Self-reported speed of eating and 7-year risk of type 2 diabetes mellitus in middle-aged Japanese men

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Abstract

Objective: This cohort study investigated the association between eating speed and the incidence of type 2 diabetes in middle-aged Japanese men.

Materials/Methods: Participants were 2,050 male employees of a metal products factory in Japan. We measured self-reported categorical eating speed. The incidence of diabetes was determined in annual medical examinations over a 7-year period. The association between eating speed and the incidence of diabetes adjusted for multiple variables (age, family history of diabetes, smoking, alcohol drinking, habitual exercise, and presence of hypertension and hyperlipidemia) was evaluated using Cox proportional hazards models.

Results: The prevalence of obesity (BMI ≥ 25 kg/m^2) across the categories of eating speed (slow, medium, and fast) was 14.6, 23.3, and 34.8%, respectively, and a faster eating speed was associated with a higher prevalence of obesity. During the study, 177 participants developed diabetes. Crude incidence rates (/1,000 person-years) across the three categories of eating speed were 9.9, 15.6, and 17.3, respectively. Multivariate-adjusted hazard ratios (95% CI) across the categories were 1.00 (reference), 1.68 (0.93-3.02), and 1.97 (1.10-3.55), respectively, and eating speed was associated with the risk of diabetes (p for trend = 0.030). After further adjustment for BMI, a significant association was not observed.

Conclusions: Eating speed was associated with the incidence of diabetes. Since these associations were not significant after adjusting for BMI, eating speed may act via its effect on body weight. Eating speed is a controllable risk factor, and eating slowly could be an acceptable lifestyle intervention for the prevention of diabetes mellitus.

Abbreviations

BMI, body mass index; HbA1c, glycated hemoglobin; JDS, the Japan Diabetes Society;

NGSP, the National Glycohemoglobin Standardization Program; HOMA-IR, homeostasis

model assessment of insulin resistance; DHQ, diet history questionnaire.

1. Introduction

The prevalence of type 2 diabetes mellitus is similar in Asian and Western countries even though the prevalence of obesity is lower in Asia [1]. The high incidence of diabetes in the relatively lean Asian population may be explained, in part, by the presence of more abdominal fat in Asians as compared with white people of similar body mass index (BMI) [2, 3]. Non-obese Asians who have low pancreatic beta cell function are also at high risk for diabetes [4-6]. Dietary factors, such as higher dietary glycemic index in Asian compared to Western people [7-14], may also be associated with a higher risk of diabetes [14].

Similar to a high glycemic index diet, fast eating speed is associated with postprandial hyperglycemia [15]. Recent epidemiological cross-sectional studies and longitudinal studies showed that eating speed is associated with obesity [16-21] and insulin resistance [22]. Fast speed of eating may cause diabetes mellitus by postprandial hyperglycemia, obesity, and insulin resistance; however, no studies have investigated the association between eating speed and the incidence of diabetes mellitus.

In this cohort study of middle-aged Japanese men, we examined the relationship between eating speed and 7-year incidence risk of diabetes mellitus.

2. Materials and Methods

2.1. Participants

The study participants were employees of a factory that produces zippers and aluminum sashes in Toyama Prefecture, Japan. Detailed information on the study population has been previously reported [6, 12, 14]. The Industrial Safety and Health Law in Japan requires that employers conduct annual health examinations for all employees. A test for diabetes mellitus

was conducted during annual medical examinations between 2003 and 2010. In 2003, 2,275 (89%) of 2,543 male employees aged 35–55 years received health examinations and responded to the diet survey. Of these 2,275 potential participants, 225 (10%) were excluded: 165 were diabetic or had high fasting plasma glucose ($\geq$ 126 mg/dL) or high glycated hemoglobin (HbA1c) levels ($\geq$ 6.5%) at the time of the baseline examination, 13 had a total daily energy intake below 500 kcal or above 5,000 kcal, and 47 did not participate in consecutive follow-up annual health examinations. Thus, 2,050 participants were included in the present study.

2.2. Data collection

The annual health examination included a medical history, physical examination, anthropometric measurements, and the measurement of fasting plasma glucose, fasting insulin, HbA1c, and serum lipid levels. Height was measured without shoes to the nearest 0.1 cm using a stadiometer. Weight was measured with participants wearing only light clothing and no shoes to the nearest 0.1 kg using a standard scale. BMI was calculated as weight/height^2 (kg/m^2). Blood pressure was measured using a mercury sphygmomanometer after the subject rested for 5 min in a seated position. All measurements were taken by trained staff.

