Effects of Vegetable Oils and C₁₈- Unsaturated Fatty Acids on Plasma Ethanol Levels and Gastric Emptying in Ethanol-Administered Rats

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Summary The effects of vegetable oils (soybean oil and coconut oil) and C₁₈-unsaturated fatty acids (oleic acid, C₁₈:₁; linoleic acid, C₁₈:₂; linolenic acid, C₁₈:₃) on plasma ethanol levels in male rats (6 weeks old) were investigated. Vegetable oils decreased and delayed the peak of plasma ethanol concentration: a dose-response to vegetable oils was observed in the concentration and time to maximum concentration of plasma ethanol but no change in disappearance time. These phenomena were observed in two conditions: 1) oral administration of vegetable oils before oral intubation of ethanol and 2) simultaneous oral administration of vegetable oils and ethanol. Similar responses were obtained in three C₁₈-unsaturated fatty acids. No changes in hepatic alcohol and aldehyde dehydrogenase isozyme (high $K_m$ and low $K_m$) activities were observed. The remaining rate of ethanol in stomach was significantly higher with administration of vegetable oils or linoleic acid. A high negative correlation between the maximum plasma ethanol concentration and the remaining rate of ethanol in stomach was found. These results suggest that the slowing of the gastric emptying is a major mechanism for the decreasing and delaying effects on plasma ethanol levels by vegetable oils. The present paper also suggests that fatty acids may participate in the decreasing and delaying actions on the peak of plasma ethanol concentration by vegetable oils.

Key Words vegetable oil, soybean oil, coconut oil, fatty acids, ethanol, ethanol metabolism, gastric emptying, alcohol dehydrogenase, aldehyde dehydrogenase, rats

It is well known that many factors change the blood ethanol response when ethanol is administered (1, 2). For instance, it has been reported that a significant decrease of blood ethanol level induced by pyruvate, glutamine, or alanine is due to the acceleration of the ethanol metabolism in the liver by decreasing the ratio of NAD to NADH (1–3), as a result of activation of mitochondrial electron transport...
by ADP following ethanol treatment (4). The factors which control the absorption, distribution and metabolism of ethanol in vivo, however, are unclear (5–9). To obtain the complete elucidation of the variation mechanisms of ethanol metabolism in vivo, it is essential to study the factors which control blood ethanol concentration. However, little is known of the underlying mechanism by which may factors change ethanol metabolism in either human or experimental animals (7–9). For intrinsic understanding of this purpose, it is crucial to examine: 1) the change of the absorption rate of ethanol through gastrointestinal tract, 2) the change of the rate of ethanol metabolism in the liver, 3) the change of ethanol clearance in the body, or 4) the change of the rate of the excretion of ethanol from urine and sweat glands.

Recently, Lieber (10) pointed out that the hepatic alcohol dehydrogenase [ADH: EC 1.1.1.1] and the hepatic aldehyde dehydrogenase [ALDH: EC 1.2.1.3] play an important role in the biosynthesis of bile acids from cholesterol. Therefore, the linkage between ethanol metabolism and the biosynthesis of bile acids has attracted attention (11, 12). Previously, we reported the decreasing and delaying actions of bovine bile on plasma ethanol levels in rats (13). As a possible mechanism for the actions, we proposed the slowing of gastric emptying and/or the decrease of the intestinal absorption rate of ethanol (13). Further, our previous paper suggested that bile acid, which was contained in about 18.5% of bovine bile, may participate in the variation effect of bovine bile on plasma ethanol levels (13).

According to our unpublished analysis, about 37% of total fatty acids was comprised in bovine bile. The contribution and magnitude of each substance to the changing actions of plasma ethanol level by bovine bile are not clear. It is very important that these possibilities are studied from a physiological point of view. Therefore, we planned to examine effects of fats and fatty acids on plasma ethanol levels. Effects of fats and fatty acids on ethanol metabolism, however, have not been elucidated systematically (14–18).

