Effects of intravenous fructose on gastric emptying and antropyloroduodenal motility in healthy subjects

Stevens JE, Doran S, Russo A, O'Donovan D, Feinle-Bisset C, Rayner CK, Horowitz M, Jones KL. Effects of intravenous fructose on gastric emptying and antropyloroduodenal motility in healthy subjects. Am J Physiol Gastrointest Liver Physiol 297: G1274–G1280, 2009. First published October 1, 2009; doi:10.1152/ajpgi.00214.2009.—Gastric emptying (GE) of glucose is regulated closely, not only as a result of inhibitory feedback arising from the small intestine, but also because of the resulting hyperglycemia. Fructose is used widely in the diabetic diet and is known to empty from the stomach slightly faster than glucose but substantially slower than water. The aims of this study were to determine whether intravenous (iv) fructose affects GE and antropyloroduodenal motility and how any effects compare to those induced by iv glucose. Six healthy males (age: 26.7 ± 3.8 yr) underwent concurrent measurements of GE of a solid meal (100 g ground beef labeled with 20 MBq 99mTc-sulfur colloid) and antropyloroduodenal motility on three separate days in randomized order during iv infusion of either fructose (0.5 g/kg), glucose (0.5 g/kg), or isotonic saline for 20 min. GE (scintigraphy), antropyloroduodenal motility (manometry), and blood glucose (glucometer) were measured for 120 min. There was a rise in blood glucose (P < 0.001) after iv glucose (peak 16.4 ± 0.6 mmol/l) but not after fructose or saline. Intravenous glucose and fructose both slowed GE substantially (P < 0.005 for both), without any significant difference between them. Between t = 0 and 30 min, the number of antral pressure waves was less after both glucose and fructose (P < 0.002 for both) than saline, and there were more isolated pyloric pressure waves during iv glucose (P = 0.003) compared with fructose and saline (P = NS for both) infusions. In conclusion, iv fructose slows GE and modulates gastric motility in healthy subjects, and the magnitude of slowing of GE is comparable to that induced by iv glucose.

IT IS GENERALLY RECOGNIZED that, in health, gastric emptying is regulated tightly, primarily as a result of feedback inhibition generated by interaction of nutrients with the small intestine, the magnitude of which is dependent on the length and region of small intestine exposed to nutrient (36, 37), so that the overall rate of entry of nutrients into the small intestine approximates ~2 kcal/min in healthy subjects (5, 28, 36, 45). The motor correlates of the slowing of gastric emptying induced by the presence of nutrients in the small intestine include relaxation of the proximal stomach (2), suppression of antral motility (22), and stimulation of phasic and tonic pyloric contractions (23). Monosaccharides empty from the stomach more slowly than water or isotonic saline because of small intestinal feedback (17), but there may be subtle differences between them. In particular, fructose, when given as intragastric loads to monkeys (45), empties more rapidly than glucose. The slightly more rapid rate of emptying of oral fructose compared with oral glucose has also been demonstrated in humans (11, 17, 24, 52).

In addition to intraluminal mechanisms, there is evidence that plasma monosaccharide concentrations may also affect gastric motility and emptying (21, 40, 50, 51). This could potentially contribute to the discrepant effects of monosaccharides on gastric emptying. In particular, it is well established that acute hyperglycemia, induced by intravenous glucose, has major, reversible effects on gastrointestinal motor function. Marked hyperglycemia (~16–20 mmol/l) slows gastric emptying in healthy subjects (40, 51) and patients with type 1 (14, 50) and type 2 (25) diabetes, when compared with euglycemia (~4 mmol/l). Even blood glucose concentrations that are within the normal postprandial range (i.e., 4–8 mmol/l) affect gastric emptying in both healthy volunteers and uncomplicated type 1 patients (51). As with the effects of small intestinal nutrients, the slowing of gastric emptying induced by acute hyperglycemia is associated with suppression of antral pressure waves (3, 50) and their propagation (50), stimulation of basal and phasic pyloric pressure waves (13), an increase in proximal gastric compliance (20, 21), and suppression of duodenal motor activity (49). Acute hyperglycemia also affects the perception of sensations arising from the gut, including fullness and nausea (20, 21, 31, 38). In view of the well-documented effects of hyperglycemia, it is surprising that there is no information about the potential effects of intravenous fructose on gastric emptying or gastric motility.

