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PDF issue: 2026-11-10

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(Citation)
American Journal of Health Promotion,39(7):1046-1050

(Issue Date)
2025-09

(Resource Type)
journal article

(Version)
Accepted Manuscript

(Rights)
Kabata D, Matsumiya S, Yamanouchi F, et al. Caloric Intake at Lunch Does Not Mediate the Impact of Eating Rate on Weight Gain Among Healthy Adults: Longitudinal Cohort Study. American Journal of Health Promotion. 2025;39(7):1046-1050. Copyright © The Author(s) 2025. DOI: https://doi.org/10.1177/08901171251335778

(URL)
https://hdl.handle.net/20.500.14094/0100495936

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Title Page

Caloric Intake at Lunch Does Not Mediate the Impact of Eating Rate on Weight Gain among Healthy Adults: Longitudinal Cohort Study

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Author Contributions

Study concept and design: Kabata, Matsumiya, Yamanouchi, and Koretsune.

Acquisition, analysis, or interpretation of data: All authors.

Drafting of the manuscript: Kabata.

Critical revision of the manuscript for crucial intellectual comment: All authors.

Statistical analysis: Kabata.

Administrative, technical, or material support: Koretsune.

Study supervision: Kabata and Koretsune.

Conflict of Interest

Kabata received the consultation fees from Daihatsu Co., Ltd. Matsumiya, Yamanouchi, Saeki, Kajimoto, Kaimori, Tanaka, Zeniya, and Koretsune are an employee of Daihatsu Co., Ltd. There are no other potential conflicts of interest regarding this submitted work.

Financial Support

This study did not receive any funding.

Ethical Approval and Informed Consent Statements

Institutional Review Board II reviewed and approved the study protocol at the National Hospital Organization, Osaka National Hospital, Japan (approval No. 23115). All data were anonymized to ensure participant confidentiality and privacy. Employees were also allowed to opt-out and have their data removed from the study.

Data Availability Statements

The data utilized in this study includes employee information and cannot be shared publicly due to confidentiality and privacy considerations.

Abstract

Purpose: This study aims to evaluate causal relationships among eating rate, caloric intake, and weight gain using longitudinal data and to clarify how caloric intake mediates the effect of eating rate on weight gain.

Design: A longitudinal cohort study with a four-year follow-up.

Setting: Data were collected from Daihatsu Co., Ltd. employees, a major Japanese automobile manufacturer with four plants in Japan.

Subjects: Of the 12,816 working-age Japanese individuals who underwent health checkups in 2020, 7,784 without excess weight or non-communicable diseases were included.

Intervention: The eating rate was self-reported, and caloric intake was calculated based on cafeteria meal choices.

Measures: Annual change in body mass index (BMI), calculated as the difference in BMI between consecutive years.

Analysis: Mediation analyses using a mixed-effects model were conducted to assess whether caloric intake mediates the effect of eating rate on BMI.

Results: Caloric intake in the fast-eating and ordinal-eating groups exceeded that of the slow-eating group by 19.21 kcal [9.28, 29.13] and 10.01 kcal [1.20, 18.81],

respectively. Higher caloric intake was associated with a more significant BMI increase, rising by 0.09 [0.06, 0.13] per interquartile range increase. The proportion of mediated impact among the impact of eating rate is 0.19% [0.09, 0.32].

Conclusion: Faster eating independently contributes to BMI increases, with minimal mediation by caloric intake. Interventions targeting eating pace may prevent obesity, though the study population may limit generalizability.

Keywords

Obesity

Excess weight

Caloric intake

Eating rate

Mediation analysis

Purpose

Excess weight is closely linked to dietary habits, mainly eating rate. A meta-analysis by Ohkuma et al. has shown a positive association between fast eating and excess weight, with studies suggesting that fast eating and caloric intake independently contribute to this condition.[1] However, most prior research on this relationship has been cross-sectional, highlighting overlaps between fast eating and excess weight without clarifying causality. Rapid eating may lead to weight gain, but obesity-related hormonal changes could also increase hunger and caloric intake, suggesting possible reverse causation.[2] While significant associations between fast eating and excess weight persist after adjusting for caloric intake, the extent to which caloric intake mediates this relationship remains unclear. To address these gaps, we conducted mediation analysis using longitudinal data.