This paper describes, thus, 1) effects of timing of the administration of vegetable oils or C18-unsaturated fatty acids on the plasma ethanol concentration in rats, 2) effects of vegetable oils or C18-unsaturated fatty acids on the time course of plasma ethanol concentration, 3) effects of soybean oil on ADH and ALDH isozyme (high $K_m$ and low $K_m$) activities in the rat liver, and 4) effects of vegetable oils and linoleic acid (C18:2) on the gastric emptying rate of ethanol.

**EXPERIMENTAL**

**Animals.** Specific-pathogen-free male Sprague Dawley rats (6 weeks old: 157 ± 9 g (n = 182), in M ± SD; Japan SLC, Inc., Hamamatsu) were used. The animals were normally housed in the cage maintained at a temperature of 24 ± 1°C and at a relative humidity of 55 ± 5%. Lighting was controlled automatically for 8:00 to 20:00. Animal diet (CE-2 type, CLEA Japan, Tokyo) and once-boiled water were given to the rats ad libitum. The rats were starved to 17:00 and deprived of diet for 16 h and water for 1 h before the experiment. The present studies were carried out...
Conditions of administration of vegetable oils or C₁₈-unsaturated fatty acids and ethanol. 1) The timing of oral administrations: The following three conditions were employed (13): (1) oral administration of the soybean oil (1.0 g·kg⁻¹ BW) or linoleic acid (0.5 g·kg⁻¹ BW) 30 min before oral intubation of ethanol (1.0 g·kg⁻¹ BW, as 20% solution), (2) simultaneous oral administration of soybean oil (1.0 g·kg⁻¹ BW) and ethanol (1.0 g·kg⁻¹ BW), and (3) oral administration of soybean oil (1.0 g·kg⁻¹ BW) 30 min after oral intubation of ethanol (1.0 g·kg⁻¹ BW). In the control rats, 0.9% NaCl solution instead of the soybean oil or linoleic acid was orally given with the solution volume being equal, via a stomach tube to the rats.

2) Intraperitoneal injection of ethanol: We also determined the time course of plasma ethanol concentration when ethanol (1.0 g·kg⁻¹ BW) was intraperitoneally administered 30 min after oral intubation of soybean oil or coconut oil (1.0 g·kg⁻¹ BW) and linoleic acid (0.5 g·kg⁻¹ BW) to the rat. After the oral intubation or intraperitoneal injection of ethanol, plasma ethanol concentrations in rats were assayed at constant interval (13, 20).

Dose of vegetable oils and linoleic acid. Rats received 0.125, 0.25, 0.5, 1.0, or 2.0 g·kg⁻¹ BW of soybean oil and 0.25, 0.5, or 1.0 g·kg⁻¹ BW of coconut oil by intragastric intubation. Linoleic acid (C₁₈:2) of 0.125, 0.25, 0.5, or 1.0 g·kg⁻¹ BW was administered to rats by intragastric intubation. Thirty minutes after intragastric intubation, ethanol (1.0 g·kg⁻¹ BW) was administered orally via a stomach tube to the rats. In the control rats, an equivalent volume of 0.9% NaCl solution was given instead of vegetable oils or linoleic acid in the same manner.

Analysis of plasma ethanol concentration. After the administration of ethanol to rats, blood samples (~25 µl) were collected with heparinized microcapillary tubes from the tail vein or artery, at constant intervals, and centrifuged promptly at 10,000 rpm for 3 min at 2°C. During experiments, we performed with the least possible pain or discomfort to the animals (19). The supernatant fraction (=plasma) was used as the sample for measuring ethanol concentration. The plasma ethanol concentrations were analyzed by a gas chromatograph (model 163, Hitachi Co., Tokyo) with a flame ionization detector. The analytical conditions were as follows (13, 20): column packing, PEG-20 M (Gasukuro Kogyo Co., Tokyo); stainless steel column, 2 mm × 2 m; column temperature, 80°C; flow rate of nitrogen gas, 25 ml·min⁻¹; flow rate of hydrogen gas, 25 ml·min⁻¹; air pressure, 0.5 kg·cm⁻²; injection temperature, 90°C; and injection volume, 1 µl.

