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ORIGINAL RESEARCH

Habitual Night Eating Was Positively Associated With Progress of Arterial Stiffness in Chinese Adults

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BACKGROUND: Night eating has been associated with an elevated risk of obesity, dyslipidemia, and cardiovascular disease. However, there is no longitudinal study on whether habitual night eating, regardless of diet quality and energy intake, is associated with arterial stiffness, a major etiological factor in the development of cardiovascular disease.

METHODS AND RESULTS: The study included 7771 adult participants without cardiovascular disease, cancer, or diabetes mellitus prior to dietary assessment by a validated food frequency questionnaire in 2014 through 2015. Participants were categorized into 3 groups based on self-reported night-eating habits: never or rarely, some days (1–5 times per week), or most days (6+ times per week). Arterial stiffness was assessed by brachial-ankle pulse wave velocity at baseline and repeatedly during follow-ups. Mean differences and 95% CIs in the yearly change rate of brachial-ankle pulse wave velocity across the 3 groups were calculated, adjusting for age, sex, socioeconomic status, total energy intake, diet quality, sleep quality, and other cardiovascular disease risk factors. At baseline, 6625 (85.2%), 610 (7.8%), and 536 (6.9%) participants reported night eating as never or rarely, some days, or most days, respectively. During a mean 3.19 years, we observed a positive association between night-eating frequency and progression of arterial stiffness (*P* trend=0.01). The adjusted difference in brachial-ankle pulse wave velocity change rate between the group that ate at night most days and the group that never or rarely ate at night was 14.1 (95% CI, 0.6–27.5) cm/s per year. This association was only significant in women, but not in men (*P* interaction=0.03).

CONCLUSIONS: In an adult population free of major chronic diseases, habitual night eating was positively associated with the progression of arterial stiffness, a hallmark of arteriosclerosis and biological aging.

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Key Words: arterial stiffness ■ meal timing ■ night eating ■ pulse wave velocity

The association between individual dietary components, as well as overall dietary patterns, and cardiovascular health has been well-documented.[1] Eating patterns, such as eating late at night, have been associated with higher odds of having cardiovascular risk factors, such as increased visceral adiposity, obesity, and dyslipidemia.[2] In a prospective observational study, men with night-eating habits had a higher risk of developing coronary heart disease, relative to men who did not eat late at night.[3] The underlying mechanism connecting night eating and coronary heart disease might involve higher energy intake, change in appetite, and disturbed circadian clock.[4] Currently, literature on habitual night eating is sparse, and no prospective population study has examined the association between night eating and preclinical,

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CLINICAL PERSPECTIVE

What Is New?

- The first large-scale, prospective population study on the association between night eating and preclinical cardiovascular disease.
- Habitual night eating was associated with more rapid progression of arterial stiffness in women.
- This association was independent of total energy consumption, diet quality, insomnia, menopausal status, and other major risk factors for cardiovascular disease.

What Are the Clinical Implications?

- Night eating, independent of other lifestyle behaviors, plays a critical role in the prevention of chronic disease.
- Potential underlying biological and physiological mechanisms are warranted in future studies.
- Guidelines may consider recommendation for timing of meals in addition to nutrients, foods, and meal patterns.

Nonstandard Abbreviations and Acronyms

baPWV	brachial-ankle pulse wave velocity
DASH	Dietary Approaches to Stop Hypertension
FFQ	food frequency questionnaire

pathological changes in cardiovascular disease (CVD), such as arterial stiffness.

Arterial stiffness characterizes pathological changes in the artery wall, including an increase in wall thickness and lumen diameter, and a decrease in elasticity and resilience.[5] It is a significant predictor and preclinical sign of CVD, with prognostic values independent of traditional CVD risk factors.[6] Brachial-ankle pulse wave velocity (baPWV) is a common measure for arterial stiffness, especially in large-scale cohort studies in the Asian population.[7] It has been validated against carotid-femoral PWV, the gold standard of arterial stiffness measurement, and has been found to have predictive value for CVD.[8–10] Our aim was to determine, longitudinally, the relationship between habitual night eating and progression of arterial stiffness among ≈8000 adults without major chronic diseases (ie, CVD, diabetes mellitus, and cancer), adjusting for overall diet quality, sleep parameters, and other potential confounders.

METHODS

Data used herein and analytic code will be made available from the corresponding author upon reasonable request and approval.

