (per 10,000 person-years)	6.93 (4.72–10.2)	9.61 (8.84–10.4)	14.4 (7.21–28.8)	-
Model 1†	0.605 (0.406–0.900)‡	Reference	2.049 (1.013–4.144)‡	0.006
Model 2	0.548 (0.352–0.855)‡	Reference	2.272 (1.066–4.840)‡	0.003
Model 3	0.552 (0.354–0.861)‡	Reference	2.261 (1.061–4.817)‡	0.003

*Model 1 was adjusted for BMI in 2006.
†Model 1 was adjusted for weight in 2006.
Model 2 was further adjusted for age (continuous), sex, smoking status (current or non-smoker), alcohol consumption (current or non-alcohol consumer), physical activity (inactive or active), fatty food intake (<1 time/week, 1–3 times/week, or >3 times/week), snoring frequency (never, occasionally, or frequently), heart rate (continuous), fasting blood glucose (continuous), low-density lipoprotein cholesterol (continuous), high-density lipoprotein cholesterol (continuous), high-sensitivity C-reactive protein (continuous), hypertension (yes or no), diabetes (yes or no), myocardial infarction (yes or no), and stroke (yes or no), and was based on model 1.
Model 3 was further adjusted for anti-diabetic agents (yes or no), anti-hypertensive agents (yes or no), and anti-lipidemic agents (yes or no), and was based on model 2.
‡$P < 0.05$.

the overall pattern remained consistent with that in the main analysis. Compared with stable waist circumference, waist circumference loss was associated with a lower risk of incident AF (HR 0.795, 95% CI 0.640–0.987; Supplementary Table 9).

Subgroup Analysis

In the subgroup analysis, the associations between weight loss and AF risk remained consistent across key clinical subgroups, including individuals ≥60 years of age, men, individuals with overweight/obesity at baseline, and individuals with hypertension. Compared with the stable group (BMI change: −3 to 3 kg/m^2), weight loss (BMI decrease ≥3 kg/m^2) was associated with a diminished AF risk, with HRs of 0.525 (95% CI: 0.280–0.983), 0.565 (95% CI: 0.359–0.890), 0.595 (95% CI: 0.393–0.901), and 0.553 (95% CI: 0.341–0.897) across the respective subgroups. In contrast, weight gain (BMI increase ≥3 kg/m^2) was associated with an elevated AF risk, with HRs of 1.633 (95% CI: 1.049–2.543), 1.497 (95% CI: 1.042–2.152), 1.120 (95% CI: 0.666–1.885), and 1.127 (95% CI: 0.695–1.828), respectively. Additionally, in individuals without overweight/obesity (BMI <24 kg/m^2) at baseline, weight gain (BMI increase ≥3 kg/m^2) was associated with a significantly elevated AF risk (HR: 1.731, 95% CI: 1.103–2.717). Similarly, among individuals without hypertension, weight gain was associated with elevated AF risk with respect to the stable group (HR: 1.832, 95% CI: 1.150–2.917). No significant interaction was observed between age, sex, baseline overweight/obesity status, or hypertension and the association of weight loss with AF risk (P for interaction >0.05, Figure 3).

We conducted a detailed analysis of the effects of BMI changes on AF risk across various physical activity levels (Supplementary Table 10). In both the inactive and regularly active groups, the number