Plasma glucose levels were measured enzymatically using an Abbott glucose UV test (Abbott Laboratories, Chicago, IL, USA), and plasma insulin levels were determined by radioimmunoassay (Shionogi Co., Tokyo, Japan). HbA1c was measured by high-velocity liquid chromatography using a fully automated hemoglobin A1c analyzer (Kyoto Daiichi Kagaku, Kyoto, Japan). Quality control of the HbA1c measurements was performed using the standard certified by the Japan Diabetes Society (JDS), and values of HbA1c were converted to the values of the National Glycohemoglobin Standardization Program (NGSP) using the formula provided by JDS; HbA1c (NGSP) = HbA1c (JDS) + 0.4 [23]. Total cholesterol and

triglycerides were measured using an enzymatic assay. HDL-cholesterol was measured using direct methods. Insulin resistance was calculated by the homeostasis model assessment (HOMA) method using the formula: HOMA-IR = fasting insulin (μU/mL) $\times$ fasting plasma glucose (mg/dL)/405 [24].

A questionnaire was used to collect information about smoking, alcohol drinking, habitual exercise, family history of diabetes, medical history of hypertension, dyslipidemia, diabetes, and the use of antidiabetic medication. Dietary habits, total energy intake (kcal/day), and total fiber intake (g/day) were assessed using a self-administered diet history questionnaire (DHQ) [25]. The DHQ was developed to estimate the dietary intakes of macronutrients and micronutrients for epidemiological studies in Japan. A detailed description of the methods used for calculating dietary intakes and the validity of the DHQ have been reported previously [25-27]. Estimates of dietary intake for 147 food and beverage items, energy, and nutrients were calculated using an ad hoc computer algorithm developed for the DHQ that was based on the Standard Tables of Food Composition in Japan [28]. In the DHQ, the speed of eating was self-reported by the answer to the question; "How fast is your rate of eating (speed of eating)?" chosen from five semi-quantitative categories; "very slow," "relatively slow," "medium," "relatively fast," and "very fast." The validity and reproducibility of the self-reported speed of eating data using the DHQ have been reported previously [17, 19]. Self-reported speed of eating showed a high level of agreement with that reported by a friend; the percentages of exact and adjunct categories of answers (for example, very fast and fast were regarded as agreed) were 46% and 47%, respectively, which indicated high levels of agreement between self- and friend-reported rates of eating [17]. Repeatability for self-reported eating speed was assessed by repeating the questionnaire survey after 1 year, and the k statistics were 0.63 in men and 0.67 in women [19].

2.3. Diagnosis of diabetes

Fasting plasma glucose and HbA1c were measured during the annual medical examinations. According to the definition of the American Diabetes Association [29] and the JDS [23], the diagnosis of diabetes was confirmed by at least one of the following observations: 1) a fasting plasma glucose concentration of $\geq$ 126 mg/dL, 2) a HbA1c (NGSP) of $\geq$ 6.5%, or 3) treatment with insulin or an oral hypoglycemic agent.

2.4. Statistical analysis

Since the numbers of participants who reported their speed of eating as "very slow" or "very fast" were small (n = 19 and 155, respectively), we divided the participants into three categories of speed of eating as follows: slow (very slow and relatively slow), medium, and fast (relatively fast and very fast).

The mean values at baseline examination, such as age, BMI, blood pressure, plasma glucose levels, and serum lipids, in each category of eating speed were determined. The mean of metabolic parameters adjusted for age and BMI in each category of eating speed were calculated through an analysis of covariance. Fasting insulin, HOMA-IR, and triglycerides were converted logarithmically for analysis. We calculated the adjusted rate ratios for the prevalence of obesity (BMI $\geq$ 25 kg/m^2) according to the three categories of eating speed using a multiple logistic regression analysis.