Study Population

The analysis was based on 2 ongoing, population-based cohorts in Tangshan City, China: the Kailuan study I and the Kailuan study II. A detailed description of this cohort can be found elsewhere.[7,11] Briefly, the Kailuan study I spanned from 2006 through 2007 and included 101 510 participants; the Kailuan study II ranged from 2008 to 2010, and included 35 865 participants. Participants from both cohorts were followed biannually. This study was approved by the Ethics Committee of the Kailuan Medical Group, Kailuan Company, Tangshan, China. All participants provided their written informed consent. At baseline and at each follow-up visit, physical examinations and laboratory analyses were performed, anthropometric measures were assessed, and questionnaires regarding socioeconomic status and lifestyle factors were completed by the participants. Medical records and death certificates were reviewed annually. Starting in 2010, arterial stiffness was measured in a subcohort of participants, as detailed previously.[7,12] Dietary data were collected in 2014 using a validated food frequency questionnaire (FFQ), and the data were used for the current analyses.[13] Inclusion criteria in the current study were: (1) participation in the 2014 examination; (2) completion of the dietary assessment; and (3) 2 baPWV measurements ≥3 months apart. Of the 9073 participants who met these criteria, we excluded participants with established CVD or cancer (confirmed by medical record review) or diabetes mellitus (defined as either having fasting blood glucose concentration ≥7.0 mmol/L or using antidiabetic drugs) prior to 2014[14] because participants diagnosed with these conditions were likely counseled to change their dietary habits (n=1302). After applying these exclusion criteria, 7771 participants were included in our analyses (Figure).

Assessment of Night-Eating Frequency

Night-eating behavior/frequency was assessed via a multiple-choice question, "How many days do you usually eat at night in a typical week?" The possible answers were, "never or rarely," "1 to 2 times weekly," "3 to 5 times weekly," and "almost every day." Night eating was defined as the consumption of food and beverages, excluding water, after 8 PM and before 5 AM, in the 2 hours before bedtime, or after going to bed. In the current study, we merged the answers, 1 to 2 times weekly and 3 to 5 times weekly into 1 category "some days" because of the small sample size in

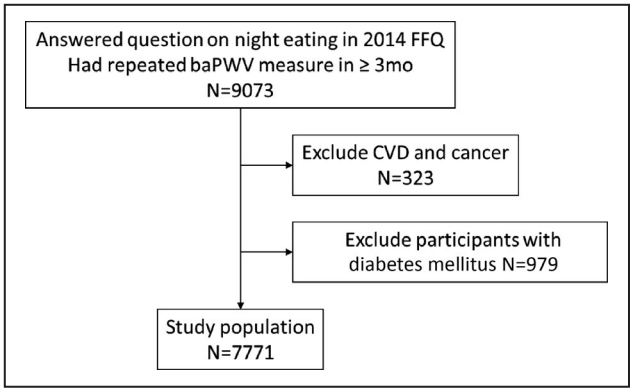

Figure. Flow chart of this study.
BaPWV indicates brachial-ankle pulse wave velocity; CVD, cardiovascular disease, including myocardial infarction, stroke, and heart failure; and FFQ, food frequency questionnaire.

each group (n=443 in the 1–2 times weekly group and n=167 in the 3–5 times weekly group). The night-eating frequency was thus classified on an ordinal scale of "never or rarely," "some days," and "most days."

Assessment of Arterial Stiffness Change

Arterial stiffness was assessed twice at baseline and during follow-up (mean difference between 2 assessments 3.19 years; interquartile range, 1.73–4.72 years) using baPWV, a simple and validated measurement that has been widely used in Asian cohort studies.[15] A detailed description of the assessment has been described elsewhere.[7] Briefly, participants were instructed to refrain from smoking and alcohol consumption 1 day prior to their visit to the clinic. On the examination day, participants were asked to rest for 5 minutes and then to lie down on the examination coach. Four cuffs were placed, 1 on each side of the brachial area and 1 on each ankle. The pulse transit times from brachial to ankle of both sides were read, and the transit velocities (cm/s) were calculated using a BP-203RPE III networked arteriosclerosis-detection device (Omron Healthcare, Pudong, Shanghai, China). Higher velocity indicates greater stiffness of the artery wall. The mean baPWV of 2 repeated readings was recorded. Finally, the mean baPWV of both the right and left sides was used in our analysis.