Parameters for the time course of plasma ethanol concentration. To analyze quantitatively the time course of plasma ethanol concentration, as shown schematically in Fig. 1, we introduced the following five empirical parameters (13): C(max), T(max), T(disapp), K(app), and Σ.

Assays of liver cytosolic alcohol dehydrogenase (ADH) and mitochondrial aldehyde dehydrogenase (ALDH) isozyme (high Kₘ and low Kₘ) activities. Enzyme activities were determined spectrophotometrically; ADH at 37°C and pH 9.0 with
Fig. 1. Schematic representation of five empirical parameters estimated from the time course of plasma ethanol concentration. C\text{max} (in %): maximum ethanol concentration; T\text{max} (in min): the time to reach maximum ethanol concentration; T\text{disapp} (in min): disappearance time of plasma ethanol; k_{\text{app}} (in min\text{ }^{-1}): apparent first order rate constant of plasma ethanol elimination process; \Sigma (in arbitrary unit): integral area under the time course of plasma ethanol concentration. p[EtOH] shows plasma ethanol concentration.

ethanol as substrate (21) and ALDH (22) as described earlier (13, 20). In ALDH assay, high $K_m$ isozyme activity was calculated by subtracting low $K_m$ enzyme activity from the combined activity. Protein contents of the subcellular fractions were determined by the method of Lowry et al. (23).

**Measurements of gastric emptying.** The soybean oil (1.0 g·kg\textsuperscript{-1} BW), coconut oil (1.0 g·kg\textsuperscript{-1} BW), or linoleic acid (0.5 g·kg\textsuperscript{-1} BW) were administered to rats orally. Thirty minutes after the intubation, ethanol (1.0 g·kg\textsuperscript{-1} BW) was administered orally (13). In the control group, 0.9\% NaCl solution of an equivalent volume was administered orally to rats. The rats were decapitated 20 min after intubation of ethanol. The stomach of the rat was excised promptly with ligations at the cardia and the pylorus. After weighing the stomach with contents, ethanol concentration in stomach was analyzed with a gas chromatograph as described already. The samples for analyzing plasma ethanol concentration were collected from a cervical artery, with decapitation. The remaining rate of ethanol in stomach was calculated using the following equation (13, 20): (content of ethanol in stomach/total content of ethanol administered) × 100.

**Analysis of fatty acids in vegetable oils.** The analysis of fatty acid in vegetable oils was carried out using the method of Aveldano et al. (24).

**Chemicals.** Ethanol used was super special grade (Wako Pure Chemical Co., Tokyo). Soybean oil, coconut oil, and three fatty acids (oleic acid: C\textsubscript{18:1}; linoleic acid: C\textsubscript{18:2}; linolenic acid: C\textsubscript{18:3}) were purchased from Sigma Chemical Co. (St. Louis). \textbeta;NAD was obtained from Oriental Yeast Co. (Tokyo). All other chemicals used were of reagent grade and used without further purification.

**Statistical analysis.** Data were expressed as M±SE. The statistical significance of the data was evaluated by Student’s t-test for unpaired comparison or Dunnett’s test. Differences were considered significant when $p$ was < 0.05.
RESULTS

Effects of the timing of oral administration of vegetable oils on plasma ethanol level