We calculated the crude incidence rates and HRs for diabetes according to the three categories of eating speed. The Cox proportional hazard model was used to calculate HRs. Adjustment for possible confounders was performed sequentially: for age (Model 1); plus family history

of diabetes (no, yes), alcohol consumption determined by the DHQ (nondrinker, occasional, consumed < 20 g/day, consumed ≥ 20 g/day), smoking status (never, ex-smoker, or current smoker), habitual exercise (no, yes), treatment of hypertension (no, yes), and treatment of dyslipidemia (no, yes) (Model 2); plus each of several stipulated variables (Models 2A-2D). Statistical analyses were conducted using the Statistical Package for the Social Sciences (SPSS version 12.0J; Tokyo, Japan). A p-value of < 0.05 was deemed statistically significant.

3. Results

3.1. Baseline examination

The mean participant age at baseline was 45.9 years and the mean BMI was 23.4 kg/m^2 (Table 1). Eating speed was associated with metabolic abnormalities; the participants who ate faster were younger and had a higher BMI, higher blood pressure, higher insulin resistance, higher triglycerides, and lower HDL-cholesterol. However, these cross-sectional associations between eating speed and metabolic abnormalities were not significant after adjustment for age and BMI.

The prevalence of obesity across the three categories of eating speed is shown in Table 2. The age-adjusted rate ratios of obesity in the medium and fast eating speed groups were 1.7 and 3.0, respectively (Model 1), and were significantly higher compared to the slow group. These associations were similar even after further adjusting for lifestyle factors (Model 2).

3.2. Follow-up study

During the 7-year follow up (11,311 person-years, mean follow-up time was 5.5 ± 1.8 years), we documented 177 cases of diabetes (15.6/1,000 person-years). Among these, 97 diagnoses were based on high fasting plasma glucose levels, 79 were based on high HbA1c, and one

participant had been treated with hypoglycemic medication.

The crude incidence rates (per 1,000 person-years) across the categories of eating speed from slow to fast were 9.9, 15.6, and 17.3, respectively (Table 3). In the age-adjusted model (Model 1), speed of eating was significantly associated with the incidence of diabetes (p for trend = 0.028), and the age-adjusted HR for the fast eating speed group was significantly higher as compared with the slow eating speed group. Further adjustments for a family history of diabetes, smoking, alcohol intake, habitual exercise, the presence of high blood pressure, and dyslipidemia at baseline (Model 2) did not affect the HRs. The relationships remained significant even after additional adjustments for dietary factors (Models 2B) and for HbA1c at baseline (Model 2C). However, the relationships were not significant after further adjustment for BMI (Model 2A).

4. Discussion

This study investigated the association between speed of eating and the incidence of type 2 diabetes in middle-aged Japanese men. The results indicated that self-reported speed of eating was associated with the incidence of diabetes, and faster speed of eating was associated with a higher risk. The associations between eating speed and the incidence of diabetes were not significant in models with additional adjustment for BMI. Eating speed was reported to be associated with body weight [16-21], and eating speed may affect the incidence of diabetes through its effect on body weight. Eating speed is controllable, and eating slowly would be an easy and acceptable target for lifestyle intervention to prevent type 2 diabetes mellitus.

In this study, the fast speed of eating group had a significantly higher risk of diabetes in

age-adjusted models or models adjusted for lifestyle factors, such as smoking, alcohol drinking, and habitual exercise. In models with additional adjustment for BMI, the associations between eating speed and the incidence of diabetes were not significant. Body weight has also been reported to be associated with speed of eating [16-21]. A fast eating speed might be associated with diabetes through its effects on body weight gain in a causal pathway, and adjustment for BMI could be an over-adjustment. Furthermore, higher BMI itself is a strong risk factor for type 2 diabetes, and it may be difficult to detect the effects of other risk factors beyond obesity.

In our baseline examination, fast eating was associated with a higher BMI and higher prevalence of obesity, which is consistent with previous studies [16-21]. Difference in total energy intake could be one of the mechanisms underlying the relationship between eating speed and BMI. Fast eating may cause overeating before the stomach senses fullness due to a lack of satiety in the hypothalamic system [30]. However, in our study and a previous study that evaluated eating speed and total energy intake [18, 22], a linear trend between eating speed and total energy intake was not observed although eating speed was linearly associated with BMI. A previous study indicated that relative under-reporting of energy intake increased with increasing BMI [31, 32], which may have affected our results. Furthermore, eating speed was associated with BMI even after adjustment for total energy intake [18], indicating that total energy intake may explain only part of the relationship between eating speed and BMI. Another potential mechanism underlying the association is insulin resistance. A fast eating speed could lead to a higher blood glucose/insulin response. This increased response may cause insulin resistance, which was associated with abdominal fat gain in a prospective study [33].