Assessment of Covariates

Personal and social demographic data, including age, sex, marital status, occupation type, education level, smoking habits, alcohol consumption, physical activity, antihypertensive drug usage, menopause status for women, and sleep quality were collected via self-reported questionnaire.[13] A validated semiquantitative FFQ was used to obtain habitual dietary intake in the past year.[13] Overall diet quality was assessed via the Dietary Approaches to Stop Hypertension (DASH) diet-quality score as detailed elsewhere.[16,17] Height, weight, and systolic blood pressure were measured on site by trained field workers (ie, physicians and nurses). Sleep parameters, including sleep duration, snoring, and insomnia, were assessed via a separate questionnaire as detailed elsewhere.[18] Physical activity was assessed by the validated Chinese version of the International Physical Activity Questionnaire Short Form.[19] Height and weight were measured by trained nurses, and were used to calculate body mass index (BMI). Fasting blood glucose, low-density lipoprotein-cholesterol, and high-density lipoprotein-cholesterol were quantified using a Hitachi 747 auto-analyzer (Hitachi, Tokyo, Japan); a blood sample was collected after an overnight fast (>8 hours). Details of the collection and assessment methods can be found in a previous publication.[13]

Statistical Analysis

All statistical analyses were performed in SAS 9.4 (SAS Institute, Cary, NC). Baseline characteristics across the 3 night-eating groups were compared using a 1-way ANOVA for continuous variables and a chi-square test for categorical variables. Data were presented as mean±standard deviation for continuous variables, and percentage for categorical variables.

The outcome in this analysis was the annual change rate of baPWV, as calculated by the difference of 2 values of baPWV (cm/s) divided by the follow-up duration (years). Greater change rates indicate a faster progression of arterial stiffness. Multiple linear regression models were used to calculate and compare adjusted means of baPWV change rate across night-eating habits, with the never or rarely group as the reference group. Model 1 adjusted for age and sex; model 2 further adjusted for baseline baPWV, total energy intake, and DASH diet-quality score; model 3 further adjusted for physical activity (low, moderate, or high), marriage (single or married), occupation (blue-collar or white-collar worker), education level (high school and below, or college and above), alcohol consumption (yes/no), smoking status (yes/no), BMI, systolic blood pressure, antihypertensive drug use (yes/no), fasting blood glucose, low-density lipoprotein-cholesterol and high-density lipoprotein-cholesterol; and model 4 further adjusted for sleep duration, insomnia (yes/no), snoring (yes/no), and breakfast frequency. Mean difference in baPWV annual change rate compared to the reference group with 95% CI was calculated. We further used linear regression to test the trend of arterial stiffness progression with an ordinal increase in night-eating frequency by including it as a continuous variable. In sensitivity analyses, we further excluded

900 participants who had 2 baPWV measurements in <1 year and 404 participants with insomnia.

We tested multiplicative interaction between night-eating frequency and age, sex, BMI, hypertension, and DASH diet-quality score, in relation to baPWV change rate, adjusted for the same set of covariates as our final model (model 4), and conducted relevant subgroup analysis when the interaction term was significant.

RESULTS

The mean age at baseline was 45.7±10.3 years, and 71.4% participants were men. In total, 14.8% of participants reported night eating some days or most days. Compared with those who never or rarely ate at night, participants with night-eating habits were younger, more likely to have longer sleep duration, higher BMI, and higher low-density lipoprotein-cholesterol concentration (Table 1).

During a mean 3.19 years of follow-up, frequent night-eating behavior was associated with more rapid progression of arterial stiffness, as indicated by annual change rate of baPWV, after adjustment for age, sex, baseline baPWV, total energy intake, overall diet quality, physical activity, marriage, employment, education level, alcohol consumption, smoking status, BMI, systolic blood pressure, antihypertensive drug use, fasting blood glucose, low-density lipoprotein-cholesterol, high-density lipoprotein-cholesterol, sleep duration, insomnia, snoring, and breakfast frequency (*P* trend=0.01; Table 2). The adjusted difference in change rate of baPWV between participants who ate at night most days and those who never or rarely ate at night was 14.1 (95% CI, 0.6–27.5) cm/s per year (Table 2).

This association was not modified by age, BMI, hypertension, or DASH diet-quality score (*P* interaction >0.1 for all). We found a significant interaction between night-eating frequency and sex, in relation to change in baPWV during follow-up (*P* interaction=0.03; Table 2 and Table S1). A significant association between night-eating behavior and annual change rate of baPWV was observed in women (*P* trend=0.002), but not in men (*P* trend=0.36). Further adjustment for menopausal status in women did not result in material change in the association between night-eating behavior and annual change rate of baPWV (Table 2). The observed association also persisted in the subgroup analyses stratified by menopausal status (*P* interaction=0.92; Table S2). A detailed baseline comparison between men and women is presented in Tables S3 and S4. We performed multivariable analysis separately for men and women, adjusting for the same set