Figure 2A shows the time courses of the plasma ethanol concentration when soybean oil (1.0 g·kg⁻¹ BW) was administered orally 30 min before oral intubation of ethanol (1.0 g·kg⁻¹ BW). In this condition, as shown in “Before” of Fig. 3A–D, the soybean oil decreased the Cmax and Σ to 0.72 and 0.75 times, respectively, and delayed the Tmax to 1.92 times compared with the control. After 220 min of ethanol administration, the contents of ethanol remaining in the stomach were assayed. The ethanol was not detected. Further, we observed the inner walls of stomach in full details, but no definite differences between the soybean oil and the control groups were observed. Similar results, as shown in Fig. 2B, were also observed by the concurrent intubation of ethanol and soybean oil. As shown in “Simultaneous” of Fig. 3A–C, soybean oil (1.0 g·kg⁻¹ BW) decreased the Cmax to 0.84 times and delayed the Tmax to 1.84 times compared with the control, whereas Tdisap showed no significant changes with the dose of soybean oil. Figure 2C and “After” of Fig. 3 show the time courses of the plasma ethanol concentration when soybean oil 30 min after oral intubation of ethanol (1.0 g·kg⁻¹ BW) was administered orally. Both Cmax and Tmax did not change with the dose of soybean oil. Tdisap also did not change.

Effects of dose of the soybean oil on the plasma ethanol levels

Figure 4 shows the time course of the plasma ethanol concentrations when 0, 0.25, 0.50, or 1.0 g·kg⁻¹ BW of the soybean oil were administered orally 30 min before oral intubation of ethanol (1.0 g·kg⁻¹ BW). The data of 2.0 g·kg⁻¹ BW of the soybean oil were similar with result of 1.0 g·kg⁻¹ BW. Therefore, we did not

Fig. 2. Effects of soybean oil on the time course of plasma ethanol concentration under various conditions. ○, control; ●, soybean oil administration group. A: Soybean oil (1.0 g·kg⁻¹ BW) was administered orally 30 min before oral intubation of ethanol (1.0 g·kg⁻¹ BW). B: Soybean oil (1.0 g·kg⁻¹ BW) and ethanol (1.0 g·kg⁻¹ BW) were simultaneously administered orally. C: Soybean oil (1.0 g·kg⁻¹ BW) was administered to the rats orally 30 min after intubation of ethanol (1.0 g·kg⁻¹ BW). p[EtOH]: plasma ethanol concentration. Each value is represented as M±SE (n = 6–12 at each group).
Fig. 3. Effects of the timing of oral administration on plasma ethanol parameters. All data were estimated from each curve shown in Fig. 2. A, $C_{\text{max}}$; B, $T_{\text{max}}$; C, $T_{\text{disap}}$; and D, $\Sigma$. Before, Simultaneous, and After are as A, B, and C in Fig. 2, respectively. All values are shown as M±SE. *$p<0.05$, **$p<0.01$, and ***$p<0.001$ (vs. Control).

Fig. 4. Time courses of plasma ethanol concentration for various dosages of vegetable oils. Ethanol (1.0 g·kg$^{-1}$·BW$^{-1}$) was administered orally to the rats 30 min after gastric intubation of soybean oil (A) and coconut oil (B). Each plasma sample was prepared from tail blood. A: The dose (g·kg$^{-1}$·BW$^{-1}$) of the soybean oil=0 (n=10), 0.25 (n=6), 0.5 (n=6), and 1.0 (n=12) for ◦, ●, △, and ▲, respectively. The data of 2.0 g·kg$^{-1}$·BW$^{-1}$ of the soybean oil (n=6) are omitted in the figure. B: The dose (g·kg$^{-1}$·BW$^{-1}$) of the coconut oil=0 (n=19), 0.5 (n=5), and 1.0 (n=5) for ◦, ●, and △, respectively. All data are shown as M±SE.

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Fig. 5. Dose-response of soybean oil to each plasma ethanol parameter. A: \( C_{\text{max}} \), B: \( T_{\text{max}} \), C: \( T_{\text{disp}} \), and D: \( \Sigma \), integral area under the time course of plasma ethanol concentration. All data were estimated from each curve shown in Fig. 4. Values are shown as M±SE.