Fructose consumption has increased markedly in the last 20–30 yr (17, 54). Fructose is sweeter than isonenergetic glucose and, as such, offers the same level of sweetness for a lower energy burden (15, 18). Fructose is used widely in the type 2 diabetic diet and has largely replaced sucrose in a number of processed foods, particularly beverages (15, 57). The glycemic response to fructose is also substantially less than to glucose (4, 9, 24, 33, 57). There are also substantial differences in the effects of oral glucose and fructose on the release of gastrointestinal hormones, including insulin (57) and the “incretin” hormones glucagon-like peptide (GLP)-1 (33, 55, 57) and glucose-dependent insulinotropic-polypeptide (GIP) (57). Fructose-induced insulin release is glucose dependent in that insulin secretion following intravenous (10) and oral (48) fructose is greater during hyper- than during euglycemia.

The primary aims of this study were to determine whether intravenous fructose affects gastric emptying and antropyloroduodenal motility and how any effects compare to those induced by iv glucose. Six healthy males (age: 26.7 ± 3.8 yr) underwent concurrent measurements of GE of a solid meal (100 g ground beef labeled with 20 MBq 99mTc-sulfur colloid) and antropyloroduodenal motility at three separate days in randomized order during iv infusion of either fructose (0.5 g/kg), glucose (0.5 g/kg), or isotonic saline for 20 min. GE (scintigraphy), antropyloroduodenal motility (manometry), and blood glucose (glucometer) were measured for 120 min. There was a rise in blood glucose (P < 0.001) after iv glucose (peak 16.4 ± 0.6 mmol/l) but not after fructose or saline. Intravenous glucose and fructose both slowed GE substantially (P < 0.005 for both), without any significant difference between them. Between t = 0 and 30 min, the number of antral pressure waves was less after both glucose and fructose (P < 0.002 for both) than saline, and there were more isolated pyloric pressure waves during iv glucose (P = 0.003) compared with fructose and saline (P = NS for both) infusions. In conclusion, iv fructose slows GE and modulates gastric motility in healthy subjects, and the magnitude of slowing of GE is comparable to that induced by iv glucose.

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rododental motility and, if so, how these effects compare to those induced by intravenous glucose.

**MATERIALS AND METHODS**

**Subjects**

Six healthy men (mean age: 26.7 ± 3.8 yr, body mass index: 26.4 ± 1.4 kg/m²) were studied. Subjects were randomly selected from volunteers who responded to advertisements posted on university notice boards. Subjects were asked to maintain a normal diet for three days before each study, and smokers were required to abstain from tobacco for at least 12 h before each study day. No subject had a history of diabetes mellitus, gastrointestinal disease or surgery, significant respiratory, cardiac, or hepatic disease, chronic alcohol abuse, gout, or epilepsy. No subject was taking medication known to influence gastrointestinal function.

Each subject provided written, informed consent prior to involvement. The protocol was approved by the Human Ethics Committee of the Royal Adelaide Hospital, and all studies were performed in accordance with the Declaration of Helsinki.

**Experimental Protocol**

Each subject underwent three randomized, single-blind studies, separated by at least three days. During each study, subjects received an intravenous infusion of either fructose or glucose (0.5 g/kg body wt dissolved in sterile water as 0.2 g/ml, for both monosaccharides) or placebo (saline 0.9% wt/vol), administered over 20 min, i.e., the total volume infused varied between subjects depending on body weight. The dose of the monosaccharides was selected on the basis of previous studies (1, 10, 12).

On each study day, subjects attended the Department of Nuclear Medicine, PET and Bone Densitometry at ~0800 h following an overnight fast (14 h for solids and 12 h for liquids). A silicone-manometric assembly (~4 mm diameter) (Dentsleeve, Adelaide, SA, Australia) was introduced into the stomach via an anaesthetised nostril and allowed to pass through the stomach and into the duodenum by peristalsis (47). The manometric catheter consisted of 16 sideholes (channels) spaced at 1.5-cm intervals, comprising six antral sideholes (channels 1–6), a 4.5-cm sleeve sensor (channel 7), two sideholes on the back of the sleeve sensor (channels 8 and 9), seven duodenal sideholes (channels 10–16), and an infusion port (the latter was not used) (47). The correct position of the catheter, i.e., with the sleeve sensor straddling the pylorus, was monitored by continuous measurement of the transmucosal potential difference (TMDP) between the most distal antral channel (channel 6; ~40 mV) and the most proximal duodenal channel (channel 10; ~0 mV). For this purpose, an intravenous cannula filled with sterile saline was placed subcutaneously in the posterior aspect of the forearm and used as a reference (47). All channels were perfused with degassed, distilled water, except for the two TMDP channels, which were perfused with degassed, isotonic saline at 0.15 ml/min. Two intravenous cannulae were inserted into antecubital veins on opposing arms, one for blood sampling and the other for intravenous infusion of fructose, glucose, or saline.