Table 1. Baseline Characteristics Across Night-Eating Habits

Night Eating	Never or Rarely (n=6625)	Some Days (n=610)	Most Days (n=536)	*P* Value
Age, y	46.1±10.5	42.4±8.7	44.1±9.3	<0.001
Male, %	69.7	80.5	82.1	<0.001
College or above, %	17.6	18.4	11.6	0.03
Manual labor, %	80.5	81.6	89.0	0.002
Married, %	95.2	91.5	94.6	0.02
Current smoker, %	39.7	50.3	42.6	<0.001
Current drinker, %	35.8	57.2	46.4	<0.001
Physical exercise, %	24.1	24.3	25.6	0.009
Antihypertensive drug, %	10.1	6.6	7.4	0.006
Sleep duration, h	7.86±1.61	7.83±1.68	8.05±1.91	0.04
Insomnia, %	4.6	11.1	6.1	<0.001
Frequent snoring, %	11.1	14.9	11.0	0.02
Everyday breakfast, %	82.9	61.5	82.8	<0.001
Total energy intake, kcal/d	1668±562	1724±624	1685±559	0.08
DASH diet-quality score	26.1±4.7	26.1±4.5	26.1±4.6	0.91
baPWV, cm/s	1408±275	1385±241	1401±234	0.10
Body mass index, kg/m^2	24.8±3.3	24.9±3.3	24.4±3.1	0.02
Systolic blood pressure, mm Hg	130±17	129±15	129±16	0.05
Fasting blood glucose, mmol/L	5.31±0.61	5.25±0.63	5.30±0.62	0.13
Low-density lipoprotein cholesterol, mmol/L	2.96±0.76	2.86±0.76	2.87±0.72	<0.001
High-density lipoprotein cholesterol, mmol/L	1.41±0.43	1.40±0.60	1.40±0.34	0.71

baPWV indicates brachial-ankle pulse wave velocity; and DASH, Dietary Approaches to Stop Hypertension.

Table 2. Difference of Brachial-Ankle Pulse Wave Velocity Change Rate (cm/s per year) According to Night-Eating Frequency in Women, Men, and Total Participants

	Never or Rarely	Some Days	Most Days	*P* for Trend
Women (n=2222)				
N	2007	119	96	
Model 1	0 (reference)	36.1 (11.3–61.0)*	27.2 (0–54.5)	0.003
Model 2	0 (reference)	38.8 (13.1–64.5)*	29.8 (1.7–57.9)*	0.002
Model 3	0 (reference)	34.2 (7.5–61.0)*	34.3 (4.7–64.0)*	0.002
Model 4	0 (reference)	32.5 (5.0–59.9)*	35.1 (5.4–64.8)*	0.002
Further adjusting for menopausal status	0 (reference)	32.2 (4.7–59.6)*	35.1 (5.3–64.8)*	0.003
Men (n=5549)				
N	4618	491	440	
Model 1	0 (reference)	3.4 (–10.2 to 17.1)	4.3 (–9.7 to 18.3)	0.47
Model 2	0 (reference)	1.9 (–11.0 to 15.7)	4.0 (–10.1 to 18.1)	0.55
Model 3	0 (reference)	–0.1 (–14.8 to 14.5)	6.9 (–8.2 to 22.0)	0.43
Model 4	0 (reference)	4.1 (–11.3 to 19.5)	6.3 (–8.9 to 21.4)	0.36
Total (n=7771)	6625	610	536	
Model 1	0 (reference)	11.0 (–0.9 to 22.9)	9.8 (–2.6 to 22.3)	0.04
Model 2	0 (reference)	10.4 (–1.8 to 22.5)	10.0 (–2.6 to 22.6)	0.04
Model 3	0 (reference)	8.9 (–4.0 to 21.7)	14.3 (0.9–27.8)*	0.02
Model 4	0 (reference)	12.2 (–1.2 to 25.6)	14.1 (0.6–27.5)*	0.01

Model 1 adjusted for age and sex; model 2 further adjusted for baseline brachial-ankle pulse wave velocity, total energy intake (quartiles), and Dietary Approaches to Stop Hypertension diet-quality score; model 3 further adjusted for physical activity (low, moderate, or high), marriage (single or married), employment (blue-collar or white-collar worker), education level (high school and below, or college and above), alcohol consumption (yes/no), smoking status (yes/no), antihypertensive drug (yes/no), body mass index (quintile), systolic blood pressure (quintile), fasting blood glucose (quintile), low-density lipoprotein-cholesterol (quintile) and high-density lipoprotein-cholesterol (quintile); model 4 further adjusted for sleep duration (hours), insomnia (yes/no), snoring (yes/no), and breakfast frequency. Menopause status was categorized as no menopause, developed menopause, or postmenopausal during follow-up.

*P difference <0.05 compared with "never or rarely" ate-at-night group.