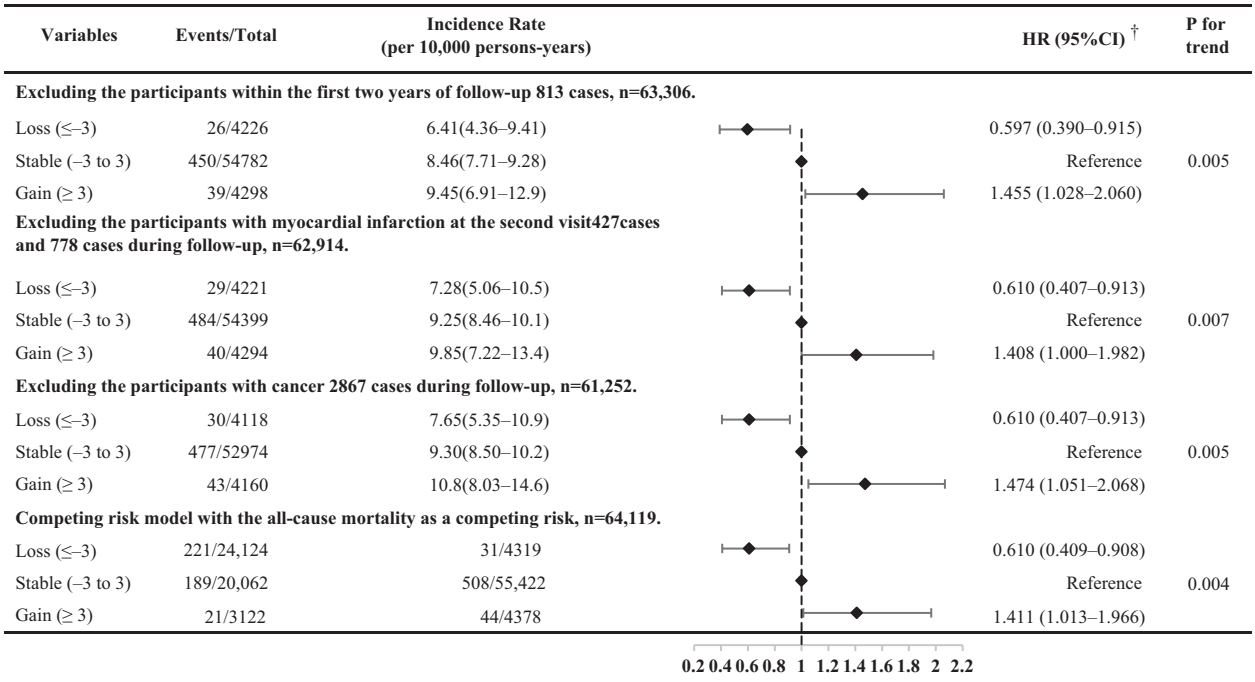

Figure 2 Sensitivity Analysis: Association Between Weight Loss and Atrial Fibrillation Risk.
†Model was adjusted for BMI in 2006, age, sex, smoking status, alcohol consumption, physical activity, fatty food intake, snoring, heart rate, fasting blood glucose, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, high-sensitivity C-reactive protein, hypertension, diabetes, myocardial infarction, stroke, anti-diabetic agents, anti-hypertensive agents, and anti-lipidemic agents.

of AF events in the weight loss and weight gain groups was fewer than ten cases. However, among individuals with occasional physical activity, the risk of AF was significantly lower in the weight loss group than the stable group, with an HR of 0.559 (95% CI: 0.345–0.903).

Dose–Response Relationship Between BMI and Atrial Fibrillation Risk

When BMI change was modeled as a continuous variable in a multivariable Cox regression model, each 1-unit increase in BMI change was associated with a 6% higher AF risk (HR: 1.060; 95% CI: 1.018–1.103). Additionally, for each 1-SD increase in BMI change, AF risk increased by 14.6% (HR: 1.146; 95% CI: 1.044–1.258). Beyond BMI changes, we also examined the effects of baseline BMI in 2006 on AF risk. Participants were categorized into underweight, normal weight, overweight, and obese groups by baseline BMI. With respect to normal weight, the adjusted HRs for incident AF were 0.364 (95% CI: 0.090–1.472) in the underweight group, 1.239 (95% CI: 0.993–1.546) in the overweight group, and 1.672 (95% CI: 1.295–2.159) in the obese group (Supplementary Table 11).