Subjects were positioned supine until the catheter was in the correct position, when they were seated with their back against a gamma camera. They then consumed the test meal, which comprised 100 g ground beef, labeled with 20 MBq 99mTc-sulfur colloid chicken liver, followed immediately by 25 ml water (to clear the esophagus of food). The meal was consumed within 5 min, and the time of meal completion was defined as t = 0 min. Immediately after the meal, the intravenous infusion (glucose, fructose or saline) was commenced for 20 min. Gastric emptying and antropyloroduodenal motility were monitored between t = 0 and 120 min.

**Measurement of Blood Glucose Concentrations**

Venous blood samples were obtained immediately before the intravenous infusion (t = −5 min) and then at 0, 5, 10, 15, 30, 45, 60, 90, and 120 min. Blood glucose concentrations were determined immediately using a portable blood glucose meter (Medisense Companion 2 meter; Medisense, Waltham, MA).

**Measurement of Gastric Emptying**

Gastric emptying was measured scintigraphically. Radioisotopic data were acquired at 1-min intervals for the first hour and at 3-min intervals thereafter. Data were corrected for subject movement, radio-nuclide decay, and γ-ray attenuation (8). Regions of interest were drawn for the total stomach, which was subsequently divided into proximal and distal stomach regions. Gastric emptying curves for total, proximal, and distal stomach regions, expressed as percent retention over time, were then derived. From the gastric emptying curves, the intragastric retention at 0, 15, 30, 45, 60, 75, 90, 105, and 120 min was derived. The amounts remaining in the stomach at 45 min were compared because blood glucose concentrations were expected to return to baseline around that time (24). The lag phase was determined visually as the time before radioactivity appeared in the duodenum (8, 26) and the time for 10% emptying.

**Measurement of Antropyloroduodenal Motility**

Pressure waves were analyzed only when the sleeve sensor was positioned correctly across the pylorus, according to previously defined TMPD criteria, and their amplitude was ~10 mmHg. Intraluodenal pressures were recorded at 10 Hz using custom software (HAD, written by Assoc. Prof. G. S. Hebbard, Royal Melbourne Hospital, Melbourne, Victoria, Australia), written in LabVIEW 3.1.1 (National Instruments, Austin, TX), and stored for subsequent analysis. Data were converted for analysis (MAD, written by Prof. C. H. Malbert, Institut National de Recherches Agronomiques, Rennes, France) and artefacts eliminated by visual inspection of each recording. Variables assessed were the following: 1) number and amplitude of antral pressure waves (waves in any of the last 3 antral side holes of amplitude >10 mmHg), 2) number and amplitude of isolated pyloric pressure waves (IPPWs) (IPPWs recorded by the sleeve sensor in the absence of a pressure wave of onset within 5 s of the pyloric wave, occurring in the antral or duodenal side holes, of amplitude >10 mmHg), and 3) number and amplitude of duodenal pressure waves (waves in any of the first 3 duodenal side holes of amplitude >10 mmHg), using custom software (written by Prof. A. Smout, Utrecht, The Netherlands). The number and amplitude of pressure waves was determined manually by visual inspection of manometric recordings, and measurements of amplitude were corrected by subtraction of baseline pressure recordings. Because of the intermittent position of the sleeve sensor, basal pyloric pressure was not recorded. The number of antral pressure waves observed in the first 30 min following intravenous glucose and fructose was too small to allow meaningful analysis of patterns of antral wave propagation.

**Statistical Analysis**

Data (blood glucose, gastric emptying, and IPPWs) were evaluated using repeated-measures ANOVA and are shown as means ± SE. The number and amplitude of both antral and duodenal pressure waves were analyzed using Wald statistics with generalized estimating equations (GEE) (35, 58) on the basis of chi-square distribution to adjust for missing values. Manometric data (IPPWs, antral, and duodenal pressure waves) were analyzed in 30-min time intervals from t = 0–120 min, with a 15-min baseline (i.e., t = −15–0 min). For the mean pressure analyses, the data were log transformed, and a normal distribution was used for the GEE. For the mean number of wave analyses, a Poisson distribution with log link was used; however, the data were overdispersed, so a negative binomial distribution with log link was used instead. A compound symmetry covariance structure was assumed for all analyses. Measurements from the last three sideholes in the antrum were grouped for the analysis of number
and amplitude of antral pressure waves. Likewise, measurements from the first three sideholes in the duodenum were grouped for the analysis of number and amplitude of duodenal pressure waves. For analysis of the number and amplitude of antral and duodenal pressure waves, data are expressed as means with upper and lower 95% confidence interval (95% CI) limits. Student’s t-tests were used to assess paired comparisons. A P value <0.05 was considered significant in all analyses.