Values are adjusted mean differences (95% CIs).

of covariates in model 4. For women only, being single and presence of snoring were significantly associated (P<0.05), whereas for men only, higher total energy intake, smoking, and higher fasting blood glucose concentration were significantly associated (P<0.05) with faster progress of arterial stiffness.

Significant associations persisted after we excluded participants with short follow-up duration (<1 year); the adjusted difference in annual change rate of baPWV comparing the 2 extreme night-eating groups was 10.4 (95% CI, 1.4–19.4) cm/s per year in the total population (P trend=0.04; Table 3). Further, the trends

Table 3. Sensitivity Analyses of Adjusted Difference of Brachial-Ankle Pulse Wave Velocity Change Rate (cm/s per year) According to Night-Eating Frequency

	Never or Rarely (n=6625)	Some Days (n=610)	Most Days (n=536)	*P* for Trend
Propensity score adjusted (n=7771)	0 (reference)	8.6 (0.9–16.3)*	8.8 (0.7–17.0)*	0.01
Excluding repeated measurement in <1 y	0 (reference)			
Women (n=2062)	0 (reference)	4.5 (–17.6 to 26.6)	23.1 (–0.2 to 46.4)	0.05
Men (n=4809)	0 (reference)	–3.2 (–12.7 to 6.4)	5.9 (–3.1 to 15.0)	0.35
Total (n=6871)	0 (reference)	–0.1 (–9.4 to 9.1)	10.4 (1.4–19.4)*	0.04
Excluding participants with insomnia				
Women (n=2026)	0 (reference)	33.5 (3.7–63.4)*	35.0 (3.1–66.9)*	0.005
Men (n=5341)	0 (reference)	3.9 (–12.5 to 20.3)	6.1 (–9.5 to 21.7)	0.39
Total (n=7367)	0 (reference)	11.6 (–2.8 to 25.9)	13.6 (–0.4 to 27.7)	0.02

Model adjusted for age, baseline brachial-ankle pulse wave velocity, total energy intake (quartiles), Dietary Approaches to Stop Hypertension diet-quality score, physical activity (low, moderate, or high), marriage (single or married), employment (blue-collar or white-collar worker), education level (high school and below, or college and above), alcohol consumption (yes/no), smoking status (yes/no), antihypertensive drug (yes/no), body mass index (quintile), systolic blood pressure (quintile), fasting blood glucose quintile), low-density lipoprotein-cholesterol (quintile), high-density lipoprotein-cholesterol (quintile), sleep duration (hours), insomnia (yes/no), snoring (yes/no), and breakfast frequency.

*P difference <0.05 compared with the "never or rarely" ate-at-night group.

Values are adjusted mean differences (95% CIs).

persisted after we excluded participants with insomnia (*P* trend=0.02; Table 3).

DISCUSSION

In a population without major chronic diseases, women, but not men, who reported eating most nights had a more rapid increase in baPWV than those who never or rarely ate at night. The observed association was independent of total energy consumption, overall diet quality, and other major risk factors for CVD.

This is the first large-scale longitudinal study, to the best of our knowledge, which examined the association between night-eating habits and arterial stiffness, an important risk factor and preclinical marker for CVD. Physical stiffening of arteries significantly contributes to an increased risk of developing hypertension, coronary heart disease, and stroke.[6] Although recognized as part of the natural process of aging, arterial stiffness progression rates vary among individuals and may be accelerated by lifestyle behaviors such as night eating. A meta-analysis, including 14 673 participants, showed that the addition of baPWV to a model incorporating the Framingham risk score significantly increases the predictive power for future CVD risk.[10] To date, less attention has been paid to the progression of arterial stiffness. This study took advantage of repeated measures of baPWV to estimate the speed of vascular aging as our main outcome.

Our findings are consistent with previous studies that found night eating was associated with CVD risk factors. In a clinical study including 52 participants who completed 7 days of wrist actigraphy and food logs, energy consumed after 8 PM was positively associated with higher BMI after adjustment for age, sleep duration, and sleep timing.[20] Night eating, defined as having dinner immediately before bed or having snacks after dinner, has been reported to result in a 2.37-fold (95% CI, 1.71–3.29) higher risk of having obesity, and a 1.49-fold (95% CI, 1.14–1.94) higher risk of having dyslipidemia, after adjustment for age, smoking habits, alcohol consumption, physical activity, breakfast intake, and hypertension.[2] Similarly, in a large-scale prospective cohort study, men who reported eating late at night had a 55% (95% CI, 5%–129%) higher risk of developing coronary heart disease, compared with those who did not eat late at night, after adjustment for age, demographic factors, and physical activity.[3] These studies, together with the current findings, suggest that habitual night eating—independent of other lifestyle behaviors, dietary quality, and sleep quality—plays a critical role in the prevention of chronic disease. However, because of the small number of studies, the 2015 Dietary Guidelines for Americans did not make a recommendation for the timing of meals.[21] Future studies with large case numbers and long follow-up durations are needed to study the direct association between food-intake distribution patterns, specifically night-eating habits, and health outcomes.