In restricted cubic spline analyses, higher BMI (2006 and 2010) and greater BMI change were linearly associated with elevated AF risk (all P for overall association <0.05; all P for nonlinearity >0.1), thus indicating no evidence of a nonlinear relationship (Supplementary Figure 2).

Discussion

In this large prospective cohort study, weight loss was significantly associated with diminished risk of incident AF, whereas weight gain was associated with elevated risk, particularly among individuals with normal baseline weight. The inverse association of weight loss with AF risk was most evident in participants with overweight or obesity at baseline, as well as in older adults, men, and individuals with hypertension. A clear dose–response relationship was observed between both baseline BMI and BMI change and AF risk. Together, these findings suggested that the relationship between weight change and incident AF is not uniform, but depends on both baseline adiposity and the direction of weight change. In addition, both

Subgroups	BMI Change, kg/m²	Events/Total	Incidence Rate (per 10,000 persons-years)	HR (95%CI) †	P for interaction
Age, years					
< 60	Loss (≤-3)	17/3454	5.05 (3.14–8.12)	0.720 (0.432–1.201)	0.120
	Stable (-3 to 3)	255/45,865	5.66 (5.01–6.40)	Reference	
	Gain (≥ 3)	19/3533	5.54 (3.53–8.68)	1.206 (0.722–2.015)	
≥ 60	Loss (≤-3)	14/865	20.0 (11.8–33.7)	0.525 (0.280–0.983) *	
	Stable (-3 to 3)	253/9557	30.7 (27.2–35.8)	Reference	
	Gain (≥ 3)	25/845	35.6 (24.0–52.6)	1.633 (1.049–2.543) *	
Sex					
Female	Loss (≤-3)	7/1010	7.20 (3.43–15.1)	0.788 (0.346–1.792)	0.328
	Stable (-3 to 3)	81/12191	6.79 (5.46–8.44)	Reference	
	Gain (≥ 3)	6/1248	4.93 (2.22–11.0)	1.002 (0.429–2.338)	
Male	Loss (≤-3)	24/3309	7.76 (5.20–11.6)	0.565 (0.359–0.890) *	
	Stable (-3 to 3)	427/43,231	10.3 (9.40–11.4)	Reference	
	Gain (≥ 3)	38/3130	13.0 (9.48–17.9)	1.497 (1.042–2.152) *	
Baseline Overweight/Obesity Status (BMI ≥ 24 kg/m²)					
No	Loss (≤-3)	2/384	5.57 (1.39–22.3)	0.678 (0.166–2.766)	0.135
	Stable (-3 to 3)	144/21,935	6.83 (5.80–8.04)	Reference	
	Gain (≥ 3)	27/2772	10.3 (7.09–15.1)	1.731 (1.103–2.718) *	
Yes	Loss (≤-3)	29/3935	7.82 (5.44–11.3)	0.595 (0.393–0.901) *	
	Stable (-3 to 3)	364/33,487	11.3 (10.2–12.5)	Reference	
	Gain (≥ 3)	17/1606	11.2 (6.94–18.0)	1.120 (0.666–1.885)	
Hypertension					
No	Loss (≤-3)	10/2005	5.18 (2.79–9.62)	0.720 (0.360–2.766)	0.212
	Stable (-3 to 3)	190/32,708	5.96 (5.17–6.87)	Reference	
	Gain (≥ 3)	22/2721	8.39 (5.53–12.7)	1.832 (1.150–2.917) *	
Yes	Loss (≤-3)	21/2295	9.92 (6.47–15.2)	0.553 (0.341–0.897) *	
	Stable (-3 to 3)	315/22,506	14.9 (13.3–16.6)	Reference	
	Gain (≥ 3)	22/1635	14.7 (9.71–22.4)	1.127 (0.695–1.828)	

Figure 3 Associations of Weight Loss and Atrial Fibrillation Risk, Stratified by Age, Sex, Overweight/Obesity, and Hypertension.