Furthermore, most prior studies rely on self-reported measurements. Eating rates are typically evaluated through questionnaires, while caloric intake is determined based on the Diet History Questionnaire (DHQ)3. Although our study also relied on self-reported eating rates, we utilized precise caloric intake data automatically calculated from meal choices at an employee cafeteria. This approach is expected to

enhance the reliability of our findings and contribute robust evidence to clarify the role of eating habits in excess weight and the mediating effect of caloric intake.

Methods

Design

This longitudinal cohort study utilized longitudinal data from Daihatsu Co., Ltd., collected over four years (January 2020 and December 2023). To ensure confidentiality, all data were anonymized, and employees were allowed to opt-out, removing their data from the study.

Sample

The study included working-age Japanese individuals without excess weight or a history of non-communicable diseases. Of the 12,816 employees who underwent health checkups in 2020, 7,784 were eligible. These individuals had a body mass index (BMI, kg/m^2) <25 and no treatment for hypertension, diabetes, or dyslipidemia, nor any history of stroke, cardiovascular disease, or kidney-related conditions in 2020. Participants also had not received specific health guidance programs for metabolic syndrome management. The follow-up, which started at the beginning of observation in 2020, continued for each individual until they met any of the following conditions: developing a non-communicable disease, receiving specific health guidance, or discontinuing health checkups for reasons such as retirement.

Measures

Health data were collected from mandatory annual health checkups, which included assessments like height, weight, chest X-rays, blood pressure, and blood tests. The primary outcome was the change in BMI per year, calculated as the difference in BMI between consecutive years. Secondary outcomes included the annual change in systolic blood pressure (SBP, mmHg), diastolic blood pressure (DBP, mmHg), hemoglobin A1c (HbA1c, %), triglycerides, high-density lipoprotein (HDL) cholesterol (mg/dL), low-density lipoprotein (LDL) cholesterol (mg/dL), and uric acid levels (mg/dL). These secondary outcomes were included to assess broader metabolic effects related to eating behavior and weight gain.

Eating rate, the exposure variable, was self-reported through a questionnaire. Participants were asked, “Do you eat faster than other people?” with response options categorized into three levels: slow, ordinal, and fast. Caloric intake per meal was specifically assessed for lunch, based on cafeteria menu choices. Each meal item’s caloric value was pre-determined using Japan’s Standard Tables of Food Composition.[4] The median caloric intake of meals chosen over the year preceding each health checkup was used for analysis.

Analysis

Baseline characteristics were summarized using medians and interquartile-range (IQR) for continuous variables and counts and proportions for categorical variables, stratified by eating rate groups. Key baseline characteristics included age, sex, occupational category, physical activity, commuting method, work location, alcohol consumption, smoking status, regular exercise, and prior health checkup data. These variables were included to identify potential confounders and describe population differences. Annual BMI changes were also summarized by the eating rate group.

A mediation analysis based on the Baron & Kenny framework assessed whether caloric intake mediated the relationship between eating rate and BMI change.[5] The series of analysis was consisted of the four models assessing (i) the association between eating rate and caloric intake, (ii) the non-linear impact of caloric intake on the annual BMI changes, (iii) the impact of eating rate on the annual BMI changes without adjustment for caloric intake, and (iv) the impact of eating rate on the annual BMI changes with adjustment for caloric intake. For these assessments, we performed mixed-effects models with random intercepts, adjusting for potential confounders that could influence both caloric intake and health outcomes. Specifically, we adjusted for age, sex, occupation, physical activity, commuting method, work location, alcohol

consumption, smoking status, and regular exercise, as these factors are known to be associated with dietary habits and metabolic health. Additionally, we controlled for baseline measures from the previous health checkup and the year of the health checkup to account for pre-existing health conditions and temporal trends. These adjustments were made to minimize bias and ensure that the estimated impact of eating rate and caloric intake was not confounded by imbalances in these characteristics. The non-linear relationship was examined using the restricted-cubic-spline method. Missing data were addressed using multiple imputations by predictive mean matching.