RESULTS

All subjects tolerated the saline and glucose studies well. During fructose infusion, four subjects volunteered epigastric discomfort (mild and transient), and, of these, two reported nausea.

Blood Glucose

Baseline blood glucose did not differ between study days (saline 5.4 ± 0.3 mmol/l; glucose 5.5 ± 0.3 mmol/l; fructose 5.2 ± 0.2 mmol/l) (Fig. 1). Mean blood glucose (calculated using 30-min blood glucose levels from t = 0–120 min) was greater following glucose (7.5 ± 0.3 mmol/l) compared with both saline (5.5 ± 0.2 mmol/l; P = 0.0008) and fructose (5.3 ± 0.2 mmol/l; P = 0.0002) infusions, with no difference between saline and fructose (P = 0.47). During glucose infusion, the peak blood glucose concentration was 16.4 ± 0.6 mmol/l, and the time to peak was 20 ± 3 min. At t = 120 min, blood glucose was less after glucose compared with both saline (P = 0.0005) and fructose (P = 0.009) infusions, with no difference between saline and fructose (P = 0.35).

Gastric Emptying and Intragastric Distribution

The overall gastric emptying pattern approximated a linear function after an initial lag phase (Fig. 2A). The lag phases, as measured by the arrival of food in the duodenum, were 18.7 ± 2.9 min, 34.7 ± 7.3 min, and 35.3 ± 9.8 min for saline, glucose, and fructose studies, respectively (P = NS for all comparisons), although there were trends for the lag phase on the saline day to be shorter than the glucose (P = 0.07) and fructose (P = 0.09) days. Similarly, there was no significant difference in the time for 10% of the meal to empty between either saline (35.5 ± 5.0 min) or fructose (47.8 ± 11.9 min) with glucose (52.7 ± 7.3 min) although the time was shorter (P = 0.04) after saline compared with glucose. There was a treatment-by-time interaction (P = 0.0001) for total gastric emptying; gastric emptying from the total stomach was slower with both glucose and fructose compared with saline between t = 45–120 min (P < 0.005 for all), with no significant difference between glucose and fructose. From t = 0–45 min, there was a treatment-by-time interaction (P = 0.0003) for total gastric emptying; at t = 45 min, gastric emptying was slowest with glucose compared with both fructose (P < 0.05) and saline (P < 0.0001) although fructose was slower than saline (P < 0.0001). In five of the six subjects, gastric emptying was substantially slower after intravenous fructose than after intravenous saline, and, in the remaining one, gastric emptying was comparable. Between t = 45 and 120 min, there was no difference in total gastric emptying between the three treatments: saline 55.7 ± 2.7%, glucose 58.2 ± 6.3%, and fructose 53.2 ± 6.3% (P = NS for all).
There was a treatment-by-time interaction ($P = 0.0001$) for proximal gastric emptying (Fig. 2B); retention in the proximal stomach was greater with glucose compared with saline (from $t = 15–90$ min; $P < 0.05$ for all) and greater with fructose compared with saline (from $t = 15–105$ min; $P < 0.01$), with no difference between glucose and fructose.

There was a treatment-by-time interaction ($P = 0.0001$) for distal gastric emptying (Fig. 2C); retention in the distal stomach was initially greater after saline than both glucose (from $t = 15–30$ min; $P < 0.002$) and fructose (from $t = 15–45$ min; $P < 0.01$), with no difference between glucose and fructose (from $t = 15–45$ min). Retention in the distal stomach was subsequently less with saline compared with both glucose (from $t = 75–120$ min; $P < 0.001$) and fructose (from $t = 90–120$ min; $P < 0.05$), with no difference between glucose and fructose (between $t = 105$ and 120 min).