†Model was adjusted for BMI in 2006, age, sex, smoking status, alcohol consumption, physical activity, fatty food intake, snoring, heart rate, fasting blood glucose, low-density lipoprotein cholesterol, high-density lipoprotein cholesterol, high-sensitivity C-reactive protein, hypertension, diabetes, myocardial infarction, stroke, anti-diabetic agents, anti-hypertensive agents, and anti-lipidemic agents.

*P < 0.05 for comparison with the stable weight group.

baseline BMI and BMI change showed clear dose–responsive associations with incident AF. Overall, these findings support the importance of long-term weight management in AF prevention.

Previous studies have established obesity and weight gain as important risk factors for AF, in agreement with our findings. In contrast, evidence regarding the benefit of weight loss has been less consistent. Cohort studies have shown that maintaining an ideal BMI is associated with diminished AF risk [21], and bariatric surgery-induced weight loss has been associated with diminished AF incidence in individuals with obesity [13, 22]. However, whether weight loss in the general population confers long-term protection against incident AF has remained unclear. Our study extends the existing literature by demonstrating that, in a large community-based cohort, sustained decreases in BMI and body weight were associated with decreased AF risk, and that an approximately 10% weight reduction might be a clinically meaningful target [23].

Notably, the association between weight change and AF differed by baseline weight status. Among individuals with overweight or obesity at baseline, weight loss was associated with decreased AF risk, thus supporting weight reduction as a potentially effective preventive strategy in this population. In contrast, among participants with normal baseline weight, weight gain was associated with increased AF risk, thus suggesting that prevention of upward weight drift might also be important. Our findings suggested that the implications of weight change should be interpreted in the context of baseline adiposity rather than in isolation.

Subgroup analyses further suggested potentially enhanced benefits of weight loss in high-risk populations, including older adults and those with hypertension. These groups are relatively likely to have accumulated atrial remodeling and to have elevated baseline susceptibility to AF, thus potentially making the effect of weight reduction more detectable. We also observed a stronger inverse association in men than women, in line with previous studies showing a stronger relationship between elevated BMI and AF risk in men [24, 25]. Possible explanations include a higher prevalence of obstructive sleep apnea and more severe adiposity-related atrial fibrosis in men than women [11, 26, 27]. The statistically significant association observed only in the occasional physical activity subgroup should not be overinterpreted. Given the relatively small numbers of events in the inactive and regularly active subgroups, the absence of statistical significance in these strata might reflect limited statistical power rather than true heterogeneity in the effect of weight loss. In addition, no significant interaction was observed between weight change and physical activity subgroup. Nevertheless, these subgroup findings should be interpreted cautiously and confirmed in future studies.

The dose–response relationship observed herein further supports the biological relevance of weight control in AF prevention. Obesity has been identified as a major AF risk factor [28–30], and Mendelian randomization analyses have suggested a causal link between higher BMI and AF [31]. In our study, both higher baseline BMI and greater BMI increase were associated with progressively higher AF risk, thus indicating that the association is graded rather than threshold dependent. This pattern suggests that even modest weight improvement might be beneficial, whereas progressive weight gain might incrementally increase AF risk.

The pathophysiological mechanisms linking obesity and AF are complex and not yet fully elucidated. Several potential pathways have been proposed. First, obesity might contribute to the development of AF by inducing structural remodeling of the left atrium [28, 32, 33]. Second, abundant epicardial adipose tissue, frequently observed in individuals with obesity, might directly alter the atrial electrophysiological substrate by locally modulating autonomic tone, releasing pro-fibrotic adipokines, and promoting oxidative stress pathways implicated in AF pathogenesis [34, 35]. Third, obesity is associated with a state of chronic low-grade systemic inflammation, which has been independently associated with increased AF risk [2, 36]. Additionally, weight gain often coexists with the development of cardiovascular comorbidities, such as hypertension and heart failure, which further elevate AF risk. Notably, weight loss has been associated with improvements in diastolic function, decreases in systemic inflammatory markers, and decreases in pericardial fat volume, all of which might contribute to the beneficial effects of weight reduction on AF risk [11]. Collectively, these systemic and local changes might drive atrial structural remodeling, including fibrosis and left atrial enlargement, as well as electrical remodeling, thus ultimately promoting AF initiation and maintenance. Therefore, the beneficial association of weight loss observed in our study is biologically plausible and might reflect the combined reversal of both systemic and local atrial stressors that contribute to AF development.