Furthermore, to estimate the proportion of the effect of eating rate on secondary health outcomes mediated by caloric intake, we conducted the causal mediation analyses adjusting for similar covariates.[6] The causal mediation analyses were performed for not only the primary outcome and for the secondary outcomes, the annual difference in SBP, DBP, HbA1c, triglycerides, HDL cholesterol, LDL cholesterol, and uric acid levels, we examined the mediated impact of caloric intake on them similarly to the primary outcome, except for excluding each baseline variable of them and including the prior year's BMI was included as the covariate.

All analyses were conducted using R (https://cran.r-project.org/).

Results

Baseline Characteristics

Baseline characteristics in 2020 for the 7,784 participants are summarized in Supplementary Table 1, stratified by eating rate categories from the 2021 checkup. The eating rate distribution was as follows: slow (919 individuals), ordinal (4,035 individuals), and fast (2,704 individuals), with 126 missing data points. Changes in BMI from 2020 to 2023, categorized by eating rate, show that faster eaters tended to have higher baseline BMI values and slightly greater BMI increases over time (Supplementary Figure 1).

Mediation Analysis

Figure 1A depicts the multivariable-adjusted association between eating rate and caloric intake assessed by model (i). A faster eating rate was linked to greater calorie consumption, with the ordinal-eating group consuming 10.01 kcal more per meal [95% confidence interval (CI): 1.20, 18.81] and the fast-eating group consuming 19.21 kcal more [9.28, 29.13], compared to the slow-eating group. Figure 1B highlights the relationship between caloric intake and BMI increase over one year, examined by model (ii), showing a BMI increase of 0.09 [0.06, 0.13] for an IQR increase in caloric intake.

Figure 1C compares the annual BMI increase across eating rate groups both without and with adjusting for caloric intake, assessed by model (iii) and (iv). Without an adjustment in the model (iii), the ordinal and fast-eating groups exhibited BMI increases of 0.05 [0.02, 0.09] and 0.07 [0.04, 0.11], respectively, compared to the slow-eating group. These trends remained unchanged after caloric intake adjustment in the model (iv), with increases of 0.05 [0.01, 0.08] and 0.07 [0.03, 0.10], respectively.

Causal mediation analysis showed that caloric intake mediates only 0.19% [0.09, 0.32] of the eating rate's impact on BMI changes (Supplementary Figure 2). The mediated impact was similarly small for other outcomes, except for triglycerides and LDL cholesterol, where mediation proportions were 5.79% [1.82, 12.75] and 2.55% [0.72, 4.33], respectively.

Discussion

Our findings confirm that faster eating rates are independently associated with BMI increases, yet the mediating role of caloric intake was negligible. This aligns with prior research indicating that rapid eating may lead to higher BMI through behavioral, metabolic, or physiological pathways beyond energy intake alone, such as impaired glucose tolerance and insulin resistance.[1,7] While fast eaters consumed more calories at lunch, this difference did not explain their greater weight gain. Additionally, our study,

161 which calculated caloric intake based on actual selections, demonstrated similar results
162 when compared to previous research that relied on self-reported intake measurements,
163 providing stronger supporting evidence for earlier findings.

164 Several limitations merit consideration. First, we relied on self-reported eating
165 speed, which may have introduced misclassification bias. Second, our sample
166 comprised healthy, working-age Japanese adults, potentially limiting external validity to
167 other demographics. Third, we measured only lunch-based caloric intake, thereby
168 underestimating total daily consumption. Nonetheless, the study's strengths include
169 objective caloric measurements, longitudinal data, and extensive covariate adjustment.
170 Future research should investigate the influence of rapid eating on metabolic markers,
171 such as postprandial insulin or gut peptide responses, to elucidate underlying
172 mechanisms.[8] Exploring alternative dietary patterns or smartphone-based monitoring of
173 overall intake may also help clarify whether fast eating is linked to excess calories at
174 other meals. Moreover, randomized trials testing slower eating interventions could
175 definitively determine causality.