**Antropyloroduodenal Manometry**

**Antral pressure waves.** There was no significant difference in the number of antral waves at baseline between the three studies [saline: 4.9 (95% CI: 1.7, 14.6); glucose: 2.8 (95% CI: 2.4, 3.3); fructose: 1.9 (95% CI: 1.0, 3.9)] (Fig. 3A). There was a significant treatment-by-time interaction between the three infusions ($P < 0.0001$). There was a significant increase in the number of antral waves following the meal during saline, glucose, and fructose ($P < 0.0001$ for all) infusions. The rise in the number of antral waves from baseline occurred promptly following meal ingestion (i.e., $t = 0–30$ min) during infusion with saline ($P = 0.02$) but not during glucose ($P = 0.21$) or fructose ($P = 0.65$), i.e., the stimulation of antral waves was attenuated by infusion of glucose and fructose. In contrast, the rise in the number of antral waves was not evident until $t = 30–60$ min after fructose ($P < 0.0001$) and $t = 90–120$ min after glucose ($P < 0.0001$). Over the 120-min period, the number of antral waves was greater after saline compared with both fructose ($P = 0.002$) and glucose ($P < 0.0001$) infusion and greater for fructose compared with glucose ($P = 0.0004$).

The amplitude of antral pressure waves at baseline was higher for saline [33.9 mmHg (95% CI: 24.8, 46.4)] than glucose [17.5 mmHg (95% CI: 11.7, 26.2)] ($P = 0.01$) and tended to be greater compared with fructose [20.6 mmHg (95% CI: 16.4, 25.8)] ($P = 0.09$), with no significant difference between fructose and glucose ($P = 0.49$). Following meal ingestion, there was no significant difference in the amplitude of antral pressure waves between the three infusions (data not shown).

**IPPWs.** There was no difference in the number of IPPWs at baseline between the three studies (saline 2.0 ± 0.6; glucose: 3.7 ± 1.8; fructose: 1.3 ± 1.0) (Fig. 3B). There was a significant ($P = 0.0001$) time effect for the duration of the study. There was an increase in the number of IPPWs from baseline to 30 min immediately following meal ingestion (i.e., $t = 0–30$ min) during infusion with glucose ($P = 0.003$) but not with saline ($P = 0.21$) or fructose ($P = 0.06$), i.e., the number of IPPWs was increased during infusion of glucose but not fructose or saline. The rise in the number of IPPWs was not evident until $t = 30–60$ min for both saline ($P = 0.018$) and fructose ($P = 0.0002$). For the period $t = 0–120$ min, there was no treatment effect on the number of IPPWs.

There was no significant difference in the amplitude of IPPWs at baseline between the three treatments (data not shown). There was a significant ($P = 0.0001$) time effect for the amplitude of IPPWs with the three treatments for the duration of the study. There was a significant increase in the amplitude of IPPWs from baseline immediately following meal ingestion (i.e., $t = 0–30$ min) during infusion with saline ($P = 0.02$), glucose ($P = 0.0005$), and fructose ($P = 0.04$). The magnitude of the increase in amplitude at $t = 0–30$ min was greater with glucose compared with fructose ($P = 0.05$) but not saline ($P = 0.37$), and there was no difference in the amplitude of IPPWs at $t = 0–30$ min between saline and fructose ($P = 0.28$) (data not shown).

**Duodenal pressure waves.** There was no significant difference in the number of duodenal waves at baseline between the
three infusions [saline: 11.1 (95%CI: 5.4, 22.7); glucose: 7.8 (95%CI: 4.4, 13.7); and fructose: 6.83 (95%CI: 3.35, 13.95)] (Fig. 3C). There was a significant increase in the number of duodenal waves from baseline following meal ingestion (i.e., t = 0–30 min) during infusion with saline (P < 0.0001) and glucose (P < 0.0001) but not fructose (P = 0.27). However, for the 120-min period, the number of duodenal waves was not significantly different between the three infusions.

There was no significant difference in the amplitude of duodenal pressure waves at baseline or after the meal between the three infusions (data not shown).

**DISCUSSION**

This study establishes that, when given intravenously, fructose has the capacity to slow gastric emptying and modulate antropyloroduodenal motility in healthy subjects and that the magnitude of these effects is comparable to those induced by an identical intravenous glucose load. In particular, intravenous administration of fructose resulted in a transient slowing of gastric emptying and intragastric distribution of a solid meal, associated with suppression of antral and duodenal pressure waves and (nonsignificant) stimulation of IPPWs.

