

—Short Communications—

Age-Related Changes in Postprandial Plasma Glucose in Type 2 Diabetes

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Glucose tolerance is known to decrease with advancing age. Spence¹ in 1920 was the first to document that the glucose metabolism was impaired in subjects over the age of 60 yr. Since this initial observation, numerous reports have appeared showing that glucose tolerance declines as a function of age. The decline begins in the third or fourth decade of life and continues through the entire adult life span². This age-related decline in the glucose metabolism appears to have only a small effect on the fasting plasma glucose concentration, which rises by 1 mg/dl per decade. On the other hand, following oral glucose ingestion, the 1h plasma glucose response has been shown to increase by 4~14 mg/dl (mean=9 mg) per decade and the 2h plasma glucose value by 1~11 mg/dl (mean=5 mg/dl) per decade³. Therefore, two-thirds of elderly diabetic patients have postchallenge hyperglycemia during oral glucose tolerance tests without fasting hyperglycemia. Some investigators say that elderly type 2 diabetic patients with fasting hyperglycemia also have increased postprandial hyperglycemia relative to their younger counterparts⁴. But, whether or not this is true remains to be proven.

Daily blood glucose profiles were measured in 98 type 2 diabetic patients treated with diet alone. The subjects were divided into two groups according to whether they were <65 years old (middle-aged group, mean age; 54.7 ± 8.3 y old) or ≥65 years old (elderly group, mean age; 72.8 ± 5.7 y old). They were considered to have overt diabetes only if their fasting

plasma glucose concentrations were greater than 126 mg/dl on 2 separate occasions⁵. All patients were admitted to our hospital and were prescribed a weight-maintaining diet (25~30 kcal/kg standard body weight). Patients with before breakfast plasma glu-

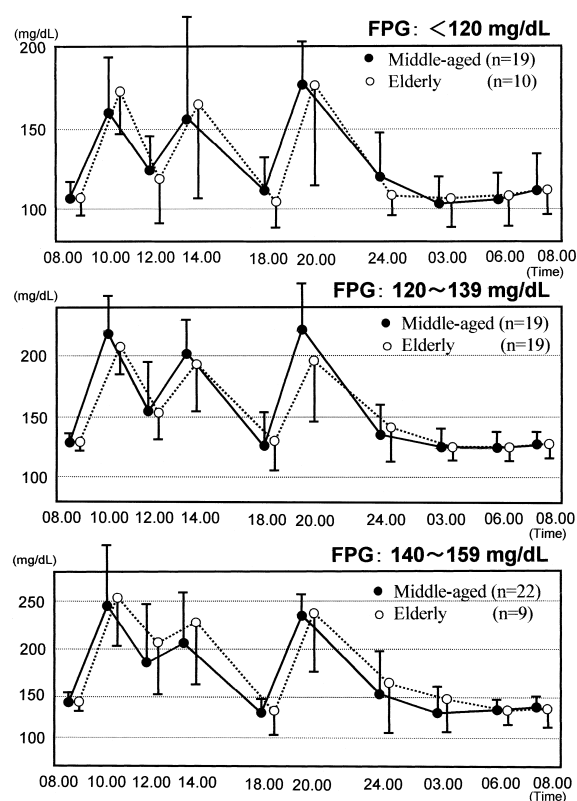


Fig. 1 Daily plasma glucose profiles during treatment with diet alone in elderly (≥65 yr.) diabetic patients and in middle-aged (<65 yr.) diabetic patients. Mean ± SD

Table 1 Standardized partial correlation (r) and F values between plasma glucose levels and explanatory variables in backward stepwise multiple regression analysis (F value; 2.0 or more)

Dependent variables	Explanatory variables					
	Age	08.00h PG	Urinary CPR/day	Duration of diabetes	BMI	24h Ccr
10.00h PG	—	0.70 (65.5) ***	—	—	- 0.14 (2.5)	—
14.00h PG	—	0.47 (25.3) ***	—	0.34 (13.0) ***	—	—
20.00h PG	—	0.47 (20.7) ***	—	0.15 (2.1)	- 0.24 (5.4) *	—

* p < 0.05, *** p < 0.0001, PG: plasma glucose

r (F value)

cose concentrations ≥ 160 mg/dl were excluded. Plasma glucose concentrations were determined at 8.00 (before breakfast), 10.00, 12.00 (before lunch), 14.00, 18.00 (before dinner), 20.00, 24.00, 3.00, 6.00, 8.00 hours. The subjects were divided into 3 subgroups, according to their 8.00 hours plasma glucose values (≤ 119 mg/dl, 120~139 mg/dl, 140~159 mg/dl). Plasma glucose concentrations were measured by the autoanalyzer method.

Statistical analysis was carried out using the Student t test. Backward stepwise multiple regression analysis was used to investigate the relationship between postprandial plasma glucose concentrations and explanatory variables. Explanatory variables were as follows: age, 8.00 hours plasma glucose value, daily urinary excretion of CPR, duration of diabetes, BMI and 24 h Ccr. Data in the text, tables and figures are expressed as mean \pm SD.

As shown in **Fig. 1**, daily plasma glucose profiles did not differ between elderly patients and middle-aged patients in any of the three groups. **Table 1** shows the standardized partial correlation coefficients and F values between postprandial plasma glucose levels and explanatory variables. The 8.00 hours plasma glucose value positively correlated with plasma glucose values at 10.00, 14.00 and 20.00 hours. Duration of diabetes positively correlated with plasma glucose values at 14.00 and 20.00 hours. BMI negatively correlated with plasma glucose values at 10.00 and 20.00 hours. No association was observed between age and postprandial plasma glucose values.

Daily plasma glucose profiles did not differ between elderly patients and middle-aged patients in overt type 2 diabetes mellitus. The glucose intolerance of aging appears to be due to an increase in peripheral insulin resistance that occurs with aging. The exact

mechanism for this defect remains unknown, but is probably associated with post receptor defects in insulin action². Abnormalities of insulin release, insulin binding and suppression of hepatic glucose production by insulin do not seem to play a major part in the glucose intolerance associated with aging⁶. On the other hand, overt type 2 diabetes is characterized by excessive increases in plasma glucose levels after ingestion of either glucose or meals, elevations superimposed on fasting hyperglycemia. These large glycemic excursions result from a failure in inhibition of hepatic glucose production in combination with a reduction in glucose uptake by the muscles^{7,8}. These abnormalities result from a combination of inadequate insulin secretion and insulin resistance by both the liver and muscles. The failure to appropriately suppress plasma glucagon levels probably contributes to persistently elevated hepatic glucose production. Thus the underlying pathophysiologic mechanisms of type 2 diabetes mellitus suggest that there are both qualitative and quantitative differences between the glucose intolerance of aging and overt type 2 diabetes. There is no evidence that the age-related increase in postprandial blood glucose levels is found in overt elderly type 2 diabetes as far as we are aware.

In conclusion, this study suggests that the age-related increase in postprandial glucose levels has no clinical significance in overt type 2 diabetes.

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