176 From a public health standpoint, mindful eating strategies—focusing on slower
177 chewing, satiety awareness, and meal-related behavioral cues—have demonstrated
178 potential in reducing overeating and facilitating long-term weight control.[9,10] Workplace

wellness programs and health education campaigns could integrate such approaches to reduce obesity risk, although additional evidence is needed on how best to sustain slower eating behaviors over time.

“SO WHAT?”

What is already known on this topic?

Fast eating and caloric intake are independently associated with the prevalence of excess weight. However, the causal relationships between them and the degree to which caloric intake mediates the relationship between eating speed and weight gain remain unclear.

What does the article add?

Faster eating independently causes weight gain over time, with minimal mediation by caloric intake. This highlights that eating rate itself directly contributes to weight control.

What are the implications for health promotion practice or research?

Interventions focusing on slowing eating speed may be an effective approach to curbing weight gain and preventing obesity, independent of caloric intake adjustments.

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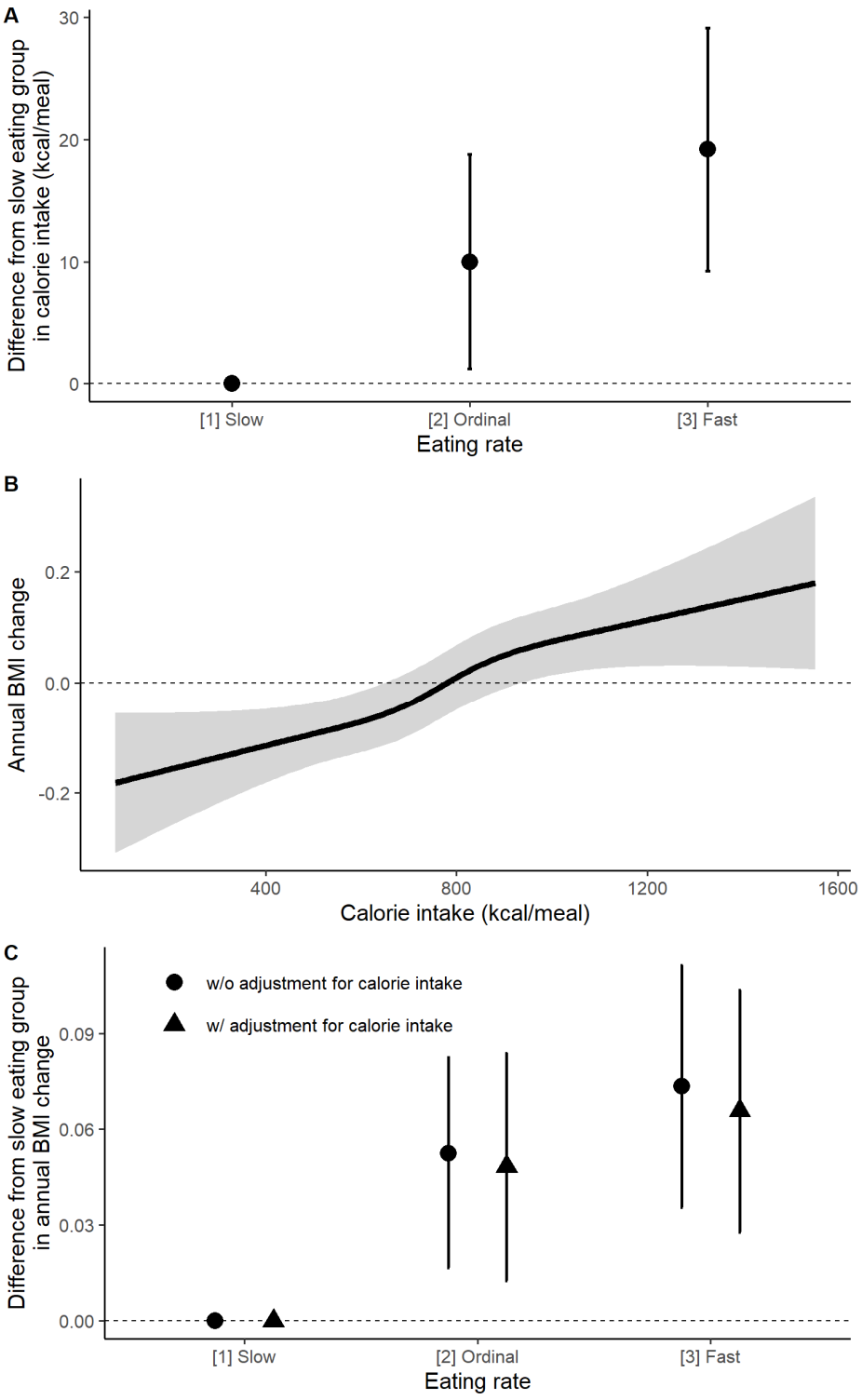
Figure Legends