The demonstrated effects of intravenous glucose, resulting in peak plasma glucose concentrations of ~16 mmol/l, on gastric emptying (40, 50, 51) and antropyloroduodenal motility (13, 49, 50) are consistent with previous reports. As has been shown, these effects were transient, indicating that they were secondary to hyperglycemia. The effects of intravenous glucose and fructose to slow gastric emptying evident during the initial phase of emptying, which as is to be expected for the solid meal studied, were dominated by the lag phase (8). This was anticipated given that the infusions were administered over 20 min, immediately after meal consumption, and that effects on emptying and motility would only be expected when blood concentrations of glucose (13, 40) and fructose were elevated. Hence, the timing and duration of the infusions diminished the capacity to observe any effect on the postlag emptying slope. Because patterns of emptying from the proximal stomach closely paralleled emptying from the total stomach, it is possible that differences in intragastric distribution affected motility. This appears, however, unlikely given that acute hyperglycemia has been shown to stimulate pyloric (13) and suppress antral (19) motility in the fasted state and that differences in distal stomach content were modest. The mechanism(s) mediating the effects of hyperglycemia on gastric motility are poorly defined but appear to involve nitric oxide pathways (34). Glucose-dependent neurons are also known to be present in the myenteric plexus (39) and central nervous system (44). The effect of acute hyperglycemia, to slow gastric emptying, is apparently not mediated by changes in plasma osmolality (46). Although plasma fructose was not quantified, the dose used was identical to that in a previous study (10), and the effects of fructose on gastric emptying/motility are probably, like glucose, dependent on the plasma concentrations. As expected, intravenous fructose had no effect on plasma glucose (1, 10, 56), nor would it be expected to affect insulin secretion (10). Intravenous glucose has no effect on the secretion of GLP-1 or GIP, which are released by enteral glucose and account for the so-called “incretin effect” (43). Hence, it appears that both glucose and fructose have the capacity to slow gastric emptying both as a result of their interaction with the small intestine (11, 17, 24, 52) and also the elevation of plasma monosaccharide concentrations. Accordingly, although we did not observe differences in the magnitude of the delay in gastric emptying when equivalent doses of monosaccharides were given intravenously, this does not exclude a difference following oral administration, particularly because ingested fructose is absorbed from the intestine much more slowly (via glucose transporter 5) than glucose and, hence, results in lower plasma concentrations than oral glucose (via sodium-glucose cotransporter 1) (30, 53). It should be recognized that we studied only a relatively small number of subjects, and the possibility that there are modest differences in the effects of intravenous fructose and glucose, which may potentially contribute to the slightly more rapid gastric emptying of oral fructose compared with oral glucose, cannot be excluded (11, 17, 24, 45, 52) although this may well be explicable by the differential effects on gut hormone release, particularly GLP-1 (33, 47, 57). Four subjects experienced transient, mild epigastric discomfort during intravenous fructose infusion. This has been reported during rapid fructose infusion in much higher doses (0.5 g/kg in 5 min to 1.5 g/kg in 60 min) (12), and its cause remains uncertain. In the present study, the severity and duration of discomfort did not compromise the completion of the experiments, and the effects of fructose on gastric emptying appeared consistent in both symptomatic and asymptomatic subjects. While in view of our observations that it would be of interest to evaluate the effects of different, particularly lower doses of fructose and glucose, it should be recognized that the blood glucose concentrations observed after intravenous glucose (peak ~16 mmol/l) are comparable to those observed in uncomplicated type 2 diabetes patients after oral 50-g or 75-g glucose loads (32). Because of the substantial effect of the menstrual cycle on gastric emptying (6), we elected to study only males. Hence, our observations should be extrapolated to females circumspectly.

That fructose has the capacity to slow gastric emptying as a result of its plasma levels is not surprising given that intravenous administration of high-caloric nutrients (parenteral nutrition) has been reported to delay solid (41) and liquid (7) gastric emptying in humans. It has been suggested that this reflects the stimulation of gastric acid secretion by the amino acid content of the parenteral feed (29, 42), with a concomitant suppression of pancreatic and biliary secretion and a reduction in the buffering capacity of the duodenum (7). Energy intake may also be suppressed during parenteral nutrition, possibly as a result of slower gastric emptying (27). Intravenous administration of high-dose amino acids has also been shown to decrease antral motility (16). As with fructose, the mechanisms mediating these effects remain to be determined.

In conclusion, in healthy subjects, intravenous fructose has the capacity to slow gastric emptying and modulate gastric motility; the magnitude of the delay in gastric emptying appears comparable to that induced by intravenous glucose.

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