Somewhat unexpectedly, the significant association between night-eating habits and baPWV annual change rate was significant in women, but not in men. Menopause status did not significantly modify this association. However, this finding might be limited by the small sample size of postmenopausal women (n=438) in this cohort. We cannot exclude the possibility that the sex difference in association between night-eating habits and baPWV annual change rate is a chance finding. This result could also be because of the differences in lifestyle and health status between men and women. For example, the prevalence of insomnia was higher in women relative to men. Although we adjusted for these factors in the model, we still cannot totally exclude the possibility of residual confounding. However, these data suggest there may be sex differences in mental and physical adaptation to night eating. Women have been reported to experience higher postmeal satiety and dietary restraint than men.[22] Sex differences are also present in the association between the intake of macronutrients and alcohol and body fat indicators.[23] For example, 1 study reported an inverse association between fat intake and BMI, and between fat intake and waist circumference in men, but not in women.[23] The circadian rhythm of plasma cortisol concentrations also exhibits sex differences: Women have a higher age-related elevation in the morning, and a more abrupt ending of the quiescent period than men.[24] Early studies found that circadian misalignment—suppressed and delayed melatonin secretion—would increase estrogen concentration and function of the estrogen receptor.[25] Finally, although the findings are limited, certain aspects of jejunal motility during sleep were modulated by sex.[26] These lifestyle and biological differences between women and men may impact patterns associated with arterial stiffness and CVD risk.

Night eating may influence a number of variables that could result in an acceleration of arterial stiffening. Night eating leads to a disturbance or shift in an individual's cardiac clock and overnight-fasting period. Glucose, epinephrine, and cortisol all exhibited significant impacts on endogenous circadian rhythm.[27] Circadian misalignment could further alter endocrine function. Moreover, eating close to bedtime would necessitate digestion during sleep, contributing to sleep disturbance and imposing an extra burden on the pancreas[28] and adipose tissue.[29] The

potentially resulting hyperglycemia, elevated nonesterified fatty acid level, and insulin resistance could result in elevated oxidative stress and inflammation, contributing to endothelial dysfunction and arterial stiffness progression.[30] Previous studies on meal timing (eg, breakfast frequency and overall eating frequency) and CVD and mortality, also reported significant associations.[3,31] The observational data support potential underlying biological and physiological mechanisms underpinning the effect of the biological clock on cardiovascular health.

Night eating may indirectly accelerate arterial stiffness as a result of insomnia (eg, difficulty falling sleep). However, controlling for sleep-quality assessments, including sleep duration, snoring status, insomnia, and breakfast habits had little effect on the results. Night eating may be associated with the intake of excess energy or specific foods associated with less healthy dietary patterns. However, no significant difference was observed in total energy intake or diet quality across different night-eating groups, and the association observed with arterial stiffness remained significant after adjustment for total energy intake and diet quality. Night eating might be an indicator of other lifestyle characteristics that would promote arterial stiffening, such as sedentary lifestyle, smoking, and alcohol consumption. In our cohort, controlling for these variables had little effect on the statistical significance of the results, although because of the observational study design, residual confounding cannot be avoided.

Limitations

This study focused on a population without CVD, cancer, and diabetes mellitus at the time of recruitment because diagnosis of these diseases would likely change a person's eating habits. Thus, our findings may not be generalizable to individuals with these chronic conditions. Another limitation of this study was the self-reporting of night-eating habits. Participants may not be able to correctly recall or may misreport their night-eating frequencies. The definition of night eating in our study did not consider the number of calories consumed. However, total energy was adjusted in all models. This study also lacked information on participants' stress levels, mood disorders, chronotypes, and the exact timing of each meal, bedtime, and work hours. A systematically delayed biological clock may contribute differently to arterial stiffness than an additional meal or snack at night. Further, only a single measure, baPWV, was used to measure arterial stiffness. Other indicators, such as the carotid-femoral PWV, which is the current gold standard measure of arterial stiffness, and the augmentation index, which characterizes the amplitude of the reflected blood wave, should be further examined. However, baPWV has been validated for use in large epidemiological cohorts,[7,15] and has previously been demonstrated to have significant prediction value for CVD.[10] Data were not available for diet behaviors, such as number of meals per day, snacks, and types of food that were consumed at night; or measures of circadian rhythms, which would have allowed for further exploration of the sex-specific difference we observed. Another limitation is that only 29% of the study population was women. This is because participants were recruited from the Kailuan Company, a coal-mining company where the majority of employees are men. Nevertheless, the large sample size gave power for sex-specified analyses. Strengths of this study include the large sample size, availability of a wide range of behavioral and biological variables, and longitudinal follow-up of arterial stiffness progression.