Figure 1. Results of mediation analysis

Figure 1A presents the multivariable-adjusted relationship between eating rate and caloric intake. Each point indicates the point estimate of the difference from the slow-eating group, and error bars represent the 95% confidence intervals. Figure 1B displays the multivariable-adjusted association between caloric intake and annual BMI change. The solid line represents the connected point estimates, and the shaded gray area indicates the 95% confidence intervals. Figure 1C shows the multivariable-adjusted impact of eating rate on annual BMI change. Circular points represent point estimates of the difference from the slow-eating group without adjusting for caloric intake, while triangular points represent estimates adjusted for caloric intake. Error bars indicate the 95% confidence intervals for each.

Figure

Figure 1. Results of mediation analysis



Supplementary Tables

Supplementary Table 1. Demographical and clinical characteristics and lifestyle habits in 2020

		Eating rate			
	Overall, N = 7,784	[1] Slow, N = 919	[2] Ordinal, N = 4,035	[3] Fast, N = 2,704	Missing, N = 126
Age	39.93 (32.99, 49.02)	39.03 (32.00, 49.98)	39.97 (33.00, 49.93)	39.87 (33.03, 48.02)	39.48 (31.29, 51.00)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
Sex					
Female	9.1% (709)	14% (130)	9.1% (368)	6.6% (178)	26% (33)
Male	91% (7,075)	86% (789)	91% (3,667)	93% (2,526)	74% (93)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
Physical activity on the job					
[1] Low	37% (2,853)	41% (379)	36% (1,452)	38% (1,018)	29% (4)
[2] Medium	18% (1,373)	15% (137)	18% (740)	18% (492)	29% (4)
[3] High	45% (3,427)	44% (398)	46% (1,836)	44% (1,187)	43% (6)
(Missing)	1.7% (131)	0.5% (5)	0.2% (7)	0.3% (7)	89% (112)
Commuting methods					
Walking	14% (1,098)	18% (162)	14% (577)	13% (355)	22% (4)
Bicycle	10% (794)	10% (93)	11% (427)	10% (272)	11% (2)
Train/Bus	17% (1,322)	18% (162)	17% (702)	17% (454)	22% (4)
Car/Motorbike	58% (4,454)	55% (501)	58% (2,324)	60% (1,621)	44% (8)

(Missing)	1.5% (116)	0.1% (1)	0.1% (5)	<0.1% (2)	86% (108)
Area					
Osaka1	43% (3,326)	48% (438)	42% (1,712)	43% (1,169)	5.6% (7)
Osaka2	13% (1,020)	10% (96)	14% (562)	12% (319)	34% (43)
Kyoto	5.8% (452)	5.0% (46)	5.5% (223)	6.8% (183)	0% (0)
Shiga1	20% (1,585)	21% (193)	21% (834)	21% (558)	0% (0)
Shiga2	17% (1,332)	16% (146)	17% (704)	18% (475)	5.6% (7)
Others	0.9% (69)	0% (0)	0% (0)	0% (0)	55% (69)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
Alcohol intake					
Non-drinker	26% (1,992)	30% (272)	27% (1,086)	23% (630)	25% (4)
Occasional	20% (1,502)	21% (189)	19% (774)	20% (533)	38% (6)
<3days/week	21% (1,636)	21% (194)	21% (834)	22% (604)	25% (4)
>=3days/week	14% (1,087)	12% (111)	14% (575)	15% (400)	6.3% (1)
Everyday	19% (1,442)	16% (151)	19% (759)	20% (531)	6.3% (1)
(Missing)	1.6% (125)	0.2% (2)	0.2% (7)	0.2% (6)	87% (110)
Smoking status					
Never	48% (3,702)	55% (507)	49% (1,985)	44% (1,199)	69% (11)
Past	19% (1,478)	17% (158)	18% (729)	22% (590)	6.3% (1)
Current	32% (2,480)	27% (252)	33% (1,314)	34% (910)	25% (4)
(Missing)	1.6% (124)	0.2% (2)	0.2% (7)	0.2% (5)	87% (110)
Regular physical activity					