show the data in Fig. 4. The dose-dependent decrease in \( C_{\text{max}} \) and \( \Sigma \), and delay of \( T_{\text{max}} \) were observed except the dose of 2.0 g·kg\(^{-1}\) BW. \( T_{\text{disp}} \) was insensitive to the change of the soybean oil dosage. Figure 5 shows the effects of various dosage of soybean oil on \( C_{\text{max}} \) (A), \( T_{\text{max}} \) (B), \( T_{\text{disp}} \) (C), and \( \Sigma \) (D) as plotted against the soybean oil dose. The soybean oil of 0.25, 0.50, 1.0, and 2.0 g·kg\(^{-1}\) BW decreased the \( C_{\text{max}} \) to 0.86 (\( p < 0.01 \)), 0.83 (\( p < 0.01 \)), 0.72 (\( p < 0.001 \)), and 0.69 (\( p < 0.001 \)) times, respectively, compared with control value. Similar phenomenon was also observed in the \( \Sigma \) values. The \( T_{\text{max}} \) increased with the increase of soybean oil dose in the range of 0.25 to 1.0 g·kg\(^{-1}\) BW. The soybean oil of 0.25, 0.50, 1.0, and 2.0 g·kg\(^{-1}\) BW delayed the \( T_{\text{max}} \) to 1.26 (\( p < 0.01 \)), 1.62 (\( p < 0.001 \)), 1.92 (\( p < 0.001 \)), and 1.91 (\( p < 0.001 \)) times compared with that estimated in soybean oil-free rats, respectively. The \( T_{\text{disp}} \) was independent of various soybean oil dose. Although not shown in figure, \( k_{\text{(app)}} \) values were constant (1.4–1.5 \( \times 10^{-2} \) min\(^{-1} \)) in the dose range up to 2.0 g·kg\(^{-1}\) BW of soybean oil.

As seen in Table 1, the fatty acid composition of the soybean oil was in the following order: linoleic acid (54.8%) > oleic acid (20.9%) > palmitic acid (12.8%) > linolenic acid (7.0%) > stearic acid (3.5%) > myristic acid (0.3%). Therefore, we examined whether C\(_{18}\)-unsaturated fatty acids such as oleic acid, linoleic acid, and linolenic acid affect the plasma ethanol levels. The results are shown in Fig. 6. These fatty acids decreased and delayed the peak of plasma ethanol concentration (Fig. 6). Further, as shown in Figs. 6 and 7, linoleic acid of 0.125, 0.25, 0.5, and 1.0 g·kg\(^{-1}\) BW decreased the \( C_{\text{max}} \) to 0.92 (\( p < 0.05 \)), 0.85 (\( p < 0.01 \)), 0.72 (\( p < 0.001 \)), and 0.69 (\( p < 0.001 \)) times, respectively, compared with the
Table 1. Fatty acid composition of soybean oil and coconut oil (%).

<table>
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<th>Fatty acid</th>
<th>Soybean oil</th>
<th>Coconut oil</th>
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<tbody>
<tr>
<td>Capric acid (C_{10:0})</td>
<td>—</td>
<td>6.3</td>
</tr>
<tr>
<td>Lauric acid (C_{12:0})</td>
<td>—</td>
<td>46.0</td>
</tr>
<tr>
<td>Myristic acid (C_{14:0})</td>
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<td>18.4</td>
</tr>
<tr>
<td>Palmitic acid (C_{16:0})</td>
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<td>7.8</td>
</tr>
<tr>
<td>Stearic acid (C_{18:0})</td>
<td>3.5</td>
<td>2.1</td>
</tr>
<tr>
<td>Oleic acid (C_{18:1})</td>
<td>20.9</td>
<td>4.7</td>
</tr>
<tr>
<td>Linoleic acid (C_{18:2})</td>
<td>54.8</td>
<td>1.3</td>
</tr>
<tr>
<td>Linolenic acid (C_{18:3})</td>
<td>7.0</td>
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Fig. 6. Time courses of plasma ethanol concentration for three kinds of C_{18}-unsaturated fatty acids. Experimental conditions are as in Fig. 4. The dose (g·kg^{-1} BW) of linoleic acid (C_{18:2}) = 0 (n = 10), 0.125 (n = 8), 0.25 (n = 8), and 0.50 (n = 9) for □, ●, △, and ▲, respectively. These data are shown in the center. The data of the linoleic acid = 1.0 g·kg^{-1} BW are omitted for non-dose-dependence. The dose of oleic acid (C_{18:1}) and linolenic acid (C_{18:3}) = 0 and 0.5 g·kg^{-1} BW for □ and ▲, respectively. All data are shown as M±SE.