A previous study showed a significant positive association between eating speed and HOMA-IR in middle-aged Japanese men even after adjustment for BMI [22]. In our baseline examination, the variables associated with insulin resistance, such as triglycerides, fasting insulin, and HOMA-IR, were associated with eating speed in univariate models but not in BMI-adjusted models, and the association between eating speed and insulin resistance beyond obesity was controversial. The associations between eating speed and body weight have been evaluated by longitudinal studies [20, 21] as well as cross-sectional studies [16-19], and the overall evidence indicates that fast speed of eating would cause body weight gain. However, in the present and in a previous study, the association between eating speed and insulin resistance was evaluated cross-sectionally, and we could not evaluate the relationships beyond body weight changes. Laboratory studies have demonstrated that fast eating, or rapid nutrient delivery, increased serum insulin levels and decreased glucose disappearance rate [15], which supports the hypothesis that fast eating may directly affect insulin resistance. Further study is needed to evaluate how the risk factors for diabetes related to eating speed, such as obesity, insulin resistance, and postprandial glucose metabolism, would affect one another and cause type 2 diabetes.

The strengths of this study include a relatively large sample size, and the fact that it was the first study to examine the relationship between eating speed and the incidence of diabetes. Moreover, several previous cohort studies used information on incidence of diabetes collected from self-administered questionnaires, whereas our conclusions are based on more reliable data obtained from annual medical examinations and fasting blood glucose and HbA1c.There were some limitations to this study. First, the eating speed was self-reported, and it may not have reflected the objective speed of eating. However, some previous reports have shown that the validity and reproducibility of eating speed data as assessed by DHQ are good [17, 19].

Furthermore, many participants in the present study could compare their speed of eating to those of other people because they usually ate lunch together at the staff canteen. Second, the sample included only people who were employed. Poor health may exclude some individuals from working; thus, the prevalence of diabetes may be lower in our sample than in the general Japanese population. Third, we did not determine whether the diabetes that developed was type 1 or type 2. However, the study participants were middle-aged men and, as the condition was detected in an annual medical check-up, with relatively mild diabetes being found, it is most likely that the cases were type 2 diabetes. Furthermore, this study was observational; an interventional study may provide us with additional information on the causal relationship between eating speed and diabetes mellitus.

In conclusion, the speed of eating was associated with incidence of type 2 diabetes in middle-aged Japanese men. The speed of eating is a controllable risk factor, and eating slowly could be an acceptable lifestyle intervention for the prevention of type 2 diabetes mellitus.

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Table 1. Baseline characteristics of study participants according to speed of eating categories in 2,050 Japanese men

	All (n = 2,050)		Slow (n = 239)		Medium (n = 921)		Fast (n = 890)		P [a]	P [b]
			Speed of eating							
Age	45.9	± 6.0	47.0	± 6.0	46.5	± 6.0	45.1	± 5.9	< 0.001	
Height (cm)	169.5	± 6.0	169.0	± 6.4	169.1	± 6.0	170.1	± 5.8	< 0.001	
Weight (kg)	67.4	± 9.4	63.5	± 9.0	66.3	± 8.7	69.6	± 9.6	< 0.001	
Body mass index (kg/m^2)	23.4	± 2.9	22.2	± 2.8	23.1	± 2.8	24.0	± 2.8	< 0.001	
Systolic blood pressure (mmHg)	122.1	± 16.7	120.7	± 16.9	121.0	± 16.4	123.6	± 16.8	0.002	0.115
Diastolic blood pressure (mmHg)	79.1	± 12.0	78.2	± 12.0	78.4	± 11.7	80.0	± 12.1	0.008	0.163
Fasting plasma glucose (mg/dL)	92.6	± 9.6	91.3	± 9.6	92.7	± 9.4	92.9	± 9.8	0.091	0.375
HbA1c (%)	5.4	± 0.4	5.4	± 0.4	5.4	± 0.4	5.4	± 0.4	0.297	0.306
Plasma insulin (μU/mL)	5.0	(3.0-7.0)	4.0	(3.0-6.0)	5.0	(3.0-7.0)	5.0	(3.0-8.0)	< 0.001	0.161
HOMA-IR	1.12	(0.70-1.69)	0.89	(0.63-1.41)	1.12	(0.70-1.62)	1.18	(0.77-1.80)	< 0.001	0.395
Total cholesterol (mg/dL)	207.2	± 33.6	204.3	± 31.2	207.3	± 33.6	207.8	± 34.3	0.368	0.819
Triglycerides (mg/dL)	102	(69-152)	88	(61-132)	98	(68-147)	106	(73-167)	< 0.001	0.161
HDL-cholesterol (mg/dL)	58.1	± 14.6	61.7	± 16.0	58.5	± 14.6	56.6	± 14.0	< 0.001	0.155
Total energy intake (kcal/day)	2201	± 611	2208	± 607	2175	± 594	2226	± 628	0.201	
Dietary fiber intake (g/day)	10.9	± 4.4	11.3	± 4.5	10.8	± 4.4	10.9	± 4.3	0.249	
Family history of diabetes (%)	13.5		16.3		12.4		13.9		0.252	
Smoking status (%)									< 0.001	
Nonsmoker	26.0		23.8		29.8		22.8			
Ex-smoker	19.7		22.6		15.6		23.0			
Current smoker	54.3		53.6		54.6		54.2			
Alcohol drinking (%)									0.181	