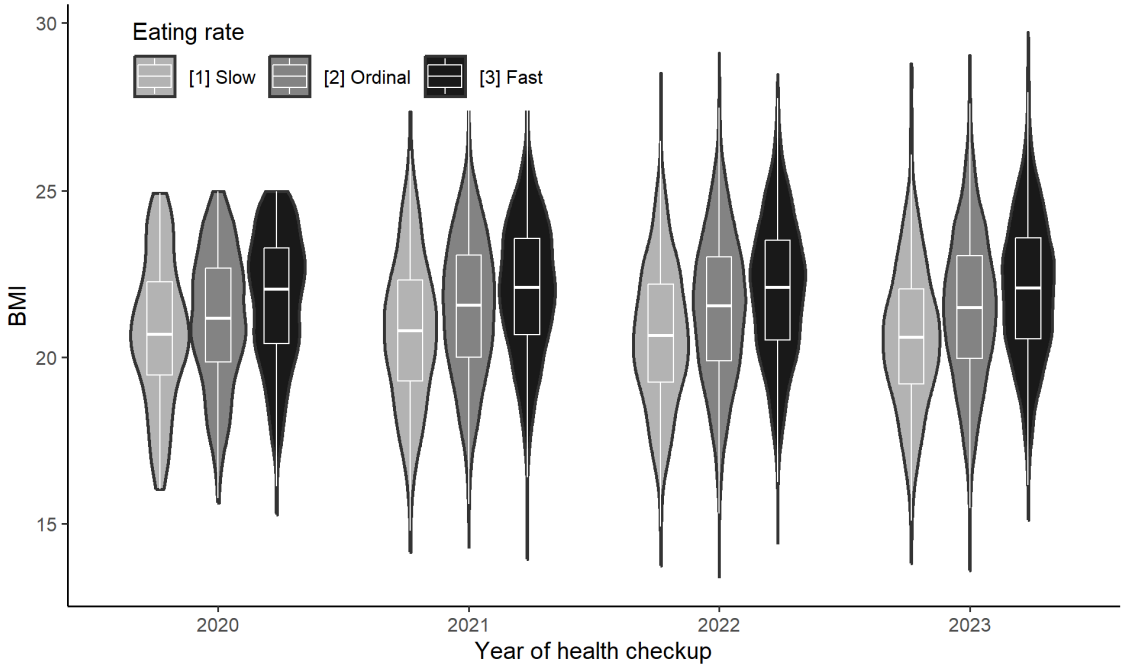
No	73% (5,619)	74% (676)	74% (2,975)	72% (1,952)	100% (16)
Yes	27% (2,044)	26% (241)	26% (1,056)	28% (747)	0% (0)
(Missing)	1.6% (121)	0.2% (2)	<0.1% (4)	0.2% (5)	87% (110)
BMI	21.50 (20.05, 22.98)	20.72 (19.23, 22.06)	21.37 (19.94, 22.88)	22.00 (20.53, 23.30)	21.17 (19.54, 22.63)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
SBP	115.00 (108.00, 123.00)	113.00 (105.00, 122.00)	115.00 (108.00, 123.00)	116.00 (109.00, 124.00)	113.50 (106.00, 124.75)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
DBP	74.00 (67.00, 81.00)	73.00 (66.00, 80.00)	74.00 (67.00, 80.00)	75.00 (68.00, 81.00)	69.50 (64.00, 79.00)
(Missing)	0% (0)	0% (0)	0% (0)	0% (0)	0% (0)
HbA1c	5.50 (5.30, 5.70)	5.50 (5.30, 5.70)	5.50 (5.30, 5.70)	5.50 (5.30, 5.70)	5.30 (5.10, 5.55)
(Missing)	31% (2,394)	34% (312)	31% (1,237)	30% (806)	31% (39)
Triglycerides	75.00 (55.00, 110.00)	72.00 (51.00, 105.00)	75.00 (56.00, 110.00)	76.00 (56.00, 111.00)	71.00 (56.00, 117.00)
(Missing)	31% (2,391)	34% (312)	31% (1,235)	30% (805)	31% (39)
HDL cholesterol	62.00 (53.00, 73.00)	64.00 (55.00, 75.00)	62.00 (53.00, 74.00)	62.00 (53.00, 72.00)	63.00 (54.50, 75.00)
(Missing)	31% (2,391)	34% (312)	31% (1,235)	30% (805)	31% (39)
LDL cholesterol	119.00 (100.00, 140.00)	116.00 (97.50, 136.00)	119.00 (100.00, 140.00)	120.00 (101.00, 140.00)	117.00 (100.00, 135.50)
(Missing)	31% (2,391)	34% (312)	31% (1,235)	30% (805)	31% (39)
Uric acid	5.90 (5.00, 6.60)	5.70 (5.00, 6.50)	5.80 (4.90, 6.60)	5.90 (5.20, 6.70)	6.00 (4.40, 6.80)
(Missing)	31% (2,393)	34% (312)	31% (1,236)	30% (806)	31% (39)