The □ values also showed dose-dependent decrease by linoleic acid except the dose of 1.0 g·kg^{-1} BW. On the other hand, 0.125, 0.25, 0.5, and 1.0 g·kg^{-1} BW of linoleic acid delayed $T_{\text{max}}$ to 1.17 ($p < 0.01$), 1.39 ($p < 0.001$), 1.77 ($p < 0.001$), and 1.86 ($p < 0.001$) times compared with the control, respectively (Fig. 7). The $T_{\text{disap}}$ was independent of the dosage of linoleic acid. These results were consistent with those obtained in the soybean oil, as shown in Figs. 4 and 5.

Effects of soybean oil on ADH and ALDH isozyme (high $K_m$ and low $K_m$) activities

Although data are not shown, there was no significant change in the ADH and ALDH isozyme (high $K_m$ and low $K_m$) activities when the soybean oil group was statistically compared with the control.

Effect of vegetable oils or linoleic acid on plasma ethanol levels when ethanol was injected intraperitoneally

We analyzed the time courses of plasma ethanol concentrations when ethanol

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Fig. 7. Dose-response of linoleic acid to each parameter. A, B, C, and D are as in Fig. 5. All data were estimated from each curve shown in Fig. 6. Experimental conditions are as in Fig. 4. Values are shown as M±SE.

(1.0 g·kg⁻¹ BW) was intraperitoneally administered 30 min after oral intubation of soybean oil (1.0 g·kg⁻¹ BW), coconut oil (1.0 g·kg⁻¹ BW), or linoleic acid (0.5 g·kg⁻¹ BW). In these conditions, plasma ethanol levels were independent of these substances (data not shown).

**Effects of vegetable oils or linoleic acid on gastric emptying**

As shown in Figs. 2, 4, and 6, more effective time of the soybean oil, coconut oil, or linoleic acid to plasma ethanol level was 20–40 min after the intubation of ethanol. Therefore, the gastric emptying of ethanol was analyzed quantitatively at 20 min after intubation of ethanol. Vegetable oils (1.0 g·kg⁻¹ BW) or linoleic acid (0.5 g·kg⁻¹ BW) was orally administered 30 min before oral intubation of ethanol (1.0 g·kg⁻¹ BW); the effects of these substances on the gastric emptying are shown in Fig. 8. Clearly, a significant decrease of plasma ethanol concentration was observed in the soybean oil group (Fig. 8A). This result approximately agreed with that of Figs. 2–5. As shown in Fig. 8B, the stomach content of the soybean oil group was 2.24 times higher than that of the control. Ethanol concentration in stomach was 1.85 times higher in the soybean oil group than in the control (Fig. 8C). The remaining rate of ethanol in stomach of the soybean oil group was 4.0 times higher than the control (Fig. 8D), suggesting slowing of gastric emptying induced by vegetable oils. Similar results were observed in the case of coconut oil or linoleic acid (data not shown). In these conditions, as shown in Fig. 9, plasma ethanol concentration was correlated highly with stomach content (A), ethanol
Fig. 8. Effects of soybean oil on plasma ethanol concentration (A), stomach content (B), ethanol concentration in stomach (C), and remaining rate of ethanol in stomach (D). Ethanol (1.0 g·kg⁻¹ BW) was administered orally to the rats 30 min after oral intubation of soybean oil (1.0 g·kg⁻¹ BW). These data were obtained 20 min after oral administration of ethanol. The dose of soybean oil-free (n=12) and 1.0 g·kg⁻¹ BW (n=12) for □ and ■, respectively. Values in parentheses indicate the ratio of the value of soybean oil=1.0 g·kg⁻¹ BW to that of soybean oil-free. *** p<0.001 (vs. Soybean oil-free). Each value is shown as M±SE.