To conclude, in this community-based prospective longitudinal study, habitual night eating was associated with more rapid progression of arterial stiffness in women without CVD, cancer, and diabetes mellitus during the observational period. Future studies with more detailed measures of eating habits, meal timings, and work and leisure patterns, as well as sex-specific endocrine measures, are needed to further examine the association between food-intake patterns during waking hours and cardiovascular risk factors, as well as CVD risk.

ARTICLE INFORMATION

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Disclosures

None.

Supplementary Materials

Tables S1–S4

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Supplemental Material

Table S1. Adjusted mean ± standard error of brachial-ankle pulse wave velocity change rate (cm/s per year) across night eating habits in women, men, and total participants.

	Never or rarely	Some days	Most days	p for trend
Women (n=2,222)				
Model 1	14.2±3.0	50.3±12.3	41.4±13.6	0.003
Model 2	13.0±3.2	51.8±12.8	42.8±14.0	0.002
Model 3	36.0±25.6	70.2±28.6	70.4±29.1	0.002
Model 4	31.2±30.8	63.6±32.4	66.3±33.4	0.002
Men (n=5,549)				
Model 1	9.9±2.1	13.4±6.6	14.2±6.8	0.47
Model 2	10.1±2.1	12.0±6.7	14.1±6.9	0.55
Model 3	-4.6±22.9	-4.8±23.7	2.2±24.0	0.43
Model 4	-12.6±26.2	-8.5±26.5	-6.3±27.0	0.36
Total (n=7,771)				
Model 1	6.6±1.9	17.6±5.9	16.5±6.2	0.04
Model 2	5.9±1.9	16.3±6.0	15.9±6.3	0.04
Model 3	20.3±16.2	29.2±17.1	34.6±17.3	0.02
Model 4	12.6±18.7	24.8±19.2	26.6±19.6	0.01

Model 1 adjusted for age and sex;
Model 2 further adjusted for baseline baPWV, total energy intake (quartiles) and Dietary Approaches to Stop Hypertension score;
Model 3 further adjusted for physical activity (low, moderate, or high), marriage (single or married), employment (blue-collar or white-collar worker), education level (high school and below, or college and above), alcohol consumption (yes or no), smoking status (yes or no), antihypertensive drug (yes/no), body mass index (quintile), systolic blood pressure (quintile), fasting blood glucose (quintile), low-density lipoprotein-cholesterol (quintile) and high-density lipoprotein-cholesterol (quintile);
Model 4 further adjusted for sleep duration (h), insomnia (yes or no), snoring (yes or no), and breakfast frequency.
*p-difference <0.05, compared to “never or rarely” ate at night group.

Table S2. Adjusted difference of brachial-ankle pulse wave velocity change rate (cm/s per year) according to night eating frequency in women, stratified by menopause status.

Menopause status	Never or rarely	Some days	Most days	p for trend	p for interaction
					0.92
No (n=1,345)	n=1200	n=88	n=57		
	0 (reference)	11.6 (-37.2, 60.3)	37.8 (6.6, 69.0)*	0.02	
Yes (n=438)	n=412	n=11	n=15		
	0 (reference)	34.3 (-137, 206)	85.1 (-61.4, 232)	0.19	

Model adjusted for age, baseline baPWV, total energy intake (quartiles), Dietary Approaches to Stop Hypertension (DASH) score, physical activity (low, moderate, or high), employment (blue-collar or white-collar worker), education level (high school and below, or college and above), alcohol consumption (yes or no), smoking status (yes or no), antihypertensive drug (yes/no), body mass index (quintile), systolic blood pressure (quintile), fasting blood glucose quintile), low-density lipoprotein-cholesterol (quintile), high-density lipoprotein-cholesterol (quintile), sleep duration (h), insomnia (yes or no), snoring (yes or no), and breakfast frequency;
*p-difference <0.05, compared to “never or rarely” ate at night group.

Table S3. Baseline characteristics comparison between women and men.