Never	16.8	18.8	16.6	16.5	
Occasional	10.3	5.4	10.3	11.6	
Drinking < 20 g/day	30.4	29.3	31.2	30.0	
Drinking ≥ 20 g/day	42.4	46.4	41.9	41.9	
Habitual exercise - Yes (%)	27.3	26.8	28.2	26.5	0.702
Medical treatment - Hypertension (%)	5.8	5.0	6.9	4.7	0.110
Medical treatment - Dyslipidemia (%)	4.1	0.8	4.1	4.9	0.018

Data are mean ± standard deviation, median (interquartile range), or %.

[a] One-way analyses of variance for continuous variables and chi-squared test for categorical variables.

[b] One-way analyses of covariance after adjusting for age and body mass index.

Table 2. Prevalence and adjusted rate ratio of obesity according to speed of eating categories in 2,050 Japanese men

| | Speed of eating | | | | | P for trend |
	Slow		Medium		Fast	
Prevalence of obesity (%)	14.6		23.3		34.8	
Rate ratio of obesity						
Model 1	1.00	(reference)	1.78	(1.21-2.63)	3.16 (2.15-4.64)	<0.001
Model 2	1.00	(reference)	1.73	(1.17-2.57)	3.07 (2.08-4.53)	<0.001

Model 1, adjusted for age; Model 2, adjusted for age, family history of diabetes, smoking, alcohol drinking, habitual exercise, and presence of hypertension and hyperlipidemia at baseline.

Multiple logistic regression models were used for the analyses.

Table 3. Incidence and adjusted hazard ratio of type 2 diabetes according to speed of eating categories in 2,050 Japanese men

	Speed of eating					P for trend
	Slow		Medium		Fast	
N	239		921		890	
Number of incident cases	13		80		84	
Person-years of follow-up	1308		5120		4850	
Incidence rate (/1,000 person-years)	9.9		15.6		17.3	
Hazard ratio (95%CI)						
Model 1	1.00	(reference)	1.64	(0.91 - 2.95)	1.96	(1.09 - 3.51)	0.028
Model 2	1.00	(reference)	1.68	(0.93 - 3.02)	1.97	(1.10 - 3.55)	0.030
Model 2A; Body mass index	1.00	(reference)	1.43	(0.79 - 2.58)	1.47	(0.82 - 2.66)	0.357
Model 2B; Total energy intake, dietary fiber intake	1.00	(reference)	1.69	(0.94 - 3.05)	1.98	(1.10 - 3.56)	0.030
Model 2C; HbA1c	1.00	(reference)	1.56	(0.86 - 2.82)	1.92	(1.06 - 3.47)	0.027
Model 2D; Fasting plasma glucose	1.00	(reference)	1.43	(0.79 - 2.56)	1.71	(0.95 - 3.10)	0.069

Model 1, adjusted for age; Model 2, adjusted for age, family history of diabetes, smoking, alcohol drinking, habitual exercise, and presence of hypertension and hyperlipidemia at baseline.

Models 2A-2D, adjusted for Model 2 variables + each stipulated variable.

The Cox proportional hazard model was used for the analyses.