Strengths and Limitations

This study has several strengths. First, it was based on a large prospective cohort with long-term follow-up, and it had sufficient statistical power to evaluate incident AF. The assessment of weight change over a 4-year interval before AF follow-up strengthened the temporal relationship between exposure and outcome. In addition, our evaluation of both baseline BMI and longitudinal BMI/body weight change enabled assessment of stratified and dose–response associations, and it adjusted for a broad range of potential confounders.

Several study limitations should also be acknowledged. First, because of the observational study design, causality cannot be established. Although we adjusted for multiple confounders, potential residual confounding remains possible. Unintentional weight loss might partly reflect frailty or occult disease. However, additional sensitivity analyses excluding participants with elevated risk of reverse causation yielded similar results, thereby supporting the robustness of the observed association.

Second, the study population was drawn primarily from urban communities in northern China and consisted predominantly of men, thus potentially limiting the generalizability of the findings. In addition, the number of underweight participants (BMI <18.5 kg/m^2) and AF events in this subgroup was very small, and no underweight participants were classified in the weight-loss group. Therefore, we were unable to reliably evaluate whether further weight loss in underweight individuals is associated with increased AF risk, and our findings should not be generalized to this population.

Third, asymptomatic AF might have been missed, thus potentially leading to underestimation of incidence. However, such misclassification would have been likely to bias the results toward the null.

Conclusion

In conclusion, weight change was significantly associated with incident AF in this large prospective cohort. Sustained weight loss, particularly among individuals with overweight or obesity at baseline, was associated with diminished AF risk, whereas weight gain among those with normal baseline weight was associated with elevated risk. The association was most pronounced in older adults, men, and participants with hypertension, and both baseline BMI and BMI change showed clear dose–response relationships with AF risk. These findings support long-term weight management as a potentially important strategy for AF prevention.

Data Availability Statement

The data supporting the findings of this study are available on request from the corresponding author. Additionally, the codebook and analytical code are available on request.

Ethics Statement

This study was conducted in accordance with the ethical principles of the Declaration of Helsinki and was reviewed and approved by the Ethics Committee of Kailuan General Hospital (approval No. [2006]5). Written informed consent was obtained from all participants before their inclusion in the study.

Author Contributions

HML and YL: Conceptualization, Data curation, Formal analysis, Writing—original draft. OHIC: Methodology, Validation, Writing—review & editing. GT: Supervision, Project administration, Writing—review & editing

SLW: Resources, Investigation, Data collection. TL: Supervision, Funding acquisition, Writing—review & editing. All authors read and approved the final manuscript.

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Conflicts of Interest

Tong Liu is the Deputy Editor-in-Chief of Cardiovascular Innovations and Applications. Tong Liu was not involved in the peer review, editorial evaluation, or decision to accept this manuscript for publication. The other authors declare no conflicts of interest.

Clinical Trial Number

Not applicable.

Consent for Publication

Not applicable. The Kailuan Study uses anonymized cohort data, and no individually identifiable information is included in this article.

Supplementary Material

Supplementary materials for this paper can be found at the following links:

https://cvia-journal.org/wp-content/uploads/2026/05/CVIA_521_SUPPLEMENTARY_FIGURES.pdf

https://cvia-journal.org/wp-content/uploads/2026/05/supplementary_tables.docx.

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