	Women n=2222	Men n=5549	p
Age, y	46.4 ± 9.8	45.4 ±10.5	<0.001
College or above, %	32.7	10.4	<0.001
Manual labor, %	61.1	90.1	<0.001
Married, %	93.7	95.6	0.006
Current smoker, %	1.44	56.0	<0.001
Current drinker, %	15.9	46.9	<0.001
Physical exercises, %	19.6	16.3	<0.001
Antihypertensive drug, %	8.0	10.3	<0.001
Sleep duration, h	7.86 ± 1.40	7.87 ±1.74	0.70
Insomnia, %	8.9	3.8	<0.001
Frequent snore, %	7.5	12.9	<0.001
Everyday breakfast, %	92.2	76.7	<0.001
Total energy intake, kcal/d	1564 ± 471	1720 ± 598	<0.001
DASH diet quality score	26.6 ± 4.2	25.9 ± 4.8	<0.001
baPWV, cm/s	1302 ± 267	1447±259	<0.001
Body mass index, kg/m^2	24.0 ± 3.3	25.1 ± 3.2	<0.001
Systolic blood pressure, mmHg	122 ± 16	133 ± 16	<0.001
Fasting blood glucose, mmol/L	5.21 ± 0.59	5.34 ± 0.62	<0.001
Low-density lipoprotein-cholesterol, mmol/L	2.83 ± 0.76	2.99 ± 0.76	<0.001
High-density lipoprotein-cholesterol, mmol/L	1.44 ± 0.41	1.39 ± 0.45	<0.001

BaPWV, brachial-ankle pulse wave velocity; DASH, Dietary Approaches to Stop Hypertension.

Table S4. Baseline characteristics across night eating habits in women versus men.

	Women, n=2222			Men, n=5549		
	Never or rarely N=2007 (90.3%)	Some days N=119 (5.4%)	Most days N=96 (4.3%)	Never or rarely N=4618 (83.2%)	Some days N=491 (8.9%)	Most days N=440 (7.9%)
Age, y	46.7 ± 9.8	42.2 ± 8.2	45.8 ± 10.1	45.9 ± 10.8	42.5 ± 8.8	43.7 ± 9.1
College or above, %	32.0	52.6	30.0	10.9	9.1	5.8
Manual labor, %	61.1	52.6	69.7	89.7	89.7	94.9
Married, %	93.9	88.1	94.4	95.9	92.4	94.7
Current smoker, %	1.5	0.9	0.0	55.8	62.0	51.5
Current drinker, %	15.4	24.4	15.2	44.4	65.0	53.0
Physical exercises, %	19.2	21.1	26.9	26.6	25.1	25.3
Antihypertensive drug, %	8.4	4.3	5.3	10.9	7.2	7.9
Sleep duration, h	7.86 ± 1.37	7.89 ± 1.51	7.84 ± 1.82	7.86 ± 1.72	7.81 ± 1.72	8.10 ± 1.93
Insomnia, %	8.6	12.7	10.9	2.9	10.7	5.1
Frequent snore, %	7.5	9.4	5.8	12.7	16.2	12.2
Everyday breakfast, %	93.2	74.8	92.6	78.3	58.3	80.7
Total energy intake, kcal/d	1564 ± 473	1608 ± 491	1511 ± 412	1716 ± 593	1754 ± 651	1724 ± 580
DASH diet quality score	26.6 ± 4.2	26.6 ± 4.1	26.4 ± 4.8	25.8 ± 4.9	26.0 ± 4.6	26.0 ± 4.6
baPWV, cm/s	1304 ± 267	1256 ± 214	1312 ± 309	1453 ± 266	1416 ± 237	1420 ± 210
Body mass index, kg/m^2	24.1 ± 3.3	23.8 ± 3.2	23.4 ± 3.4	25.2 ± 3.2	25.2 ± 3.3	24.7 ± 3.0
Systolic blood pressure, mmHg	122 ± 16	118 ± 13	118 ± 17	134 ± 16	132 ± 15	131 ± 14.4
Fasting blood glucose, mmol/L	5.22 ± 0.59	5.14 ± 0.61	5.18 ± 0.62	5.35 ± 0.62	5.28 ± 0.63	5.32 ± 0.61
Low-density lipoprotein-cholesterol, mmol/L	2.84 ± 0.77	2.63 ± 0.70	2.88 ± 0.71	3.01 ±0.76	2.92 ± 0.76	2.86 ± 0.73
High-density lipoprotein-cholesterol, mmol/L	1.43 ± 0.33	1.50 ± 1.12	1.45 ± 0.31	1.40 ± 0.46	1.37 ± 0.36	1.38 ± 0.35

BaPWV, brachial-ankle pulse wave velocity; DASH, Dietary Approaches to Stop Hypertension.