discussion

The present result clearly showed that vegetable oils such as soybean oil and coconut oil changed plasma ethanol levels in ethanol-administered rats (Figs. 2-5). The present study also suggests that the slowing of gastric emptying may be involved in the effects of vegetable oils on plasma ethanol level in vivo. In fact, the contents and concentrations of ethanol remaining in stomach after ethanol intubation...
Fig. 9. Relation between plasma ethanol concentration and stomach content (A), ethanol concentration in stomach (B), or remaining rate of ethanol in stomach (C). Experimental conditions are as in Fig. 8. ○, control; ●, soybean oil = 1.0 g·kg⁻¹ BW; △, coconut oil = 1.0 g·kg⁻¹ BW; and ▲, linoleic acid = 0.5 g·kg⁻¹ BW. Each slope was estimated by the least-squares method for linear regression function.

Fig. 10. Relation between remaining rate of ethanol in stomach and stomach content (A) or ethanol concentration in stomach (B). Experimental conditions are as in Fig. 8. Symbols are as in Fig. 9. The slope in A was estimated by the least-squares method for linear regression function. The curve simulated in B was estimated by the least-squares method for non-linear regression function.

From these results, it is suggested that the slowing of gastric emptying was one
of the major factors that served to decrease and delay actions by vegetable oils.

As shown in Figs. 6 and 7, C18-unsaturated fatty acids also changed plasma ethanol levels. Generally, it is well accepted that various dietary components such as carbohydrates, proteins, fats, amino acids, and organic acids, delay gastric emptying (25–29). For instance, the slowing of gastric emptying by fats in food is considered to be mediated by the anions of long-chain fatty acids set free during the digestion of the triglycerides in the duodenum (25). Hunt (25) reported that ionic detergents such as fatty acids, bile acids, and chelators stimulated the duodenal membrane receptors and slowed gastric emptying through the binding of ionized calcium. Hunt (25) also reported that several ionic detergents slowed gastric emptying by decreasing the dimensions of the lateral intercellular space between the duodenal enterocytes. This space gives a signal that indirectly slows gastric emptying through nerves or hormones such as gastrin, cholecystokinin, secretin, gastric inhibitory peptide (GIP), glucagon, and vasoactive intestinal polypeptide (VIP) (30–32). On the basis of these findings, we suppose that the slowing of the gastric emptying by vegetable oils or C18-unsaturated fatty acids is in part mediated by the interaction between the anion of the fatty acids and duodenal receptors.

Generally, ethanol can be absorbed into the body through the whole of the gastrointestinal tract from the mouth to the rectum. The main regions for absorption are the duodenum and jejunum, with a lower but appreciable uptake from the stomach and the large intestine and minimal absorption from the mouth (8). On the other hand, the amount of ethanol absorbed through the gastric mucosa will be dependent, in part, on the level of retention of the alcoholic liquor in the stomach (8). If the pylorus allows the rapid movement of the alcohol through to the jejunum and duodenum, absorption through the stomach walls would be expected to represent only a minor route for ethanol to enter the body water compartments (8). Since the rate of transfer of ethanol from the stomach to the small intestine markedly influences the rate at which ethanol is absorbed, the motility of the stomach is often a major rate-limiting factor in ethanol absorption (32–34). In this case, the rate of absorption of ethanol from the small intestine depends on the rate of gastric emptying (35). Therefore, when gastric emptying is slow, the absorption of ethanol is delayed and peak plasma ethanol concentration is reduced (34).

The excretion of ethanol from the urine or expiration must be considered as other factors which affect the plasma ethanol concentration. These factors could not be determined directly in this study. However, it is indicated that the proportion of ethanol excreted or expired directly was smaller, and that there was little significance (9, 13). Therefore, these factors seem less important.

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