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The baseline characteristics and lifestyles during 2020 were assessed in the 2021 checkup, except for the clinical laboratory values evaluated in the 2020 checkup. The “overall” column refers to all participants combined, regardless of eating rate category.

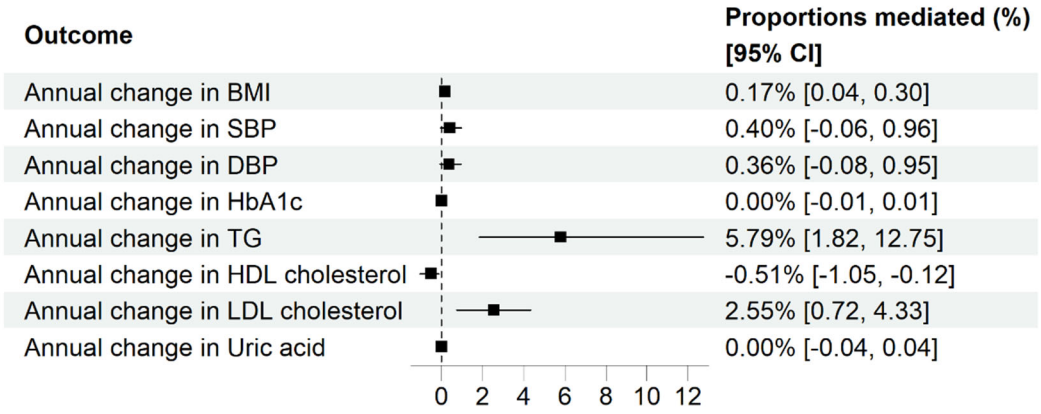
Supplementary Figures

Supplementary Figure 1. Transition of BMI distribution among each eating rate group



The box plot displays the 25th, 50th, and 75th percentiles as the bottom, middle, and top lines of the box, respectively. The whiskers extend to the upper limit of the third quartile plus 1.5 times the interquartile range and to the lower limit of the first quartile minus 1.5 times the interquartile range. The eating rate was assessed at each annual checkup and used to categorize participants into groups.

Supplementary Figure 2. Proportions of mediated impact of caloric intake on weight gain and other outcomes



Each point represents the estimated proportion of the impact of eating rate on annual changes in BMI and other outcomes that are mediated by caloric intake, with error bars indicating the 95% confidence intervals. The results suggest that, for all outcomes, the effect of eating rate mediated through caloric intake is relatively small.